Specialty Skills in Emergency Surgery and Trauma (CT1–2)

Participant handbook
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We would like to thank Mölnlycke Health Care and Nycomed for their support of the Emergency Surgery and Trauma portfolio.

We are very grateful to CareFusion, Edwards Life Sciences, Ethicon, Smith & Nephew and WL Gore & Associates who have provided equipment in support of the Specialty Skills in Emergency Surgery and Trauma course.
The Specialty Skills Project (SSP) is one of the most recent and progressive initiatives developed at The Royal College of Surgeons of England with the aim of enhancing surgical training in the United Kingdom. It has been in production for several years. During that time it has undergone many changes, including to its title. From the very start, however, it aimed to create practical courses for each of the surgical specialties, to tie in with both the Modernising Medical Careers (MMC) initiative and the new surgical curriculum. The emphasis has always been on producing hands-on practical courses (accompanied by a handbook and DVD) with very few – if any – didactic lectures. Obviously, this was an enormous and ambitious task, but now it is complete, I believe it has been worth the effort.

One of the challenges was to define and categorise the separate surgical subspecialties within the broad field of surgery. We also had to decide which specialties should have their own distinct SSP course, and where to place other subspecialties that have less clearly defined boundaries. Countless discussions and meetings were held, as a result of which a list was drawn up that mirrored as closely as possible the subspecialties recognised by the different Specialist Advisory Committees. Now that this task has been completed, cross-referencing has been greatly facilitated between the new curricula and other existing courses at the College.

There has been a great deal of confusion and change within the nomenclature of training grades. ‘Core’ training (CT) is used for the early years (usually the first two years) of specialty training (ST). As a generic model, a surgical trainee now passes through Foundation Training (F1–2), Core Training (CT1–2) and Specialty Training (ST3–8). This differs from the ‘run-through’ prototype recently seen in practice, which comprised Foundation (F1–2) and Specialty (ST1–8) training. The new terminology has been agreed with, and approved by, the Postgraduate Medical Education and Training Board (PMETB) and I very much hope there will be no further adjustments. It is important to recognise that the SSP is aimed at trainees who are entering a particular subspecialty, although it is well known that the required levels of experience for entry differ between specialties. Clearly, it is impossible to be completely prescriptive when defining specific fields of expertise, but we have tried to target the appropriate levels within the SSP project.

The different courses evolved at different rates over the years. Some were based on introductory courses that formed part of the College portfolio; others were created entirely from scratch. Working parties were assembled, bringing together tutors from the Education Department and surgical consultants. They collaborated on every course, including the one you are about to attend. In addition, advice was sought from a professional medical educator at all stages of development, both to establish validated learning outcomes and to ensure that the delivery of each course was educationally robust.
Some of the handbooks have been written and edited entirely by the course convenor, and many include contributions from various faculty members, therefore their content reflects learning on the course. Furthermore, each handbook has been peer-reviewed by consultants and senior trainees who were nominated by the president of each Specialist Society, thus ensuring that the content of each course is factually accurate and pitched at an appropriate level. Most of the illustrations, photographs and line-drawings were specifically produced or commissioned, and some were reproduced, with permission, from published textbooks and other sources. For completeness, most of the handbooks provide suggestions for further reading and identify any useful current websites, dovetailing neatly with the College’s other educational products.

This major undertaking has involved an enormous amount of hard work and organisation, and many people have played a part in its success. To them, I offer my unreserved gratitude. The greatest thanks go to Karen Day who, as project coordinator, worked relentlessly to meet endless deadlines. Without her, this project would never have come to fruition. Although some frustrations were inevitable, it was a pleasure to be involved with a project like this and to work with Karen and all the specialty leads and other members of the team. I very much hope this momentum continues into further, more advanced, phases of the project.

I am sure that these SSP courses will fully achieve their intended aims, and that the accompanying handbooks and DVDs will prove invaluable for your own preparation and revision, and for your future career in surgery.

I welcome any feedback you may have.

Jonathan Hyde

June 2009
This course aims to deliver hands-on training in Emergency and Trauma Surgery for Core and Specialist Trainees at the early to intermediate stages of training. The course is mapped to the ISCP and has been developed jointly by the Education Department at The Royal College of Surgeons of England and the course faculty. This handbook will be a useful resource for the participants, not only during, but also after the course as a reference text.

We have been successful in enlisting the help and support of contributors with both national and international reputations in the field of Emergency and Trauma Surgery and I would like to thank them for their efforts in completing this handbook. Our special thanks to Karen Day, the Publishing Editor and her assistant Katerina Georgiou for managing this project so successfully; to Ruth Warne, Specialty Programmes Coordinator, and her colleagues who have worked very hard to put the course together. Michelle Kimberlin and her colleagues in the surgical resources department deserve a special mention as the course could not run without their input.

We hope you enjoy the course.

Alastair Simpson and Adam Brooks
August 2009
• Wear surgical gloves of the correct size when handling tissues.
• Never handle sharps directly.
• Hand sharp instruments to assistants in a safe manner, preferably in a kidney dish or suitable container.
• Remember that safety of the assistant is your responsibility too.
• Immediately dispose of used needles or blades in the sharps container provided.
• Dispose of excised tissue in appropriate containers.
• Keep the operative field tidy, without extraneous instruments or equipment lying around. Never leave sharps lying unattended in or near the operative field.
• Check the integrity of each instrument yourself before you use it. This is particularly important for electrical equipment such as diathermy and laparoscopic instruments.
• Set the operating table at the correct height. Whether sitting or standing, your horizontal forearm should be approximately the same height as the operative field, to minimise fatigue (see Figure 1).

Adapted from *Intercollegiate Basic Surgical Skills course Participant handbook* with permission.
Introduction

In 2005/06 there were over half a million emergency general surgical admissions in the United Kingdom requiring over 300,000 hospital bed days. This included 33,000 appendicectomies, around 15,000 cases of acute pancreatitis and over 3000 cases of diverticular perforation. Many of these patients were critically ill and during this period 990 died of acute pancreatitis, 1671 died of either a perforated or a bleeding duodenal ulcer, and 1934 died from complications of diverticular disease.

Initial approach

It is important to differentiate between those patients who are acutely unwell and require synchronous assessment and resuscitation and those who are not physiologically compromised, in whom a more measured approach can be taken. A standard approach (Figure 2) with correct initial assessment allows prioritisation of clinical management, investigations and communication.

Occasionally, signs of impending decompensation may be subtle and it is better to fully evaluate rather than to under-appreciate a patient who rapidly deteriorates.

Resuscitation

All emergency surgery patients require resuscitation: this is not limited to those in the emergency room or the intensive care unit. Assessment of ABC (airway, breathing and circulation) can be done rapidly, and in the unstable patient resuscitation will need to be undertaken at the same time as management procedures.

Resuscitation maybe as simple as providing oxygen, inserting an intravenous line and giving intravenous fluid to replace losses, or it may require the full spectrum of advanced procedures, including airway management, central access and blood transfusion.

Patients with different pathologies will have diverse requirements. For example, in small bowel obstruction or pancreatitis there will be significant fluid losses that will require aggressive early fluid resuscitation, whereas in a
large acute gastrointestinal bleed early transfusion and correction of coagulopathy is vital, and in severe sepsis the surviving sepsis care bundles should be commenced. The response to resuscitation should be monitored and resuscitation should be tailored appropriately to the individual patient.

Communication and senior input is important when looking after seriously ill patients. Early referral to critical care and/or an outreach team is valuable. Emergency surgery is an integral part of resuscitation in some patients.

Investigations

Investigations need to be focused towards the initial diagnosis to confirm or rule out the working diagnosis, or as appropriate work-up for anaesthesia. “Routine” investigations should not be performed unless they can be justified as they may add expense and unnecessary delay.

Initially start with basic urine and blood tests and progress to plain radiological tests, followed by advanced radiological investigations to confirm the diagnosis and plan surgery if required.

Remember that investigations are valuable but should not delay definitive management in an unstable patient.

Initial management

Continuously re-evaluate the patient at each stage in the assessment process and revise the management plan according to changes in his or her physiological condition or results.

Ask the following questions:
• Does the patient require further investigation?
• Does the patient require further resuscitation?
• Does the patient require a different treatment strategy (conservative or surgical)?

Document a management plan for every emergency surgery patient and inform seniors and nursing staff. The plan may be straightforward, as in Box 1.

<table>
<thead>
<tr>
<th>Box 1 Plan for diagnosis of perianal abscess</th>
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<tbody>
<tr>
<td>Incision and drainage under general anaesthetic</td>
</tr>
<tr>
<td>Book for theatre ✓</td>
</tr>
<tr>
<td>Consent ✓</td>
</tr>
<tr>
<td>Nil by mouth until surgery</td>
</tr>
<tr>
<td>Analgesia ✓</td>
</tr>
<tr>
<td>Antibiotics ✓</td>
</tr>
<tr>
<td>Low molecular weight heparin ✓</td>
</tr>
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</table>

But in critically ill patients the plan may be more complex, as shown in Box 2.

<table>
<thead>
<tr>
<th>Box 2 Plan for diagnosis of intra-abdominal sepsis, colonic perforation/severe sepsis and acute renal failure</th>
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<tbody>
<tr>
<td>Urgent referral to critical care outreach team</td>
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<tr>
<td>High-flow oxygen</td>
</tr>
<tr>
<td>Arterial blood gases</td>
</tr>
<tr>
<td>Intravenous access ✓</td>
</tr>
<tr>
<td>Blood cultures ✓</td>
</tr>
<tr>
<td>Analgesia</td>
</tr>
<tr>
<td>Intravenous fluid resuscitation</td>
</tr>
<tr>
<td>Urinary catheter (maintain urine output 0.5 mL/kg/h)</td>
</tr>
<tr>
<td>Urgent CT scan ✓</td>
</tr>
<tr>
<td>Book theatre</td>
</tr>
<tr>
<td>Consent, including stoma</td>
</tr>
</tbody>
</table>

Update the plan frequently and inform staff and seniors of any change. Do not be reluctant to ask for help from seniors and consultants. Be clear and concise – it may be
worth planning what you are going to say before you make the call. If it is simply to keep them informed, tell them that is why you are calling; if you need help with assessment and resuscitation, make that clear.

**Operative management**

Up to 50% of emergency patients will require surgery to address their pathology. Ensure that the appropriate preoperative/pre-anaesthesia work-up has been performed and discuss the case with the anaesthetist. Book the case with theatre, ensuring that any special information (eg regarding hepatitis B or morbid obesity) is clearly communicated with the theatre team and the appropriate urgency category is used. Consent needs to be taken by a surgeon capable of performing the procedure and the operative side must be marked. Continue to liaise with critical care or outreach team regarding the preoperative and postoperative care. Take every opportunity to get into the operating room with the patient you have seen and assessed.

**LEARNING POINTS: Initial approach**

- A standard approach to emergency surgery patients provides a framework upon which to base resuscitation, investigations and management decisions.
- Information needs to be gathered efficiently and appropriate resuscitation and management commenced.
- In the unstable patient resuscitation will need to be undertaken at the same time as management procedures.
- Communication with seniors and the whole team is vital (Figure 3).

**Further reading**


![Figure 3: The emergency surgery communication circle.](image-url)
Introduction
Approximately 7% of people will develop acute appendicitis at some point in their lifetime and appendicectomy is the most common emergency surgical operation, with over 40,000 being performed each year in England alone. Although generally considered a “benign” disease, complication rates following appendicitis can be high (up to 30% in some series). In 2005, 118 patients in England and Wales died of acute appendicitis; two of them were teenagers and three were in their 20s, so the need for careful assessment and rapid resuscitation must always be borne in mind.

History and examination
The classic picture of abdominal pain in appendicitis is that it starts in the centre of the abdomen and is aching in nature. It then migrates to the right iliac fossa and becomes sharp in nature. However, many patients present with a different pattern of pain because of the variable position of the appendix. A systematic review showed that the features in the history that are most predictive of appendicitis are:

- a relatively short history;
- fever;
- pain migration.

Other important symptoms include:

- malaise;
- loss of appetite;
- nausea or vomiting;
- diarrhoea (if the appendix tip lies against the bowel).

Atypical presentations to remember include children who are generally irritable and off their food, and confused, shocked, elderly patients. Although pain is the usual presenting feature, it is not absolutely universal, especially at the extremes of age.

It is essential to think about other potential diagnoses. If the patient is female, a full gynaecological history (especially the date of the last menstrual period) should be taken. In older patients it is important to consider the possibility of malignancy. Diabetic ketoacidosis, acute pancreatitis and a perforated peptic ulcer can all mimic acute appendicitis in their early stages, so these too should be considered. The differential diagnosis of suspected appendicitis is shown in Table 1.

On examination, the patient may potentially be septic and shocked, especially if they have presented late, or are elderly or immunocompromised (eg diabetics).

The systematic review mentioned earlier identified the following examination features as having the highest specificity and sensitivity for acute appendicitis:

- temperature over 37.7°C;
- localised (rather than diffuse) tenderness;
- indirect tenderness (where the pain is worst at the point of maximal tenderness when the patient is palpated in the left iliac fossa; also known as Rovsing’s sign);
Specialty Skills in Emergency Surgery and Trauma (CT1–2)

Table 1: Differential diagnosis of suspected appendicitis. Those that can cause the patient to deteriorate rapidly are shown in italics. Adapted from Humes D.J., Simpson J. Acute appendicitis BMJ 2006;333:530–534

<table>
<thead>
<tr>
<th>SURGICAL</th>
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<tbody>
<tr>
<td>Perforated colonic tumour</td>
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<tr>
<td>Pancreatitis</td>
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<tr>
<td>Perforated peptic ulcer</td>
</tr>
<tr>
<td>Intestinal obstruction</td>
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<tr>
<td>Mesenteric adenitis</td>
</tr>
<tr>
<td>Diverticulitis</td>
</tr>
<tr>
<td>Acute cholecystitis</td>
</tr>
<tr>
<td>Rectus sheath haematoma</td>
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<tr>
<td><strong>GYNAECOLOGICAL</strong></td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
</tr>
<tr>
<td>Ruptured ovarian follicle</td>
</tr>
<tr>
<td>Salpingitis/pelvic inflammatory disease</td>
</tr>
<tr>
<td>Torted ovarian cyst</td>
</tr>
<tr>
<td>Mittelschmertz</td>
</tr>
<tr>
<td><strong>MEDICAL</strong></td>
</tr>
<tr>
<td>Diabetic ketoacidosis</td>
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<tr>
<td>Gastroenteritis</td>
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<tr>
<td>Terminal ileitis</td>
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<tr>
<td>Pneumonia</td>
</tr>
<tr>
<td><strong>UROLOGICAL</strong></td>
</tr>
<tr>
<td>Ureretic colic</td>
</tr>
<tr>
<td>Pyelonephritis</td>
</tr>
<tr>
<td>Urinary tract infection</td>
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- rebound or percussion tenderness;
- guarding.

Investigations

The diagnosis of acute appendicitis remains essentially a clinical one, but certain investigations are useful in making the diagnosis or excluding others on the differential list. These are summarised below.

*Remember that all women of child-bearing age presenting with abdominal pain are assumed to have a ruptured ectopic pregnancy – until proven otherwise.*

Therefore an urgent urinary test for β-human chorionic gonadotropin (hCG) is essential.

**Blood tests**

A raised white cell count, neutrophilia and a raised C-reactive protein (CRP) are most predictive for appendicitis. Amylase and blood glucose should also be checked to investigate for pancreatitis and diabetic ketoacidosis.

**Urine**

White cells and protein may well be due to bladder irritation by an inflamed retrocaecal appendix – they should not be assumed to indicate a urinary tract infection.

**Radiology**

**Ultrasound:** This is non-invasive, cheap and easily available and involves no doses of radiation. However, it is operator dependent and its sensitivity and specificity are only moderate (approximately 86% and 81%, respectively), so it can neither exclude nor confidently predict acute appendicitis. It is most useful in stable female patients to help exclude gynaecological pathology.

**CT scanning:** This has a higher sensitivity and specificity (around 94% and 95%, respectively) and it enables imaging of the rest of the abdomen to look for other diagnoses.
appendicitis

(eg a caecal tumour or diverticulitis). However, it carries a significant radiation dose and, although the sensitivity is high, 1 in 20 patients with appendicitis will have a negative CT. It should be considered in patients with suspected appendicitis in whom the diagnosis is unclear and cannot be confirmed by clinical and laboratory findings.

Initial management

As discussed in the introduction above, some patients with acute appendicitis can be extremely septic and unwell, particularly if they are immunocompromised. Therefore, the careful systematic approach advocated in this book is essential.

Analgesia

Analgesia should be given as soon as possible – the hypothesis that analgesia may mask the pain and lead to an incorrect diagnosis has no evidence base and is a myth. Patients should be prescribed intravenous fluids and thromboprophylaxis and kept “nil-by-mouth” until reviewed by a senior.

Antibiotics

Perioperative antibiotics have been shown to reduce the wound infection and intra-abdominal abscess rate although no consensus has been reached about the best regimen. Unlike analgesia, they potentially mask the examination signs, therefore they should not be started until the decision to operate has been made by a senior. There is no good evidence that antibiotics are a feasible alternative treatment in themselves; the only treatment for appendicitis remains surgery.

The operation

Timing of the operation

After the first 36 hours after the onset of symptoms, the risk of perforation is 16–36% and increases by approximately 5% in every 12-hour period. It is safe to wait and observe stable patients in whom the diagnosis is uncertain, but once the diagnosis has been made surgery should be performed as soon as possible and further delays should be avoided.

Operative approach

Compared to the open approach, laparoscopic appendicectomy has been associated with a reduced wound infection rate, a shorter hospital stay and a faster return to work – but there is a higher rate of intra-abdominal abscesses. However, the quality of the evidence is poor and definite conclusions cannot be made. Many units favour the laparoscopic approach in women in particular because of the ability to perform a diagnostic examination first.

Appendicitis during pregnancy

Perforated appendicitis is associated with a fetal loss rate of 20–35% and a maternal mortality of about 4%. It is therefore essential to consider and exclude the diagnosis early in any pregnant woman presenting with abdominal pain and/or systemic illness.

Diagnosis can be difficult because as the uterus grows, the appendix moves progressively superolaterally so that by late pregnancy it may be as high as the right upper quadrant and therefore mimic gall-bladder pain. In addition, as the uterus grows, the two layers of peritoneum become separated. This reduces the somatic sensation of pain and the ability to localise pain on examination. The classic features of sharp pain with localised guarding may therefore not be present. The imaging modality of choice is still ultrasound, but if the diagnosis remains unclear an urgent MRI scan should be performed. CT should be avoided because of the radiation dose.
LEARNING POINTS: Appendicitis

- Appendicitis is not a benign disease – young people still die of it and complications are common.
- All women of child-bearing age with abdominal pain have a ruptured ectopic pregnancy until proven otherwise, so an urgent β-hCG is essential.
- The diagnosis remains a clinical one – investigations are useful but they cannot always exclude or confirm the diagnosis.
- Once the diagnosis has been made, do not delay surgery because the complication rate rises with increasing length of time from symptom onset.
- All patients with a diagnosis of appendicitis should have antibiotics but they are not a substitute for surgery.

Further reading

Introduction
Appendicitis is one of the commonest surgical emergencies. The diagnosis remains clinical and the treatment remains surgical. Increasingly, surgeons are utilising laparoscopic techniques. However patient and technical factors may lead to conversion, and therefore an understanding of the open approach is imperative.

Step 1
- Palpation of the abdomen in an anaesthetised patient may identify an appendix mass.
- A Lanz incision is made over McBurney’s point.
- Subcutaneous fat is dissected to expose the external oblique muscle, which is divided in the line of the fibres.
- The internal oblique muscle can be split with blunt dissection.

Step 2
- The peritoneum can be picked up by clips, ensuring that there is nothing beneath, and divided to allow entry to the peritoneal cavity.
- Any turbid fluid or pus can be sampled and sent for microscopy and culture.
- Try to locate the base of the appendix at the point of convergence of the taenia coli.

Step 3
- Once identified, the appendix mesentery containing the appendicular artery should be clipped, divided and tied with an absorbable braided suture (Vicryl™).
• The base of the appendix is crushed with a heavy clip and the clip is placed slightly higher on the appendix.

**Step 4**
- The base of the appendix is suture ligated.
- The appendix is then divided under the attached clip with a scalpel blade and the suture is cut.
- Some surgeons advocate burying the appendix stump using a Z-stitch or purse-string, but this is not necessary.
- Return the caecum to the peritoneal cavity.

**Step 5**
- Once any remaining fluid has been sucked out the wound should be closed in layers.
- The edges of the peritoneum are identified and picked up with up to four clips. The peritoneum is then closed using a continuous 3/0 absorbable suture.
- The internal oblique muscle fibres can be loosely approximated with some interrupted stitches.

**Step 6**
- The external oblique defect must be securely repaired. This is done with a continuous 3/0 absorbable suture.
Step 7

- The skin can be closed with a continuous subcuticular absorbable suture.
- If the wound has been highly contaminated then consider closing with an interrupted suture or skin clips.
Upper gastrointestinal haemorrhage

Introduction
Acute upper gastrointestinal (GI) haemorrhage leads to approximately 2500 new admissions each year in the UK. It has a mortality rate of 11% in patients admitted to hospital and rises to 33% in patients who are already inpatients.

Bleeding from the upper gastrointestinal tract presents with a range of symptoms, including haematemesis, melaena and haematochezia. Depending on the size of the bleed there may be symptoms and signs of haemodynamic instability. Biochemically, a disproportionate rise in serum urea may be detected, due to the increased uptake and metabolism of amino acids that results from haemoglobin breakdown.

