Clinical Pathways in Emergency Medicine

Volume II

Suresh S. David
Editor

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Preface

In the history of humankind, Medicine has never been more exciting and challenging than in the twenty-first century. One of the great challenges of being a contemporary academic clinician is to find ways to correlate pertinent Basic Sciences to clinical application, at the bedside. When I set out to prepare *Clinical Pathways in Emergency Medicine*, it evoked a thought for contemplation. 'Do we need one more book in the specialty of Emergency Medicine?' That helped to harness an unprecedented approach: from the perspective of a nascent, yet inquisitive emergency physician who is keen to understand the rationale of occurrence, manifestation, and management of acute clinical conditions. And this book differs significantly by providing an algorithm at the end of each chapter, which, at a glance, provides a roadmap for the journey ahead.

Clinical Pathways in Emergency Medicine is an international congregation of contributors, who have offered their expertise which has immensely flavored the global approach to Emergency Medicine. The authors include a remarkable blend of colleagues, friends, former students, and new stars on the horizon of Emergency Medicine. A multi-author manuscript of this nature cannot be delivered without the dedication exhibited by them. In addition to being luminaries from around the globe, they are among the most progressive clinicians in various sub-specialties of Emergency Medicine. And I could not have wished for a better bunch of Section Editors, who superbly orchestrated the creation and revision of manuscripts. Each one of them is an enviable embodiment of clinical excellence.

Sound clinical experience, coupled with knowledge, based on authoritative books and peer-reviewed publications, remains the foundation, on which clinical management needs to be built. In my three decades of clinical practice, I have been humbled multiple times, by the way in which anecdotal experience and written literature is flouted by the human body.

Today's dogma becomes tomorrow's heresy. *Clinical Pathways in Emergency Medicine* is a compendium of contemporary evidence-based knowledge. However, no book remains perfect and a shrewd clinician knows very well that the practice of medicine, based out of a book, has its own limitations. Nevertheless, I am optimistic that this edition of the book would facilitate satiation to the hunger for knowledge among increasing numbers of aspirants in the field of Emergency Medicine.

Kerala, India — Prof. Suresh S. David

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Orthopaedics

Chapter 1
Back Pain

Ruth-Mary deSouza and Shumontha Dev

Key Points
- Red flags indicate potentially serious causes of back pain.
- Serious causes of back pain may be neurological or non-neurological.
- The time course of neurological symptoms is an important factor in clinical decision making.
- Suspected cord compression or cauda equina compression should be investigated urgently.

Introduction

Back pain is one of the most common presentations to the emergency department (ED). A small proportion of these will be due to spinal emergencies, which need to be rapidly identified, investigated and referred for a definitive management plan. This chapter gives an overview of neurological and non-neurological causes of back pain that may present to an emergency medicine physician. This chapter does not cover management of patients with chronic mechanical back pain who have had serious pathology ruled out.

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Table 1.1 Red flags for acute back pain, indicating potentially serious causes of back pain [1]

Neurological:
Sphincter dysfunction
Gait dysfunction
Saddle anaesthesia
Severe or progressive motor weakness
Significant neurological deficit on examination
Systemic:
Age <20 or >55 years
History of malignancy
Systemic illness
Immunocompromised status
Weight loss
Intravenous drug abuse
Steroid use
Structural deformity
Non-mechanical pain (no relief with bed rest, analgesia and nocturnal pain)
Fever
Thoracic pain

History

In the ED, the focus is to rule out dangerous causes of back pain, neurological or non-neurological. The 'red flag' system was initially developed to exclude serious pathology in lower back pain of less than 3 months duration. It has been modified to include pain outside the lower back [1] (Table 1.1) but does not provide a comprehensive assessment of back pain. Yellow flag symptoms are psychosocial factors suggesting chronicity and disability [2]. Ruling out serious pathology in the thoracic or cervical spine or non-neurological pathology involves targeted additional questions. From the neurological aspect, these can be simplified into:

- Pain – localised/diffuse, nocturnal, relation to activity, effect of analgesia, presence of radicular pain
- Weakness – upper/lower limb, proximal/distal, time course
- Sensory disturbance – dermatomal/non-dermatomal, 'heavy/dead' limb, inadvertent injuries, time course
- Sphincter disturbance – urinary retention (painful or painless), urinary incontinence/altered stream sensation, faecal incontinence, sexual dysfunction, saddle anaesthesia, time course
- Function – fine motor tasks, mobility, time course of decline

From the general history:

- Previous spinal pathology and details of any treatment
- Features of infection

- Features of malignancy or history of past malignancy
- Systemic enquiry, medical history, drug history
- Travel, drug use, immune status, occupation

The time course of symptoms is of importance in the history and is a key factor for tertiary specialties when making decisions regarding investigation, management and admission.

Examination

- Inspect the back for any deformities, surgical scars, skin breaches, signs associated with occult spina bifida and other abnormalities.
- Palpate the midline to elicit bony tenderness. Palpate the paraspinal muscles.
- Test movements of the back (flexion, extension, lateral flexion and rotation).
- A neurological examination of the upper limbs, lower limbs and sphincters.
- Distinguish whether the patient is myelopathic, has features of cauda equina compression, acute spinal cord injury, radicular features or has mixed findings.
- Table 1.2 compares myelopathy and cauda equina.

Cauda Equina Syndrome

- Cauda equina syndrome (CES) is a neurosurgical emergency that requires prompt surgery to prevent irreversible damage to the lower limbs and sphincters.
- It is a clinical diagnosis with supporting radiological findings.
- CES is associated with a high proportion of medico-legal claims [3]. Clear documentation should be undertaken including timings of events.
- CES most commonly results from a prolapsed lumbar intervertebral disc, although may be caused by any pathological compression on the cauda equina.
- CES can be defined as complete or impending. In complete CES, painless urinary retention with overflow incontinence and saddle anaesthesia are well established and usually irreversible.
- In impending CES, urinary disturbance with or without saddle anaesthesia are present. Urinary disturbance is a subjective feature and may include difficulty initiating passing urine, altered stream sensation, dribbling, incontinence and sensation of incomplete bladder emptying. In real life, this is often indistinct and confounded by other factors such as pain. It can often be difficult to distinguish new sphincter symptoms in patients who already have sphincter disturbance due to other medical conditions such as urological disorders [4].
- CES, including impending CES, should be approached in a time critical manner to prevent further neurological deterioration.
- Urgent magnetic resonance imaging (MRI) scans of the lumbo-sacral spine must be obtained for suspected cauda equina (Figs. 1.1 and 1.2).

Table 1.2 Comparison of symptoms and signs in myelopathy and cauda equina compression

	Myelopathy	Cauda equina compression
Explanation	Refers to compression of the spinal cord (i.e. from C1 to L1, where the cord terminates and the conus begins) resulting in characteristic symptoms and upper motor neuron signs in the upper and/or lower limbs and sphincters depending on the level affected. Axial pain is not always present and the absence of severe back pain should not result in this diagnosis not being explored	Refers to compression of the peripheral spinal nerve roots from L1 and below – i.e., after the termination of the spinal cord at the conus medullaris. This leads to lower motor neuron signs in the lower limbs and sphincter dysfunction
Symptoms	Neck/back pain (variably present and often not the dominant feature of the patient's complaint)	Low back pain (variably present)
	Clumsiness of the hands/loss of dexterity	Bilateral leg pain
	Brachialgia	Lower limb motor deficit
	Numbness of the hands and feet	Perineal sensory disturbance
	Difficulty passing urine/incontinence	Urinary retention/hesitance/ incontinence
	Gait and balance difficulties – important feature	Sexual dysfunction
Physical examination findings	Increased tone	Leg, ankle or foot weakness depending on the affected level
	Arm/leg weakness	Reduced straight leg raise and nerve root stretch causing pain
	Reduced grip strength	Painless urinary retention
	Decreased pain, temperature and vibration sense, especially at the extremities	Reduced perineal sensation on pinprick and light touch testing in sacral dermatomes
	Hyperreflexia	Impaired sensation of catheter tug and catheter passage
	Hoffman's sign positive – flicking the distal phalanx of the middle finger causes flexion of other fingers	Faecal incontinence
	Babinski sign positive	
	Lhermitte sign positive – neck flexion leads to electric shock like pain in the arms	
	More than three beats of clonus	
	Gait instability	

Note that the features of sphincter dysfunction differ between an upper and lower motor neuron lesion. In myelopathy, a neurogenic bladder from uncoordinated detrusor and sphincter activity occurs and in cauda equina, an insensate bladder occurs. A detailed description of this falls outside the scope of this chapter, but it is important to remember that sphincter and sexual function are important features of the history and the presence and duration of these contributes towards triaging urgency of imaging and treatment

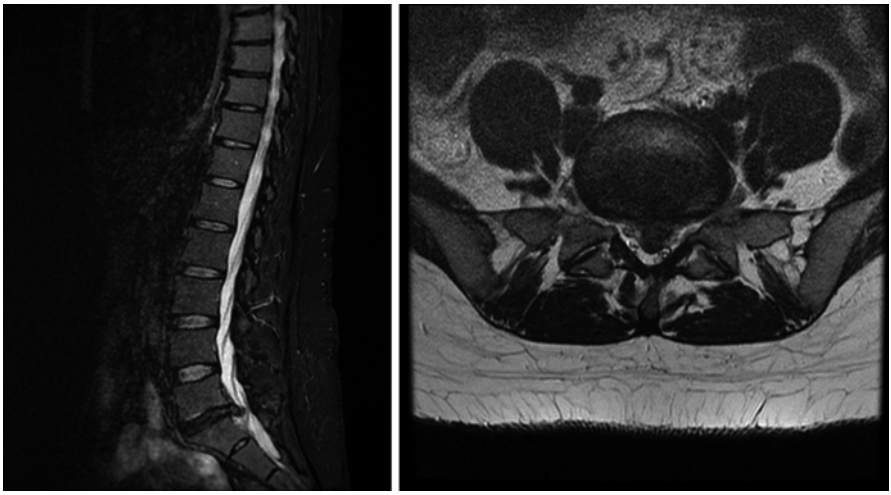

Fig. 1.1 T2 sagittal and axial MRI scan of the lumbar spine demonstrating a prolapsed L5/S1 intervertebral disc with significant cauda equina compression. Clinically this patient had cauda equina syndrome

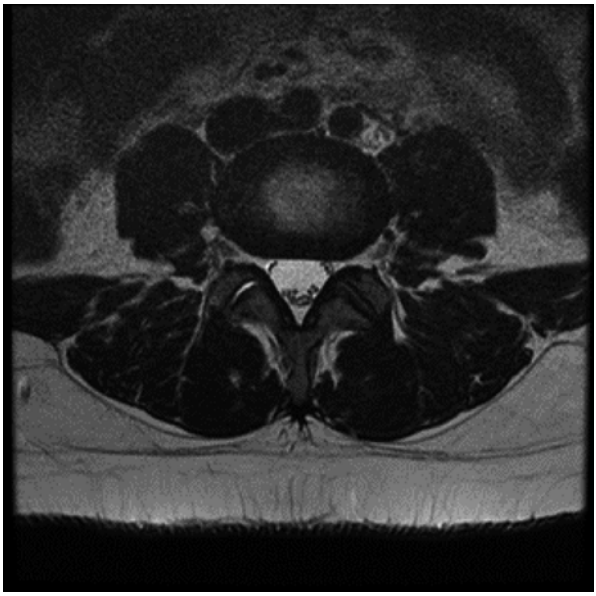

Fig. 1.2 T2-weighted MRI of the lumbosacral spine demonstrating an axial slice through a healthy level for comparison with the axial image of a prolapsed disc in Fig. 1.1

Key Points
Documentation for suspected CES

- Timings of any limb weakness, bilateral leg pain and sphincter disturbance
- Neurological examination of the lower limbs
- Sharp and light touch sensation of the saddle region
- Results of post void bladder scan for residual volume
- If catheterised, sensation of catheter being placed, sensation of catheter tug and residual volume

Traumatic Thoracolumbar Fractures

- Acute management of suspected thoracolumbar fractures includes a primary and secondary survey according to trauma protocols.
- Examination points to focus on are the presence of haemodynamic changes associated with spinal cord injury (only relevant to fractures above T6), midline bony tenderness, limb neurology, priapism and sensorimotor or reflex function of the sphincters.
- Investigations for suspected thoracolumbar fractures can initially be via plain radiographs or CT (Fig. 1.3) [5]. In 'trauma call' settings, the patient is likely to get a body CT that includes reconstructions of the thoracolumbar spine.
- An MRI is required in the presence of neurological deficit.
- If an unstable injury is suspected from a high impact trauma mechanism or clinical examination, log roll until stability is established. In patients with ankylosing spondylitis and a suspected spinal fracture, do not force them into positions outside what is neutral for them as they often have highly unstable injuries and fixed abnormal postures.
- Thoracolumbar fractures can be managed conservatively or operatively.
- In the setting of trauma, where a spinal fracture is excluded on CT scans, but the patient still has unexplained sensory or motor neurology that is not improving, or a fracture is present but does not explain the neurology, it is important to investigate this further with an MRI scan to rule out spinal epidural haematoma (Fig. 1.4). Emergency neurosurgical management for certain cases of haematoma is possible.

Key Points
Imaging thoracolumbar fractures

- A normal radiograph in the presence of bony midline tenderness and trauma does not exclude a fracture – progress to CT
- A CT with or without a fracture that does not explain neurological deficit should be followed with an MRI to exclude haematoma

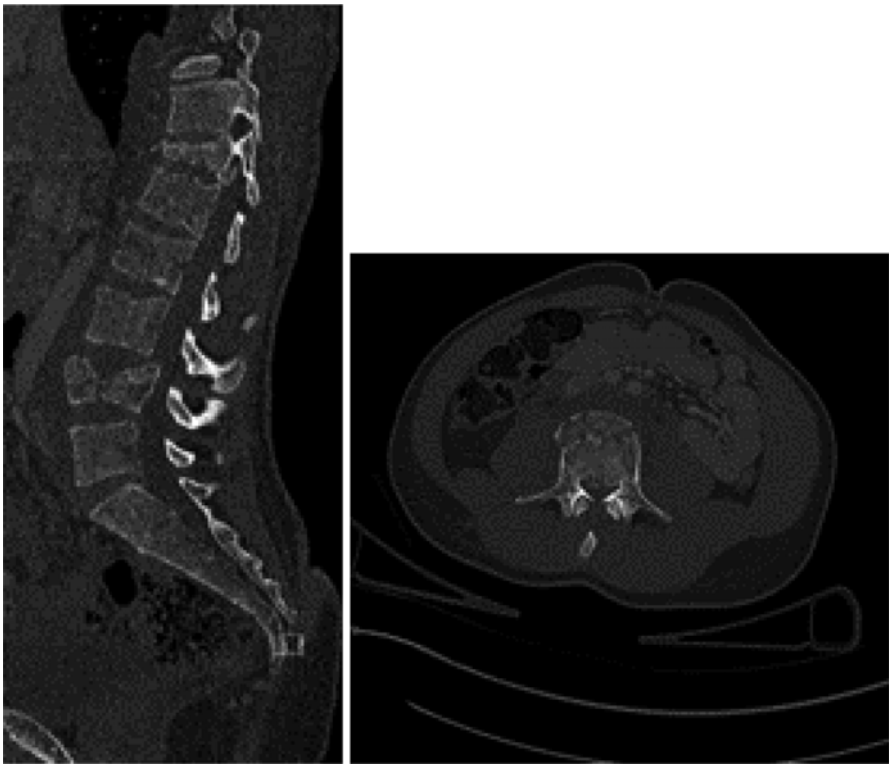

Fig. 1.3 CT spine, sagittal and axial view, showing unstable burst fractures of T12 and L4. They are unstable as they involve two columns

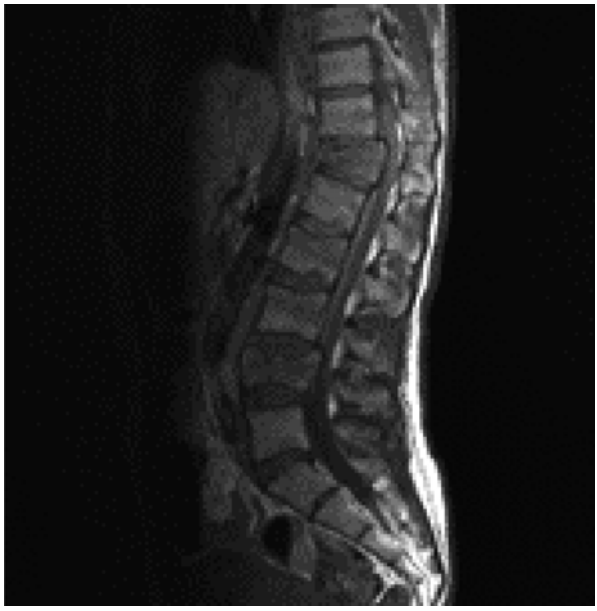

Fig. 1.4 T1 sagittal MRI of the same patient as Fig. 1.3 showing a fracture-related haematoma anterior to the cauda equina at L1–3. CT is good for showing bony anatomy. MRI is required for assessment of soft tissues, haematoma and discoligamentous integrity

Osteoporotic Wedge Fractures

- The most common location for osteoporotic fractures is the spine.
- Osteopenia is a radiological term meaning "reduced radio density' and implies 30 % or greater reduction in total bone mass.
- Osteoporosis (OP) means decreased amounts of normal bone leading to increased susceptibility to fracture.
- OP-related spinal fractures are usually in the upper to mid thoracic spine, can be multiple and are usually axial loading fractures with a stable wedge morphology.
- The history for OP wedge fractures is usually acute back pain without neurological compromise after a minor trauma. Multiple OP fractures over time may lead to kyphosis and loss of height.
- The clinical features of OP wedge fractures are localised back pain, tenderness on palpation and progressive kyphotic deformity. Neurological compromise is not typical of OP wedge fractures.
- Initial investigations involve radiographs or CT of the thoracolumbar spine and a bone profile. Another imaging which may be useful is a bone mineral density ('DEXA') scan.
- Keep an open mind regarding the differential diagnosis for an OP fracture – it may be a pathological fracture secondary to another underlying disease.
- Management of OP wedge fractures is centred around controlling pain and preventing further OP bone loss [6]. Medical therapies are analgesics, nutritional supplements, physical therapy and bone protection. Bracing whilst mobilising is an option and is solely directed at pain relief and comfort.
- Elective vertebral body augmentation (vertebroplasty or kyphoplasty) is only helpful for management of pain from a wedge fracture in selected cases [7].

Spondylodiscitis and Epidural Abscess

- Pyogenic discitis in adults is most commonly from *Staphylococcus aureus* and streptococcal species.
- It typically presents with localised back pain and fever. Neurological deficit, if present, can be radicular or from direct cord/cauda equina compression due to an epidural abscess.
- Infection can arise in the vertebral column by haematogenous spread, direct inoculation or spread from neighbouring tissues.
- Patient factors increasing susceptibility to infection are diabetes, steroids, systemic infection, immunocompromise, renal failure and dialysis and recent spinal surgery.
- Initial investigations of note are peripheral blood counts, inflammatory markers, blood cultures and systemic examination with targeted imaging. In suspected spinal infection, the imaging of choice is an MRI scan with and without contrast.

- Treatment of discitis is usually medical, with at least 6 weeks of targeted intravenous antimicrobial therapy with biochemical, radiological and clinical monitoring of response. Source identification is important in preventing recurrence. Bed rest is required until the patient is pain free [8].
- Indications for a neurosurgical referral are epidural abscess and neurological deficit from any source of neural compression. Epidural abscess is usually evacuated emergently (Fig. 1.5) [9].

Key Points
- Spondylodiscitis with no neural compression or associated abscess can be managed medically.
- Surgical referral for epidural abscess, neural compression or significant vertebral erosion.
- Identification of an organism allows targeted treatment.

Tuberculosis of the Spine

- Spinal tuberculosis is the most common osseous involvement in TB. It may be the sole/first TB presentation. Spinal TB usually has an insidious course.
- TB initially affects the anterior aspect of the vertebrae and spreads back to involve the posterior vertebral body, ligaments and posterior elements of the vertebral canal. Paraspinal and psoas involvement is common and abscesses within the latter may need drainage.

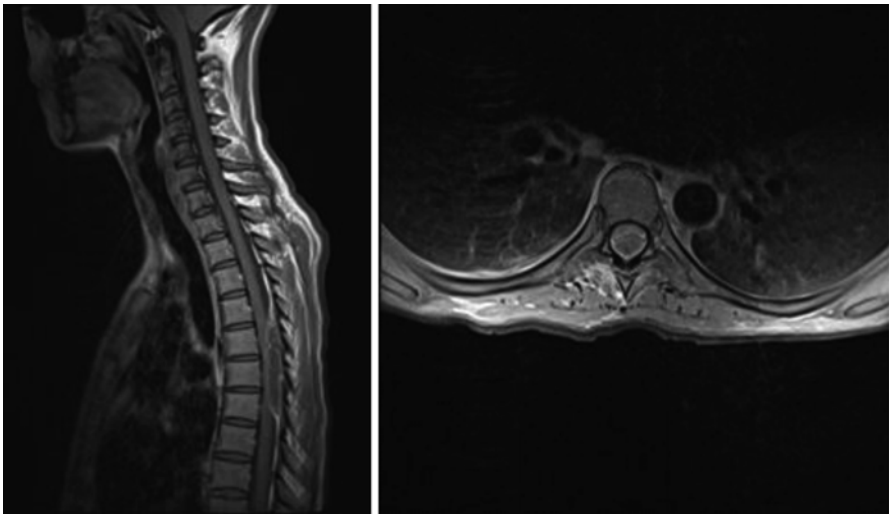

Fig. 1.5 MRI (T1 with contrast, sagittal and axial) demonstrating an enhancing epidural mass from T4–T8 causing spinal cord compression. This was an abscess

- The initial step is to confirm TB. If this cannot be achieved peripherally, vertebral biopsy may be required.
- The patient should be commenced on TB treatment and dexamethasone.
- Typically spinal TB is managed under the medical team. Surgical involvement in spinal TB is rare and only applicable in specific cases with neurological compression, severe pain and deformity [10].

Cervical Myelopathy

- Most commonly cervical myelopathy is of degenerative aetiology and affects middle-aged and elderly patients.
- Cervical spine involvement is seen in over 80 % of patients with rheumatoid arthritis and these patients may develop cervical myelopathy.
- It presents with progressive weakness, numbness and clumsiness in the hands and feet. It can also affect the thoracic spine although less frequently than the cervical spine. Patients with cervical myelopathy will report they are dropping objects and unable to perform fine motor tasks, are falling over and have gait disturbance. Neck and arm pain are also common but not necessary for the diagnosis of myelopathy [11].
- On examination, the picture will be of upper motor neuron signs and radicular features depending on the nerve root affected (Table 1.2).
- Cervical myelopathy in the context of degenerative disease is likely to come to the emergency department when an acute-on-chronic deterioration occurs. Typically, this is precipitated by a minor trauma or physical activity.
- Obtain an urgent MRI for patients with acute neurological deterioration (Fig. 1.6).
- Whilst most cases of cervical myelopathy will be degenerative in origin, it is important to consider alternative causes depending on the history and examination [12]. Non-degenerative causes include trauma, inflammatory disease, abscess, primary malignancy and metastatic spinal cord compression.
- In patients with compressive myelopathy and recent change in their condition, discuss with neurosurgery regarding surgical suitability.

Key Points

- The time course of symptoms and any acute deterioration is important in deciding a management plan.
- There is a wide differential for cervical myelopathy.

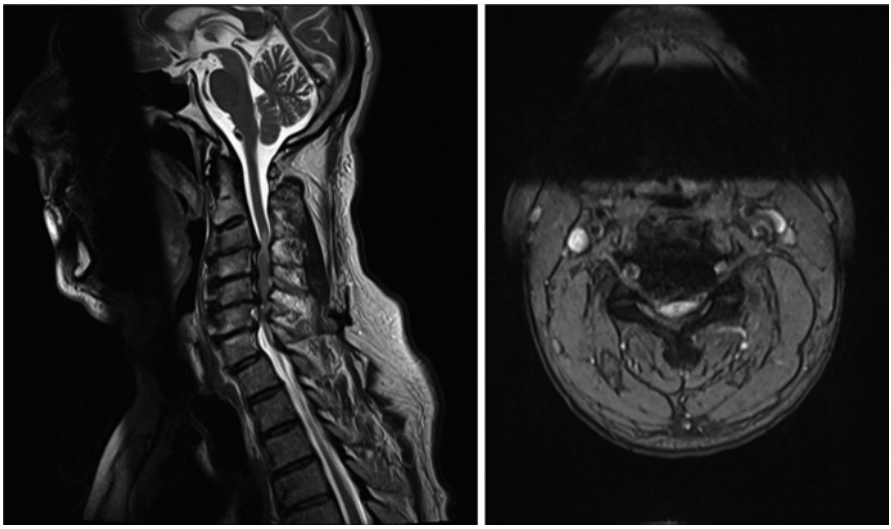

Fig. 1.6 T2 sagittal and axial MRI scan of the cervical spine demonstrating multilevel degenerative osteodiscal disease at C3/4, C4/5, C5/6 levels causing marked canal stenosis and thecal impingement. There is evidence of signal change within the spinal cord at the level of C3/4 and also at C5/6. This patient presented with cervical myelopathy

Metastatic Spinal Cord Compression

- The most common cancers that metastasise to the spine are breast, lung and prostate. Myeloma is also a cause of malignant cord compression. Metastatic spinal cord compression (MSCC) can be a first presentation of cancer.
- Symptoms suggestive of MSCC include cervicothoracic pain, progressive lumbar pain, localised, unremitting and nocturnal pain [13]
- Signs include radicular pain, limb weakness, sensory disturbance, gait and sphincter disturbance [13].
- Current guidelines state that patients with suspected MSCC and no neurological deficit require a whole spine MRI to be performed in adequate time to allow definitive treatment to take place within 1 week – this would mean obtaining the MRI in time to allow for multidisciplinary input prior to any intervention [13].
- Patients with suspected MSCC and neurological deficit require an MRI whole spine urgently so that definitive treatment can be initiated within 24 h [13].
- Definitive treatment may be oncological or surgical.

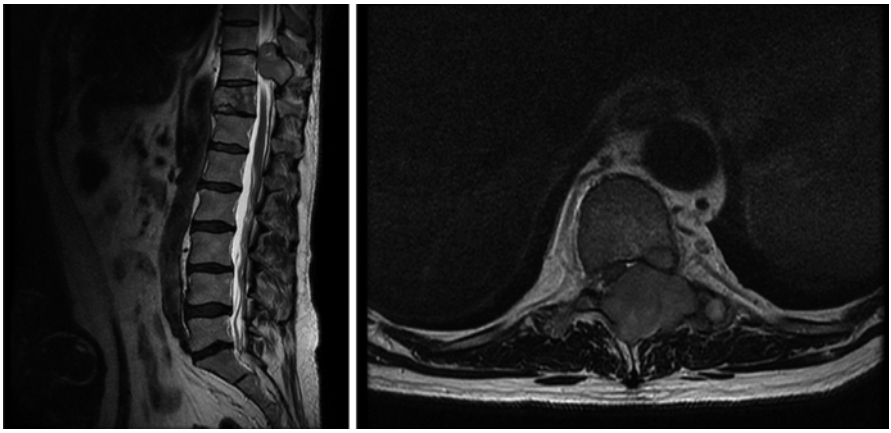

Fig. 1.7 T2 sagittal and axial MRI scan demonstrating an extradural mass at 10 causing cord compression. There is associated destruction of the pedicles, vertebrae and paraspinal tissue at this level. There is cord compression

- Patients with neurological deficit should be on bed rest if there is concern regarding spinal stability until discussed with a neurosurgeon.
- Patients with MSCC should be commenced on dexamethasone, with blood sugar monitoring and gastric protection.
- There is level 1 evidence that surgical management for specific and selected cases of MSCC (Fig. 1.7) can result in significantly improved neurological outcome versus patients treated with radiotherapy [14].

Key Points
Information that is useful to have at the time of MSCC referral:

- Primary cancer (if known), the grade and stage and prognosis of the primary.
- If the primary is unknown, further investigations include CT body, myeloma screen, prostate specific antigen, breast and skin examination.
- Performance status.
- Comorbidities.

Management of the Post Spinal Surgery Patient

- Patients may present to the ED after spinal surgery.
- The main problems to be aware of are wound breakdown, wound infection, CSF leak, recurrence of the original pathology and instrumentation-related problems.
- In the history it is important to obtain details of the surgery performed, the original indication for this and whether any spinal instrumentation was used.
- The wound should be examined and swabbed.
- There is no indication for starting prophylactic antibiotics in the case of CSF leak without signs of infection.

Non-spinal Causes of Back Pain

- Non-spinal causes of back pain can essentially be considered as those emanating from cardiovascular disease and those from abdominal disease.
- Aortic dissection and abdominal aortic aneurysms have been reported to present with sharp interscapular pain and lower back pain. Dissection is best investigated with a contrasted CT of the aorta. Abdominal aortic aneurysms are initially investigated with ultrasound and if this is not available, CT. Aortic pathology can affect downstream arteries to the lower limbs resulting in ischaemic legs, which can sometimes be mistaken for neurological deficit in a patient presenting with back and leg pain.
- Pancreatitis and pancreatic cancer can present with epigastric pain radiating to the back. Acute pancreatitis is associated with a number of risk factors, is diagnosed with raised amylase and not typically associated with any neurological dysfunction. Pancreatic malignancy is associated with weight loss, vomiting and obstructive jaundice.
- Renal colic can be associated with strong back pain and is diagnosed on urinary findings and CT imaging. Renal causes of back pain should not be associated with any neurological findings.
- Psoas abscesses, being retroperitoneal, can cause back and flank pain. Psoas abscesses are often associated with TB although they can exist in the context of any intra-abdominal infection.
- Figure 1.8 provides a flow chart to approach back pain in the ED.

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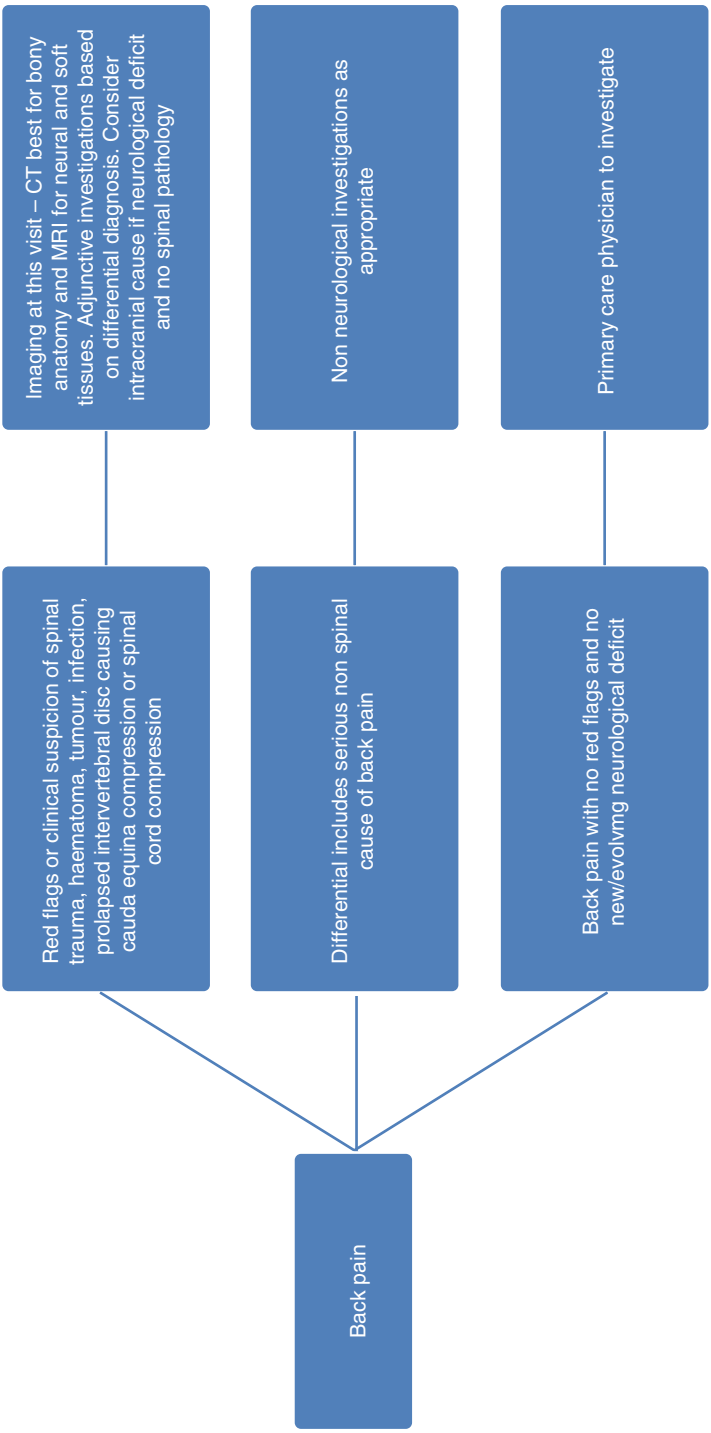

Fig. 1.8 Flow chart outlining management pathway for back pain presenting to the ED

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Chapter 2
Hand

Carlos Lojo Rial and Shumontha Dev

Introduction

Hand injuries are a frequent reason for attendance to the emergency department (ED) worldwide. Common literature estimates 5–10 % of attendances in the USA and an estimated 20 % in Europe [1]. Moreover, they appear to be the leading cause of occupational injuries in the USA [2] and in other countries like India with incidence reported of 361/10,000 industrial workers per year with 50 % of those secondary to hand entrapment [3]. Lacerations are the most frequent, followed by fractures, sprains and contusions. These injuries and associated disabilities have devastating consequences, if treated inappropriately in the ED.

History and examination are paramount in hand injuries [13]:

- A good history will help differentiate simple and complicated injuries.
- The mechanism of injury will allow adequate investigations to be performed.
- Essential to ascertain hand dominance of the patient as well as occupation in view of the potential disability resulting from the injury.
- Time of onset as well as the location of the injury is also critical to elaborate a safe management plan.
- Thorough examination, once appropriate analgesia has been administered, allows review of the range of motion and function.

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Table 2.1 Early management of penetrating injuries

Irrigation under pressure
Debride de-vitalised tissue
Consider risk factors affecting wound healing or infection risks
Co-morbidities
Tetanus vaccination status
Possibility of foreign bodies
Dirty wound
Consider time of the injury

- Neurovascular examination of the hand is essential, before analgesia and postreduction of any fractures.
- Postreduction x-ray is vital to ascertain the results.

All open injuries must be examined thoroughly. Guidelines on early management of penetrating injuries are widely agreed (Table 2.1) [2].

Hand Fractures

Thumb Fractures

Pathophysiology

Falls are the most common cause of thumb injuries. Most fractures result from an axial loading onto a partially flexed thumb. Approximately 80 % of the thumb fractures involve the base of the metacarpal bone. The Bennett's fracture is a two-part intra-articular fracture with a dislocation or subluxation secondary to the traction caused by the abductor pollicis longus tendon [2]. The Rolando fracture, on the other hand, is a Y or a T shaped comminuted intra-articular fracture at the base of the first metacarpal bone [3].

Clinical Features

Patients present with swelling, and with point tenderness, difficulty in performing normal range of motion and rotational deformity. Joint laxity should be assessed but swelling may prevent appropriate examination. A second examination may be performed after nerve block to ascertain degree of laxity.

Investigations

It is advisable to obtain dedicated thumb views and scaphoid views if the tenderness or the mechanism is not clear.

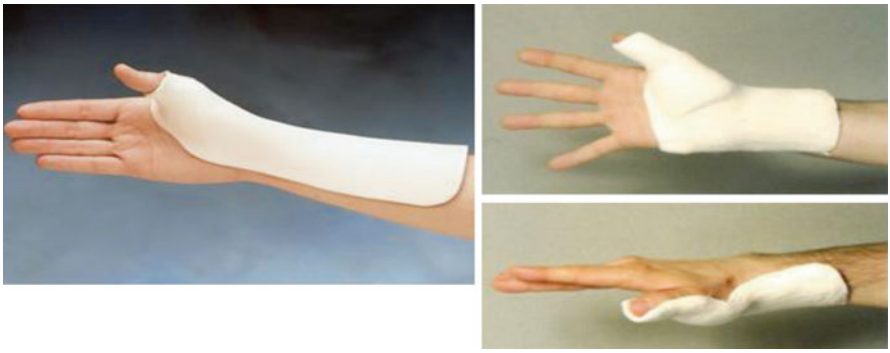


Fig. 2.1 Thumb immobilisation splints for interphalangeal, meta-carpophalyngeal and wrist joint

Table 2.2 Thumb injuries requiring surgical consultation in the ED

Key points
Emergency hand consultation in thumb injuries
Open fractures
Rotational deformity
Angulation >30°
Difficult reductions
Unstable fractures (Bennett and Rolando): step off >2 mm may require surgery

Management

Closed extra-articular shaft fractures can safely be treated by closed reduction depending on the degree of angulation and immobilised using a spica splint (Fig. 2.1). Indications for consultations with the hand surgeons include complex fractures or injuries with affected functions (Table 2.2) [3]. Suspicious injuries with negative x-rays should be immobilised and referred to the outpatient's hand clinic.

Prognosis

Unstable fractures are high risk for osteo-degenerative changes and are associated with significant disability.

Metacarpal Fractures

Pathophysiology

Metacarpal injuries are common in the ED and represent 30 % of all hand fractures in the US. They result from axial loading to the head, direct trauma and crush injuries.

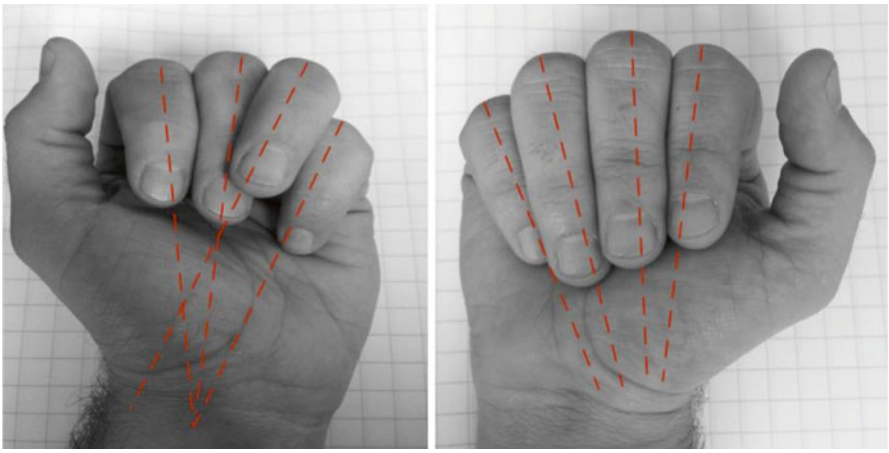


Fig. 2.2 Cascade of fingers visible when making a fist, associated with MC and phalangeal fractures

Table 2.3 Considered acceptable angulation in metacarpal fractures

Digits	2	3	4	5
MC shaft fracture angulation	0°	0°	20°	30°
MC neck fracture angulation	10°	15°	30°	40°

Clinical Features

Patients present with tenderness, swelling and loss of normal movement. There can be shortening and a cascade of fingers can be visible in attempting to make a fist (Fig. 2.2). Examination should identify complication caused by the injury such as rotational deformities, neurovascular injury, compartment syndrome and open components that would all require a hand surgeon consultation.

Specific Fractures

Boxer's fractures (fifth MC fractures) are common metacarpal neck fractures and represent about 20 % of hand injuries. Examination reveals dorsal angulation, with palpable metacarpal head over the volar aspect and shortening of the metacarpal length. The degree of angulation acceptable for MC fractures is controversial (Table 2.3). Conservative management would allow a range of 40–50° but some suggested that up to 70° is acceptable without affecting the outcome (Fig. 2.3).

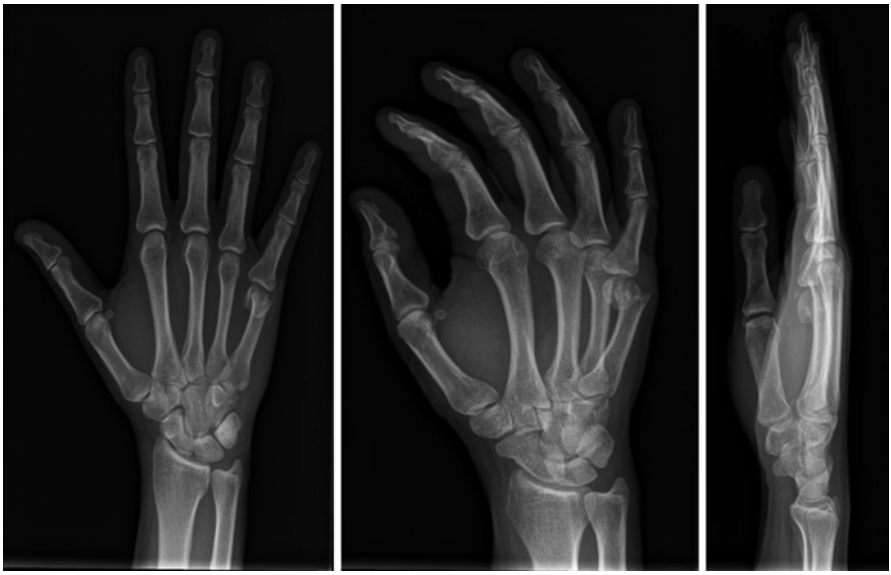


Fig. 2.3 Fifth metacarpal neck fracture or commonly called Boxer's fracture

Investigations

Three radiographic views (AP, Oblique and Lateral) are necessary for adequate assessment of metacarpal injuries. All three views are crucial to avoid missing a fracture (Fig. 2.4).

Classification

Metacarpal (MC) fractures of the non-thumb are divided into head, neck, shaft and base fractures.

Treatment

All MC fractures require reduction, splinting and referral. The most common method after immobilisation would be a Position Of Safe Immobilisation (POSI) volar slab or Radial (for digit 2–3) and Ulnar gutters (for digit 4–5). The ideal position will be wrist extension at 30°, MCP joint at 90° flexion and extended interphalangeal at 0° (Fig. 2.5). Complicated injuries should be referred to hand surgeons (Table 2.4).

Boxer's fracture: Closed reduction is performed using the Jahss or the 90–90 manoeuvres. A wide degree of angulation is considered acceptable in these frac-

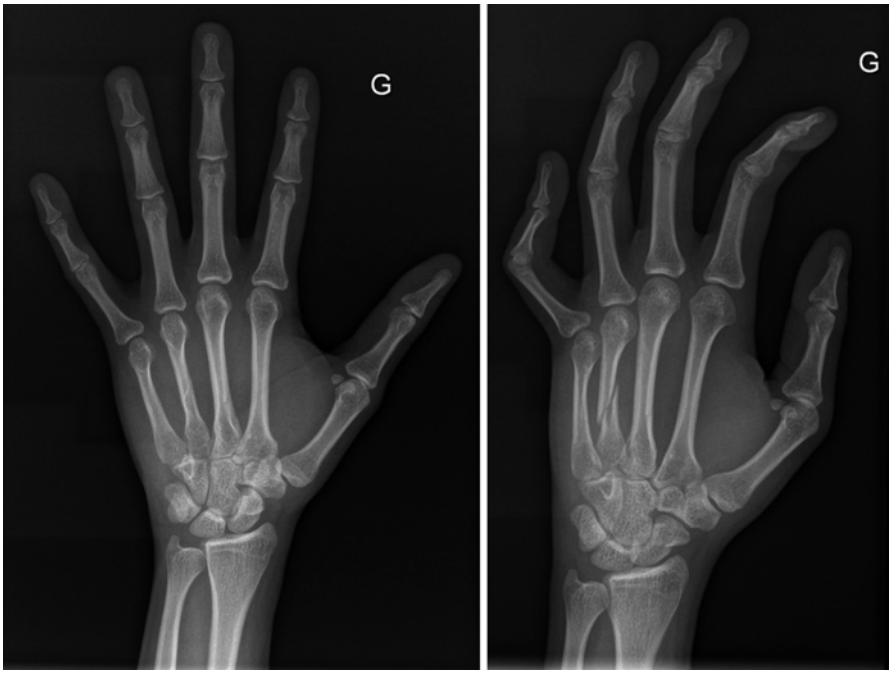


Fig. 2.4 Importance of at least two views: fourth metacarpal fracture almost not visible in the AP view and very obvious in the oblique view

tures. A Cochrane review suggested that there was no evidence of function reduction of unacceptable deformity in those with more marked mal-union. Immobilisation will be as per other non-thumb MC fractures with ulnar gutter or POSI volar slab.

Prognosis

Head fractures are associated with cartilage involvement and joint disruption. Literature review suggests no significant difference in outcomes in non-thumb metacarpal fractures in splinting methods and excellent outcome in fifth MC fractures treated conservatively.

Fractures of Phalanges

Pathophysiology

Phalangeal fractures are extremely common. The mechanism of injury varies from sports injuries in young adult to machinery and falls in older adults. High-energy injuries are more likely to require specialist input early on in the management. Complicated fractures should be referred for surgical input (see Table 2.5).

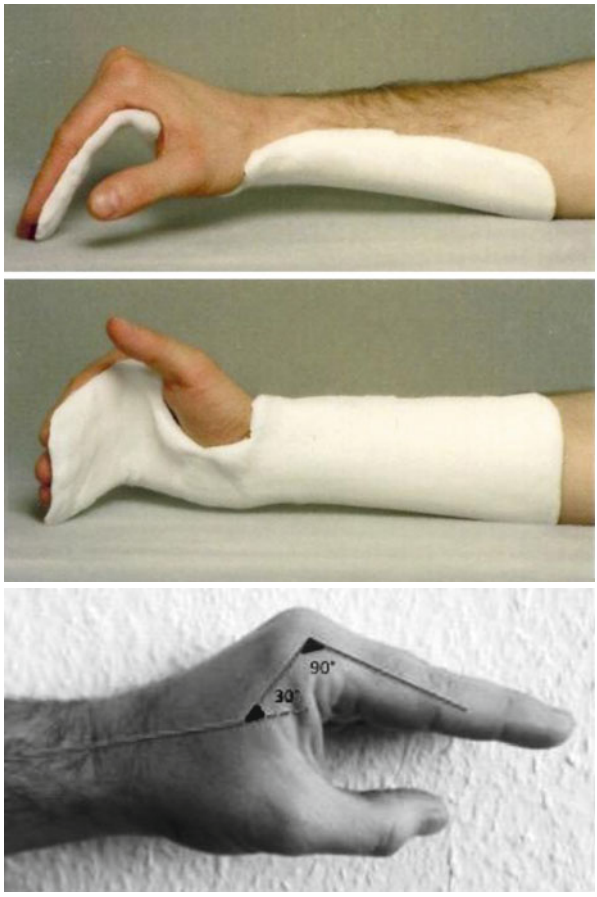


Fig. 2.5 POSI volar slab plaster of Paris and ideal position for immobilisation

Table 2.4 Metacarpal injuries requiring hand surgeon's consultation in the ED

Key points
Emergency hand consultation MC injuries
Open fractures
Rotational deformity
Difficult reductions
Fight bite injuries associated with Boxer's fractures

Table 2.5 Criteria for urgent surgical referral in phalangeal fractures

Key points
Urgent surgical referral criteria for phalangeal fractures
Amputations
Open fractures
Irreducible fractures
>10° angulation
2 mm shortening
Rotational deformity
Complicated fractures
Significant soft tissue injury associated
Intra-articular fracture with >30 % of articular surface involvement

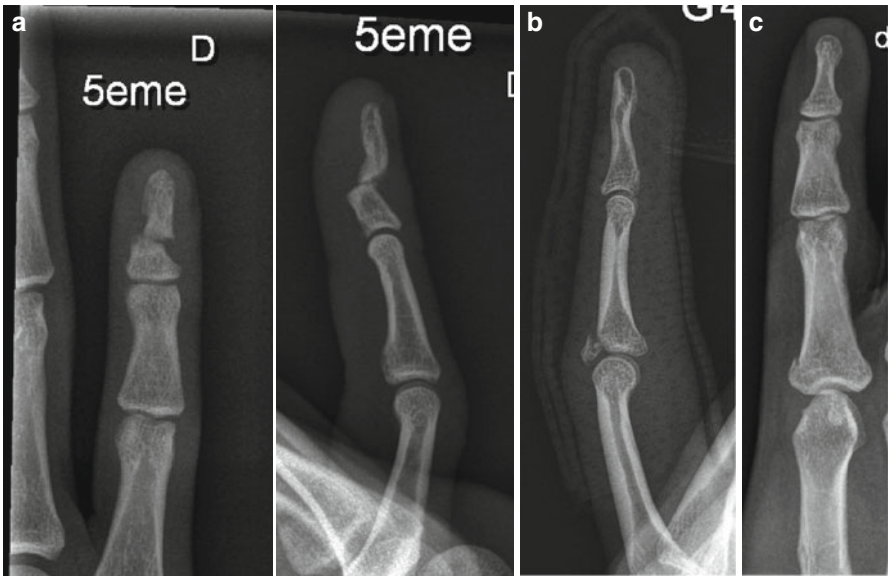


Fig. 2.6 Example of (**a**) distal, (**b**) middle and (**c**) proximal phalangeal fractures

From a management point of view, simple fractures are divided into two categories (Fig. 2.6):

- Distal fractures and
- Middle and proximal fractures

Clinical Features

Common presentations are pain with swelling, rotational deformity (especially in middle and proximal fractures) and loss of motion. Proximal fractures usually have a volar angulation due to the flexion of the proximal fragment from the interossei muscles and an extension of the distal fragment. Middle fractures can either have a volar or dorsal displacement depending on the fracture site with regards to the FDS (flexor digitorum superficialis). They are often associated with soft tissue injuries that must not be overlooked during the examination.

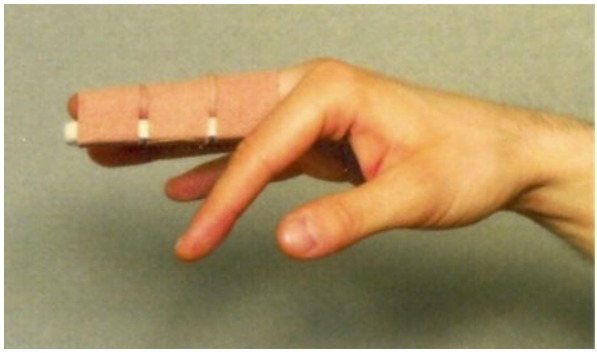


Fig. 2.7 Volar digital splint

Investigations

Two views, AP and lateral, radiographs are recommended.

Classification

This is divided into tuft, shaft or base fractures. Oblique shaft fractures are more likely to be unstable.

Treatment

Displaced closed distal phalanx fractures need to be reduced and immobilised by a volar digital splint for 2–3 weeks and referred to hand clinic (Fig. 2.7). Tuft fractures are often associated with nail plate avulsion or nail bed injuries and the plate may have to be removed. Lacerated nail bed should be sutured and can be splinted using the avulsed nail or gauze.

There is evidence to show that low-risk open distal fractures do not need prophylactic antibiotics unlike high-risk fractures [4–9].

Displaced proximal and middle phalanx fractures require closed reduction and splinting. The current literature suggests that middle fractures can be immobilised for 2–3 weeks with a dynamic splint or buddy taping if the result of the reduction is adequate. Proximal fractures, because of the instability of the oblique fractures, tend

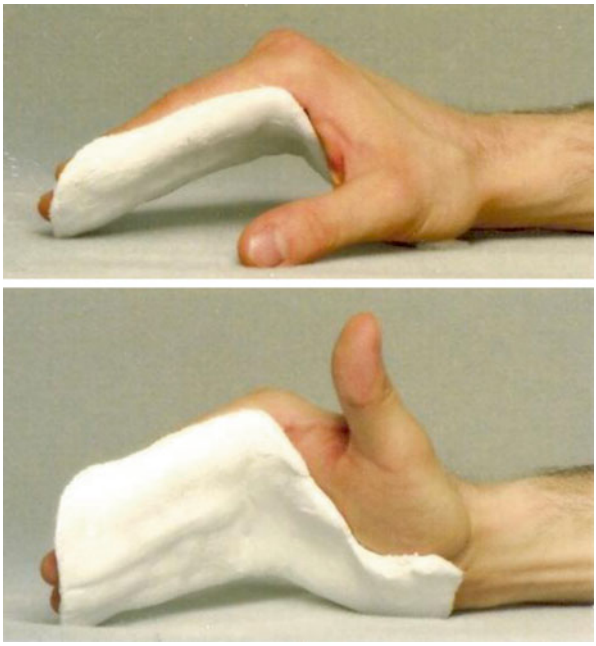


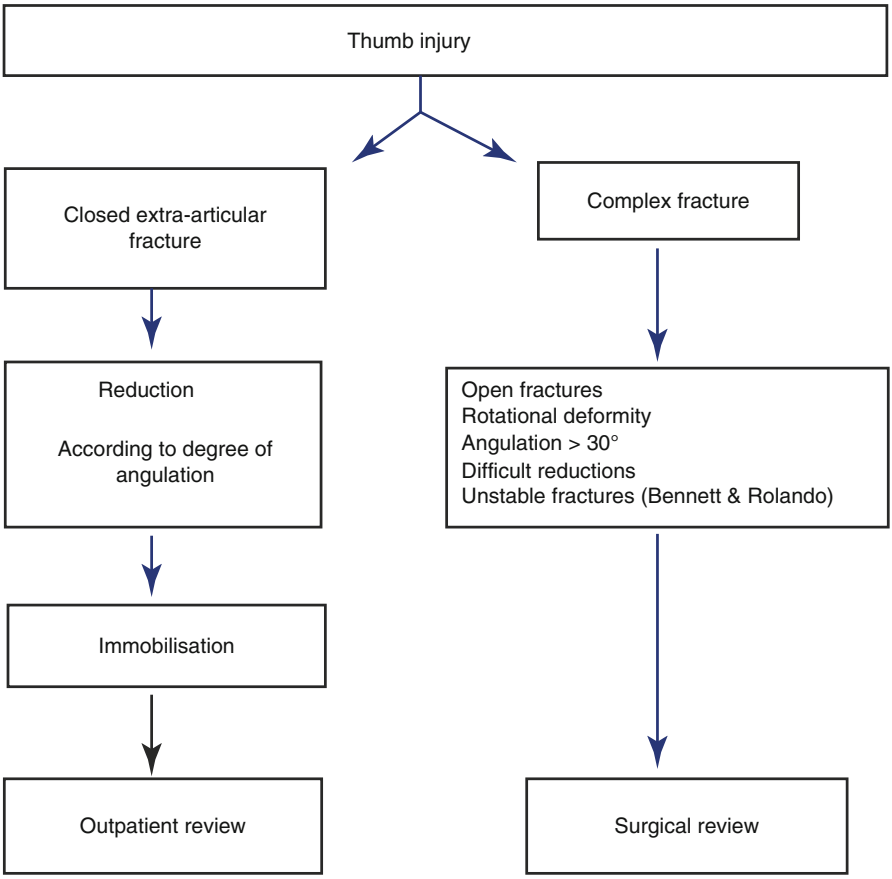
Fig. 2.8 Ulnar gutter splints

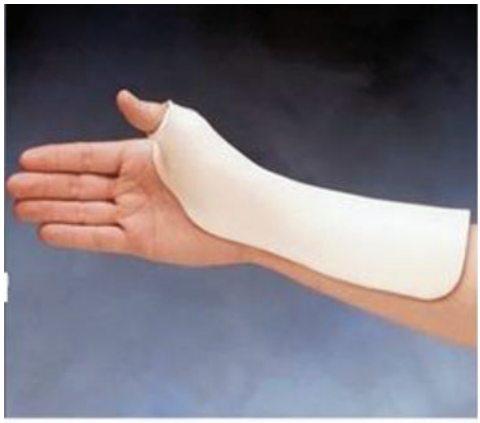
to be immobilised using a radial or an ulnar gutter splints for 2–3 weeks. Some hospitals will immobilise both middle and proximal phalanx fractures using POSI without wrist immobilisation (Fig. 2.8). Complicated phalangeal injuries should be referred to hand surgeons (Table 2.5).