Risk factors
Risk factors for bleeding may be present in the patient’s history (e.g., use of non-steroidal anti-inflammatory drugs (NSAIDs) or varices) but even in the absence of significant information or clinical signs upper gastrointestinal bleeding should always be suspected in the unstable patient with an unexplained drop in haemoglobin.

Causes
The most common cause is peptic ulceration (35%). The ulceration is an inflammatory process, with simple oozing caused by damage to submucosal vessels and more severe bleeding caused by the ulcer eroding into an artery. The majority of peptic ulcer bleeding will stop spontaneously, but ulcers with a diameter of more than 1 mm are more likely to require endoscopic or surgical intervention. The location of the ulcer also influences its natural history: ulcers on the posterior wall of the duodenum involving the gastroduodenal artery or on the lesser curve of the stomach are more likely to re-bleed and require surgical intervention.

Bleeding from a Mallory–Weiss tear is caused by retching or vomiting and is usually self-limiting, although occasionally endoscopic intervention is required.

Bleeding from oesophageal or gastric varices is the other important cause of upper gastrointestinal bleeding. It should be suspected in patients with a history or peripheral stigmata of liver disease. Bleeding from oesophageal or gastric varices is often severe and a third of patients will die. Prognosis is related to the severity of the underlying liver disease rather than the severity of the bleed.

Other rare causes of upper gastrointestinal bleeding are listed in Table 2.

Risk factors for death and the Rockall scoring system
Rockall’s 1995 prospective, multicentre audit of upper gastrointestinal haemorrhage admissions in the UK identified patient, clinical and endoscopic factors that predicted death. This led to the development of an eponymous scoring system known as the Rockall score (see Table 3).
### Table 2: Causes of upper gastrointestinal haemorrhage dependent on anatomical location

<table>
<thead>
<tr>
<th>OESOPHAGUS</th>
<th>STOMACH</th>
<th>DUODENUM</th>
<th>SMALL BOWEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oesophagitis</td>
<td>Peptic ulcer</td>
<td>Peptic ulcer</td>
<td>Peptic ulcer</td>
</tr>
<tr>
<td>Barrett’s ulcer</td>
<td>Gastritis</td>
<td>Aortoenteric fistula</td>
<td>Crohn’s</td>
</tr>
<tr>
<td>Varices</td>
<td>Dieulafoy’s lesion</td>
<td>Neoplasia</td>
<td>Meckel’s</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Varices</td>
<td>Haemobilia</td>
<td>Neoplasia</td>
</tr>
<tr>
<td>Mallory–Weiss tear</td>
<td>Vascular malformation</td>
<td>Post ERCP</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neoplasia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ERCP: endoscopic retrograde cholangiopancreatography.

### Table 3: The Rockall scoring system (the need for endoscopic findings to accurately measure the Rockall score precludes its use in early risk stratification). Each variable is scored and the total score calculated by simple addition

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>SCORE</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Systolic BP &gt;100, pulse &lt;100</td>
<td>(systolic BP &gt;100, pulse &gt;100)</td>
<td>(systolic BP&lt;100, pulse &gt;100)</td>
<td>(systolic BP&lt;100, pulse &gt;100)</td>
</tr>
<tr>
<td>Shock</td>
<td>Shock</td>
<td>Tachycardia</td>
<td>Hypotension</td>
<td>–</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>Nil major</td>
<td>–</td>
<td>Cardiac failure</td>
<td>Renal failure</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Mallory–Weiss tear</td>
<td>All other diagnoses</td>
<td>Upper gastrointestinal malignancy</td>
<td>–</td>
</tr>
<tr>
<td>Major SRH</td>
<td>None or dark spot</td>
<td>–</td>
<td>Blood in upper gastrointestinal tract</td>
<td>–</td>
</tr>
</tbody>
</table>

BP: blood pressure; SRH, stigmata of recent haemorrhage.
Early management

The priority of managing acute upper gastrointestinal haemorrhage should be to correct fluid losses and restore haemodynamic stability. Oxygen should be given. Intravenous access must be obtained and routine laboratory tests carried out. In the high-risk patient, arterial blood gas analysis should be performed. Bleeding severity and clinical risk can then be determined based on the patient’s haemodynamic status, age and comorbidity.

Endoscopic therapy

Patients with mild or moderate bleeding who have responded to fluid replacement and who remain haemodynamically stable can have their endoscopy as a semi-elective procedure, ideally within 24 hours of admission.

Endoscopy should be performed urgently in patients who have major sustained haemorrhage. The endoscopy should be performed in an endoscopy unit unless the patient is very unstable or the bleed occurs “out of hours”, when a well-staffed operating theatre with resuscitation equipment and anaesthetic support may be preferable. In the very unstable patient endotracheal intubation to prevent pulmonary aspiration should be considered.

There are three aims for the endoscopy in cases of upper gastrointestinal haemorrhage:
- to determine the cause of bleeding;
- to identify those patients at risk from re-bleeding;
- to treat the underlying cause.

A range of endoscopic techniques for non-variceal bleeding are available (Table 4). It is difficult to determine which modality is the best and local practice will be determined by local expertise and resources. In cases where one modality fails to control bleeding a second modality can be used during the same endoscopy.

<table>
<thead>
<tr>
<th>MODALITY</th>
<th>Injection</th>
<th>Fibrin glue</th>
<th>Sclerosants</th>
<th>Alcohol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heat</td>
<td>Heat probe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clip</td>
<td>Endo Clip™</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Medical therapy

Acid suppression using high-dose proton pump inhibitors (eg omeprazole 80 mg stat., then infusion 8 mg hourly for 72 hours) following successful endoscopic haemostasis of major ulcer bleeding has been shown to reduce rates of re-bleeding and should be commenced in all patients with a confirmed non-variceal upper gastrointestinal bleed. There is no convincing evidence for the use of histamine H₂-receptor antagonists. Somatostatin may have some benefit in reducing acid secretion and splanchnic blood flow, but there is no evidence to support its routine use.

Surgical management

Active non-variceal upper gastrointestinal haemorrhage that cannot be stopped by endoscopic intervention usually requires an urgent surgical operation. In cases of re-bleeding the decision to operate can be more complex. Surgery for gastrointestinal haemorrhage carries a high morbidity and mortality and the decision about whether to operate or not must be made on a patient-by-patient basis. In most cases endoscopy will provide the surgeon with the site of bleeding. The most common site of ulcer bleeding to require surgical intervention is a chronic posterior duodenal
ulcer involving the gastroduodenal artery. The principle of surgical management for a bleeding posterior ulcer is to under-run the bleeding vessel.

**Radiological intervention**

Selective arterial angiography can be used to investigate and treat upper gastrointestinal bleeding. When endoscopy has failed to identify the site of bleeding, angiography of the coeliac artery or mesenteric artery can be used to locate and treat the site of bleeding by embolisation or coiling. Selective angiography has also been used to treat ulcer bleeding following failed endoscopic haemostasis. For patients with significant medical comorbidities who are unfit for surgery, an endovascular approach may offer an alternative to repeat endoscopic intervention in the haemodynamically stable patient.

**Follow-up**

Following a confirmed ulcer bleed, patients should receive a treatment dose of proton pump inhibitor plus *Helicobacter* eradication. In cases with associated use of NSAIDs or aspirin use, these drugs should be stopped and, if essential, future NSAID and aspirin therapy must be undertaken in conjunction with a proton pump inhibitor.

Patients who have bled from a gastric ulcer should be re-endoscoped to confirm healing and to exclude a malignancy. Routine re-endoscopy of a non-surgically treated bleeding duodenal ulcer is not necessary unless evidence of arterial bleeding was seen at endoscopy – in which case endoscopic evidence of resolution prior to discharge is desirable.

**LEARNING POINTS: Upper gastrointestinal haemorrhage**

- Age, comorbidity, shock, endoscopic stigmata of recent bleeding and re-bleeding are all independent predictors of death from non-variceal upper gastrointestinal haemorrhage.
- There are three aims of endoscopy in cases of upper gastrointestinal haemorrhage: to determine the cause of bleeding; to identify patients at risk from re-bleeding; and to treat the underlying cause.
- Active non-variceal upper gastrointestinal haemorrhage that cannot be stopped by endoscopic intervention needs an urgent surgical operation.
- Acid suppression using high-dose proton pump inhibitors should be commenced in all patients with a confirmed major ulcer bleeding.
- The average mortality from the first episode of variceal bleeding is 50%.

**Further reading**


Technical skills

Perforated peptic ulcer

Introduction
Diagnosis of a perforated or bleeding peptic ulcer may not be made until a laparotomy is performed. All emergency laparotomies should be performed following the principles outlined below. Individual techniques may differ.

Step 1
- Ensure the patient is positioned supine and the abdomen is correctly prepared and draped.
- Make a midline laparotomy incision using a scalpel.
- Divide the peritoneum between two clips using scissors to prevent an iatrogenic enterotomy.
- Remove any contaminated fluid, and remember to send a sample for microscopy and culture.

Step 2
- To establish effective control of haemorrhage, or if there is excessive contamination, it may be necessary to pack the four intraperitoneal quadrants.
- A thorough and systematic examination of the intraperitoneal space and its contained organs and structures should be performed.

Step 3
- In this case a perforation is identified on the anterior wall of the stomach.
- Posterior-wall-bleeding duodenal ulcers will require a longitudinal enterotomy in line with the length of the duodenum to gain access.
• Such enterotomies should be closed at 90° to the original incision to prevent stricture formation.

Step 4
• If the area around a gastric perforation is suspicious of malignancy, a biopsy should be taken and gastrectomy considered.
• Otherwise, place an appropriate number of transverse sutures at 90° to the length of the wound.
• Clip but do not tie the suture ends.
• Take deep bites of the tissue (the edges of a perforation are often friable, with reduced tensile strength, allowing superficial sutures to pull out).

Step 5
• Identify or dissect free a viable tongue of omentum.
• Place the omentum over the perforation.
• Secure in position by tying the sutures.

Step 6
• Examine the repair to ensure it is completely closed. Extra sutures can be placed at this time.
• Remove any remaining packing material. Any further contamination should be sucked or washed away.
• Drains should not be necessary.
Step 7
- Employ a running mass closure technique to the laparotomy wound using two monofilament loop 1-0 or 0-0 sutures.
- Begin at either end of the wound, securing the suture using the loop.
- Place each suture bite through the abdominal fascia, 1 cm deep and 1 cm away from the last bite.
- A suture four times the length of the wound is required.

Step 8
- The skin should be closed with individual metal clips.
- Ensure the procedure and findings are carefully documented.
- Construct a management plan that includes chasing results for all microbiology and pathology specimens and re-examining the patient postoperatively within 4 hours.
Introduction
Acute lower gastrointestinal haemorrhage is defined as acute bleeding emanating from the gastrointestinal tract, distal to the ligament of Treitz. Lower gastrointestinal bleeding constitutes 20% of all cases of acute gastrointestinal haemorrhage. The majority of bleeds occur in the elderly and male populations. The correlation with age is probably associated with the increased incidence of colonic diverticular disease and angiodysplasia in these groups.

Typically, lower gastrointestinal haemorrhage is self-limiting with a reported mortality rate of 2–4%. Increased mortality and stratification for risk of re-bleeding is based on the presence of cardiovascular instability, significant associated comorbidities, or the use of regular anticoagulant, antiplatelet or non-steroidal anti-inflammatory drugs (NSAIDs).

History, examination and differential diagnosis

Diagnosis
The duration, frequency and colour of blood passed per rectum may help determine the severity and location of bleeding. Clinically, the most common presentation of lower gastrointestinal bleeding is haematochezia, although melaena, haemodynamic instability, anaemia, and abdominal pain can be seen. The stability of the patient and the rate of bleeding dictate the order in which diagnostic procedures should be conducted. Resuscitation efforts should take place concurrently with the initial evaluation of the patient to prevent complications of blood loss. An initial haematocrit of less than 35%, the presence of abnormal vital signs 1 hour after initial medical evaluation, and gross blood on initial rectal examination are independent predictors of severe lower gastrointestinal bleeding and an adverse outcome.

History
An accurate history may help elucidate the bleeding source. Abdominal pain tends to occur in the presence of ischaemia or inflammatory bowel disease, therefore risk factors for thrombus should be identified. Antecedent constipation or diarrhoea, the presence of diverticulosis, previous radiation therapy, recent polypectomy and a family history of colon cancer should all form part of the enquiry.

Physical examination
Physical examination should focus on assessment of loss of intravascular volume, a possible bleeding source and comorbid conditions (which may affect subsequent suitability for investigation or intervention). All patients presenting with lower gastrointestinal bleeding should have a documented digital rectal examination, commenting on the presence of anorectal lesions and stool colour. Despite
presenting features and findings on physical examination, most patients with lower gastrointestinal bleeding will warrant a full examination of the colon.

**Aetiology**

**Diverticular disease**

Diverticular disease is the most common aetiology of major lower gastrointestinal haemorrhage, comprising 20–55% of all cases. Diverticulosis is rare in people under 40 years of age, but is seen in up to 65% of patients over the age of 85. Clinical presentation generally is with acute, painless haematochezia and in most cases resolves spontaneously. However, up to 25% of patients will re-bleed and require emergency intervention. The pathophysiology is thought to be due to repeated trauma to the vasa recta which run across the diverticular dome. As bleeding frequently stops spontaneously, the diagnosis is often presumptive, made following exclusion of other pathologies. Preventative strategies include a high-fibre diet and avoidance of NSAIDs.

**Ischaemic colitis**

Ischaemic colitis accounts for up to 20% of lower gastrointestinal bleeding. Typical presentation is with bloody diarrhoea and associated abdominal pain. The colon is predisposed to ischaemic insult due to poor collateral circulation. Watershed areas, including the splenic flexure and rectosigmoid junction, harbour a particularly tenuous blood supply. Patients tend to be elderly with advanced atherosclerosis or cardiac disease. Colonic endoscopy is the investigation of choice for colonic ischaemia. Colonoscopic findings include oedema, haemorrhage and ulceration with a sharp line of demarcation between normal and abnormal mucosa. Most cases resolve spontaneously with supportive treatment; however 15–20% of patients will develop gangrene that requires surgical intervention and have a significant risk of death.

**Angiodysplasia**

Angiodysplasia are gastrointestinal vascular ectasias, estimated to be the source of lower gastrointestinal bleeding in approximately 11% of cases. They can occur anywhere along the gastrointestinal tract but are found predominantly in the caecum and ascending colon. Lesions can be multiple and incidental findings in 2% of non-bleeding patients aged over 65. The classic endoscopic appearance is of a red, flat lesion with ectatic blood vessels radiating from a central feeding vessel. Angiography is considered more sensitive than colonoscopy for detecting angiodysplasia. The clinical presentation includes iron deficiency anaemia with faecal occult blood, melaena or painless haematochezia that may be intermittent and clinically indistinguishable from diverticular bleeding.

**Anorectal disease**

Haemorrhoidal disease comprises 5–10% of fresh lower gastrointestinal bleeding. It is usually intermittent, associated with bowel movements, and rarely significant, but it highlights the importance of proctoscopy as an early assessment tool. Most haemorrhoidal bleeding will stop with conservative measures. Patients with significant refractory haemorrhage may require endoscopic or surgical intervention. Rectal ulcers may be the result of faecal impaction, rectal trauma or rectal prolapse, resulting in significant rectal bleeding requiring endoscopic therapy. Radiation colitis is most often seen following radiation therapy for prostate or gynaecological cancer and typically results in chronic, low-grade bleeding.

**Neoplasms**

In most cases the bleeding associated with neoplasia is occult. Patients present with anaemia, weight loss and change in bowel habit. Laboratory values usually demonstrate a hypochromic microcytic anaemia with associated iron deficiency. Massive haemorrhage is uncommon, accounting for only 10–15% of lower gastrointestinal bleeds.
Polypectomy
Clinically relevant bleeding occurs in 1–6% of patients undergoing colonoscopic polypectomy. Bleeding at the time of polyp excision is amenable to immediate endoscopic haemostasis. Delayed bleeding typically occurs within a week but can occur up to 3 weeks following the original procedure. Risk factors for post-polypectomy bleeding include large polyps, sessile morphology and right colon location. Most patients with post-polypectomy bleeding present with mild to moderate blood loss, allowing for conservative management.

Small bowel pathology
Up to 15% of lower gastrointestinal haemorrhage is the result of small bowel pathology. Angiodysplasia is the commonest cause, followed by lymphoma, erosions/ulcers and Crohn’s disease. Diagnosis is difficult owing to the inability of common investigative procedures to adequately visualise the small intestine. Enteroscopy, barium contrast radiography and capsule endoscopy are appropriate diagnostic tools. However, as a result these patients often have more diagnostic procedures, blood transfusions and days in hospital than those with either upper or lower gastrointestinal bleeds.

Diagnostic procedures
The diagnostic and therapeutic approach to patients with severe lower gastrointestinal haemorrhage remains controversial, with patients often being managed in accordance with site-specific protocols. Several strategies

<table>
<thead>
<tr>
<th>PROCEDURE</th>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colonoscopy</td>
<td>Therapeutic possibilities</td>
<td>Bowel preparation required</td>
</tr>
<tr>
<td></td>
<td>Diagnostic for all sources of bleeding</td>
<td>Requires on-call endoscopy service</td>
</tr>
<tr>
<td></td>
<td>Efficient and cost-effective</td>
<td>Invasive</td>
</tr>
<tr>
<td>Angiography</td>
<td>No bowel preparation</td>
<td>Requires active bleeding for successful examination</td>
</tr>
<tr>
<td></td>
<td>Therapeutic possibilities</td>
<td>Serious complications are possible</td>
</tr>
<tr>
<td></td>
<td>May be superior for patients with severe bleeding</td>
<td>False positives</td>
</tr>
<tr>
<td>Radionuclide scintigraphy</td>
<td>Non-invasive</td>
<td>Variable accuracy</td>
</tr>
<tr>
<td></td>
<td>Sensitive to low rates of bleeding</td>
<td>Not therapeutic</td>
</tr>
<tr>
<td></td>
<td>No bowel preparation</td>
<td>May delay therapeutic intervention</td>
</tr>
<tr>
<td></td>
<td>Diagnosis must be confirmed with endoscopy/surgery</td>
<td></td>
</tr>
<tr>
<td>CT scan</td>
<td>Non-invasive</td>
<td>Not therapeutic</td>
</tr>
<tr>
<td></td>
<td>Efficient and cost-effective</td>
<td>May delay therapeutic intervention</td>
</tr>
<tr>
<td></td>
<td>Diagnostic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>May localise source for future angiography/surgery</td>
<td></td>
</tr>
</tbody>
</table>

Table 5: Advantages and disadvantages of common investigative techniques of lower gastrointestinal haemorrhage
are available but depend largely on staff experience and skills.

A nasogastric tube may be placed in an attempt to exclude an upper gastrointestinal source. If following gastric lavage frank blood is aspirated, this is an indication of upper gastrointestinal bleeding. However, a clear aspirate does not exclude an upper gastrointestinal lesion. In severe lower gastrointestinal bleeding, particularly where there is evidence of cardiovascular instability, many clinicians would argue the merits of upper gastrointestinal endoscopy to either exclude this region as a source or potentially to halt the life-threatening haemorrhage. The relative value and appropriate order in which further investigations should be performed has been hotly debated. Table 5 highlights the merits and drawbacks of available investigative techniques.

Surgical management

Surgical intervention is required in a minority of patients with lower gastrointestinal haemorrhage. The surgical options depend on whether the bleeding source has been identified preoperatively (Figure 4). If the source is known, then it is possible to perform segmental resection. If the source remains unknown then an upper and lower gastrointestinal endoscopy should be performed on the anaesthetised patient, assuming this has not been carried out preoperatively. At laparotomy, it is often difficult to identify the bleeding source because blood refluxes into the bowel proximally as well as distally. On-table colonic lavage and endoscopy/enteroscopy may help identify the source.

Figure 4: Management algorithm for acute severe lower gastrointestinal haemorrhage that does not resolve spontaneously. Resuscitation should remain ongoing through each of these steps.
If the bleeding source remains unclear, a subtotal colectomy with end ileostomy is the procedure of choice. Surgeons must be aware that blind segmental resection and emergency subtotal colectomy is associated with substantial rates of re-bleeding (in up to 33% of cases) and mortality (33–57%). Anastomosis after resection for haemorrhage will depend on the stability of the patient and the site of potential anastomosis.

**LEARNING POINTS: Lower gastrointestinal haemorrhage**

- Lower gastrointestinal haemorrhage is bleeding originating within the bowel, distal to the ligament of Treitz.
- Lower gastrointestinal haemorrhage is a symptom of the elderly with multiple aetiologies.
- In the majority of cases it is self-limiting, but in a small number of cases it can lead to overt blood loss.
- In the majority of patients assessment of the entire colon is warranted.
- Suitability of diagnostic procedures is defined by the knowledge and skills held at individual centres.

It is likely that CT angiography will play an increasing role in the diagnosis of lower gastrointestinal haemorrhage, but therapeutic intervention will remain in the hands of the endoscopist, radiologist and surgeon.

**Further reading**


Technical skills

Small bowel resection

Introduction
Small bowel resection should be performed in almost all cases of perforation. Only the smallest perforations with otherwise healthy surrounding tissue are amenable to direct suture repair. Small bowel trauma should be suspected in cases of penetrating abdominal injury, crush injuries to the torso and mechanisms of injury that involve sudden deceleration (road traffic injuries and significant falls).

Step 1
- Following identification of a small bowel injury, contamination secondary to free gastrointestinal content should be kept to a minimum.
- Ensure all the small bowel and its associated mesentery is checked because multiple perforations/tears are common.