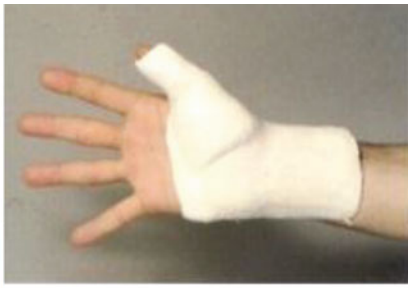
Prognosis

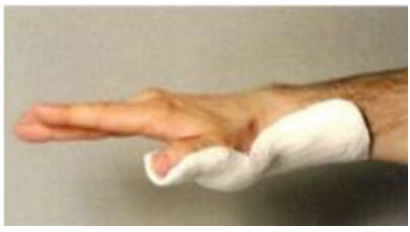
Loss of motion is the most common complication associated with fractures with articular involvements or those requiring extensive dissection. Prolonged immobilisation is associated with significant functional impairment making early rehabilitation extremely important.

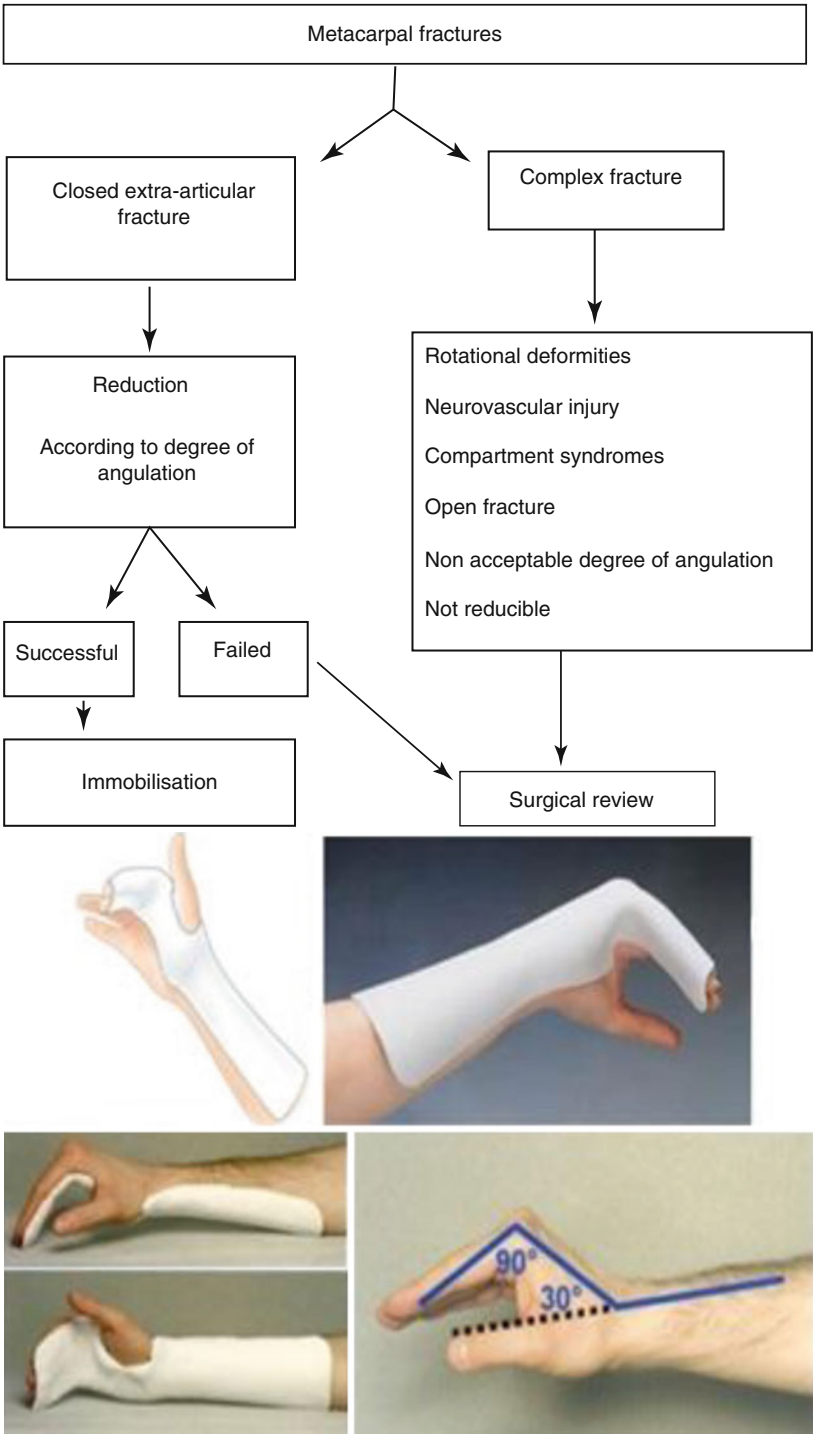
Difficult reduction of displaced fractures (malrotation, angulation and shortening) may result in mal-union requiring surgical treatment. Non-union is uncommon but may happen in distal fractures but rarely need fixation unless symptomatic.

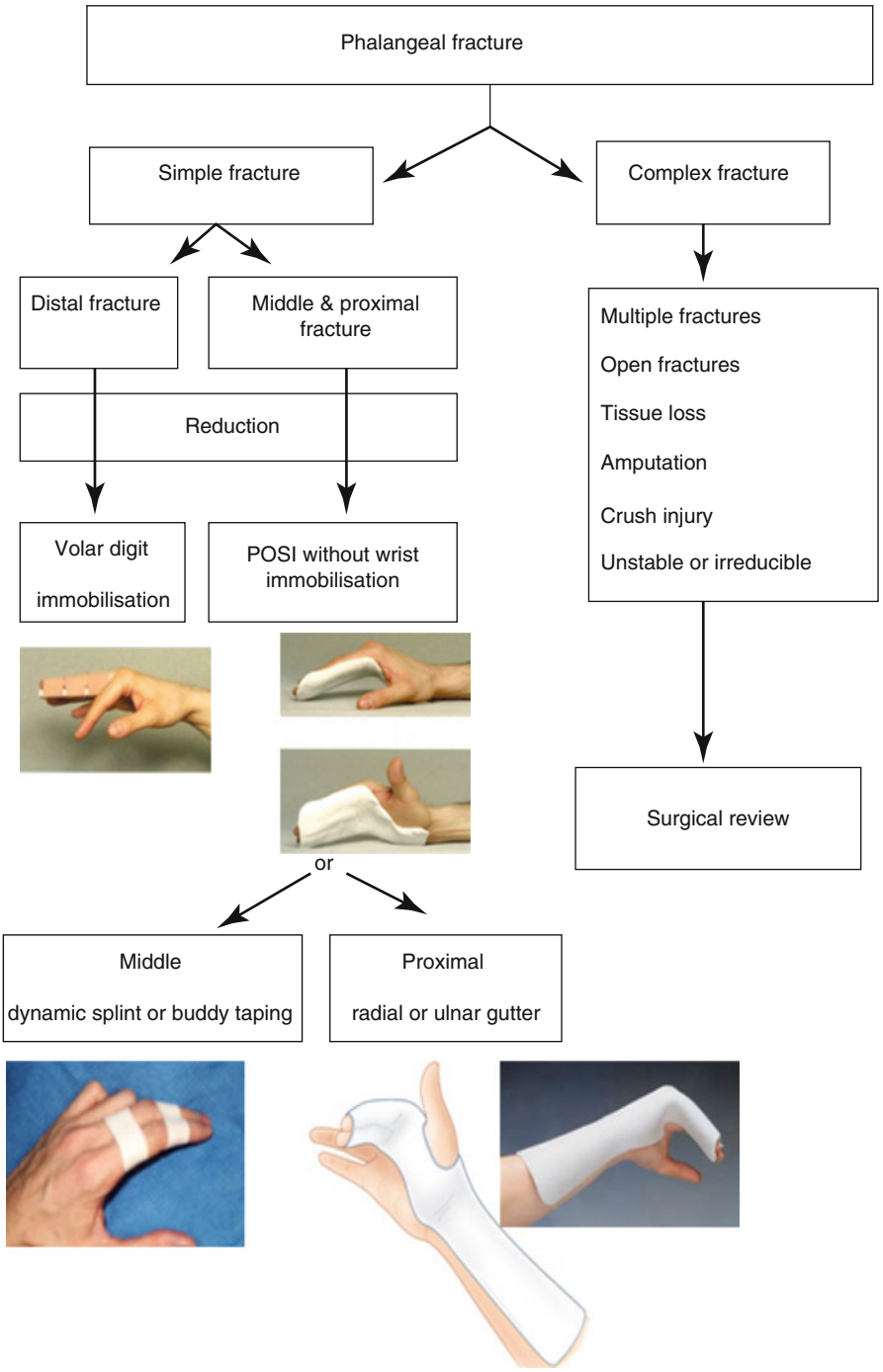

Thumb injury
Closed extra-articular fracture
Complex fracture
Reduction
According to degree of angulation
Open fractures
Rotational deformity
Angulation > 30°
Difficult reductions
Unstable fractures (Bennett & Rolando)
Immobilisation
Outpatient review
Surgical review








Metacarpal fractures
Closed extra-articular fracture
Complex fracture
Reduction
According to degree of angulation
Rotational deformities
Neurovascular injury
Compartment syndromes
Open fracture
Non acceptable degree of angulation
Not reducible
Successful
Failed
Immobilisation
Surgical review
90°
30°


Phalangeal fracture
Simple fracture
Complex fracture
Distal fracture
Middle & proximal fracture
Multiple fractures
Open fractures
Tissue loss
Amputation
Crush injury
Unstable or irreducible
Reduction
Volar digit immobilisation
POSI without wrist immobilisation
Surgical review
or
Middle
dynamic splint or buddy taping
Proximal
radial or ulnar gutter

Dislocations

All dislocations should have an x-ray to ascertain whether associated fractures are present.

MCP Dislocations

Most dorsal MCP dislocations are easily reducible. Complex fractures dislocation have a palpable volar metacarpal head and are very difficult to reduce and often require surgical input due to the tightening of both the lumbricals and ligaments around the injury. Simple reduction should be immobilised at15° flexion in a POP for review in fracture clinic.

Thumb Dislocation

Thumb dislocations tend to happen with a fall onto it or from hyperextension. These injuries can be associated with a deeper injury due to the axial loading and should be reduced and immobilised in a scaphoid plaster with a hand clinic follow up.

Phalanx Dislocation

Phalangeal dislocations may be associated with collateral ligament injury and may require different immobilisation (Fig. 2.9). Simple reducible dislocation can be immobilised using finger tapping and follow up should be arranged in hand clinic.

Amputations

Acute treatment of amputation should include fragment storage, haemorrhage control, analgesia and wound care. Most amputations should have an urgent surgical review in the ED.

Ligamental Injuries

Ligamental injuries are not surgical emergencies and may therefore be treated with delayed repair. Treatment may differ from hospital to hospital. Simple ligamental injuries can be reviewed in hand clinic at a later stage.

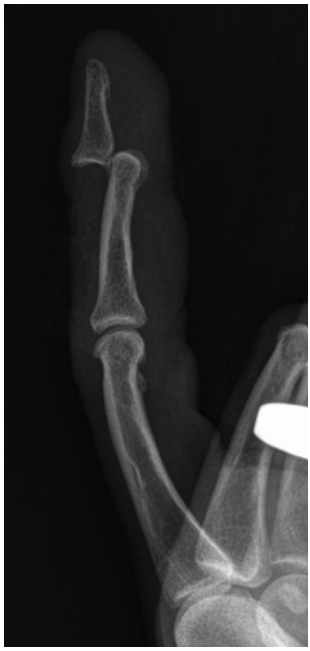


Fig. 2.9 Distal phalanx dislocation

Emergency treatment of open ligamental injury involves analgesia and thorough wash outs to reduce the risk of wound infections in open injury prior to delayed repair usually within 7–10 days. Complex ligamental injuries should be referred to hand surgeons immediately.

Flexor Tendon Injury

Pathophysiology

Usually from laceration or forced extension during finger flexion.

Clinical Features

The patient is unable to flex the DIP or PIP joint, IP joint at the thumb or the wrist.

Investigations

Imaging should be performed to exclude fracture-dislocations and foreign body if open.

Management

Flexor tendon injuries should be immobilised and referred to the hand clinic within 7 days unless associated with a complex injury. A partial tear may not require any surgical intervention.

As discussed earlier, all open wound should be irrigated and cleaned. Wounds can be closed in simple injuries and splinted until the outpatient review. Antibiotic cover should be given as well as tetanus prophylaxis in accordance with local protocols.

Extensor Injuries

Clinical Features

This injury is suspected when the patient cannot extend at the MCP joint, Thumb, IP joint of the thumb or wrist. If the whole hand is affected, an ulnar nerve injury must be excluded.

Investigations

Imaging should be performed to exclude fracture-dislocations and foreign body if open.

Management

In open cases, the wound can be explored and injury repaired depending on local expertise. Otherwise, delayed repair should be organised as per flexor injuries. The hand should be immobilised until hand clinic review within 7 days. Digit 2–5 should be immobilised in a volar splint (MCP 10–15° flexion and IP straight) and thumb injuries should be put in a spica splint.

Prognosis

Immobilisation can last 4–6 weeks post repair with usually good outcome.

Mallet Finger

Pathophysiology

This is an injury of the extensor tendon at its insertion site at the distal phalanx secondary to a forced flexion of the distal phalanx (Photo). The deformity can be due to a rupture or an avulsion fracture at the distal phalanx (Fig. 2.10).

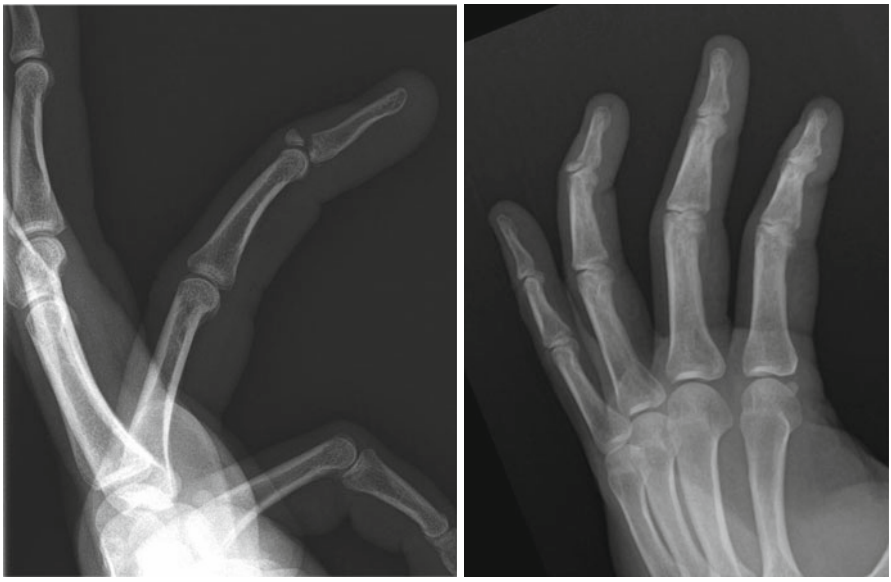


Fig. 2.10 Avulsion fracture of distal phalanx of ring finger associated with 2 mm displacement commonly called Mallet Finger injury

Clinical Features

The patient presents with an inability to extend at the DIP. It can be associated with a fracture, which can be either open or closed.

Investigations

An x-ray of the digit should be performed to ascertain if a fracture is present, and if so, how much of the joint may be affected.

Management

The digit should be immobilised using a Mallet splint for 6 weeks. Splinting can also be performed using a tongue depressor. The splinting can either be dorsal or volar (Fig. 2.11). The Proximal joint should not be immobilised and the distal joint should not be hyperextended due to a rare but potential risk of skin necrosis.

Operative management may be used in those with a fracture involving 40–50 % of the articular surface. Simple open mallet injuries can either be repaired in the ED or in theatres depending on local expertise.

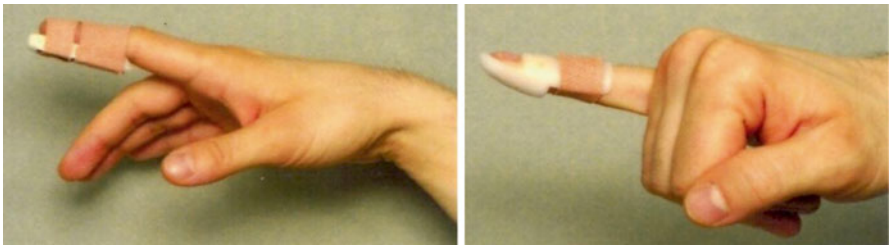


Fig. 2.11 Mallet splint or zimmer splint

Prognosis

Delay in treatment should not affect the outcome. Some patient may require longer immobilisation, if the weakness is still present.

Digital Collateral Ligament Injury

The collateral ligaments play an important role in the stability of joints. The degree of laxity should be compared with the opposite finger. If a weakness in one of the ligaments is found during stress testing, the joint must be immobilised for a period of 10–14 days in the same manor as a proximal phalangeal fracture using a gutter splint.

Soft Tissue Injury of the Hand

Compartment Syndrome

It is a rare but potentially devastating injury making early referral is essential. There are various causes and the course maybe insidious (Table 2.6). There are four potential areas at risks, the thenar and hypothenars, the three palmar spaces and the four interossei spaces. Thorough examination and high degree of suspicions are necessary to prevent long-term damage (Table 2.7). Compartment pressure is not reliable and should not be performed in the ED. Early referral if clinical suspicion is essential.

Lacerations

Simple lacerations should be closed in the ED. Historically, wound older than 6 h should be treated with delayed closure. However, there are no absolute contraindications for primary closure. The history and examination of the wound are the most important factors dictating the management strategies.

Table 2.6 Causes of compartment syndrome in the hand

Key points
Causes compartment syndrome in the hand
Crush injuries seen in machinery accidents
High-pressure injection injuries
Extravasation of IV contrast and arterial line misplacement
Rarely following prolonged immobilisation in MC fractures

Table 2.7 Six Ps associated with compartment syndrome

Key points
Suspicious of compartment syndrome
Pain
Pallor
Paraesthesia
Pulselessness
Paralysis
Poikilothermia (temperature changes)

Closure is usually performed using non-absorbable interrupted sutures. However, there is no current evidence suggesting any advantages of absorbable versus non-absorbable sutures being used. Suture removal depends on the site of the laceration but it is generally 10–14 days. More days should be allowed in sites of potential tension such as the palm that may require up to 21 days. All at-risk wounds should be referred for hand surgeon review [10].

Degloving Injuries

All degloving injuries should be referred for an urgent surgical review.

Fight Bite Injury

Those injuries are high-risk injuries associated with structural and infectious complications such as tenosynovitis and osteomyelitis and should be referred for surgical review. In fact the literature suggests an associated complication rate of about 75 % (joint violation, tendon injury, fracture and cartilage injury).

Table 2.8 Signs associated with pyogenic flexor tenosynovitis

Kanavel's signs
Symmetrical swelling
Tenderness of flexor tendon
Passive extension very tender
Fixed flexion of affected finger

Deep Tissue Infections

Pyogenic Flexor Tenosynovitis

It is an infection of the flexor tendon sheath associated with a penetrating injury usually at the crease of the digit, as this is the most superficial part of the tendon. The presentation is insidious as this could have happened several weeks back and may present as a simple redness over the palmar aspect of the finger.

Diabetes, immunosuppression and intravenous drug use are the main risk factors. The classical sign is "Kanavel's signs" (Table 2.8) [10–12].

These should all be referred for urgent exploration and IV antibiotics.

Complications are very severe; the infection can spread down the sheath and infect the radial bursa (if thumb infection) or the ulnar bursa (other four digits). In 50–80 % of patients both bursa communicate so the infection can spread to the whole palmar aspect creating a "Horseshoe" abscess. This can spread further down and infect deep tendons and lead to severe damage and sepsis.

Wrist Trauma

The wrist injuries are one of the most common injuries worldwide. Distal radio ulnar injuries will be covered in the upper limb section.

Carpal Bone Fractures

Apart from scaphoid fractures, carpal bone fractures are very rare. The most common mechanism of injury is the fall onto an outstretched hand (FOOSH). And the examination is often difficult due to pain and deformities. It is important to identify whether the injury is likely to be carpal or distal upper limb to order the right imaging modality [14].

Scaphoid Fractures

Pathophysiology

Scaphoid injuries present following two potential type of injuries: FOOSH and the so-called "kick back injury" common with hard handle or steering wheel injuries.

Clinical Features

The most common presentations are pain, swelling over the radial side of the wrist and diminished grip strength was also identified in over 50 % of cases. On examinations, pain can be elicited over the anatomical snuffbox and the palmar aspect of the scaphoid. There can also be pain on longitudinal pressure of the thumb and ulnar deviation of the wrist. The sensitivity of those symptoms is a 100 % but specificity is variable.

Investigation

Specific scaphoid views (AP/Lateral Right and Left oblique) should be performed but negative x-rays do not exclude such injuries (Fig. 2.12). Highly suspicious injury should be treated as potential fractures in view of the serious complication encountered in missed fractures.

Classifications

There are various classifications based on anatomical position (Mayo, Russe and Herbert's based on the stability of the fracture) that do not add any value into the emergency treatment.

Treatment

All non-displaced fractures should be immobilised in a scaphoid POP with hand clinic review in 10–14 days, although some would advocate a follow up imaging within 7 days [15]. They may require further 6 weeks depending on local protocols. Delayed established fractures and should be referred to hand clinic. Suspicious mechanism with minimal tenderness and negative or inconclusive imaging should be immobilised with a thumb splint and reviewed in hand clinic.

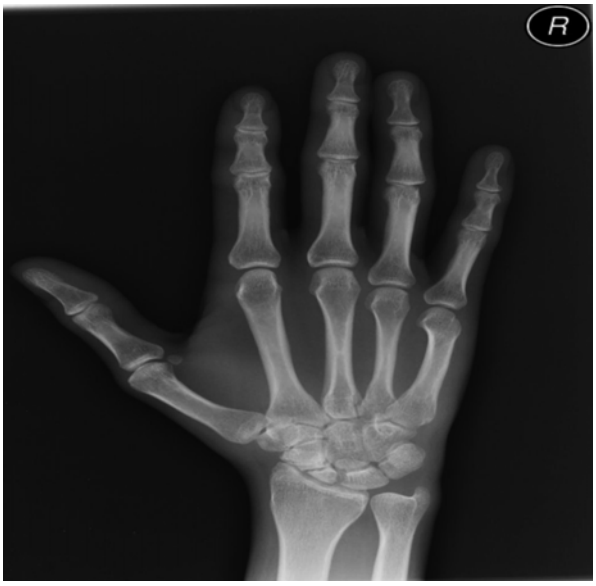


Fig. 2.12 Scaphoid fracture of the right hand

Table 2.9 Adverse event associated with scaphoid fractures

Main adverse event associated with scaphoid fractures
Delayed union
Osteonecrosis
Pseudoarthrosis
Arthrosis
Instability
Carpal joint collapse

Prognosis

Complications of delayed or inappropriate fractures management can lead to significant functional restriction (Table 2.9).

Triquetral Fractures

Pathophysiology

These are the second most common carpal fracture after scaphoid fractures. The mechanism is believed to be a fall with dorsiflexion and ulnar deviation.

Clinical Features

Focal pain over the dorsal aspect of the triquetrum suggests an avulsion fracture.

Investigations

Three views radiography should be performed and may be identified in a lateral and oblique view. If the injury is suspected, CT may be performed.

Classification

Most commonly seen are body fractures, less likely are volar avulsion and dorsal cortical fractures.

Treatment

Although operative treatments may be considered in the future, immobilisation with POP slab for 4–6 weeks with organised review is the treatment of choice in the acute phase.

Prognosis

There is excellent prognosis after 6–8 weeks with reduced pain and return to normal wrist motion. In poor outcome, luno-triquetral injury should be considered.

Trapezium Fractures

Usually, trapezium fractures result from an axial loading onto the thumb. It can be associated with Bennett's fracture. Non-displaced fractures can be immobilised with a thumb splint or spica splint for 4–6 weeks. In cases of displaced fractures, there is some argument for earlier hand surgical review to ascertain earlier operative options in young patients to reduce potential arthritis secondary to the displacement.

Wrist Instability and Collapse

Lunate Dislocation (Perilunate Dissociation)

This is a rare injury resulting from high-energy impact fall on a wrist extension and ulnar deviation. Common presentations are similar to all FOOSH with swelling and tenderness but can also be associated with median nerve paraesthesia.

The lateral x-ray would demonstrate the up lifting from the rest of the carpal bones and the concave surface facing the volar aspect (spilled tea cup appearance). The lunate is neither articulated with the radius or the capitate as opposed to perilunate dislocation that demonstrates a radial articulation.

All lunate dislocation should be referred for surgical review. There are associated with median nerve injury, avascular necrosis, complex regional pain syndrome and rarely progressive weakness. Patient may find it difficult to do push ups or bear any weight across the wrist.

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Chapter 3
Lower Limb

David Paradise and Savvas Papasavvas

Key Points
- Key priorities in the management of neck of femur fractures are analgesia, early preoperative optimisation and early post-surgical mobilisation.
- Management of femoral shaft fractures should ensure haemodynamic resuscitation and haemorrhage control as priorities due to potential for significant blood loss.
- Imaging of knee and ankle can be used appropriately by application of simple decision rules aimed at reducing unnecessary radiographs.

Introduction

The lower limb is responsible for bearing significant load in many everyday activities. The anatomy of the limb and joints reflects how these loads are translated during movement. As a result of its key role in movement, injury to the lower limb can result in significant morbidity and in some cases mortality.

- Global projections indicate that the number of hip fractures alone will rise from 1.66 million in 1990 to 6.26 million by 2050 [1].
- Average length of stay in the United Kingdom for lower limb injury is 9.1 days versus 3.0 for upper limb injury [2].

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This chapter will cover the most common lower limb injuries that are likely to be encountered in everyday practice. Each of the most common clinical presentations is covered, and associated pathophysiology, investigations and immediate management options are outlined. As with all trauma presentations, continual assessment for life-threatening injury should precede detailed limb and joint examination.

For all the injuries described in the chapter, the following key factors will improve the diagnosis:

- Focus on thorough history and examination.
- Attempt to translate mechanism into suspected injury patterns.
- Combining anatomical knowledge with an understanding of the injury mechanism will improve diagnosis.

Neck of Femur Fracture

These fractures occur in the proximal portion of the femur, specifically from the most proximal point to 5 cm distal of the greater trochanter.

Pathophysiology

- Incidence increases with age.
- Most common in elderly women.
- Most likely mechanism is a fall onto the affected side.

Clinical Features

Diagnosis of hip fractures will be initially based on mechanism likely to result in a fracture, such as a fall onto the affected side.

- If fractured, palpation over the anterior femoral neck and the greater trochanter may elicit pain.
- Fractures will cause pain on rotation of the affected limb.
- Many patients will not be able to bear weight, but this is not universally the case.
- Shortening and external rotation of the affected limb are important signs but are not always present.

Differential Diagnosis

- Pubic rami fractures
- Acetabular fractures

Investigations

Consider pelvic or hip imaging in acutely or chronically confused patients following fall where a good history and examination are difficult.

If the fracture line is difficult to identify on initial AP pelvis and lateral hip x-ray, look carefully for asymmetry of trabeculations and Shenton's lines [3]. If doubt persists, then consider early CT or MRI; undisplaced fractures are often missed especially if patients can bear weight following the injury.

Fracture classification is based on the level at which the fracture is in comparison to the insertion of the joint capsule. The joint capsule inserts along the intertrochanteric line. Fractures are defined as either intracapsular or extracapsular. Extracapsular fractures can be further defined as trochanteric or subtrochanteric (Fig. 3.1).

Intracapsular fractures are more prone to complications as they may interrupt vascular supply to the femoral head resulting in avascular necrosis. The head is often very fragile and may provide difficulties for any surgical fixation. Definitive management decisions are based on many factors, but displacement of the fracture plays a major part. The degree of displacement is assessed using the Garden classification [4]; see Table 3.1; type 3 and 4 fractures carry the worst prognosis.

Treatment

The priorities are:

1. Analgesia (increasing opioid requirements in elderly patients means that nerve blocks such as the fascia iliaca block [5] may provide adequate analgesia without potential for opioid-based side effects)

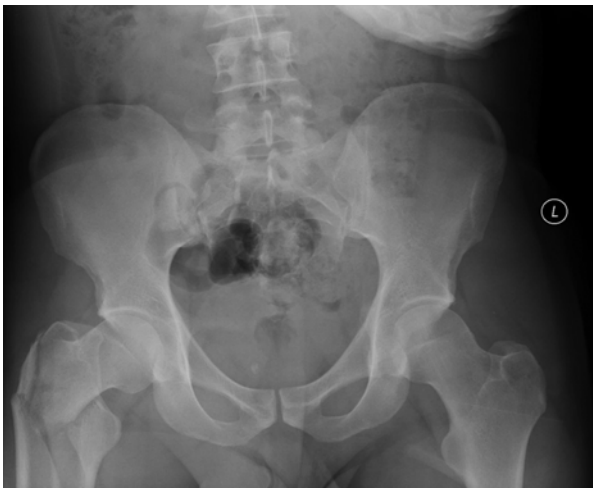


Fig. 3.1 Right comminuted intertrochanteric fracture

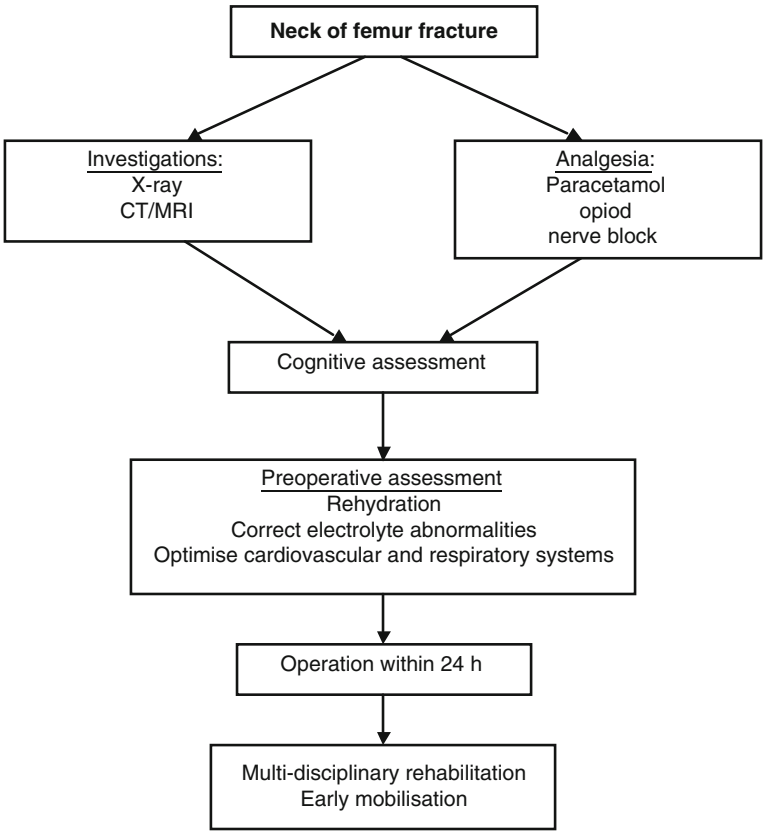
Table 3.1 Garden classification

Type 1	Inferior cortex not completely broken, but trabecular lines are angulated with valgus impaction
Type 2	Fracture line complete but no angulation and no displacement of fracture seen
Type 3	Obvious fracture line, associated femoral head rotation in the acetabulum, slightly displaced
Type 4	Fracture is fully displaced

Adapted from Garden [4]

2. Investigations to reflect the individual patients likely needs preoperatively.
3. Resuscitation and preoperative optimisation should begin at the earliest opportunity. A management algorithm can be seen below.

Neck of Femur Algorithm



Early input from a multidisciplinary team, including a geriatrician, has been shown to improve outcomes [6]. The goal of management is to operate at the earliest opportunity to allow the patient to mobilise early in the post-operative period.

Surgical Management

- NICE in the United Kingdom [7] recommends that displaced intracapsular fractures (types 3/4) should undergo hemiarthroplasty or total hip replacement.
- For type 1/2 injuries, closed reduction and internal fixation is often the operative choice [7].
- Trochanteric fractures are commonly managed with extramedullary devices such as the sliding hip screw.
- Extratrochanteric fractures will require intermedullary nails [7].

Prognosis

Neck of femur fracture carries high mortality and morbidity.

- UK mortality one-third in first year post injury.
- Significant rehabilitation required.
- Annual cost of hip fractures in the United Kingdom is £2 billion [7].

Prevention

- Falls prevention
- Improving bone density in elderly female patients [8]

Femoral Shaft

Pathophysiology

- High-energy transfer mechanisms are likely in young people.
- May result from simple falls in the elderly/frail.
- Assume femoral shaft fractures are associated with significant other injuries.

Clinical Features

Obvious deformity and swelling to the femur associated with significant energy transfer trauma should raise the possibility of femoral fracture.

Differential Diagnosis

Large hematoma and soft tissue injury may result in significant limb swelling but are not likely to impair weight bearing.

Investigations

AP and lateral x-rays should be performed but can be delayed until traction splints are applied and hemodynamic stability is restored (Fig. 3.2). Many forms of splint are readily available and may have already been applied by prehospital

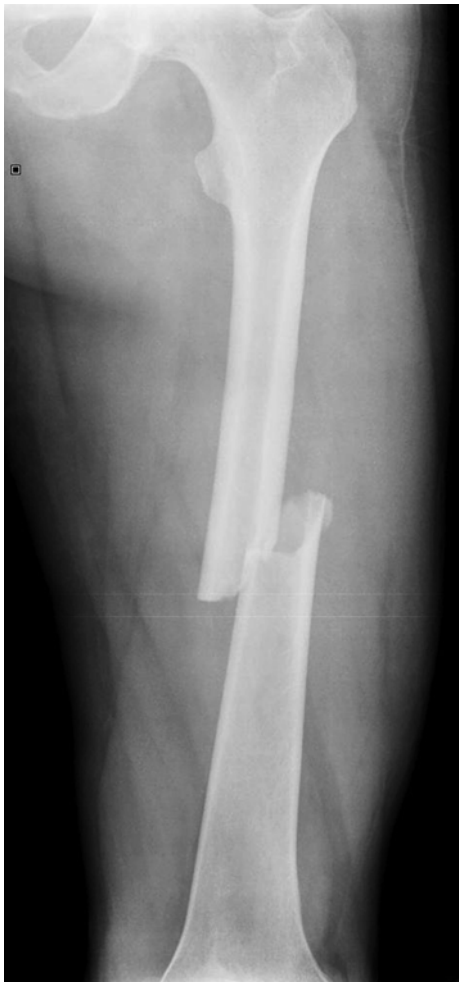
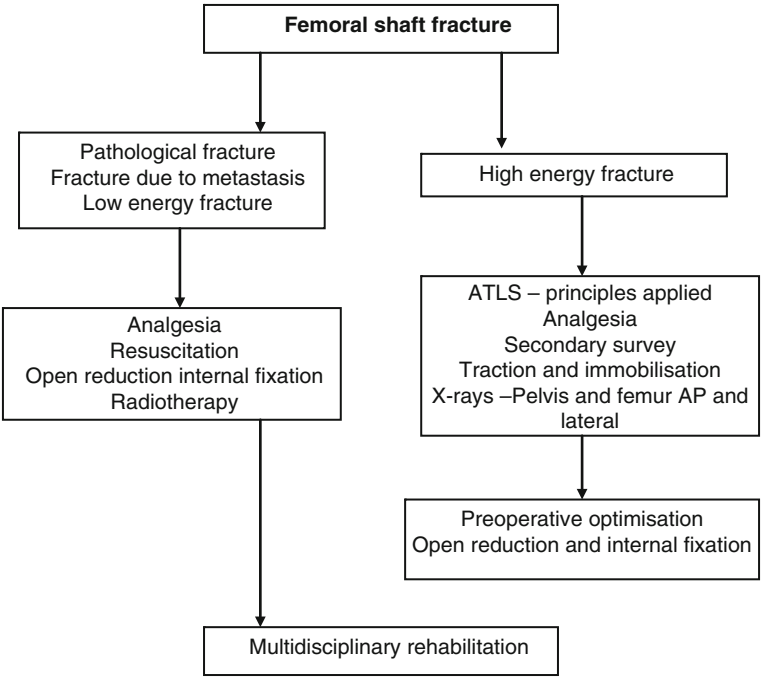


Fig. 3.2 Mid-shaft fracture of the left femur

practitioners. In this case, if position and effect are considered sufficient, leave in situ.

Femoral Shaft Fracture Algorithm



Treatment

- Consider analgesia as appropriate.
- Active resuscitation and haemorrhage control.
- Control haemorrhage by reduction of fracture reducing potential for ongoing bleeding.
- Apply traction device to maintain reduction of fracture.

A treatment algorithm can be seen above.

Definitive management will require specialist opinion and likely operative management for intermedullary nail.

Prognosis

Recovery will depend on severity of the original injury. Fracture union should occur over a period of 10–14 weeks depending on mechanism and severity of injury. Many factors will affect full recovery including reduction in length of affected leg and reduction in power of hamstrings. Studies indicate a wide range time to return to work, one study suggesting up to 30 weeks as an average [9].

Prevention

Accident prevention strategies should be supported by health care services and government agencies with the aim of reducing these types of injury. For low-energy accidents, falls prevention work may also be of benefit.

Knee

Anatomy

Four ligaments support the articulation of the femur on the tibia. The anterior and posterior crucial ligaments reduce anterior and posterior displacement of the tibia relative to the femur. The lateral and medial collateral ligaments prevent the knee being forced to open up in a lateral or medial direction.

Pathophysiology

There are multiple components to the knee, and each will be injured by differing mechanisms.

Clinical Features

Injury to any component of the knee will often result in significant swelling and pain making examination difficult. An appropriate mechanism of injury associated with inability to bear weight or reduced range of movement of the knee may be present. Careful examination of the joint line and ligaments should be attempted if pain allows in an attempt both to achieve more accurate diagnosis and provide an assessment of stability. Decisions about imaging are equally problematic. Imaging rules developed by Stiell et al. are designed to reduce unnecessary radiographs [10].

Imaging of the knee is only required if patients meet any of the following criteria:

1. Age 55 or older
2. Isolated bony tenderness on palpation of patella (no bone tenderness of knee other than patella)
3. Bony tenderness of proximal head of fibula
4. Flexion less than 90°
5. Inability to bear weight immediately after incident and for four steps in the emergency

Condylar Fractures

Fractures in the distal portion of the femur between or at the level of the condyles are likely secondary to significant force. Often this type of fracture will be significantly displaced due to the action of gastrocnemius on the distal portion of the femur (Fig. 3.3). Unless there is no displacement, all fractures at the level of the condyles will require operative fixation.

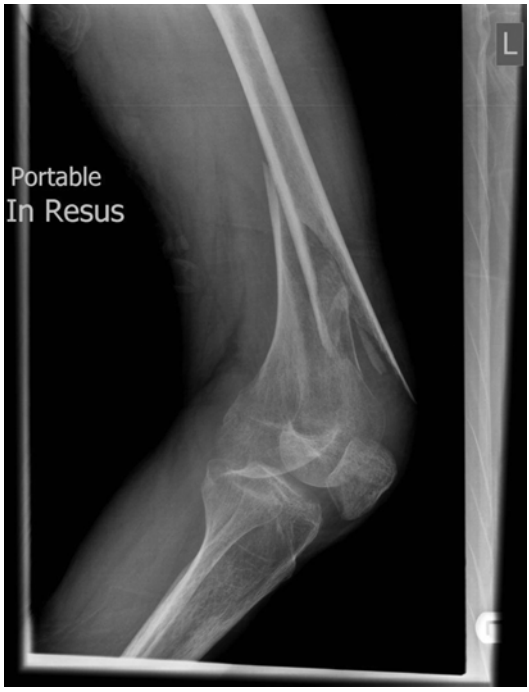


Fig. 3.3 Comminuted left distal femur fracture

Ligamentous Injury

Ligaments provide the majority of stability to the knee joint, and significant morbidity can result from injury.

Pathophysiology

Valgus or varus force through the knee is likely to result in ligamentous injury.

Clinical Features

Signs of injury such as swelling and pain are significant findings in themselves but also make detailed examination of the ligaments and their integrity difficult. Examination should include ligamentous testing; posterior and anterior drawer tests and testing of collateral ligamentous integrity should all be completed if possible. Often if ligaments are significantly damaged, examining these ligaments immediately or in the time initially following the injury may not demonstrate laxity before pain created by the test is too great.

Treatment

Management is initially conservative with immobilisation and support as required.

Prognosis

Further assessment and investigation including imaging such as MRI can often be left until initial phase of injury is improving. Surgical repair may be avoided with good physiotherapy-led rehabilitation.

Patella Injury

Patella Dislocation

This is the loss of congruity in the patellofemoral joint.

Pathophysiology

The stability of this joint is dependent on the depth of the grove in which the patella runs, known as the trochlear groove.

Clinical Features

The history will be of sudden-onset pain and swelling to the knee with inability to mobilise. The patella will be seen or felt to be out of place by the patient. This type of injury often results from changing direction on a planted foot. Many patients will experience recurrent episodes and may have relocated the patella prior to arrival in the emergency department.

Treatment

Immediate management is to relocate the patella. Flexion of the hip and medial pressure on the patella whilst extending the knee should relocate the patella into the trochlear grove. The knee should be supported in extension to encourage the recovery of supporting structures.

Prognosis

If dislocations are recurrent, surgical intervention may be considered.

Patella Fracture

A direct blow onto the patella itself can result in a fracture of the patella.

Clinical Features

If fracture results in separation of fragments, then the extensor mechanism of the knee will be impaired.

Investigations

AP x-rays can often be misleading as bipartite patella can appear as a fracture, and fractures that are undisplaced are difficult to detect. Some displacement is commonly seen in the lateral view (Fig. 3.4).

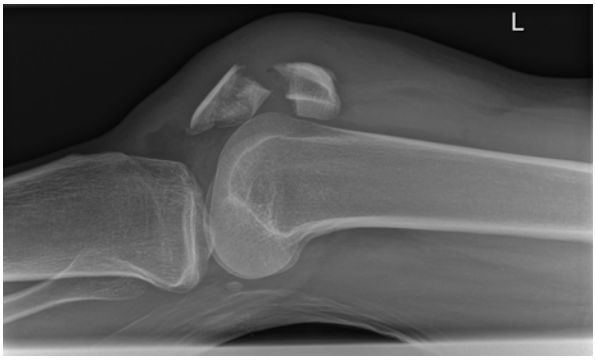


Fig. 3.4 Displaced left patella fracture

Treatment

- Injured leg should be placed in above-knee POP cast.
- Operative management likely if patellofemoral surface is disrupted or significant separation of fragments.

Tibial Plateau Injury

The tibial plateau is a significant joint surface that bears full body weight during all load-bearing exercise. As such any damage to its surface will result in significant morbidity unless managed in an appropriate way.

Pathophysiology

- Valgus stress under axial loading in a patient who is 50 years of age

There is often association between tibial plateau fractures and soft tissue and ligament damage; stress testing for this type of injury is important [11].

Investigations

The fat-blood interface (FBI) sign may be present in 35 % of intra-articular, tibial plateau fractures. This is seen on lateral view x-rays and is a fluid level that develops due to the lower density of fat compared to the blood that lies below it.

Treatment

- Initial long leg plaster of Paris cast.
- Operative management is likely to be indicated unless frail or minimal displacement.

Tibia and Fibula Shaft Fractures (*Figs. 3.5 and 3.6*)

- Fractures affecting the shaft of the tibia are likely to affect the fibula as well.
- Injuries are likely as a result of high, energy transfer.
- Significant danger of compartment syndrome developing. Ensure specific advice regarding compartment syndrome is given to both patient and staff.
- Initial management should be elevation and application of POP back slab.

Injuries to the Ankle

- Common presentation in the majority of emergency departments.
- A combination of bony, ligamentous and cartilaginous structures interacts to ensure stability and flexibility.
- The joint consists of the talus bone sitting in between the distal ends of the tibia and fibula and is held in place by the strong ligaments.

Pathophysiology

Injury occurs when the talus is forcibly moved in an abnormal direction within the confines of the bones and ligaments that surround it.

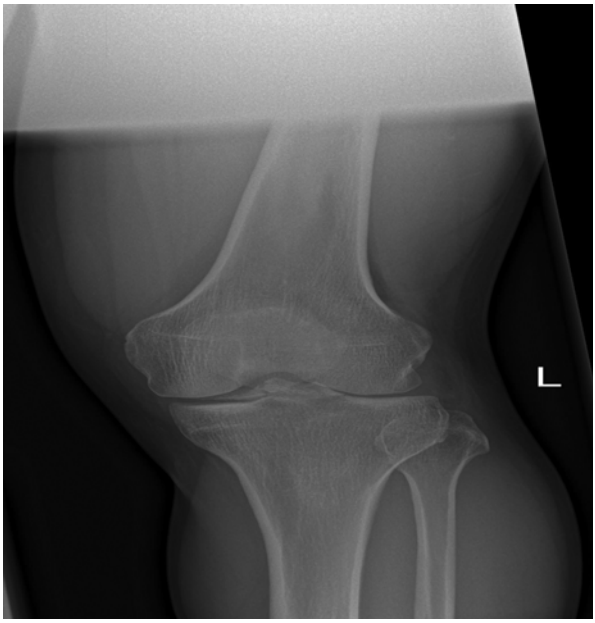


Fig. 3.5 Left tibial plateau fracture

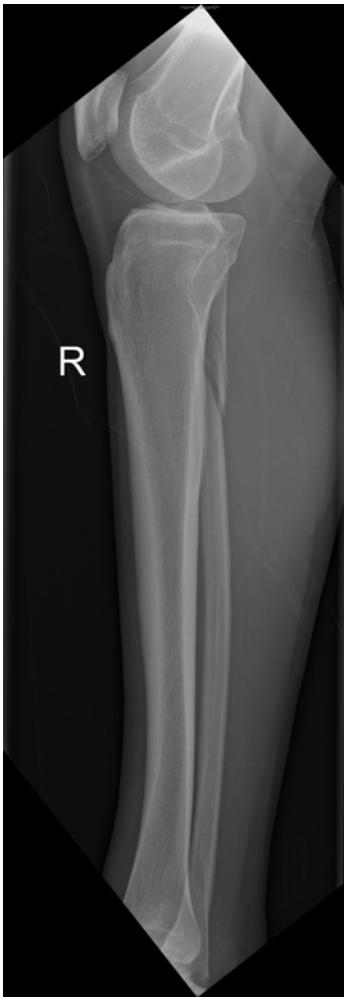


Fig. 3.6 Right proximal fibula fracture

Clinical Features

A good history for ankle injury should include:

- Previous injury and mobility prior to injury
- Mechanism of injury – specifically position of ankle at time of impact or injury
- Initial ability to mobilise following injury
- Other joint/bone problems following incident

If the deformity is suggestive of dislocation or significantly displaced fracture, this should dictate prompt neurovascular assessment. Urgent reduction prior to x-ray should be undertaken if any concern is present regarding vascular compromise secondary to ankle injury.

Investigations

The Ottawa ankle rules aim to reduce inappropriate radiological investigations. X-ray is only required when pain in the malleolar region is associated with three criteria which can be seen below [12]. The x-ray in Fig. 3.7 shows a bi-malleolar fracture of the right ankle with significant talar shift. Furthermore, Fig. 3.8 demonstrates a tri-malleolar fracture of the right ankle, where the posterior component is also fractured (Table 3.2).

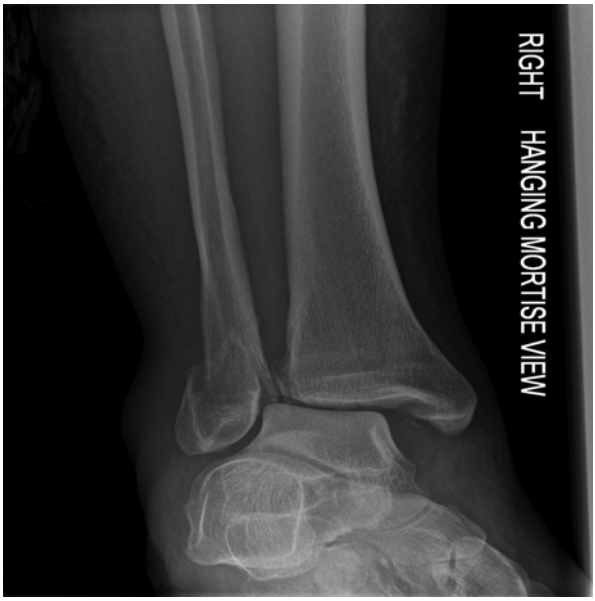


Fig. 3.7 Bi-malleolar fracture of the right ankle

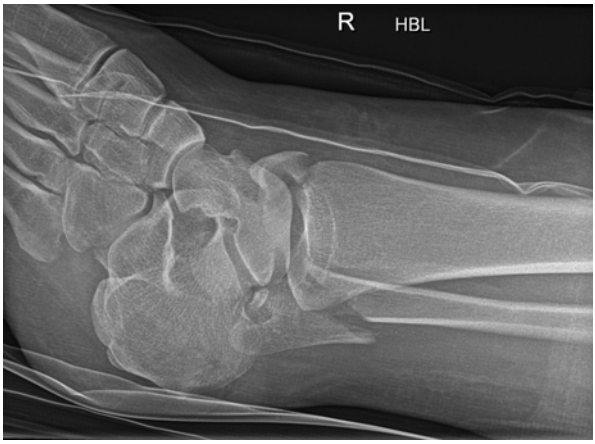


Fig. 3.8 Tri-malleolar fracture of the right ankle

Table 3.2 Indicators for radiological investigation in ankle injury

Tenderness over the distal 6 cm of the posterior or tip of the lateral malleolus
Tenderness over the distal 6 cm of the posterior or tip of the medial malleolus
Inability to bear weight immediately after the accident or in the emergency department

Adapted from Stiell et al. [12]

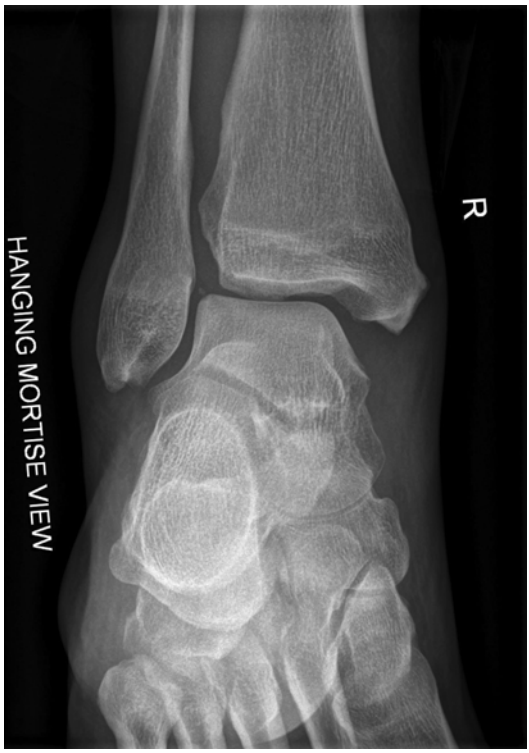


Fig. 3.9 Interruption of the interosseous membrane

Treatment

Stability of an ankle fracture must be assessed to ensure appropriate management is undertaken. Fractures of the fibula with either a severe injury to the contralateral ligament or medial malleolus are inherently unstable. These fractures are likely to benefit from surgical fixation. Figure 3.9 shows interruption of the interosseous membrane but no visible fracture, causing instability of the ankle.

Table 3.3 Ankle fracture patterns likely to require operative fixation

Fracture dislocations
Fractures with significant talar shift
Bi-malleolar and tri-malleolar fractures
Lateral malleolar fractures with ligamentous injury medially

Conservative management of reduction and stabilisation with plaster of Paris below-knee 'back slab' will improve healing and allow better pain control to be achieved. This is possible only for those fractures that are minimally displaced and considered stable. Fracture patterns that are likely to require operative fixation are listed below [13]. All ankle fractures even those that are discharged from the emergency department require early orthopaedic review for definitive management planning (Table 3.3).

Differential Diagnosis

- Ankle ligament injury (these can result in long-term pain and reduced mobility for the patient and should be treated with appropriate care; the lateral ligaments are especially at risk from inversion injuries)

Achilles Tendon Rupture

Pathophysiology

Overflexion of the ankle joint producing sudden stressed extension of the Achilles tendon can result in partial or complete tears to the Achilles tendon.

Clinical Features

- Sudden-onset posterior calf or ankle pain with associate inability to mobilise; patients may describe a snapping sensation.
- Step or loss of continuity of the tendon itself with associated tenderness.
- Complete rupture may result in swelling or bunching in the calf.
- The Simmonds-Thompson test, where plantar flexion is elicited by compression of the calf, may show reduced or lost range of movement when compared with the opposite side [14].

Treatment

Initial management should be analgesia and patient being placed in an equinus cast so that the foot is held in plantar flexion. All patients with diagnosed or suspected Achilles tendon injury should be reviewed at the earliest opportunity by an orthopaedic specialist for consideration of surgical intervention [15].

The Foot

Anatomy

There are multiple bones and ligaments in the foot that allow it to absorb significant loading forces during weight bearing exercise. The tarsal, metatarsal and phalanx bones are grouped into hindfoot, midfoot and forefoot for descriptive purposes. A working knowledge of the bony and ligamentous anatomy will improve clinical examination and diagnosis.

Hind Foot: Calcaneal Fractures

Pathophysiology

The calcaneum forms the heel of the foot and is often injured following falls when the injured party lands on their feet.

Clinical Features

- Tenderness and swelling over the calcaneum following fall onto feet.
- Calcaneal injury should raise concern regarding injury at any level above this especially to the acetabulum, pelvis and spine.

Imaging

- Specific radiographs for calcaneal fracture request both lateral and axial views (Fig. 3.10).
- The extent of any injury can often be significantly greater than indicated by the radiograph.
- Low-energy mechanisms can result in radiological findings that can be missed.

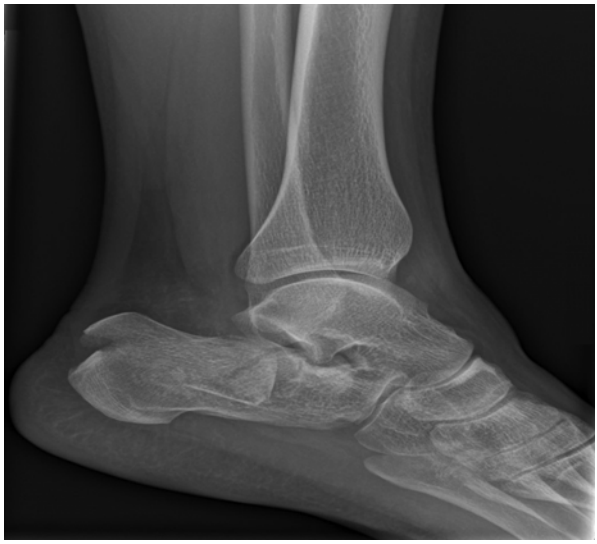


Fig. 3.10 Fracture of the left calcaneum

Table 3.4 Indicators for radiological investigation of foot fractures

Indicators for radiological investigation of foot fractures
Bony tenderness over the base of the 5th metatarsal
Bony tenderness over the area of the navicular
Inability to bear weight at the time of the injury or in the emergency department

- Look for flattening of Bohler's angle, which indicates displacement of the posterior area of the bone [16].
- Gold standard imaging modality is CT [17].

Treatment

Management should include an early orthopaedic opinion because management decisions regarding operative intervention need to be made.

Midfoot and Forefoot Fractures

Investigations

Stiell et al. determined that foot x-rays should only be ordered if there is any midfoot pain and any of the features seen in the table below [18]. Toe phalanx fracture and dislocation should be determined by inspection and palpation and may not adhere to the foot rules for x-ray (Table 3.4).

Fifth Metatarsal Fractures

Pathophysiology

Avulsion fractures are by far the most common of all fifth metatarsal fractures. These occur when there is inversion of the ankle and are commonly associated with lateral malleolar fractures. The bony attachment of peroneus brevis is avulsed as the peroneus muscles attempt to correct inversion. It is very often missed as focus is drawn by pain and swelling to the ankle.