Step 2
- Note the visible vascular arcades radiating through the mesentery. Proper identification of this blood supply is essential to ensure viable bowel remains following resection.
- Create a window in the mesentery adjacent to the bowel and a safe distance from the perforation.

Step 3
- Using a linear cutting stapler, seal and separate the small bowel.
- If the vascular arcades are difficult to identify, hold the mesentery up to a light – the arcades will create an easily identifiable shadow.
Step 4

- Having correctly resected the small bowel on one side of the perforation, repeat steps 2 and 3 on the opposite side of the perforation.
- If a stapling device is not available, place a soft bowel clamp on the healthy bowel and a crushing clamp closest to the perforation. You can now use a scalpel to divide the bowel.

Step 5

- You should now be left with an isolated segment of bowel containing the original perforation.
- Identify the blood vessels supplying this segment.

Step 6

- Place a clamp across these vessels.
- Divide the vessels above the clamp.
- Remove the damaged specimen and send to pathology for review.

Step 7

- Ensure haemostasis by either tying or transfixing the vessels held in the clamp.
Step 8

- The two bowel ends can be anastomosed or returned temporarily to the abdomen if a damage-control laparotomy is being performed.
- A postoperative ileus is not uncommon due to significant handling of the small bowel.
Acute pancreatitis

Introduction
Acute pancreatitis is defined as an acute inflammatory process of the pancreas, usually with rapid onset of pain and tenderness, accompanied by vomiting and systemic inflammatory response syndrome (SIRS) and sometimes involves regional tissues and remote organ systems, as well as elevation of pancreatic enzymes in the blood or urine (Atlanta, 1992). The condition usually occurs in those aged over 30 years and is rare in children. The incidence in the UK is approximately 50–100 per million of the population and it continues to rise. The condition causes significant morbidity. The mortality rate is 10% overall (25–90% in severe cases) and the elderly are at increased risk (mortality rate 28% in people aged over 60).

The three commonest causes in the West are cholelithiasis (42%), alcohol (25%) and idiopathic (15%). Other causes include post-ERCP (endoscopic retrograde cholangiopancreatography) (4%), trauma, steroids, autoimmune disease, metabolic abnormalities (hyperlipidaemia, hypercalcaemia, hypothermia) and drugs (eg didanosine, diuretics).

Pathogenesis
Two theories exist regarding the pathogenesis.
1. Mechanical obstruction ± hypersecretion: This leads to intrapancreatic bile reflux and activation of proteases, coupled with hypersecretion.

2. Toxic and metabolic causes: These lead to immaturity and disruption of normal acinar separation of lysosomes and protease activation within acinar cells.

Terminology
Mild acute pancreatitis (80% cases)
This is classically associated with minimal organ dysfunction (see below) and an uneventful recovery. The predominant feature is interstitial oedema of the gland. The attack usually resolves rapidly with administration of analgesia and intravenous fluid resuscitation.

Severe acute pancreatitis (20% cases)
Organ failure and/or local complications such as necrosis (± infection), or pseudocyst or abscess formation may occur. These lead to a severe life-threatening illness.

History
Acute pancreatitis usually presents with a sudden onset of severe continuous epigastric pain that classically radiates to the back. The pain may be associated with early nausea and profuse vomiting. There is often a history of previous attacks or gallstone disease.

Examination
Typical examination features are shown in Table 6. Chest examination may reveal a sympathetic pleural effusion (in
Specialty Skills in Emergency Surgery and Trauma (CT1–2)

20% of cases) and abdominal bruising is a late sign of severe disease.

<table>
<thead>
<tr>
<th>Table 6: Typical examination findings in Acute pancreatitis (AP)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FEVER</strong></td>
</tr>
<tr>
<td>Hypovolaemic shock</td>
</tr>
<tr>
<td>Mild abdominal distension</td>
</tr>
<tr>
<td>Grey-Turner sign</td>
</tr>
</tbody>
</table>

**Diagnosis**

The diagnosis is usually made in the presence of a convincing history coupled with a rapid rise in serum amylase levels (especially over 1000 IU/l). The rise in pancreatic lipase (sensitivity and specificity >90%) may be slower but is more sustained. Raised amylase concentrations may be caused by any condition that results in an acute abdomen. It is particularly important to rule out other conditions requiring immediate surgery, such as intestinal obstruction, a perforated peptic ulcer or an ectopic pregnancy. Medical causes of a raised amylase, such as renal failure, diabetic ketoacidosis and familial macroamylasaemia, should also be ruled out.

**Scoring systems**

The variability of clinical behaviour seen in acute pancreatitis has led to the development of systems to predict severity. Scoring systems are universally accepted despite their accuracy being overstated and their use in different populations and settings. There has also been a lack of training for users.

The commonest system in use is the Modified Glasgow criteria (Blamey, et al. 1984), which uses eight factors to predict the development of severe disease (Table 7). In three prospective studies, the modified Glasgow scoring system had a sensitivity of only 50–70% and a positive predictive value (PPV) of 46–60%. This means that between 30% and 50% of severe attacks will be missed and 50% of those predicted to have severe acute pancreatitis will have an uncomplicated attack.


<table>
<thead>
<tr>
<th><em><em>AST/ALT</em> &gt;200 U/L</em>*</th>
<th><strong>WCC &gt;15 × 109/L</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate dehydrogenase</td>
<td>Urea &gt;16 mmol/L</td>
</tr>
<tr>
<td>&gt;600 U/L</td>
<td>(not responding to therapy)</td>
</tr>
<tr>
<td>Calcium &lt;2 mmol/L</td>
<td>Glucose &gt;10 mmol/L</td>
</tr>
<tr>
<td>(not responding to therapy)</td>
<td>(in absence of diabetes)</td>
</tr>
<tr>
<td>PaO2 &lt;60 mmHg</td>
<td>Albumin &lt;32 g/L</td>
</tr>
</tbody>
</table>

*Alanine aminotransferase to serum aspartate aminotransferase ratio.

Other systems used to predict severe disease include the APACHE II score on admission (positive predictive value 46%) and raised C-reactive protein (CRP) levels in excess of 150 mg/dL.

**Organ dysfunction**

Organ dysfunction is defined as an alteration of organ function in acute illness such that homeostasis cannot be
maintained without intervention. Prospective multicentre trials have demonstrated that the majority of patients who develop severe acute pancreatitis have evidence of early systemic organ dysfunction at the time of admission. Furthermore, worsening organ failure was associated with a high mortality. Hence there is now a shift in emphasis towards repeated clinical assessment, aimed at picking up early organ dysfunction which occurs in the majority of patients at presentation. Thus the emphasis is on diagnosing as opposed to predicting severe pancreatitis.

UK guidelines
The current UK guidelines relating to scoring systems were published by Johnson et al. in 2005. Table 8 summarises which features predict a severe acute attack within 48 hours of admission.

### Table 8: Features predicting a severe acute attack within 48 hours of admission. Reproduced from Johnson CD *et al.* UK guidelines for the management of AP. Gut 2005; 54 (Sup 3)

<table>
<thead>
<tr>
<th>TIME</th>
<th>CLINICAL IMPRESSION OF SEVERITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial assessment</td>
<td>Body mass index &gt;30 Pleural effusion on chest x-ray APACHE II score &gt;8</td>
</tr>
<tr>
<td>24 hours after admission</td>
<td>Glasgow score 3+ Persisting organ failure C-reactive protein &gt;150 mg/dL APACHE II score &gt;8</td>
</tr>
<tr>
<td>48 hours after admission</td>
<td>Glasgow score 3+ C-reactive protein &gt;150 mg/dL Persisting organ failure</td>
</tr>
</tbody>
</table>

### Management

**Resuscitation and immediate treatment**
- Oxygen.
- Pain relief and antiemetics.
- Nil by mouth.
- Nasogastric tube (if vomiting is a prominent feature).
- Intravenous fluids for replacing the fluid deficit plus ongoing losses (as well as maintenance fluid and electrolyte requirements).
- Fluid balance chart.
- Enoxaparin plus thromboembolic deterrent
- Urinary catheterisation.
- Regular monitoring and repeated assessment.

**Baseline investigations**
- Bloods (full blood count, urea and electrolytes, liver function tests, C-reactive protein, calcium, lactate dehydrogenase and glucose).
- Group and save.
- Arterial blood gases.
- Erect chest x-ray to rule out a perforated viscus.
- Supine abdominal x-ray if abdominal distension is a prominent feature (and will detect pancreatic calcification in recurrent cases of acute-on-chronic pancreatitis).
- Pregnancy test in women of child-bearing age.
- Dipstick urine or midstream urine.
- ECG to rule out medical causes of upper abdominal pain.

**Imaging**
- Ultrasound (look for gallstones, dilated intra/extrahepatic ducts, rule out other pathology).
- Early CT (to confirm diagnosis and grade severity):
  - Grade A – Normal pancreas
  - Grade B – Local/diffuse enlarged; small intraperitoneal fluid collections
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- Grade C – Above + peripancreatic inflammation and <30% gland necrosis
- Grade D – Above + single extrapancreatic fluid collection and 30–50% gland necrosis
- Grade E – Above + extensive extrapancreatic fluid collection, pancreatic abscess and >50% necrosis.

- Late CT (5+ days) (identify pancreatic necrosis and infection).

Treatment

**Mild acute pancreatitis:** Patients presenting with mild acute pancreatitis may be nursed on the general ward and treated with intravenous fluids and analgesia. Oral fluids and diet may be commenced after a few days. There is no place for antibiotic therapy in these patients.

**Severe acute pancreatitis:** Patients with severe acute pancreatitis require nursing in the high-dependency unit or intensive care. They require full monitoring, regular blood gases and, if indicated, early ERCP (endoscopic retrograde cholangiopancreatography) and sphincterotomy within 72 hours (severe gallstone pancreatitis). Contrast-enhanced CT scans are performed at 1 week and serially to monitor progress. Threat to life within the first few days is hypovolaemic shock. In the first week it is multiorgan failure. From the second week onwards it is sepsis.

**Complications**

The commonly encountered complications are listed in Table 9.

**Infected necrosis**

Incidence rates are 40–70% in pancreatic necrosis. Infected necrosis presents with worsening clinical condition, usually 2 weeks after the onset of an acute attack. Diagnosis is made by CT and fine-needle aspiration. In 11 published series spanning a total of 541 patients, mortality rates range from 7.3% to 61.1% with an overall rate of 20%. The treatment options include open necrosectomy and laparostomy, radiological drainage and lavage, or laparoscopic drainage and debridement procedures.

<table>
<thead>
<tr>
<th>Table 9: Complications of acute pancreatitis</th>
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<tbody>
<tr>
<td><strong>LOCAL</strong></td>
</tr>
<tr>
<td>Gastroparesis</td>
</tr>
<tr>
<td>Gastrointestinal haemorrhage</td>
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<tr>
<td>Necrosis of surrounding organs</td>
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<td></td>
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<tr>
<td><strong>Late</strong></td>
</tr>
<tr>
<td>Pseudocyst formation</td>
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<tr>
<td>Pancreatic necrosis</td>
</tr>
<tr>
<td>Pancreatic abscess</td>
</tr>
<tr>
<td>Chronic pancreatitis</td>
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</tbody>
</table>
LEARNING POINTS: Acute pancreatitis

- In most patients acute pancreatitis is a self-limiting condition.
- Scoring systems help predict the severity of the ensuing attack of acute pancreatitis.
- The majority of patients who develop severe acute pancreatitis have evidence of early systemic organ dysfunction at the time of admission to hospital.
- Repeated clinical assessment is essential and aims to pick up early organ dysfunction in these patients.
- The emphasis is on the prevention of recurrence.

Further reading


Intestinal obstruction

Introduction
Intestinal obstruction is defined as a restriction to the normal passage of gastrointestinal contents and is one of the commonest conditions encountered by surgeons, accounting for 5–10% of all admissions. Intestinal obstruction is further subdivided into two distinct groups, with patients presenting with either a paralytic or mechanical obstruction.

Definitions

Paralytic obstruction
This is defined as a functional problem with either disordered or absent gastrointestinal motility. It is often referred to as an adynamic or paralytic ileus. The aetiology of paralytic obstruction is complex, with many factors coexisting. The commonest precipitating factor is recent surgery. Research suggests that the combined effect of several different factors during the postoperative period produce a paralytic obstruction. These include both pharmacological (eg anaesthetic agents and opioid analgesia) and inflammatory factors (eg bowel manipulation and inflammatory mediator release) as well as altered neural reflexes after surgery. Other than postoperative ileus, important causes of paralytic obstruction include systemic infections, metabolic disturbances and neurological disorders.

Mechanical obstruction
This occurs when a mechanical component blocks the normal passage of gastrointestinal contents. It can be divided into two groups, depending upon the anatomical site of obstruction – namely the small or the large bowel. The commonest causes of small and large bowel obstruction are shown below (Tables 10 and 11). In the developed world adhesions related to previous surgery are by far the commonest cause of all obstructions, whereas hernia’s and atypical infections are more prevalent in other countries.

Table 10: Causes of small bowel obstruction

<table>
<thead>
<tr>
<th>Adhesions</th>
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<tbody>
<tr>
<td>Hernias</td>
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<tr>
<td>Crohn’s disease</td>
</tr>
<tr>
<td>Neoplasms</td>
</tr>
<tr>
<td>Intussusception</td>
</tr>
<tr>
<td>Volvulus</td>
</tr>
<tr>
<td>Superior mesenteric artery syndrome</td>
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<tr>
<td>Gallstone ileus</td>
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<tr>
<td>Ischaemic strictures</td>
</tr>
<tr>
<td>Foreign bodies</td>
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<tr>
<td>Intestinal atresia</td>
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</tbody>
</table>
### Table 11: Causes of large bowel obstruction

<table>
<thead>
<tr>
<th>Cause</th>
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<tbody>
<tr>
<td>Neoplasms</td>
</tr>
<tr>
<td>Hernias</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Colonic volvulus</td>
</tr>
<tr>
<td>Faecal impaction</td>
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<tr>
<td>Colon atresia</td>
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<tr>
<td>Diverticular disease</td>
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<tr>
<td>Endometriosis</td>
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</table>

### Closed-loop obstruction

This is a specific form of mechanical obstruction characterised by massive distension within a segment of bowel, due to a combination of complete obstruction distally and the presence of proximal valve-like mechanism, allowing the bowel segment to fill, but preventing reflux. This situation is commonly seen with distal large bowel obstructions where a competent ileocaecal valve allows the proximal large bowel to receive contents but prevents reflux. The large bowel, usually the caecum, becomes grossly distended; perforation may occur if the obstruction is not relieved.

### Pathophysiology

If the intestine is obstructed by a mechanical occlusion, the bowel distal to the obstruction continues to empty and becomes collapsed. Conversely, the bowel proximal to the site of obstruction dilates due to the continuing passage of gas (swallowed air) and gastrointestinal secretions. This produces an ideal environment for bacteria, resulting in overgrowth and fermentation. The byproducts of these processes create gradients for both gaseous diffusion and the passage of fluids and electrolytes (osmosis) from the bowel wall into the lumen. This distension impairs the blood supply to the bowel wall, increasing the risk of ischaemia. If the obstruction is not resolved, mucosal ulceration and eventually perforation may also occur.

### Recognition

The clinical findings vary between patients depending upon the cause of obstruction and location. There are four principal clinical features present in the majority of cases of intestinal obstruction.

1. **Pain**

   Commonly pain is the earliest symptom noted in mechanical obstruction. It is often described as “colicky” due to episodes of vigorous peristalsis occurring in an attempt to overcome the physical obstruction. Stretch receptors within the visceral peritoneum are stimulated by the dilated bowel, giving rise to pain that is poorly localised to original embryological origins (eg foregut, midgut and hindgut). Occasionally, as the obstruction persists, normal peristalsis ceases and flaccidity occurs, which in turn reduces symptoms of pain. Where pain is intense, constant and well localised, a perforation must be suspected. There is often no pain in a paralytic ileus owing to the absence of effective peristalsis.

2. **Abdominal distension**

   Abdominal distension is entirely dependent on the anatomical region of bowel involved. A high small-bowel obstruction may present with only minimal abdominal distension as there is limited amount of proximal bowel available for dilatation. In reverse, an obstruction located within large bowel may present with marked abdominal distension owing to the presence of dilatation of the large and small bowel.

3. **Constipation**

   As a clinical feature of intestinal obstruction, constipation varies greatly between individual people. In cases of small bowel obstruction it is often a late feature, as the bowel distal to the site of obstruction will continue to empty while peristalsis occurs. However, in large bowel obstruction inability to pass a motion is an early feature because there
is often little bowel distal to the site of obstruction. Absolute constipation – the failure to pass faeces or flatus – is common in these patients and is confirmed by finding an "empty" rectum on digital rectal examination.

4. Vomiting
Early onset of vomiting is a clear feature of proximal bowel obstruction, but delayed or no vomiting signifies a more distal site of obstruction. In the later stages of obstruction vomiting may become faeculent owing to the decomposition of food or the presence of altered blood within the bowel lumen.

Other features
In combination with these clinical features, a history of previous abdominal surgery is important to note as it raises the possibility of postoperative adhesions being the underlying cause. During clinical examination, signs of dehydration may become apparent. The loss of fluid within the gastrointestinal tract should not be underestimated in these patients. Particular attention should be applied to signs of hypovolaemia (eg dry mouth, lethargy, tachycardia, hypotension and oliguria). Detailed examination of hernial orifices is important in order to exclude an incarcerated or strangulated hernia. Erythema, tenderness and non-reduction over a hernial mass suggest the presence of strangulation.

Investigations
Blood tests
Simple blood tests are important tools in investigating these patients. Monitoring the trend in white cell count is vital. A rising count is often associated with a deteriorating patient. An acute elevation suggests infarction or perforation. Monitoring the biochemical markers for renal function is equally important as this enables accurate assessment of renal function and fluid balance. These tests also give valuable information on serum electrolyte concentrations, which are commonly altered in intestinal obstruction owing to substantial losses from gastrointestinal secretions and through osmotic effects within the bowel lumen.

Radiological investigations
Radiological investigations include an erect chest x-ray to exclude perforation. This is usually localised under the hemidiaphragm when the patient is in the erect position. Supine abdominal x-rays may confirm the diagnosis of obstruction with the presence of distended bowel loops. These films may also help to locate the site of obstruction. For example, a grossly distended stomach and duodenum may point to a proximal site of small bowel obstruction, and distended jejunal loops are distinguished by the presence of valvulae conniventes across the width of the bowel (see Figure 5), whereas the ileum is relatively featureless on plain films. Distended large bowel can be distinguished from

Figure 5: Plain x-ray showing small bowel obstruction. Note the presence of valvulae conniventes running across the entire width of the bowel.
small bowel by the presence of the haustrations of the taenia coli, which do not pass along the entire width of the bowel, and its peripheral location (see Figure 6). In approximately 5% of patients signs of intestinal obstruction are not seen on plain abdominal x-rays due to the bowel being completely distended with fluid, and no gas being present.

Figure 6: Plain x-ray showing large bowel obstruction. Note the presence of the haustrations of the taenia coli which do not run across the entire width of the bowel

Contrast studies
Recent research highlights the role of contrast studies in identifying patients suitable for conservative treatment. The introduction of water-soluble contrast through a nasogastric tube may be tracked on serial radiographs in order to identify a clear site of obstruction. Computed tomography (CT) scanning is becoming more important in the investigation of intestinal obstruction. This modality can be combined with the ingestion of water-soluble contrast in order to identify the site and cause of the obstruction. This information is invaluable to the modern surgeon in being able to plan the correct course of treatment.

Management

Resuscitation
The key to managing these patients is resuscitation. Obstructed patients with significant hypovolaemia have been shown to have significantly higher morbidity and mortality rates. Replacement of losses should be delivered through intravenous fluid infusions. Initially large volumes of fluid may be required to replenish third-space losses within the abdomen. In essence, fluid losses must be replaced in addition to normal daily requirements. Urinary catheterisation and use of fluid balance charts are often required to assess fluid balance and guide replacement therapy. Central venous catheterisation may be required to fine-tune treatment in patients with congestive cardiac failure or severe hypovolaemia.

Bowel decompression
Decompression of the bowel often accompanies fluid resuscitation. The passage of a nasogastric tube reduces the risk of aspiration from vomiting by decompressing the stomach. It also allows realistic measurements of gastrointestinal losses. Ideally these losses should be measured through regular aspiration and subsequent free drainage. Usually good fluid balance, together with adequate restoration of electrolyte losses and gastric decompression with a nasogastric tube, is sufficient to allow either an ileus or an adhesional mechanical obstruction to resolve conservatively.

Surgery
The major difficulty in managing intestinal obstruction is knowing when to operate. Patients with small bowel intestinal obstruction and no obvious history to indicate
intra-abdominal adhesions require an early laparotomy in order to protect the small bowel from damage by other causes. Other indications for surgery include patients with suspected perforation or ischaemia (as a result of strangulation), and those at high risk of developing either perforation or ischaemia (e.g., a closed-loop obstruction or an irreducible hernia). Lastly, surgery is also indicated in patients where conservative management has failed, but there is considerable difficulty deciding exactly when conservative treatment has failed. In essence, the decision is made by assessing the patient regularly and reviewing results from various investigations. Recent research has shown that in expert hands laparoscopic surgery can be performed on patients suffering from simple obstruction, but most surgeons perform an open laparotomy.

The reason for operating in these patients is to decompress the bowel, to release the cause of obstruction and to assess the viability of the affected bowel. It may include resection of strangulated or perforated bowel, and further surgery relating to the cause of obstruction.