Investigations

The x-ray may be difficult to interpret due to normal variants mimicking fractures; however, the fracture line will normally run at right angles to the bone's long axis.

Treatment

Management of avulsion fractures is below-knee plaster of Paris or walking boot.

Prognosis

Recovery will be 5–7 weeks. If significantly displaced or accompanied by other injury, then orthopaedic consultation and operative fixation may be considered.

The Jones fracture of the fifth metatarsal is often more distal on the bone and likely to be a stress-type fracture. Careful management is required due to significant chance of non-union [19].

Phalanx Fractures

If phalanx fractures have minimal displacement or angulation, no treatment is required, and buddy strapping to adjacent toe may improve pain. Significant displacement may benefit from reduction. Ensure no evidence of open fracture is present.

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Chapter 4
Spinal Injury

Michael Trauer, Jignesh Tailor, and Shumontha Dev

Key Points
- Early rapid assessment and initial appropriate imaging are essential in spinal injury.
- Cervical spine immobilisation is important to prevent further damage.
- SCIWORA is a more likely diagnosis in children under 8 years of age.

Adult Cervical Spine Injury

Cervical spine (C-spine) injuries may result in a serious long-term disability, and thus their management in the acute situation is both incredibly important and subject to litigation:

- Good multidisciplinary approach to patients with cervical spine injuries.
- Understanding of the indications.
- Familiarisation with the guidelines that are available for imaging in C-spine injuries is essential within a busy emergency department.

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It should be established early:

- Whether the patient's mental status is normal.
- Alcohol, drugs, head injury and shock can all alter the pain perception, and therefore, injury should be assumed in these types of patients.

Hence, a proper history is A MUST as shown in table below.

Key Points
A: altered mental state – check whether drugs or alcohol is on board.
M: mechanism – is there potential for injury?
U: underlying conditions – are there high-risk factors for fractures?
S: symptoms – is there pain or paraesthesia?
T: timing – when did symptoms start?

Who to Image?

Senior emergency doctors should be familiar with current local guidelines for C-spine imaging so that patients can be swiftly assessed and any unnecessary, uncomfortable and potentially harmful immobilisation removed. At the time of writing, the UK guidelines as published by NICE in 2014 (based on the NEXUS and Canadian rules) can be summarised as follows [1].

CT imaging of the spine should be performed on the following 'high-risk' groups [2]:

- GCS <13 or intubated (go straight to CT)
- Multiregion trauma or head injury requiring CT imaging or surgery
- Focal peripheral neurological deficit or paraesthesia in the upper or lower limbs (know your dermatomes and myotomes)
- Dangerous mechanism or >65 years old + any neck pain and visible supraclavicular or painful thoracic injury

Key Points
Dangerous mechanisms include:

- Fall from a height of >1 m or five stairs
- Axial load to the head, for example, diving
- High-speed motor vehicle collision
- Rollover motor accident
- Ejection from a motor vehicle
- Accident involving motorised recreational vehicles
- Bicycle collision

Furthermore, CT imaging of the cervical spine should be considered:

- If plain film imaging is inadequate
- If suspicion or certainty of abnormality of plain film
- In patients with dementia
- If new neurological signs or symptoms
- If severe neck pain (>7/10 severity)
- If significantly reduced range of neck movement
- In patients with known vertebral disease

CT should cover the area from cranio-cervical junction to the thoraco-cervical junction.

If none of the above 'high-risk' features are present, at least one of the following 'low-risk' features must be present for the clinician to be able to attempt to clear the neck clinically as follows [3, 4]:

- Simple rear end
- Ambulatory at any time since accident
- Sitting up in ED
- Delayed onset neck pain
- No midline cervical spine tenderness

Patients that are unable to rotate their neck 45° in both directions or report severe pain (>7/10 severity) on doing so should have cervical spine imaging performed.

It is important to note that the use of the clinical decision rule may lead to patients being discharged without imaging with the following 'insignificant' cervical spine injuries:

- Isolated spinous process fracture not involving the lamina
- Isolated osteophyte fracture (not corner or teardrop fracture)
- Isolated transverse process fracture not involving the facet joint
- Simple vertebral compression fracture (<25 % loss of height)

Key Points
Modified Canadian cervical spine rule [3]:

- GCS <15 on assessment in the ED
- Paralysis, focal neurological deficit or paraesthesia in the extremities
- Patients with abnormal vital signs (systolic BP <90 mmHg or respiratory rate outside of the range 10–24 breaths per minute)
- Urgent requirement to identify a cervical spine fracture
- Severe neck pain (>7/10 severity)
- Patients with a dangerous mechanism of injury and either a visible injury above the clavicles or a severely painful thoracic injury even if there is no neck pain or tenderness

- Patients with neck pain and any of the following high-risk factors:
- Fall from >1 m or five stairs
- An axial load to the head, e.g. diving
- High-speed motor vehicle collision
- Rollover motor vehicle accident
- Ejection from a motor vehicle
- Accident involving motorised recreational vehicles
- Bicycle collision
- Age 65 years or more
- Injured more than 48 h earlier
- Reattending with the same injury
- Known vertebral disease (RA, ankylosing spondylitis, spinal stenosis)

The above indications for imaging only apply to adults who are not intoxicated and do not have significant distracting injuries.

If no high-risk and at least one low-risk features are present, remove the collar and test movements:

- If the patient is unable to flex laterally 45° left and right or this causes severe pain or upper limb paraesthesia, then re-immobilise and request three-view cervical spine x-rays (AP, lateral C-spine and odontoid peg views).
- If the x-rays are inadequate, suspicious or definitely abnormal, then perform CT imaging of the C-spine.

How to Read C-Spine X-Rays

It is important for the emergency physician to be confident in clearing C-spine x-rays (Figs. 4.1, 4.2 and 4.3) as a report may not be forthcoming and immobilisation needs to be removed as soon as possible. A systematic approach will avoid missing abnormalities, and a commonly used system is the ABCD system:

- *A* – adequacy and alignment
- *B* – bones
- *C* – cartilage
- *D* – dense soft tissues

In respect to adequacy, the C-spine should be visualised from the atlanto-occipital joint to the junction of C7 and T1. A swimmer's view may be necessary to visualise the lower cervical vertebrae.

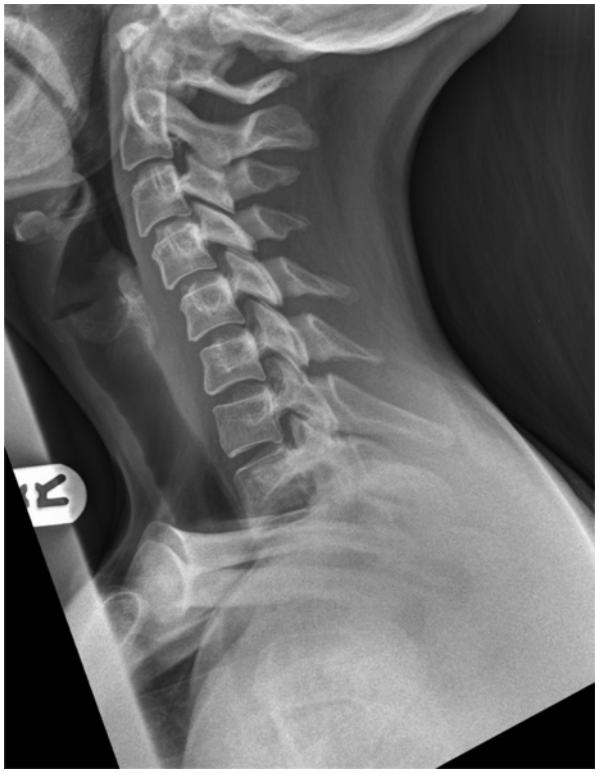

Fig. 4.1 Lateral view of normal C-spine

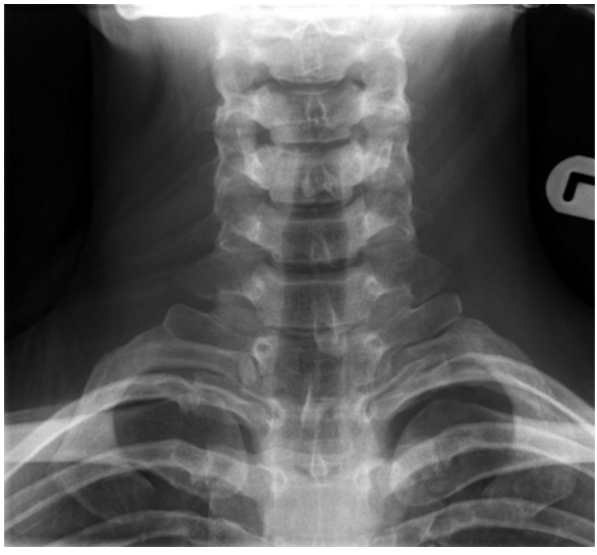

Fig. 4.2 AP view of normal C-spine

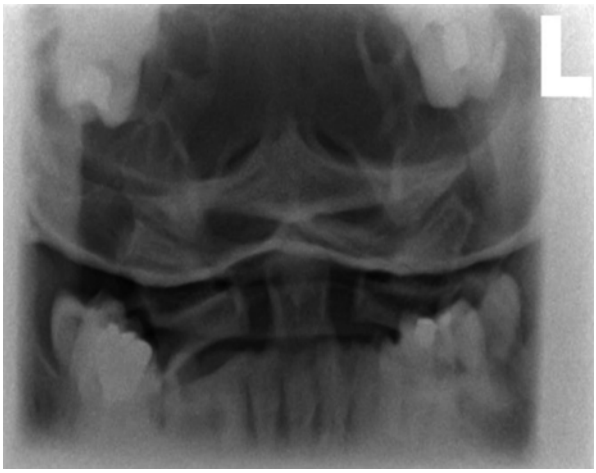

Fig. 4.3 Odontoid (open-mouth) peg view of normal C-spine

For assessment of alignment, four longitudinal lines along the lateral view should be traced:

- Anterior vertebral line (anterior longitudinal ligament)
- Posterior vertebral line (posterior longitudinal ligament (PLL), anterior border spinal canal)
- Spino-laminar line (posterior border spinal canal)
- Spinous process line (tips of the spinous processes)

Also, three longitudinal lines along the AP view should be traced:

- Spinous process line (should be equally spaced)
- Foraminal lines (asymmetry could indicate uni- or bi-facet dislocation)

On the peg view:

- Borders of the lateral masses of C1 and C2 should be aligned.
- If the overhang of C1 on C2 is >7 mm, the transverse atlantal ligament (TAL) may be disrupted. This is also known as the 'rule of Spence' for assessing transverse ligament integrity.

On assessment of the bones:

- Outline of each vertebra should be traced, noting any discontinuities or irregularities.
- Any subtle avulsion fractures should be looked for along the anterior longitudinal ligament on lateral view.

In respect of the cartilages:

- Each disc from C2 to C3 caudally should be uniformly spaced.
- Distance from the anterior dens to the posterior border of the anterior arch of C1 should be <3 mm (may be a sign of anterior atlanto-axial subluxation associated with TAL disruption).

The following limits apply for the dense soft tissues:

- C1–C3: <7 mm or 30 % vertebral body width
- C4–C7: <22 mm or 100 % vertebral body width

Specific Fracture Patterns

The fractures of the C1 vertebra can be classified as follows:

- *Type I*: involving a single arch
- *Type II*: burst fracture (the classic Jefferson fracture)
- *Type III*: lateral mass fractures

Sir Geoffrey Jefferson, a British neurosurgeon, originally described the Jefferson fracture in 1920. It was classically described as a four-point (burst) fracture of C1. However, the term is now more commonly used to describe the more common three- or two-point fractures. It typically occurs after an axial load to the head. Although it is an unstable fracture, there tends to be normal neurology as the spinal canal is wide at the upper cervical spine.

Odontoid peg fractures can be categorised as follows [5]:

- Type 1: tip of peg – this is rare and usually not an isolated fracture and may be manifestation of atlanto-occipital dislocation.
- Type 2: base of peg – unstable and often require operative fixation.
- Type 3: extending into the body of C2 – stable

A *hangman's fracture* (Figs. 4.4 and 4.5):

- Is a bilateral fracture through the pars interarticularis of C2, with traumatic subluxation of C2 on C3.
- Typically occurs from hyperextension of the neck (e.g. the forehead hitting a windscreen).
- Levine classification (modified from Effendi et al.) is used to grade adult hangman's fractures based on the degree of subluxation (and whether angulation) of C2 and C3.

There are broadly three types as follows:

- *Type I*: ≤3 mm subluxation – stable and fracture lines may not be seen (IA).
- *Type II*: >3 mm subluxation or ≤3 mm with >15° angulation (IIA, rare) – signifies disruption of the C2/C3 disc and PLL and, therefore, unstable injury.
- *Type III*: subluxation of C2/C3 facets – signifies bilateral facet capsule disruption.

As with Jefferson fractures, the neurological examination may be normal in patients with hangman's fractures as the spinal canal is wide at this point.

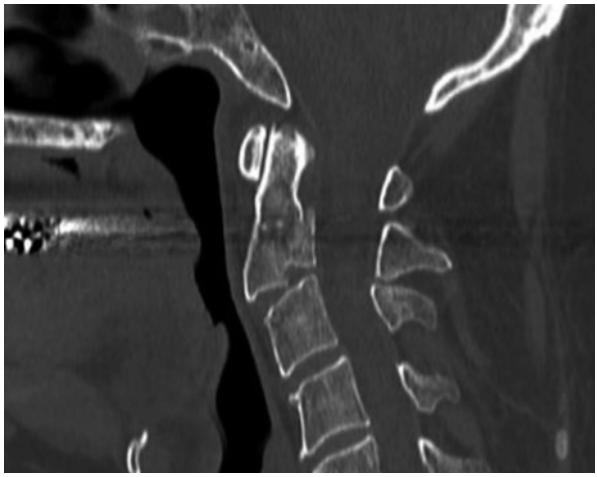

Fig. 4.4 CT scan lateral view showing hangman's fracture

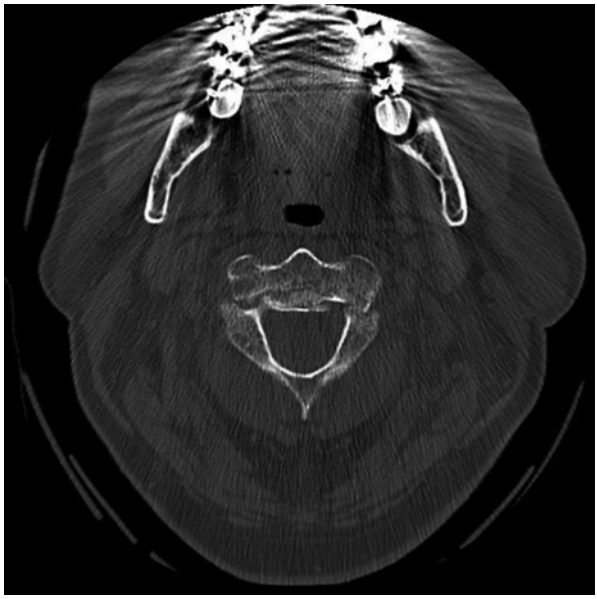

Fig. 4.5 CT scan AP view showing hangman's fracture

The nature of *subaxial (C3–C7) fractures* is usually determined by the mechanism of injury and the position of the neck at the time of injury [6]:

- Flexion
  - Acting alone: look for unilateral/bilateral facet dislocation.
  - With compression: look for anterior vertebral body fractures.
  - With distraction: look for torn PLL + dislocated/locked facets.

- Extension
  - Acting alone: look for spinous process fractures.
  - With compression: look for fractures in lateral mass or facet.
  - With distraction: look for torn ALL + retrolisthesis of superior vertebrae on inferior one.
- Neutral position
  - With compression: look for burst fracture.
  - With distraction: associated with complete ligamentous disruption (usually unstable).

In the lower cervical spine, a *burst fracture*:

- Involves both the anterior and posterior end plates of the vertebral body
- Usually occurs following vertical compressive forces on the C-spine

Remember that around 20 % of patients with major spine injury will have a second spinal injury at another level, which may be noncontiguous. Therefore, consider imaging the rest of the spine with AP/lateral x-rays or CT if a C-spine injury is found [7].

Spinal Cord Injury

As explained above, some injuries may produce fractures of the spinal column, but may not injure the spinal cord (particularly those at the C1/C2 level). Injuries to the spinal cord are classified as [11]:

- *Complete* – no motor or sensory function is preserved in S4–S5 segments (no voluntary anal contraction, anal sensation or sensation in S4–S5 dermatome).
- *Incomplete* – motor or sensory function is preserved below the neurological level.

Clinically, incomplete lesions may produce one of the following syndromes, depending on the location of the injury in the spinal cord.

Central cord syndrome (injury to the central part of the cord) is the most common type of incomplete spinal cord injury:

- Usually from hyperextension injury in an older person with osteoarthritis of the spine
- Disproportionately greater motor deficit in the upper extremities (centrally located arm tracts) than the lower
- Sensation variably spared

Brown-Sequard syndrome (hemi-section of the cord):

- Ipsilateral motor paralysis below the lesion (due to corticospinal tract lesion) and posterior column function (proprioception and vibratory senses), with contralateral loss of pain and temperature sensation (due to spinothalamic tract lesion).
- Prognosis for recovery is good.

Anterior cord syndrome (cord infarction in the territory of the anterior spinal artery):

- Quadriplegic (lesion above C7), with dissociated sensory loss below the lesion – loss of pain and temperature sensation bilaterally, with preserved proprioception, two-point discrimination and vibration sense (posterior column function)

Posterior cord syndrome (involves the dorsal column of the spinal cord):

- Loss of proprioceptive vibratory sense with sensory and motor functioning spared
- A rare syndrome

The emergency doctor should be aware of these clinical syndromes and act promptly to protect the spine with immobilisation, arrange appropriate imaging and discuss with neurosurgical colleagues at an earliest opportunity.

Indications for MRI Cervical Spine

MRI scan should be used to exclude cervical spine injury in adults following blunt trauma if any of the following criteria are met:

- Neurological signs and symptoms referable to the cervical spine
- Suspicion of vertebral artery injury (e.g. spinal column displacement, foramen transversarium or lateral process fracture, posterior circulation syndromes)

MRI scan should also be used to exclude cervical spine injury in adults with severely restricted neck movement or severe pain ($\geq$7/10) despite a normal CT scan.

Paediatric C-Spine Injury

There is evidence that children are less likely than adults to suffer neurological injury with cervical spine trauma, although when neurological injury does ensue it often occurs with fracture-dislocations. Neurological sequelae can result from facet dislocations, with bilateral dislocated facets generally affecting the spinal cord.

Several differences between children and adults affect patterns of neck injury as children [7]:

- Have large heads relative to their bodies and the fulcrum around which flexion occurs is higher in the neck (C2–C3 as opposed to C5–C6 in adults).
- Are prone to higher neck injuries than older children and adults (particularly under the age of 8).
- Have neck muscles and ligaments that are underdeveloped and lax.
- Have facet joints in a more horizontal plane, predisposing them to anteroposterior dislocation.
- Have vertebral bodies tapered anteriorly, giving a wedged appearance.
- Have tip of the odontoid peg that ossifies between ages 3 and 6 and fuses at age 12, and the cartilaginous synchondrosis at the junction of the peg and body of C2 is visible up to age 11.

None of these normal variants should be mistaken for a fracture.

Key Points
Anatomical considerations:

- Large head and higher fulcrum
- Weak musculature and greater compliance
- Facet joints more horizontal
- Anterior 'wedged' appearance
- Normal odontoid cartilage

Due to the greater carcinogenic potential of radiation in children, there is generally a higher threshold for imaging:

- If the child falls into a 'high-risk' group (as in adults), CT imaging of the C-spine is advised.
- Children who do not present with high-risk features but who have sustained a head injury and have neck pain usually have C-spine x-rays if there was a dangerous mechanism of action or if the neck range of movement cannot be assessed safely (see algorithm children).
- For children over 10 years of age, treat as adults.
- For children under the age of 10, the open-mouth peg view should be omitted and only perform CT if the GCS is <8 or there is focal neurology.
- Limited CT should be considered or perform an MRI scan if available.

Children under 8 years old are at a higher risk of SCIWORA (spinal cord injury without radiographic abnormalities). In children with this disorder:

- MRI scan can show ligamentous or disc injury, complete spinal cord transection and spinal cord haemorrhage.

- It is important to understand that, if one injury is found, it is necessary to consider imaging the whole spine to look for other injuries.
- Generally, it is considered that injuries associated with SCIWORA are stable lesions, and immobilisation for up to a period of 3 months may be required.

Thoracolumbar Injuries

Thoracic fracture-dislocations are uncommon [8]:

- Often associated with severe cord injury as a result of the narrow canal and the 'watershed' area for blood supply.
- Majority of injuries occur at T10–L2 levels, at the junction of thoracic kyphosis and lumbar lordosis.
- Anterior wedging of the vertebral body in the anterior column results from a compression fracture.
- The middle and posterior columns are not involved.
- Compression fractures are stable and do not cause neurological deficit.
- Patients may be treated with a hyperextension brace.
- Wedge fractures of the thoracolumbar regions can be potentially unstable if >50 % loss of anterior height, and these will require open reduction and internal fixation.

A 'burst' fracture of the thoracolumbar region:

- Associated with loss of anterior and posterior height.
- Involves the anterior and middle columns, with fracture fragments displaced into the neural canal.
- Often, there is mechanical stability, but these injuries can still cause spinal cord damage.
- As a result of retropulsion of bone into the canal, burst fractures may require laminectomy and posterior stabilisation.

A flexion-distraction injury, also known as a 'Chance' fracture of the thoracolumbar area:

- Is associated with 'lap' seatbelts, with complete failure of bony structures.
- Involves the anterior, middle and posterior columns of the posterior ligaments and may occur through the bone or through the soft tissue.
- Injuries that occur through the bone may be treated with a hyperextension cast.
- Ligamentous disruption may need posterior spinal arthrodesis with compression system.

Disruptions of all three columns can result from a combination of anterior compression, with distraction and rotation. It is associated with significant instability, and neurological deficit, and can include dural tears and intra-abdominal injuries.

With dislocations:

- All three columns are disrupted, causing instability due to multiple forces, including rotation, compression and sheer forces.
- Due to the instability, there is associated neurological deficit with dislocations.
- Treatment may consist of stabilisation of the spine to allow early mobilisation.

Hyperextension of the lumbar spine:

- Causes an anterior vertebral body avulsion fracture, with fractures of the posterior columns, including spinous process, lamina and possibly pedicles.
- Fractures are stable.

A spine is defined as being unstable if it is not able to bear weight without deforming the anatomic structure or causing pain [9–11]. The stability of the bony spine can be thought of in terms of a three-column concept:

- Spinal structure can be divided into three columns, from anterior to posterior.
- Anterior half of the vertebral body and anterior longitudinal ligament comprise the anterior column.
- Posterior half of the vertebral body and posterior longitudinal ligament comprise the middle column.
- The third column is comprised of the posterior elements including the pedicles and facets.

Another way of thinking about the three columns is with the vertebral body and disc forming one column and each of the two facets posterolaterally being a separate column.

Key Points
Classification of thoracolumbar spine fractures:

- Compression
- Burst fracture
- Flexion-distraction injuries
- Extension injuries
- Dislocations

Other possible fractures that can occur:

- Transverse process fracture: from blunt trauma, commonly L5
- Spinous process avulsions: from distractions, but stable injuries
- Facet fractures: uncommon, but may occur in patients with prior laminectomies or stress fractures

Emergency Department Management of Spinal Injuries

The important principles of ATLS (Advanced Trauma Life Support) [11] should be followed in managing any patients that may have a suspected spinal injury:

- Patient should be immobilised as much as possible to prevent secondary spinal injuries.
- Cervical spinal immobilisation would include a firm collar, sand bags or 'blocks', with tapes.
- Although spinal boards may be used to transport patients from the scene of the incident to hospital, a patient should be removed from the spinal board as soon as it is possible.
- Followed by imaging as discussed earlier.

In those patients that may have sustained significant spinal cord injuries, *spinal shock* is a possibility:

- Occurs within the first 24 h in a patient with spinal cord injury, with complete lack of deep tendon reflexes and anal reflexes
- Usually reverses itself within a day

Neurogenic Shock

- Occurs as a result of loss of sympathetic function, manifesting profound hypotension from significant cervical or high thoracic spinal cord injury.
- Occasionally, there is bradycardia due to unopposed vagal activity.
- Can be a devastating complication, leading to organ dysfunction and death, if it is not promptly recognised and treated.
- After the initial treatment of shock, e.g. due to haemorrhage, it may be necessary to give pressor agents to achieve haemodynamic stability.

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Chapter 5
Upper Limb Disorders

Shumontha Dev and Savvas Papasavvas

Key Points
- Always perform two-view plain imaging to exclude fractures or dislocations.
- Pain relief, reduction and immobilisation of the affected part are essential in managing fractures or dislocations of upper limb injuries.

Introduction

The upper extremity or upper limb extends from the deltoid region to the hand. It consists of four major components: a girdle formed by the clavicle and scapulae, the arm, the forearm and the hand. The upper limb is supported by muscles connected to the ribs and vertebrae. The focus of this chapter is the management of injuries.

Initial Assessment of Upper Limb

The initial assessment of the upper limb should follow the look, feel, movement format. It cannot be overemphasised that as will any trauma the principles of ATLS (Advanced Trauma Life Support) take priority before concentrating on the specific limb assessment and management.

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Clavicle Injuries

Being the most proximal bone of the upper limb, the clavicle provides the leverage and support required for the shoulder girdle components. The clavicle should be routinely examined as part of assessment of the shoulder joint. A fracture may occur after a fall onto the outstretched hand (FOOSH). It is most common at the junction of the medial two-thirds and the lateral one-third of the clavicle. Characteristically, a fracture of the clavicle leads to an inferior displacement of the distal component (Fig. 5.1), as it is weighed down by the whole arm [2].

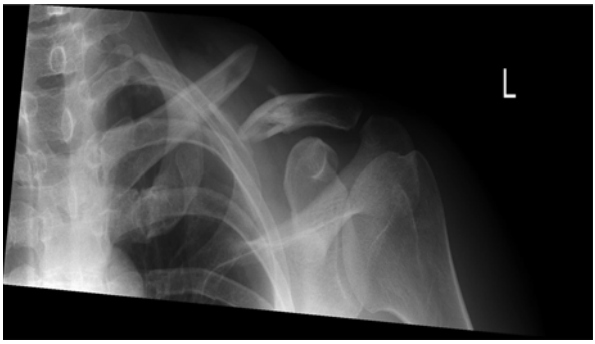
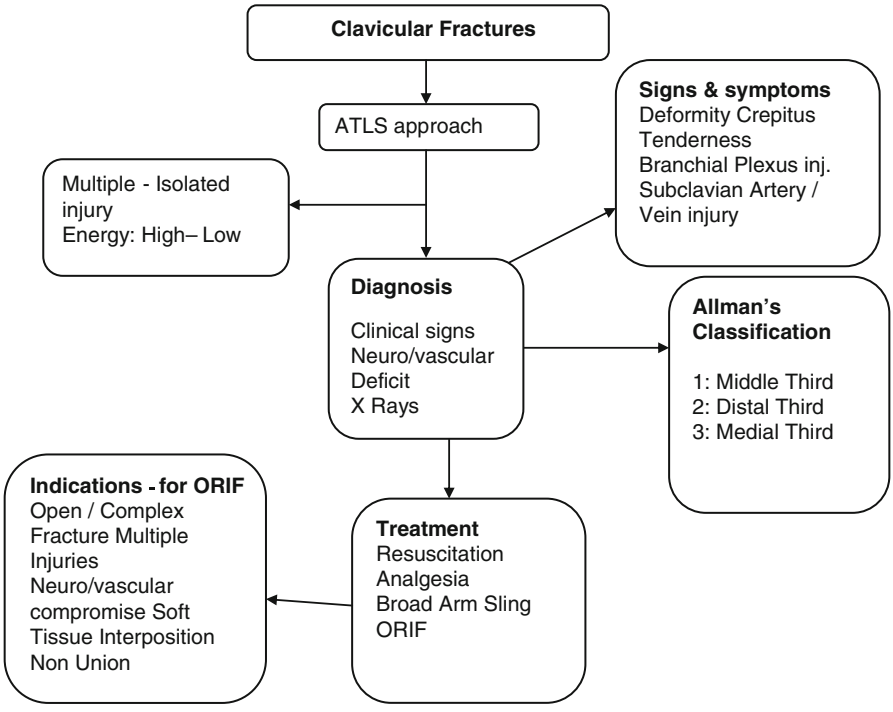


Fig. 5.1 Old mid-clavicular fracture with inferior displacement of the distal end



Acromioclavicular Joint

The acromioclavicular (AC) joint [1] is the junction between the acromion (a section of the scapula that forms the highest point of the shoulder) and the clavicle.

The acromioclavicular joint should be assessed as part of the examination and with standard shoulder X-rays. A common injury to the ACJ is dislocation, often known as shoulder separation (Fig. 5.2). ACJ dislocations commonly arise as a result of collision sports. The most common mechanism of injury is a fall on the tip of the shoulder or FOOSH.

A loss of alignment of the inferior surfaces of the clavicle and acromion indicates disruption of the acromioclavicular ligaments at the ACJ. An additional disruption of the coracoacromial ligament results in separation of the entire scapula from the clavicle.

Although assessment should be with plain radiography, a minor ligamentous disruption may not be detectable as alignment is not lost.

Key Points

- A disruption of the acromioclavicular ligaments results in loss of alignment of the clavicle and acromion inferior surfaces.
- An additional disruption of the coracoacromial ligament results in separation of the entire scapula from the clavicle.
- A low-grade ligament injury may not be visible on a plain X-ray.

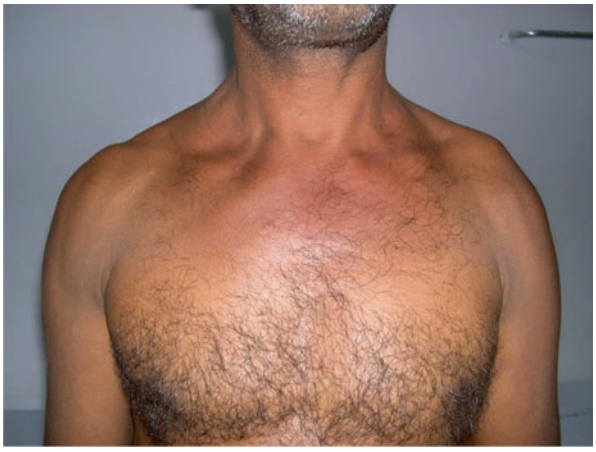
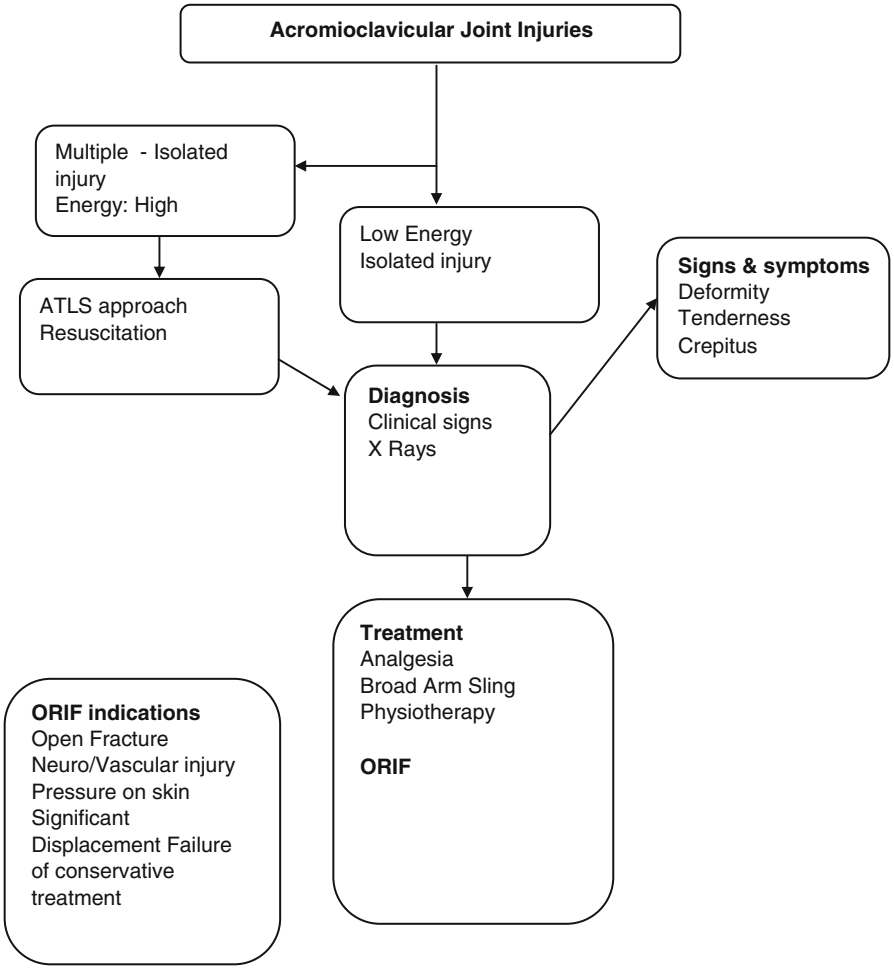


Fig. 5.2 Left AC joint disruption (Courtesy: Prof. SS David, Pushpagiri Medical College Hospital, India)



Shoulder Joint

The shoulder is made up of three bones, the clavicle (collarbone), the scapula (shoulder blade), and the humerus (upper arm bone), with associated muscles, ligaments and tendons. The shoulder joint is the articulation between the bones of the shoulder and typically refers to the glenohumeral joint. The shoulder joint needs to be sufficiently mobile for the wide range actions of the arms and hands. Furthermore, it needs to be stable enough to allow for actions such as lifting, pushing and pulling. The shoulder examination, along with all other joint examinations, follows the general pattern of 'look, feel, move' with additional special tests.

Three special tests, the impingement, apprehension and the scarf test, can be performed on the shoulder:

- Impingement test: performed by placing the shoulder out at 90° with the arm hanging down, pressing back on the arm and checking for any pain.
- Apprehension test: similarly performed but with the arm faced upwards and pushed back on the arm. The patient may be apprehensive about the movement as the joint feels 'unstable'.
- Scarf test: performed with the elbow flexed to 90°, with the patient's hand placed on their opposite shoulder and pushed back. Again look for any discomfort.

In the context of trauma to the shoulder joint, there are two standard plain radiological views essential to assess the joint. These views are the anterior-posterior (AP) view and the lateral or 'Y-view'. Also, an 'axial' view (Fig. 5.3) is an alternative to the Y-view if the patient is able to tolerate holding the arm in abduction.

Key Points

- Standard views in the context of trauma are AP and Y-views.
- Anterior dislocation is much more common than posterior dislocation.
- Anterior dislocation results in the humeral head lying anterior to the glenoid and inferior to the coracoid process.

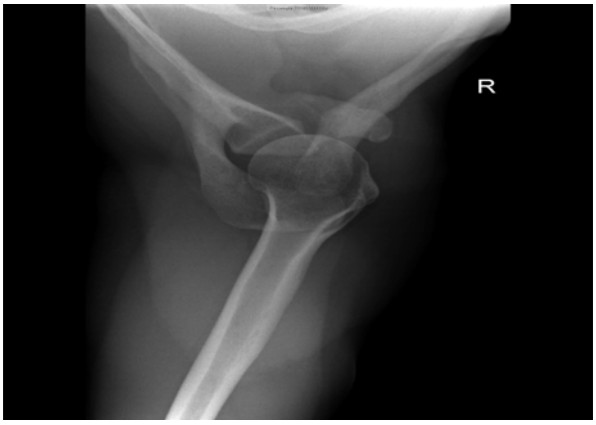


Fig. 5.3 Axillary view of the normal right shoulder

The shoulder is the most commonly dislocated joint in the body [3]. Most shoulder dislocations occur anteriorly (Figs. 5.3 and 5.4). However, they may also occur posteriorly, inferiorly or anterior-superiorly. Usually, anterior dislocations are associated with trauma with the arm abducted and in external rotation. Posterior dislocations are commonly associated with electric shocks and epileptic seizures. Those patients that have sustained a previous shoulder dislocation are more prone to redislocation. This may be as a consequence of the tissue not healing properly or because the tissue stretches out and becomes more lax. Factors such as age and rotator cuff tears and fractures of the glenoid show a clear correlation to redislocation. Hence, younger patients (those aged 20 years) have a much higher frequency of redislocation than patients over the age of 50.

On examination of a dislocated shoulder, the range of motion (ROM) is poor and the patient is usually in intense pain. In anterior dislocation of the shoulder, the arm is in slight abduction and external rotation, and the prominent humeral head can be felt anteriorly, with the void seen posteriorly. It is important to note that posterior shoulder dislocations can be easily missed as the patient usually keeps the arm in internal rotation and adduction. An orthogonal view radiograph is essential to the normal views in excluding posterior dislocation. On occasions, it may be necessary to perform CT scan [4] (Fig. 5.5).

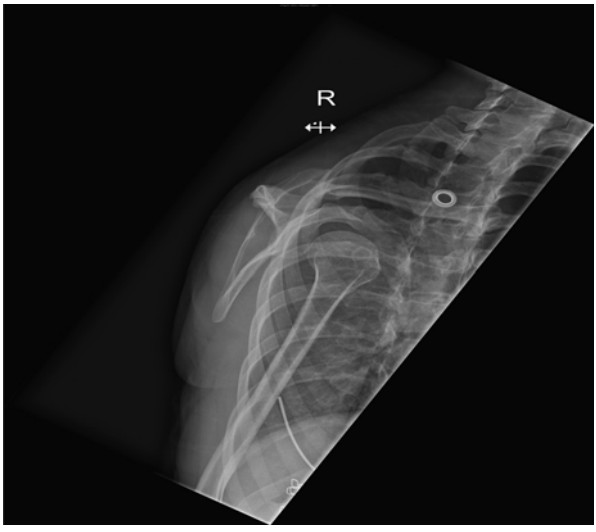


Fig. 5.4 Anterior dislocation of the right shoulder

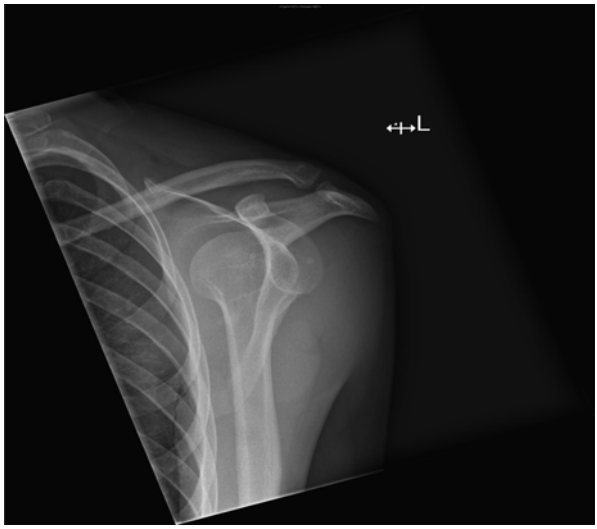
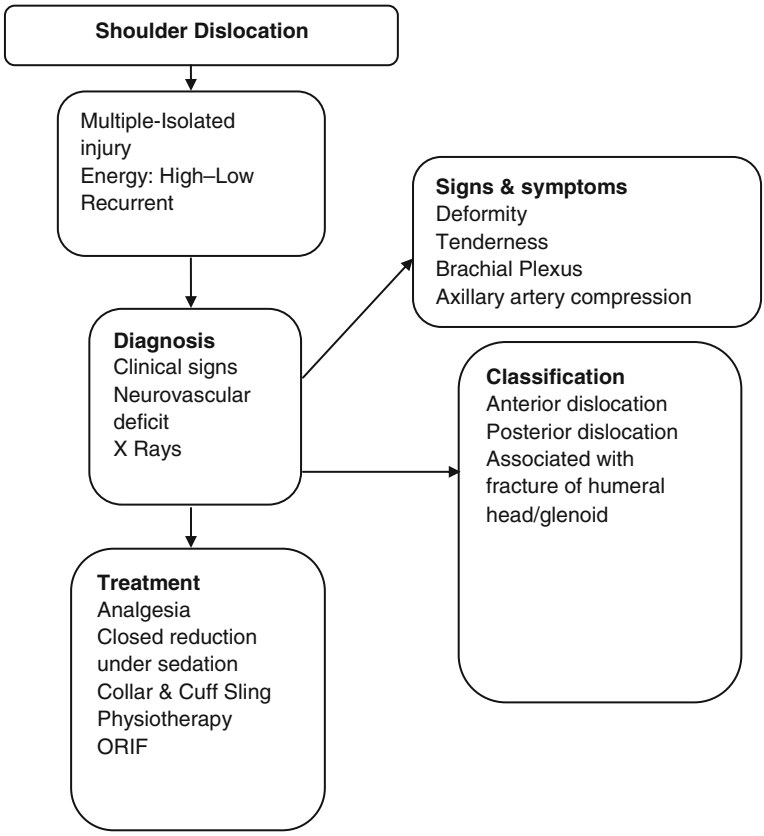


Fig. 5.5 Anterior dislocation of the left shoulder



Scapula

The scapula provides attachment for:

Scapula fractures are relatively uncommon [5]. As scapula injuries are often found when not suspected clinically, careful attention is needed when looking at the radiographs, otherwise subtle fractures can be missed (Fig. 5.6).

The glenoid cartilage can be injured due to shoulder dislocation. This 'Bankart' lesion is not visible usually on X-rays. However, the fracture is most often seen on an X-ray taken following reduction of a glenohumeral joint dislocation [6].

Key Points

- In the context of trauma, careful attention is required in checking the scapula.

Humerus

Fractures of the humerus are common at the surgical neck, and a fracture line may extend into the humerus head with separation of the tubercles. Commonly, humerus fractures are caused by direct trauma to the arm or shoulder or by axial loading that has been transmitted through the elbow (Fig. 5.7). Majority of proximal humeral fractures can be managed nonoperatively. Rarely, vascular or nerve injuries are associated with proximal humerus fractures, especially radial nerve injury. Therefore, neurovascular

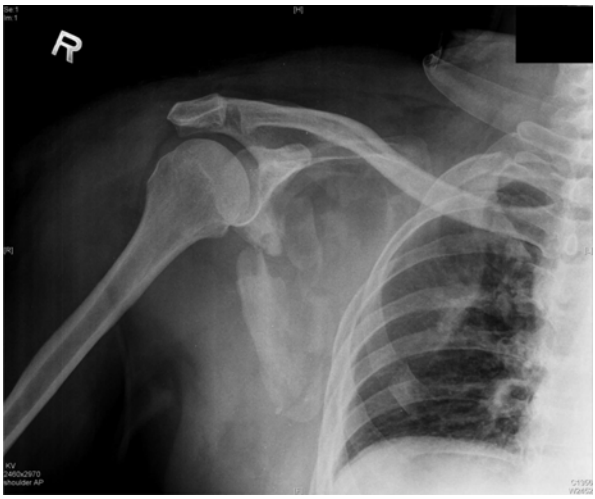


Fig. 5.6 Scapular fracture (Courtesy: Prof. SS David, Pushpagiri Medical College Hospital, India)

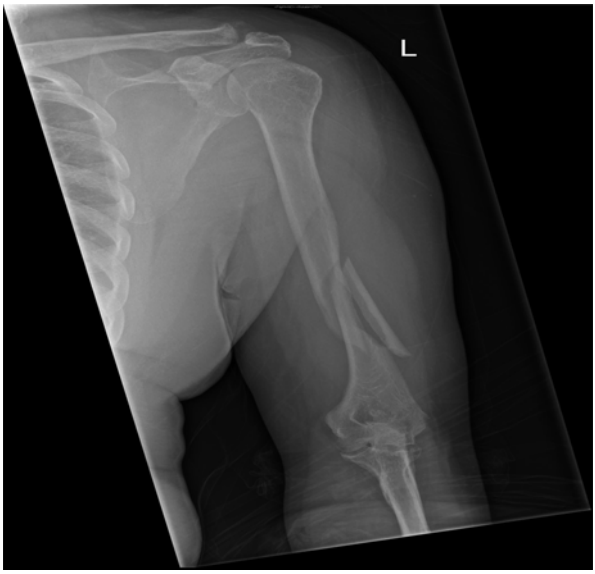


Fig. 5.7 Mid-shaft, comminuted fracture

examination is essential. There is usually pain with palpation or movement of the shoulder or elbow. Also, ecchymosis and oedema may be present.

An anteroposterior and lateral views of the humerus, together with transthoracic and axillary views of the shoulder, should be adequate to visualise a fracture. A CT scan may be required in certain cases.

Minimally displaced fractures are treated conservatively. Often, distal comminuted fractures require surgical management due to damage of the vasculature of the humeral head (Fig. 5.8).

In the emergency department, the important aspect of management of humeral fractures is adequate analgesia and immobilisation with a sling. For humeral shaft fractures, a wrap splint (humeral brace) will stabilise the fracture. As reduction is usually difficult, this is not necessary and a 30–40° angulation is acceptable as the shoulder is able to compensate. However, if the fracture is segmental or the vasculature is compromised, surgical fixation, by either intramedullary nailing or plating, is necessary.

Key Points
- Surgical neck is the commonest site of humerus fracture.

A common complication of proximal shaft fracture is adhesive capsulitis. This can be prevented by early rehabilitation with physiotherapy. Avascular necrosis of

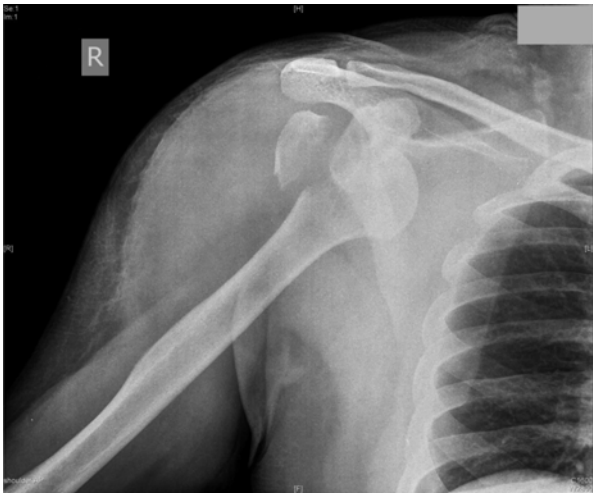


Fig. 5.8 Comminuted fracture humeral head

the humeral head is a realistic possibility. With humeral shaft fractures, a likely complication is radial nerve injury, mainly neurapraxia that can take several months to resolve.

Elbow

The elbow joint is the synovial joint between the humerus in the upper arm and radius and ulna in the forearm. The elbow region consists of the olecranon, the elbow pit and the lateral and medial epicondyles. Two main compartments of the elbow are the anterior and posterior compartments:

- Brachial artery and the ulnar and median nerves are within the anterior compartment. Commonly, this compartment is affected by dislocations and can be a clinical concern in regard to brachial artery disruption and median or ulnar nerve entrapment. When a dislocation occurs, the close proximity of the ulnar nerve to the medial epicondyle allows for the increased likelihood of entrapment.
- Radial nerve and triceps brachii muscle are in the posterior compartment.

The centres of ossification become visible from 6 months to 12 years of age and in early adulthood fuse to the humerus, radius or ulna. These are important points to be considered when interpreting X-rays. X-ray appearances of the normal elbow joint are essential for the identification of elbow injuries. Often, elbow injuries have characteristic radiological appearances, which may only be detected by the presence of soft tissue abnormalities.

Key Points
- Elbow injuries have characteristic appearances.
- Soft tissue abnormality is often the only evidence of bone injury.
- An awareness of elbow development is essential when considering paediatric elbow injuries.

On lateral radiograph, there is often a visible triangle of low density lying anterior to the humerus. This is the anterior fat pad which lies within the elbow joint capsule in a normal structure.

Elbow X-ray: Radial Head Fracture
- Commonly adult patient.
- Lateral image shows an anterior fat pad that is raised away from the humerus but does not show a fracture.
- A visible posterior fat pad is *ALWAYS ABNORMAL*.
- An AP image will show a visible radial head fracture.

The radial head may dislocate from the capitulum of the humerus on its own or in combination with dislocation of the ulna from the trochlea. Dislocations of the radial head may be more subtle to identify, but the latter is more straightforward to confirm. The anterior humerus line is a line extending from the anterior edge of the humerus and should pass through the capitulum with at least one-third of the capitulum seen anterior to it. The midline of the radial shaft, the radiocapitellar line, is an important landmark, as it should pass through the middle of the capitulum.

Key Points
- Elbow injuries have characteristic appearances.
- The only evidence of bone injury may often be the soft tissue abnormality.
- In paediatric cases, development of ossification centres is essential when considering elbow injuries.

A joint effusion is indicated if the anterior fat pad is raised away from the humerus or if a posterior fat pad is visible between triceps and the posterior humerus (Fig. 5.9). When the aetiology is trauma, this finding is due to haemarthrosis (blood in the joint) secondary to a bone fracture. Often, this may be the only X-ray sign of a bone injury.

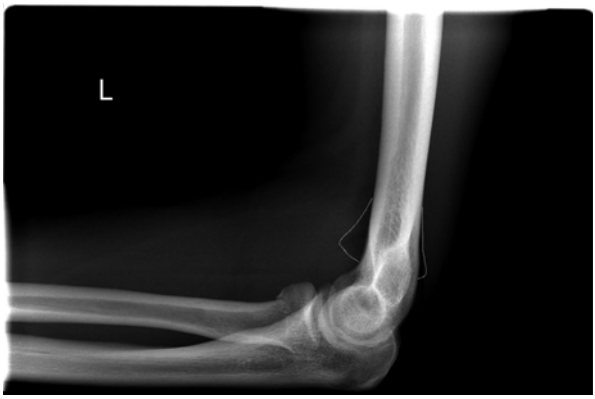


Fig. 5.9 Left elbow joint showing anterior and posterior fat pads

In an adult, a post-traumatic effusion without a visible bone fracture usually indicates a radial head fracture, whereas in a child, it indicates a supracondylar fracture of the distal humerus. An inflammatory cause should be considered if there is a joint effusion but no history of trauma.

Elbow X-ray: Supracondylar Fracture
- A child patient.
- A fracture visible at the distal humerus.
- Raised fat pads away from the humerus suggest joint effusion (haemarthrosis).
- Powerful triceps muscle posteriorly displaces the ulna – taking the capitulum (**C**) with it.
- Capitulum lies behind the anterior humerus line.
- Essential that at least one-third of the capitulum should lie in front of the anterior humerus line.

Elbow Dislocation

Elbow dislocations are not a rare presentation to the ED [7] either due to sport-related injury or high-speed vehicular accidents. The mechanism of injury is usually a fall on an extended or hyperextended elbow.

As with all dislocations, the position of the distal segment in relation to the proximal part defines a dislocated elbow:

- The most common elbow dislocation is posterior or posterolateral (Fig. 5.10). Therefore, the ulna is posterior to the humerus in a posterior elbow dislocation.
- Other types of elbow dislocations, anterior, medial, lateral or divergent, also occur, rarely.
- Radius and ulna dislocate from the humerus in different directions in a divergent dislocation. The strong musculoligamentous complex binding the ulna and radius is completely disrupted.

Clinically, most patients with elbow dislocations are in severe pain.

- Usually swelling of the elbow.
- The affected forearm is held with the opposite hand. The elbow is held in slight flexion, and the forearm appears foreshortened. The olecranon is prominent posteriorly and palpable.
- Vital to check the neurovascular function of the forearm and hand before any attempt at manipulation of the joint. All findings should be clearly documented.
- Sensation of pain and light touch distal to the elbow needs to be checked first.

The median, ulnar and radial nerves need to be tested for muscle power distal to the elbow.

- Median nerve can be tested by asking the patient to touch the thumb with the little finger.
- Interosseous muscle function, innervated by the ulnar nerve, can be tested by asking the patient to spread the fingers apart.
- Radial nerve is attested by asking the patient to extend the thumb, fingers and wrist.
- At the elbow, the brachial artery needs to be palpated.
- At the wrist, both the ulnar and radial arteries need to be felt.

An attempt should be made to reduce elbow dislocations as soon as possible. In the ED, reduction can be performed under procedural sedation, with the patient in the supine position. During reduction, an assistant should apply steady traction to the upper aspect of the humerus, whilst the other person applies gentle traction to the elbow. The elbow is gently extended whilst counterforce is applied to the side of displacement. To accomplish reduction, the elbow is gently flexed and a loud ‘clunk’ is palpated and heard. At times, reduction cannot be established under sedation due to entrapped fragments with the joint space, and open reduction under general anaesthetics is required [8–10].

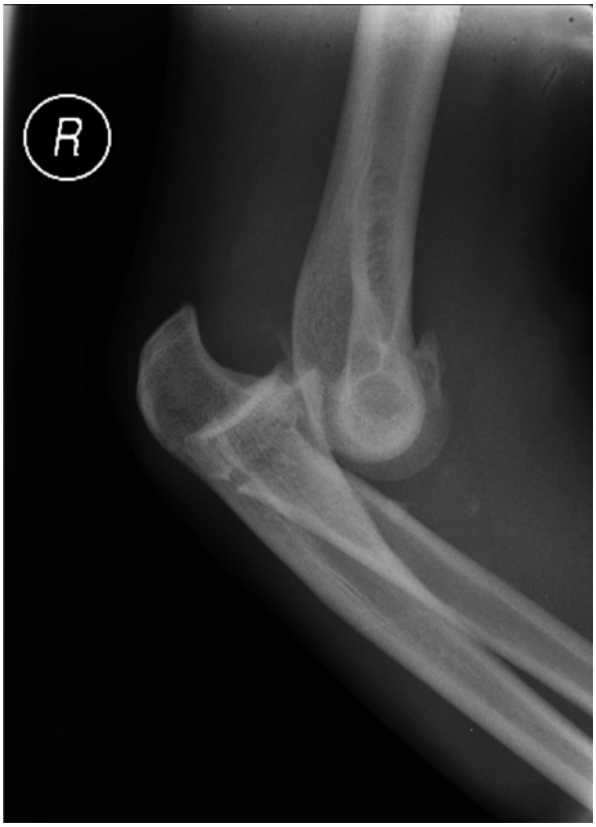


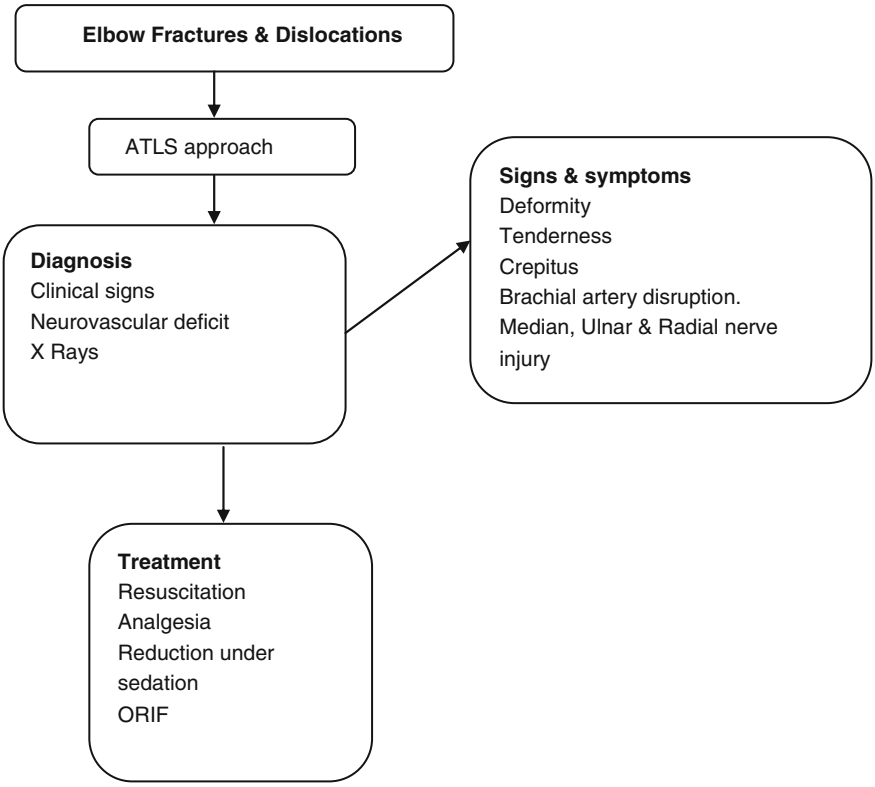
Fig. 5.10 Posterior dislocation of the right elbow

After reduction of elbow dislocations, it is essential to begin early active motions within 1 week. Recurrent dislocation of the elbow is rare. There is a loss of mild terminal extension in all patients that have suffered elbow dislocations.

The Forearm: Radius and Ulna

In forearm injuries, the mainstay of treatment is stable anatomic reduction with preservation of mobility, predominantly through operative intervention. Injury to the forearm includes direct trauma, fall from a height, road traffic accidents and sporting injuries. However, the most likely cause is FOOSH with the forearm pronated.

Assessment of the forearm must include careful neurological (specifically motor and sensory functions) and vascular status, especially in relation to the extent of swelling of the forearm. Compartment syndrome needs to be excluded in a patient with a tense compartment and neurological signs.



Mechanism of the injury and the age of the patient define typical fracture patterns in the forearm bones. Osteoporotic fractures of the distal radius are common in the elderly. However, in children, 'buckle' or 'greenstick' type (visible fracture on one side and buckle on other) injuries are common due to bone compliance. Buckling without a visible fracture line is termed a 'torus' injury.

An anatomical unit is formed by the radius and ulna, with an interosseous ligament throughout their length. It is stabilised at the elbow and wrist, thus forming a ring. A fracture of the shaft of one of these bones with visible shortening will lead to dislocation at the wrist or elbow of the other. An ulnar shaft fracture with shortening causes the radius to dislocate at its point of weakness at the elbow (known as the Monteggia fracture-dislocation). However, if the radius fractures with shortening, it will cause the ulna to dislocate at its point of weakness at the distal radioulnar joint (known as the Galeazzi fracture-dislocation).

Greenstick Fracture

- Buckling of the palmar (volar) cortical surface of the radius.
- Visible fracture through the dorsal cortex of the radius.
- Ulna is normal.

Torus Fracture
- Buckled radius
- No visible fracture line

Stabilisation is required for all displaced adult forearm fractures because no comparable result is achievable by other means of management. There are a number of indications for operative treatment, including fractures of both radius and ulna (Fig. 5.11), Monteggia and Galeazzi fracture dislocations (Figs. 5.12 and 5.13), isolated radius fractures, displaced ulna shaft fractures, delayed union or non-union, open fractures and pathological fractures.

Wrist

A complex joint, the wrist bridges the hand to the forearm. The wrist comprises the distal ends of the radius and ulna, eight carpal bones and the proximal portions of the five metacarpal bones. These bones participate in complex articulations that allow variable mobility of the hand. The wrist joint has a complex configuration of ligaments, without sacrificing stability. FOOSH, motor vehicle accident or sports contact injury is the commonest reason for wrist fractures and dislocations as a result of axial loading on the outstretched palm and extended wrist.

Posterior-anterior (PA) and lateral are standard wrist views. On occasions, further views are helpful so that the eight overlapping bones are more easily seen.

The wrist comprises the scaphoid, lunate, triquetrum, pisiform, trapezium, trapezoid, capitate and hamate bones. The radiocarpal, distal radioulnar and carpometacarpal joints can also be considered part of the wrist. When assessing the wrist, it is important to assess the bones and the joint spaces separating them.

The term distal radius fractures properly cover all fractures of the distal articular and metaphyseal areas. Restoration to the prior level of functioning should be the main aim of the treatment and specific goals will differ depending on the activity of the patient and not simply by age. Distal radius fractures are probably the most common presentation to an emergency department. The mechanism of injury differs in the young and the elderly. High-energy injuries are more common amongst the younger age group whilst low-energy injuries being more common in the older age group (Fig. 5.14).

The median nerve is always compressed after a fall on the palmar aspect of the hand that results in a distal radius fracture, and it is important to specifically assess and examine the median nerve function and document appropriately. Anatomic reduction is recommended in a patient who is active in recreation or engages in forceful activities at work. Three parameters are relevant in the decision to reduce the intra-articular step off (less than 2 mm), dorsal tilt (less than 10°) and radial length (less than 5 mm).

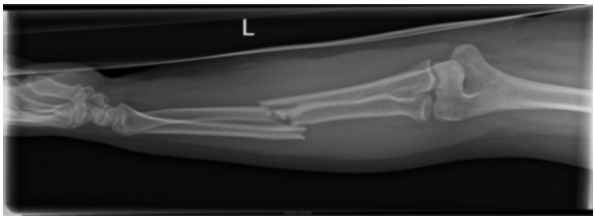


Fig. 5.11 Mid-shaft forearm fracture of left radius and ulna

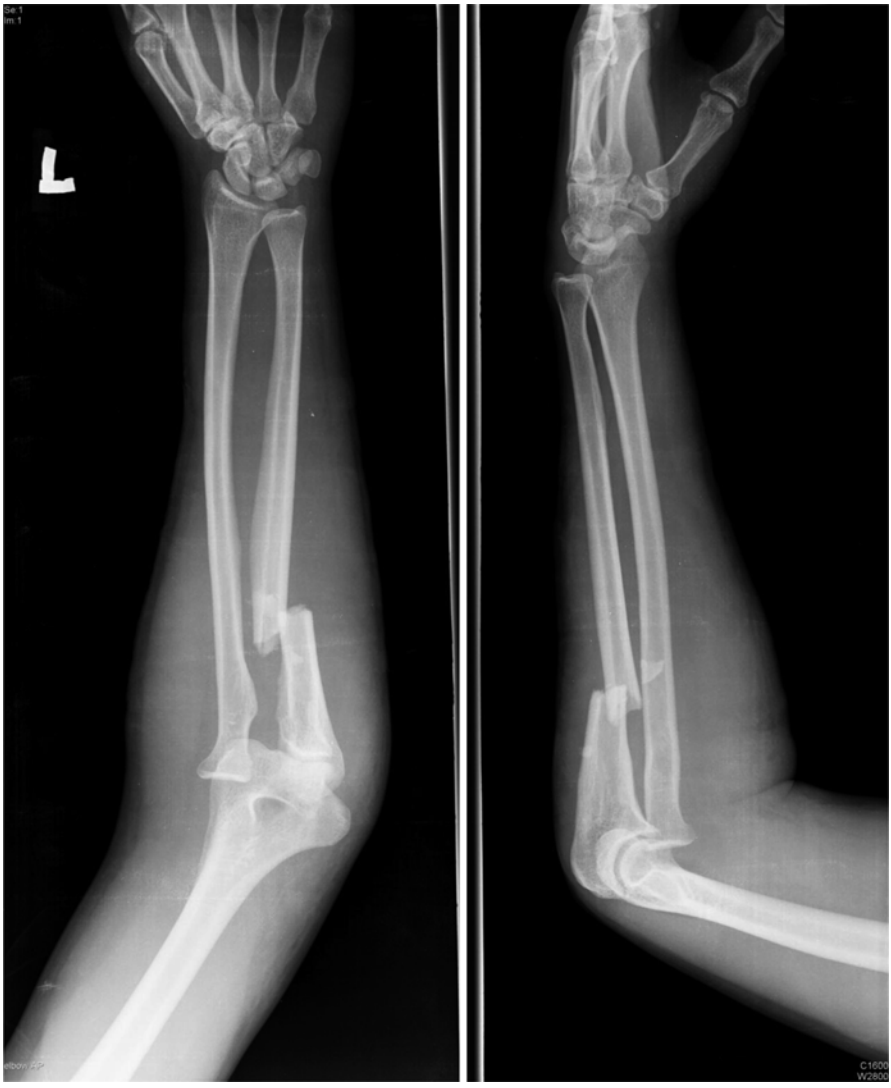


Fig. 5.12 Monteggia fracture (Courtesy: Prof. SS David, Pushpagiri Medical College Hospital, India)

Rigid immobilisation is the mainstay of treatment in the nonoperative management. However, surgical treatment has been reserved for displaced, irreducible distal radius fractures or reducible but unstable fractures. There are a number of operative techniques, including closed reduction and percutaneous pinning, external fixation, dorsal plating and fragment-specific fixation.

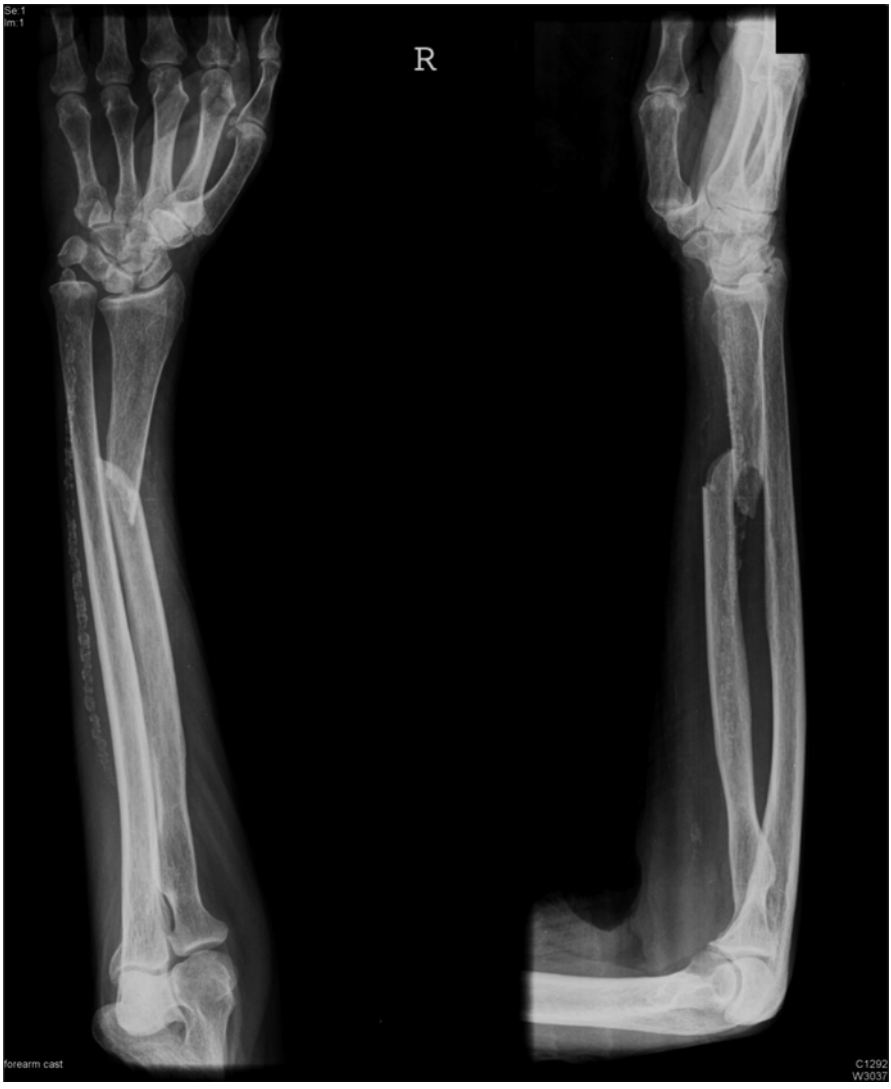


Fig. 5.13 Galeazzi fracture (Courtesy: Prof. SS David, Pushpagiri Medical College Hospital, India)

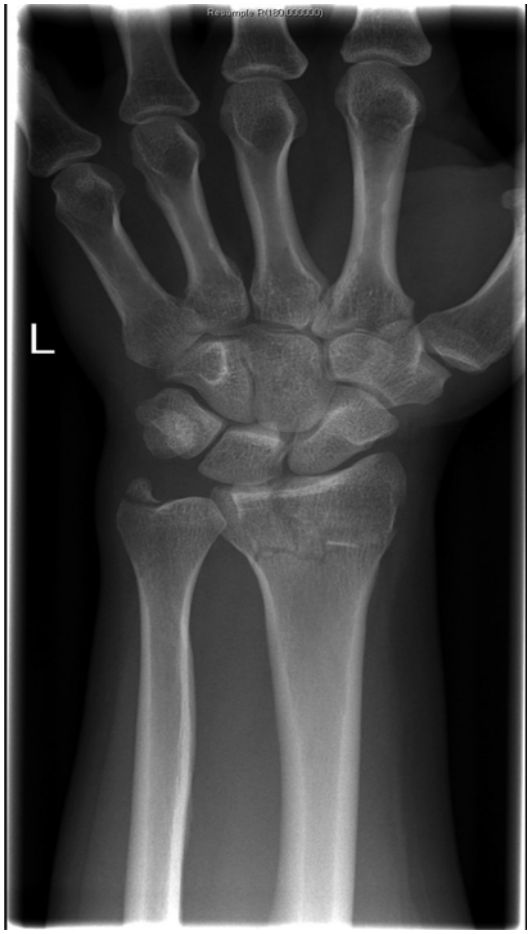
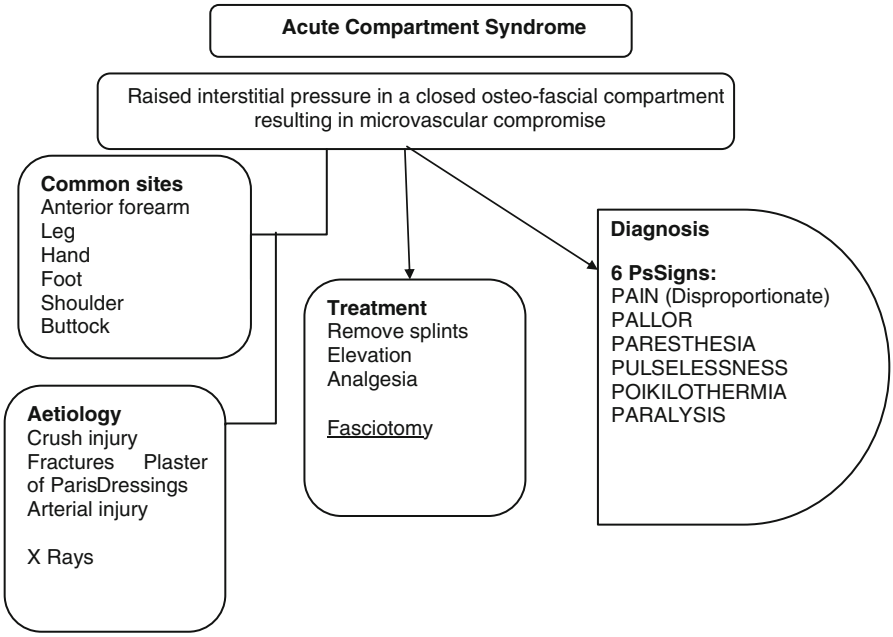


Fig. 5.14 Intra-articular fracture of the left distal radius

Acute Compartment Syndrome

An acute compartment syndrome occurs when there is an increase in the interstitial tissue pressures within an enclosed fascial envelope, preventing adequate tissue oxygenation and can cause cellular necrosis of the compartment contents. This can occur due to an injury or condition, such as blunt or penetrating trauma, infection, burns or vascular injury. Compartment syndrome most commonly involves the leg and forearm [11, 12]. There is usually pain that is out of proportion to the injury; there is pain with passive movement of the compartment musculature, with tense compartments on palpation, paraesthesia and ultimately paralysis. Considering compartment syndrome as a possibility may be the most important factor leading to

its diagnosis. As it is a difficult diagnosis, based on suspicion and clinical features, objective intra-compartmental pressure measurements may be assessed as absolute readings or as values relative to diastolic blood pressure. An absolute tissue pressure of 45 mmHg constitutes an acute compartment syndrome. Treatment of compartment syndrome can be nonoperative or operative. Nonoperative treatment includes simply removing compressive dressings and elevating the affected limb to reduce swelling. Operative treatment involves performing a fasciotomy and decompression of the forearm.



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Part II
Paediatrics

Chapter 6
Acute Neurological Emergencies in Children

Indumathi Santhanam and Sangeetha Yoganathan

Introduction

Clinical presentation of neurological emergencies in children and adolescents can be viewed as two temporal patterns, acute and acute recurrent, and they arise due to a large variety of causes. Knowledge of the spectrum presenting to an emergency department is vital in optimising the quality of care delivered, and the guidelines developed should target the commonest presenting problem categories. This chapter commences with the clinical approach to a child with altered sensorium and proceeds to discuss the salient causes – raised intracranial pressure, status epilepticus, stroke, acute movement disorders and neuromuscular emergencies in children, from the emergency physicians' perspective.

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Clinical Approach to a Child with Reduced Level of Consciousness

Key Points
- Level of consciousness of a critically ill or injured child is rapidly assessed at the bedside by using an AVPU or ACDU scale as a part of the primary survey.
- The GCS score is a more reliable and objective way of assessing the level of consciousness of a child.
- Respiratory pattern, size and reaction of pupils along with eye movements and the movement of the limbs to a painful stimulus provide clues to the anatomic site and nature of the injury in a comatose child.

Introduction

Consciousness is the state of full awareness of self and one's relationship to the environment. Consciousness is divided into two components, namely, arousal (wakefulness or vigilance) and awareness (awareness of the environment and of the self) [1]. Impairment of consciousness can be either an activated mental state such as hallucination, illusion, delusion and delirium or reduced mental state such as obtundation, stupor and coma.

Pathophysiology

Arousal is determined by reticular activating system and projection to thalamus. Awareness is regulated by thalamus, cerebral cortex, their white matter connections and functioning reticular system. Disorders of consciousness result from interference with either or both of these systems [2, 3]. Causes for altered mental status in children are summarised in Table 6.1.

Clinical Features

Level of consciousness of a child is assessed at the bedside by the responses of the patient to the examiner. History taking is crucial to determine the possible aetiology in a child with unexplained coma (Table 6.2).

Rapid assessment of sensorium in ED is performed using AVPU or ACDU scale stated below:

AVPU scale

A, alert; V, responding to voice; P, responding to pain; U, unresponsive

ACDU scale

A, alert and oriented; C, confused; D, drowsy; U, unresponsive

Table 6.1 Causes of coma in children

Infective
Meningitis, encephalitis, cerebral malaria, neurocysticercosis, tuberculous meningoencephalitis
Inflammatory
Acute disseminated encephalomyelitis, autoimmune encephalitis, vasculitis
Metabolic
Hyponatraemia, hypoglycaemia, inborn error of metabolism, hepatic encephalopathy
Reye syndrome
Vascular
Ischemic stroke, haemorrhagic stroke, cerebral venous thrombosis
Trauma
Traumatic brain injury, inflicted neurotrauma
Drugs, toxins and envenomation
Hypertensive encephalopathy
Brain tumours
Nonconvulsive status epilepticus

Table 6.2 History in a child with altered mental status

Onset (abrupt, gradual)
Recent complaints (fever, headache, focal weakness, seizures)
Recent head injury
Previous medical illnesses (seizures, renal failure, heart disease, collagen vascular diseases)
Previous psychiatric illness
Access to drugs (sedatives, psychotropic drugs)

Table 6.3 Rapid neurological examination in a comatose child

Eye opening, motor response, verbal response
Optic fundi
Pupillary reactions
Spontaneous eye movements
Oculocephalic responses (cervical trauma excluded)
Corneal responses
Gag reflexes
Skeletal muscle tone
Deep tendon reflexes
Signs of meningeal irritation

Detailed assessment of neurological status is performed using Glasgow coma scale in older children and modified Glasgow coma scale in infants (discussed in management of raised ICP chapter). Examination should focus on the evaluation of airway, breathing and circulation. Document the vitals, pattern of respiration, external markers of trauma, markers of systemic illness and evidence of drug exposure such as abnormal breath odour or needle marks. Rapid neurological examination will help in diagnosing the depth and localization of coma. Neurological assessment in a comatose child is shown in Table 6.3. Acute brain injury of any aetiology can result in locked-in syndrome, minimally conscious state, vegetative state and brain death [4].

- Coma: awareness of self or environment, sleep-wake cycle, purposeful motor movement and response to pain do not exist.
- Vegetative state: sleep-wake cycle is intact, although there is no response to pain, purposeful motor movement or awareness of self and environment.
- Minimally conscious state: limited awareness of self and environment, intact sleep-wake cycle, limitation of purposeful motor movement and response to pain exist.
- Locked-in syndrome: presence of awareness of self and environment, intact sleep-wake cycle and response to pain but quadriplegic with preserved eye movements.
- Brain death: no awareness of self or environment, no response to pain, no sleep-wake rhythm, no spontaneous respiration, no motor response and has absent brainstem reflexes.

Signs with Localising Value in Coma

In a comatose child, *clues to the anatomic site* and nature of the injury include:

- Respiratory pattern
- Pupillary response
- Eye movements
- Position or movements of the limbs

  Based on the pattern of respiration, the localization of coma can be made as follows:

  – Cheyne-Stokes breathing – diencephalon
  – Neurogenic hyperventilation – high brainstem
  – Apneustic breathing – bilateral pontine
  – Cluster and ataxic breathing – pontomedullary junction
  – Apnoea – ventrolateral medulla

  Based on the pupillary responses, coma localization is shown below:

  – Small reactive pupils – diffuse encephalopathy and metabolic, diencephalic pathology
  – Large, fixed pupils with hippus – pretectal lesion
  – Unilateral dilated and fixed – uncal herniation
  – Mid-position and fixed – midbrain lesion
  – Pinpoint pupils – pons

Based on the motor response assessment, the presence of hemiparesis indicates contralateral cerebral hemisphere involvement, decorticate posturing in upper mid-brain damage and decerebrate posturing in upper pontine damage.

False Localising Signs

False localising signs are signs produced distant or remote from the expected anatomical locus of pathology [5]. List of false localising signs in neurological examination are stated below [5, 6]:

- Unilateral or bilateral sixth nerve palsy in raised ICP
- Trigeminal or facial nerve palsy in raised ICP
- Third nerve palsy and Kernohan notch phenomenon in uncal herniation
- Unilateral papilloedema with contralateral optic atrophy
- Hemidiaphragmatic palsy – ipsilateral medullary compression
- Pseudo-internuclear ophthalmoplegia – myasthenia gravis
- Pseudo-locked-in syndrome – Guillain-Barré syndrome

Investigations

Child brought with altered mental status to ED should be evaluated based on the differential diagnosis considered after history and examination. List of investigations to be considered in a child with coma are:

- Blood glucose
- Urine ketones
- Serum electrolytes
- Complete blood count
- Prothrombin time, activated partial thromboplastin time
- Urea, creatinine
- Liver function test
- Blood-borne virus screen
- Blood culture
- Electroencephalography
- CT brain/MRI brain ± MRA/MRV
- Arterial blood gas
- Blood ammonia
- Arterial lactate
- Serum amino acids, acylcarnitine profile
- Urine organic acids
- Urine toxicology, blood lead
- CSF analysis

Treatment

- Stabilise airway, breathing and circulation.
- Check blood glucose and correct hypoglycaemia with 2 ml/kg of 10 % dextrose if identified.
- Seizures and raised ICP are managed as per protocols discussed elsewhere in this chapter.
- In a child with fever and altered sensorium, initiate therapy with meningitic dose of third-generation cephalosporins (Ceftriaxone 100 mg/kg) and 10 mg/kg of acyclovir every 8 hours awaiting the CSF reports.

- Maintain euthermia and correct coagulopathies if detected.
- In children with suspected inborn error of metabolism, maintain glucose infusion rate of 8–10 mg/kg/min, correct metabolic acidosis, sodium benzoate and phenyl acetate if hyperammonaemia is detected and peritoneal or haemodialysis in refractory cases.
- Consider therapy with megavitamins in suspected mitochondrial cytopathies.
- Control hypertension in hypertensive encephalopathy.

Raised Intracranial Pressure: Causes and Management

Key Points
- Rapid identification and appropriate treatment of raised ICP are essential to prevent secondary brain injury.
- Mean arterial pressure (MAP) – Intracranial pressure (ICP) = Cerebral perfusion pressure (CPP).
- A normal fundoscopy examination does not rule out raised ICP.

Introduction

Raised intracranial pressure (ICP) often results in significant morbidity and mortality. Early identification and appropriate goal-directed management of raised ICP in the ED can often prevent secondary brain injury.

Pathophysiology

Brain, blood and cerebrospinal fluid (CSF) within the cranial vault contribute to intracranial pressure. In normal individuals, intracranial pressure ranges between 6 mmHg in infants to 10–15 mmHg in adults [7]. According to the Monro-Kellie doctrine, any increase in the volume of one component results in a compensatory decrease in the volume of the remaining two components thereby attempting to maintain normal ICP [8]. Raised ICP occurs when the compensatory mechanism fails.

Commonest causes of raised ICP in children are listed in the Table 6.4.

Clinical Features

Clinical manifestations in infants and children with raised ICP of varied aetiology are depicted in Table 6.5. In children less than 5 years of age, modified Glasgow

Table 6.4 Causes for raised ICP in infants and children

Increase in intracranial volume	Obstruction to blood flow	Obstruction to CSF
Brain tumours (primary or metastases)	Cerebral venous sinus thrombosis	Hydrocephalus (obstructive or communicating)
Intracranial haemorrhage	Arterial ischaemic stroke	Ventriculoperitoneal shunt dysfunction
Cerebral oedema (meningoencephalitis, inborn error of metabolism, toxin exposure, prolonged seizures)		

Table 6.5 Clinical symptomatology in infants and children with raised ICP

Young infants	Older children
Incessant cry	Headache, especially in the morning
Bulging fontanelle	Diplopia
Convergent squint (lateral rectus palsy)	Hemiparesis
Posturing	Projectile vomiting
Seizures	New onset of squint
	Seizures
	Ataxia

Table 6.6 Modified Glasgow coma scale for infants

1. Eye opening
Spontaneous-4
Speech-3
Pain-2
None-1
2. Motor response
Purposeful movements-6
Localizes pain-5
Withdrawal to pain-4
Flexor response-3
Extensor response-2
None-1
3. Verbal (modified for infants)
Cooing or babbling appropriately-5
Consolable cry-4
Inconsolable cry-3
Grunting or moaning to pain-2
None-1

coma scale (GCS) is used for the assessment (Table 6.6). Motor response is elicited by applying supraocular, sternum or nail bed pressure. As ICP worsens, the following clinical findings may be noted [9]:

- Profound fall in mental status (pain responsive or unresponsive in AVPU scale) or consciousness level less than ≤8 in the GCS scale.

- Abnormal respiratory pattern (hyperventilation, irregular respiration or apnoea).
- Abnormal posture (decorticate, decerebrate or complete flaccidity).
- Abnormal doll's eye response.
- Abnormal pupils (unilaterally or bilaterally dilated or unresponsive pupils).
- Cushing's response (bradycardia and hypertension) occurs due to impending herniation of the brainstem. As a preterminal sign, it requires urgent intubation, controlled hyperventilation and anti-oedema measures.
- Papilloedema is a late sign. A normal fundoscopy examination does not rule out raised ICP.

Anticipate the risk of herniation in raised ICP and evaluate for the pattern of clinical findings that help in recognition of the level of herniation. Children with raised ICP often have eye signs of subtle status epilepticus. Eyes should be carefully assessed to identify eye lid twitch, conjugate deviation and nystagmus. On the other hand, unrecognized seizure activity increases ICP. The pupils are checked for pupillary size, equality and response to light, and the fundus is examined for papilloedema.

Investigations

- Computerized tomography (CT) of the brain is the investigation of choice following resuscitation.
- A cranial CT may appear normal even if the child has clinical evidence of ICP as seen during diffuse axonal injury.
- CT is ideal in identifying bleed, fracture, hydrocephalus, obliteration of cisternal spaces, compressed ventricles, mass lesion and midline shift [10].
- Magnetic resonance imaging (MRI) of the brain is indicated when CT does not explain neurological findings.
- Indications for neuroimaging in an ED are as follows:
  - Clinical suspicion of nonaccidental injury
  - Post-traumatic seizure (no past medical history of epilepsy)
  - GCS <14 on initial assessment or if <1 year, GCS <15
  - GCS <15, 2 hours after injury
  - Suspected skull fracture or tense fontanelle
  - Focal neurological deficit
  - Aged <1 – bruise, swelling or laceration >5 cm on the head

Estimation of Optic Diameter

A rise in ICP directly affects the perioptic nerve space, which leads to an increase in optic nerve diameter [11]. Through transorbital sonography, the normal diameter of the optic nerve sheath is seen to be 3 mm. Increase in diameter of 5 mm is diagnostic of ICP.

Transcranial Doppler (TCD)

TCD is a non-invasive tool that measures velocity of blood flow in the middle cerebral arteries and helps in early detection of ICP [12].

Invasive ICP Monitoring

Invasive ICP monitoring is indicated in patients with GCS ≤8, patients with traumatic brain injury with abnormal CT brain on admission and intracranial mass lesions [13].

Treatment

- Resuscitation of raised ICP in the paediatric emergency department is not dependent on aetiology.
- Energetic management using an ABCDE approach along with management of seizures if present is the backbone of treatment.
- Maintain higher MAP to ensure adequate CPP.
- Adjunctive measures such as sedation, analgesia and maintenance of metabolic parameters help to improve the outcome.
- Discuss the need of neurosurgical intervention.
- If features of pulmonary oedema are unmasked during fluid therapy, an inotrope is initiated, and intubation and ventilation should be provided.
- Care is taken to maintain euglycaemia. Hyper- or hypoglycaemia can worsen outcome.

Specific Measures in Management

- Head end is elevated 30° up in the neutral position.
- Adequate pain and sedation after intubation is mandatory to avoid raise in ICP secondary to pain and agitation. Fentanyl infusion is initiated @1 μg/kg/min.
- Catheterization not only helps to monitor urine output, it also prevents aggravation of ICP caused by the noxious sensation of a full bladder.
- Fever can increase cerebral metabolic rate. Paracetamol suppository can help to treat fever and additionally provide pain relief.
- Hypertonic saline bolus causes a reduction in cerebral oedema by induction of an osmotic gradient.
- It also has the benefit of increasing intravascular volume without causing diuresis and worsening shock in an acutely ill child in emergency settings!
- Infuse 3 mL/kg of 3 % or 2.7 % (premade) hypertonic saline solution over 10–20 min (Na increase of 2–3 mmol/L). A greater increase may occur if there

is a large diuresis. A sodium rise of <10 mmol/L in 24 h is acceptable, but serum sodium should not exceed 150 mmol/L.
- Mannitol causes reduction in cerebral oedema through induction of an osmotic gradient, and 20 % mannitol is infused at a dosage of 0.25–1 g/kg over 10–20 min. Since it causes diuresis leading to dehydration and shock, it should be used with caution.
- Ventilation is titrated to maintain eucapnia (PaCO2 35–40 mmHg). Hyperventilation is used briefly in children who have clinical features of cerebral herniation. Prolonged hyperventilation is not recommended; it can precipitate decreased cerebral perfusion leading to brain ischaemia.
- Call for neurosurgical help when herniation is recognized and plan decompressive craniectomy.
- External ventricular drainage can be attempted in children with poor GCS and hydrocephalus or VP shunt dysfunction.

Status Epilepticus

Introduction

Status epilepticus (SE) is defined as "seizures persisting for more than 30 min or series of seizures occurring consecutively without an intervening period of full recovery of consciousness [14]". However, the operational definition of SE is any seizures lasting for more than 5 min in duration [15]. Status epilepticus can be categorized as follows [16, 17]:

- Early or impending SE: 5–30 min
- Established SE: 30–60 min
- Refractory SE: Seizures persisting despite treatment with adequate doses of two or three initial antiepileptic medications.
- Super refractory SE: Seizures that persists for more than 24 h on anaesthetic therapy or seizure recurrence on weaning off anaesthesia.

Seizures that do not cease in 5 min are less likely to terminate without intervention. Hence, a child who is convulsing on arrival in to the ED is more likely to continue to convulse unless actively treated. Non-convulsive status epilepticus (NCSE) needs to be considered in children with failure to regain baseline mental status after a convulsion necessitating appropriate intervention.

Pathophysiology

Glutamate is the excitatory amino acid neurotransmitter, and gamma-aminobutyric acid (GABA) is the inhibitory neurotransmitter. Most seizures terminate spontaneously within 2 min, due to GABA-mediated inhibition that occurs in response to

Table 6.7 Clinical approach to a child brought with SE

History	Clinical examination
Seizures: age at onset, semiology, duration, frequency	Airway, breathing, circulation
Provoking factors: fever, withdrawal of antiepileptics	Level of consciousness
Antiepileptic drugs: dose, duration, compliance, response, adverse effects	Eye deviation, impaired DEM, pupillary abnormalities, eyelid flutter, papilloedema
Developmental history: normal or delayed	Facial weakness
Toxin exposure	Abnormal tone and posture
Head trauma	Hyperreflexia
Perinatal risk factors	Extensor plantar
Any systemic illness	Signs of meningeal irritation
Family history of epilepsy or febrile seizures	Description of convulsive event
Details of prehospital treatment	Rule out PNEE
	Neurocutaneous markers
	Facial dysmorphism

DEM Doll's eye movement, *PNEE* psychogenic non-epileptic events

seizures. Ongoing seizure activity results in loss of protective effects mediated by GABA. GABA receptors are either destroyed or recycled in to the cellular membrane. At the same time, continued seizure activity results in mobilization of excitatory N-methyl-D-aspartate receptors leading to continuing status epilepticus [18]. Benzodiazepines bind to GABA-A receptors and promote neuronal inhibition. Since the number of active GABA-A receptors decreases as an episode of SE progresses, first dose of benzodiazepines should be administered as early as possible for seizure termination.

Clinical Assessment

History taking in a child brought convulsing to ED, and focused neurological examination are crucial in the management of SE (Table 6.7).

Airway compromise in SE occurs due to loss of protective airway reflexes and glottic spasm.

Respiratory failure occurs because of muscle fatigue secondary to convulsive activity of the muscles of respiration and respiratory depressant effect of drugs used in the management of status epilepticus. An unstable and obstructed airway associated with ineffective ventilation for greater than 5 min can cause:

- Severe hypoxia
- Shock
- Myocardial dysfunction
- Increased risk of prolonged seizure activity

Failure to provide effective ventilatory support during the management of SE is perhaps one of the most frequent causes of morbidity and mortality. Correction of hypoxia during the management of SE is mandatory for intact neurological survival.

Shock could occur due to a wide variety of causes in convulsing children.

During SE, cerebrovascular resistance falls due to hypoxia, resulting in severe derangement of cerebral autoregulation. Cerebral perfusion becomes directly dependent on systemic blood pressure. Within the first ½ h of SE, blood pressure rises. Later blood pressure either becomes normal or hypotensive. Circulatory compromise that occurs in convulsing children are secondary to seizure activity and other causes such as sepsis or hypovolaemia complicating SE which severely deranges cerebral physiology. Hypoglycaemia can also cause severe disruption of autoregulation of cerebral blood flow leading to adverse neurological outcomes.

Investigations

In a child with new onset of seizures presenting as SE, the following investigations are recommended [19]:

- Blood glucose
- Serum electrolytes
- Electroencephalography
- CT brain or MRI brain
- Complete blood count, blood culture and lumbar puncture (if febrile)
- Urine and serum toxicology screening (if exposure to toxins is suspected)
- Genetic testing (karyotyping to look for ring chromosomes)
- Metabolic testing with arterial blood gases, ammonia, lactate, serum amino acids, acylcarnitine profile, urine organic acids, CSF lactate, sugar and neurotransmitters (to rule out inborn error of metabolism)

In a child with epilepsy on treatment and presenting with SE, therapeutic drug level monitoring is also recommended.

Management in the ED

- Ensure patent airway (position, suction, adjuncts, BMV).
- Provide 100 % oxygen.
- Check glucose and correct hypoglycaemia (2 mL/kg of 10 % dextrose).

- IV or IO access if no delay (otherwise buccal midazolam or PR diazepam can be used).
- Follow *Algorithm* 1 for steps involved in the management of status epilepticus.
- Tepid sponging and paracetamol IV/PR if fever is documented.
- Malignant hypertension may require treatment.
- Cefotaxime/ceftriaxone if suspected meningitis; acyclovir is added in suspected encephalitis and erythromycin in addition if aetiology unclear.
- Treat shock if present with fluids +/− inotropes.
- Lumbar puncture if there is reduced level of consciousness.
- Signs of raised intracranial pressure – consider mannitol 0.25 g/kg.

Drugs

Benzodiazepines

- Benzodiazepines are the most potent and effective first-line drugs in the management of SE. Administration of benzodiazepines within first 10 min of onset of seizure is beneficial due to the properties of GABA receptors and its allosteric modulators [20].
  - Lorazepam is the benzodiazepine of choice as it controls seizures within 3 min in 50 % of patients. Its potency and efficacy are comparable to diazepam; it has a longer duration of antiseizure effect and hence reduces the risk of recurrence [21, 22].
  - Midazolam is extremely efficacious, has a rapid onset of action and controls seizures in 90 % of patients. However, its shorter half-life may result in an increased risk of recurrence. Midazolam also has an added risk of hypotension.
- Presence of apnoea is not a contraindication to the administration of benzodiazepines.

Phenytoin

- Phenytoin is the second-line drug in patients not responding to the initial two doses of benzodiazepines.
- Since it is poorly soluble in water and precipitates in dextrose containing solutions, it is infused in normal saline (dose of 20 mg/kg) at the rate of 1 mg/kg/min with maximum rate of 50 mg/min.
- Antiseizure threshold in the brain is reached within 10–30 min after infusion.

- Phenytoin is potentially cardiotoxic. Significant proportion of SE patients are at risk for cardiac dysfunction as it decreases the force of cardiac contraction producing hypotension and predisposes to cardiac arrhythmias.
- If pulmonary oedema or myocardial dysfunction is anticipated or occurs during phenytoin infusion, consider using levetiracetam and/or sodium valproate after the initial doses of benzodiazepines. Fos-phenytoin, a water-soluble phospho-ester of phenytoin, has been considered less cardiotoxic than phenytoin and may be safer.

Levetiracetam

- Levetiracetam acts through calcium channels, glutamate receptors and GABA modulation. It is administered as 20 mg/kg IV over 15 min (maximum, 3 g).
- Levetiracetam is a good alternative antiseizure drug, and its efficacy had been compared against phenytoin in randomized trials.
- In some children, it may cause reversible behavioural changes.

Valproic Acid (VPA)

- Valproic acid, a broad-spectrum anticonvulsant, acts by modulating sodium and calcium channels, as well as inhibitory GABA transmission. Dose of sodium valproate is 25 mg/kg bolus.
- Sodium valproate has less sedation, good cardiovascular profile and lower risk of respiratory failure compared to other anticonvulsants. It seems to be a safer option in children presenting with or at risk of co-existing developing cardiac dysfunction such as prolonged SE or SE with septic shock. It is safer in settings without immediate access to mechanical ventilation.
- Sodium valproate should be avoided if child has signs of liver diseases, metabolic diseases or haemostatic abnormalities.
- In adults with SE, VPA was found to be effective in termination of SE and well tolerated as compared with phenytoin.
- Termination of SE varied from 65 to 100 % in patients treated with intravenous sodium valproate [23].

Barbiturates

- Barbiturates are useful in management of refractory status epilepticus. It depresses neuronal excitability by enhancing the GABA receptor response. Depression of mental status and respiratory failure are common. Recommended dose of phenobarbitone is 20 mg/kg.

- Thiopentone sodium can be administered at a loading dose of 5 mg/kg followed by an infusion of 3–5 mg/kg/h.

Super Refractory Status Epilepticus

Consequences of prolonged seizures are oxidative stress, mitochondrial dysfunction, neuroinflammation and activation of signalling molecular pathways resulting in cerebral damage. There are no randomized trials in literature for treatment of super refractory SE. Various drugs tried in the management of super refractory SE are [18]:

- Midazolam infusion
- Propofol
- Ketamine
- Magnesium
- Pyridoxine
- Immunotherapy: steroids and intravenous immunoglobulin
- Ketogenic diet
- Deep brain stimulation
- Transcranial magnetic stimulation
- Vagal nerve stimulation therapy
- Resective neurosurgery

Pyridoxine

- Pyridoxine at 30 mg/kg may be beneficial in super refractory SE and in children with SE due to ALDH7A gene mutations [18]. Pyridoxine infusion may cause bradycardia, apnoea and hypothermia.

Midazolam Infusion

Midazolam, as a continuous infusion of 1 μg/kg/min with increments of 1 μg/kg/min every 5 min, is recommended until seizures are terminated [24].

Propofol

- Propofol exerts seizure control through GABA-A agonistic action, N-methyl-D-aspartate receptor antagonistic action, calcium and sodium channel modulation.

- Loading dose of 2 mg/kg followed by an infusion of 2–10 mg/kg/h is recommended.
- Tachyphylaxis, propofol infusion syndrome and hypotension are adverse effects observed on propofol infusion.

Topiramate

- Topiramate has an N-methyl-D-aspartate receptor antagonistic action and has been used in the management of refractory SE in various studies and found to be efficacious [25].

Non-convulsive Status Epilepticus

- Evidence of conjugate deviation of eyes, lid twitch, nystagmus or unilateral clonic movements in an unresponsive child helps to recognize subtle status epilepticus.
- Subtle SE must be anticipated in children who developed sudden unresponsiveness or who had seizures and had not regained baseline consciousness. More commonly, convulsive SE evolves into subtle SE during resuscitation.
- Cessation of overt motor movements alone should not be considered as achievement of complete seizure control.
- Continuous video EEG monitoring is recommended to diagnose NCSE and titrate drugs in accordance with resolution of electrographic abnormalities.
- Continuation of the aggressive management of the airway, breathing and circulation with the same drug protocol as for convulsive status epilepticus until all therapeutic goals are achieved is recommended.

Treatment of Specific Causes of SE

Central nervous system infections, cortical malformation, head trauma, cerebral oedema, space-occupying lesion, haemorrhage, toxins, hypoxia, hypertensive encephalopathy, inborn error of metabolism, electrolyte imbalances and autoimmune encephalitis can all produce seizures that are difficult to control. Assessment of patient for possible aetiology is performed simultaneously during the management of refractory seizures (Fig. 6.1).

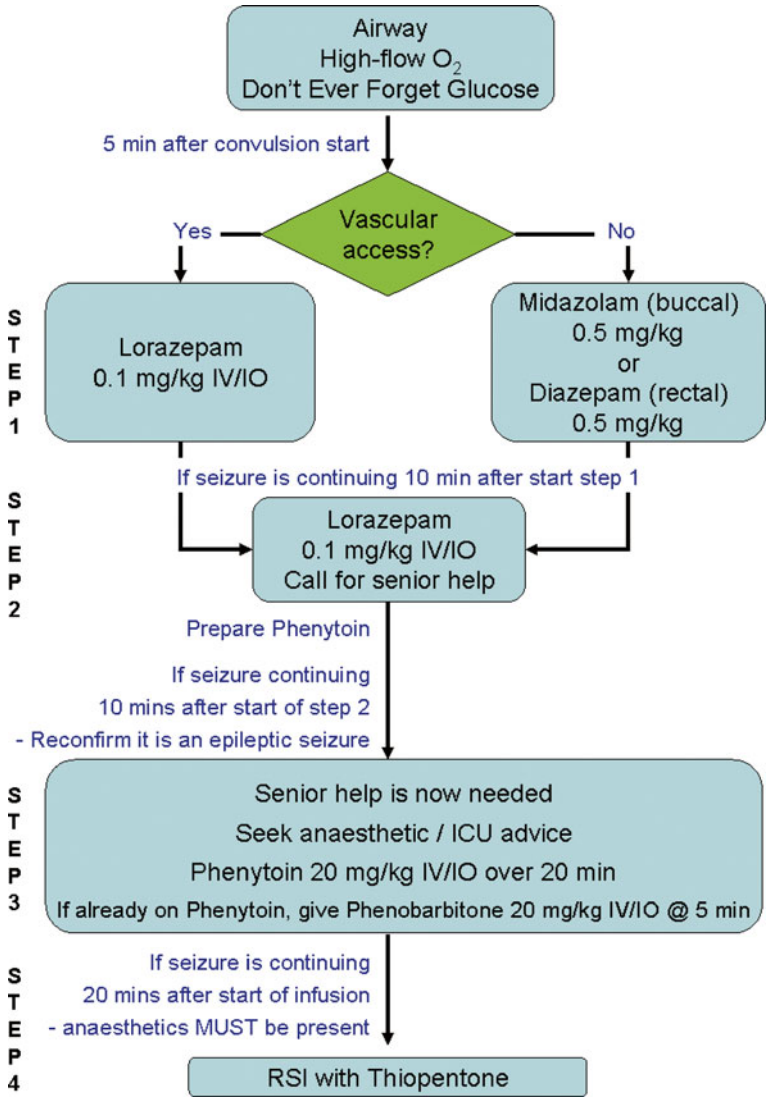

Fig. 6.1 ED algorithm for management of status epilepticus

Stroke in Children

Key Points
- There is equal prevalence of ischaemic and haemorrhagic stroke in children in contrast to adults where ischaemic stroke predominates.
- Aetiology for stroke in children is usually multifactorial and diverse.

Introduction

Stroke is defined as "rapidly developing signs of focal or global disturbance of cerebral function, lasting more than 24 h, with no apparent causes other than of vascular origin" [26]. Prevalence rate of stroke in adults varied from 64 to 270 per 100,000 among the different regions of India [27]. Stroke incidence rate of 1.2–2.3 per 100,000 was documented in children from a hospital-based database [28]. Incidence of childhood cerebral venous thrombosis (CVT) is 0.3 per 100, 000 children per year for term birth to 18 years of age and nearly half of the cases occur in neonates [29]. Perinatal stroke refers to the cerebrovascular lesions that occur between 20 weeks of gestation and 28 days of life. In contrast to adults where ischaemic stroke cases are significantly higher than haemorrhagic stroke, childhood stroke has almost equal prevalence of ischaemic and haemorrhagic stroke [30]. About 80 % cases of perinatal stroke are ischaemic, and remaining cases are attributed by haemorrhagic stroke or CVT [31].

Pathophysiology

Aetiologies of stroke in children are different from adult population. Aetiology for stroke in children is usually multifactorial and diverse which are summarized in Table 6.8. Stroke results from interruption of blood flow resulting in infarction. Central area where ischaemia is severe and infarction develops rapidly is the core area, and penumbra refers to the marginally perfused area surrounding the core which has the capacity to recover if perfusion is restored early. Stroke management aims at the preservation of perfusion and function of the penumbra.

Clinical Features

Stroke occurring due to involvement of large arteries results in seizures, altered sensorium, headache and focal deficits depending on artery affected. Small vessel-mediated stroke can lead to hemiplegia and movement disorders.

Table 6.8 Aetiology of stroke in children

Arterial	*Venous*
Cardiovascular	Sinusitis, otitis media, mastoiditis, pyogenic meningitis
Cyanotic and rheumatic heart disease	Dehydration
Cardiomyopathies	Inherited and acquired prothrombotic states
Cardiac surgeries	Head injury
Haematological	Brain tumour
Sickle cell anaemia	Systemic diseases
Protein C, S deficiency	SLE, thyrotoxicosis, Behçet's disease
Antithrombin III deficiency	
Polycythaemia	
Thrombocytosis	
Methylterahydrofolate reductase deficiency	
Leukaemia	
Lupus anticoagulant	
Factor V Leiden mutation	
Elevated lipoprotein A	
Vasculopathies	
Post varicella	
Moyamoya syndrome	
Infection associated	
Trauma	
Arterial dissection	
Metabolic	
Homocystinuria	
Hyperlipidaemia	

SLE Systemic lupus erythematosus

Commonest age of cerebral venous thrombosis (CVT) is infancy (50 %), and it usually occurs after an acute diarrhoeal episode. Diffuse signs and seizures are more likely than focal signs in infants with CVT. Infants present with lethargy, jitteriness, seizures and hemiplegia, and older children might present with focal signs. Focused history taking and examination are essential in ED as shown in Table 6.9.

Children with haemorrhagic stroke present with any of the following clinical symptoms:

- Thunderclap headache, vomiting or neurological symptoms
- Increased ICP
- Seizures
- Focal signs

Table 6.9 Assessment of a child with stroke

History taking	Clinical examination
Head and neck trauma	Assessment of airway, breathing, circulation
Unexplained fever/malaise	Vital signs and hydration
Recent infection	General examination
History of cardiac illness	Skin: rashes, Raynaud's phenomena
History of haematological disorders	Signs of infective endocarditis
Varicella in last 12 months	Stigmata of neurocutaneous syndromes
Head and neck irradiation	Dysmorphology
Drug ingestion (aspirin, oral contraceptives, phencyclidine)	Anaemia
Developmental delay	Stature and Marfanoid habitus
Migraine	Signs of connective tissue or systemic vasculitis
Family history (hyperlipidaemia, stroke or myocardial infarction at young age, hematologic disease, migraine and intellectual disability)	Signs of trauma – head, neck or intraoral

- Rapid deterioration in consciousness
- Posterior fossa bleed – Gaze abnormality, ataxia and rapid coma

Rapid neurological examination must be carried out in any infant or child presenting with stroke as it helps in the anatomical localization. It includes the assessment of level of consciousness, speech assessment, cranial nerves examination, motor and sensory examination and evaluation for cerebellar signs.

Investigations

Causes for stroke in children are multifactorial, and at least 50 % of them have an identifiable risk factor [32]. Aetiological evaluation of a child brought with stroke to ED is shown in Table 6.2.

Treatment

Supportive Care

Stroke management aims at neuroprotection, and measures are attempted to maintain adequate oxygenation, ventilation and hydration, correction of systemic

hypotension (target BP 50th–90th centile), maintaining euglycaemia, euthermia, control of seizures and management of raised ICP.

Perinatal Stroke

Class I evidence exists for correction of low platelet count, factor replacement in coagulation factor deficiency, vitamin K administration in haemorrhagic disease of newborn and placement of ventriculoperitoneal shunt in hydrocephalus after intracerebral haemorrhage [31]. Class II evidence exists for supplementation of folate and B vitamins in methyltetrahydrofolate reductase deficiency, correction of dehydration, correction of anaemia, evacuation of intracerebral haematoma and neurorehabilitation in perinatal stroke. Anticoagulation is considered in neonates with underlying prothrombotic state, multiple emboli or propagating thrombus [31].

Haemorrhagic Stroke

Efforts are directed to stabilize airway, breathing and circulation. Measures should be initiated to reduce the risk of rebleeding and to treat the haemorrhage itself. Effective control of systemic hypertension, management of seizures and raised ICP are paramount. Definitive therapy must be planned after the child's condition stabilizes. Conventional surgery, radiosurgery or endovascular obliteration of aneurysms and arteriovenous malformation (AVM) are effective for children with bleed due to AVM rupture. Class I evidence exists for factor replacement in children with severe coagulation factor deficiency [31]. In children with supratentorial intracerebral haematoma, surgical evacuation is not recommended.

Ischaemic Stroke

Adequate hydration, oxygenation, correction of shock and periodic transfusion are recommended in children with sickle cell anaemia. Revascularization surgery and aspirin are indicated in children with recurrent stroke due to moyamoya disease. Unfractionated or low molecular weight heparin is indicated in children with arterial dissection, failure of aspirin prophylaxis and cardiac thromboembolism [31]. Children with cardiac failure must be initiated on antifailure medications, and surgery should be attempted in all cardiac disorders with feasible surgical treatment [31].

Prognosis

Reported mortality rates varied between 5 and 13 % [32, 33]. Altered sensorium at presentation, seizures and completed middle cerebral artery stroke are associated with poor outcome in children [33]. Long-term morbidities that have been reported in children with stroke are neurological deficits, seizures, speech deficits, movement disorders and behavioural problems.

Acute Movement Disorders in Children

Key Points
- Dystonia refers to an involuntary movement resulting from simultaneous contraction of agonist and antagonist group of muscles.
- Dystonia severity can be graded from grade 1 (comfortable sitting and normal sleep to grade 5 (status dystonicus).
- Differentials for status dystonicus include neuroleptic malignant syndrome, malignant hyperthermia and drug-induced acute dystonia.

Introduction

Dystonia refers to an involuntary movement resulting from simultaneous contraction of agonist and antagonist group of muscles. This hyperkinetic movement disorder is characterized by abnormal repetitive twisting movements, posture or both [34]. Status dystonicus (SD) is a movement disorder emergency which is often underdiagnosed and if untreated can result in significant morbidity and mortality. No consensus definition on the duration or frequency of dystonic spasms to be labelled as status dystonicus exists [35]. Status dystonicus is defined as an increase in the frequency or duration of dystonic spasms that requires hospitalization, and only about 100 cases have been reported in literature [36].

Pathophysiology

SD occurs commonly in children between 5 and 15 years of age with chronic dystonia or rarely can have an acute onset in a previously normal child. Childhood disorders presenting with dystonia and are at risk for dystonic storm are cerebral palsy, Wilson disease, glutaric aciduria, autoimmune encephalitis,

Japanese encephalitis, drug-induced, basal ganglia stroke, systemic lupus erythematosus, acute necrotizing encephalopathy of childhood, subacute sclerosing panencephalitis and neurodegenerative disorders [37, 38]. Provoking factors reported for SD are febrile illness, pain due to any aetiology, change in medications, stress, surgical procedures, gastro-oesophageal reflux, constipation and trauma [36].

Basic pathophysiology is the loss of inhibitory functions at multiple levels, namely, cortex, brain stem and spinal cord. In addition, there is an imbalance in basal ganglia networks and loss of excitation of inhibitory pathways from cortex which results in loss of surround inhibition. Loss of surround inhibition is associated with overflow of contraction into other muscles during a motor task [39].

Clinical Features

Clinical evaluation starts with focused history taking. Antenatal risk factors, perinatal asphyxia, kernicterus, developmental delay, jaundice in past, exposure to drugs or toxins and family history should be checked. With regard to dystonia, history of onset, duration, distribution, aggravating factors, medications and response to treatment have to be documented. During SD, there are uninhibited and overflow contraction of muscles leading on to various clinical symptoms that are summarized in Table 6.10 [36]. Autonomic symptoms are rare. Fractures, gastrointestinal bleeding and sepsis are other complications encountered in SD.

Lumsden et al. have devised a scale for assessment of dystonia severity based on the clinical and laboratory parameters [40]. Grading of dystonia severity based on Lumsden scale is depicted below:

Grade 1 – Comfortable sitting and normal sleep.
Grade 2 – Irritable, dystonia interferes with sitting but not lying.
Grade 3 – Dystonia interferes with lying and sleep is disturbed.
Grade 4 – Evidence of early multi-organ failure and metabolic decompensation.
Grade 5 – Life threatening with full-blown metabolic decompensation and multi-organ failure.

Table 6.10 Clinical manifestations in children with status dystonicus

1. Sensorium	Usually remains intact. Altered level of consciousness may be present in heredodegenerative disorders or in children with hypoxemia due to respiratory failure
2. Bulbar muscles	Dysphagia, dysarthria, risk of aspiration, airway compromise
3. Diaphragm	Respiratory failure
4. Generalized dystonia	Pain, sleep disturbances, exhaustion, hyperthermia, rhabdomyolysis, myoglobinuria, dehydration, renal failure

Description of grade 3/5 and 4/5 of the above grading system probably elucidates the pre-status dystonicus and status dystonicus phases, respectively.

Differential Diagnosis

Neuroleptic Malignant Syndrome (NMS)

It is an idiosyncratic reaction with reported incidence of 0.2 % [41]. NMS can occur in children, and both genders are affected. Rapid changes in doses of neuroleptic medications, such as metoclopramide, prochlorperazine, tetrabenazine, lithium and pimozide, are risk factors for developing NMS [36, 41]. Major criteria for diagnosis of NMS are fever, rigidity and elevated creatine kinase level. Minor criteria include tachycardia, abnormal blood pressure, tachypnoea, altered consciousness, diaphoresis and leucocytosis. Presence of three major or two major and four minor suggests a diagnosis of NMS [42]. Treatment of children with NMS includes supportive care, ceasing the precipitant drug, dopamine agonist or dantrolene, and managing psychosis with electroconvulsive therapy [36, 41, 42].

Malignant Hyperthermia

It is a rare disorder occurring in children with mutations in gene encoding ryanodine receptor following exposure to halogenated inhalational anaesthetics and depolarizing muscle relaxants. Clinical syndrome is characterized by rapid onset hyperthermia, rigidity, rhabdomyolysis, labile BP, tachycardia, increased sweating, hyperkalaemia, metabolic acidosis and hypercapnia. Supportive care, withdrawal of triggering agents and dantrolene are the management options.