Distinguishing viable from non-viable bowel may not be clear-cut. Non-viability is determined by three cardinal signs:
- loss of peristalsis;
- discoloration (green or black bowel);
- loss of pulsations.

Doubtful bowel may recover once the obstruction is cleared. In such cases, the bowel can be wrapped in warm, moist packs for 10 minutes. If normal colour and peristalsis return, the bowel is often deemed viable.

Where a segment of bowel is resected, the next course of action adopted by surgeons depends on the site of obstruction. In cases of small bowel obstruction it is common practice to perform a primary anastomosis because the small bowel has an excellent blood supply. In contrast, the large bowel has a relatively poor blood supply and thus primary large bowel anastomoses are at higher risk of leakage. It is up to individual surgeons to decide whether to attempt primary anastomosis in these patients.

A common strategy adopted by most surgeons after a segmental large bowel resection is to bring both ends of the bowel out of the abdominal wall as an end stoma (proximal segment) and a mucous fistula (distal segment). If the distal segment does not reach the surface it can be closed and left within the abdomen as a redundant stump (Hartmann’s procedure). There is much debate on the best type of primary anastomosis to use in obstructed patients. At present, evidence suggests no real difference in leakage rates between hand-sewn and stapled anastomoses. With time and experience most surgeons feel comfortable with a particular style of anastomosis and are able to perform this to a high standard.

**LEARNING POINTS: Intestinal obstruction**

- Intestinal obstruction is a common condition that includes both paralytic and mechanical obstruction.
- Mechanical obstruction is further divided into small or large bowel obstruction depending upon the exact site of the problem.
- There are four key clinical features of intestinal obstruction: pain, vomiting, distension and constipation.
- Radiological investigations can help with making a diagnosis, finding the underlying cause of obstruction and predicting whether some obstructions will resolve spontaneously.
- Adequate fluid resuscitation is vital and is directly related to patient survival.
- Surgery is indicated in patients where conservative treatment has failed or when the patient shows signs of peritonism and/or strangulation. There should be a low threshold to operate on patients with small bowel obstruction in the absence of previous surgery.
Further reading
Abbas S, Bissett IP, Parry BR. Oral water-soluble contrast for the management of adhesive small bowel obstruction. *Cochrane Database of Systematic Reviews* 2007 (1).
Introduction
Infections are a major cause of morbidity and mortality in surgical patients. Among all hospitalised surgical patients, the prevalence is estimated at 7–10%. Trauma patients are at high risk of wound contamination and devitalisation of tissues, which can lead to uncontrolled levels of bacterial colonisation, particularly if the injury includes perforation of the gastrointestinal tract or severe burns. Postoperative patients are more likely to suffer immune dysfunction and may be at risk of developing a systemic inflammatory response.

Table 12: Classification of surgical site infections (adapted from Berard et al. 1964)

<table>
<thead>
<tr>
<th>CLASSIFICATION</th>
<th>CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clean</td>
<td>Elective, not emergency, non-traumatic, primarily closed</td>
</tr>
<tr>
<td></td>
<td>No acute inflammation</td>
</tr>
<tr>
<td></td>
<td>No break in technique</td>
</tr>
<tr>
<td></td>
<td>Respiratory, gastrointestinal, biliary and genitourinary tracts not entered</td>
</tr>
<tr>
<td>Clean–contaminated</td>
<td>Urgent or emergency case that is otherwise clean</td>
</tr>
<tr>
<td></td>
<td>Elective opening of respiratory, gastrointestinal, biliary or genitourinary tract with minimal spillage (e.g., appendectomy) not encountering infected urine or bile</td>
</tr>
<tr>
<td></td>
<td>Minor technique break</td>
</tr>
<tr>
<td>Contaminated</td>
<td>Non-purulent inflammation</td>
</tr>
<tr>
<td></td>
<td>Gross spillage from gastrointestinal tract</td>
</tr>
<tr>
<td></td>
<td>Entry into biliary or genitourinary tract in the presence of infected bile or urine</td>
</tr>
<tr>
<td></td>
<td>Major break in technique</td>
</tr>
<tr>
<td></td>
<td>Penetrating trauma &lt;4 hours old</td>
</tr>
<tr>
<td>Dirty</td>
<td>Chronic open wounds to be grafted or covered</td>
</tr>
<tr>
<td></td>
<td>Purulent inflammation (e.g., abscess)</td>
</tr>
<tr>
<td></td>
<td>Preoperative perforation of respiratory, gastrointestinal, biliary or genitourinary tract</td>
</tr>
<tr>
<td></td>
<td>Penetrating trauma &gt;4 hours old</td>
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</tbody>
</table>
response with multiorgan failure in the presence of an infection. Cosmesis and function can also be compromised by infection.

Among surgical patients the reported rate of colonisation with methicillin-resistant Staphylococcus aureus (MRSA) is increasing. This has led to an escalation in MRSA-associated postoperative infections. Moreover, Clostridium-related infections – ranging from simple antibiotic-related diarrhoea to fulminant pseudomembranous colitis – are beginning to assume greater importance to the surgeon.

Epidemiological information regarding surgical infection is difficult to collect because there is wide variability among procedures, surgeons, patients and local microbiology. Despite this challenge, estimates suggest an incidence of infection following surgery at almost 20%.

**Definitions**

It is important to able to characterise which types of infection are likely to need rapid assessment and prompt surgical management. Less severe infections can be managed simply with observation and antibiotics. Surgical infections can be classified in a number of different ways depending on their site, chronicity or the type of infecting agent.

**Surgical site infection (SSI)** is defined as an infection occurring within 30 days of an operation (or within 1 year if an implant is used) that affects either the incision or deep tissue at the operation site.

According to this description, infections are classified as secondary to a surgical intervention but may arise either in the hospital or in the community following discharge (Table 12).

**Recognition**

**Clinical features and signs**

Life-threatening infections usually present with systemic signs of sepsis but an insidious presentation is not infrequent. Assessment and recognition of surgical infection begins with knowledge of the patient’s history. Details of any recent surgery, history of trauma, previous infections, antibiotic use, immunosuppression, steroid use and any other relevant comorbidities are important clues to the source of infection. Clinical examination includes a thorough assessment of major systems, potential sites of infection and review of the observational charts.

Skin and soft tissue infections as well as intra-abdominal infections share some common clinical features. Patients typically describe increasing levels of pain at the site of the infection and are generally unwell, with anorexia and nausea or vomiting. Clinical examination may reveal cardinal signs of inflammation: heat (calor), pain (dolor), erythema (rubor), swelling (tumor) and possible fluctuance. A swinging pyrexia indicates an infective collection that is typically associated with the systemic inflammatory response syndrome (SIRS).

**Cellulitis**

All wounds should be examined to establish the extent of infection and presence of cellulitis, but in particular following synthetic graft placement (Table 13). Cellulitis is usually indicated by erythema with tissue oedema, and margins should be marked to assess response to treatment. The usual causative organisms are streptococci or staphylococci. In the absence of a subcutaneous collection these infections usually respond to the administration of antibiotics, although close observation is warranted if there are any features of systemic toxicity or a poor response.

**Fluid collections**

Fluid collections usually exhibit fluctuance or discharge of pus and require drainage. Deep space collections may be heralded by physical signs including faecal or purulent discharge; persistent ileus or fistula formation, and cardiac rhythm abnormalities. Some collections may be
managed conservatively taking into account the individual patient factors; however, there is a risk that left untreated these collections can become the source for sepsis and therefore require either radiological or surgical drainage. Intraperitoneal fluid collections irritate the peritoneal lining and usually result in peritonitis. Clinical examination reveals involuntary abdominal wall guarding and, in severe cases, percussion tenderness. These signs may be diminished in patients with immunosuppression or who are taking steroid medication. In cases of lower abdominal abscess secondary to appendicitis or diverticulitis, a palpable mass may be evident and pelvic abscesses may be detected as a “boggy” swelling on digital rectal or vaginal examination.

**Clostridium difficile-associated infections**

Features of these infections can range from mild diarrhoea to life-threatening pseudomembranous colitis. Severe infections are usually characterised by abdominal pain, signs of peritoneal irritation, diarrhoea with or without the passage of blood, and the symptoms and signs of systemic toxicity (such as fever, anorexia, nausea and
malaise). Full blood count analysis often reveals a markedly elevated number of white cells. Some patients with fulminant pseudomembranous colitis and toxic megacolon may complain of little or no diarrhoea owing to paralytic ileus. In these patients abdominal pain and distension are the predominant features.

Resuscitation

People presenting with features of septic shock require urgent assessment and management initially focusing on defects in their airway, breathing and circulation.

Clinical features such as oliguria, confusion, decreased conscious level and hypotension should raise the suspicion of systemic sepsis. Supplementary oxygen and analgesia should be instigated immediately and fluid resuscitation may need to be aggressive depending on the level of intravascular volume depletion. A urinary catheter should be placed and the insertion of a central venous catheter considered in order to evaluate the response to resuscitation.

Broad-spectrum antibiotics are indicated once appropriate cultures for microbiological analysis have been obtained. Antibiotic therapy can be refined once the causative organism has been identified. If invasive monitoring is required then the patient should be managed in a critical care setting.

Investigations

Clinical presentation, history and examination will guide investigation. Venous blood should be sent for a full blood count, urea and electrolytes, liver function and a group and save. Inflammatory markers are frequently elevated in patients with infection, in particular the leukocyte count and C-reactive protein. Chronic infection may lead to a fall in the serum albumin.

Pus specimens from discharging wounds or abscesses and faeces from patients with diarrhoea should be obtained and sent for microscopy, culture, *Clostridium difficile* toxin assay and antimicrobial sensitivity. Radiological imaging includes an erect chest radiograph, CT or magnetic resonance imaging (MRI) and can be useful if deep collections are suspected.

Management

Key principles in the management of surgical infections are:

- drainage of abscesses;
- debridement of non-viable tissue;
- definitive management of ongoing bacterial contamination.

Skin and soft tissue infections with an associated collection can be categorised as uncomplicated or complicated.

Superficial abscesses

Without involvement of deep tissues or underlying patient factors complicating the response to treatment, these can be managed with simple incision and drainage. This is optimally managed in the operating theatre under aseptic conditions. General or regional anaesthesia is usually required to achieve sufficient debridement, depending on the site of the abscess. An incision over the point of maximal fluctuance will achieve drainage of pus, which can be sent for microscopy, culture and antibiotic sensitivity. Irrigation with water or saline and removal of any non-viable tissue with Volkmann’s spoon is usually sufficient to prepare for healing by secondary intention. This can be encouraged by removal of the overlying skin (de-roofing) or by light packing with an absorbent dressing to keep the skin edges apart.

Complicated soft tissue infections

Complicated soft tissue infections involving ulcers, burns or major abscesses require thorough assessment and a multidisciplinary approach to management involving reconstructive and wound care specialists.
Superficial wound collections
These can be drained by removing selected stitches or clips and gentle probing to allow drainage of pus. Wounds should then be allowed to heal by secondary intention. In some cases, debridement may be necessary. Wounds undergoing sharp debridement require regular evaluation to assess the progression or improvement of tissue necrosis. Antibiotics should be administered if spreading cellulitis or systemic illness is noted.

Intra-abdominal collections
These usually occur as a result of a defect in the intestinal wall resulting in the passage of organisms into the peritoneal cavity. Ensuing peritonitis can be localised or diffuse. Management of intra-abdominal abscesses includes maintenance of balanced fluid, nutritional, and electrolyte status and broad-spectrum antimicrobials. In instances of appendicitis or diverticulitis with resulting abscess, initial radiological investigation should determine suitability for ultrasound or CT-guided percutaneous drainage. Remember to send a sample of the aspirate for microbiology. Open surgical drainage is indicated if percutaneous drainage is technically not feasible or if the patient’s condition remains unstable despite drainage and antimicrobial therapy.

Clostridium difficile-associated infections
These infections are manifested by profuse diarrhoea and usually respond to treatment with oral preparations of metronidazole or oral vancomycin in severe disease. However, surgical intervention is indicated in the presence of toxic dilatation, perforation, signs of organ failure, shock, requirement for vasopressors, worsening CT scan findings in the face of medical management, signs of peritonitis, or a lack of response to maximal appropriate medical management within 24–72 hours. Mortality in this group ranges between 30% and 80%. It is important to recognise patients who are not responding to medical treatment because delayed surgical intervention is the factor that has the highest association with mortality.

Typically the colon is oedematous and patients may have ascites. The colon may well be distended to the point of perforation or infarction, but its appearance should not dissuade the surgeon from performing the procedure of choice – which is a total colectomy with end ileostomy. If the integrity of the tissues permits, a rectal “stump” may be created to allow antibiotic irrigation of the remaining rectum and restoration of intestinal continuity at a later date.

Necrotising soft tissue infections
Necrotising soft tissue infections are an uncommon but potentially fatal condition that affects fascial planes, leading to necrosis, systemic toxicity and invariably death without prompt supportive and surgical management. Even with supportive and surgical care mortality is estimated at 25%. Classifications for necrotising soft tissue infections add little value to diagnosis and management when in fact these conditions share aetiology and pathophysiology. Initial management is identical: rapid assessment, resuscitation, administration of antibiotics and decisive operative intervention.

Establishing a timely diagnosis can be notoriously difficult. Clinical signs can seem innocuous in the early stages, resulting in delayed diagnosis. Once the manifestations of severe infection become apparent, the progression of the infection to systemic toxicity and multiorgan dysfunction may not be halted even by massive surgical debridement.

The causal organisms usually involve a synergistic combination of aerobic and anaerobic bacteria. Many of these bacteria are normal human commensals, thus highlighting the particular danger in immunosuppressed patients.

Clinical features can be divided into early and late symptoms and signs:
- **Initial signs** may only reveal simple cutaneous erythema or cellulitis. These appearances, together
with fever and leukocytosis and definite risk factors, should raise the suspicion of possible necrotising soft tissue infection. Other early features include pain, induration and skin anaesthesia.

- **Late and worrisome features** indicating progression include extension of erythema, crepitus, skin blistering and ecchymosis. Physiological changes can include tachycardia, pyrexia, shock, metabolic acidosis, clotting disorders, hypoalbuminaemia, acute renal failure, confusion and a decrease in level of consciousness.

A summary of these early and late features is given in Table 14.

<table>
<thead>
<tr>
<th>Table 14: Features of necrotising soft tissue infections</th>
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<tbody>
<tr>
<td><strong>EARLY FEATURES</strong></td>
</tr>
<tr>
<td>Pain</td>
</tr>
<tr>
<td>Cellulitis</td>
</tr>
<tr>
<td>Swelling</td>
</tr>
<tr>
<td>Induration</td>
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<tr>
<td>Skin anaesthesia</td>
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</table>

The use of adjunctive tests to aid diagnosis is of limited value in the critically ill patient and the diagnosis remains for the most part clinical. However, it is possible to delineate the extent of tissue involvement and the presence of soft tissue gas with computed tomography or magnetic resonance scanning. These modalities should not be employed if a significant delay to treatment is likely to occur.

Individuals suspected to have a necrotising soft tissue infection require rapid assessment with simultaneous resuscitation. Decisive surgical management is the key to a favourable outcome. Exploration should be undertaken in theatre and will begin with exposure of the soft tissue planes. Lack of bleeding and discharge of turbid fluid from the subcutaneous tissues indicates a likely necrotic infection. Discoloration or frank necrosis confirms the diagnosis. Additional exploration beyond the deep fascia is necessary to examine muscle and tissue planes at this level. Wide debridement should ensue, with exposure of healthy tissue that bleeds on contact. The temptation to adopt a conservative approach in favour of a more cosmetic outcome can result in the need for repeated debridement. Wounds are irrigated and dressed, typically with ribbon gauze soaked in an antibiotic such as proflavine. Further exploration and dressing changes should be planned for 24–36 hours to assess the response to debridement and the need for further debridement.

Adequate wound care is vital to ensure prompt closure. Specialist reconstructive surgical involvement and specialist wound care nursing support can provide expertise to promote wound healing and closure. Vacuum-assisted closure devices may be employed to facilitate wound closure where skin grafting is not appropriate. Patients may require elective colonic defunctioning if there is likely to be contamination of wounds with faeces.

**Learning Points: Surgical Infections**

- Infections in surgical patients are common and are a major cause of mortality and morbidity.
- Infections can occur primarily or secondary to a surgical procedure.
- It is important to recognise an infection that is rapidly progressive with risk of irreversible tissue damage.
- Resuscitation and supportive care play a major part in reducing the risk of multiorgan failure.
- Patients with sepsis should be assessed thoroughly for occult sources of infection.
- Complicated abscesses favour a multidisciplinary approach.
- Patients with *Clostridium difficile*-associated infections benefit from early surgical assessment.
Further reading


**Introduction**

Vascular trauma in the United Kingdom is predominantly blunt in nature. Fewer than 10% of polytrauma patients have a vascular injury, however, there is a steady increase in the incidence of penetrating injury from firearms and knives in particular. Some vascular injuries can be complex and present some of the most challenging problems for the surgeon.

Blunt injury can be direct or indirect. Direct blunt trauma to the artery is more common than indirect arterial injury, where the artery is injured by shear or distraction forces following joint dislocation and/or bony fracture and displacement. Acceleration or deceleration forces from vehicular accidents and falls from height also cause indirect arterial injury, often leading to aortic disruption. The artery is disrupted from inside to outside. The intima is the least elastic layer and will therefore tear, resulting in an intimal flap. This may cause immediate thrombosis and obstruction or cause delayed obstruction. As the vessel is further deformed, the media is progressively disrupted until only the elastic adventitia maintains continuity. Finally the vessel is completely disrupted.

Penetrating injury may be caused by a stab wound, firearm injury (which may be of low or high energy) or blast injury. Iatrogenic penetrating injury is increasingly common due to percutaneous transluminal vascular procedures and may account for up to 40% of all penetrating vascular trauma. The result of penetrating injury is partial or complete transection of a vessel, and flow distal to the injury is interrupted. There is often brisk bleeding and/or the formation of a haematoma. In high energy gunshot wounds, arteries distant from the immediate track of the bullet may be damaged within a wide area of devitalised and contaminated tissue due to the cavitation effect. Shotgun injuries also cause widespread tissue injury with multiple sites of penetration, and bomb blasts may lead to a combination of all of the effects mentioned above, with additional thermal injury.

**Recognition**

The pre-hospital information should provide details about the mechanism of injury and the type (pulsatile and bright red or oozing and dark) and the estimated amount of any blood loss. Any pre-existing vascular disease should be noted.

The examination should be performed in the context of the ATLS® resuscitation protocol with the proviso that any obvious substantial and on-going extremity bleeding should be controlled as a priority. This is recognised in the UK by the adoption of ‘<C>’ before ABCDE in the trauma algorithm in many trauma texts. Active bleeding should be controlled by direct pressure and packing. Tourniquets are now being used in military practice for extremity haemorrhage control and this is likely to become more common in civilian practice, especially in view of the relatively short pre-hospital times.
Clinical signs of vascular injury may be divided into hard and soft signs as shown in Table 15. Where there is any doubt or where the injury makes distal pulses difficult to evaluate, arterial Doppler pressure measurement is very useful. An arterial pressure index (systolic arterial pressure distal to the injury in the involved limb, divided by the systolic pressure in an uninvolved arm) of more than 0.9 reliably excludes significant occult arterial injury.

**Table 15: Signs of vascular injury**

<table>
<thead>
<tr>
<th>HARD SIGNS OF VASCULAR INJURY</th>
<th>SOFT SIGN OF VASCULAR INJURY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active pulsatile bleeding</td>
<td>History of severe blood loss</td>
</tr>
<tr>
<td>Shock, with ongoing bleeding</td>
<td>Diminished distal pulses</td>
</tr>
<tr>
<td>Absent distal pulses</td>
<td>Injury of anatomically adjacent or related structures</td>
</tr>
<tr>
<td>Symptoms and signs of acute limb ischaemia: the six ‘P’s (pain, paralysis, paraesthesia, pallor, pulselessness and poikilothermia)</td>
<td>Small non-expanding haematoma</td>
</tr>
<tr>
<td>Expanding or pulsatile haematoma</td>
<td>Multiple fractures and extensive soft tissue injury</td>
</tr>
<tr>
<td>Bruits/thrills over the area of injury</td>
<td></td>
</tr>
</tbody>
</table>

There are also other considerations to be borne in mind in the assessment of vascular injury. The site of vascular injury and the presence of pre-existing vascular disease, both of which influence the collateral circulation, will determine the extent of and the severity of the sequelae of vascular injury. Injury to accompanying veins, local nerves and bones, as well as the level of soft tissue contamination, will all exacerbate the vascular injury. Symptoms and signs may change as a previously patent artery that has been damaged begins to thrombose and occlude. Thrombus may then propagate and cause increasingly severe acute ischaemia as collateral vessels become involved. Nerve and muscle damage occurs within 4–6 hours and most muscle impairment is irreversible within 12 hours, hence regular and repeated clinical examination is necessary where vascular injury is likely.

**Resuscitation**

In vascular injury, in particular, it is important to strike the correct balance between pre-operative optimisation of a surgical patient and the need for surgery as part of the resuscitation process. In a haemodynamically stable patient with a vascular injury, minimal or moderate fluid resuscitation is indicated, permitting relative hypotension, as is the case in the ruptured abdominal aortic aneurysm. In an unstable patient resuscitation is best conducted in the operating theatre.

Other resuscitation techniques, in addition to controlling active bleeding with direct pressure, packing or tourniquets, include reduction of (fracture) dislocations and displaced fractures, and stabilisation of bony injury to better contain and limit the bleeding and to protect the soft tissues from further trauma.