Acute Dystonic Reaction

It occurs in children on therapy with dopamine-blocking neuroleptics and antiemetics. Clinical presentation can be focal or generalized. Laryngeal muscles are usually affected resulting in laryngeal dystonia. Oculogyric crisis, cervical dystonia, blepharospasm and focal limb dystonia are the other presentations reported in acute dystonic reaction. Consciousness is preserved. Treatment is supportive with withdrawal of precipitant drug and use of benztropine or diphenhydramine.

Investigations

Investigations are targeted to identify the aetiology if not established already, to screen for sepsis as it is one of the most common triggering agents and to monitor the complications in an established SD (Table 6.11).

Table 6.11 Summary of investigations in a child with status dystonicus

To diagnose the aetiology	To look for triggering agent	To monitor complications
MRI brain	Blood count	Serum creatine kinase
Serum copper	Blood culture	Urine myoglobin
Serum ceruloplasmin	Chest X-ray	Serum electrolytes
Urine copper excretion	Urine culture	Arterial blood gases
Blood lactate, ammonia	Blood borne virus screen	Blood urea, creatinine
Urine organic acids		Urine myoglobin
Plasma amino acids		Upper gastrointestinal (GI) endoscopy in case of GI bleed
CSF lactate, pterins, neurotransmitters, protein, sugar, PCR for viruses, antimeasles antibodies		Electrocardiography
Acylcarnitine profile, mitochondrial and DYT gene mutation studies		Echocardiography

MRI magnetic resonance imaging, *CSF* cerebrospinal fluid, *PCR* polymerase chain reaction, *GI* gastrointestinal

Treatment

Supportive Measures

In a child admitted with SD, care should be taken to stabilize airway, breathing and circulation. Supportive measures in the management of SD are proper positioning, catheterization to monitor urine output, bowel care with laxatives, adequate nutrition with nasogastric feeds or parenteral nutrition, antibiotics for sepsis, ensure adequate sleep with chloral hydrate or benzodiazepines and adequate analgesia [36].

Medical Management

Randomized trials for management of SD do not exist. In established SD, clonidine can be administered by oral, enteral or intravenous route and dose is titrated in accordance with clinical response. If dystonic spasms are refractory, midazolam infusion, use of non-depolarizing muscle relaxants, propofol infusion and barbiturates are considered. Anti-dystonic medications that are used in the treatment of SD are benzodiazepines (diazepam, clonazepam), trihexyphenidyl, tetrabenazine, haloperidol, pimozide, baclofen, levodopa, carbidopa-levodopa, benztropine, benzhexol, sulpiride, lithium, bromocriptine, valproate, carbamazepine, primidone, phenytoin, acetazolamide, gabapentin, chlorpromazine, olanzapine, clozapine, risperidone and botulinum toxin injection [36, 43].

Surgical Management

Intrathecal baclofen pump, deep brain stimulation of globus pallidus, pallidotomy and thalamotomy are the surgical procedures beneficial in the management of refractory SD [36].

Specific Treatment for Underlying Aetiology

Low-lysine diet for glutaric aciduria, copper-free diet and zinc for Wilson disease, megavitamins for mitochondrial disease, steroids and immunoglobulins in autoimmune encephalitis are paramount in the management.

Prognosis

Mortality was reported as 10 % in a study and predictors of mortality in their cohort were tonic phenotype and male gender [43].

Neuromuscular Emergencies in Children

Key Points

- Guillain-Barre syndrome (GBS) and myasthenic crisis are among the common neuromuscular emergencies in children and adolescents.
- GBS is an immune-mediated neuromuscular paralysis in which the antibodies are directed against the gangliosides of peripheral nerves.
- Autoimmune myasthenia usually presents with myasthenic crisis.
- Easy fatigability with diurnal variation of symptoms differentiates MG from other LMN disorders.

Introduction

Lower motor neuron (LMN) comprises the anterior horn cell, root, plexus, peripheral nerve, neuromuscular junction and muscle. Acute motor weakness can result in emergency department admission in children. Guillain-Barre syndrome (GBS) and myasthenic crisis are among the common neuromuscular emergencies in children and adolescents. Annual incidence of myasthenia is 0.2–2 patients per 100,000, and GBS is 0.5–1.5 per 100,000 [44, 45]. Up to one-fourth of all MG patients have

disease onset in childhood or adolescence [46, 47]. There is lack of data on incidence of myasthenic crisis in paediatric population. Prevalence of myasthenic crisis in adults with myasthenia gravis varies between 15 and 50 % [44]. Risk of recurrent crisis is high with MuSK antibody-associated MG.

Pathophysiology

GBS is an immune-mediated neuromuscular paralysis in which the antibodies are directed against the gangliosides of peripheral nerves. Antibodies develop following natural infection (respiratory or gastrointestinal) or immunization in two-thirds of cases [48]. Antibodies bind to epitopes on the Schwann cells resulting in complement activation and myelin destruction. Another proposed mechanism is through cell-mediated immunity where T cells act against Schwann cells resulting in activation of macrophages and release of inflammatory mediators [49]. GBS is further subclassified into acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), acute inflammatory demyelinating polyneuropathy (AIDP), Miller Fisher syndrome (MFS) and Bickerstaff encephalitis based on the electro-clinical features.

Myasthenia is a congenital or acquired syndrome. Congenital myasthenic syndromes are inherited and rarely present with crisis. Neonatal myasthenia due to passive transfer of maternal antibodies can result in respiratory depression. Autoimmune myasthenia is characterized by the presence of antibodies directed against acetylcholine receptor (AchR) or muscle-specific tyrosine kinase (MuSK) antibodies. AchR antibodies cross-link with receptors at the postsynaptic membrane leading to endocytosis and degradation. Myasthenia with autoantibodies usually presents with myasthenic crisis, an emergency characterized by respiratory muscles weakness, bulbar muscles weakness or both with need for mechanical ventilation.

Clinical Features

Hallmark symptoms of LMN disorders that facilitate visit to ED are progression of weakness, bulbar or respiratory paralysis or severe pain. More than half of children with GBS have a history of preceding respiratory or gastrointestinal symptoms within 3 weeks of onset of weakness. GBS is diagnosed based on the Asbury et al. criteria [50] (Table 6.12).

Pattern of muscle weakness differentiates AchR from MuSK antibody-associated disease, and easy fatigability with diurnal variation of symptoms dif-

Table 6.12 Criteria for diagnosis of Guillain-Barre syndrome [50]

Definite	Supportive	Doubtful
1. Progressive weakness involving both upper and lower limbs	1. Symptoms progressive up to 4 weeks	1. Pulmonary dysfunction at onset but mild limb weakness
2. Areflexia	2. Symmetry of symptoms	2. Predominant sensory signs at onset
	3. Mild sensory symptoms or signs	3. Bladder or bowel symptoms at onset
	4. Cranial nerve involvement	4. Fever at onset
	5. Dysautonomia	5. Definite sensory level
	6. Presence of pain	6. Slow progression
	7. Albuminocytological dissociation in CSF	7. Marked asymmetry of weakness
	8. Typical electrophysiological features	8. Persistent bladder or bowel dysfunction
		9. Increased number of mononuclear cells in CSF (>50 × 106/L)
		10. Polymorphonuclear cells in CSF

CSF cerebrospinal fluid
Adapted from Asbury and Cornblath

ferentiates MG from other LMN disorders. Limb muscles being more involved than bulbar muscles, predominant ptosis, extraocular muscle weakness and neck extensor being more involved than neck flexors characterize the clinical phenotype of AChR-MG. Triggering factors for myasthenic crisis are infection, surgery, emotional stress, high environmental temperature, aminoglycoside therapy and coexistence of other autoimmune disorders [44].

Focused clinical examination in MG crisis and GBS is essential to check for reduced single breath count, poor chest expansion, use of accessory muscles, hypophonia, airway collapse due to oropharyngeal weakness, pooling of oral secretion, bifacial weakness and jaw weakness.

Differential Diagnosis

Other causes of acute onset motor weakness in infants, children and adolescents are summarized below:

- Anterior horn cell: Poliomyelitis and non-polio enterovirus infection
- Peripheral nerve: Porphyritic polyneuropathy, diphtheritic polyneuropathy and toxic neuropathies

- Neuromuscular junction: Botulism, spider envenomation and organophosphate poisoning
- Muscle: Inflammatory myopathies, metabolic myopathies, hypokalaemic or hyperkalaemic periodic paralysis

Investigations

In a child brought to ED with underlying neuromuscular pathology and crisis, blood should be send for complete blood count, C reactive protein and culture to rule out sepsis which is the most common triggering agent. Serum electrolytes must be sent to exclude hypokalaemic or hyperkalaemic periodic paralysis. Calcium, serum creatine kinase (CK), liver enzymes, arterial blood gases and thyroid-stimulating hormone should also be tested. Serum CK is elevated in inflammatory myopathies. ECG is done to look for arrhythmias in children with GBS. Chest X-ray is needed to rule out aspiration pneumonia and thymic enlargement.

AChR and MuSK antibodies testing are done in patients with myasthenic crisis. Detailed nerve conduction studies with repetitive nerve stimulation test and single fibre EMG should be planned to characterize the type of GBS and myasthenic syndrome. Magnetic resonance imaging of spine with contrast is done in suspected cases of polio or non-polio enterovirus myelitis. CSF samples analysis should be analysed for albumin-cytological dissociation and viral PCR. Screening for porphyria and heavy metals are done in appropriate cases.

Respiratory function indices such as vital capacity <10 mL/kg, negative inspiratory force <20 cmH_2O and positive expiratory force <40 cmH_2O indicates myasthenic crisis.

Treatment

Supportive Care for Children with Neuromuscular Emergencies

In an unstable child, emergency physician should take a focused history and perform rapid clinical examination with simultaneous efforts to resuscitate airway, breathing and circulation. Stabilize the child and monitor for progression of weakness with efforts to prevent fatal complications. Frequent monitoring is done to watch for respiratory paralysis, bulbar muscle paralysis and dysautonomia. Proper positioning of the child with frequent position change is needed to prevent decubitus ulcers, contractures and pulmonary embolism. Nociceptive pain in acute phase due to inflammation in GBS is often treated with paracetamol, analgesics, gabapentin and carbamazepine. However, a recent Cochrane review concluded that there is no sufficient evidence to support the use of any pharmacological intervention for

pain management in patients with GBS [51]. Prophylaxis for deep vein thrombosis is also recommended.

Specific Treatment

GBS

Intravenous immunoglobulins (IVIg) 400 mg/kg/day administered for 5 days or plasma exchange (PE) is the recommended treatment for children with GBS who are non-ambulant. A Cochrane review concluded that in children, according to low-quality evidence, IVIg probably hastens recovery compared with supportive care alone [52]. In this Cochrane review, other trials comparing IVIg and PE were also analysed, and it was found that efficacy of IVIg and PE wad similar.

Myasthenic Crisis

Removal of precipitating agent and treatment of infection with five sessions of plasma exchange (removal of 1–1.5 times plasma volume on each session) or human IVIg (0.4 gm/kg/day for 5 days) and corticosteroids (prednisolone 1 mg/kg/day) are recommended [53].

Prognosis

Nearly 15 % of children with GBS require mechanical ventilation. Prognosis of GBS in children is excellent with complete recovery in majority [45].

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Chapter 7
Eczema, Allergy and Anaphylaxis

Nikki Biggs and Sophie Vaughan

Allergic and Anaphylactic Reactions

Key Points

- Allergic or atopic patients have a predisposition to mount exaggerated immune responses to common environmental allergens such as pollen, insect venom or certain foods such as peanuts.
- Symptoms can consist of a spectrum of respiratory, cardiovascular, gastrointestinal and dermatological manifestations.
- Anaphylaxis is defined as a serious or life-threatening generalised allergic reaction with rapid onset, involving airway, breathing or cardiovascular compromise.
- Allergy assessment is mandatory in any child presenting with anaphylaxis, and a tailored management plan is needed, based on an individual risk assessment.

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Aetiology

The aetiology of allergy and anaphylaxis can be considered in four mains groups: foods, drugs, venom (insect bites, bee stings, etc.) and idiopathic. Foods are the most common precipitant in children [1].

The aetiology is variable depending on whether the onset is community or hospital based. Those presenting to an emergency department are more likely to have food, venom or idiopathic allergic reactions. Hospital-based episodes are more likely to be drug induced.

Epidemiology

The epidemiology of anaphylaxis has been challenged in the past by inconsistencies in the definition [2]. The incidence of allergic illnesses is on the increase [1], which has been attributed to changes in environmental factors such as air pollution and tobacco exposure, respiratory viruses and obesity [3].

Despite overall numbers of those suffering from allergy increasing, it is thought anaphylaxis, specifically and particularly in the very young, is under-diagnosed as the presentation is less specific and can mimic a number of other pathologies such as sepsis, GI upset and viral illnesses [4].

There is no overall figure for the frequency of anaphylaxis in the UK, but it is estimated that 1 in 1,333 of the population has experienced it at some point in their life [5]. The American College of Allergy, Asthma and Immunology Epidemiology of Anaphylaxis concluded that the overall frequency of anaphylaxis lies between 30 and 950 cases per 100,000 persons per year [1]. Anaphylaxis is fatal in 0.65–2 % of cases, causing estimated 20–30 deaths per year in the UK [1, 6].

Pathogenesis

Most cases of anaphylaxis are the result of mast and basophil cell activation via allergen-specific IgE molecules. Activation of such cells results in the release of chemical mediators such as histamine, tryptase and cytokines that produce the symptoms of disease in some or all of the target organs.

IgE-dependent reactions are usually precipitated by the more common allergens: food, medications and venoms. There are some reactions that are not IgE mediated and these are termed anaphylactoid reactions [4]; the precipitants for which are very varied but include radiocontrast media and non-steroidal (NSAIDs) [3].

Mast cells can be directly activated through precipitants such as exercise, cold, sunlight and ethanol and medications such as opioids [3]. Idiopathic anaphylaxis: anaphylaxis, when there is no apparent trigger, should raise the possibility of an underlying immunological condition such as mastocytosis.

Presentation

The classical presentation of an allergic reaction is that of an onset of symptoms minutes to hours after exposure. The onset of IgE-mediated symptoms usually occurs within 2 h and food allergens, specifically, within 30 min [2]. When anaphylaxis is fatal, death usually occurs very soon after onset, typically after 5–35 min; drug-induced reactions occur quicker than venom-induced ones, which occur quicker than food-induced reactions. Death has never occurred more than 6 h after onset after contact [1].

Initial symptoms can vary with aetiology and may include any of the following: pruritus around the face and mouth, a strange taste in the mouth, flushing, urticaria (local or generalised), facial angioedema, dry cough, nasal congestion, sneezing, feeling of tightness in the throat, shortness of breath, wheezing, nausea, vomiting, abdominal cramping, feeling faint and loss of consciousness in severe cases.

The younger the child, the vaguer the symptoms can be, with children presenting as pale and floppy with or without a history of previous reactions, which can be challenging to the diagnostician. As children get older, they are more likely to develop respiratory and oropharyngeal symptoms. Such episodes can present more in keeping with life-threatening asthma [1]. Adolescents are more likely to present in a similar way to adults, with a higher propensity for cardiovascular compromise and collapse.

In older children, anxiety/panic attacks can cause diagnostic confusion because of the combination of associated feeling of apprehension or 'sense of impending doom'. Tachypnoea, flushing and gastrointestinal symptoms can occur in both.

Syncope can also be a confusing presenting complaint, but simple vasovagal events are usually quick to resolve with simple measures and are unlikely to be associated with any of the symptoms listed above.

Pruritus, especially of the palms, feet and head, is often an impending sign of a more severe reaction and/or anaphylaxis, but it is important to recognise that cutaneous symptoms are absent in approximately 20 % of cases [6].

It is thought that many patients do not receive optimal medical management for anaphylaxis, for a variety of reasons, including the difficulty in differentiating it from other less severe histamine-releasing reactions and a lack of understanding in making the diagnosis [5].

Anaphylaxis is highly likely when any one of the following three criteria is fulfilled [2]:

1. Acute onset of an illness (minutes to hours) with involvement of the skin, mucosal tissues or both, e.g. generalised urticaria, hives, flushing or mucosal swelling
   And at least one of the following:

   (a) Respiratory compromise: dyspnoea, stridor and bronchospasm
   (b) Cardiovascular compromise: hypotension and collapse

2. Two or more of the following that occur rapidly after exposure to a likely antigen for that patient (minutes to hours):

   (a) Involvement of the skin or mucosal tissues
   (b) Respiratory compromise

(c) Cardiovascular compromise
(d) Persistent gastrointestinal symptoms, e.g. crampy abdominal pain, nausea and vomiting

3. Hypotension after known exposure to a known allergen for that patient.
   Hypotension for children is defined as systolic blood pressure <70 mmHg from 1 month to 1 year, <70 mmHg + (2 × age) from 1 to 10 years and <90 mmHg from 11 to 17 years.

Severe allergic reactions and anaphylaxis can both run a biphasic course, the quoted incidence varying between 6 % and 11 %, with up to 3 % of these being severe in nature [2, 3].

Risk Factors

Patients, who have had an anaphylactic reaction, have a strong likelihood of having a further one. The risk of developing anaphylaxis seems to be dependent on the severity and character of previous reactions.

Asthma, particularly when severe, or poorly controlled, is strongly associated with anaphylaxis [7], and almost all fatal cases occur in patients with asthma [2]. Additional risk factors include reactions to peanuts specifically, the amount of allergen, not being at home at the time of reaction, those who have a biphasic course and those on concurrent medication such as β-blockers [3]. Mast cell disorders, although rare in children, are also associated with an increased risk of severe or fatal anaphylaxis [7].

The very young, as they are unable to describe their symptoms, and the adolescent population, for their associated increased risk-taking behaviour, are also at higher risk [3].

Exercise-induced anaphylaxis (EIA) is a rare variant of illness where physical exertion itself seems to trigger and often involves concomitant ingestion of a specific food such as shellfish, celery or wheat [3]. Other cofactors thought to amplify an acute anaphylactic reaction, which are thought to be involved in up to 20 % of cases [3], include acute intercurrent infection, fever, emotional stress and premenstrual status in females. Concomitant ingestion of ethanol and/or NSAIDs enhances intestinal permeability and allergen absorption and hence can also augment a reaction [3].

Acute Management of Anaphylaxis (See Flowchart for a Brief Overview)

The initial treatment is hinged on early recognition of the severity of the reaction, which can sometimes be challenging should the child not be known to have allergies. A structured ABC (airway, breathing and circulation) approach should be used.

Removal of the Allergen

- The need to consider this is more important should the reaction occur in an inpatient setting, i.e. removing latex gloves and stopping an antibiotic infusion.
- Those with a reaction precipitated by inhaled/touched allergen should consider changing clothes, washing the hand/face, etc., to avoid ongoing exposure.

Adrenaline

- Intramuscular adrenaline is the cornerstone of treatment [6]; the α-adrenergic effects increase peripheral vascular resistance, blood pressure and coronary artery perfusion whilst reducing angioedema and urticaria. The β1-adrenergic effects increase heart rate and contractility, and the β2-adrenergic effects mediate bronchospasm and inhibit release of inflammatory mediators [2].
- Using adrenaline early is associated with a better outcome [1].
- It is underused in the treatment of anaphylaxis [3, 7].
- There are no absolute contraindications for using adrenaline; however, it should always be given after making an individual risk assessment. The side effects such as pallor, tremor, headache and palpitations are usually indicative that a therapeutic dose has been delivered. Serious adverse reactions, such as ventricular arrhythmias and hypertensive crises, usually only occur during an overdose of adrenaline [3].
- It should be delivered intramuscularly which delivers a peak concentration within 8–10 min.
- Nebulised adrenaline can be considered and has been found to be helpful with perioral or airway swelling, but its bioavailability renders it less effective for other manifestations of anaphylaxis.
- Intravenous adrenaline is warranted should repeated doses of intramuscular adrenaline be required or should there be an insufficient response. This should ideally be done with invasive monitoring in place.

Fluids

- Fluids should be run in parallel with treatment with adrenaline, in 20 ml/kg boluses, and response to these boluses should be monitored.
- Should the child be in need of more than 40 ml/kg, sedation to allow intubation, ventilation and invasive monitoring ± inotropic support should be considered.

B_2 Agonists

- Salbutamol delivered via a spacer and metered-dose inhaler or by nebuliser should be given for any wheeze; however, adrenaline should still be the first-line therapy should the child present with significant respiratory compromise and bronchospasm.

H_1 Antagonists

- Whilst this forms part of the management plan for allergic and anaphylactic reactions in most departments, there is no evidence of their efficacy [2]. It is thought that they help relieve itching, flushing, angioedema and nasal and eye symptoms [3]. It is usually the first-line treatment for mild to moderate allergic reactions.
- Their average onset of action is 30 min and should be given promptly if a child has been exposed to a known allergen or develops symptoms but should not delay the delivery of adrenaline if the child is in anaphylaxis.
- Cetirizine is potent and fast-acting antihistamine and should be considered as the drug of choice.

Corticosteroids

- These should not be considered a first-line treatment as their onset of action is not quick enough (4–6 h). They 'switch off' transcription of activated genes that encode pro-inflammatory proteins and in doing so help in reducing the incidence of biphasic reactions [3].

Observation/Monitoring

Protracted uniphasic anaphylaxis is uncommon but can last for days [3]. However, for the vast majority, close observation of children, who did not require adrenaline, for 4 h would be considered sufficient.

Those with a severe-enough reaction to need adrenaline should be observed for 6–12 h depending on how quickly their condition stabilises.

Patients with a severe or protracted course or those needing more than one dose of adrenaline would warrant admission.

Diagnosis and Investigation

A pattern of symptom onset and exposure in association with raised tryptase and histamine levels during the time of acute illness and raised IgE levels to the suspected antigen are usually sufficient to make the diagnosis.

Plasma histamine is elevated for a brief period of time but is unstable and difficult to measure in the clinical setting [4]. It is recommended that tryptase levels be measured as soon as possible during an acute reaction and then ideally within 1–2 h but no longer than 4 h from the onset of symptoms [5]. The half-life of tryptase is 2 h, so levels may well be back to normal within 6–8 h [1]. Levels within an acute setting that remain within normal limits do not rule out the clinical diagnosis [3].

When the diagnosis is uncertain, skin prick testing or serum-specific IgE for the common agents [6] may be performed if the diagnosis still remains unclear. There is no consensus currently on the optimal time after a reaction to perform these tests, although skin prick testing cannot be performed when the patient has recently had antihistamine, so it is commonly performed at a later stage as part of an allergy clinic review [3].

Chronic Management

All children who have had an episode of anaphylaxis should be considered for a referral to an allergy specialist service. When compared to those receiving standard care, they benefit from reduced incidence rates as a result of effective avoidance and a higher probability of successful adrenaline autoinjector use [5]. Those whose initial reaction was due to food, drugs or venom are expected to experience five to six more reactions in their lifetime. This is reduced to one to two in those with food-induced reactions under specialist care, and in those with drug-induced reactions, the recurrence rate is almost eradicated. The incidence of idiopathic anaphylaxis remains unchanged [5].

Current research, specifically within the paediatric population, has shown promising results from early introduction of peanut from 4 months of age showing significantly reduced peanut allergy at 60 months of age compared to a peanut-avoidance group [8]. Further studies are also looking at early introduction of allergenic foods in the diet between 3 and 6 months of age [9].

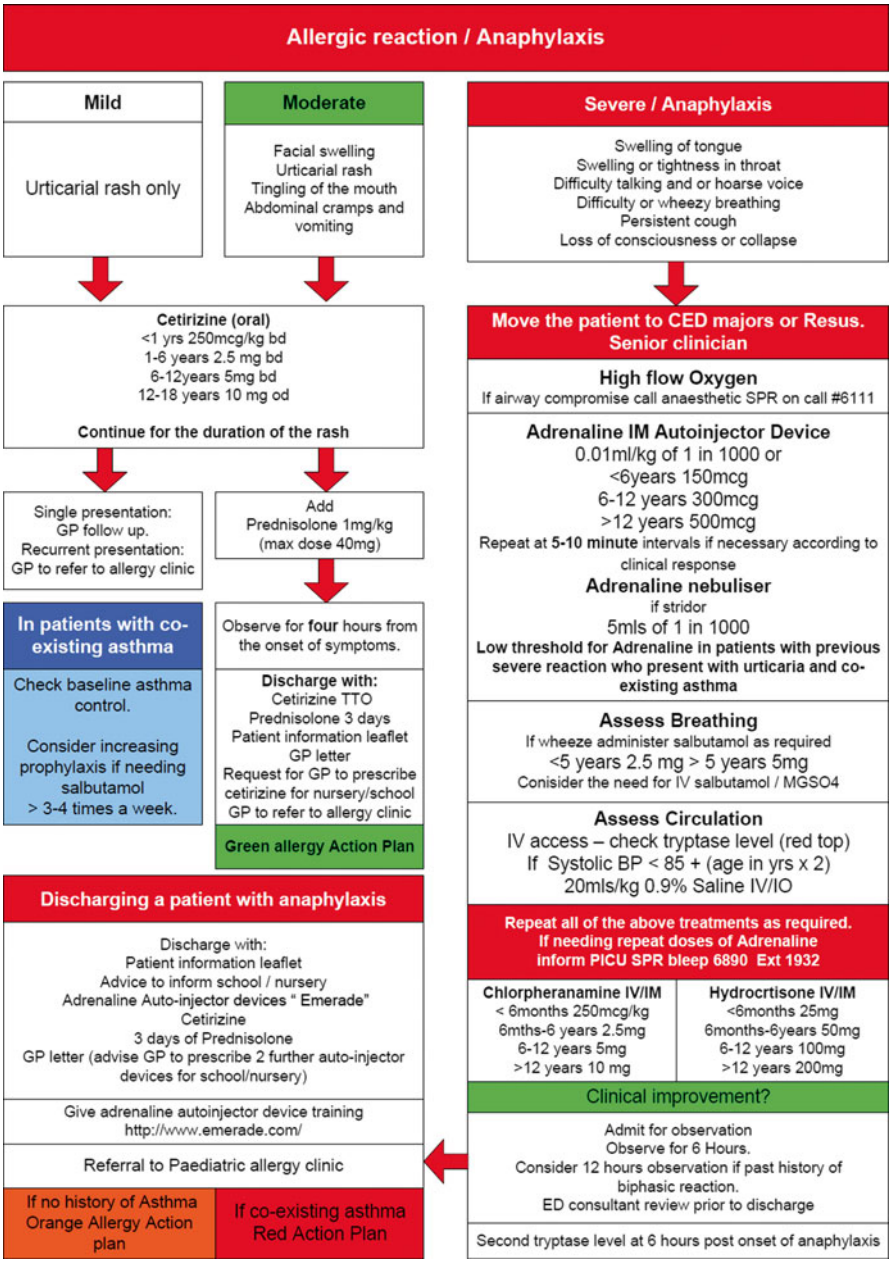
Agents such as omalizumab, a recombinant DNA-derived IgG monoclonal antibody, are also in early stages of development for allergy and anaphylaxis. It binds to free human IgE in the blood and interstitial fluid and to membrane-bound IgE. It is currently used in the management of steroid-resistant asthma.

Prevention

All patients should be educated about allergen avoidance if known. It is important to advice patients on allergies that commonly occur together, e.g. latex and kiwi fruits [6].

Should the child have needed adrenaline, or presented with a moderate reaction and have underlying asthma, an adrenaline autoinjector should be considered. The patient and carers should be trained in its use, ideally with a personalised action plan at discharge and regular review. The treatment plan should also include advice on symptom recognition and guidance on when to use mediation [6]. Optimal management of concurrent medical problems, asthma especially, is also essential.

Emergency management of an allergic reaction/anaphylaxis



Atopic Eczema/Dermatitis

Key Points
- Nine out of ten children who attend the ED with atopic eczema or dermatitis will be <5 years of age.
- The aetiology is often multifactorial (genetic, allergy and infection).
- There is an early and a late stage.
- Distribution is age dependent.
- Treatment is based on the severity/control at the time of presentation.
- About 60 % of children will have grown out of eczema by the time they are teenagers.

Aetiology

There is usually a family history of atopy in about two-thirds of the cases. Breastfeeding is thought to offer some protection in children of atopic families.

Triggers

- Irritants: Soap and detergents.
- Infection: Bacterial and viral. *Staphylococcus aureus* infections secondary to its endotoxins have been implicated. Vesicular or punched-out ulcerated lesions could suggest eczema herpeticum.
- Aeroallergens: Pollen, animal fur, house-dust mite.
- Stress.
- Overheating.
- Synthetic clothing.
- Food allergens.

Clinical Features

The early stage is characterised by erythema, scaling, itching and weeping. The late stage is characterised by lichenification and pigmentation changes.

Distribution

- Infancy: The face and scalp predominate followed by trunk and limb extensor surfaces.
- Childhood: Flexural creases of the elbow and the knee.
- Adolescents: Flexural creases of the elbow/knees, face, hands and feet.

Management [10]

This depends on the severity of the presentation. Three presentations are common (mild, moderate or severe flare). An ongoing itch is the biggest sign of under-treatment of the inflammation, and the ED physician should consider stepping up the acute treatment and/or consider infection. In all presentations advice the parents and the children on how to try and avoid known triggers. The mainstay in the management of eczema flare is emollients, corticosteroids and antihistamines.

Emollients (e.g. Diprobase and Epaderm)

Apply emollients generously. Do not rub onto the skin; just apply smoothly. A 500 g tub of emollient should last for about 2 weeks. Some may cause irritation, and if so consider trialling alternatives. If used in conjunction with a corticosteroid cream, wait for 15 min after the application of the steroid ointment before applying an emollient.

Corticosteroid Ointment

Three potencies	Mild: 1 % hydrocortisone (face and body)
	Moderate: Eumovate (body only)
	Potent: mometasone (body only)

Steroid ointments are applied as fingertip units (FTU: amount of cream squeezed from the tube along an adult's fingertip). An FTU is sufficient to cover an area of the skin twice of the flat of an adult's hand with the fingers together.

Antihistamines

Antihistamines not advised routinely.

Consider a 1-month trial of a non-sedating antihistamine to children with severe flare or those with moderate/mild flare when there is severe itching or urticaria. If successful continue for 3 months, and review the need thereafter.

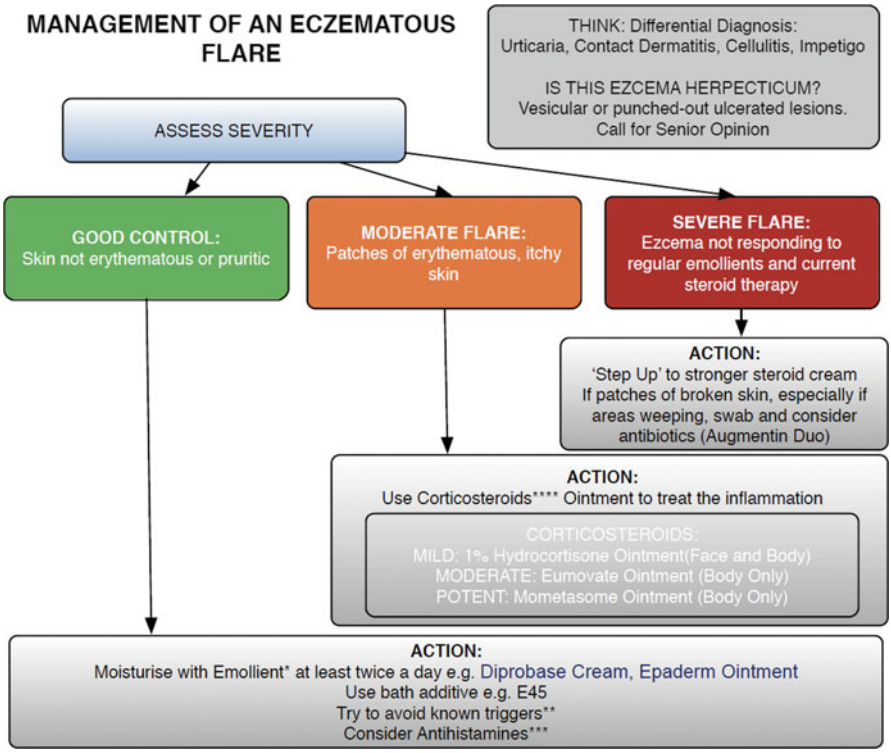
If the itching is severe enough to cause sleep disturbance and the child is over 6 months of age, then consider a 7–14-day trial of a sedating antihistamine. This can be repeated for subsequent flare-ups.

Mild flare or good control (skin not erythematic or pruritic): Moisturise with emollient at least twice a day, e.g. Diprobase Cream or Epaderm Ointment. Use a bath additive such as E45.

Moderate flare (patches of erythematous, itchy skin): Use corticosteroid ointment to treat the underlying inflammation in addition to the above. Consider antihistamines.

Severe flare (eczema not responding to regular emollients and current steroid therapy): 'Step up' to stronger steroid cream. Swab areas of weeping skin, and consider antibiotics such as Augmentin as secondary infection with *Staphylococcus aureus* may lead to the development of scalded skin syndrome. Add antihistamines to the regime. If eczema herpeticum (*herpes simplex* 1: causative agent) is a concern, get an opinion from a senior clinician, and antiviral such as acyclovir may be added to the above regime if the lesions are widespread or if the child is clinically unwell.

ED management of an eczematous flare



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Chapter 8
Gastrointestinal Emergencies

Shanthi Sangareddi

The most common gastrointestinal emergencies encountered in the emergency department are acute gastroenteritis and gastrointestinal bleeding, and these two entities are addressed in this chapter.

Section A: Acute Gastroenteritis

Key Points
- Diarrhoea is commonly caused by viral infections. Antibiotics are not needed for most diarrhoea.
- Oral rehydration therapy reduces mortality.
- Assessment of dehydration is vital. Some dehydration is managed with ORS and severe dehydration with intravenous fluids.
- Dysentery results from bacterial infections.

Introduction

Acute gastroenteritis (AGE) is a common emergency in children. It usually presents with vomiting and diarrhoea. The burden of diarrhoeal disease is very high all over the world. It is an important cause of mortality (9 % of all deaths in children less than 5 years of age) leading to 580,000 deaths per year globally [1]. In India more than 2.3 million children died in 2004 of which 334,000 are attributable to

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Table 8.1 Common causes of AGE

Viruses	Bacteria	Parasite
Rotavirus Caliciviruses Norwalk-like viruses Enteric adenoviruses	Enterotoxigenic *E. coli*, *Shigella* *Salmonella* *Yersinia enterocolitica* *Campylobacter jejuni* Cholera	*Entamoeba histolytica* *Giardia lamblia* Cryptosporidium

diarrhoeal diseases [2, 3]. 23 % of global deaths among children <5 years of age are due to rotaviral infections that occurred in India [4].

Diarrhoea is defined as the passage of three or more than three liquid or loose stools per day or more frequent passage than is normal for the individual [5]. If the duration of an episode of diarrhoea is less than 14 days, it is called acute diarrhoea.

Aetiology

Most diarrhoeal episodes occur due to contaminated water or food and spreads by the faeco-oral route. The common causes of AGE are given in Table 8.1 [6].

Systemic infection, urinary tract infection and viral hepatitis can also cause diarrhoea. Drug-induced diarrhoea is another common problem.

Pathophysiology

Enteropathogens produce diarrhoea in two ways. Some pathogens (cholera, enterotoxigenic *E. coli*, staphylococci) produce toxins and cause non-inflammatory diarrhoea. These toxins affect the small intestine. They usually cause secretory diarrhoea. The stool microscopy is usually normal.

Rotavirus invades the microvilli and destroys it reducing the absorptive area of the intestine. Other organisms (*Shigella* and *Salmonella*) invade the colonic mucosa and produce an inflammatory reaction resulting in dysentery.

History: The history should include:

- The duration of diarrhoea
- Number of stools per day
- Nature of the stool
- Presence of blood and mucus in the stools
- Vomiting
- Fever
- Thirst
- Urine output in the last 6 h
- If child is able to drink or eat
- Lethargy, irritability and unresponsiveness
- Measles in the past 3 months

Table 8.2 Features of dehydration as per WHO

Some dehydration (2 or more of the following signs should be present)	Severe dehydration (2 or more of the following signs should be present)
Irritable	Lethargy or unconsciousness
Sunken eyes	Sunken eyes
Skin pinch goes back slowly	Skin pinch goes back very slowly
Drinks eagerly	Drinks poorly or not able to drink

Vomiting without diarrhoea is not AGE. Other causes of vomiting should be ruled out.

Clinical examination: Helps to assess the hydration status of the child. Conventionally dehydration is classified as mild, moderate and severe based on the % of weight loss. In infants and young children, it corresponds to 5, 10 and 15 % and in older children to 3, 6 and 9 %.

World Health Organization (WHO) classifies dehydration as no dehydration, some dehydration and severe dehydration (Table 8.2) [7].

Hypovolemic shock due to AGE is identified by the presence of:

- Altered mental status
- Effortless tachypnoea
- Tachycardia
- Cold extremities
- Weak pulses
- Capillary refill time >3 s with normal blood pressure (compensated shock) or low blood pressure (hypotensive shock)

It is important to differentiate between severe dehydration and shock as the latter requires more aggressive therapy.

Complications of AGE

1. Hypovolemic shock
2. Haemolytic uraemic syndrome
3. Electrolyte imbalance: hyponatraemia, hypernatraemia and hypokalemia
4. Septic shock
5. Acute renal failure

Investigations: are not needed for most children with diarrhoea

Stool microscopic examination – RBCs and neutrophils are seen in dysentery. Entamoeba trophozoite is diagnostic of amoebiasis. Presence of giardia trophozoite or cyst indicates giardiasis.

Stool culture in infective colitis.

Hanging drop preparation for cholera.

Serum electrolytes, BUN and creatinine in children presenting with hypovolemic shock.

Blood culture if sepsis is suspected.

Management

The advent of oral rehydration salt (ORS) in the 1970s has revolutionized the management of diarrhoeal diseases and reduced the mortality significantly. Oral rehydration therapy (ORT) using a solution made from ORS is the cornerstone of treatment of diarrhoea.

The WHO recommendation is discussed below.

Diarrhoea with No Signs of Dehydration

Plan A

1. Continue feeding.
2. Give extra fluids.
3. Tell when to return.
4. Give zinc supplements (20 mg for >6 months of age; 10 mg for 2–6 months of age – once a day for 14 days).

Home available fluids like buttermilk, tender coconut water, plain water, lemon water or milk can be given. If child is exclusively breast-fed, give ORS and boiled and cooled water in addition to breast milk.

Additional ORS should be given after each loose stool (<2 years: 50–100 ml and >2 years: 100–200 ml).

Diarrhoea with Some Dehydration

Plan B

A child with some dehydration has a fluid deficit of 5–10 %. Treat this by giving oral rehydration solution 75 ml/Kg over 4 h. Give frequent small sips from a cup. If the child vomits, stop giving ORS for about 10 min. Then restart and give small quantity more slowly. If the child is breast-fed, continue to breast feed. After 4 h, child is reassessed for the degree of dehydration and treated accordingly. Child is started on feeds. Ongoing losses should be replaced by ORS 10 ml/kg per loose stool.

Diarrhoea with Severe Dehydration

Plan C

This represents >10 % fluid deficit. Child needs 100 ml/kg fluids. This is given intravenously as Ringer's lactate (preferred) or normal saline.

For children

<1 year – 30 ml/kg in 1 h[1] and 70 ml/kg in 5 h.
>1 year – 30 ml/kg in half an hour[1] and 70 ml/kg in two and a half hours.

Reassess the child every 30 min–1 h. After giving 100 ml/kg fluid, the degree of dehydration is reassessed and treated accordingly. Once the child's sensorium improves, she can take ORS (about 5 ml/kg/h), and breast milk can be started. Ongoing losses should be replaced.

If there are no facilities to start IV in a peripheral centre or if IV access is difficult, a nasogastric tube is introduced, and ORS is given at the rate of 20 ml/kg/h for 6 h and child is referred to a higher centre.

Hypovolemic shock: Shock treatment starts with opening the airway if child is in pain or unresponsive state and administering 100 % oxygen using a non-rebreathing mask. IV access is quickly established with a wide bore needle. If access is not obtained within three attempts or 90 s, an intra-osseous (IO) access should be obtained.

Compensated shock: The first bolus of 20 ml/kg of normal saline or Ringer's lactate is given over 20 min.

Hypotensive shock: The first bolus is given as fast as possible by a push-pull method using a three-way stopcock.

The child is reassessed. If shock persists, further 20 ml/kg fluid boluses are given. Most children will respond to two or three fluid boluses. Up to 80–100 ml/kg of fluids can be safely given in hypovolemic shock in the first 1–2 h. These patients tolerate fluids well and rarely need inotropes or intubation.

Capillary blood glucose estimation should be done. If there is hypoglycaemia, 5 ml/kg of 10 % dextrose should be administered to treat it. A maintenance fluid containing dextrose should be started separately.

Suspect Septic Shock in a Child with Diarrhoea

- If child does not respond to fluid boluses.
- Develops respiratory distress during fluid therapy.
- If there are no clinical signs of dehydration but child has signs of shock.
- If child has wide pulse pressure (warm shock). Hypovolemic shock is characterized by narrow pulse pressure (cold shock).

If sepsis is suspected, child may need inotropes and antibiotics in addition to fluid therapy.

Antibiotics are not routinely indicated for diarrhoea. They are indicated only in:

1. Cholera (a single dose of doxycycline is recommended- 2–4 years: 50 mg; 4–5 years: 100 mg)

[1] Repeat once if radial pulse is very weak or not detectable.

2. Severe acute malnutrition (SAM)
3. Severe dehydration in young infants <2 months.

Dysentery: Is passing blood in stools. It may be associated with mucus, tenesmus and fever. Patients may have anorexia and lose weight rapidly. Though dysentery is less common than watery diarrhoea, it causes more morbidity and mortality. About 10 % of all diarrhoeal episodes in children under 5 years old are dysentery, but these cause 15 % of all diarrhoeal deaths [7]. Severe dysentery occurs in malnourished children, non-breast-fed babies and those who had measles in the preceding month. It is quite often due to bacterial infections. *Shigella*, *E. coli* and *Campylobacter jejuni* are the organisms which commonly cause dysentery. *Clostridium difficile* can cause pseudomembranous colitis. *Shigella* infection can present with high fever, febrile convulsions, encephalopathy and septic shock. Amoebic dysentery is uncommon in young children.

Differential Diagnosis for Dysentery

1. Intussusception – These children will develop intestinal obstruction and gangrene of the bowel if not identified early.
2. Polyps

Children with dysentery should be admitted:

- If they have signs of dehydration
- If they are <2 months old
- SAM children
- Those who are toxic and have abdominal distension and tenderness

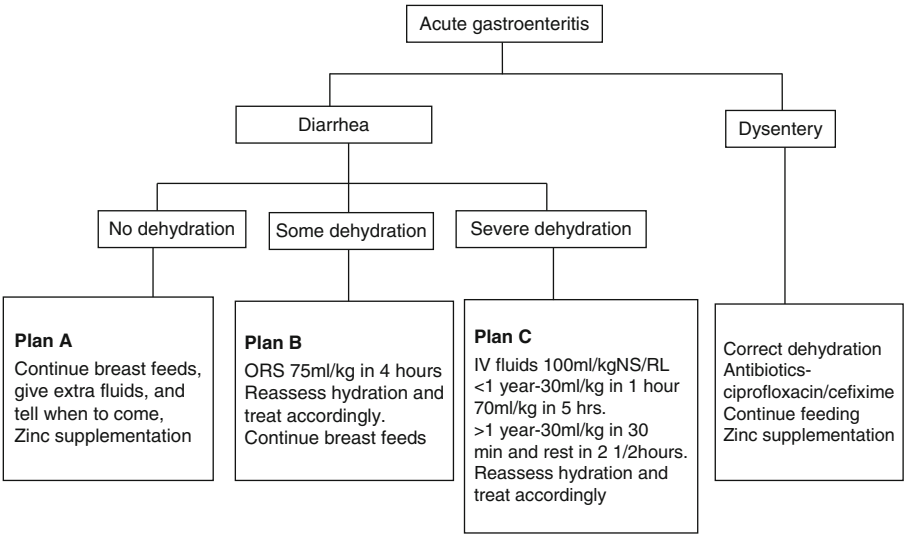
Treatment for Dysentery

1. Fluids: Fluids/ORS should be given as appropriate for their hydration status.
2. Antibiotics

   In the outpatient setting, the first-line drug is oral ciprofloxacin 15 mg/kg two times daily for 3 days [8]. If there is no improvement in 2 days, oral cefixime is given for 5 days [9].

   In hospitalized children, IV/IM ceftriaxone 100 mg/kg once daily for 5 days is given [9].
3. Continue feeding.
4. Give zinc supplementation.

Approach to gastroenteritis



Section B: Gastrointestinal Bleed

Key Points
- UGIB presents as haematemesis or melaena.
- LGIB presents as haematochezia.
- Care of airway, breathing and circulation is the priority in haemodynamically unstable children.
- Identification as variceal or non-variceal bleeding is important as treatment is different.

Introduction

Gastrointestinal bleeding (GIB) is a common medical emergency. It is classified as upper gastrointestinal bleeding (UGIB) and lower gastrointestinal bleeding (LGIB) based on whether the bleeding is originating above or below the Treitz ligament.

UGIB presents as haematemesis (bright red/coffee coloured) or melaena (dark red/black/tarry stool with offensive odour). The LGIB presents as haematochezia (bright red blood per rectum) or sometimes as maroon-coloured stool. “Red currant jelly” stool is seen in intussusception and Meckel’s diverticulum. All red-/black-coloured stools are not due to bleeds. Ingestion of beet root, tomatoes and food colouring agents can result in red stool. Black stool can be a result of ingestion of iron, blue berries, charcoal, bismuth salts and lithium. Hence, a stool test for occult

Table 8.3 Causes of gastrointestinal bleed

	Infants	Children
Upper GIB	Swallowed maternal blood	Gastritis and gastric ulcer
	Late haemorrhagic disease of the newborn	Dengue
	Sepsis with disseminated intra vascular coagulation (DIC)	Sepsis with DIC
	Stress bleed	Stress bleed
	Gastritis and gastric ulcer	Fulminant hepatic failure
	Oesophagitis	Cirrhosis with portal hypertension
	Bleeding diathesis	Extrahepatic portal vein obstruction
	Drug induced	Mallory-Weiss syndrome
		Drug induced
Lower GIB	Intussusception	Infectious colitis
	Meckel's diverticulum	Polyps
	Cow's milk protein allergy	Anal fissure
	Infectious colitis	Inflammatory bowel disorder
	Hirschsprung's colitis	Meckel's diverticulum
	Necrotising enterocolitis	Henoch-Schonlein purpura
	Bleeding diathesis	Bleeding diathesis
	Vascular malformation	Vascular malformation
	Anal fissure	Haemorrhoids

blood is indicated to confirm blood in stool. Young infants can swallow maternal blood or blood from nasopharyngeal trauma or epistaxis leading to a positive guaiac test in stool. History will help differentiate these causes.

The causes of gastrointestinal bleed vary with age and are depicted in Table 8.3.

An Approach to GIB

Ask these questions:

(A) Is the child haemodynamically stable?
(B) What is the type of bleeding – UGIB or LGIB?
(C) What is the aetiology of bleeding and the possible site of bleeding?

Is the Child Haemodynamically Stable? If No, Start Resuscitation

- First assess the physiologic status of the patient by doing a cardiopulmonary cerebral assessment. Irrespective of the aetiology, if the physiologic status is abnormal as evident by shock, intervene.

- Position the airway of the child and administer 100 % oxygen using a non-rebreathing mask. Intubation may be needed if child is hypoventilating or to protect the airway in a child with Glasgow coma scale <8.
- Get intravascular access with a wide bore needle and take blood for investigations, group and crossmatch. Two lines are needed.
- Give 20 ml/kg of NS or RL over 20 min for compensated shock and by 'push and pull' method for hypotensive shock till the BP improves. Reassess and repeat fluid boluses. If there is no improvement with 40–60 ml/kg, 10 ml/kg of packed RBCS should be given. In exsanguinating bleed, fresh whole blood may be given. Fresh frozen plasma (FFP) should be administered in patients who have received more than 1.5 times circulating blood volume, even if there is no bleeding [10]. Platelet transfusion may be required in an actively bleeding child if platelet count is <50,000 [11]. Warming the blood prior to transfusion will help prevent hypothermia.
- A nasogastric tube is introduced. This helps to identify the type of bleeding, ongoing bleeding, to empty the stomach and to facilitate endoscopy if needed.
- Intravenous erythromycin is administered before EGD at a dose of 5 mg/kg in children to help to empty the stomach contents [12]. Erythromycin is useful in bleeding ulcer and in bleeding associated with portal hypertension [13, 14].
- Child should be kept nil by mouth.
- A Foley catheter is introduced to monitor urine output.

Determine the Type of Bleeding: Upper GIB or Lower GIB?

- UGIB presents as haematemesis or melaena, rarely as haematochezia if bleeding is massive.
- A bloody nasogastric aspirate or coffee-coloured blood confirms upper GIB [15]. However, a non-bloody aspirate may be seen in 25 % of UGIB.
- Blood urea nitrogen (BUN) may be increased in UGIB as blood proteins are broken down to urea by intestinal bacteria and is absorbed. A highly elevated BUN: Creatinine may indicate UGIB but not specific as it may also reflect prerenal azotaemia secondary to volume depletion.

Find Out the Aetiology and Possible Site of Bleeding

A thorough history and clinical examination will help arrive at the possible cause of bleeding. Investigations are needed to confirm the site of bleeding. Table 8.4 lists the history and the possible aetiology.

Sexual abuse may present with rectal bleed. It should be considered if unusual features in the history or examination are present.

Table 8.4 History and possible aetiology

History	Possible aetiology
Retching	Mallory-Weiss
Abdominal pain	Peptic ulcer, gastritis, Henoch-Schonlein purpura, infectious colitis, dengue
Jaundice	Decompensated liver disease
Epistaxis	Swallowed blood
Drug intake (aspirin, NSAIDS, steroids, anticoagulants)	Peptic ulcer, gastritis
Fever	Infectious colitis, dengue,
Umbilical sepsis or catheterization in the newborn period	Extrahepatic portal venous obstruction (EHPO)
Volume of bleeding, dizziness, fatigue, syncope	Severity of bleed
Any other bleeding tendency like purpura, petechiae, bleeding from venipuncture sites	Thrombocytopenia, DIC
Bleeding following injections, minor trauma, shedding of teeth, spontaneous bleeds	Bleeding diathesis
Past history	Peptic ulcer, liver disease, gastro-oesophageal reflux disease, EHPO
Family history	Bleeding diathesis, hereditary haemorrhagic telangiectasia

Clinical Examination

Table 8.5 shows the important clinical signs that should be looked for in a child with GI bleeding.

Rectal examination is mandatory in all patients. It helps to diagnose polyps, intussusception and any mass. Inspect the perianal area for fissures, fistulas, skin breakdown or evidence of trauma. Inspection of the vomitus and stool is important. If vomitus is coffee coloured, active bleed is unlikely. It often indicates that the patient has had a recent bleeding. If the stool is red or maroon or melaena, it usually indicates active bleeding.

Investigations

The following investigations are useful to arrive at a diagnosis and for management of these patients.

Complete blood count may show leucocytosis as a stress response; haematocrit is usually normal soon after a bleed. A fall is seen only after 48–72 h. Stool microscopy will show pus cells in colitis. Stool culture may be indicated in infectious colitis. A guaiac test in stool or vomitus should be done to confirm bleed. Blood urea nitrogen (BUN) and creatinine, liver function tests, prothrombin time, partial thromboplastin time, fibrinogen, D-dimer, electrolytes, blood grouping and typing are helpful in management.

Table 8.5 Clinical signs in a child with GI bleed

Vitals, cardiopulmonary cerebral assessment
Orthostatic hypotension – a fall in systolic BP>20 mm of Hg or of diastolic BP>10 mmHg on standing
Signs of shock
Anaemia, pallor
Clubbing-inflammatory bowel disorders
Jaundice, palmar erythema, asterixis, foetor hepaticus
Purpura, petechiae, gum bleeds
Nasopharyngeal and oropharyngeal examination
Pigmentation of the lips, Peutz-Jeghers syndrome
Telangiectasia of the skin, mucus membranes and lip (Osler-Weber-Rendu disease)
Splenomegaly – portal hypertension, may be absent in a child with EHPO when there is active bleeding
Hepatosplenomegaly, prominent veins over the abdomen, ascites, caput medusae – cirrhosis with portal hypertension
Epigastric tenderness-gastritis, peptic ulcer
Abdominal mass, intussusception
Foci of sepsis

Imaging Studies

Chest x-ray may show right-sided pleural effusion in dengue. Plain abdominal x-ray can reveal pneumatosis intestinalis and portal air in necrotising enterocolitis.

Barium contrast studies (barium swallows, upper GI series, small bowel follow-throughs or barium enemas) may detect oesophagitis, IBD or polyps. They are contraindicated if perforation is suspected.

Ultrasonogram of the abdomen can identify ascites and target sign in intussusception.

Endoscopy

1. Oesophagogastroduodenoscopy (OGD) is the investigation of choice for UGIB. OGD can detect the source of bleeding in 90 % of the children if done within 24 h [12]. However endoscopy may have to be done as an emergency procedure soon after resuscitation if bleeding cannot be arrested by other measures.
2. Colonoscopy is useful in LGIB. It can detect polyps, ulcerative colitis and diverticulum. A good bowel preparation with polyethylene glycol lavage solution by mouth or nasogastric tube is essential.
3. Push enteroscopy and double-balloon enteroscopy are useful if bleeding cannot be identified by EGD.

Special studies for LGIB include contrast CT scan of the abdomen, radiolabeled red cell scan (can detect bleeding as low as 0.1 ml/min), Meckel's scan, arteriography (can only detect bleeding >0.5 ml/min) and capsule endoscopy.

Explorative laparotomy/laparoscopy may be needed in severe cases.

Management

Stabilization of airway, breathing and circulation is the priority as mentioned earlier.

Non-variceal Bleeding

Stop the offending drugs.

Proton pump inhibitors (PPI) are the drugs of choice for non-variceal GIB. Loading dose of 2 mg/kg IV followed by 0.2 mg/kg/h for 72 h [16]. H2 receptor antagonists such as IV ranitidine 1 mg/kg/dose q6h can also be used [17]. Patients with ulcer bleeding should not be treated with H_2 receptor antagonists [12]. Antacids are given at the dose of 1 ml/kg/dose q2–4 h. Sucralfate has been found to be useful in preventing stress bleeds. It forms a protective coat on the gastric mucosa. The dose is 1 g/1.73 m^2 given orally [17]. It is not given during acute bleeding.

Treatment of the cause is important. Helicobacter pylori infection should be suspected in patients who have GIB of unknown aetiology and should be treated with triple drugs twice a day for 1–2 weeks. A combination of PPI with amoxicillin and metronidazole or clarithromycin is given. Vitamin K is administered if there is coagulopathy and in late haemorrhagic disease of the newborn. It arrests bleeding within 2 h in the latter disease. DIC may need transfusion of blood products like cryoprecipitate, fresh frozen plasma and packed RBC.

If peptic ulcer bleeding does not resolve with medical management endoscopic electrocautery, injection of epinephrine, embolization of blood vessels, fibrin sealants and endoclips or surgery may be needed. Biopsy is taken, if warranted.

Variceal Bleeding

Resuscitation is done as mentioned above. In variceal bleeding, too aggressive volume expansion can result in a rebleeding [18]. A restricted transfusion strategy (transfusion when the haemoglobin level below 7 g per deciliter) is recommended for acute UGIB [19].

Medical Management

Octreotide-a somatostatin analogue acts by splanchnic vasoconstriction. The dose is 1–2 μg/kg IV bolus followed by 1–2 μg/kg/h as infusion [20]. The efficacy of octreotide was equivalent to terlipressin [21]. Vasopressin IV 0.01 U/kg/min has also been used. However, side effects like ischaemia to the bowel, heart and kidneys limit its use.

Antibiotic prophylaxis with third-generation cephalosporin or with fluoroquinolone for 5–7 days should be given to any cirrhotic patient with GI bleeding. It decreases mortality and the probability of rebleeding [12]. In children, PPI therapy should probably be initiated in case of oesophageal/gastric varices rupture [12] as gastritis is frequently associated with portal hypertension [22]. Fresh frozen plasma or factor VIIa is probably not indicated in GI bleeding in patients with cirrhosis [12].

Balloon tamponade is tried if emergency endoscopy is not possible. The Sengstaken-Blakemore tube has three ports. It is introduced into the stomach, and the gastric balloon is inflated. The tube is then withdrawn till there is resistance which indicates that the tube is in the gastro-oesophageal junction. The oesophageal balloon is then inflated. The position of the balloon should be checked radiographically. Traction (using a 0.5 kg weight or a 500 mL plastic bag of IV fluid) is maintained via a pulley system above the patient's head. For small children, a large adult size Foley catheter 12 FG (with a 30 cc balloon) may be used [23]. It may be safer to intubate the children prior to balloon tamponade.

Self-expandable metal stents (SEMS) are used as an alternative to balloon tamponade. It is inserted over an endoscopic placed guide wire using a stent delivery device [24].

Endoscopy: Intubate patients with massive bleeding and/low GCS prior to endoscopy. Variceal band ligation (VBL) is superior to endoscopic sclerotherapy (EST). Injection of cyanoacrylate glue is used for gastric varices.

Trans jugular intra hepatic portosystemic shunt (TIPS) by an interventional radiologist is another option. An intrahepatic metallic stent is placed between the intrahepatic portions of the portal vein and the hepatic vein, creating a nonselective portocaval shunt.

Shunt surgery or gastric transection is the final option if other measures fail.

Primary Prophylaxis and Secondary Prophylaxis for Variceal Bleed

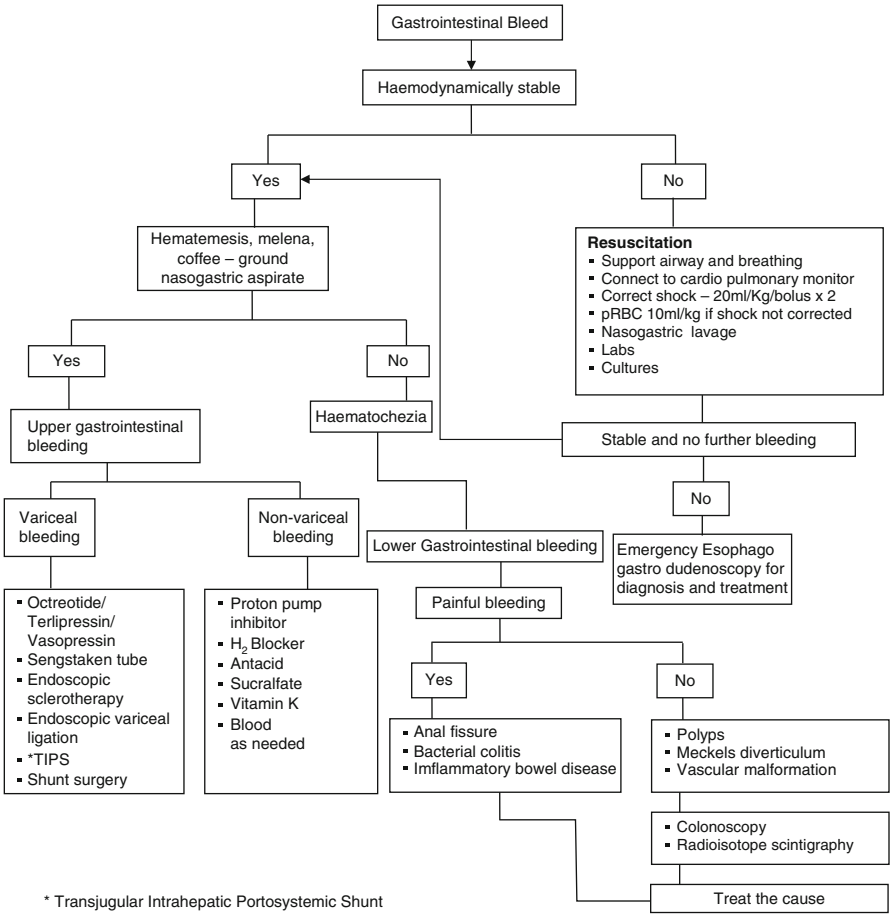
Propranolol, VBL and EST are useful in that order.

Lower Gastrointestinal Bleeding

Most bleeds stop spontaneously. If low or intermittent bleed, do an urgent colonoscopy preferably within 8 h of presentation. Approximately 10 % of cases of massive haematochezia are related to UGIB, justifying EGD before colonoscopy [25]. An emergency scintigraphy to search for a Meckel's diverticulum and/or a surgical exploration should be undertaken if GI endoscopic findings are normal or haemodynamic consequences persist.

Colonoscopic treatment includes injection therapy (epinephrine or saline), electrocoagulation, haemoclips and band ligation. Polyps are resected using sigmoidoscopy. Surgery is needed for Meckel's diverticulum. Infective colitis is treated with appropriate antibiotics. HUS may need supportive therapy and renal replacement therapy. Inflammatory bowel disorders are treated with steroids. Vascular malformations may need arterial embolization.

Approach to gastrointestinal bleed



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Chapter 9
Haematology and Oncology: Common ED Presentations

Dwynwen M. Roberts and N. Udayakumar

Key Points
- A new diagnosis of a previously undiagnosed haematological or oncological condition may occur in the ED.
- Emergency physicians need to possess basic understanding of common haemato-oncological conditions that could present to the ED in order to apply a systematic approach in managing them.

Haematology

Introduction

Children with haematological conditions are very likely to present to the emergency department (ED). Presentation either can at first be an innocuous sign or symptom but after full blood count could reveal serious diagnosis or as a consequence of underlying haematological condition like sickle cell disease.

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The Bleeding or Bruising Child

Bleeding or bruising excessively is a relatively common presentation. In many children, no pathological cause is found. On occasion it may imply serious underlying illness. Symptoms vary from a non-blanching rash due to petechiae or purpura, bruising without an obvious cause, mucous membrane bleeds or excessive bleeding following minor procedures like dental extraction to more significant symptoms such as joint or limb pain and swelling from haemarthrosis or muscle haematomas, respectively.

Clinical Features and Assessment

History

Careful history taking is very important, particularly the duration of bleeding or bruising. History of months or years suggests a congenital or inherited condition whereas shorter history of days is more likely to be an acquired condition. Constitutional symptoms such as lethargy, anorexia or weight loss may point to bone marrow failure [1]. A history of preceding infection could point towards acute leukaemia or immune thrombocytopenia. Presence of abdominal pain could suggest Henoch-Schonlein purpura [1].

Family History

An inherited disorder such as clotting abnormalities is familial [2].

Medication

Prescribed (or otherwise) medications may lead to excessive bleeding or aplastic anaemia like valproic acid [1].

Examination

- In general examination, look for common sites of infection such as chest, ear, nose and throat. Document the site, number and severity of any bruises or petechiae. Look for bleeds particularly gingival membrane.
- Examine the abdomen for organomegaly.
- Examine for lymphadenopathy.

Investigations

All children will need a full blood count and a coagulation profile [1]. Other investigations will be based on the clinical condition of the child, the suspected differential diagnosis and following the result of the initial blood count and coagulation screen.

Non-blanching Rash

Due to national campaigns on the dangers of meningitis and meningococcal sepsis promoting the 'tumbler test', many children will present to the ED with a non-blanching rash.

A non-blanching rash is defined as a rash which does not fade on pressure. The rash may consist of petechiae (spots <3 mm diameter) or purpura (spots >3 mm diameter). Although some will have severe illness, most will have a benign cause. However, it is important to be proactive in assessing these children so as not to miss a serious underlying clinical condition.

Prompt assessment should be done. If signs of septicaemia, shock or meningitis are present, the child should be treated for meningococcal disease, and treatment should involve senior clinicians.

If in doubt, TREAT! If not, then the differential diagnosis for non-blanching rash should be considered (Table 9.1). A full history and examination should be undertaken.

Immune Thrombocytopenia

It is a common presentation in childhood. A platelet count of less than 20×10^9/L is present with a normal white blood cell count and haemoglobin [1].

Table 9.1 Differential diagnosis for non-blanching rash

Meningococcal or other septicaemia
Viral illness
Immunisation reaction
Mechanical causes such as cough or vomiting – causes petechial spots in the distribution of the superior vena cava
Direct pressure or trauma
Henoch-Schonlein purpura
Haemato-oncological causes of non-blanching rash such as idiopathic/immune thrombocytopenia and acute leukaemia

Treatment for non-blanching rash is directed towards the underlying condition

Pathogenesis

Autoantibodies to platelets are formed causing destruction and their removal from the circulation [1].

Clinical Features

It is characterised by sudden onset of petechiae or purpuric spots or bruising. The rash is often noticed on the legs, but can be widespread. It is often preceded by a viral illness [1]. Rarely, a child will present with severe bleeding or intracranial haemorrhage.

Investigations

A full blood count will show isolated thrombocytopenia [3]. Clotting screen is normal. Blood film may show giant platelets.

Treatment

Treatment is often reassurance with supportive treatment for any underlying/ongoing viral illness [1]. Repeat the full blood count within a few days to ensure no new features.

Prognosis

Virtually, all will resolve within a few weeks. Before discharge, parental advice should be provided to keep a watchful eye on their child to prevent injuries (particularly head injuries) from sporting or leisure activities.

Acute Leukaemia

One of the most common forms of cancer in children is acute leukaemia and accounts for approximately 30 % of childhood cancers [4], often diagnosed in the ED whilst the child is being assessed for unexplained bruising or bleeding or a non-blanching rash. Acute lymphoblastic leukaemia is the commonest (five times more common than acute myeloid leukaemia) [4].

Acute Lymphoblastic Leukaemia

The commonest childhood leukaemia which has now a survival rate of >85 % [1, 4].

Pathogenesis

Most occur spontaneously. Immunoglobulin and T-cell receptor gene rearrangement in lymphoid precursor cells [1].

Clinical Findings

Most symptoms are non-specific such as fever and fatigue. There may be bruising, non-blanching/purpuric rash or bleeding [1, 4]. Lymphadenopathy may be present. Bone pain is common. Other symptoms include headache or painless testicular enlargement. Other rare symptoms are confusion due to leukostasis from an extremely elevated white blood cell count or visual disturbance due to retinal infiltration or haemorrhage [1, 4].

Investigations

A full blood count should be done. If this is abnormal, a blood film will be done. On the blood film, lymphoblasts are present and a normal or depressed white blood cell count. Most will be anaemic and thrombocytopenic.

Differential Diagnosis

1. Other leukaemias
2. Aplastic anaemia
3. Non-Hodgkin's lymphoma

Treatment

The local paediatric oncology centre should be contacted immediately to discuss further investigation and management. Treatment is based on multidrug chemotherapy using drugs such as steroids, methotrexate, vincristine, cyclophosphamide and cytarabine, initially 6–12 months of intensive blocks of chemotherapy followed by 1–2 years of low-intensity maintenance. Most children are treated according to national treatment protocols [1].

Acute Myeloid Leukaemia (AML)

Serious form of childhood leukaemia.

Pathogenesis

The cause is unknown. However, there may be environmental or hereditary factors associated with the development of AML. There is abnormal proliferation of myeloid precursor cells.

Clinical Findings

Presenting signs and symptoms are very similar to ALL.

Investigations

The full blood count will show an extremely elevated white blood cell count, which may be in excess of 200×10^9/L.

Treatment

Immediate referral to the local paediatric oncology centre is recommended. There is a risk of leukostasis with extremely high white blood cell count which is treated by commencing anti-leukaemic treatment as soon as possible. Patients may need platelet transfusion to avoid cerebral haemorrhage.

Chemotherapy is commenced as soon as possible and as intensively as possible. Haemopoietic stem cell transplant may be used in some patients. Survival has much improved, but still lags behind ALL.

Benign Haematological Conditions

Children will also present to the ED with consequences of their underlying haematological condition such as certain haemoglobinopathies or with a new discrete haematological illness such as venous thromboembolic disease which is a rare but important condition.

Sickle Cell Disease

One of the most commonly encountered haemoglobinopathies particularly in the UK and other parts of the world with significant African population is sickle cell disease (SCD) which is a genetic illness with structural abnormality of haemoglobin.

Pathogenesis

A haemoglobin variant – haemoglobin S (HbS) – is produced. This is due to substitution of valine for glutamic acid as the sixth amino acid in the beta globin chain of haemoglobin [5].

In SCD the HbS gene is inherited from both parents, and the child therefore exhibits symptoms and signs of the disease [1]. In sickle cell trait, child inherits HbS from one parent and HBA from other parent and is a carrier [1].

Clinical Findings

Children will present to the ED with symptoms related to complications of their illness. Red cells containing HbS deform or sickle when deoxygenated, causing vaso-occlusive crisis [1]. Episodes of acute pain localised in bone or soft tissue are the most common type of vaso-occlusive crisis. Such crises are often precipitated by an intercurrent illness, dehydration or exposure to cold [1, 5]. Occasionally, they can present with chest crisis due to pulmonary sickling (acute chest syndrome) often characterised by chest pain, respiratory distress and hypoxaemia [1, 5]. Examination of these children should look at precipitating factors like infections. Chest x-ray will show new infiltrates.

If a sickle cell patient presents with collapse and hypotension, consider splenic sequestration. Whilst rare, patients do present to the ED with this condition, which usually occurs in early childhood. Children are profoundly anaemic [1]. There is massive splenic enlargement.

Other complications that may occur in a child with SCD are stroke, mesenteric infarction and priapism [5].

Treatment

Provide immediate pain relief (Table 9.2). Oral analgesia such as paracetamol and NSAIDS is used regularly. However, patients will need adequate doses of intravenous opioids such as morphine. This is often administered via patient-controlled devices. Many emergency departments will have patient-specific guidelines for pain relief as well as access to acute pain services.

Table 9.2 Analgesia guideline

Mild pain	Paracetamol 20 mg/kg 4–6 hourly	Ibuprofen <3 months 5 mg/kg 8 hourly	
Moderate pain	Paracetamol and ibuprofen as above	Dihydrocodeine <4 years 0.5 mg/kg >4 years 1 mg/kg	Oral morphine 1–12 months 0.1–0.2 mg/kg >1 year 0.2–0.4 mg/kg
Severe pain	Rx as for mild and moderate pain	Intranasal fentanyl as per protocol	Intravenous morphine 0.1–0.2 mg/kg

Patients will need to be adequately hydrated and oral fluids be encouraged. However, if this is not possible, then intravenous fluids will be required. Antibiotics should be considered if there is fever and/or signs of infection. Oxygen if there are respiratory symptoms or low saturations. The haemoglobin concentration may drop by 1–2 g/dL during a crisis. However, top-up transfusions are rarely required [1].

Treatment of acute chest syndrome is aimed as relieving pain. However, antibiotics are usually required. On occasion, exchange transfusion is preformed to reduce the percentage of sickling HbS in the blood.

These patients are often seriously unwell and support from haematologist and PICU team will be required [1, 5].

Blood transfusion will be required and is life-saving in splenic sequestration.

Principles of treatment are the same in a child with stroke, mesenteric infarction and priapism, i.e. pain relief, treatment of precipitating factors, adequate fluids, expert help from haematologists and in severe and serious cases exchange transfusion.

Prognosis

Symptoms usually settle over a few days and discharge can be planned once pain is controlled with simple analgesia.

Many patients live with an element of pain and therefore recognise their symptoms well and can often manage their pain control at home.

Venous Thrombosis and Thromboembolism

Now, children are increasingly presenting to the ED with signs and symptoms of VTE, and hence, emergency physicians should be familiar with this condition. It occurs often as a consequence of improved treatment of conditions that previously had a poor outcome [1]. Occasionally, completely well children can present with VTE.

One of the commonest risk factors is an indwelling central venous access device used in the treatment of malignancies. Other risk factors include systemic lupus erythematosus, sickle cell disease, malignancy, trauma, oral contraceptive pill use, infection and thrombophilia. Idiopathic thrombosis is rare in children.

Pathogenesis

Hypercoagulability, endothelial injury and stasis leading to Virchow's triad are the processes that lead to VTE [6]. Predisposing factors as described above will promote this action.

Clinical Findings

Clinical features are often related to the location of the thrombosis/embolism. Hot, swollen, painful limb (upper or lower) suggests deep venous thrombosis (DVT) [1, 6]. Pleuritic chest pain, shortness of breath, elevated heart rate, hypoxia and occasionally haemoptysis will indicate pulmonary embolism (PE) [1, 6]. Headache, focal neurological signs such as limb weakness and seizures are signs of cavernous sinus venous thrombosis (CSVT) [1, 6].

Investigations

Aimed at the presumed diagnosis – Doppler ultrasound for DVT, CT pulmonary angiogram for PE, CT or MRI venogram for CSVT. D-dimer may be useful if negative. However, this must be used in conjunction with risk-assessment strategies. If D-dimer is negative, but a high index of clinical suspicion remains, then other investigations must be done to confirm or exclude the diagnosis of VTE.

Treatment

Symptomatic relief of symptoms – pain relief, oxygen if hypoxic, control of seizures. Remove central venous access devices once other venous access is obtained [1, 6].

Anticoagulation Therapy

Low molecular weight heparin is preferred as parents can be taught to administer. Warfarin is difficult to use in children due to variable dosages with age and weight. Duration of treatment varies from 6 weeks to 3 months, but local guidelines/haematologist should be consulted. In life-threatening VTE such as PE, thrombolysis treatment can be considered and may be life-saving.

Prognosis

Most patients will recover without any complication. A small proportion will go on to develop pro-thrombotic syndrome which is a chronic complication of VTE causing symptoms that vary from mild oedema of the limb to chronic pain and ulceration due to chronic venous insufficiency [6]. Recurrence is rare.

Prophylaxis

Is used in some clinical settings like underlying malignancy or known thrombophilia who are at high risk [1]. Low molecular heparin such as tinzaparin subcutaneously at a dose of 50 unit/kg daily is recommended.

Oncological Emergencies

Introduction

Children will present to the ED with complications of treatment of their malignancy. Most hospitals will have local treatment guidelines for these conditions. Emergency physicians should be familiar with the commonest cancer-related emergencies.

Oncological emergencies can be defined as acute conditions that are caused by cancer or its treatment, which require rapid intervention to avoid subsequent mortality and morbidity. Table 9.3 provides a summary of the different oncological emergencies that can present to an emergency department.

Tumour Lysis Syndrome

One of the most serious and potentially life-threatening oncological emergencies in children is tumour lysis syndrome (TLS). It causes metabolic derangements due to rapid cancer cell necrosis [1]. It mainly occurs in haematological cancers but can also happen in solid tumours.

Pathogenesis

Very large amounts of intracellular contents are released into the bloodstream. This leads to hyperkalaemia, hyperuricaemia and hyperphosphataemia with secondary hypocalcaemia [7]. Eventually, this will cause urate nephropathy and acute renal failure.

Table 9.3 Summary of oncological emergencies

Emergency	Associated cancer or cause	Signs and symptoms
Metabolic		
Tumour lysis syndrome	Haematologic malignancies; cancers with rapidly growing tumours, particularly acute leukaemias and high-grade lymphomas	Azotaemia, acidosis, hyperphosphataemia, hyperkalaemia, acute renal failure, hypocalcaemia
Haematologic		
Febrile neutropenia	Chemotherapy-associated bacterial or fungal infections	Temperature greater than 101 °F (38.3 °C), absolute neutrophil count less than 500 per mm^3 (0.5×10^9 per L)
Hyperviscosity syndrome	Waldenström's macroglobulinaemia, multiple myeloma, leukaemia	Spontaneous bleeding, 'sausage-like' haemorrhagic retinal veins, neurologic defects, serum viscosity levels greater than 5 cP
Structural		
Superior vena cava syndrome	Lung cancer, metastatic mediastinal tumours, lymphoma, indwelling venous catheters	Cough; dyspnoea; dysphagia; head, neck or upper extremity swelling or discoloration; development of collateral venous circulation
Side effects from treatment agents		
Diarrhoea	Chemotherapy	Dehydration, poor skin turgor, dry mucous membranes, weight loss
Extravasations	Current chemotherapy infusion	Pain and erythema at infusion site, swelling, necrosis, contractures
Obstipation	Narcotic medications, chemotherapy (specifically neurotoxic agents)	Abdominal pain, constipation, hard stools every 3–5 days

Adapted with permission [10]

TLS may occur due to the treatment of the malignancy such as cytotoxic therapy or radiotherapy. It can also occur following the administration of steroids for another reason or spontaneously such as after surgery. Other predisposing factors are decreased urine output, dehydration or pre-existing renal failure [1, 7]. Children with certain malignancies, e.g. acute lymphoblastic leukaemia and B- and T-cell non-Hodgkin's lymphoma, are more at risk of developing TLS than others.

Clinical Features

Symptoms associated with the severe metabolic derangement usually occur within 5 days of starting chemotherapy. Symptoms maybe non-specific such as GI upset and lethargy or those that may more directly be attributable to the metabolic abnormalities such as cardiac arrhythmias and failure, muscle cramps, tetany and seizures [1, 7, 9].

Diagnosis

There is no specific criterion to define TLS. However, it is recognised to be the metabolic consequences of the treatment of certain malignancies. Not all the biochemical components are initially present, and early treatment will prevent progression to full-blown TLS [8]. Patients who develop TLS will begin to show biochemical abnormalities within 4–6 h of starting appropriate treatment for the malignancy.

Investigations

Renal function, bone profile, uric acid, ECG

Treatment

Hyperhydration: isotonic saline up to 3 l/m^2/day and can be increased up to 4 l/m^2/day if no signs of fluid overload. Caution with hyperhydration in children with very low Hb and high WBC count as they are at a risk of haemodilution and dropping Hb further.

Allopurinol (100 mg/^{m2} PO 8 hourly) or urate oxidase (rasburicase, 0.2 mg/kg IV OD) for elevated uric acid could be required [8, 9].

Monitor trend of electrolytes regularly. Treat potassium, if over 5.5 mmol/l, with standard hyperkalaemia treatment.

Haemofiltration may be required. It is important that these patients are discussed with their oncology centre early.

Superior Vena Cava Obstruction (SVCO)

SVCO is defined as a clinical picture of compression or complete obstruction of the superior vena cava by a mediastinal mass. It is a haemato-oncological emergency [1].

Causes

Anterior mediastinal mass secondary to lymphoblastic lymphoma or non-Hodgkin's lymphoma is the commonest cause.

Clinical Findings

The clinical presentation does not reflect the degree of obstruction. Patients may present with respiratory, neurological or cardiovascular symptoms or signs. Respiratory symptoms include chest pain, cough, shortness of breath, inability to lie flat and stridor. Clinically, the patient may be tachypnoeic, cyanotic, wheezy and hypoxic and have diminished breath sounds on auscultation. Cardiovascular system examination may show facial oedema, distended veins over the neck and chest as well as hypertension.

There could be neurological symptoms such as headache and dizziness. Occasionally, they may appear confused or anxious, due to raised intracranial pressure or cerebral oedema which will be manifested by papilloedema. The child may also experience seizures or syncope [1]. Airway obstruction may occur in up to 60 % of patients.

Management

1. Avoid distressing the child, i.e. intravenous access and obtaining blood for diagnostic tests must be done with as little fuss as possible [9].
2. Avoid general anaesthesia or sedation as much as possible and closely monitor the vital signs [9].
3. Avoid inappropriate or unnecessary investigations. These may worsen respiratory compromise and ultimately lead to clinical deterioration and cardiorespiratory arrest [1].

A chest x-ray will be required to identify the size of the mass and associated pleural effusion which needs to be drained.

Immediate review is needed by a senior anaesthetist due to the high risk of airway compromise [8]. If intubation is required for life-threatening obstruction, then an ENT surgeon must be present along with equipment to manage difficult and surgical airway [8]. The patient will need to be transferred to an oncological centre for further treatment and assessment as soon as possible.

Treatment

Treatment is directed at the underlying cause which may be chemotherapy or radiotherapy. Steroids should be initially avoided to prevent precipitating TLS unless advised by the child's oncologist [1].

Prognosis

Whilst superior vena cava compression and any accompanying airway obstruction are potentially life threatening, the underlying causative malignancies have a good outcome and prognosis [1].

Febrile Neutropenia or Neutropenic Sepsis

It is defined as a temperature of higher or equal to 38 °C (OR), other signs or symptoms consistent with clinically significant sepsis, in patients having anticancer treatment who have a neutrophil count of 0.5×10^9/L or lower. Empirical antibiotics as guided by local antibiotic policies should be given immediately if febrile neutropenia/sepsis is suspected in all ill patients even if they do not strictly fulfil the defining criteria of febrile neutropenia.

Presentation

Typically 5–10 days after last chemotherapy when the trough of neutrophil count is reached [9]. Children who have severe neutropenia may be afebrile [9]. They are often systemically unwell with warm peripheries and low blood pressure [9].

Management

The patient must be examined systematically looking for signs of infection. This includes examining central venous access devices for exit or tunnel infection and the mouth for mucositis. In addition as a minimum, ask about the perineal area [1, 9]. Infection screen should be carried out (culture blood and indwelling catheters, chest x-ray and consider other potential sources such as urine or stool based on history and examination) [8]. Send urgently full blood count, urea and electrolytes, liver function tests, clotting screen, C-reactive protein and serum lactate.

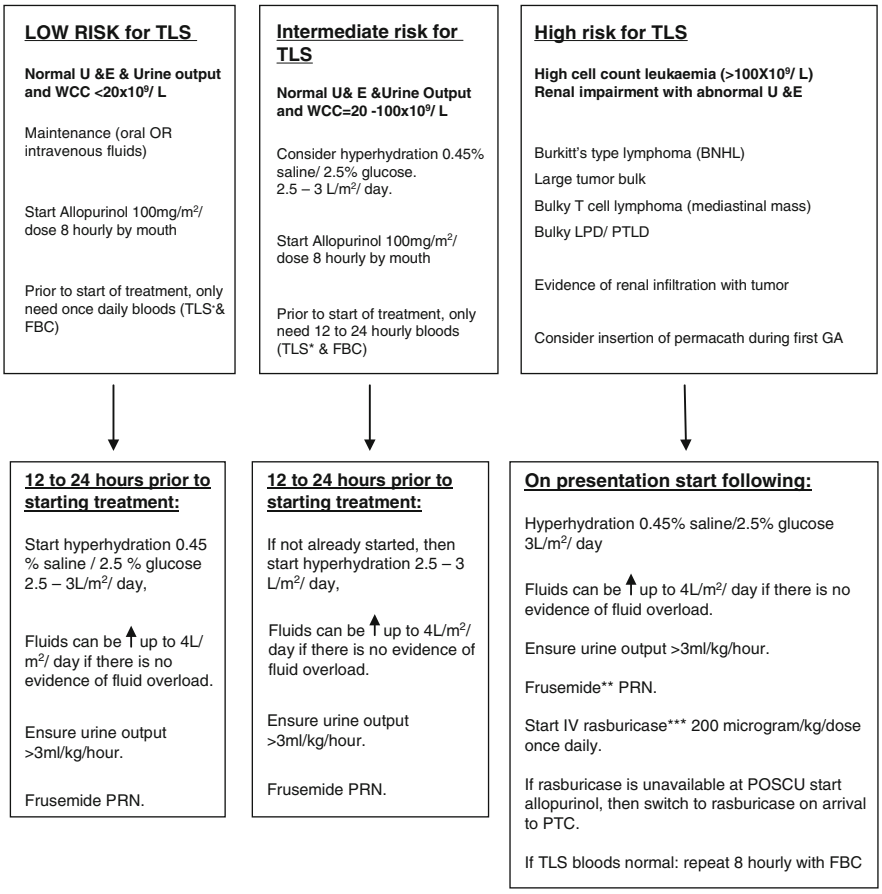
Treatment

Monitor vital signs closely as the patient may deteriorate quickly. Start immediate broad-spectrum antibiotics as per local guidelines. Usually, two agents are recommended, such as piptazobactam 90 mg/kg 6 hourly and gentamicin 7 mg/kg once a day [9]. Fluid resuscitation may need large volumes up to 60–100 ml/kg along with early inotropic support [9]. Admit to a paediatric intensive care unit is advised, if found to be in septic shock or severe sepsis. Also discuss the case with the local oncology centre.

Prognosis

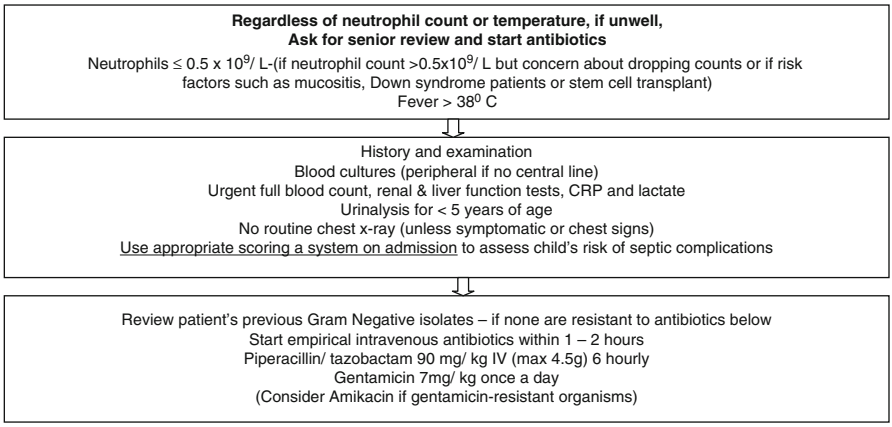
This will be dependent on response to treatment.

TLS Flow Chart 1: Prevention of Tumour Lysis Syndrome (TLS): Prior to Starting Treatment



*TLS blood = urate, U&E, K, Ca, phos and FBC
**Discuss with PTC (patients at risk of leukostasis, dehydration may worsen leukostasis)
*** Rasburicase can trigger haemolysis in G6PD deficiency.

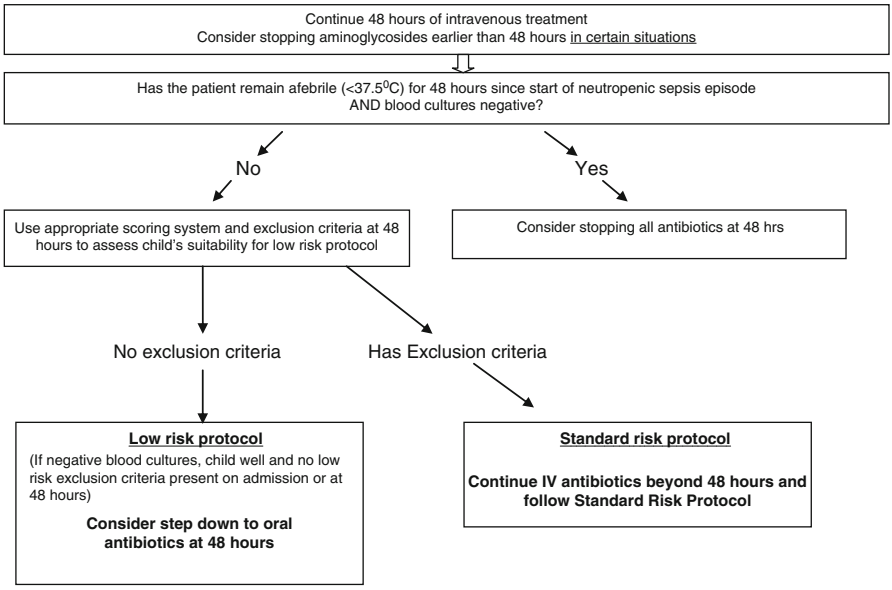
Summary of the Emergency Management of Neutropenic Sepsis



Aminoglycosides should not be used in the following groups:

- Bone tumour patients – empirical treatment is ciprofloxacin with piperacillin/ tazobactam – if endoprosthesis in situ, add teicoplanin also.
- Patients with established or potential renal impairment.

If Gram-positive organism is considered, add teicoplanin or vancomycin to the treatment. For patients with penicillin allergy, use IV ciprofloxacin 10 mg/kg (max 400 mg) every 8 h instead of piperacillin/tazobactam.



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Chapter 10
Metabolic and Endocrine Emergencies in Children

Yasmin Baki

Diabetic Ketoacidosis

Key Points
- Diabetic ketoacidosis is a paediatric emergency with significant mortality and morbidity, in particular from cerebral oedema.
- The focus of management is early diagnosis and the correction of the metabolic abnormality by carefully administering fluids and insulin to reverse ketosis and acidosis.

Introduction

Biochemical criteria for diagnosis:

Acidosis	pH <7.3 ± HCO3 <15 mmol/l
Hyperglycaemia	Blood glucose >11 mmol/l
Ketones	Blood ketones >3 mmol/l

Cerebral oedema, hypokalaemia and aspiration pneumonia are all potential causes of mortality in diabetic ketoacidosis (DKA). Five to ten children die from cerebral oedema secondary to DKA per year in the UK [1]. Therefore, children

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should be managed by senior clinicians with careful clinical and biochemical monitoring of response to treatment. The 2012–2013 National Diabetes Audit identified 25,000 children and young people with type 1 diabetes [2]. Almost one in five children and young people are in DKA at diagnosis of diabetes [3]. Nine percent of children and young people with type 1 diabetes in the UK experienced at least one episode of DKA in 2009–2010 [3].

Pathophysiology [4, 5]

Absolute or relative insulin deficiency causes DKA. Insulin deficiency results in an increase in counterregulatory hormones, including glucagon, cortisol, catecholamines and growth hormone. This hormonal imbalance leads to an accelerated catabolic state with increased glucose production by the liver and kidney resulting in hyperglycaemia. Impaired peripheral glucose utilisation also results in hyperglycaemia and hyperosmolality. In addition, lipolysis occurs due to insulin deficiency and high counterregulatory hormones. This increases serum free fatty acids with production of large quantities of ketone bodies and consequent metabolic acidosis. Hyperglycaemia exceeds the usual renal threshold and with raised ketones causes osmotic diuresis. This osmotic diuresis combined with ketone-induced nausea and vomiting leads to severe fluid depletion and life-threatening electrolyte imbalance. High urine output due to glycosuria continues until extreme volume depletion leads to a critical decrease in renal blood flow and glomerular filtration resulting in potential renal failure. Lactic acidosis from hypoperfusion and sepsis can also contribute to life-threatening acidosis. Unless interrupted by exogenous insulin, there is a significant risk of death.

Young age (<2 years), delayed diagnosis and low socioeconomic group are risk factors for first presentation with DKA at diagnosis [4]. In children with known diabetes, risk factors for DKA include insulin omission, poor control, previous DKA, intercurrent illness, psychiatric problems including eating disorders, challenging social/family circumstances, adolescence and failure of insulin pump therapy.

Clinical Features

Children with a first presentation of diabetes will often have a history of polyuria, polydipsia and weight loss. Also consider DKA in children with known diabetes presenting with abdominal pain, vomiting, tachypnoea or confusion.

Clinical features of DKA include:

- Dehydration and tachycardia
- Deep sighing respiration (Kussmaul) with breath smelling of acetone
- Abdominal pain, nausea and vomiting
- Confusion, drowsiness and progressive reduction in conscious level with eventual coma

Investigations

First line:

- Blood glucose – laboratory and near patient
- Urea and electrolytes (blood gas gives initial electrolytes)
- Blood gas (venous samples give similar pH/pCO_2 to arterial samples)
- Near patient blood ketones (superior to urine ketones)

Second line:

- FBC (often DKA is associated with high white cell count in response to stress so does not necessarily reflect sepsis).
- If febrile or clinically septic, look for a source of infection. Consider CXR, CSF, throat swab, blood cultures, urine and MC&S.

Assessment [4, 6, 7]

1. Degree of dehydration:

Mild 3 %	Only just clinically detectable
Moderate 5 %	Fry mucous membranes, reduced skin turgor
Severe 8 %	As moderate + sunken eyes, prolonged capillary refill time
+ Shock	Poor perfusion, thready fast heart rate, BP drop (late sign)

Weight is a good marker of degree of dehydration.

2. Conscious level:
   Glasgow coma score (GCS) should be evaluated at least hourly.
   If reduced conscious level, consider cerebral oedema or coma related to degree of acidosis. Signs of raised intracranial pressure suggest cerebral oedema (see later section).
3. Full examination to look for signs of sepsis or ileus. If abdominal pain, consider surgical opinion
4. Consider PICU/HDU if:
   - Severe acidosis pH <7.1 + hyperventilation
   - Severe dehydration + shock
   - Reduced GCS with risk of aspiration from vomiting
   - Children <2 years
   - Need for high level of nursing care (1:1) not available in current location

Monitoring:

- Strict fluid balance (consider urinary catheter). Twice daily weights can be useful.
- Hourly capillary blood glucose.

- Capillary blood ketones 1–2 h (if not available urinary ketones).
- Hourly observation including BP. ECG monitoring.
- Initial hourly GCS.

Management (Fig. 10.1)

Airway, breathing and circulation [8]:

(A) Ensure airway is patent. If level of consciousness is reduced, insert airway devices and seek anaesthetic assessment. Insert a nasogastric tube and aspirate and then place on free drainage.
(B) One hundred percent oxygen via facial mask.
(C) Assess circulation. Obtain intravenous access and blood tests.
If in shock, give 10 ml/kg normal saline (0.9 % saline) as a bolus and reassess. Repeat to a maximum of 30 ml/kg. There is no evidence that colloids or other volume expanders are superior to normal saline.

Fluids

1. Volume
Once circulating volume is restored, calculate fluid deficit:

$$\text{Deficit}\left(\text{ml}\right) = \%\ \text{Dehydration} \times \text{Body weight}\left(\text{kg}\right) \times 10$$

$$\text{Fluid Requirement} = \text{Maintenance} + \text{Deficit} - \text{Fluid boluses given}$$

Do not use >8 % in deficit calculations because overestimation of the fluid deficit is dangerous. Beware using capillary refill time as the only indicator for fluid bolus administration as hypocarbia causes peripheral vasoconstriction.

Maintenance should be calculated over 48 h so that total volume is given evenly. Recent UK guidelines [6] recommend the use of lower than standard maintenance volumes to reduce the risk of cerebral oedema.

Maintenance in DKA using the 'reduced volume' volumes [1]:

- Weight <10 kg, give 2 ml/kg/h
- Weight 10–40 kg, give 1 ml/kg/h
- Weight >40 kg, give a fixed volume of 40 ml/h.

2. Type of fluid

- Initially give 0.9 % saline with 20 mmol of KCl in a 500 ml bag.
- Once blood glucose <14 mmol/l, add glucose to the bag starting with 5 % dextrose.

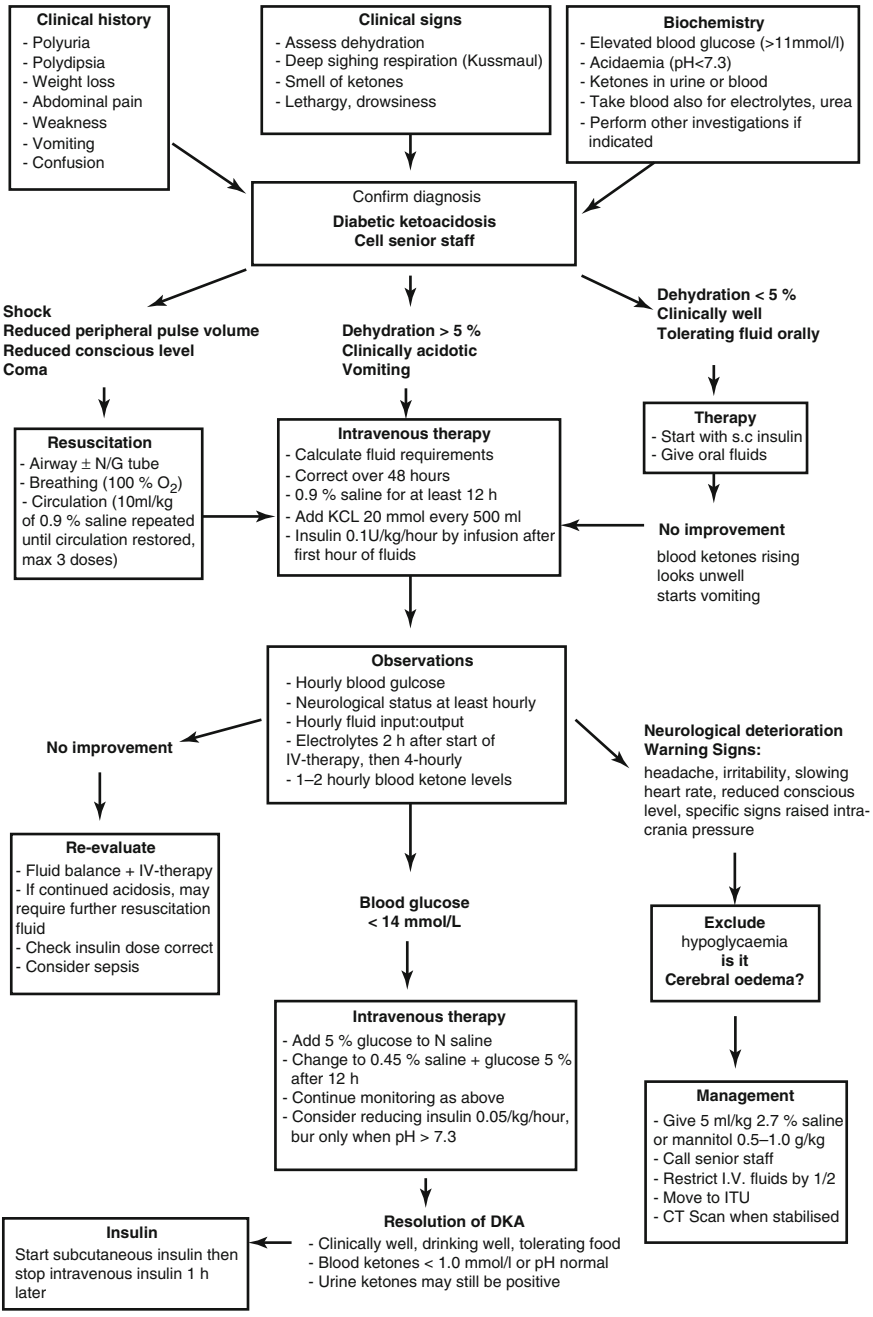

Fig. 10.1 Algorithm for the management of diabetic ketoacidosis [6]

- *After 12 h*, if plasma sodium is stable or increasing, change to 0.45 % saline and 5 % dextrose with 20 mmol KCl. If sodium is falling, continue with normal saline (± dextrose relating to blood glucose).
- Corrected Sodium = Measured Sodium + 0.4 × (BG – 5.5) [9]
- Add potassium to fluids immediately unless anuria is suspected. Potassium levels will fall with insulin, and children with DKA will be potassium depleted. Therefore, ensure that 20 mmol of KCl in each 500 ml bag.
- Monitor urea and electrolytes 2 h after initial treatment and then four hourly.

In severe dehydration, *keep patients nil by mouth. Consider nasogastric tube if there is evidence of gastric paresis*.

3. Insulin [4, 6, 7]

- Do not start insulin until intravenous fluids have been running for at least 1 h. There is evidence to suggest that cerebral oedema is more likely if insulin is started early.
- Administer insulin at continuous low dose by adding 50 units of soluble insulin (Actrapid) to 50 ml of normal saline in a pump running at 0.1 unit/kg/h.
- Once blood glucose <14 mmol/l – add 5 % dextrose to bag. Do not reduce insulin (needed to switch off ketogenesis).
- If blood glucose <4 mmol/l – give bolus 2 ml/kg 10 % dextrose and increase glucose concentration of infusion. Do not stop insulin while glucose is being infused.
- Once pH >7.3+ blood glucose <14 and dextrose containing fluid is being given, consider reduce insulin infusion rate but not <0.05 Units/kg/h

Continue intravenous treatment until the patient is drinking well and tolerating food. Change to subcutaneous insulin once blood ketones <1; urinary ketones may not have disappeared completely. Stop the intravenous insulin infusion 10 min after fast acting subcutaneous insulin to avoid rebound high blood glucose levels (60 min if long acting subcutaneous insulin given).

If acidosis is not correcting consider:

- More insulin needed
- More fluid required
- Sepsis
- Hyperchloraemic acidosis (Cl >80 % Na) [10]
- Salicylates or other recreational drugs

Cerebral Oedema

Risk factors for cerebral oedema [4]:

- Severe hypocapnia at presentation (<2 kPa)
- Younger age
- First presentation

- Elevated serum urea at presentation
- Bicarbonate administration
- Rapid fall in corrected sodium
- High fluid volume given in first 4 h
- Early insulin administration in first hour of treatment

Clinical presentation:

- Headache
- Change in neurological status (restlessness, irritability, increased drowsiness)
- Specific neurological signs (cranial nerve palsies)
- Abnormal posturing, reducing GCS (fits, papilloedema, respiratory arrest)
- Bradycardia, hypertension and decreasing oxygen saturations

Management [8]:

- Hypertonic saline 3 % or mannitol intravenously.
- Rule of 3: 3 ml/Kg of 3 % HNS will increase the plasma Na by 3 mmol/l.
- Restrict intravenous fluids (half maintenance and replace over 72 h).
- Neuroprotective measures – elevate bed head to 30°.
- CT scan to rule out haemorrhage/thrombosis requiring neurosurgical intervention.
- Need to be transferred to PICU for further care.

Prevention [1, 5]

Children with type 1 diabetes admitted with DKA require education regarding:

- Potential precipitants, e.g. infection
- Risks of insulin omission (check adherence)
- Early warning symptoms
- Written sick day rules
- Use of bedside blood ketone meters (more accurate than urine ketones)
- Management of high ketones and sugars

It is also important to address psychosocial issues, in particular eating disorders, and supporting and educating families and schools are vital.

Prognosis

The mortality rate in children from DKA is 0.15–0.30 % [1, 4]. Cerebral oedema accounts for 60–90 % of all DKA deaths [1, 4]. In addition, 10–25 % of survivors of cerebral oedema have significant residual morbidity [1, 4]. There can be subtle evidence of brain injury, for example, memory deficits, even if there are no overt signs during DKA.

Hypoglycaemia

Key Points
- Persistent, severe or recurrent hypoglycaemia can be the presentation of inherited metabolic diseases and endocrine disorders.
- Accurate diagnosis is essential, and it is thus imperative that the correct specimens are obtained at time of diagnosis.
- Prompt treatment is imperative in order to avoid long-term neurological damage or death.

Introduction

Hypoglycaemia is a common finding in paediatrics and needs prompt investigation and treatment. Severe hypoglycaemia is associated with seizures, loss of consciousness and potential risk of death. Therefore, all children who are unwell should have a blood glucose level checked. Hypoglycaemia should be treated as a medical emergency and careful monitoring of both blood glucose and electrolytes should be initiated following treatment. Close clinical monitoring, with particular focus on conscious level, is also essential. A cause for the hypoglycaemia must be elucidated from full investigation with a hypoglycaemia screen.

Pathophysiology

The ability to maintain a normal plasma glucose level depends on:

- Normal endocrine system
- Functionally intact enzymes for glycogenolysis, glycogen synthesis, gluconeogenesis and utilisation of other fuels for oxidation and storage
- Adequate supply of endogenous fat, glycogen and potential gluconeogenic substrates, such as amino acids

Hypoglycaemia triggers physiological counterregulatory mechanisms including [15]:

- Inhibition of insulin and increased glucagon levels resulting in hepatic glycogenolysis with reduced glucose utilisation.
- Release of cortisol increasing protein breakdown and encouraging hepatic gluconeogenesis.
- Release of growth hormone reducing insulin sensitivity and reducing glucose utilisation.
- Release of catecholamines increasing lipolysis and free fatty acid production with resultant increase in ketones. Catecholamines also inhibit insulin release.
- Lipolysis raising free fatty acid levels which act as an alternative fuel to glucose and inhibit glucose utilisation while stimulating glucose production. Thus, the presence of ketones indicates the use of fat as an energy store.

Table 10.1 Clinical presentation of hypoglycaemia [11]

Autonomic	Neuroglycopenic	Behavioural
Pale	Headache	Irritability
Sweaty/clammy	Confusion	Mood change
Tremor	Poor concentration	Erratic behaviour
Restlessness	Loss of consciousness	Combative behaviour
Palpitations	Visual/speech disturbance	
Nausea	Seizures	

Disruption of any of these counterregulatory processes can lead to hypoglycaemia. During hypoglycaemia, children and young adults release catecholamines, cortisol and growth hormone at higher glucose levels than adults and are therefore more susceptible to hypoglycaemia. Neuroglycopenia thus develops at higher glucose concentrations in youth and is more frequent in young children. Neonates and young children are more vulnerable to glucose drops after short fasts because of less developed substrate supplies.

Clinical Features

Hypoglycaemia is defined as blood glucose <2.6 mmol/l. This must be confirmed with a laboratory blood glucose level. This can be associated with clinical symptoms or may be asymptomatic. Symptoms vary between individuals and according to age. Initially, children with hypoglycaemia present with an adrenergic response. This includes pallor, sweating, restlessness and tremor. Subsequently more neuroglycopenic symptoms and signs are prominent, such as headache, confusion, visual disturbance and seizures.

Young infants are more likely to present with neurological features such as jitteriness, apnoea, hypotonia, feeding difficulties, irritability, abnormal cry and convulsions. Older children may have more subtle behavioural changes. Important features in the history are enumerated in Table 10.1.

Important features in the history:

- Age
- Feeding history
- Birth history – prematurity, intrauterine growth retardation (IUGR) and maternal diabetes
- Persistent neonatal jaundice
- Tolerance to fasting/illness
- Drugs – insulin, B-blockers and inhaled steroids
- Alcohol
- Family history of metabolic disorder
- Previous pregnancy loss
- Consanguinity

Examination findings:

- Height/weight/BMI – short stature (growth hormone deficiency).
- Dysmorphism – midline defects (panhypopituitarism), features of Beckwith-Wiedemann Syndrome (hyperinsulinism).

- Hepatomegaly – glycogen storage disease.
- Encephalopathy.
- Cataracts (galactosaemia).
- Appearance of genitalia – undescended testis, microphallus (panhypopituitarism).
- Skin pigmentation – hyperpigmentation (Addison's disease).
- Odour of baby and urine (maple syrup urine disease).
- Look for signs of sepsis.

Differential Diagnosis (Table 10.2)

Investigations [12, 13]

- Near patient and laboratory blood glucose
- Ketones – 3OHbutyrate (can be bedside)
- Blood gas
- Lactate
- Free fatty acids
- Insulin

Table 10.2 Causes of hypoglycaemia [12]

Endocrine
Hyperinsulinism
Adrenal insufficiency
Hypopituitarism
Growth hormone deficiency
Metabolic
Disorders of fatty acid oxidation and carnitine transport
Disorders of carbohydrate metabolism
Disorders of organic acid metabolism
Disorders of gluconeogenesis
Other causes
Neonatal complications: prematurity, birth asphyxia, infants of diabetic mother (secondary hyperinsulinism), IUGR
Biochemical hypoglycaemia is common in the neonatal period (<72 h after birth) prior to establishing feeds. Therefore, the full hypoglycaemia screen is not usually needed, unless the hypoglycaemia does not response to a feed or there is persistent/recurrent hypoglycaemia
In neonates, hypoglycaemia is commonly due to sepsis, severe illness, maternal diabetes or IUGR
Drugs
Liver and multi-organ failure
Sepsis
Gastroenteritis
Idiopathic ketotic hypoglycaemia [13]
This is the most common cause for hypoglycaemia in children, after the neonatal period. It affects children between the ages of one and 5 years. This is usually precipitated by an often relatively mild illness, reducing food intake, in an otherwise well child and children often respond well to treatment with glucose. It is a diagnosis of exclusion

- Random cortisol
- Growth hormone
- Acylcarnitine blood spot (MCAD-Guthrie card)
- Serum amino acids
- Urea and electrolytes and liver function tests
- Ammonia on ice
- Urine:
  - Ketones
  - Organic acids
  - Amino acids
  - Reducing substances

It is helpful to separate ketotic and non-ketotic patients in order to determine aetiology and prioritise blood tests if there are insufficient blood/urine samples.

Absent ketones in blood or urine tests:

- Blood glucose and lactate
- Blood gas
- Insulin (raised in hyperinsulinism/suppressed in fatty acid oxidation defects)
- BetaOH butyrate
- MCAD blood spot
- Urine metabolic screen (organic and amino acids) and reducing substances

Ketones present in blood/urine tests:

- Blood glucose and lactate
- Blood gas
- Random cortisol
- Growth hormone (GH)
  - Low GH and low cortisol suggest hypopituitarism/adrenal failure.
  - Normal GH and cortisol with hepatomegaly indicates glycogen storage disease.
  - In normal GH and cortisol without hepatomegaly, consider ketotic hypoglycaemia.
- Urine metabolic screen and reducing substances (Table 10.3)

Table 10.3 Interpretation of blood/urine results in hypoglycaemia [12]

Specimen	Test	Interpretation
Urine		
Urine Dipstick/ laboratory results	Glucose	
	Ketones	Low/absent in:
		Fatty acid oxidation defect
		Hyperinsulinaemia
	Reducing substances	Galactosaemia/fructosaemia (particularly if glucose dipstick negative)
	Amino acids and organic acids	Deranged in:
		Organic acidaemia
	Toxicology screen	Urea cycle disorders

(continued)

Table 10.3 (continued)

Specimen	Test	Interpretation
Blood		
Bedside/ laboratory results	Glucose (near patient/lab)	<2.6 mmol/l = hypoglycaemia
	Ketone bodies (beta-hydroxybutyrate) (Ketostix/lab)	Low/absent in:
		Fatty acid oxidation defect
		Hyperinsulinaemia
	Lactate	Raised in:
		Metabolic liver disease
		Prolonged convulsion
		Glycogen storage disorders 1
		Defects of gluconeogenesis
		Congenital lactic acidaemia (organic acidaemias)
		Sepsis
	Free fatty acids	Raised in fatty acid oxidation defects
	Carnitine/acylcarnitine (Guthrie/lab)	Raised in:
		Fatty acid oxidation defect
		Organic acidaemia
	Ammonia	Raised in:
		Urea cycle disorders (metabolic alkalosis)
		Organic acidaemias
		Liver dysfunction
		Hyperammonaemia syndrome
	Cortisol	Low levels:
		Hypoadrenalism
		ACTH deficiency/hypopituitarism
	Insulin and C-peptide	*Abnormal if insulin present even at normal laboratory levels in hypoglycaemia*
		Absence of C-peptide suggests exogenous insulin administration
	Growth hormone	Low levels:
		GH deficiency
		Panhypopituitarism
	Amino acids	Deranged in:
		Specific amino acid (aa) disorders (ketogenic and gluconeogenic aa raised in ketotic hypoglycaemia. Low plasma aa in anabolic patients/poor protein reserves)
	U&E	Low Na, high K – low glucocorticoids
	LFT	Deranged in:
		Sepsis
		Liver disease
		Metabolic defects (e.g. glycogen storage disease)
	Poisoning (alcohol, salicylates)	

(continued)

Table 10.3 (continued)

Specimen	Test	Interpretation
Blood gas	Capillary blood gas	Metabolic acidosis:
		Fatty acid oxidation defects (MCAD, LCAD)
		Defects in ketogenesis
		Sepsis
		Glycogen storage disorders
		Organic acid disorders
Microbiology	Blood culture	Sepsis

Management

Assess airway, breathing and circulation as per APLS [14].

If blood glucose <2.6 mmol/l, obtain intravenous access and take blood samples as above.

1. *If patient is conscious* and able to tolerate oral intake, give a trial of oral carbohydrates, such as 10–15 g of fast-acting oral carbohydrates (two to three dextrose tablets). If uncooperative but conscious, give Glucogel®/Dextrogel® (fast-acting sugary gel containing 10 g glucose) by squirting contents into the side of each cheek (buccal) evenly and massaging gently from the outside. This should not be used in an unconscious or fitting child.

   Recheck blood glucose in 10–15 min. If improving and blood glucose >2.6 mmol/l, give 15 g slow-acting carbohydrate, e.g. slice of toast or 200 ml of milk and monitor to ensure normoglycaemia is maintained. If blood glucose has not recovered, give one more trial of oral fast-acting carbohydrate before commencing intravenous treatment.
2. If patient is *unconscious*, give 2–5 ml/kg 10 % dextrose as a bolus intravenously followed by IV glucose infusion 0.9 % saline/10 % dextrose ±electrolytes at maintenance rate. Recheck blood glucose 10 min post IV infusion. Need close monitoring of blood glucose and conscious level as there is a risk of recurrence of hypoglycaemia.