**Investigation & interventional radiography**

Chest and pelvis x-rays may have been performed as part of the trauma protocol as well a Focused Assessment with Sonography for Trauma (FAST) scan. These may indicate vascular injury in the chest and pelvis. In unstable patients with hard signs of vascular injury, and in particular active bleeding or distal ischaemia, the next stop should be theatre. This will often be the case in penetrating vascular injury.

In more stable patients, and especially those with blunt injury, the skeletal injuries may also have been imaged and may indicate the possibility of extremity vascular injury. In stable patients, computed tomography with arterial phase contrast (CTA) has become the investigation of choice.
vascular trauma and the principles of vascular surgery

This examination is particularly valuable in the neck, chest, abdomen and pelvis but the extremities can also be imaged and it opens the door for interventional radiological procedures in vascular trauma. Where an interventional suite is available on a 24 hour basis, angio-embolisation of arterial bleeding from pelvic vessels and from vessels supplying the solid abdominal organs is possible. This may be done as a stand-alone procedure for haemorrhage control or as an adjunct to a surgical procedure, such as pelvic packing or a laparotomy and packing. Temporary vascular control may be achieved with percutaneous balloon occlusion of the vessel proximal and distal to the injury pending definitive surgical repair. Thoracic aortic disruption can also be detected and managed endovascularly by stent graft insertion, as can some of the more inaccessible vessels in the thoracic outlet and neck.

Diagnostic angiography has largely been supplanted by CTA but is an integral part of the interventional radiographic therapeutic process described above. Other imaging modalities of limited use in trauma are colour duplex ultrasound and magnetic resonance angiography and are only used for surveillance in a patient managed expectantly or for screening and diagnosis in the absence of hard signs.

Management & the principles of vascular trauma surgery

With few exceptions surgery should be performed under general anaesthesia with the patient placed supine on the operating table with the arms abducted to 90° – the ‘trauma-T’ position. Systemic heparinisation is not indicated but antibiotic prophylaxis is established in vascular trauma. The skin should be widely prepared to include the anatomical area proximal and distal to the injury. This is particularly important in vascular trauma as exposure is the key to success and proximal and distal control may have to be obtained in adjacent anatomical areas, for example, in a groin injury it may be necessary to go via the abdomen to achieve proximal control. An uninjured leg may also have to be prepared for the harvest of long saphenous vein in the event of a bypass or interposition graft being required.

The artery proximally and distally must be identified and clamped some centimetres from the site of the injury to allow room to inspect and adequately débride the vessel. There is often more extensive intimal damage than might be expected from inspection of the outside of the vessel and all must be repaired or resected until normal intima is reached. The inflow and outflow (back-bleeding) of the proximal and distal ends of the injured vessel must be checked and any clot removed by careful use of an embolectomy catheter. The cleared vessel must then be flushed proximally and distally with heparinised saline.

Repair of the vessel may be direct suture, in the case of a simple laceration, taking care not to create a stenosis, or by patch angioplasty with local or long saphenous vein (or rarely a prosthetic material). An end-to-end anastomosis may be preferred after resection of a short length of damaged artery. This often requires further mobilisation of the vessel ends to achieve approximation without tension. Most commonly an interposition graft is required and in some cases a bypass graft is indicated.

The competing priorities in a trauma patient with an extremity vascular injury are the total physiological burden of injury versus the need to revascularise the extremity within the 4–6 hour window before the onset of neuro-muscular degeneration. Where the total burden of injury threatens the life of the patient, surgery must be limited to the minimum possible to save life and limb. In this damage control surgery mode, arterial shunting may be employed on a temporary basis until the patient’s physiology can withstand further definitive arterial reconstructive surgery. Likewise, arterial repair should precede bony fixation (it pays to anticipate any lengthening that may occur with alignment of the bones) and débridement of all devitalised and contaminated soft tissue is necessary. Finally, coverage
of the arterial repair is essential if the wound is to be left open initially and closed by delayed primary suture.

Fasciotomies should be performed in injured limbs with vascular injuries. There are few, if any, disadvantages in doing so and it should be performed before revascularisation. Venous reconstruction improves the outcome of arterial reconstruction and should also be performed before arterial reconstruction, where it can be done simply and quickly. Reperfusion injury should be anticipated and the anaesthetist informed before releasing vascular clamps. Measures to protect the kidneys should be taken by creating a diuresis, alkalinising the urine, monitoring the serum potassium and other techniques.

In damage control surgery, ligation and amputation are also options when required to save the life of the patient with obvious sequelae for the affected limb. Amputation is a two consultant decision, should involve the patient and family members and like all surgical decisions, depends on various factors. These factors include the skill-set of the surgeon, the injury burden of the patient and the physiology of the injured patient (is damage control surgery required?) and the number of casualties to be treated in the available facilities.

LEARNING POINTS: Vascular trauma and the principles of vascular surgery

- Manage substantial ongoing haemorrhage with pressure, packs or tourniquets – the \textit{C} in \textit{ABCDE}
- Clinical evaluation is key in determining vascular injury
- Surgical or radiological intervention is mandatory in all haemodynamically unstable patients and/or those with hard signs of vascular injury
- An arterial pressure index of more than 0.9 distal to the extremity trauma site reliably excludes occult vascular injury
- Symptoms and signs of vascular injury may evolve with time and regular repeated clinical examination is necessary
- In unstable patients surgery is part of resuscitation, whilst in stable patients permissive hypotension is indicated
- CTA is the investigation of choice in stable patients and is the gateway to interventional radiological procedures
- Wide exposure, often across anatomical areas, facilitates proximal and distal control of the injured vessels
- The artery will often have suffered more intimal damage than the adventitial layer and this must be resected or repaired before the final arterial repair
- Damage control techniques (shunting, ligation and amputation) must be employed where the physiological burden of injury prevents definitive repair
- Arterial continuity should be restored before bony fixation
- Fasciotomies should be liberally used and consideration given to early venous repair and reperfusion injury after restoration of continuity

Further reading
Introduction

Acute limb ischaemia (or acute critical ischaemia) is a deterioration in the blood supply of a previously stable leg that results in rest pain and/or other features of severe ischaemia of less than 2 weeks’ duration. Incidence is between 13 and 17 per 100,000 of the population per year, which amounts to 30 or 40 cases annually presenting to an average district general hospital serving a population of 250,000. The risk of amputation and a prolonged hospital stay is associated with a high cost to the community and this can only be minimised by rapid clinical assessment, recognition, appropriate resuscitation and a clear understanding of the available investigations and therapeutic management options run by a 24-hour vascular consultant-led service.

Aetiology

The aetiology of acute limb ischaemia is occlusion of a native artery or bypass graft. This is most commonly caused by embolism and in-situ thrombosis. There are a number of other, rarer causes (listed in Table 16). Emboli arise in, and become dislodged from, the heart or more proximal vessels. Today 80% of cardiac emboli arise as a result of atrial fibrillation in ischaemic heart disease; mural thrombus following myocardial infarction accounts for the remainder. Larger emboli impact at a site of peripheral arterial bifurcation, particularly in the common femoral and popliteal arteries, thereby occluding flow. Smaller emboli impact in the distal vessels of the foot, resulting in acute “blue-toe syndrome”. Emboli may consist of a platelet-rich thrombus in the case of cardiac embolism, or an atherosclerotic plaque or cholesterol-rich emboli from proximal atherosclerotic vessels.

Table 16: The aetiology of acute limb ischaemia. Adapted from ‘Management of Acute Limb Ischaemia’ in A Companion to Specialist Surgical Practice: Vascular and Endovascular Surgery Eds Beard JD & Gaines PA

<table>
<thead>
<tr>
<th>EMBOLISM</th>
<th>THROMBOSIS</th>
<th>OTHER CAUSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial fibrillation</td>
<td>Atherosclerosis</td>
<td>Dissection</td>
</tr>
<tr>
<td>Mural thrombus</td>
<td>Bypass graft occlusion</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>Trauma</td>
<td></td>
</tr>
<tr>
<td>Valvular vegetations</td>
<td>Pro-thrombotic conditions</td>
<td>Popliteal entrapment</td>
</tr>
<tr>
<td>Proximal aneurysms</td>
<td>Popliteal aneurysm</td>
<td>Compartment syndrome</td>
</tr>
<tr>
<td>Atherosclerotic plaque</td>
<td>–</td>
<td>External compression</td>
</tr>
</tbody>
</table>
In-situ native arterial vessel or graft thrombosis is now the most common cause of acute limb ischaemia owing to the reduction in rheumatic heart disease and the anticoagulation of patients in atrial fibrillation. The population is ageing and there is a commensurate increase in atherosclerosis. Thrombosis is usually caused by flow arrest at the site of an atherosclerotic stenosis in a native artery or following a reduction in graft flow from a stenotic lesion within the conduit, compromised outflow or reduced inflow. In a prosthetic graft, thrombosis may develop in the absence of a stenotic lesion. Distal embolisation from a popliteal aneurysm occluding the tibial vessels or thrombosis of the aneurysm itself is a particularly malignant cause of acute limb ischaemia. In 20% of patients no source of thromboembolism will be found.

**Definitions**

*Acute limb ischaemia* (acute critical ischaemia) is defined above. Patients with in-situ native arterial vessel thrombosis often present with less severe ischaemia than those with acute limb ischaemia from embolic disease, possibly because of pre-formed collateral vessels around diseased arterial trunks. The condition can be considered as a subset of acute limb ischaemia often known as *acute-on-chronic* or *acute subcritical limb ischaemia*.

*Critical limb ischaemia* is defined as a deterioration in the blood supply of a limb resulting in rest pain or tissue loss of more than 2 weeks’ duration. It is usually associated with a history of peripheral vascular disease, or *chronic limb ischaemia* and ankle brachial pressure indices of <0.3 or an absolute pressure at the ankle of <50 mmHg.

**Recognition**

The most important factor affecting the outcome of the leg is the severity of ischaemia at presentation. A clinical classification of acute limb ischaemia has been developed which is designed to stratify limbs into defined groups for decision-making purposes and it has been shown to correlate with outcome. The classification is summarised in Table 17.


<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>PROGNOSIS</th>
<th>FINDINGS</th>
<th>DOPPLER SIGNALS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Sensory loss</td>
<td>Muscle weakness</td>
</tr>
<tr>
<td>I Viable</td>
<td>Not immediately threatened</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>IIA Non-viable or threatened</td>
<td>Salvageable if promptly treated</td>
<td>None or minimal</td>
<td>None</td>
</tr>
<tr>
<td>Marginal</td>
<td></td>
<td>(toes)</td>
<td></td>
</tr>
<tr>
<td>IIB Non-viable or threatened</td>
<td>Salvageable only with immediate</td>
<td>Moderate</td>
<td>Mild or moderate</td>
</tr>
<tr>
<td>Immediate</td>
<td>revascularisation</td>
<td>(foot &amp; calf)</td>
<td></td>
</tr>
<tr>
<td>III Unsalvageable or irreversible</td>
<td>Major tissue loss and permanent</td>
<td>Profound</td>
<td>Profound</td>
</tr>
</tbody>
</table>
However, it is difficult to distinguish between categories IIa and IIb and between categories IIb and III. Therefore, there are two critical decisions:

- **first**, to determine whether the limb is viable or not viable and in need of urgent therapeutic intervention;
- **second**, to determine whether the limb is salvageable with immediate revascularisation or non-salvageable and in need of primary amputation.

Complete occlusion of a proximal artery in the absence of pre-formed collaterals results in the classic clinical presentation of **pain**, **paralysis**, **paraesthesia**, **pallor**, **pulselessness** and **pallor or perishing cold**. Such a limb is non-viable but eminently salvageable. The pain is severe and resistant to analgesics. Sensorimotor deficits indicate nerve and muscle ischaemia. Even in more prolonged acute limb ischaemia that results in tender muscles and tense compartments (indicating muscle necrosis) and the development of a mottled appearance which blanches on pressure (due to deoxygenated blood pooling in the capillaries) the limb may still be salvageable with prompt intervention. If flow is not restored quickly the mottling becomes fixed due to propagation of thrombus in the arteries distal to the occlusion and rupture of the capillaries. At this stage the ischaemia is irreversible and the limb is unsalvageable.

Despite early recognition of acute limb ischaemia and expedited revascularisation procedures the limb loss rate is about 10–30% and the 30-day mortality is approximately 15%. Prompt treatment is the most important factor in saving a non-viable leg. It has been shown that the interval between onset of acute limb ischaemia and exploration is proportional to the amputation rate (6% if within 12 hours; 12% if between 12 and 24 hours; 20% if more than 24 hours). It is also recognised that the limb salvage rate is lower in thrombosis than in embolism, and that the mortality is higher in embolic acute limb ischaemia due to underlying cardiac disease. Finally, immediate anticoagulation on diagnosis with therapeutic levels of heparin reduces morbidity and mortality.

### Resuscitation

Patients presenting with acute limb ischaemia are usually in a poor state of health. They are often dehydrated, in cardiac failure, hypoxic and in pain. These issues should be addressed as part of the initial management with oxygen, judicious intravenous fluids and analgesia via an infusion pump. Intravenous heparin (5000 units) should be given immediately and an infusion commenced to maintain the activated partial thromboplastin time (APTT) at twice to three times above normal to restrict thrombus propagation. The latter is often poorly managed as it involves regular re-reviews of the patient and blood samples to be sent to the laboratories every 2 hours initially. Heparin does not dissolve embolic material or thrombus, but is associated with an improved outcome.

### Investigations

A history from the patient may reveal relevant cardiac disease such as myocardial infarction and/or atrial fibrillation, or a known malignancy or other prothrombotic tendency. There may also be a history of peripheral vascular disease, previous bypass procedures, aneurysmal disease or multiple deep venous thromboses. Examination of the patient’s contralateral limb may reveal absent pulses or a popliteal aneurysm.

Venous bloods should be sent for a full blood count, electrolytes and glucose. An ECG and chest radiograph may be useful in diagnosing and managing cardiac arrhythmias and aneurysms in the chest. If a prothrombotic
tendency is suspected, a thrombophilia screen needs to be taken before heparin is given (if possible) and discussed with the haematologists.

Additional more complex investigations may be indicated, but they should not delay surgery in a non-viable or threatened acutely ischaemic limb. A CT angiogram will reveal the nature and extent of the thromboembolic disease and may better direct surgery, as well as possibly detecting aortic thrombus and aneurysmal disease in suspected embolic disease. Echocardiography is necessary to exclude a cardiac source of embolism. Duplex and angiography are useful, especially in in-situ native arterial vessel and bypass graft thrombosis, and thrombolysis may be attempted in selected patients.

Management

Once a non-viable, threatened, or irreversibly ischaemic limb is recognised the therapeutic options depend on the severity of the ischaemia. Clearly unsalvageable limbs need primary amputation, but in a few patients, who are in a moribund state, the leg problem heralds the end of life and terminal care is indicated.

Revascularisation of a late presentation of acute limb ischaemia is dangerous, as the hypoperfused limb causes systemic acid–base and electrolyte disorders that impair cardiac function. On reperfusion of a large mass of ischaemic tissue the increased release of blood with a low pH and a high potassium into the systemic circulation – together with myoglobin which impairs renal function and the systemic inflammatory reaction caused by neutrophil activation – can lead to multiorgan dysfunction syndrome and death. The reperfusion injury also causes swelling of the muscles within the fascial compartments and further neuromuscular damage within the limb itself.

Management of the acutely ischaemic limb also depends, to some extent, on the aetiology. In an acute, marble-white extremity with neurosensory deficit, embolism is more likely, especially where there is no previous history of peripheral vascular disease, no atrial fibrillation, and a normal contralateral limb. Urgent embolectomy, after resuscitation and systemic heparin therapy, is indicated in these patients to prevent limb loss. Angiography is not indicated as it delays revascularisation. Femoral and/or below-knee popliteal exploration is necessary in the leg and brachial exploration in the arm. Consideration should always be given to fasciotomies in the calf or forearm, ideally before embolectomy.

In the majority of patients with acute limb ischaemia the picture will not be so clear. These patients have acute-onset pain but no paralysis and little or no sensory loss. They often have a history of peripheral vascular disease and absent or reduced pulses in the contralateral limb. There may be surgical scars suggesting bypass procedures. These limbs are often viable and there is time to investigate and plan the therapeutic options. Here duplex, CT angiography and arteriography are of value, as thrombectomy is less likely to reperfuse the atherosclerotic limb and formal arterial bypass is often required. Thrombectomy can be combined with on-table angiography, direct intra-arterial thrombolysis and balloon angioplasty to achieve reperfusion in difficult situations.

Angiography and catheter-directed thrombolysis has a role to play in these situations. Arguably it is less invasive than surgery and can re-open smaller arteries as well as the main arterial trunks and bypass grafts. It can also unmask atherosclerotic stenoses, the probable cause of the in-situ thrombosis, and direct further treatment, such as radiological balloon or surgical angioplasty. Severe haemorrhage is a complication of intra-arterial thrombolysis and the risk of intracranial haemorrhage is 1–2%.
Compartment syndrome

Compartment syndrome is a condition in which there is increased pressure within a limited space which compromises circulation and tissue function in that space. Reperfusion of ischaemic muscle can result in considerable swelling of compartmentalised muscle groups, especially in the calf, forearm and thigh. Other causes of compartment syndrome are listed in Table 18.

Table 18: Causes of acute compartment syndrome. Adapted from reference 3 in the text: ‘Acute Compartment Syndrome of the Limb’ Kostler W, Strohm PC & Sudkamp NP

<table>
<thead>
<tr>
<th>Vascular</th>
<th>Vascular injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Reperfusion injury</td>
</tr>
<tr>
<td></td>
<td>Haemorrhage</td>
</tr>
<tr>
<td></td>
<td>Phleghmasia caerulesa dolens</td>
</tr>
<tr>
<td>Orthopaedic</td>
<td>Fractures</td>
</tr>
<tr>
<td></td>
<td>Fracture surgery</td>
</tr>
<tr>
<td></td>
<td>Pulsatile lavage</td>
</tr>
<tr>
<td>Soft tissue</td>
<td>Crush injury</td>
</tr>
<tr>
<td></td>
<td>Burns</td>
</tr>
<tr>
<td></td>
<td>Prolonged compression</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Arterial puncture in anticoagulated patients</td>
</tr>
<tr>
<td>Occasional</td>
<td>Overuse of muscles</td>
</tr>
</tbody>
</table>

Symptoms and signs

The symptoms and signs of compartment syndrome include disproportionate pain which is exacerbated by passive stretching of the involved muscles, a swollen, tense compartment (or compartments) and rapid progression of these signs. Paraesthesia follows and paralysis and pulselessness are late signs. Knowledge of the nerves within the fascial compartments and their motor and cutaneous distributions may aid early diagnosis. Clinical suspicion is the mainstay of early diagnosis and is vital for preventing an adverse outcome, but this can be difficult in patients who are less than fully conscious. Compartment pressures can be measured if there is clinical uncertainty. The normal pressure is 10–12 mmHg. Absolute compartment pressures above 20–30 mmHg are usually abnormal. The mean compartmental perfusion pressure is the mean arterial pressure minus the compartment pressure, and it should be greater than 70 mmHg. All relevant compartments must be measured. The creatinine kinase may also be massively elevated where there is muscle damage or ischaemia.

Resuscitation

Resuscitation involves supplemental oxygen to improve tissue oxygenation and all circumferential dressings should be removed. Hypotension should be treated to improve the compartmental perfusion pressure. The treatment for a compartment syndrome is fasciotomy. Fasciotomy is most commonly necessary in the leg, and here a four-compartment fasciotomy is indicated, performed through generous skin incisions. “Percutaneous” fasciotomy is dangerous. Prophylactic fasciotomy should be performed where the arterial supply has been interrupted for 4–6 hours or more, in crush injuries, in patients with major vein ligation (especially around the knee and in the calf) and in patients who undergo open fracture fixation near a compartment at risk. The fasciotomy should be performed prior to attempts at revascularisation or bony fixation.

Missed compartment syndrome results in damaged muscle replacement with scar tissue. Subsequent contracture and traversing peripheral nerve neuropathy leads to severe and permanent dysfunction.
LEARNING POINTS: Acute limb ischaemia

• The non-viable or threatened limb is in urgent need of intervention.
• The unsalvageable limb needs primary amputation.
• Heparin is part of resuscitation and prevents thrombus propagation and improves outcomes in acute limb ischaemia. It does not dissolve emboli or thrombi. The APTT must be regularly monitored.
• Embolus and thrombus in acute limb ischaemia are often managed differently but can be difficult to distinguish.
• Embolectomy is the mainstay of treatment in embolic acute limb ischaemia.
• Angiography and thrombolysis may be considered, as well as thrombectomy and bypass surgery, in thrombotic acute limb ischaemia.
• Always consider fasciotomies in acute limb ischaemia.

Further reading
Technical skills

Vascular embolectomy

Introduction
Acute ischaemia of the upper or lower limb represents a vascular surgery emergency. Embolectomy remains the cornerstone of treatment. Preoperatively, ensure the patient is fully heparinised. Successful embolectomy may mean the difference between the survival or loss of the patient’s limb and all the associated quality of life and cost implications of this. In the following example a porcine aorta is used as the femoral vessels are very small.

Step 1
- Having exposed the vessel in question (usually the femoral or brachial artery) it is imperative to gain both proximal and distal control.
- This is achieved by dissecting out the exposed portion of the vessel from all surrounding tissues; the same principle applies to any visible branches.

Step 2
- Care should be taken to limit contact with the vessel itself because this may lead to vasospasm or iatrogenic damage.