If unable to obtain IV access in unconscious child, give IM glucagon:

- <25 kg or <8 years ½ vial (0.5 mg)
- >25 kg or >8 years full vial (1 mg)

Glucagon is fast acting, so there should be a response within 5 min. Potential side effects include nausea and vomiting so manage the patient in the recovery position subsequently. If there is a history of alcohol use, glucagon may be ineffective as glycogen hepatic stores will be depleted.

If the blood glucose is still <2.6 mmol/l or the GCS has not improved:

- Give second bolus 5 ml/kg 10 % dextrose.
- Increase rate of infusion to 6 mg/kg/min (see formula below).

- Increase glucose concentration. Central access is required for glucose concentrations >12.5 %.
- If possible adrenal insufficiency, give IV hydrocortisone 4 mg/kg.
- Monitor urea and electrolytes regularly.
- Repeat blood glucose initially after 5 min and then 15 min then every 30 min.

A high insulin requirement may indicate hyperinsulinism (>10 mg/kg/min).

If a child remains unconscious after the blood glucose has corrected, consider other causes such as cerebral oedema, head injury, adrenal insufficiency and drug overdose.

$$\text{Glucose in mg/kg/min} = \frac{\%\,\text{dextrose solution} \times \text{ml/hr}}{\text{Weight}(\text{kg}) \times 6}$$

Other metabolic considerations:

- Profound encephalopathy, intractable seizures or apnoea will require intubation and ventilation.
- Circulatory failure can occur requiring intravascular volume expansion and potentially inotropes.
- Metabolic acidosis. If intractable, correct pH using sodium bicarbonate.
- Hyperammonaemia – consider sodium benzoate. Discuss with specialist metabolic team. May need dialysis.
- Monitor for cerebral oedema
- If known condition, use emergency plan provided by metabolic team.

Prognosis

Studies report that repeated severe hypoglycaemia adversely affects cognitive domains in particular long-term memory, attention and verbal IQ. Structural brain abnormalities, such as mesial temporal sclerosis, have been reported in young children with repeated hypoglycaemic seizures. Neurocognitive defects, including impaired auditory and sensory evoked responses, have been reported at the time of hypoglycaemia in infants and young children [15]. This emphasises the need for early diagnosis and treatment.

Prevention [15]

In order to prevent further episodes of hypoglycaemia from occurring, it is important to establish a cause for the hypoglycaemia. Prior to discharge, the child must be able to tolerate a reasonable time between feeds (at least 4 h) without blood glucose dropping below 3 mmol/l. Children and their families need multidisciplinary team support to manage their condition. Dietician input and clear management plans regarding care during illness and potential illness triggers are essential. The child

and family need education about the management of hypoglycaemia with supplies of fast-acting carbohydrate and IM glucagon available. In addition, the child and family should know how to monitor glucose levels. School involvement is also important.

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Chapter 11
Neonatal Emergencies

Christina L. Cochran and Parul P. Soni

Key Points
- Neonatal Resuscitation Program (NRP) guidelines should be employed for management of a newborn in distress [1].
- Always consider nonaccidental trauma in newborns presenting to the emergency department (ED).
- Prognosis is often dependent on the underlying aetiology or degree of illness.
- Overall, early recognition and management will improve long-term outcome.

Section A: Common Assessment and Treatment Elements

Introduction

Newborns presenting with respiratory illness should be evaluated promptly as respiratory failure can precipitate quickly. There are many aetiologies of respiratory illness in the newborn period, of which the most common will be reviewed.

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Pathophysiology

- Respiratory distress syndrome (RDS) is caused by insufficient surfactant production, most commonly in preterm infants.
  - Insufficient surfactant causes inappropriate alveolar expansion.
  - Decreased alveolar expansion leads to respiratory distress.
- Bronchiolitis is the result of a viral infection affecting the lower respiratory tree.
  - Bronchioles become inflamed and increase secretion production.
  - Common viral aetiologies include respiratory syncytial virus, human metapneumovirus, rhinovirus, enterovirus, and coronavirus [2].
- Meconium aspiration syndrome (MAS) results from aspiration of meconium at the time of delivery.

Clinical Features

- Initial signs of respiratory distress include tachypnoea and increased work of breathing (Table 11.1)
- As distress progresses, newborns are at risk of developing respiratory failure and apnoea.
- RDS presents in the first days of life (Table 11.2)
- Bronchiolitis is a clinical diagnosis based on physical exam and history [3].
  - Newborns will have increased work of breathing with crepitations and rhonchi on exam.

Differential Diagnosis

- Consider RDS, bronchiolitis, sepsis, bronchopulmonary dysplasia (BPD), pulmonary hypertension, pneumonia, MAS, and pertussis.

Table 11.1 Signs of respiratory distress versus respiratory failure

Respiratory distress	Tachypnoea
	Retractions
	Abdominal accessory muscle use
	Nasal flaring
	Tracheal tugging
Respiratory failure	Bradypnoea
	Apnoea
	Hypoxia/hypoxemia
	Carbon dioxide retention
	Respiratory acidosis

Table 11.2 Comparison of clinical features and management of RDS and bronchiolitis

	RDS	Bronchiolitis
Age	First 2 days of life	First 2 years of life
Aetiology	Decreased surfactant	Viral infection
Signs/symptoms	Tachypnoea, increased work of breathing, hypoxia	Tachypnoea, increased work of breathing, apnoea, hypoxia
Diagnosis	CXR, clinical	Clinical
Management	Supplemental oxygen, surfactant	Suctioning, supplemental oxygen

Table 11.3 Chest x-ray findings in common newborn respiratory diseases

	Chest x-ray finding
Respiratory distress syndrome	Ground-glass opacities
	Decreased lung volumes
	Air bronchograms
Pneumonia	Focal opacities
	Diffuse infiltrates
Foreign body aspiration	Air trapping on the side of the foreign body

- History of meconium presence in amniotic fluids should raise concern for MAS.
- When considering pulmonary hypertension, conduct thorough cardiac evaluation.

Investigations

- Chest radiograph (CXR) should be obtained in patients suspected to have RDS, pneumonia, and foreign body aspiration (Table 11.3).
- CXR is not indicated in patients with bronchiolitis [3].
- Obtain nasal secretions to confirm pertussis infection.
- If suspected, infectious workup should be completed including blood, urine, and cerebrospinal fluid studies (CSF).

Treatment

- Intervention can be tailored to a degree of respiratory distress (Table 11.4).
- Surfactant should be provided to patients with RDS [4].
- Supportive care, including suctioning and supplemental oxygen, is the treatment of choice for bronchiolitis.
  - Salbutamol, normal saline, and hypertonic saline nebulisers have not been shown to improve outcome in bronchiolitis [3].
- For patients without clear viral aetiology, consider antibiotic coverage.
- If suspicious for pertussis, start prophylactic antibiotics.

Table 11.4 Recommended interventions based on level of respiratory distress

	Intervention
Apnoeic	Bag-valve-mask ventilation
	Intubation and mechanical ventilation
Severe distress	Continuous positive airway pressure (CPAP)
	Intubation and mechanical ventilation
Moderate distress	High-flow nasal cannula
	Vapotherm
	Continuous positive airway pressure (CPAP)
Minimal distress	Trial supplemental oxygen via nasal cannula

Prognosis

- Newborns in respiratory distress will require observation and treatment on an inpatient service.
- Newborns with severe RDS are at risk of requiring long-term supplemental oxygen and developing BPD.
- Bronchiolitis has an overall good prognosis with resolution of symptoms once the virus infection resolves.

Prevention

- Prevention of bronchiolitis focuses on reducing virus transmission.
- Avoidance of preterm delivery unless clinically indicated will decrease the risk of RDS.

Section B : Neonatal Cardiac Emergencies

Introduction

Congenital cardiac deformities may present in the first hours to days to weeks of life. Prompt recognition of a cardiac aetiology is imperative, as management from cardiogenic shock differs from management of other aetiologies of newborn shock.

Pathophysiology

- Congenital heart defects are a result of abnormal embryogenesis
- Defects can be classified as cyanotic (Table 11.5) and non-cyanotic (Table 11.6).
- Cyanosis is a result of right to left shunting of non-oxygenated blood into systemic circulation.

Table 11.5 Characteristics of cyanotic heart lesions

Name	Truncus arteriosus	Transposition of the great vessels	Tricuspid atresia	Tetralogy of Fallot	Total anomalous pulmonary venous return
Description	Single arterial trunk supplying pulmonary and systemic circulation	Pulmonary artery supplying systemic circulation; aorta supplying pulmonary circulation	Absence or underdevelopment of the tricuspid valve	Overriding aortic arch, pulmonary atresia, VSD, right ventricular hypertrophy	All four pulmonary veins do not connect correctly with the left atrium
Diagnosis	Echo, CT angiogram	Echo, CXR showing "egg on a string." CT angiogram	Echo	Echo, CXR showing boot-shaped heart	CT angiogram, cardiac MRI
Management	CR support, diuretic therapy, surgical intervention	CR support, PGE1, surgical intervention	CR support, PGE1, surgical intervention	Tetralogy spell: 100 % oxygen, morphine, vasopressor; CR support, surgical intervention	CR support, PGE1, supplemental oxygen, fluid support, surgical intervention

VSD ventricular septal defect, *Echo* echocardiogram, *CT* computerised tomography, *CXR* chest x-ray, *CR* cardiorespiratory, *PGE1* prostaglandin E_1

Table 11.6 Non-cyanotic heart defects

	Coarctation of the aorta	Ventricular septal defect (VSD)
Presentation	Cool BLE extremities	Time of presentation dependent on the size of VSD
	Decrease pulses in the BLE	Signs of heart failure or fluid overload of the lungs
	Decreased blood pressure in the BLE	
Diagnosis	Echocardiogram	Echocardiogram
	CT angiogram or MRA of the heart/aorta	CXR may demonstrate cardiomegaly
Management	Prostaglandin E_1	Haemodynamic support
	Haemodynamic support	Closure of the defect
	Surgical repair	

BLE bilateral lower extremities

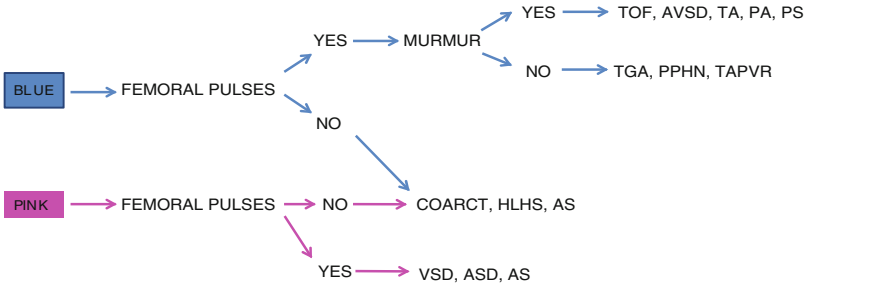
Clinical Features

- Newborns may present with a variety of findings (Table 11.7).

Table 11.7 Possible clinical features associated with congenital heart disease

History	Vital sign abnormalities	Physical exam findings
Abnormal foetal ultrasound	Tachycardia (>160 bpm)	Shock
Poor/difficult feeding	Bradycardia (<80 bpm)	Decreased central/peripheral pulses
Cyanosis	Hypoxia (<90 % on RA)	Poor perfusion
Emesis	Tachypnoea	Increased work of breathing
Poor weight gain	Bradypnoea	Pathologic murmur
Maternal diabetes	Hypertension	Lethargy
Maternal hypertension	Hypotension	Hepatomegaly
Maternal medication		Crackles on lung auscultation
Family cardiac history		

bpm beats per minute, *RA* room air



TOF: Tetralogy of Fallot AVSD: Atrioventricular Septal Defect TA: Truncus Arteriosus PA" Pulmonary Atresia
PS: Pulmonary Stenosis TGA: Transposition of the Great Arteries PPHN: Persistent Pulmonary Hypertension
TAPVR: Total Anomalous Pulmonary Venous Return Coarct: Coarctation HLHN: Hypoplastic Left Heart Syndrome
AS: Aortic Stenosis ASD: Atrial Septal Defect

Fig. 11.1 Assessment of infant colour and central pulses

Differential Diagnosis

- Serious bacterial infection (SBI) should be considered in all neonates presenting to the ED.
- Metabolic abnormalities, respiratory infections, and feeding difficulties can present similarly.

Investigations

- Assess colour of the newborn and central/peripheral pulses (Fig. 11.1).
- Measure four extremity blood pressures and pulse oximetry.
  - In the setting of coarctation, lower extremity blood pressures and pulse oximetry will be decreased compared to right upper extremity.
- CXR should be obtained to assess the cardiac silhouette.

- Obtain electrocardiogram to assess for rhythm disturbances.
- Echocardiogram is used to assess cardiac anatomy and function.
- Consider blood gas to assess pH and arterial oxygenation.
- Detailed maternal and prenatal history, including ultrasound results, should be gathered.
  - Many maternal conditions and medications can be associated with congenital heart disease [5].

Treatment

- Supplemental oxygen should be provided.
- Haemodynamic support should be provided with fluids, inotropes, and chronotropes as clinically indicated.
  - The degree of fluid resuscitation is dependent on the underlying cardiac defect.
- Prostaglandins should be initiated to delay closure of the ductus arteriosus in patients suspected of having a shunt-dependent defect.
  - Initial dose – Prostaglandin E1: 0.05 mcg/kg/min
- Initiate inhaled nitric oxide if pulmonary hypertension is suspected.
- Cardiology consult should be obtained as soon as cardiac defect is suspected.

Prevention

- Improved management of maternal diabetes and hypertension may decrease congenital heart defects.

Section C: Serious Bacterial Infections (SBI)

Introduction

Any newborn with concern for infectious process should be evaluated and treated immediately. SBIs in neonates include, but not limited to, urinary tract infections (UTIs), bacteraemia, sepsis, meningitis, pneumonia, pyelonephritis, and cellulitis. Neonates with bacterial infection are at high risk for long-term morbidity and mortality [6]. Low threshold for workup of an SBI should be maintained when evaluating children less than 29 days old.

Pathophysiology

- Newborns have reduced defence against bacterial infections secondary to an immature immune system.
- The two most common bacteria responsible for infection in newborns are group B streptococcus (GBS) and *Escherichia coli* (*E. coli*) [7].
- UTIs are the most common bacterial infection in this patient group.
- Necrotising enterocolitis (NEC) is a result of intestinal ischaemia, leading to bacterial overgrowth and gut necrosis [8].

Clinical Features

- Rectal temperature greater than 38° C is suggestive of infection.
- Infants may present with hypothermia, lethargy, increased sleeping, poor feeding, jaundice, vomiting, changes in their respiratory pattern, or abnormal vital signs.
- Detailed prenatal and antenatal history should be obtained.
  - Risk factors for infection include prolonged rupture of membranes, maternal fever, maternal infection, and lack of appropriate treatment for maternal GBS.
- Abdominal tenderness, distension, vomiting, bloody stool, and lethargy may indicate NEC.
- Redness, tenderness, or drainage from the umbilicus is concerning for omphalitis.

Differential Diagnosis

- Consider hypoglycaemia, metabolic dysfunction, hyperbilirubinemia, congenital heart conditions, and neurologic dysfunction when assessing a patient with the above features.

Investigations

- Workup should include CBC, blood culture, urinalysis, urine culture, and CSF studies including cell count, differential, glucose, protein, and culture.
- Consider obtaining herpes simplex virus (HSV) tests.
- In the setting of omphalitis, send culture of umbilical discharge.
- Obtain cultures prior to antibiotic initiation unless patient is clinically decompensating.
- For patients with dehydration or concern for metabolic abnormalities, obtain electrolytes and liver function panel.

- CXR should be considered in newborns with respiratory distress, and hypoxia.
- NEC diagnosis and management is reviewed in Table 11.11 [9].

Treatment

- Prompt antibiotic therapy is essential.
- Initiate broad-spectrum antibiotic coverage with ampicillin and gentamicin and/or cefotaxime.
  - Ampicillin, gentamicin, and cefotaxime dosing is dependent on age and weight [10].
  - Acyclovir covers for possible HSV infection: 20 mg/kg/dose every 8 h [10].
- Continue antibiotics until CSF, blood, and urine cultures have resulted.
- NEC management is reviewed in Table 11.11.

Prevention

- Appropriate treatment of maternal GBS infections reduces the likelihood of newborn infection [11].

Section D: Newborn Gastrointestinal Emergencies

Introduction

There are a variety of gastrointestinal (GI) emergencies that may present in the newborn period. The majority of diagnoses can be distinguished based on physical exam and radiologic evaluation. Omphalocele and gastroschisis are not discussed in depth, but ED physicians should be aware of these complications.

Hyperbilirubinemia

Pathophysiology

- Most newborns develop clinical hyperbilirubinemia [12].
- Most cases of hyperbilirubinemia are physiologic, or secondary to normal delayed conjugation and excretion of bilirubin in the newborn, though pathologic aetiologies must be considered.

- Factors contributing to higher bilirubin levels are listed in Table 11.8.
- Patients can be classified into low risk, medium risk, or high risk for complications of bilirubin (Table 11.9) [12].

Clinical Features

- Newborns with hyperbilirubinemia will appear jaundiced and may have decreased activity and desire to feed.
- Jaundice occurs in a cranial to caudal progression and presents within the first week of life, with peak from day 3 to 5.
- Jaundice presenting within the first 24 h of life is likely pathologic.

Differential Diagnosis

- Consider underlying genetic abnormalities, including Gilbert, Crigler-Najjar, and Dubin-Johnson, when diagnosing unconjugated hyperbilirubinemia.
- Also consider causes of elevated direct/conjugated bilirubin, including biliary atresia.
- Infection and sepsis can lead to increased bilirubin levels.

Table 11.8 Risk factors for hyperbilirubinemia

Birth trauma
Dehydration
Breast feeding jaundice
Breast milk jaundice (after 1 week of life)
Haemolysis
ABO incompatibility
Polycythaemia
Preterm infant
Infection
Ethnicity (Asian)

Table 11.9 Classifying newborns into risk category based on age and number of risk factors

	Gestational age		Number of risk factors
Low risk	38 weeks or older	with	0
Medium risk	38 weeks or older	with	1
	35 weeks to less than 38 weeks	with	0
High risk	35 weeks to less than 38 weeks	with	1 or more

Investigations

- Laboratory workup will demonstrate elevated unconjugated bilirubin levels.
- Consider blood type testing, direct bilirubin level, complete blood count, blood smear, liver function panel, and infectious workup.

Treatment

- Treatment for hyperbilirubinemia is based on age, risk factors, and level of bilirubin (Table 11.10).
- The goal of treatment is to prevent encephalopathy secondary to bilirubin deposition in the brain or kernicterus [12].
- Initial intervention includes oral hydration, intravenous (IV) hydration, and phototherapy.
- For high-risk cases or concern for kernicterus, exchange transfusion is employed [12].

Prognosis

- Overall prognosis of hyperbilirubinemia is very good [12].
- The incidence of kernicterus is declining with the advent of phototherapy and exchange transfusion, though patients with kernicterus continue to have long-term neurologic morbidities [12, 13].

Table 11.10 Trigger levels of total serum bilirubin for phototherapy and exchange transfusion in infants 35 weeks or greater [12]

Age of newborn	Risk level	Phototherapy level (μmol/L)	Exchange transfusion level (μmol/L)
24 h	Low	196	324
	Medium	162	282
	High	128	256
48 h	Low	258	376
	Medium	222	324
	High	192	290
72 h	Low	299	410
	Medium	265	360
	High	230	316
96 h	Low	336	428
	Medium	294	384
	High	247	324
5 days and older	Low	359	428
	Medium	307	384
	High	256	324

Prevention

- Prevention of hyperbilirubinemia focuses on appropriate hydration of the newborn and appropriate treatment of maternal infections at the time of delivery.
- Awareness of risk factors will assist in early recognition and intervention.
- Kernicterus is an entirely preventable disease though not reversible [12].

Duodenal Atresia

Pathophysiology

- Duodenal atresia occurs when there is an occlusion of the duodenum, restricting passage of materials through the small bowel.
- Overall incidence is reported at 1 in 6,000 births [14]
- This diagnosis is highly associated with trisomy 21.

Clinical Features

- Patients will present in the first days of life with bilious vomiting.
- Physical exam may reveal a scaphoid abdomen.
- Newborn may pass meconium but is unlikely to have any bowel movements.
- Prenatal history is often positive for polyhydramnios.

Differential Diagnosis

- Infection, oesophageal atresia, tracheaoesophageal fistula, pyloric stenosis, and reflux should be considered.
- Vomiting in the setting of duodenal atresia is bilious in nature, distinguishing this diagnosis from many others.

Investigations

- Abdominal x-ray will reveal a double bubble sign indicating trapped air in the stomach and proximal duodenum.
- Electrolytes, including glucose, should be obtained to assess hydration and nutritional status.

Treatment

- Management of duodenal atresia is reviewed in Table 11.11.

Prognosis

- Complications may include dependence of parenteral nutrition, poor growth, perforation of the duodenum, and stricture at the site of anastomosis [15].

Hirschsprung's Disease

Pathophysiology

- Hirschsprung's disease is the result of failed migration of neural crest cells leading to a lack of innervation in a section of the colon.
- The aganglionic colon is unable to relax, leading to constriction of that segment.
- Hirschsprung's disease is more common in males with a 4:1 male to female predominance [16].

Clinical Features

- Most newborns present with delayed passage of meconium and stool.
  - Ninety-eight percent of normal newborns pass meconium within 48 h.
- Newborns may present with abdominal distension, vomiting, or stringy stools
- Physical exam reveals a tight anal sphincter and explosive stooling with rectal exam.
- Patients may present with toxic megacolon – extreme dilation of the colon leading to distension, pain, perforation, and shock.

Differential Diagnosis

- Consider constipation, meconium ileus, duodenal atresia, or other intestinal obstruction.

Table 11.11 Characteristics of duodenal atresia, Hirschsprung's disease, and necrotizing enterocolitis

	Duodenal atresia	Hirschsprung's disease	NEC
Age	First days of life	First days of life	First months of life
Gender dominance	None	Male > female	None
Emesis	Bilious	Bilious, stool – coloured	Non-bilious or bilious
Abdominal x-ray findings	Double bubble sign	Dilation of the proximal colon, lack of air in the rectum	Pneumatosis, abdominal free air, portal venous gas
Diagnosis	Abdominal x-ray	Rectal suction biopsy	Clinical with x-ray findings
Management	NPO, nasogastric tube placement, IV hydration, surgical repair	NPO, nasogastric tube placement, IV hydration, surgical repair	NPO, nasogastric tube placement, IV hydration, initiate antibiotics, surgical consult

Investigations

- Rectal suction biopsy of the narrowed section of the colon is gold standard for diagnosis [17].
- Anal manometry and barium enema can assist with diagnosis.
- If toxic megacolon is suspected, obtain abdominal x-ray, electrolytes, complete blood count, and blood culture.

Treatment

- Management of Hirschsprung's Disease is reviewed in Table 11.11
- In the case of toxic megacolon, provide resuscitation as clinically indicated and IV antibiotics

Prognosis

- Long-term complications of Hirschsprung's disease include constipation, bowel dysfunction, and toxic megacolon [17].

Section E: Metabolic Emergencies

Introduction

Metabolic disorders can masquerade as many different diagnoses on initial presentation. In newborns, always consider underlying metabolic disorders such as

congenital adrenal hyperplasia (CAH), thyroid dysfunction, fatty acid disorders, amino acid disorders, urea cycle disorders, organic acid disorders, and glycogen storage disease.

Pathophysiology

- Metabolic emergencies in a newborn result from a range of disorders and deficiencies.
- Pathophysiology is entirely dependent on the underlying disorder.

Clinical Features

- Newborns may present with poor weight gain, feeding difficulties, lethargy, emesis, diarrhoea, and decreased movement.
- Female and male infants with CAH present with slight variation [18].
  - Females will likely have ambiguous genitalia with enlarged clitoris.
  - Males tend to present with salt wasting and electrolyte abnormalities including hyponatremia and hypokalaemia.

Differential Diagnosis

- Consider sepsis, genetic disorders, and cardiac abnormalities in these patients.

Investigations

- Newborn screening results should be reviewed.
- Glucose levels should be obtained immediately.
- Blood gas should be obtained to assess for signs of metabolic acidosis or alkalosis.
- Multiple metabolic disorders can present with electrolyte derangement.
- Obtain ACTH, cortisol, and 17-hydroxyprogesterone levels if concerned for CAH [18].
- Consider urine organic acids, serum amino acids, acylcarnitine profile, lactate, and pyruvate in non-emergent phase.
- Consider thyroid studies in patients with clinical signs or maternal history of antithyroid antibodies.

Treatment

- Provide dextrose for patients with hypoglycaemia defined as less than 2.6 mmol/L or symptomatic.
  - Oral feeds with dextrose (milk, formula) if patient tolerates.
  - IV dextrose 10 % bolus of 2 ml/kg.
  - If blood sugar remains low, consider an IV infusion of 10 % dextrose.
- Consider stress-dose steroids if CAH and adrenal crisis are suspected [10].
  - Hydrocortisone IV: 50–100 mg/m^2
- Correct electrolyte abnormalities as indicated.
  - CAH patients may require sodium chloride supplementation [18].
  - Consider sodium benzoate for sodium replacement after discussion with an endocrinologist.

Prognosis

- Many metabolic disorders require long-term dietary supplementation or restriction.

Prevention

- Newborn screening should be administered on all newborns in the first 3 days of life.

Section F: Haematologic Emergencies

Introduction

Bruising, bleeding, and petechiae are not common presenting issues in newborns, though when present, should raise concern. Broad differentials should be maintained for these clinical features.

Pathophysiology

- In neonatal alloimmune thrombocytopenia, maternal antibodies cross the placenta and target paternally derived antigens, causing destruction of the platelet.
- Autoimmune thrombocytopenia occurs when maternal antibodies target maternal and neonatal platelets.

Clinical Features

- Patients may present with easy bleeding, petechiae, and purpura.
  - Assess if males experienced prolonged bleeding with circumcision.
- Mental status changes could be indicative of intracranial haemorrhage.

Differential Diagnosis

- Consider vitamin K deficiency, neonatal alloimmune thrombocytopenia, and autoimmune thrombocytopenia.
- Also consider nonaccidental trauma, infection, underlying coagulopathy, necrotizing enterocolitis, haemophagocytic lymphohistiocytosis, and leukaemia.

Investigations

- Gather thorough history including maternal medications, illnesses, birth history, and vitamin K administration.
- Obtain complete blood count, coagulation panel.
- Maintain a low threshold for infectious workup
- Cranial computerised tomography (CT) scan or ultrasound should be completed if concerned for intracranial bleed.
- Consider haematology/oncology consult.

Treatment

- Immediately administer vitamin K if not previously given or coagulopathy is suspected.
  - Vitamin K: 0.5–1 mg intramuscularly, subcutaneously, or intravenously [10]
- Provide platelet transfusion if patient is thrombocytopenic and actively bleeding (Table 11.12).
- Consider fresh frozen plasma in setting of moderate to severe bleeding.
- If concerned for an antibody-mediated process, consider intravenous immunoglobulin (IVIg) and/or platelet transfusion [19, 20].
  - IVIg: 0.4–1 g/kg/day [19, 20].
  - Steroid infusion can be considered as adjunct therapy [19, 20].
- Antibiotic therapy for infants with suspected bacterial infection

Table 11.12 Potential triggers for platelet transfusion

Platelet level	Clinical condition
Normal	Platelet function disorder with bleeding
Platelet count <100	Major bleeding
	Disseminated intravascular coagulopathy
	Preoperative
	Sepsis
Platelet count <50	Minor bleeding
	Exchange transfusion
	Preterm infant
Platelet count <30	Asymptomatic term infant

Prognosis

- Intracranial haemorrhage and lower platelet levels are associated with increased risk of morbidity and mortality [21].
- Majority of newborns with thrombocytopenia have good outcomes [21].

Prevention

- Administration of vitamin K immediately following birth
- Close monitoring of infants born to moms with idiopathic thrombocytopenia

Section G: Neurologic Emergencies

Introduction

Seizures may present in the neonatal period as a secondary process or as a primary seizure disorder. The primary goal after stabilisation of the patient should be to uncover and manage the underlying aetiology of the seizure.

Pathophysiology

- Seizures are more frequent in preterm infants and infants with hypoxic ischemic encephalopathy (HIE) [22].
- Abnormal movements occur secondary to withdrawal from maternal drug exposure, commonly opioids.

Clinical Features

- Seizures may manifest with abnormal extremity or eye movements.
- Withdrawal symptoms are listed in Table 11.13 [23].
  - Neonatal abstinence scoring may be utilised to assess for risk of neonatal withdrawal.

Differential Diagnosis

- Aetiologies of seizure range from encephalopathy, stroke, HIE, metabolic disorders, infection, to genetic disorders [24].
- Electrolyte abnormality can present abnormal movements or behaviour.
- Nonaccidental trauma should always be considered.

Investigations

- Obtain glucose and electrolytes in infants with abnormal movements.
- Gather thorough history, including maternal history, maternal medications, maternal infections, and birth history [24].
- Obtain head imaging
- Complete infectious workup, including CSF studies, should be performed.
- Send urine and meconium toxicology screens to assess for withdrawal syndrome.

Table 11.13 Comparison of neonatal seizures and withdrawal syndrome

	Neonatal seizure	Withdrawal syndrome
Clinical features	– Abnormal focal or generalised movements – Decreased responsiveness	– Inconsolable, high-pitched crying – Diarrhoea, vomiting – Poor feeding – Yawning, sneezing – Sleep disturbance
Diagnosis	– Clinical/history – EEG – Infectious workup – CT or ultrasound of the head	– Neonatal abstinence scoring – Maternal drug history – Urine drug screen – Meconium drug screen
Management	– Phenobarbital 20 mg/kg IV[10] – Fosphenytoin 20 mg PE/kg IV[10] – Lorazepam 0.05 mg/kg IV[10]	– Supportive care – Methadone – Second line agents: phenobarbital, clonidine, lorazepam

Treatment

- If hypoglycaemia is present, correct with IV dextrose.
- Consider treating a seizing neonate with phenobarbital or phenytoin [24].
- If seizures are nonresponsive to the above medications, please consider lorazepam or levetiracetam.
- In cases of neonatal withdrawal, provide supportive care and tailor medical management based on maternally abused drug (Table 11.13).

Prognosis

- Prognosis depends on the underlying aetiology and duration of symptoms [24, 25].

Prevention

- Maternal education on the harms of drug use

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Chapter 12
Nonaccidental Injuries in Children

Kee-Chong Ng and Peter Choong-Yi Wong

Key Points
- "CALL DR" is a mnemonic for the key steps in the emergency department pathway for nonaccidental injury.
- Fractures unusual for the reported mechanism of injury or the developmental age of the child may indicate NAI.
- Infants who have suffered abusive head trauma (AHT) may present with apnoea or seizures.
- A diagnosis of NAI should not be made without due consideration of differential diagnoses such as bleeding disorders and osteogenesis imperfecta.

Introduction

The problem of nonaccidental injury is huge: the World Health Organization (WHO) estimates that in the year 2000, there were 57,000 deaths from homicide in children under 15 years old [1]. In the USA, nonaccidental injury is the third leading cause of death in children aged 1–4 years [2].

The correct diagnosis and management of nonaccidental injury in one child can potentially save many lives: at stake are the lives of the presenting victim as well as others at risk of harm from the same source. A vigilant health professional that recognises and reports it can break a cycle of recurring violence and redeem for the child decades of healthy life ahead.

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The spectrum of presentation of nonaccidental injuries is so varied and the individual needs of each patient so nuanced that no one algorithm will do the matter justice. There is however a common pathway that involves a high index of suspicion, good communication, recognition of alerting features and patterns of injury and overcoming obstacles to report suspected child abuse as mandated by the local legal framework [3–5].

The key roles of the doctor in the emergency department are to identify possible nonaccidental injury, investigate or refer onward for investigation, provide medical treatment, ensure the safety of the patient, document accurately and adequately and communicate appropriately with law enforcement and child protection agencies [6].

Definitions

- "Child abuse" or "child maltreatment" can be defined as "any act of omission or commission by a parent or caregiver that would endanger or impair a child or young person's physical or emotional well-being, or that which is judged by community values and professionals to be inappropriate" [7]. It includes the categories of physical abuse, sexual abuse, emotional abuse and neglect [1].
- "Nonaccidental injuries" usually refer to physical injuries resulting from abuse or neglect. There is no clear line separating accidental from nonaccidental injuries, but rather a continuum with a grey zone in the middle where injuries have not been deliberately inflicted but have resulted, for example, from a caregiver's failure to supervise the child or ensure the child's safety. A child's injury can be judged nonaccidental even if there was no intention to cause harm [5].

Clinical Features: General

In a child presenting with an injury, features which may raise the suspicion of nonaccidental injury include [3, 5]:

- No or vague explanation for a serious injury
- Explanation discordant with the nature/severity of the injury (e.g. a toddler with a femur fracture reported to have "bumped into a chair while walking")
- Explanation discordant with the age/developmental stage/ability of the child (e.g. a 2-week-old neonate reported to have "rolled off the bed")
- Discordant explanations from different witnesses
- Discordant explanations from the same witness at different times (changing story)

Delay in seeking consultation or unusual patterns of emergency department use are also of concern.

No injury should be assessed in isolation. All injuries should be assessed in the context of:

- The explanation given for the injury
- The child's medical history/pre-existing illnesses
- Social history
- The developmental age of the child
- Full clinical examination, for other injuries as well as signs of possible differential diagnoses (e.g. blue sclera of osteogenesis imperfecta)

Clinical Features: Bruising and Other Soft Tissue Injuries

- Bruises are the most common form of nonaccidental injuries [8].
- Nevertheless, accidental bruising is more common than nonaccidental bruising.
- The *location* of bruising is important. *Accidental* bruises tend to occur over bony prominences and anterior surfaces of the body, such as the forehead, knees and shins. On the other hand, *abusive* bruises may occur anywhere on the body, often away from bony prominences, and are often found on the face, ears, neck, abdomen, back and buttocks [8–10].
- Particular *patterns* of bruising, such as clustering of bruises, multiple bruises of similar shape and size and bruises resembling the shape of an object (such as a loop or clothes hanger), also suggest nonaccidental injury [8].
- The developmental age of the child should be considered. Accidental bruises in young infants who have not yet learnt to crawl or cruise are rare. From 6 to 12 months old, the head and face are the most common site of accidental bruises; in ambulant children, the extensor surfaces of the limbs, especially the knees and shins, are the most common site of accidental bruises [8]. "Bruising before cruising" should raise concern [11], as should bruises in older children who are not independently mobile for medical reasons (e.g. cerebral palsy).
- Accidental bruises are often small, less than 10 or 15 mm, whereas bruises in abused children are often larger [8].
- It is not possible to accurately determine the age of a bruise from its appearance or colour [9]. One study in adults and older children suggests that the appearance of yellow colour may indicate that a bruise is at least 18 h old [12].
- Examples of other soft tissue injuries where the possibility of abuse should be considered are biting marks (two opposing semicircular arcs of teeth imprints) and abrasions around the wrists and ankles (caused by rope ties).

Clinical Features: Thermal Injuries

Burns and scalds, both accidental and nonaccidental, are a common reason for infants and children to be brought to the emergency department. Several distinct

patterns of nonaccidental thermal injuries are recognised [13, 14]. These include:

- *Scalds from forced immersion in hot water*. Unlike accidental scalds, forced immersion scalds typically (but not universally) have features of uniform burn depth, sharp demarcation lines, symmetrical distribution, absence of splash marks and sparing of the flexures. A classic example would be of a toddler forcibly held down in a tub with a shallow level of hot water, thereby sustaining symmetrical scalds of both buttocks with well-defined "tide marks" reflecting the depth of immersion and "sock distribution" scalds of his ankles and feet as well (see Fig. 12.1).
- *Contact burns from hot objects*, in which the shape of the burn may suggest the object used ("branding"): e.g. clothes iron, kitchen utensil and hot metal end of a cigarette lighter. Multiple burns of uniform depth, size and shape may be present. Small round, deep, sharply demarcated burns may have been inflicted using a lit cigarette, often on parts of the body usually covered by clothing (e.g. buttocks, genitalia).

Clinical Features: Fractures

- Fractures are common in children, accounting for 8–12 % of childhood injuries. Twelve to 20 % of childhood fractures are attributed to physical abuse, far less than those due to nonaccidental trauma such as falls and road traffic accidents but a high enough proportion to warrant consideration of abuse in every case of fracture in a child [15].
- There is a strong inverse relationship between a child's age and the likelihood that a fracture is nonaccidental: the majority of abusive fractures in children are in those under 18 months old [16].

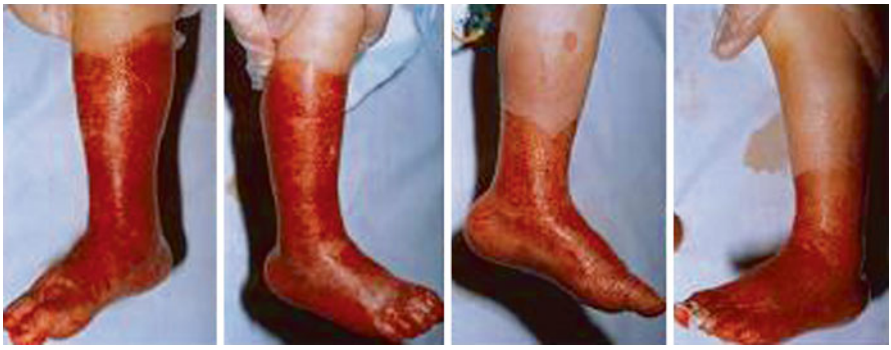

Fig. 12.1 Tide mark and sock distribution from immersion injury

Table 12.1 Strength of association of radiologic findings with child abuse in infants and toddlers

Specificity for child abuse	Type and location of fracture
High specificity	Classic metaphyseal lesions
	Rib fractures, especially posterior
	Scapular fractures
	Sternal fractures
Moderate specificity	Multiple fractures, especially bilateral
	Fractures at different stages of healing
	Epiphyseal separations
	Vertebral body fractures and subluxations
	Digital fractures
	Complex skull fractures
Common but low specificity (common in both abuse and accidental injuries)	Linear skull fractures
	Clavicle fractures
	Long bone shaft fractures
	Subperiosteal new bone formation

Adapted from Kleinman [17]

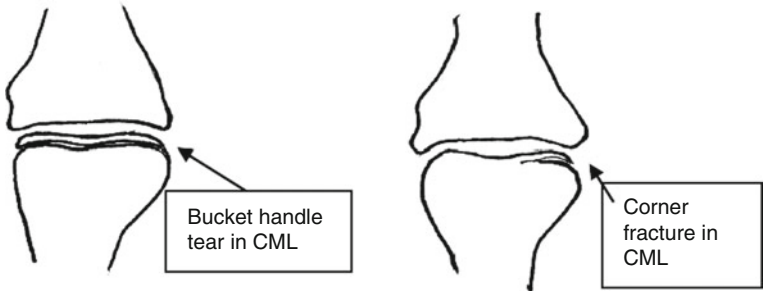

Fig. 12.2 Classic metaphyseal lesions in NAI

- Multiple fractures, especially if they are at different stages of healing, are strongly associated with abuse [16].
- Some forms of fractures are more strongly associated with abuse than others: Kleinman's classification (Table 12.1) is widely quoted and very useful [17]. It must however be remembered that no fracture is "pathognomonic" of child abuse. Fractures that are particularly associated with abuse are *rib fractures* (especially posteromedial rib fractures), *scapular fractures*, *sternal fractures* and classic metaphyseal lesions of the ends of long bones [17].
- *Classic metaphyseal lesions* (CMLs) are the most common long bone fractures found in fatally abused infants. They are planar fractures through the metaphysis, most commonly of the distal femur, proximal tibia or distal humerus; depending on the projection of the radiograph, they appear as "corner fractures" or "bucket handle fractures" [15] (see Fig. 12.2). Traction or torsion applied across the metaphysis, such as when an infant's limb is vigorously pulled or

twisted, is believed to be the causative mechanism, although the evidence for this has been challenged [18]. It should nevertheless be noted that breech delivery and splinting for congenital talipes equinovarus (club foot) may possibly cause CMLs, and metabolic bone disease may cause a similar radiological appearance.

- Excluding children involved in violent trauma/motor vehicle crashes, a meta-analysis estimated the probability of abuse being the cause of a *femoral fracture* as 0.43 (95 % confidence interval 0.32–0.54) [16].
- Supracondylar fractures of the humerus are usually accidental, although the possibility of abuse must still be considered, especially if the child is under 3 years old; *spiral/oblique fractures of the humerus*, suggesting a possible twisting mechanism, are more concerning for abuse [16].
- An estimated 30 % of paediatric *skull fractures* could be due to abuse, with some studies suggesting that skull fractures that are complex, multiple, bilateral or cross suture lines are significantly more common in abused children [16].
- Fractures usually associated with accidental causes can also be caused by child abuse.
- Differential diagnoses of abusive fractures and the role of *skeletal surveys* in the investigation of suspected abuse are discussed in subsequent sections.

Clinical Features: Abusive Head Injury

- Abusive head trauma (AHT) is the leading cause of traumatic death in infants, as well as the leading cause of death due to child abuse [19].
- The peak incidence of AHT occurs at the age of 3–4 months.
- The clinical symptoms and signs of AHT are often non-specific, such as poor feeding, decreased activity, irritability and vomiting; it is thus easily missed initially, especially as there is usually no reported mechanism of head injury.
- The infant or child may progress to develop apnoeas (with or without noticeable cyanosis), seizures, coma or cardiorespiratory arrest.
- Neuroradiological findings associated with AHT are subdural haemorrhage and cerebral ischaemia [19].
- Retinal haemorrhages are very strongly associated with AHT. A recent meta-analysis found that the presence of retinal haemorrhages in a head injured child strongly favours abuse over non-abuse (odds ratio 27.12, 95 % confidence interval 15.70–46.84) [19].
- The same authors found a very strong association between the absence of an adequate history to explain the head trauma with AHT (odds ratio 46.94 favouring abuse over non-abuse, 95 % confidence interval 12.91–170.63).
- The term "*shaken baby syndrome*" has been used to describe the triad of subdural haemorrhage, cerebral oedema and retinal haemorrhages. The presence of this triad is believed to strongly suggest abuse. Shear forces due to violent shaking of the infant's head are believed to cause tears in the fragile bridging veins in the

subdural space, leading to subdural bleeds. These forces are believed to also cause retinal haemorrhages, and acceleration/deceleration forces may account for the cerebral oedema. There is significant support for this from biomechanical studies and case series where perpetrators had admitted to only shaking infants who developed this triad of findings [20]. However, there have also been significant challenges to this theory in the scientific and legal communities. The American Academy of Paediatrics (AAP) holds the view that shaking is an important mechanism of AHT. However, recognising that blunt impact alone, or a combination of blunt impact and shaking, can also cause infant head injuries, the AAP holds the view that "shaken baby syndrome is a subset of AHT" and recommends that "paediatricians should use the term 'abusive head trauma' rather than a term that implies a single injury mechanism, such as shaken baby syndrome, in their diagnosis and medical communications" (2009 AAP policy statement) [20].

- Retinal haemorrhages while highly suggestive of shaken baby syndrome can also result from cardiopulmonary resuscitation (particularly in children less than 2 years of age), accidental head injury, blood dyscrasias and coagulation defects, severe hypertension, following birth trauma and post-seizure as well as from infections (rickettsia, cytomegalovirus and falciparum malaria) and from arterial aneurysms/arteriovenous malformations, etc.

Differential Diagnosis

- Always consider other causes when considering the possibility of nonaccidental injury.
- Bleeding disorders (e.g. haemophilia, von Willebrand disease, thrombocytopenic purpura) may present with multiple bruises, although not usually patterned bruising.
- Impetigo may be mistaken for cigarette burns; Steven-Johnson syndrome (SJS) or toxic epidermal necrosis (TEN) may present with lesions resembling scalds.
- Acid stool burns, such as those caused by the ingestion of senna laxative, can cause erythema and blistering of the buttocks mimicking immersion scalds. However, unlike immersion scalds, acid stool burns do not spare the perianal skin and intergluteal cleft and are less likely to have sharp demarcations.
- Osteogenesis imperfecta may present with multiple fractures or fractures after minimal trauma, usually accompanied by other clinical and radiological features of the disease (e.g. blue sclera in some forms, poor dentition, short stature, osteopenia on x-rays). Vitamin D deficiency rickets and osteopenia of prematurity may present with fractures in infants. Scurvy and osteomyelitis may mimic classic metaphyseal lesions [15].
- Cultural practices that do not put the child at significant harm, such as moxibustion and coining, are generally not considered as abuse by leading international child abuse experts the authors have spoken to.

Investigations

- Local guidelines may determine *where* radiological and laboratory investigations for child abuse are conducted (in the emergency department, in the paediatric clinic or as an inpatient) and exactly *which* tests are done.

Skeletal Survey

- Abused children presenting with various injuries (including one or more identified fractures) often also have clinically occult fractures resulting from abuse; a *skeletal survey* is a specialised set of at least 20 separate plain radiographs (*not* a single whole-body film) used to screen for these occult fractures in the workup of suspected abuse.
- The American Academy of Paediatrics recommends that a skeletal survey be done for:
  - All children with fractures suspected to be due to abuse
  - Children under 2 years old with any suspicious injuries [3]

More detailed guidelines, considering fracture types and age subgroups, have recently been proposed [21].

Screening for Bleeding Disorders

- For children presenting with *bruises* or intracranial haemorrhage suspected to be due to abuse, the American Academy of Paediatrics recommends a panel of:
  - Clotting screen and DIC panel (D/dimer and fibrinogen)
  - von Willebrand factor antigen
  - von Willebrand factor activity (ristocetin cofactor)
  - Factor VIII level
  - Factor IX level
  - Complete blood count with platelet count

Neuro-imaging

- An infant with suspected abusive head injury should be investigated with neuro-imaging modalities (computed tomography, magnetic resonance imaging) and also referred to an ophthalmologist early to look for retinal haemorrhages.

Management

- Protocols for the management of suspected/alleged nonaccidental injury are country and centre specific. In terms of evaluation of suspected abuse, guidelines from the American Academy of Paediatrics [3] and the UK National Institute for Health and Care Excellence (NICE) [4] are widely referred to beyond these respective countries. In many jurisdictions, healthcare providers are mandated by law to report a reasonable suspicion of child abuse to child protection services or the police. The procedures followed by these agencies upon receiving a report, and the role of various child abuse professionals throughout the process, vary between countries.
- In a child who is medically unstable, the emergency physician's priority is to stabilise the child/acutely treat the medical emergency, ahead of forensic examination [6].
- Apart from identifying possible abuse, investigating appropriately and providing medical treatment, the emergency physician has further key responsibilities of:
  - Ensuring the child's safety: this may involve admitting the child to hospital as a place of safety or securing a safe transfer to a paediatric hospital
  - Accurate and adequate documentation, as the medical records may be critical to subsequent child protection and law enforcement interventions
  - Unbiased communication with child protection and law enforcement agencies
  - Unbiased court testimony if called upon to testify in court [6]
- Where possible, an immediate child abuse paediatric consultation in the emergency department is ideal. Or else the child with suspected nonaccidental injury should be referred to a quaternary paediatric hospital with a specialist child abuse team.

A "Pathway" for the Emergency Department Evaluation and Management of Nonaccidental Injury: The "CALL DR" Mnemonic

The authors propose the novel mnemonic of "CALL DR" as an easily remembered guide to six key steps in the evaluation and management of nonaccidental injury. These steps are adapted from previously published guidance [3, 4]. See Table 12.2 for a safeguarding checklist:

C – Consider
A – Ask
L – Listen
L – Look
D – Document
R – Refer

Table 12.2 Safeguarding checklist

Is an interpreter needed?	Y	N
Has there been a delay in seeking medical help?	Y	N
Is there an unexplained or unwitnessed injury?	Y	N
Is there injury consistent with the history given?	Y	N
Is the injury consistent with the child's developmental stage?	Y	N
Are there any concerns about the child's reaction or behaviour?	Y	N
Are there any concerns about the child's care e.g. presentation, supervision?	Y	N
Are there concerns about the parent/child interaction?	Y	N
Is a safeguarding assessment required?	Y	N
If 'Yes' refer to Paeds ED Consultant or Paeds Registrar		

Consider

- Consider the possibility of child abuse in every consult with a child.
- Consider if the injury is consistent with the explanation given and the child's developmental age

Ask

- Ask how the injury occurred with open-ended questions, in a non-accusatory tone.
- Ask other relevant past medical history (previous injuries, chronic conditions), family history (bleeding disorders, bone disorders), developmental history, the child's temperament ("difficult" children are at higher risk of nonaccidental injury) and stressors in the family.

Listen

- Let the parent/caregiver, and also the child if he/she is able to, tell what happened in their own words.
- Listen attentively for points that fit the injury, as well as points that don't fit the injury.

Look

- Assess the injury/physical findings independently of the story and then decide if they are concordant or discordant. Don't try to make the injury fit the story.
- Look carefully for any other injuries or signs of possible abuse (not forgetting to check often-missed sites such as behind the ears or the groin).

- Look at the child's demeanour and interaction with the parent/caregiver (a withdrawn child or one clearly fearful of the parent/caregiver should raise concern).

Document

- Document in the clinical record exactly what was said or heard by whom and when and what was observed by you at the time of consultation.
- Document exact findings including location, nature, colour and measurements of lesions, using diagrams if possible. Consider using forensic photography where available.
- Document your concerns and why you are concerned.

Refer

- Refer to the appropriate team such as the paediatric consultant/child abuse paediatric team, social workers ± police according to local policies

Conclusion

Diligent and skilled evaluation along with non-judgemental communication is critical when a child is being evaluated for possible nonaccidental injury.

A child whose safety from further harm has not been adequately determined should not be discharged home and should be considered for admission whilst further enquiries are being carried out.

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Chapter 13
Paediatric and Newborn Resuscitation

Martin Gray and Nadeeja Koralage

Key Points
- Children have unique physiology which directs resuscitation.
- Most cardiac arrests in children are due to hypoxia.
- A systematic approach should be used when assessing children.

Introduction

Worldwide, 10.5 million children under 5 years of age die each year. Many children die of preventable causes [1]. Infectious diseases such as gastroenteritis, pneumonia and malaria are the major causes of child death in developing countries.

Resuscitation and Shock [2]

Resuscitation is the restoration of the functions of life after their loss in the presence of a shock state. Shock is the failure of tissue perfusion or of delivery of oxygen that will ultimately lead to cell death; this will in turn lead to the death of the organism if medical intervention aimed at reversing this process is not instituted. Even when life is preserved, morbidity from shock can affect many organ systems resulting in hypoxic ischaemic brain damage, acute kidney injury, gut ischaemia, cardiac failure and acute respiratory distress syndrome.

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Shock States

Hypovolaemic shock (haemorrhage, burns, dehydration)

Signs in children: tachycardia; low-volume pulse; cold, pale, clammy skin; tachypnoea; depressed conscious level and hypotension (preterminal in children)

Distributive shock (sepsis, anaphylaxis, neurogenic shock)

Two types

Warm shock: tachycardia, large-volume pulse and warm skin
Cold shock: tachycardia, low-volume pulse and cool skin

Hypotension in both scenarios is a late sign.

Cardiogenic shock (arrhythmia, heart failure)

Signs may be characterised by the nature of the arrhythmia ultimately leading to signs of low cardiac output and congestion of the lungs and viscera.

Obstructive shock (pneumothorax, cardiac tamponade, pulmonary embolism)

Signs of low cardiac output are reversed by removal of obstruction.

Principles of Fluid Resuscitation in Shock

It is not always possible to differentiate between the different shock types at the point of presentation. Once arrhythmia is excluded as a cause, a pragmatic approach to shock is to attempt fluid resuscitation as both a treatment and a diagnostic intervention. In most cases an aliquot of 20 ml/kg is the standard dose as an initial intervention. Close observation of the response to fluid can help to differentiate the shock state and guide your next move (i.e. drop in heart rate may suggest hypovolaemic or distributive shock, whereas increased heart rate might suggest cardiogenic shock). Tissue perfusion itself cannot be measured directly as an end point. Surrogate markers are used such as urine output (target of 1–2 ml/kg/h), mean arterial pressures (although this can be unreliable due to compensatory vasoconstriction) and central venous pressure trends.

Cardiorespiratory Arrest

If untreated, shock can lead to cardiorespiratory arrest. In children the outcome is extremely poor. The key to prevention is to recognise the pattern of illnesses that precede arrest and to treat them aggressively.

In the paediatric population, the most common cause of cardiorespiratory arrest is hypoxia. This can be caused by:

- Respiratory pathology – birth asphyxia, asthma, bronchiolitis and pneumonia
- Neurological dysfunction

Most other arrests are due to circulatory failure as depicted below (Fig. 13.1).

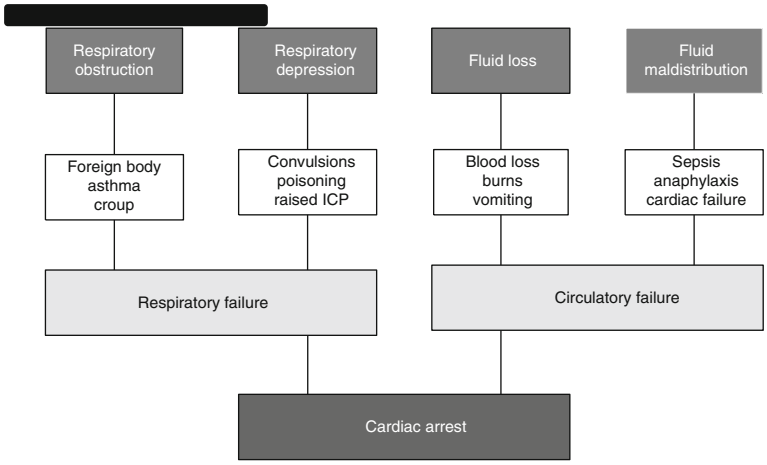

Fig. 13.1 Cardiac arrest in children: aetiologies (Adapted with permission [3])

Approach to Resuscitation

A child should first be assessed for responsiveness. This can be done by firmly holding the shoulders and squeezing and speaking loudly to assess for a response. An appropriate verbal response informs us that the airway is patent and the child has a sufficient blood pressure for mentation.

Airway

A hoarse voice or lack of a response tells us that there may be an obstruction or the airway is not patent. The airway should be examined to look for the cause of obstruction:

- A foreign body can be removed using McGill's forceps.
- Vomitus or blood can result in a noisy, gurgling respiratory effort and can be cleared by using a Yankauer catheter to direct suction, where directly visible.

Airway manoeuvres and adjuncts are simple techniques that can be used with great effect. In the unconscious patient, the aim is to maintain a patent airway, often obstructed by the tongue.

Chin lift in infants keeps the infants' head in the neutral position as overextension of the neck can obstruct the airway.

Head tilt and chin lift in children. Place one hand on the forehead to apply gentle pressure to bring the child's head into the sniffing position. Use the other hand to apply pressure under the mandible to bring the chin upwards slightly (Fig. 13.2).

If the head tilt/chin lift manoeuvre is not possible due to suspicion of c-spine injury, then a jaw thrust can be performed.

Jaw thrust – place the index and middle fingers underneath the angle of the mandible and lift the jaw forwards. This lifts the tongue away from the back of the throat.

During these manoeuvres always take care to only apply pressure to bony prominences and not soft tissues. Once an airway manoeuvre has been carried out, assess its effectiveness. Look, listen and feel for chest wall movement, breath sounds or the feel of breath against your cheek (Fig. 13.3). If supplemental oxygen is being applied, is the mask fogging with breath?

Airway adjuncts may also be used.

A Guedel airway can be sized by measuring it against the distance from the tragus of the ear to the corner of the mouth. It is inserted by opening the mouth and passing it over the tongue into the oropharynx. If a Guedel airway adjunct is tolerated, this would suggest that the child does not have a gag reflex and a definitive airway is likely to be needed.