Step 2
- Placement of vascular slings which encircle the vessel prevents unwanted haemorrhage.
- The O’Shaughnessy (right-angled) clip is particularly useful for this procedure.
- Some surgeons wisely guard against the use of red vascular slings in a surgical field that is likely to be contaminated by fresh bleeding.

Step 3
- There are different approaches to the artery, but here we describe a transverse arteriotomy.
• A stab incision should be carefully made using an 11 blade. Take care not to injure the posterior wall of the vessel.
• Extend the incision using Pott’s scissors to allow adequate access for a Foley catheter.

**Step 4**
- The Foley catheter has a balloon at the distal end which can be inflated with either air or saline. This should be tested before use.
- Assuming the embolism is distal to your arteriotomy, pass the deflated Foley down the artery, past the blockage and inflate.
- Withdraw the balloon. Adjust the pressure in the balloon to prevent stripping off the intima.

**Step 5**
- Once blood flow has been re-established, the vessel should be heparinised. Closure can use either an interrupted or a continuous technique; however, if narrowing of the vessel is a risk then a patch closure should be used.
- Here we describe a continuous technique using a Prolene™ suture.

**Step 6**
- Secure the suture at each end of the wound.
- Work towards the middle, ensuring that the first bite is upstream from adventitia to intima and the second is downstream from intima to adventitia. This ensures that the suture follows the direction of flow and an intimal flap is not raised.
Vascular emergencies: Ruptured abdominal aortic aneurysm

Introduction
Aneurysms are focal dilatations in the diameter of arteries of at least 50% that of the non-dilated adjacent vessel. Population screening suggests that the prevalence of abdominal aortic aneurysm (AAA) in the UK is approximately 7–8% among men over the age of 65. This is an incidence in the population over 65 of 35.5 per 100,000. The prevalence of AAA is six times greater in men than in women, and in women they tend to occur one decade later in life. The prevalence is increasing, and in men ruptured abdominal aortic aneurysm (rAAA) is the seventh most common cause of death in the UK.

The majority of these aneurysms remain asymptomatic until the point of rupture. Most (up to 75%) are detected during this asymptomatic phase as an incidental finding during the investigation of unrelated conditions (especially by urologists) and sometimes a patient will notice a pulsatile mass. Symptoms of an enlarging abdominal aneurysm may include backache and abdominal pain. Signs may include tenderness over an expansile, pulsatile mass in the epigastrium. Peripheral embolisation from these aneurysms is rare.

Despite increasing asymptomatic detection and a rise in elective repairs the incidence of ruptured abdominal aortic aneurysm is still rising. Rupture is associated with a 50–75% mortality rate before hospitalisation. Of those who do make it as far as a hospital, the mortality of surgery for this condition is 40–70%. Thus the cumulative mortality for a ruptured abdominal aortic aneurysm is 80–88% (as high as 95% in some series). Screening programmes have shown that aneurysm-related mortality can be reduced by 53% despite a 6% perioperative mortality rate. There is conclusive evidence now that the incidence of ruptured abdominal aortic aneurysm and its attendant death rate can be reduced by screening the at-risk population.

Recognition
Free rupture into the pericardial cavity (20%) is rapidly fatal and probably accounts for the majority of pre-hospital deaths. A rupture into the retroperitoneum (80%) may be contained by a combination of tamponade and hypotension. This is an unstable and transient phase, but provides a window of opportunity for emergency lifesaving surgery. It is arguably the surgical emergency par excellence. Left untreated, 100% of people with ruptured abdominal aortic aneurysm die within hours.

Presentation
Rupture classically presents as a triad of sudden onset of severe abdominal or back pain, hypotension and collapse or syncope, and a palpable, pulsatile abdominal mass. Other common symptoms include groin or flank pain (especially on the left side). Other signs include reduced or absent groin pulses, signs of hypovolaemia, haematuria and groin hernia (often incarcerated) as a result of the increased intra-abdominal pressure, and evidence
of retroperitoneal bleeding can be seen as a bluish discoloration of the flanks (Grey–Turner sign) and around the umbilicus (Cullen sign).

**Diagnosis**

The diagnosis of ruptured aneurysm is often not quite clearcut. It can be difficult to palpate an abdominal mass, particularly in the obese, hypotensive patient, and this frequently confuses the diagnosis. The differential diagnosis includes other abdominal catastrophes, such as severe acute pancreatitis and ischaemic bowel, as well as myocardial infarction. Further differential diagnoses include renal colic, diverticulitis, gastrointestinal haemorrhage and a perforated duodenal ulcer. An incorrect diagnosis is made in up to one-third of all cases.

**Resuscitation**

Unlike in many other conditions the principal resuscitative manoeuvre in ruptured abdominal aortic aneurysm is urgent surgical repair. While this is being arranged the patient should be administered high-flow oxygen. The patient’s blood needs to be taken for urgent cross-matching and blood samples sent to the laboratory for a full blood count, urea and electrolyte determination and a coagulation profile. Do not wait for the results to be made available. The haematologists should be informed and a massive transfusion protocol initiated in order to make large quantities of type O blood, fresh frozen plasma and platelets rapidly available. Large-bore intravenous access should be established. The anaesthetists need to be informed to assess and prepare the patient for theatre. Intravenous, radial arterial and Foley catheter placement should occur simultaneously to avoid delay in transfer to theatre. The theatre team need to be making preparations to receive the patient.

Intravenous fluid resuscitation should be kept to a minimum. The aim of permissive hypotension is the maintenance of consciousness with a systolic pressure at around 80–90 mmHg. If intravenous fluid is necessary then blood is recommended. Aggressive crystalloid fluid resuscitation can dilute coagulation factors, lower the viscosity of the blood and elevate blood pressure to a level where there is rupture of any temporary haemostasis. It may thus destabilise the patient before they arrive in the operating theatre.

**Investigation**

Evidence from the history and examination is often enough to establish the clinical diagnosis. In an unstable hypotensive patient this is all the investigation that can be performed before transfer to the operating theatre. In more stable patients a plain abdominal radiograph may already have been performed as part of the investigation of abdominal pain and will show signs of the rupture in 90% on retrospective analysis. Calcification of the aortic wall beyond its normal limits and loss of a psoas shadow is characteristic. However, it should not be performed once the diagnosis is suspected and it needs expert review. An emergency room ultrasound will confirm an abdominal aortic aneurysm, but is not sensitive to extraluminal blood and is rarely necessary or indicated.

A computed tomography angiogram (CTA), if the patient is stable enough, has become the investigation of choice in the diagnosis of ruptured abdominal aortic aneurysm. Newer-generation multislice scanners in the emergency department allow this investigation to be done within the same timescale as a plain film. They can detect extraluminal blood with a 100% positive predictive value and they provide important information on aneurysm morphology and associated abnormalities and conditions. Importantly, the evolution of ruptured endovascular aneurysm repair (rEVAR) means the patient can be considered for this form of treatment. Nevertheless, in an unstable patient, immediate transfer to the operating theatre is the only therapeutic option.
Management and communication

There is usually only one decision to be made once the diagnosis of ruptured abdominal aortic aneurysm is made—that is, whether to attempt surgical treatment or to withhold care. The majority of patients will be offered or given treatment in the form of an open or endovascular repair. In this case the driving force must always be towards theatre and the application of an arterial clamp (or towards the endovascular suite and a rEVAR if indicated). Unhelpful investigations and unnecessary delays while multiple lines are inserted must be avoided.

Communication

Decisive leadership and clear communication by the admitting surgeon, at whatever level, is paramount. Immediate involvement of a consultant vascular surgeon and anaesthetist is mandatory, and the interventional radiologist must be consulted and review the patient’s CTA if a rEVAR is to be considered. It is also helpful at this stage to speak to (and consent) the patient, if possible, and to discuss the prognosis with the patient and any relatives.

Withholding care

The decision to withhold care is made at consultant level, often in consultation with the anaesthetists and the patient. Often, a prior decision has been made not to offer the patient elective aneurysm repair due to substantial operative risks. However, when such a patient presents with a rupture, the decision must be revisited. The odds have changed in the risk–benefit analysis to certain death in the event of no intervention, compared to a slim chance with surgery. Faced with this decision many patients opt for surgery.

Scoring systems

In a climate of cost containment, the possibility of withholding repair in some patients whose death can be predicted with certainty has been investigated by several authors. Various scoring systems have been developed with the aim of identifying which patients are unlikely to survive surgery, based on several preoperative factors. Two such systems are the Hardman Index and the Glasgow Aneurysm Score.

Hardman Index: As shown in Table 19, this has five parameters and scores patients between 0 and 5. A score of 3 or more is considered to indicate a high risk of surgical mortality.

Glasgow Aneurysm Score: As shown in Table 20, this also has five parameters ranging from the patient’s age in years to that age plus 48. A score of over 95 is considered high risk.

Table 19: The Hardman Index

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age over 76</td>
<td>1</td>
</tr>
<tr>
<td>Haemoglobin 9 g/dL</td>
<td>1</td>
</tr>
<tr>
<td>Serum creatinine &gt;190 µmol/L</td>
<td>1</td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>1</td>
</tr>
<tr>
<td>Ischaemic change on ECG</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 20: The Glasgow Aneurysm Score

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years of age)</td>
<td>17</td>
</tr>
<tr>
<td>Shock</td>
<td>14</td>
</tr>
<tr>
<td>Renal disease</td>
<td>10</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>7</td>
</tr>
<tr>
<td>Myocardial disease</td>
<td>7</td>
</tr>
</tbody>
</table>
Doubt has been cast on the predictive value of these scoring systems and at best they can be regarded as useful in assisting the surgeon in making clinical decisions. Such tools can only support but never replace good clinical judgement.

### Treatment

The treatment of ruptured abdominal aortic aneurysm is open surgical repair. The patient should be supine on the table, with the skin prepared and draped before induction of anaesthesia. The priority at surgery is to gain rapid proximal control by the application of a clamp to the aortic neck. Thereafter, the operation can proceed at a more normal pace. Where the clamp is difficult to site due to a massive haematoma, pressure on (or clamping of) the supracoeliac aorta can temporarily stabilise the patient’s condition until more senior help arrives.

However, in those relatively stable patients who have undergone a CTA and have favourable anatomy, consideration can be given to rEVAR in some units with the expertise and available stent stock to allow emergency intervention. The important anatomical considerations for rEVAR are an infrarenal neck of sufficient cylindrical length (at least 15 mm) and iliacs that will permit the passage of the necessary large introducers (at least 7–8 mm in diameter). An aorto uni-iliac device with a femoro-femoral crossover graft is often used in this situation.

### LEARNING POINTS: Ruptured abdominal aortic aneurysm

- Abdominal aortic aneurysms affect 7–8% of the male population over the age of 65.
- They usually only become symptomatic when they rupture.
- Less than 50% of all ruptured aneurysm patients survive to reach hospital.
- Screening for abdominal aortic aneurysms will reduce the 80–90% mortality rate from rupture.
- Abdominal or back pain, hypotension (and collapse or syncope) and a pulsatile abdominal mass is pathognomonic of a ruptured abdominal aneurysm.
- The best investigation, in a temporarily stable patient, is emergency room CTA.
- Permissive hypotension and rapid transfer to theatre for surgical or endoluminal repair are key to resuscitation and treatment.
- Good clinical judgement is necessary to determine those patients from whom futile surgical repair should be withheld.

### Further reading

Trauma laparotomy

Introduction
The primary goals of trauma laparotomy are:

- rapid control of exsanguination;
- control and minimisation of bowel spillage;
- avoidance of the lethal triad: hypothermia, coagulopathy and acidosis.

Prior to undertaking such a process, however, it is critical that one ensures a safe, but rapid, delivery of the patient to the operating theatre. Furthermore, an open (and ongoing) communication with the rest of the trauma team should be established early on. These members include the anaesthesia and nursing teams and adjunct services such as interventional radiology and critical care teams.

Ancillary and support staff are usually limited during the time of day these cases generally arrive, therefore the trauma team leader must engage in a very proactive role to optimise patient care. Remember to identify those instruments or trays you anticipate needing; request blood-salvage systems and or fluid warmers or infusers; tell staff clearly how you would like the patient to be positioned, prepared and draped.

Unless he or she is in extremis, the patient should be prepared and draped in the sterile fashion, preferably from the chin to the knees. While the surgeon on the right of the patient should be helping the scrub nurse drape the patient and throw off suction tubing and electrocautery cables and so on, the assistant (or co-surgeon) on the left side of the patient should be setting up a self-retaining retractor. The post of the retractor is typically positioned between the chest and the abdomen on the left side of the table, to allow for maximal abdominal retraction and unobstructed access to the left chest.

General aspects
Some useful adjuncts to a successful trauma laparotomy are shown in Table 21.

Table 21: Useful adjuncts to a successful trauma laparotomy

<table>
<thead>
<tr>
<th>Adjunct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headlight</td>
</tr>
<tr>
<td>High-power suction</td>
</tr>
<tr>
<td>Argon beam coagulator</td>
</tr>
<tr>
<td>Haemostatic agents</td>
</tr>
<tr>
<td>Blood bank notification for massive transfusion</td>
</tr>
<tr>
<td>Another assistant</td>
</tr>
<tr>
<td>Intraoperative blood salvage</td>
</tr>
</tbody>
</table>

The incision should be large enough to accommodate rapid and adequate exposure for a thorough exploration. As such, the midline incision is preferred in almost all cases, spanning from xiphoid to pubis. Classic teaching ascribes the use of a knife to cut through the skin,
subcutaneous tissues and the fascia in three broad strokes. Once the preperitoneal fat is exposed, the abdominal cavity is entered with a blunt finger push just above the umbilicus, where the peritoneum is the thinnest. The peritoneal cavity is then fully opened with Mayo scissors, taking care to avoid iatrogenic injury to the liver, bowel and bladder.

Once the abdomen is opened, divide the ligamentum teres between clamps and sharply divide the falciform ligament towards the central tendon to facilitate exposure.

**Initial approach**

Packing starts in the quadrant with most suspicion for an injury and proceeds towards the area of least suspicion for an injury (usually in a clockwise fashion). Use of a large handheld Morris retractor provides adequate exposure for the operator on the contralateral side of the table to systematically pack off one quadrant at a time. Once the abdomen is adequately packed, a self-retaining retractor is quickly placed and the zones of the retroperitoneum are quickly inspected. Ensure there is not a large or expanding haematoma that needs to be addressed immediately. If packing is unsuccessful at staunching the haemorrhage, it is likely the patient has an active intraperitoneal bleed from the great vessels or the portal/retrohepatic area.

Next, the quadrants and pelvis are unpacked in a counter-clockwise fashion, beginning in the quadrant where you do not expect an injury.

Injuries to the portal structures should be explored regardless of the mechanism or appearance of the haematoma. Vascular control is first attempted with a Pringle manoeuvre (occlusion of all three portal structures with a vascular clamp or Rummel tourniquet) proximally and individual vascular control distally. In the absence of brisk, active haemorrhage, the retrohepatic area should not be explored. If required, be prepared to perform total hepatic isolation by controlling the portal, infrahepatic and suprahepatic inferior vena cava.

**Vascular injuries**

Options for simple and efficient vascular control differ depending on whether the injury is arterial or venous and how accessible the vessel is to the surgeon (Table 22). In the case of major arterial injuries, rapid control with vascular clamps is advocated. If the haematoma is somewhat intact, proximal and distal control should be obtained around the area prior to opening the haematomata. If the injury can be repaired with a simple suture, this should be chosen. However, many arterial injuries will require anastomosis or even an interposition graft. Initially, venous injuries are best controlled by manual pressure. While sponge-sticks (rolled-up sponges on ring forceps) may be best for compressing the inferior vena cava or iliac veins, small holes can be controlled with an Allis clamp. Most venous structures can be ligated with impunity. In damage-control situations, where a formal vascular repair would be time-consuming and technically difficult, temporary vascular repair might be the best option. Consider using an Argyle vascular shunt to temporarily bypass the flow in large mesenteric vessels or small, paediatric chest tubes if size appropriate.

**Solid organ injury**

**Liver**

As with any approach in trauma surgery, one must first achieve adequate exposure before investigating hepatic injuries. If not already done, the falciform ligament should be taken down, as well as the triangular ligament on the side of the injury. Packs should be placed both outside the liver between the parenchyma and the thorax and between the liver and the right retroperitoneum. In the presence of bleeding from the parenchymal cavity, this area also be packed with laparotomy pads and or absorbable haemostatic agents. The argon beam coagulator or electrocautery set to “spray” mode can be used to address parenchymal oozing and more minor bleeding. Carefully placed liver sutures will at times help to approximate edges of the injured liver parenchyma with either absorbable
haemostatic material (Gelfoam \textsuperscript{\textregistered} wafer soaked in thrombin) or omental pedicle. Should these efforts fail, a Pringle manoeuvre should be employed, followed by “direct attack” and opening of the injured parenchyma (with suturing or clipping of bleeding vessels if possible). Lateral segmentectomies can be rapidly performed with a stapler if necessary. Prior to closure, remember to place closed-suction drainage to help detect and possibly control a biliary leak.

**Spleen**

The key to a successful trauma splenectomy is rapid mobilisation of the injured spleen towards the midline by taking down its four ligamentous attachments (gastrosplenic, splenorenal, splenocolic and splenophrenic). Dividing the splenorenal ligament allows for maximal mobilisation of the spleen towards the midline, as well as up and into the surgical field. Whereas the other ligaments can be rapidly divided with either electrocautery or scissors, the gastrosplenic ligament should be taken with clips or suture ties (for the short gastrics) and suture ligature (for the splenic vessels). Many surgeons prefer to first divide this ligament rapidly with the use of clamps, then suture ties and ligature once the spleen is passed off the surgical field. Rapid division of the gastrosplenic ligament can often to lead to injury of the tail of the pancreas (leading to pancreatic leaks, fistulas and delayed bleeds) or the stomach body (resulting in necrosis of the gastric wall, leaks and abscesses); not ligating or clipping to close to

<table>
<thead>
<tr>
<th>VASCULAR STRUCTURE</th>
<th>PLAN OF ACTION</th>
<th>POTENTIAL COMPLICATIONS IF VASCULAR STRUCTURE IS LIGATED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>Repair</td>
<td>–</td>
</tr>
<tr>
<td>Infrarenal inferior vena cava</td>
<td>Repair or ligate</td>
<td>Lower extremity venostasis, oedema, deep vein thrombosis</td>
</tr>
<tr>
<td>Suprarenal inferior vena cava</td>
<td>Repair or ligate</td>
<td>Same as infrarenal (also renal failure)</td>
</tr>
<tr>
<td>Coeliac</td>
<td>Ligate</td>
<td>–</td>
</tr>
<tr>
<td>Superior mesenteric artery</td>
<td>Repair or ligate</td>
<td>bowel ischaemia</td>
</tr>
<tr>
<td>Superior mesenteric vein</td>
<td>Repair or ligate</td>
<td>bowel ischaemia</td>
</tr>
<tr>
<td>Portal vein</td>
<td>Repair or ligate</td>
<td>bowel ischaemia</td>
</tr>
<tr>
<td>Hepatic artery</td>
<td>Repair or ligate</td>
<td>Cholecystectomy if ligated</td>
</tr>
<tr>
<td>Common iliac artery</td>
<td>Repair or ligate</td>
<td>Will need extra-anatomic revascularisation of extremity</td>
</tr>
<tr>
<td>External iliac artery</td>
<td>Repair or ligate</td>
<td>Will need extra-anatomic revascularisation of extremity</td>
</tr>
<tr>
<td>Internal iliac artery</td>
<td>Ligate</td>
<td>–</td>
</tr>
<tr>
<td>External/Internal iliac vein</td>
<td>Ligate</td>
<td>Oedema, deep vein thrombosis</td>
</tr>
<tr>
<td>Renal artery</td>
<td>Repair or perform nephrectomy</td>
<td>–</td>
</tr>
<tr>
<td>Left renal vein</td>
<td>Ligate</td>
<td>–</td>
</tr>
<tr>
<td>Right renal vein</td>
<td>Repair or perform nephrectomy</td>
<td>–</td>
</tr>
</tbody>
</table>
the stomach wall can avoid these injuries. Finally, a rolled lap-pad should be used to evaluate the splenic bed for any ongoing bleeding.

**Kidney**

In the absence of severe injury that results in retroperitoneal disruption, the injured kidney has its own effective method of compression to achieve haemostasis (and thus rarely requires removal). However, patients with rapidly expanding haematomas and injuries with a suspicion for major vascular compromise often require nephrectomy. Prior to considering this, however, first check that the contralateral kidney is present. If absent, you can be even more aggressive with attempts at kidney preservation and salvage. Medial visceral rotation and mobilisation of the kidney out of the Gerota’s fascia allows for appropriate exposure and isolation of the vascular pedicle. The most expeditious method for controlling the renal vessels is with a vascular clamp or vascular-load stapler. When possible, the renal vessels (artery and vein) should be ligated separately. Finally, the ureter should be isolated and ligated, taking care to do this as distally as possible.

**Hollow organ injury**

**Small bowel injury**

The exact approach to small bowel injury depends on the presence or absence of haemodynamic instability and the severity of the associated injuries. In cases of patients in extremis, address the haemorrhage first then proceed to contain gross bowel spillage. Gross contamination may be quickly controlled with the placement of ligatures (umbilical ties or heavy silk sutures) proximal and distal to the bowel defect. Once the bleeding and contamination are addressed, the entire length of bowel must be inspected by “running” it from the ligament of Treitz to the ileocaecal valve. Take care to ensure that you examine the leafs of mesentery on both sides and down to its base. Any injuries identified can be controlled with an Allis or Babcock clamp, or even a silk suture. The surgeon should inspect the entire bowel prior to performing any resections to ensure that bowel length is optimised and conserved. Resection is most commonly performed by use of a surgical stapler followed by control of mesenteric vessels with clamps and suture ligature or ties. If the fascia is to be left open for damage-control (DC) purposes, the bowel may be left in discontinuity at the time of the first procedure (DC phase I). Otherwise, bowel continuity is restored with a stapler or hand-sewn anastomosis.

**Stomach and duodenum**

Penetrating injuries that traverse the left upper quadrant mandate a careful inspection of the posterior stomach and gastro-oesophageal junction. Injuries to the stomach are usually easily controlled with suture or clamp, and formally repaired by resection with a stapler or simple suture. The pylorus, duodenal sweep and ligament of Treitz should be carefully inspected for evidence of haematoma and, in penetrating injuries, for any defects of the bowel wall. Approaches to repair include simple oversewing of serosal defects and two-layered repair and pyloric exclusion. In the presence of haemodynamic abnormalities and or severe anatomic derangements of the pancreaticoduodenal complex, formal repair should almost always be delayed and a plan for reconstruction and completion set for DC phase III.

**Colon and rectum**

As with the “running” of the small bowel, the colon should be inspected in a systematic and consistent fashion. It is usually undertaken by inspecting the length of the large bowel from the caecum to the extraperitoneal rectum. Obvious defects are controlled with simple suture ties. Areas of concern should be mobilised and inspected fully as half of the colon circumference is retroperitoneal. Formal resection and discontinuity principles are similar to those for the small bowel. While injuries to the left colon should carry a low threshold to divert with an ostomy, those involving the
right colon can be more frequently reconstructed with an anastomosis. In true damage control, however, the colon should be left in discontinuity, with planned re-explorations undertaken in the subsequent 12–48 hours. The rectum needs to be evaluated after any penetrating injury to the pelvis. While a digital rectal examination may identify injury, a negative examination should be followed with a rigid proctosigmoidoscopy. If a rectal injury is identified, a diverting colostomy is the best treatment option.

Diaphragm
Regardless of mechanism, the diaphragm should be inspected visually and manually on both sides to fully exclude this easily missed injury. Diaphragmatic injuries should be closed with a large, absorbable suture, in interrupted fashion. The repair is often expedited and simplified by bringing the corners and centre of the defect together and up into the surgical field by approximating and evertting the edges of the laceration with Allis clamps.

Bladder
Intraperitoneal injuries of the bladder are identified by the presence of a urine leak (even with bladder decompression) or a peritoneal defect involving the pelvis. If in doubt, methylene blue or indigo carmine may be given intravenously and will result in obvious colouring of extravasated urine. Of all the intra-abdominal injuries, repair of the bladder (especially that involving the dome) is easily accomplished and can be performed with a simple one-layer suture repair (although some still advocate a two-layer closure). Catheter decompression of the bladder accompanies repair and usually remains in place for 5–7 days. There is significant variability in the use of a postoperative cystogram to guide or dictate catheter removal.

Abdominal closure
Shortly after initiating the laparotomy, the surgeon should be considering and preparing for the closure of the fascia. In the absence of the lethal triad, major vascular injuries, or questionable bowel viability, primary abdominal fascial closure is advocated (Table 23).

<table>
<thead>
<tr>
<th>PHYSIOLOGIC</th>
<th>INJURY PATTERN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ongoing hypothermia</td>
<td>Combination major vascular and bowel injury</td>
</tr>
<tr>
<td>Ongoing coagulopathy</td>
<td>Combination solid organ and vascular and/or bowel injury</td>
</tr>
<tr>
<td>Ongoing acidosis</td>
<td>Need for re-exploration for evaluation and/or reconstruction of bowel</td>
</tr>
<tr>
<td>Ongoing haemodynamic instability</td>
<td>Multiple cavity injury</td>
</tr>
</tbody>
</table>

Although there are numerous suture options, the fascia is most commonly closed with two running, large, looped sutures (PDS or polypropylene). One is typically begun on each side of the incision and the two are joined together with a knot just above the umbilicus. The subcutaneous tissues should be irrigated and the skin edges re-approximated with staples. If the decision is made to leave the abdomen open, even more options and variability exist for “closure”.

Towel clips, a running suture and a Bogota bag (intravenous fluid bag) have all been described as quick and easy closure methods when employing damage control. However, the most common is the vacuum-pack abdominal closure technique. The initial layer is a bowel bag, ventilated with numerous holes, that is then tucked underneath the edges of the fascia to protect the intra-abdominal contents. A surgical towel is placed on top of the exposed bowel bag and two flat-fluted Jackson–Pratt drains are then laid...
on top of the towel on each side near the fascia interface. An Ioban™ antimicrobial adhesive drape is then used to seal the contents of the vacuum-packed abdomen and the drains are then placed to suction.

### LEARNING POINTS: Trauma laparotomy

- The primary goals of trauma laparotomy are: rapid control of exsanguination, control and minimisation of bowel spillage, and avoidance of the lethal triad (hypothermia, coagulopathy and acidosis).
- Four-quadrant packing should be performed for excessive intra-abdominal haemorrhage.
- A methodical examination of all hollow and solid organs should be performed to avoid missed injury.

### Further reading


**Introduction**

By their very nature, traumatic injuries are unpredictable. Therefore, it is impossible to provide a complete set of instructions for how to deal with all the injuries that may be encountered. However, the principles regarding thorough investigation of the abdomen and its contents remain the same. Ensuring that a methodical approach is taken will significantly reduce the chance of a missed injury.

**Step 1**
- The patient should be positioned supine with both arms out and prepared from neck to knees.
- It is important to ensure that all required equipment is prepared, including cell salvage and suction.

**Step 2**
- If there is extensive haemorrhage then four-quadrant packing should be performed, beginning with the likely source.

**Step 3**
- Once packing is complete and the haemorrhage controlled, the process of pack removal can begin. Start with the least likely quadrant and address each injury as it is encountered.
- All of the small and large bowel must be examined and any defects addressed.

**Technical skills**

**Trauma laparotomy**

- A midline laparotomy incision is performed from the xiphisternum to the pubic symphysis.
• The remaining hollow organs and solid organs must be examined in turn for further injury. Communicate continually with the anaesthetist. If the patient’s physiological status deteriorates be prepared to curtail your operation once life-saving manoeuvres have been completed.
Damage-control resuscitation

Introduction
The lethal triad of hypothermia, acidosis and coagulopathy is often found in the severely injured patient and accounts for the incredibly high mortality seen in this population. The concept of damage-control (DC) surgery evolved from the frustration of surgeons who had to address complex injuries in patients with significant intraoperative coagulopathy. This technique involves the “temporising” of the traditional trauma laparotomy by obtaining control of surgical haemorrhage and gross bowel spillage and contamination, then leaving the operating room to correct the lethal triad (coagulopathy, acidosis and hypothermia).

This approach is now well established in both civilian and military trauma practice and is the standard of care for the severely injured patient with disturbed physiology. The concept has even carried over to the actual resuscitation of these severely injured patients, giving rise to an increasingly popular method of avoiding and/or treating trauma-associated coagulopathy (known as damage-control resuscitation).

Indications
Numerous objective and subjective data have been cited as cues and identifiers of when the surgeon should implement damage-control techniques. However, no author or group has been able to identify strict criteria to guide the clinician. As with many treatments and adjuncts to patient care in the critically injured patient, damage-control measures should be employed early to achieve maximal benefit. In keeping with this concept, it is critical that the surgeon stays out of trouble rather than attempting to get out of trouble at some later point. To this end, it is critical that the surgeon keeps the anaesthesiologist, haematologists, blood bank and nursing staff fully involved with and aware of the management of the patient.

Phases of damage control
The initial operation – Phase I
Control of haemorrhage
As with standard trauma laparotomy, the patient undergoing damage-control laparotomy should undergo packing of all four quadrants. While the initial packing should temporise the majority of bleeding, supracoeliac control may often be required to allow adequate visualisation of the precise location and source of haemorrhage. Control of aortic disruption in this area may be obtained through the gastrohepatic ligament with occlusion devices, fingers, or an aortic clamp. Once identified, a side-biting vascular clamp is useful for obtaining local control and lateral repair is the easiest and typically the quickest method of addressing these injuries. Furthermore, ligation of any truncal vessel can be considered, although some arteries and veins will tolerate this better than others.
Control of contamination
Following rapid control of haemorrhage, isolation and cessation of bowel spillage is the next critical step in DC phase I. To achieve this rapidly, 0-0 silk sutures or umbilical ties can be placed proximally and distally (they are passed along the mesenteric border, across the bowel lumen, then secured). If smaller defects are present, these may be quickly repaired with a running silk suture. For large bladder injuries, a suction device can be placed into the pelvis and a lap-pad placed over this and packed. A simple absorbable suture repair (2-0 or 3-0 chromic or Vicryl™) can be performed at the completion of the case, with revision at a later time.

Temporary abdominal closure
The most common (and probably the most simple) method for rapid abdominal “closure” is the vacuum pack technique. This approach involves the use of a non-adherent bag placed into the abdomen (isolating the bowel from suction), followed by placement of an Ioban™-wrapped towel over the bowel bag. An additional Ioban™ covers the abdominal wall and a closed suction drain is placed into the “gutters” of the subcutaneous tissue. Some trauma surgeons advocate the use of pre-packaged negative-pressure sponge devices because of the simplicity of application and recent data demonstrating superior “time to fascial closure”. Regardless of the method chosen, it is critical to remember that if the patient’s airway pressures increase by more than 10 cmH2O then the closure is too tight and should be revised.

Restoring physiology and reversing the “lethal triad” – Phase II
Hypothermia
Hypothermia affects coagulation by reducing platelet activation and adhesion and by slowing down the metabolic rate of coagulation factor enzymes, specifically the serine proteases. Treatment begins with basic care, including keeping the patient covered from head to toe and using external warming devices and radiant heat blankets. Next, intravenous fluids and blood products can be delivered through effective fluid warming devices and ventilator circuits can be warmed and humidified. Finally, invasive manoeuvres such as active internal and extracorporeal methods can be employed.

Acidosis
Acidosis inhibits key enzymatic activity in the coagulation cascade and this effect is potentiated in the presence of hypothermia. Traditional aggressive crystalloid-based resuscitation and inadequate ventilation may both worsen acidosis and coagulopathy, and care should be taken that these are avoided.

Following base deficit and lactate levels may provide some evidence that the resuscitation is heading in the right direction, but they should not be used as definitive endpoints. In fact, careful catheter-directed resuscitation might provide more gentle volume expansion with superior improvements in end organ perfusion. Sodium bicarbonate or tris-hydroxymethyl-aminomethane (THAM) may be used with caution to improve refractory acidosis in patients with severe metabolic acidosis (pH<7.20).

Coagulopathy
Trauma-associated coagulopathy is present in up to one-third of trauma patients on admission. Upon admission to intensive care, this may be even higher. Damage-control haematology directly attempts to address this process with a proactive rather than a reactive approach to transfusion of blood and blood component therapy. Although the optimum ratio of blood products to be transfused is currently being debated, it is well accepted that the use of fresh frozen plasma and platelets should start earlier and in ratios similar to that of whole blood.
**Fascial closure and definitive surgery – Phase III**

Although phase III was originally described as the take-back or “second-look” laparotomy, the patient’s injuries and physiology may necessitate several returns to the operating theatre. Some patients may be prepared to return to theatre just a few (6–8) hours after the original operation, but most will require 12–24 hours or more. However, this phase should not be delayed for longer than 72 hours.

The key steps of phase III are the removal of packing, the restoration of intestinal continuity, and re-evaluation for potential missed injuries. At this re-exploration, a nasoenteric feeding tube or percutaneous feeding tube should be placed. Unfortunately, primary fascial closure may not be possible at the initial take-back, therefore plan a return to the operating room once significant oedema has resolved. If this happens, consider aggressive diuresis upon return to the intensive care unit and another planned trip back to the operating room for primary closure. In these situations, percutaneous tubes and ostomies should be delayed or brought out lateral to the rectus to avoid complicating future fascial closure.

**Permissive hypotension**

While maintaining arterial blood pressure within “normal” ranges is a classic teaching of ATLS (Advanced Training in Life Support), such arterial pressure gradients haven been shown to impair the formation of a new clot and dislodge an existing clot. Avoiding dislodgement of such a haemostatic thrombus has been shown to reduce bleeding and reduce haemorrhage volume. Therefore, the target systolic blood pressure appears to be at (or slightly below) 90 mmHg.

**Minimising crystalloid-based resuscitation**

Whereas many providers view early administration of fluids to be the standard of care, neither animal nor clinical research literature would support this approach. In fact, recent animal data support “minimising” crystalloid resuscitation until surgical control of haemorrhage has been achieved. Furthermore, clinical trials performed to date support fluid restriction prior to obtaining control of haemorrhage. In fact, investigators who have evaluated withholding fluid until the patient is in the operating theatre advocate careful titration of initial fluid therapy to a lower than normal systolic blood pressure (≥70 mmHg instead of >100 mmHg) during active haemorrhage.

**Damage-control haematology**

Damage-control haematology (DCH) is the process of delivering large amounts of blood products in an efficient manner to patients who have been identified as having life-threatening haemorrhage. Typically, products delivered include packed red blood cells (PRBC), plasma and platelets. Adjuncts include cryoprecipitate, recombinant factor VIIa, prothrombin concentrate, intraoperative cell salvage techniques and fluid warmer/infusion systems. Both military and civilian groups recommend the delivery of PRBC, plasma and platelets in ratios similar to those in whole blood. In addition, over 25% of injured patients arrive at the trauma centre already coagulopathic, and they are...
at a markedly higher risk of mortality. As such, massive transfusion protocols that allow for the immediate and sustained release of the above-described products should be in place.

**LEARNING POINTS: Damage-control resuscitation**

- Damage-control surgery should be considered if the patient exhibits the lethal triad of hypothermia, acidosis and coagulopathy.
- Abbreviated surgical techniques include not forming an anastomosis, not exteriorising bowel, vascular shunting and leaving the abdomen open.
- Strict fluid balance should be maintained with the benefits of permissive hypotension.

**Further reading**


Introduction
Hepatic injury can occur as a result of either blunt or penetrating trauma. Conservative management is attempted in many cases but operative intervention is required if there is uncontrolled haemorrhage.

Step 1
• For a trauma laparotomy the patient should be laid supine, in a “crucifix” position, and prepared from the level of the neck to below the groin.
• Identification of the liver injury is likely to require four-quadrant packing.
• The first principle of hepatic haemorrhage control is to attempt to return the traumatised liver to its original conformation.

Step 2
• Returning the liver to its normal shape can be achieved by physical compression.
• This applies pressure to the traumatised liver surface and may reduce or arrest bleeding.

Step 3
• If physical compression fails, the second approach to haemorrhage control is packing.
• Packing should occur above and below the liver. Packing can be left in place for up to 48 hours if re-look laparotomy is planned.
• Care should be taken because tight packing can lead to hepatic necrosis.
Step 4

- If both pressure and packing fail then a Pringle manoeuvre should be attempted. This involves placing a large haemostat/vascular sling/digital pressure across both the portal vein and the hepatic artery, thus preventing inflow of blood to the liver.
- The haemostat in the Pringle manoeuvre should not be left on for longer than 30 minutes.
Introduction
Owing to the excessive haemorrhage involved with a traumatised spleen, efficient removal is crucial in order to limit blood loss. Haemorrhage in this case may also partially obscure the operating field, therefore it is imperative that the operating surgeon understands the relevant splenic anatomy. The incidence of emergency splenectomy has decreased in recent years as clinicians attempt to preserve splenic function through conservative management.

Step 1
- Midline laparotomy should be performed to allow access to other structures, followed by abdominal packing to control haemorrhage.
- Splenic bleeding can be controlled by compressing the splenic artery and vein at the hilum of the spleen. This can be done using a finger and thumb or a non-crushing intestinal clamp.

Step 2
- The spleen has a number of vascular and non-vascular attachments. The first is the lienorenal ligament.
- Standing on the right of the patient, pass your hand over the spleen and gently retract it; divide the lienorenal ligament on the lateral aspect.
- Use long-handled scissors, moving from the lower to the upper pole.

Step 3
- The spleen can now be delivered into the wound.
- The remaining attachments include the left gastroepiploic vessels at the lower pole, the short
gastric vessels passing from the upper pole and the main splenic vessels.

- In an elective splenectomy each of these vascular pedicles would be divided individually.

Step 4

- During emergency splenectomy time often dictates that all vessels should be clamped and divided quickly. Therefore, place either one or two haemostats across all vessels and divide.
- The spleen can be removed. Significant haemorrhage should have stopped.

Step 5

- Transfix the remaining vessels using a braided suture.
- Ensure that the patient receives post-splenectomy prophylaxis, including a pneumococcal, meningococcal and Haemophilus influenzae vaccine.
- Penicillin-based antibiotic prophylaxis may also be appropriate.
Technical skills

Temporal abdominal closure (vac-pac dressing)

Introduction
It is not always possible to close the abdominal wall following an emergency laparotomy and it may be necessary to return to theatre within 48 hours to inspect the operative site. In these cases a temporary abdominal dressing may be applied. Below we describe such a dressing. It has significant advantages over the classic Bogota bag, but remains relatively inexpensive and easy to construct.

Step 1
- The equipment needed to make this dressing includes a large gauze pack, two large lo-band™ dressings and two Jackson-Pratt suction drains.

Step 2
- Fold the gauze pack to the approximate size of the abdominal defect and place on the sticky side of one of the fully open lo-band™ dressings.

Step 3
- Fold the lo-band™ dressing so that the gauze pack is completely encased.
**Step 4**
- Place the dressing within the wound, ensuring that the edges tuck underneath the wound edges all the way round. This will protect the bowel and other viscera.

**Step 5**
- Lie the Jackson–Pratt drains on either side of the wound and bring out either laterally or towards the groins.
- These drains can later be attached to suction and will remove inflammatory exudates and secretions from the wound.

**Step 6**
- Open the second Ioband™ dressing and place over the entire wound.
- Ensure there is an airtight seal all the way round, otherwise the drains will be ineffective.
- Also ensure that a mesentery is created around the drains.

**Step 7**
- This dressing protects the abdominal contents and allows for easier wound care by the nursing staff.
- Dressings should be changed or removed within 48 hours.
Introduction
Thoracotomy for trauma can be performed for a variety of mechanisms, numerous indications, and in multiple locations throughout the hospital.

Resuscitative (or emergency department) thoracotomy is defined as a left lateral thoracotomy that is emergent in nature and performed in the resuscitation room shortly after arrival.

Emergent and urgent thoracotomy is classically described as thoracotomy performed in an emergent or urgent fashion in the operating theatre. Emergent and urgent thoracotomy can be performed on either side of the chest. Given the urgent nature of its use, trauma thoracotomy has a defined set of principles that are followed whether the patient is in extremis or is haemodynamically stable but warranting immediate exploration.

Emergent/resuscitative thoracotomy

Indications and rationale
The role of emergent thoracotomy is defined on the basis of the need to perform therapeutic manoeuvres and correct surgical lesions, including aortic cross-clamping, suturing cardiac wounds, and evacuation of tamponade. Based on the majority of data available, an emergent thoracotomy is indicated:

- in patients with penetrating thoracic injury and either vital signs or signs of life;
- in patients with penetrating abdominal injuries who arrive with vital signs and then deteriorate;
- in blunt trauma patients with vital signs on arrival who deteriorate en route to the operating theatre.

Technical aspects – entering the chest
In the case of an emergent thoracotomy, a left anterolateral incision is made at the level of the fourth or fifth intercostal space. If the patient is female, the breast should be retracted cephalad to gain access to the interspace. This incision provides rapid access with the simple instruments and is easily extended onto the right chest as needed. First, the skin, subcutaneous fat, and muscles are incised using a scalpel. Next, the intercostals and parietal pleura are divided in one layer using heavy scissors. At this point, a standard rib retractor is inserted with the handle directed towards the floor and axilla. If necessary, rapid extension across the midline (sternum) can be achieved with a Gigli saw or Lebsche knife.

Addressing the heart
A knife or sharp-pointed scissors are then used to initiate the pericardiotomy. The pericardial incision should be oriented parallel with the table to avoid injury to the phrenic nerve. Blood clots should be promptly and completely evacuated. Cardiac bleeding should be controlled.
immediately using digital pressure. Ventricular wounds are best repaired with 3-0 non-absorbable sutures placed in a horizontal mattress fashion (with pledgets). Many venous and atrial wounds, however, can be repaired (with a similar suture) in a simple running fashion. If large defects are present and prove to be difficult to repair in the emergency department, temporary control with balloon occlusion (a bladder catheter placed into the defect, inflated, with the external portion clamped) can be employed. If the patient is found to be in cardiac arrest, the heart may be quickly delivered into the chest for compression or defibrillated with internal paddles.

Assessing the chest cavity
Once the heart has been investigated, attention should be directed towards gross haemorrhage from the chest cavity. Lap-pads can be quickly positioned to provide compression and stop bleeding into the surgical field to allow for appropriate inspection. If the haemorrhage appears to be due to pulmonary vascular or parenchymal injury, the injured lung may be twisted around by 180°, thereby compressing the main pulmonary artery and vein ("hilar twist"). Alternatively, placement of a clamp across the hilum can effectively control bleeding. In cases of cardiac arrest or subdiaphragmatic exsanguination, the thoracic aorta should be cross-clamped inferior to the left pulmonary hilum. This is performed by first elevating the left lung superiorly, then the mediastinal pleura is incised to expose the aorta. Passage of an orogastric or nasogastric tube by the airway team can assist in quickly identifying and distinguishing the empty descending aorta from the oesophagus.

Be prepared – the checklist
Although the performance of an emergent thoracotomy (and the patient’s physiology leading to its performance) can be overwhelming, the trauma team leader should not forget the basic principles (the ABCs) for the initial care of the injured patient. The patient should be intubated immediately (if not already done), with checking of end-tidal carbon dioxide and the subsequent passage of an orogastric tube. Once the decision has been made to open the chest, a team leader should be assigned to “run” the resuscitation if the trauma team leader becomes involved in the performance of the thoracotomy.

A number of key supplies should be available regardless of the nature (resuscitative or urgent) of the thoracotomy. These include (but are not limited to) a scalpel, heavy scissors, a chest retractor, forceps, suture and a needle driver (Table 24).

### Table 24: Instrument check list for emergent thoracotomy

<table>
<thead>
<tr>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>#10 blade scalpel</td>
</tr>
<tr>
<td>2-0 silk (or Prolene™) 3-0 CV Ethibond™ sutures (+/- pledgets)</td>
</tr>
<tr>
<td>Mayo (curved) scissors and Metzenbaum scissors</td>
</tr>
<tr>
<td>Internal defibrillator paddles</td>
</tr>
<tr>
<td>Finochietto retractor</td>
</tr>
<tr>
<td>Toothed and DeBakey forceps</td>
</tr>
<tr>
<td>Long needle holder</td>
</tr>
<tr>
<td>Lebsche knife and mallet</td>
</tr>
<tr>
<td>Satinsky and DeBakey aortic clamps</td>
</tr>
</tbody>
</table>

The environment in which the operation takes place is also critical to both its measured and its unmeasured success. The chaotic environment of the trauma bay or trauma operating theatre, not to mention one in which an emergent thoracotomy is being performed, poses significant risks for exposure to blood-borne pathogens. Bear in mind the potential costs of contracting a blood-borne infection during the procedure, considering with whom and in what setting an emergent thoracotomy is warranted.
Urgent thoracotomy

A quarter of deaths from trauma are the result of severe chest injury. While many people die immediately as a result of great vessel and cardiac injuries, many will survive to arrive to the trauma centre, where they will present with urgent issues that require urgent (if not emergent) intervention. Among these are tension pneumothorax, cardiac tamponade, and large haemothoraces. However, only 15–20% of patients will require operative intervention; most simply require thoracostomy tube placement.

Indications for urgent thoracotomy include:

• 1000–1500 mL immediately returned from thoracostomy tube;
• persistent bleeding from the tube >200 mL per hour;
• identification of cardiac tamponade;
• massive air leak.

The approach, albeit somewhat less expedited, proceeds in a manner similar to that of resuscitative or emergent thoracotomy.

Further reading

Introduction
This chest wall incision can be used to access the thoracic cavity to manage pericardial tamponade, penetrating cardiac injury, severe lung contusion or great vessel injury.

Step 1
- Anterolateral thoracotomy is performed by first positioning the patient supine, ideally with the appropriate arm raised above their head.

Step 2
- An incision is then made from sternal edge to mid-axillary line in the 4th or 5th intercostal space.
- Deepen the incision and enter the intrathoracic cavity.

Step 3
- Attempt to avoid the neurovascular bundle running on the inferior margin of the rib.
- The wound can be held open using a self-retaining retractor.
- On the left this will allow the lung and pericardium to be visualised.
**Step 4**
- In order to mobilise the lung it is necessary to divide the inferior pulmonary ligament.

**Step 5**
- Once fully mobilised, the lung can be delivered into the wound.
- This will allow the identification of specific lung injury.
- Bleeding from these injuries can be difficult to identify and control.

**Step 6**
- To allow better access, a stapled tract can be created within the lung tissue.
- Ideally a linear stapler should be used.

**Step 7**
- This opens up the original injury but at the same time prevents further haemorrhage.

**Step 8**
- Bleeding vessels can now be under-run using a non-absorbable monofilament suture.
• If the injury is peripheral it may be easier to staple off a small segment of lung tissue completely.

• If bleeding continues it may be possible to perform a "lung twist" manoeuvre.

• This involves twisting the lung through 180°.
• This rotates the vascular pedicle at the hilum and stops blood flow.

Step 9
• If bleeding continues it may be possible to perform a "lung twist" manoeuvre.
Technical skills

Clamshell thoracotomy and cardiac injury

Introduction
An anterolateral thoracotomy can be extended to a clamshell incision, providing access to both sides of the thoracic cavity and the mediastinum.

Step 1
- The clamshell thoracotomy is created by performing right and left anterolateral thoracotomies, then dividing the central sternal cartilage.
- Bleeding from the internal mammary artery may occur as a result of this procedure.

Step 2
- Once the incision has been completed the ribs may be spread using two retractors.
- This completely exposes the thoracic cavity.

Step 3
- When addressing cardiac injury, note the phrenic nerve traversing the pericardial sac and the left anterior descending coronary artery running across the face of the left ventricle.
- Every attempt should be made to preserve these structures, therefore to this end a vertical
rather than a horizontal incision in the pericardium is made.

Step 4
- In this case we are able to identify a single penetrating injury to the left ventricle, just inferior to the left anterior descending coronary artery.

Step 5
- Profuse haemorrhage can be stemmed by placing a finger over the defect.

Step 6
- The defect can be closed using a non-absorbable monofilament suture with Teflon pledges attached.
- The pledges prevent the suture from cutting through the cardiac muscle.
Step 7
- Pledgets should be placed on both sides of the wound.
- A mattress suture technique is ideal for closing this type of wound.

Step 8
- Once the defect has been closed the patient will need drains placed in both sides of the thoracic cavity and one in the mediastinum.

Step 9
- There is no benefit to closing the pericardium.
- The patient should be transferred to a critical care setting.

- If sutures are unsuccessful or unavailable, or a faster closure technique is required, then penetrating cardiac wounds can be closed with a staple gun.
Introduction

Professionalism encompasses many aspects of behaviour and practice and its importance is being increasingly emphasised both in the UK and abroad. The British Intercollegiate Surgical Curriculum Project (ISCP) has a separate section on “Professional Skills and Behaviour” and trainees must demonstrate that they possess the relevant skills and attitudes before they can progress in their training. Seven essential characteristics of a surgeon are described that encompass the required attributes:

1) medical expert;
2) communicator;
3) collaborator;
4) manager;
5) health advocate;
6) scholar;
7) professional.

A detailed description of the different domains and aspects of professionalism is beyond the scope of this book. However, emergency and trauma surgery can present unique challenges in terms of professional behaviour. Although often there are no right or wrong answers, there are some common pitfalls. The aim of this chapter is to highlight a few of these and explore some of the issues involved.

Leadership and teamwork

Teamwork is essential in all branches of medicine, but particularly so in emergency surgery. Staying in contact with one’s colleagues is essential in order to maximise the efficiency of the team and ensure that the variety of tasks one is faced with get done in the appropriate order of priority. As a surgical trainee you are often no longer the most junior member of the team, and must take on a leadership role if you are to be effective. Everyone has their own style of leadership. However, certain principles apply:

- **Delegate and share appropriately:** A common problem for trainees concerns a failure to delegate appropriate tasks to others so that important tasks are sometimes missed.
- **Support colleagues:** If they feel out of their depth or afraid to make contact, then situations can arise where others manage patients beyond the limits of their competence and mistakes are inevitable.
- **Ask for help or opinions when necessary:** Colleagues would far prefer to be called upon than have a patient deteriorate.

Return to first principles. When unsure in clinical situations, take the logical approach advocated earlier in this book and on the ATLS® and CCRISP® programmes to reduce the likelihood of making basic errors.

Time management and priority setting

Managing different tasks and different priorities is one of the most difficult aspects of being a surgeon and will increase in complexity with increasing seniority. This is
particularly true in the emergency setting and out of hours when balancing the demands of operating, assessing patients in the emergency department, looking after acutely unwell patients on the wards, and dealing with managerial pressures such as waiting time targets and discharging patients in order to free up beds. Remember that whatever else occurs, patient care comes first. Although sometimes difficult, managerial pressures should not be allowed to interfere with managing a sick patient.

Successfully running an emergency depends on setting priorities and teamwork. Errors commonly occur when trainees fail to balance their workload and set appropriate priorities. Communicating closely with each other means all the members of the team get the information they need to prioritise correctly.

Informed consent

The process of informed consent is intended to give patients the information they need about their treatment, to allay unnecessary fears, to decide together on the best course of management and to obtain permission for any necessary procedures.

Although often viewed as a chore, informed consent is an integral part of patient care. Fear of the unknown is often particularly acute in an emergency situation where a previously well patient suddenly finds him or herself unwell, in an unfamiliar environment, undergoing painful and often undignified procedures. Attempting to relieve fear by giving the patient information they need is a basic act of humanity as well as being a legal requirement. It does not need to take long, and lack of time is not an excuse for doing it badly.

Two issues sometimes arise in the context of emergency surgery:

1. **Capacity**

   A person is considered unable to make a decision if, at the time it needs to be made, he or she fails to:
   - understand information relevant to the decision;
   - retain information relevant to the decision;
   - use or weigh the information; or
   - communicate the decision (by any means).

   For emergency surgery, it is not uncommon for patients to be considered “incompetent” if, for example, they are confused or not fully conscious. In these situations it is the surgeon’s duty to act in the patient’s best interests. Such decisions are best taken following discussion with a number of team members. Although any relatives should be consulted to help determine exactly what the patient’s best interests are (including their premorbid quality of life), there is no legal requirement to do so in the UK. Sometimes a patient may be alert and orientated when they present, but deteriorate in the first few hours in hospital. Documenting a careful social and premorbid history is therefore essential for determining the management plan if the patient deteriorates later. Clearly this is especially important with elderly patients.

2. **Varying information needs**

   Different patients require different levels of information and it can be difficult sometimes to judge exactly what is appropriate. This is particularly true in the emergency setting. The General Medical Council guidance is clear: patients must be told if an investigation or treatment might result in a serious adverse outcome, even if the likelihood is very small. They should also be told about less serious side effects or complications if they occur frequently.

   However, it can be difficult to judge this. Sometimes patients do not want to know (“Just do it please, Doctor”). In these situations you must still provide the information specified above, in order to help the patient make an autonomous decision. Balancing this with your other ethical duty of non-maleficence (do no further harm, including psychological harm) is one of the practical challenges encountered in emergency and trauma surgery. Note that the guidance does permit you to recommend a course of
development, provided you do not put pressure on the patient to accept your advice.

Developing professional skills

Professional skills are a crucial component of surgical practice and will be assessed throughout your training. The key to learning in this domain is through reflection and feedback. By critically analysing clinical experiences involving dimensions concerned with professionalism, one can make changes to future practice — which is, of course, learning.

Critical analysis involves taking a balanced view: what went well and options for dealing with the situation better or differently next time. Discussing it with a trainer or colleague can generate invaluable feedback. Although it is helpful if the trainer or colleague was also involved in the situation, it is not essential.

It is important to emphasise that this does not necessarily need to be a formal process. For example, it can be done in between cases or briefly after work.

Summary

Professional aspects of trauma and emergency surgery are not as clearly defined as some in this clinical field. However, there are some common pitfalls worth considering before you find yourself in a difficult situation, perhaps in the middle of the night with limited access to support and advice, and when time is of the essence.

LEARNING POINTS: Medical professionalism

- Professional behaviour must be learned and is not just instinctive.
- Providing appropriate support for colleagues is essential for patient safety.
- Work closely with the rest of your team, and remember that communication is key.
- Informed consent is an integral part of patient care, not just a legal duty.
- If in doubt, return to basic principles (eg ATLS®).

Further reading


Introduction
The magnitude of traumatic injury as a public health problem is enormous. It is now recognised as one of the most important threats to public health and safety because of the number of productive life years lost and the resultant prolonged or permanent disability and its cost to society. For a trauma system to reduce the risks and the burden of disease to individuals and to society it requires a group of related injury-orientated facilities, personnel and organisational entities operating in a coordinated way within a defined geographical area. Although the definitive treatment of life-threatening or limb-threatening injury falls largely to the surgical specialties, the view that it is a purely surgical problem has deferred to a more system-based approach spanning a continuum from prevention to rehabilitation.

The management of injured patients requires a system approach to ensure optimal care, rather than simply developing trauma centres. No single facility can provide resource-constrained optimal care for an entire community in all situations on its own. An ideal trauma system includes prevention, access, primary care, acute hospital care, rehabilitation and research activities. The term inclusive trauma system is used for this all-encompassing approach, as opposed to the term exclusive trauma system, which focuses only on the major trauma centre.

Cooperation between the various levels of trauma provision is also required in the care of the injured patient to avoid the waste of precious medical resources.

Levels of definitive care
A trauma system is based on a network of designated definitive care facilities that provide a spectrum of care to all injured patients. A centre is designated a level according to the resources it can provide to the community it provides for. A designating authority is responsible for assessing the available resources and determining the optimal number and level of trauma centres in a given area. It is not necessary to have all levels of trauma centre in a single area. For example, in an area with a large Level I centre it may not be necessary to provide Level II centres.

An effective trauma system also requires leadership, in the form of a lead hospital. This should be the highest-level hospital in the trauma system and will usually, but not exclusively, be a Level I centre. In less densely populated areas the highest designated facility may be a Level II trauma centre, and this will take on the responsibilities of leadership. In most trauma systems a combination of levels of designated trauma centre will coexist with other acute care facilities.

Level I trauma facilities
A Level I facility is a regional centre that provides tertiary trauma care resources to the whole trauma system in their region. A Level I centre should be available to all patients that require it and be capable of providing total care for
every aspect of injury, from prevention to rehabilitation. It should also provide leadership, education, training, research and system planning extending to all the hospitals caring for injured patients within their regions. Because of the large personnel and facility resources required for all these activities, they will often be university-based teaching hospitals.

**Level II trauma facilities**
A Level II trauma centre provides initial definitive trauma care regardless of the severity of injury. It may not be resourced to be able to provide the same comprehensive care as a Level I centre, therefore rapid transfer of patients with more complex injuries between centres is a requirement for any such trauma system. The Level II centre is often the most prevalent hospital trauma facility in a community and manages the majority of trauma patients. Where there is no Level I centre in the immediate region, the Level II centre will take on the responsibilities for system leadership and education.

**Level III trauma facilities**
Level III centres provide prompt assessment, resuscitation, emergency operations and stabilisation, provided by general surgeons, followed by transfer of injured patients, when required, to a centre that can provide definitive trauma care. They should have transfer agreements and standardised treatment protocols. They are situated predominantly outside urban or suburban areas in less populated communities that do not have immediate access to Level I or Level II centres.

**Level IV trauma facilities**
Level IV trauma facilities consist of advanced trauma life support in remote areas, before patient transfer to a higher level of care. It may not be a hospital but it should have a physician available. Such a clinic will provide primary care to isolated communities and, as a trauma centre, will provide optimal care given its resources. It should therefore be regarded as integral to the inclusive trauma system and have similar treatment protocols, transfer agreements and data-reporting systems as a Level III centre.

No facility within a trauma system should be without direct linkage to a Level I or Level II centre to ensure prompt transfer of seriously injured patients who are in need of a higher level of care. Similarly, there should be exchange of personnel between Level I/II and Level III/IV centres, as well as outreach to more isolated facilities in the form of education and consultation.

**Integration of definitive care into a trauma system**
Definitive care is just one aspect of the system-based approach to the management of trauma in a geographical area. It must fit into a public health framework that views injury as a disease that can be prevented and managed in a way that reduces severity. To do this a public health trauma-based system must first perform three core functions, namely assessment, policy development and assurance. Specifically, it must assess and analyse the regular and systematic collection of injury-related information to define the nature of the problem; it must then develop comprehensive policies and standards to meet goals and improve outcomes in traumatic injury; finally, it must evaluate and monitor the system components, resources, organisation, and processes and enforce adherence to policies and standards.

Having defined what a trauma system does, it remains to decide what comprises a trauma system. A trauma system consists of a variety of components which, when combined, operate in such a way as to perform the three core functions and achieve defined goals. Table 25 and Table 26 list the basic administrative and clinical components of a trauma system.
### Table 25: Basic administrative components of a trauma service. Reproduced from American College of Surgeons Committee on Trauma: Resources for optimal care of the injured patient 2006, with permission

<table>
<thead>
<tr>
<th>Component</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Leadership</strong></td>
<td>Lead agency&lt;br&gt;Advisory committee or committees&lt;br&gt;Trauma medical leadership</td>
</tr>
<tr>
<td><strong>System development</strong></td>
<td>Gap analysis to establish need&lt;br&gt;Formal trauma plan&lt;br&gt;System standards and guidelines&lt;br&gt;Processes to integrate all the components in trauma care</td>
</tr>
<tr>
<td><strong>Legislation</strong></td>
<td>Lead agency authority over regional trauma plan approval, implementation and development&lt;br&gt;Authority to set standards and adopt guidelines&lt;br&gt;Authority for designation of trauma care facilities</td>
</tr>
<tr>
<td><strong>Finances</strong></td>
<td>Financial planning, analysis and reporting processes</td>
</tr>
</tbody>
</table>

### Table 26: Basic clinical components of a trauma service. Reproduced from American College of Surgeons Committee on Trauma: Resources for optimal care of the injured patient 2006, with permission

<table>
<thead>
<tr>
<th>Component</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td><strong>Injury prevention</strong></td>
<td>Promotion of injury control</td>
</tr>
<tr>
<td><strong>Human resources</strong></td>
<td>Adequate workforce resources to allow coordinated operation of a trauma system&lt;br&gt;Evaluation of workforce adequacy and competences&lt;br&gt;Ongoing trauma-related education of workforce</td>
</tr>
<tr>
<td><strong>Pre-hospital care</strong></td>
<td>Agency responsible for pre-hospital care&lt;br&gt;Standardised certification&lt;br&gt;Ambulance and helicopter emergency service guidelines&lt;br&gt;Communication systems integrated with all emergency medical services and disaster preparedness</td>
</tr>
<tr>
<td><strong>Definitive care facilities</strong></td>
<td>Designated acute-care facilities&lt;br&gt;Designation processes using established standards&lt;br&gt;Transfer agreements to facilitate access to other levels of care&lt;br&gt;Rehabilitation facilities to provide post-acute care</td>
</tr>
<tr>
<td><strong>Information systems</strong></td>
<td>System-wide, timely data collection for a trauma registry to allow analysis of data</td>
</tr>
<tr>
<td><strong>Evaluation</strong></td>
<td>System-wide monitoring of system performance against agreed standards&lt;br&gt;Adoption of opportunities to improve trauma care</td>
</tr>
<tr>
<td><strong>All-hazards preparedness</strong></td>
<td>Integrated disaster preparedness capability involving all the emergency services, the pre-hospital facilities, the acute-care hospitals and non-trauma facilities&lt;br&gt;Performance improvement capability</td>
</tr>
<tr>
<td><strong>Research</strong></td>
<td>Active research programmes linked to specific problems identified, quality improvement or ongoing clinical efforts</td>
</tr>
</tbody>
</table>
Developing a regional trauma system

Once it is recognised that injury is a public health problem and is subject to primary (stopping the event), secondary (reduction in the severity of injury from an event) and tertiary (optimising outcomes after an event) prevention strategies, then steps can be made to develop a trauma system. Public education will help to dispel the popular misconception that major traumatic injury “will not happen to me” and that “I will be well taken care of at my local hospital” and it will engender support. This is vital to catalyse this process and the first step – legislation to establish the structure of the system – its lead authority and funding – will surely follow.

An analysis of the gap between the available resources to deal with injury and the assessed burden of injury in a defined geographical area will enable planners to identify needs and to propose solutions for a trauma system to bridge the gap. Enabling legislation for system development will then be necessary, and the establishment of a lead agency to operate and develop the system and to police standards. Public and professional collaboration will follow with the formation of an advisory body.

The lead agency will then design, implement and continue future development of the regional trauma system and define which organisational bodies and components are required, as well as setting the objectives and standards of the system and establishing a means of evaluation and enforcement of performance. This will usually be done in conjunction with the regional or supraregional advisory body, and standards will be developed with national organisations, such as the professional representative colleges.

Trauma centres will then have to be evaluated to verify that they meet standards set by the lead agency and appropriately designated as acute-care facilities or other components of the trauma system. An ongoing process of evaluation, verification and designation will help regulate the quality and consistency of the various components of the system. Data should be entered into a regional or supraregional trauma registry, and a system-wide audit process embarked upon to ensure the various elements of the system are operating in an organised and coordinated manner, and that outcomes from traumatic injury are, indeed, improving.

A process of peer review should address identified areas of improvement in the system and its personnel. External review is often also necessary in both young and more mature trauma systems. This can be accomplished by the use of a small number of key performance indicators to gauge the level of development and completeness of a trauma system.

Summary

In developing a trauma system it is clear that a multifaceted organisation is required to deliver improved injury care. The trauma centre stands at the heart of the system as the provider of definitive surgical treatment, but it must provide much more support to the system in the form of administrative leadership, medical leadership, academic expertise, research, education and outreach consultation. Furthermore, in collaboration with the lead agency, the trauma centre has the challenge of engaging all the other designated centres, acute-care facilities and non-trauma hospitals in performance improvement processes for an inclusive trauma system.

For its long-term survival an expensive, resource-intensive trauma system must be perceived by the public as another emergency service, akin to the police or fire services. A lack of public support (or knowledge) will threaten its legislative-based financing and its ability to recruit and retain a broad spectrum of highly skilled and motivated medical and administrative staff from whom a substantial degree of commitment is required.
Further reading
American College of Surgeons Committee on Trauma. *Resources for Optimal Care of the Injured Patient*. Chicago, IL: American College of Surgeons Committee on Trauma, 2006.