If a Guedel is not tolerated, a nasopharyngeal airway can be used. Visual approximation can be used to pick the correct size, and this is passed through a nostril in a direction that is perpendicular to that of the plane of the child's face. It is theoretically possible to pass the airway through the cribriform plate into the brain if a base of skull fracture is present, which is why they are avoided in children who may have sustained a head injury. Both of these adjuncts can cause local trauma and bleeding and should be avoided if passage is difficult so as not to compromise the airway further.

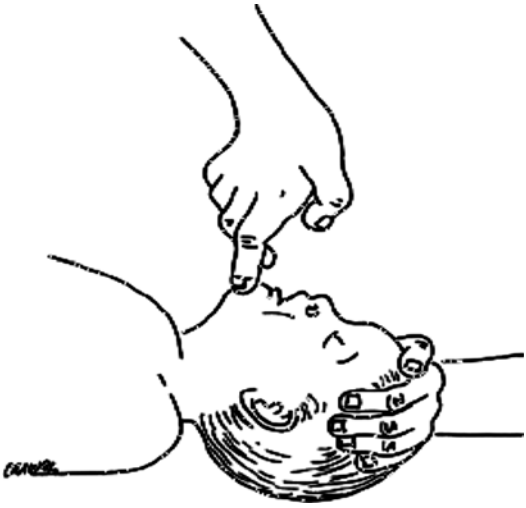

Fig. 13.2 Head tilt and chin lift in children

A child who cannot protect their own airway and who is at risk of aspiration will need intubation. This should be carried out by a practitioner who is experienced in intubating children as it has its unique technique, equipment and risks. Straight blade laryngoscopes are used to directly lift the epiglottis. This stimulation can cause a reflex bradycardia.

Breathing

Supplement high-flow oxygen via a non-rebreather mask with an oxygen reservoir.

Respiration should be assessed by inspection of the chest wall – looking for the depth and rate of breathing and signs of increased work of breathing (tracheal tug, intercostal and subcostal recession). An increased respiratory rate is sometimes the first sign of shock. Table 13.1 shows the normal resting respiratory rate for different age groups. Nasal flaring is suggestive of respiratory distress.

Inspiratory noise, stridor, suggests that there is a tracheal or laryngeal airway obstruction. Expiratory noises, or wheeze, point towards lower airway constriction. Grunting in infants is called by the need to air trap against a partially closed epiglottis

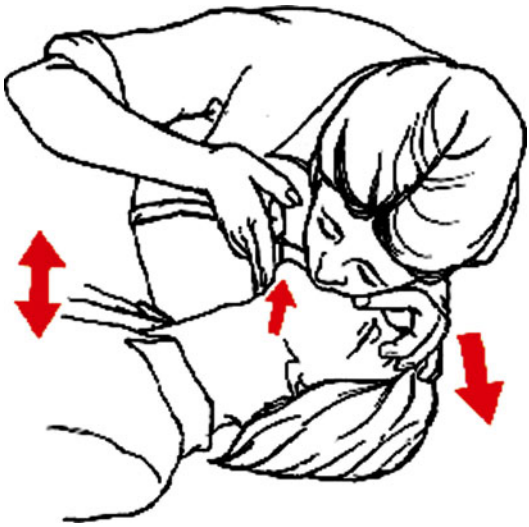

Fig. 13.3 Head tilt + chin lift + mouth-to-mouth breathing

Age (years)	Respiratory rate (breaths per minute)
<1	30–40
1–2	25–35
2–5	25–30
5–12	20–25
>12	15–20

Table 13.1 Normal respiration rates at rest by age

to prevent end-expiratory lower airway collapse in young children with stiff lungs. The volume of these added noises does not indicate the severity of the child's condition – in fact, a silent chest on auscultation is a worrying, preterminal sign.

Pulse oximetry is very helpful, especially when the child is breathing normal air. Keep in mind that normal levels may not reveal a child's true respiratory distress if they are breathing supplemental oxygen, and it is not a true marker of efficient ventilation, but oxygenation.

A hypoxic child may present as agitated or irritable. Central cyanosis is a late sign and will not occur until oxygen saturations are below 70 %. Hypercapnia can result in drowsiness or unconsciousness.

Carbon monoxide monitors are helpful to both monitor ventilation and confirm endotracheal tube placement. They can form part of the child's ventilator circuit.

In the absence of respiratory effort, or what appears to be ineffective breathing, five rescue breaths should be given via a bag-valve mask with supplementary oxygen.

A firm seal should be made around the child's mouth and nose and five slow breaths should be administered, checking for chest wall rise and fall with each. If there is an absence of chest wall movement, the child's position should be reassessed and optimised.

Circulation

Once rescue breaths have been administered if necessary, a central pulse should be assessed for rate, rhythm and character. In infants this should be done using the brachial artery and in older children using the carotid or femoral pulse.

Tachycardia can be an early sign of infection, shock, pain or respiratory distress. Bradycardia is a worrying, late sign of shock and suggestive of a preterminal state. Table 13.2 depicts the normal heart rates for different age groups.

A weak radial pulse and cool peripheries may be suggestive of poor perfusion, whereas a bounding pulse points towards carbon dioxide retention or sepsis.

Capillary refill assessment is carried out by blanching the skin by pressing the sternum firmly using a finger. This is held for 5 s before the finger is removed, and the seconds the skin takes to recolour are counted. A normal capillary refill is <2 s.

In the absence of a palpable pulse, or a rate of <60 beats per minute, chest compressions should be started immediately. This assessment should take no longer than 10 s (Fig. 13.4).

Table 13.2 Normal heart rates by age

Age (years)	Heart rate (beats)
<1	110–160
1–2	100–150
2–5	95–140
5–12	80–120
>12	60–100

Effective compressions are:

- Carried out with the child lying on their back on a flat surface.
- Done at a rate of 100–120 per minute.
- Pressure is applied over the lower half of the child's or infant's sternum.
- At a depth of a third of the patient's chest.
- With full recoil of the chest wall in between each compression.

In infants, the torso should be encircled with both hands (Fig. 13.5), with thumbs meeting over the lower half of the sternum to apply the pressure needed. This technique is best done when there is another rescuer to administer ventilations with a bag-valve mask.

If only one rescuer is present, a two-finger technique (Fig. 13.5) is used to apply compressions over the same site.

In children, the heel of one hand is placed over the lower half of the sternum, with the rescuers' elbows straight and their arm perpendicular to the child's chest.

In older children or adolescents, both hands can be used with fingers interlocking (Fig. 13.6).

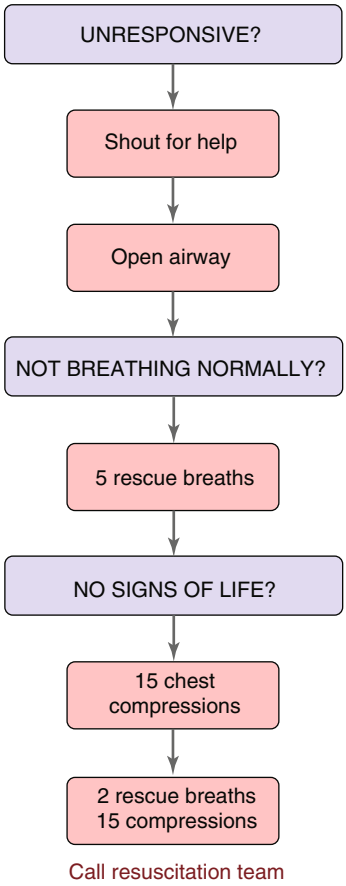

Fig. 13.4 Paediatric basic life support (Adapted with permission [3])

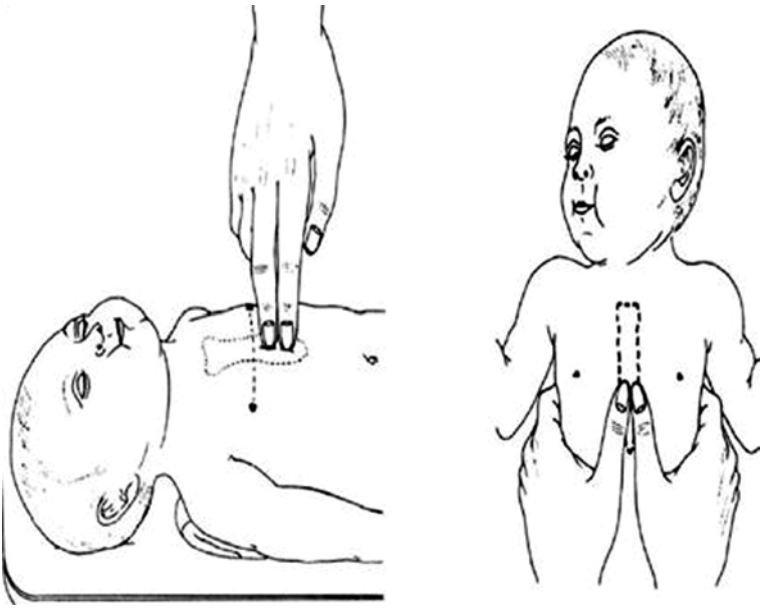

Fig. 13.5 Chest compression techniques in infants

Fig. 13.6 Chest compression techniques in adolescents

Compressions are carried out at a rate of 15 is to every two ventilations.

In a cardiac arrest, it is vital to ascertain the cardiac rhythm and perform advanced life support (Fig. 13.7).

Non-shockable Rhythms

Asystole

This rhythm is most commonly seen in children after prolonged period of hypoxia. Its appearance on the monitor is that of a wavy line. A completely flat line may indicate a disconnection so check the equipment.

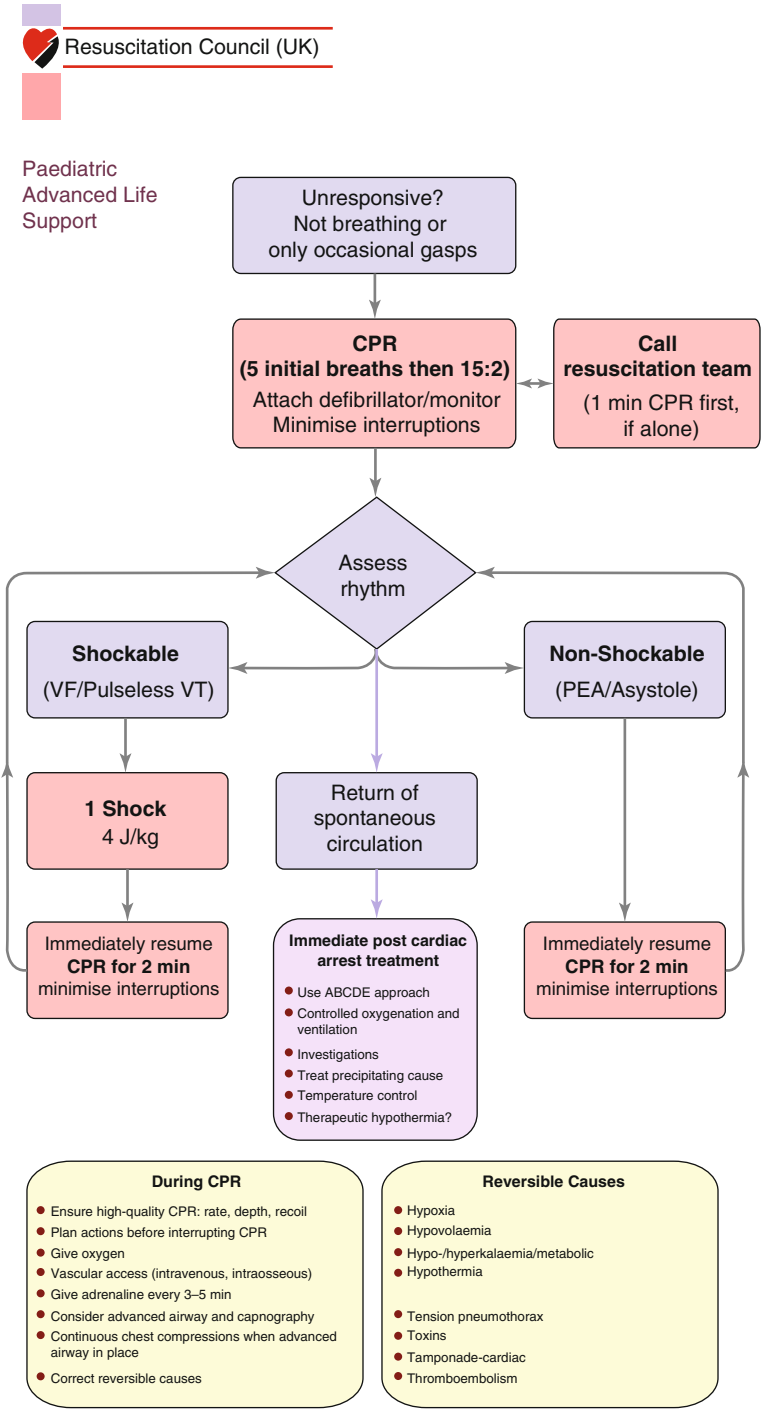

Fig. 13.7 Advanced paediatric life support algorithm [3]

Pulseless Electrical Activity (PEA)

Recognisable complexes in the absence of signs of life and a pulse are known as PEA. It may be associated with a reversible cause such as hypoxia, hypovolaemia, tension pneumothorax, cardiac tamponade, hypothermia and electrolyte disturbances. It is important to consider and treat these causes if they are suspected.

Both of these rhythms share the same management – effective compressions with ventilations. It is important that a competent clinician intubates the patient as soon as possible to allow for continuous ventilations (10–12) alongside compressions.

Adrenaline should be given as soon as a diagnosis of asystole or PEA is made. Ten micrograms/kilogram is given. This dose is repeated every 3–5 min until there are signs of life or a palpable pulse over 60 bpm.

Shockable Rhythms

Ventricular Tachycardia

This is a broad complex tachycardia which in children is mostly associated with no cardiac output.

Ventricular Fibrillation

A chaotic, irregular waveform is never associated with a cardiac output.

These rhythms require a DC shock of 4 J/kg. Following the shock, compressions and ventilations should be restarted immediately as even if a shock is successful, the first few seconds following are unlikely to result in a significant cardiac output. Adrenaline (10 μg/kg) is given after the third shock (IV or IO) and then administered with alternate cycles.

Amiodarone is given after the third and fifth shock only (5 μg/kg IV or IO).

Disability

A child's alertness can be assessed by using 'AVPU'. This is a technique which formalises whether the child is alert or responding to verbal or painful stimulus or is unconscious. A response to only pain suggests that the patient's GCS is 8 or less.

Pupils and their reflexes should also be assessed at this stage. Very dilated or pinpoint pupils with sluggish reactions are suggestive of possible overdose of benzodiazepines or opiates. Dilated pupils which are unreactive following trauma may indicate raised intracranial pressure and compression of the ophthalmic nerve.

In children who are unwell, it is vital to check blood glucose. Insulin-dependent diabetes can present with vomiting and malaise or concurrent infection. Conversely, seizures can be caused by low blood sugar levels. Low blood glucose (<3 mmol/l) is treated with a buccal glucose gel or an IV bolus of 2 ml/kg 10 % glucose.

Remember to consider and treat infective causes of reduced GCS.

Exposure

Due to their high surface area, if exposed for examination, paediatric patients lose body heat rapidly. Care should be taken to avoid the patient becoming hypothermic.

Pyrexia and hypothermia can indicate an infective cause.

A top-to-toe examination should be carried out to look for focuses of infection and rashes which can evolve quickly. Petechiae and purpura should direct the clinician to possible life-threatening bacteraemia. In some cases, a few petechiae can be seen following prolonged vomiting, but these can only be attributed to this cause if they are above the nipple line and other causes have been excluded.

While examining and treating children, it is important to always be mindful of nonaccidental injury.

Reversible Causes

Consider the possible reversible causes of cardiac arrest 4 Hs and 4 Ts as illustrated in Fig. 13.7.

Newborn Resuscitation

Contractions which push a baby through the birth canal result in periods of relative hypoxia. Before birth, babies' lungs are filled with fluid, and as a result, newborn babies are far more likely to need resuscitation due to respiratory, rather than cardiac, origin.

Dry, Warm and Assess the Baby

Wet babies will rapidly lose heat which in turn will put them at greater risks of metabolic acidosis. Dry and warm the baby using towels, under a heat lamp. Remove used, wet towels and wrap in a dry towel.

During this time, assess the baby's appearance (pink or blue in colour) and tone. (Is it limp, floppy or appears to be significantly preterm?)

Rubbing the baby dry will cause stimulation and this is normally enough to elicit normal breathing and crying.

In the absence of normal breathing, or with a heart rate of below 100 beats per minute, rescue breaths are required. Using a stethoscope is the most accurate method of assessing the heart rate.

Inflation Breaths and Compressions

Ensuring the newborn's position is optimal (neutral position) to the airway. Most newborn babies will have a prominent occiput that will flex the neck when the baby is placed on a flat surface. To overcome this, place a folded towel under the shoulders of the baby as shown in Fig. 13.8. Take extreme care not to overextend the neck as this too will compromise the baby's airway.

Suction can be used to remove blood, meconium or other fluid. Gentle suctioning using a soft catheter offers further stimulation. If no respiratory attempts are made, administer five inflation breaths.

This can be done using a soft face mask and t-piece or self-inflating bag-valve (Fig. 13.9) mask with supplementary oxygen of 4–6 l. Each breath should last for 2–3 s using no more than 30 cm of water pressure, and the chest wall should be seen to rise a little.

After five inflation breaths, reassess for spontaneous respiratory effort and heart rate. If there is no improvement, repeat the five inflation breaths, after repeating airway opening manoeuvres.

If the heart rate does not improve after this, compressions are necessary (see earlier for technique). Perform three chest compressions to each breath. The rate should be at 120 compressions per minute. Reassess the heart rate every 30 s. Follow the newborn life support algorithm as shown in Fig. 13.10.

If there is no improvement, certain drugs (BAD mnemonic) can be used to reverse intracardiac acidosis, increase volume and cardiac output and correct hypoglycaemia via umbilical venous catheter.

Fig. 13.8 Optimal airway positioning of a newborn

Fig. 13.9 BVM ventilation of the newborn

*B*icarbonate –

1–2 mmol/kg (2–4 ml/kg of 4.2 %)

This can partially correct the baby's metabolic acidosis to improve cardiac function.

*A*drenaline –

10 μg/kg (0.1 ml/kg of 1:10,000)

This can also be given via the intra-osseous route.

*D*extrose –

250 mg/kg (2.5 ml/kg of 10 %)

Blood glucose levels decrease after birth and should be managed with an infusion following an initial bolus.

Crystalloid infusion – 10 ml/kg

Bradycardia may respond to a fluid bolus. If the primary problem is hypovolaemia, a subsequent blood transfusion may be necessary.

If there is no improvement in the baby's condition after drugs are given, the outcome is likely to be poor.

It is vital to seek appropriate help when treating a seriously unwell baby. Unfortunately, resuscitation attempts are often unsuccessful once cardiopulmonary resuscitation is required. A decision to stop resuscitation attempts is a difficult one, but prolonged attempts are unlikely to be successful. If they wish to be present, it is appropriate for the child's parents to witness the attempts made to resuscitate their child.

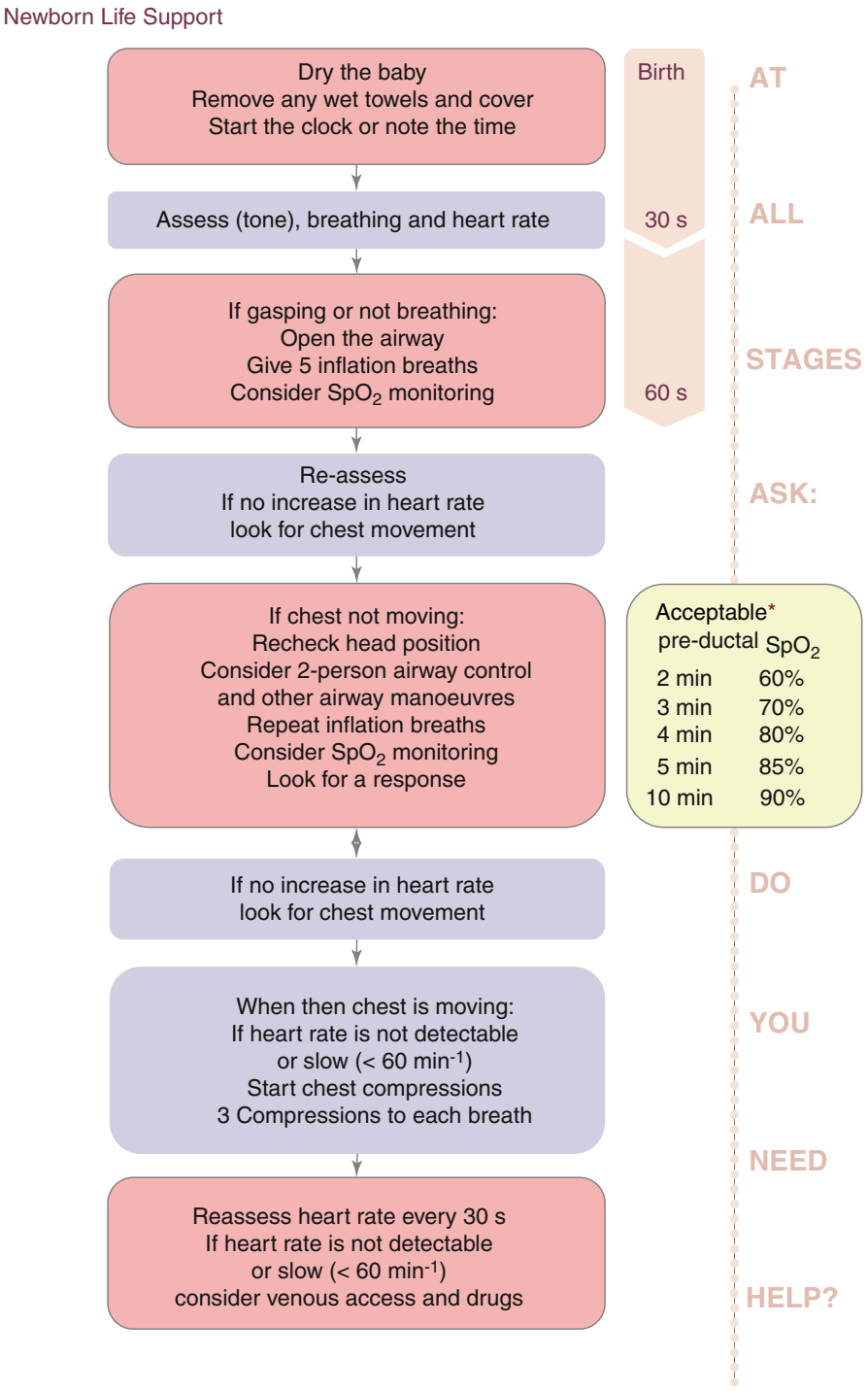

Fig. 13.10 Newborn life support algorithm (Adapted with permission [4])

A return of spontaneous circulation calls for a reassessment of all systems. The child should be transferred, when safe to do so, to an appropriate area for their ongoing stabilisation and care.

Aide memoire	
Drugs and other useful calculations (WET FLAG) in paediatric resuscitation	
Adrenaline	
Cardiac arrest (IV)	0.1 mg/kg (1:10,000)
As soon as in asystolic cardiac arrest	
After third shock in VF/VT arrest	
Anaphylaxis (IM)	>12 years of age 0.5 mg (1:1,000)
	6–12 years of age 0.30 mg (1:1,000)
	<6 years of age 0.15 mg (1:1,000)
Amiodarone after 3rd shock	
Pulseless VT and VF (IV)	5 mg/kg
Bicarbonate	1 ml/kg of 8.4 % solution if required
Calcium	0.3 ml/kg of 10 % calcium gluconate
Hyperkalaemia, hypocalcaemia	
Dextrose	2 ml/kg of 10 % solution
Magnesium	25–50 mg/kg
Torsades de pointes (form of VF)	

*W*eight: 0–12 months = 0.5 × age in months + 4
1–5 years = 2 × age in years + 8
6–12 years = 3 × age in years + 7
*E*nergy = 4 J/kg
*T*racheal tube:
Int. diameter (mm) = (age/4) + 4
Length (oral) (cm) = (age/2) + 12
Length (nasal) (cm) = (age/2) + 15
*F*luid bolus = 20 ml/kg
(aliquots of 10 ml/kg in trauma)
*L*orazepam = 0.1 mg/kg
(maximum of 4 mg)
*A*drenaline = 0.1 ml/kg of 1:10,000
*G*lucose = 2 ml/kg 10 % dextrose

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Chapter 14
Respiratory Emergencies in Children

S. Thangavelu, R.C. Sharada, and N. Balamurugan

Key Points

- Focused clinical observation is the key in the initial recognition of respiratory distress in an acutely ill child.
- Early diagnosis and urgent treatment improve the chances of recovery in critically ill child, presenting with respiratory distress or failure.
- Diagnosing the underlying aetiology is usually possible on clinical grounds, and laboratory investigations are very few and are carried out only after the initial stabilisation.
- Monitoring and management in PICU particularly ventilatory support for both invasive and non-invasive has improved the prognosis to a greater extent.

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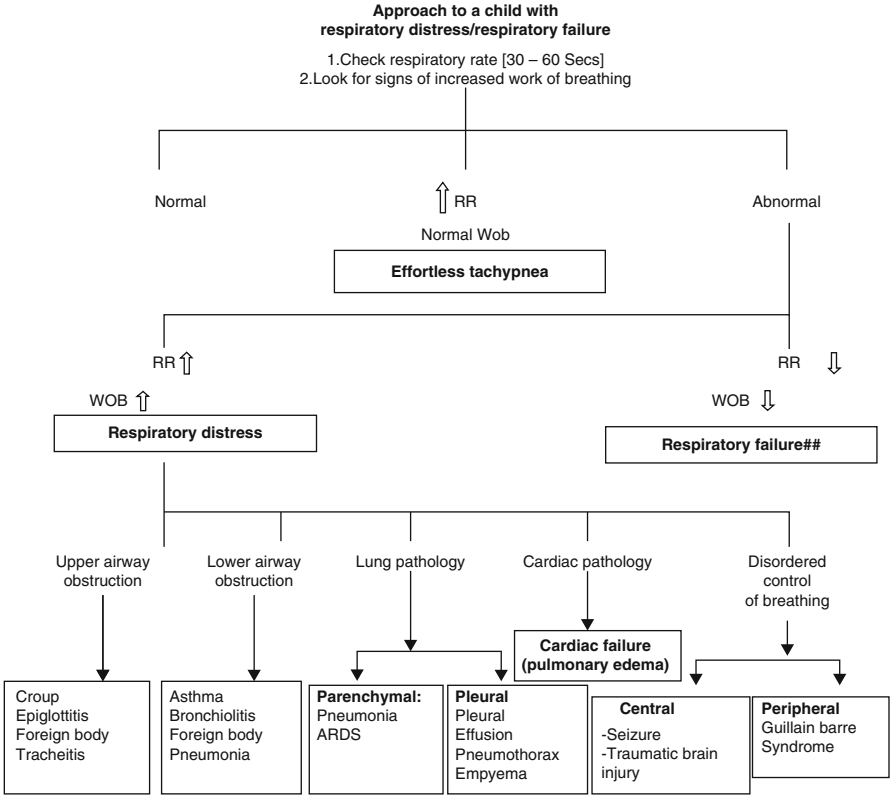
An Approach to a Child with Respiratory Distress and Respiratory Failure

Introduction

Fever and respiratory distress are the most common reasons for hospital visit by an acutely ill child. A child with respiratory distress (RD) can quickly worsen to respiratory failure (RF) and finally to cardiac arrest if left untreated [1]. Hence a precise recognition and management is essential (Flowchart 14.1). As a part of the initial primary survey, look for the three "E"– effort, efficacy and effect of breathing [2].

- *Effort of breathing* – Increased effort or work of breathing if the following is present: tachypnoea, sternal/subcostal/intercostals recession, use of accessory muscles, flaring of ala nasi and stridor/wheeze/grunt. Subcostal recession is more significant than intercostal recession. Bradypnoea is a sign of exhaustion and suggests impending respiratory arrest. Increased work of breathing may be absent in central causes like CNS depression and peripheral nervous system failure as in Guillain-Barre syndrome. Check respiratory rate for 30–60 s (Table 14.1).



Flowchart 14.1 Approach to a child with respiratory distress/respiratory failure

Table 14.1 Differences between effortless tachypnoea and respiratory distress

Effortless tachypnoea	Respiratory distress
1. RR alone increased	1. RR is increased
2. WOB normal	2. WOB increased
3. No noisy breathing	3. Noisy breathing may be present
4. No respiratory findings on auscultation	4. Diminished breath sounds, crepts or wheeze on auscultation
5. SpO_2 may be normal in room air	5. SpO_2 may be decreased
6. Occurs due to non-respiratory causes (DKA, renal failure, poisoning with salicylates or isoniazid, etc.)	6. Occurs due to respiratory causes

- *Efficacy* – Impaired efficacy of breathing is suggested by reduced chest expansion, diminished air entry and reduced SpO_2 on pulse oximeter.
- *Effort of breathing* – Cyanosis, tachycardia and altered mental status such as confusion or agitation would suggest reduced oxygen delivery. As per Integrated Management of Neonatal and Childhood Illnesses (IMNCI), fast respiratory rate (RR) was found to be the only clinical sign with high specificity and sensitivity in the diagnosis of pneumonia. RR has to be counted meticulously for 30–60 s in every child seeking medical help. Bradycardia in the presence of a respiratory distress would suggest impending cardiac arrest.

Underlying Causes of Respiratory Distress/Failure

There are many causes for respiratory distress, important ones are listed below:

1. Upper airway obstruction: Viral croup, epiglottitis, diphtheria, bacterial tracheitis, retropharyngeal abscess
2. Lower airway obstruction: Bronchiolitis, viral wheeze, asthma and foreign body airway
3. Lung parenchymal disease: Pneumonia, ARDS, collapse
4. Pleural disease: Pneumothorax (Fig. 14.1)
5. Cardiac illness: Pulmonary oedema (Fig. 14.2)
6. Neurological causes for pump failure or respiratory failure: Central causes – drug poisoning, traumatic brain injury, Guillain-Barre syndrome, etc.

Relevant Questions and Stepwise Management

Following questions should be raised, and answers sought while evaluating a child with respiratory distress or respiratory failure.

1. Does the child have features of respiratory failure or respiratory distress?
2. What is the initial management?
3. What is the anatomical diagnosis?

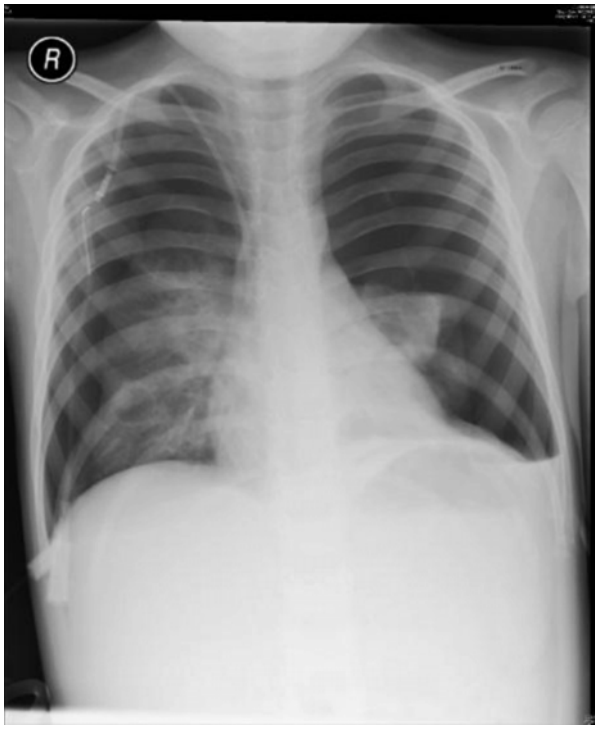


Fig. 14.1 Bilateral pneumothorax

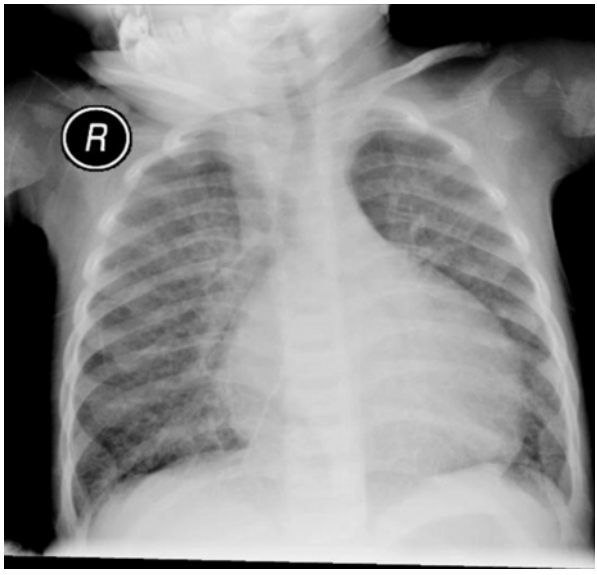


Fig. 14.2 Vitamin D deficiency cardiomyopathy with features of cardiac failure

Table 14.2 Identifying the anatomical site based on clinical signs

Anatomical diagnosis	Noisy breathing	Rise in respiratory rate	Predominant retractions	Resonance	Others
1. Upper AW obstruction	Stridor	++	Suprasternal sternal	Normal	
2. Lower AW obstruction	Wheeze	+++	Intercostal recession (ICR), subcostal (SCR)	Normal	
3.a. Lung parenchymal	Grunting	++++	ICR, SCR	Impaired	
3.b. Pleural	Same	Same	Same	Stony dullness	
3.c. Cardiac	Same	Same	Same	Normal	Gallop, murmur, hepatomegaly, oedema
4. Disordered control of breathing	No	Normal or bradypnoea	No	Normal	Shallow breathing, see-saw respiration, low voice volume

4. Which laboratory tests need to be done?
5. What is the specific management plan?

RD is an early stage of physiological disturbance. After the initial steps to correct hypoxia (this may require high-flow nasal cannula or non-invasive ventilation or intubation), specific management should be provided based on the underlying illness. Respiratory rate, respiratory effort, level of consciousness, trends in oxygen saturation via pulse oximeter (SpO_2), response to oxygen therapy and looking for signs of improvement or worsening are the most important components of monitoring.

Exact anatomical site of the underlying disease can be inferred by careful stepwise evaluation (Table 14.2).

- If the child is presenting with wheeze, absence of fever, or cardiac symptoms and past history of response to nebulisation, it will support the diagnosis of under-five wheeze or asthma in children above 5 years.
- Child (6 months to 2 years of age) with rhinorrhoea, low-grade fever, cough followed by noisy breathing, stridor and sternal and suprasternal retractions – viral croup causing stridor.
- Child above 3 years of age with high-grade fever, soft stridor, toxaemia, drooling, dysphagia with sniffing position – epiglottitis, rare but potentially fatal disease.
- Toddler developing breathlessness for the first time, dramatic onset, found to be playing with small round objects such as a button battery or a coin (Fig. 14.3) – choking due to foreign body in the airway presenting with obstructive emphysema or collapse.
- Child with high-grade fever, cough, fast breathing, grunting, subcostal and intercostal retractions, no past history of breathlessness – pneumonia. Suspect empyema if there is stony dullness and absent breath sounds on one side.

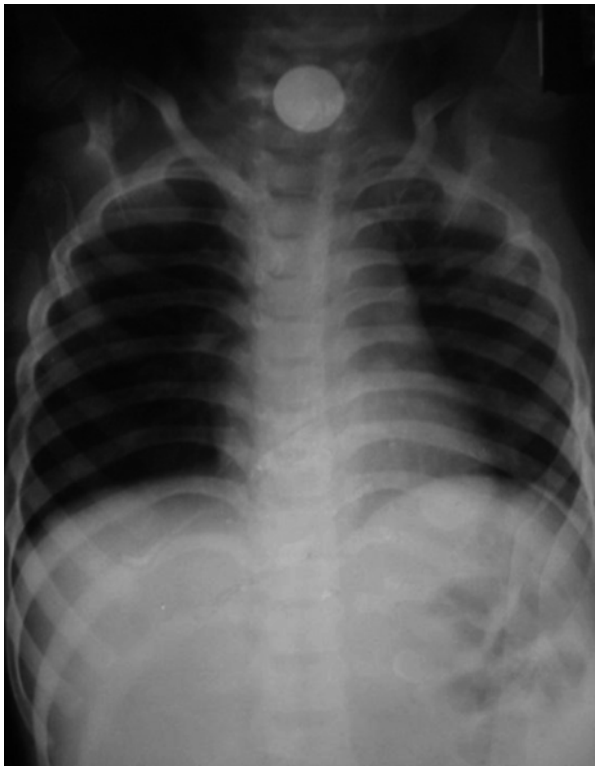


Fig. 14.3 Button Battery in the cricopharynx

- Child with acute-onset breathlessness, grunting without fever, lethargy, puffiness of face, disproportionate tachycardia, gallop with or without murmur, hepatomegaly – myocarditis or dilated cardiomyopathy.
- With the above said features, if the child has pericardial rub or muffled breath sounds and pulsus paradoxus suspect pericardial effusion.
- If the child has low voice volume, floppiness, weak cough, bradypnoea, see-saw breathing and shallow breathing, it is indicative of acute flaccid paralysis affecting respiratory muscles – respiratory failure due to disordered control of breathing.
- Similar situation in a different presentation will be seen in a child with acute seizures.

Laboratory Investigations

- Common indications for CXR in a child with respiratory distress are pneumonia, pulmonary oedema, suspected pneumothorax and foreign body inhalation. Cardiomegaly, collapse or congenital anomalies like eventration of diaphragm, congenital lobar emphysema, bronchogenic cyst or diaphragmatic hernia are sometimes seen as surprise.

- See chapter on Stridor for other disease-specific investigations.
- Blood counts and blood culture: Any radiological shadow in the background of fever is an indication for antibiotic in paediatric emergency department, but FBC and CRP will aid in suggesting a bacterial aetiology if not confirming. Blood culture is rarely positive in pneumonia unless there is septicaemia.
- In viral infections like H1N1, PCR of respiratory tract secretions is a useful investigation when a child presents with pneumonia during an epidemic situation particularly with normal WBC count and negative CRP.

Management

Specific management:

1. Management of asthma – see relevant section in this chapter.
2. For management of croup – see relevant section in this chapter.
3. Once the diagnosis of epiglottitis is suspected, PICU team, ENT surgeon and anaesthetist are involved in the management early. Antibiotics such cefotaxime should be given early. Child should be admitted in high dependency unit (HDU) or paediatric intensive care unit (PICU). Airway has to be secured early by intubation or tracheostomy because they can deteriorate very fast.
4. If pneumothorax or empyema is suspected, diagnosis should be confirmed by CXR and ultrasonography of the chest. Immediate needle thoracocentesis will relieve the respiratory distress, and it should be followed by intercostal drainage.
5. Appropriate antibiotics to treat the underlying pneumonia or empyema.
6. For management of foreign body, see relevant chapter under ENT.

Prognosis

In most of the situations, early recognition and management in HDU or PICU will help in good recovery.

Asthma in Children

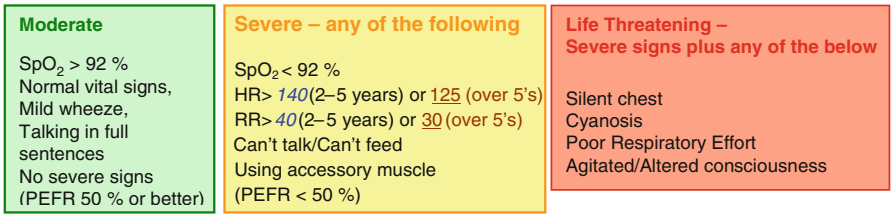
Asthma is a cause of reversible airway obstruction secondary to chronic airway inflammation and bronchial hyperreactivity. Approximately 10 % of the children are affected, and about 50 % of them will attend their GP or an ED before the age of 10 years. They often present with wheeze and shortness of breath. Occasionally the presenting complaint is that of cough at night or early morning, cough brought

on by exercise/cold weather and limitation in carrying out sporting activities (exercise tolerance).

- Look out for a family history of asthma or atopic disease such as hay fever, eczema or allergy.
- Based on the number of episodes experienced, asthma can be classified into three subgroups:

  Infrequent (<4 episodes/year) – three out of four asthmatics will fall into this category (75 % of cases).
  Frequent (episodes every 2–4 weeks) – one in five asthmatics will fall into this category (20 % of cases).
  Persistent (three or more episodes every week) – 1 in 20 asthmatics will fall into this category (5 % of cases).

Severity of Asthma [3]



Management

- Salbutamol 10 puffs via spacer for the moderate to salbutamol 2.5 (2–5 years of age)–5 mg (over 5's) nebulised (in oxygen) every 20 min for 1–2 h for the severe/life-threatening types.
- Consider adding ipratropium 250 mcg every 20 min for 1–2 h.
- Continuous oxygen by facemask or nasal cannula.
- Give prednisolone 10 mg <2 years, 20 mg or 30–40 mg (max 40 mg unless on regular oral steroids) within 1 h or hydrocortisone 4 mg/kg iv.
- Reassess after 15 min.
- Consider adding 150 mg MgSO4 to each nebuliser in the first hour if life-threatening in severity.

If poor response/deteriorating, call ED consultant, consultant general paediatrician and/or PICU team.

Consider in this order:

1. Salbutamol 15 mcg/kg IV bolus (max 250 mcg) or 5 mcg/kg in under 2s

   Reassess.

2. Magnesium 40 mg/kg IV bolus (max 2 g) over 30 min

   Reassess.

3. Salbutamol IV infusion (1–2 mcg/kg/min)

   Consider CXR.
   Blood gas and measure serum K+.
   ECG monitoring.

Indications for Ventilation

- Exhaustion.
- Worsening hypoxia.
- RR >60 despite treatment or falling RR without clinical improvement.
- Normal or rising CO_2.
- Prepare fluid bolus of 20 ml/kg and adrenaline bolus of 0.1 ml/kg of 1:10,000.

Induction

- Atropine 20 mcg/kg
- Ketamine 1–2 mg/kg or thiopentone 1–2 mg/kg
- Suxamethonium 1–2 mg/kg

Ventilation

- Largest possible cuffed ET tube
- Permissive hypercapnoea: PH >7.2, keep PIP <45 mmHg and TV 5–10 ml/kg, RR 10–15. PEEP 5–7

Sedation

- Midazolam and fentanyl
- Regular suctioning and chest physio

Discharge Criteria

- *All severe and life-threatening cases should be admitted.*
- No life-threatening signs at any time.
- On 4 hourly bronchodilators.
- SpO2 95 % or higher.

Discharge Plan [3]

- Weaning regime of bronchodilator.
- Prednisolone for 3 days.
- Consider regular inhaled steroids, i.e. beclomethasone 100–200 mcg BD, if:
  - Needing B2 agonist >3 times per week
  - Nocturnal symptoms disturbing sleep more than once per week
  - Exacerbations requiring oral steroids

In children under 5 years with viral-associated wheeze, steroids (oral and inhaled) may not be beneficial.

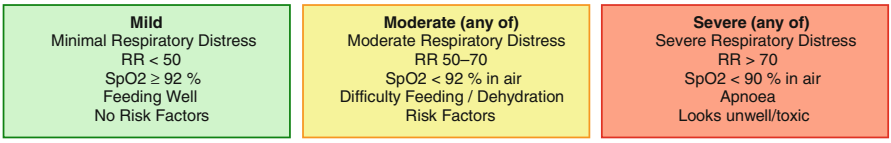
Follow-Up [3]

- GP follow-up within 48 h
- If >2 attendances to ED within 1 year or two courses of oral steroid, refer to asthma clinic for urgent review.

Bronchiolitis

Bronchiolitis is a common respiratory tract infection in infancy and a common cause for admission in young children and often leads to a lot of morbidity in infants. It is typically caused by viruses and the common aetiologic agent being the respiratory syncytial virus. Usual age group is 2 months to 2 years.

Bronchiolitis is practically defined as the first episode of wheezing in a child younger than 12–24 months who has physical findings of a viral respiratory infection and has no other explanation for the wheezing, such as pneumonia or atopy [4]. It is usually a seasonal viral illness characterised by low-grade fever, nasal discharge and dry, wheezy cough. On examination there are fine inspiratory crackles and expiratory wheeze. Within 1–2 days of these prodromal symptoms, the cough worsens and child may also develop rapid respiration, chest retractions, irritability, poor feeding and vomiting. Though, in majority of cases, the disease remains mild and recovery starts in 3–5 days, some of these children may continue to worsen and become sick, and prompt management is needed.



Factors Increasing the Likelihood of Severe Disease

- Chronological age less than 6 weeks
- History of apnoea before assessment
- Known structural cardiac anomaly (e.g. VSD)
- Known pre-existing lung disease (e.g. cystic fibrosis)
- Significant prematurity (<32 weeks)

The diagnosis of bronchiolitis is mostly clinical, and laboratory investigations have a limited role in diagnosis and management.

Bloods	*Bloods (FBC, U&E and CRP) should only be done if child is having IV line inserted. Blood cultures if fever >39 or in infants <60 days old*
CXR	If deteriorating/needing ventilatory support/needing HDU/PICU or prolonged or atypical course. Most don't need them
CapGas	If tiring or poor saturations despite FiO_2 >40 % (ABG is alternative)
NPA	For viral immunofluorescence/rapid antigen test if decision to admit

The current management primarily consists of supportive care:

- Widely accepted line of management is adequate hydration, supplemental oxygen and careful monitoring of vital signs and SpO_2.
- At this point, there is no other specific treatment for bronchiolitis for which there is a strong or convincing evidence of effectiveness and the role of each modality is debatable [5].
- Hypertonic saline nebulisation with salbutamol 2.5 mg may reduce the length of stay for admitted children.
- Consider nebulised epinephrine if considering ventilation.
- Consider 1–3-month course of montelukast (4 mg at night) [6] as it may reduce risk of relapse/recurrent wheeze.

Acute Laryngotracheobronchitis (Croup)

This is one of the most common causes of stridor in children presenting to the emergency department. It is most commonly due to a viral illness caused by parainfluenza virus which accounts for 80 % of the cases [7].

Children often present with typical barking cough and harsh stridor. These symptoms are often preceded by a viral prodrome of low-grade fever, running nose and cough. Usually the children are not toxic and they do not have drooling of saliva. The illness usually lasts for 2–5 days. If obstruction worsens, the child becomes increasingly tachypnoeic with respiratory distress indicated by retractions. Rarely respiratory failure occurs. Cyanosis, tachycardia and altered level of consciousness if present indicate that there is hypoxia and it needs prompt management. Westley croup score

is helpful in assessing the severity [8]. Croup is essentially a clinical diagnosis. Steeple sign seen in x-ray is seen in croup but is not pathognomic of the condition. Chest x-ray is done only when there are chest retractions and diagnosis is in doubt.

Treatment

- Keep the child in the mother's lap. Administer oxygen in a nonthreatening manner if needed.
- *Steroids* [9] are the drug of choice in managing croup. There is role for IV, IM, oral and inhaled corticosteroids. Their action usually starts 1–2 h after administration. Popularly a single oral or intramuscular dose of dexamethasone 0.15 mg/kg for the mild variety up to 0.6 mg/kg in severe cases is used in the emergency room/OP and is sufficient. Rarely the second dose is administered after 8–12 h based on clinical need. Oral prednisolone 1–2 mg/kg/day for 24 h is another option. Inhaled budesonide 2 mg in 3 ml normal saline also has rapid effect and is found to be useful.
- Epinephrine nebulisation is used in cases of moderate to severe croup, whenever there is evidence of respiratory distress and retractions are present even at rest. Nebulised adrenaline (epinephrine) solution 1 in 1,000 (1 mg/mL) should be given with close clinical monitoring in a dose of 400 μg/kg (maximum 5 mg) repeated after 30 min if necessary. Administration of three doses of nebulised adrenaline within 60 min has caused dysrhythmia and myocardial infarction in children. The effects of nebulised adrenaline last 2–3 h; the child should be closely monitored/observed for at least 4 h after giving.
- There is no role for antibiotics unless when croup is thought to be caused by a *Mycoplasma pneumoniae* infection.

Indications for ventilations in croup
Exhaustion
Worsening hypoxia
RR>60 despite treatment OR falling RR without clinical improvement
Normal or rising CO_2

Westley croup score						
	0	1	2	3	4	5
Stridor	Nil	When agitated	At rest			
Intercostal recession	Nil	Mild	Moderate	Severe		
Decreased air entry	Normal	Mild	Severe			
Cyanosis	None				When agitated	At rest
Consciousness level	Normal					Altered

Possible score 0–17: 0–3 = mild croup, 4–6 = moderate croup, >6 = severe croup

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Chapter 15
Surgical Emergencies in Children

Shanthi Sangareddi, Saravanakumar Paramalingam, and Zahid Mukhtar

Abdominal Pain

Key Points
- Initial stabilisation of the vitals is important followed by prompt surgical referral.
- Medical conditions can present with acute abdominal symptoms, and it is important to consider these prior to surgical referral.

Introduction

Abdominal pain is a common complaint for which children are brought to the emergency department. The cause of abdominal pain can be benign or life-threatening. It is the responsibility of the emergency physician to accurately assess and arrive at a probable diagnosis. The patient requires initial stabilisation and then referral to a surgeon if a surgical cause is suspected. Severe abdominal pain lasting for more than 6 h constitutes an "acute abdomen" [1].

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Table 15.1 Causes of abdominal pain

Acute surgical	Intra-abdominal medical	Extra-abdominal causes	Systemic illness
Appendicitis	Acute gastroenteritis	Tonsillitis	Diabetic ketoacidosis
Intestinal obstruction	Urinary tract infection	Streptococcal pharyngitis	Dengue
Visceral perforation	Peptic ulcer disease	Viral syndrome	Henoch-Schonlein purpura (HSP)
Intussusception	Renal colic	Lower lobe pneumonia	Lead poisoning
Malrotation ± volvulus	Worm infestation	Abdominal wall abscess	Porphyria
Incarcerated hernia	Constipation	Contusion of abdominal muscle or soft tissue	Heart failure
Acute scrotum (including testicular torsion)	Spontaneous bacterial peritonitis		Sepsis
Hirschsprung's disease	Pancreatitis		Infectious mononucleosis
Ovarian torsion	Cholecystitis		Abdominal epilepsy
Traumatic injuries	Gastroesophageal reflux disease		Migraine
	Acute retention of urine		
	Infantile colic		

Pathophysiology

Abdominal pain is transmitted by two types of fibres. Type A nerve fibre transmits sharp pain from skin and muscle. Type C fibre carries impulses from viscera, peritoneum and muscle and produces dull, poorly localised pain. These fibres synapse in the dorsal root ganglia; some afferent fibres cross and travel in the spinothalamic tract to the postcentral gyrus. In the gut pain is often due to stretching or tension. Ischaemic pain is due to tissue metabolites released near nerve endings.

Common Causes of Acute Abdominal Pain

Abdominal pain can be due to medical or surgical causes. Most of the medical causes of abdominal pain are due to systemic diseases or extra-abdominal causes. Table 15.1 shows the different causes of abdominal pain [2].

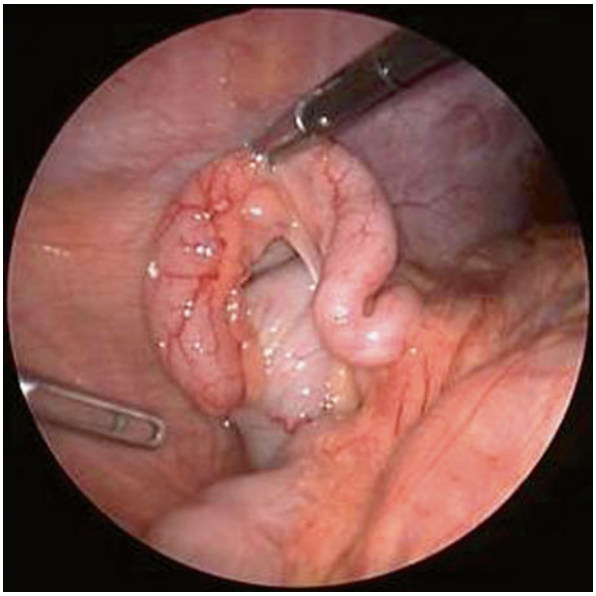


Fig. 15.1 Acute appendicitis – intraoperative view

Acute Appendicitis

This is the most common cause of the acute abdomen. The child may present with a low-grade fever, and the pain is initially periumbilical, later localising to the right iliac fossa. Stools may be normal, loose or constipated. The classical McBurney's tenderness, the psoas sign, obturator sign and Rovsing's sign may not be present in young children. The diagnosis is often difficult to make and may need repeated examination especially when there is minimal abdominal tenderness or no rigidity. Delayed diagnosis can lead to perforation in the younger child resulting in peritonitis. Diagnosis is often delayed in children <5 years and 90 % have perforation at the time of diagnosis [3]. Treatment is resuscitation with age appropriate fluids and antibiotics, followed by appendectomy using conventional or minimally invasive techniques (Fig. 15.1).

Intussusception

It is common in children <2 years with peak incidence at 6–9 months. The proximal segment of the intestine (intussusceptum) is telescoped into the adjacent distal segment (intussusception) (Fig. 15.2). Vomiting may be the earliest manifestation. The

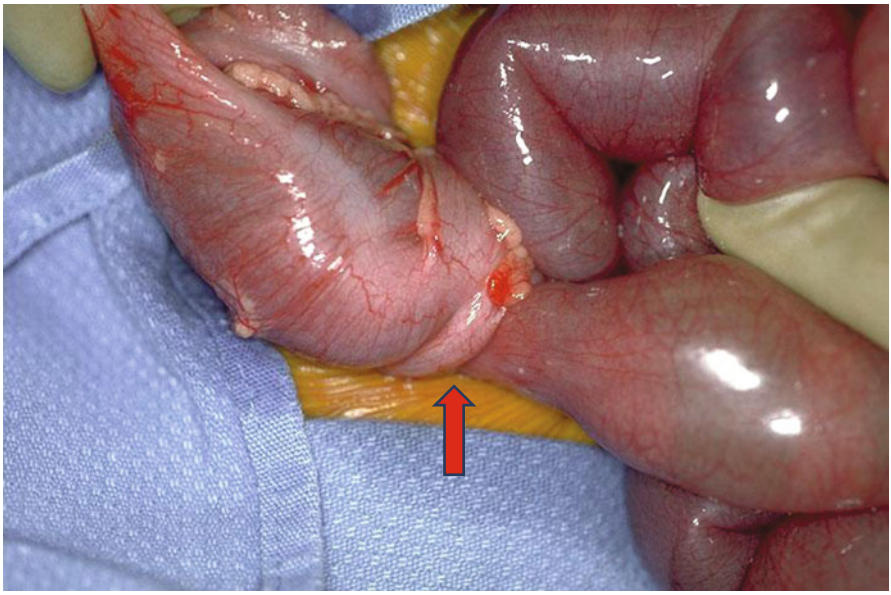


Fig. 15.2 Operative reduction of intussusception (failure of nonoperative reduction)

child may then develop colicky abdominal pain. A child with intussusception may draw the lower limbs up and cry inconsolably during episodes of pain. In between the bouts of colic, the child is listless. A mass may be palpable in the right upper quadrant in between episodes of pain. Red currant jelly stool, characteristic of the disease, is a late sign and indicates ischaemia of the intussusceptum, by which time the child is in shock. These children may be erroneously diagnosed as septic. Lethargy may be the only presenting sign in 10 % [4].

The preferred diagnostic modality is ultrasonography (USG). USG abdomen shows a doughnut or target sign in transverse images [5].

The intussusception is reduced either by nonoperative (air or saline enema) or operative reductions. If there is peritonitis, shock or signs of perforation, nonoperative reduction is contraindicated. Rarely, an intussusception can reduce spontaneously.

Peritonitis

Inflammation of the peritoneum can be primary as in spontaneous bacterial peritonitis or secondary to perforation of intra-abdominal viscera and sepsis. The child remains still and the abdomen may not move with respiration due to pain. The child could show signs of respiratory distress such as tachypnea and grunting. There is tenderness, guarding and rigidity of the abdomen, with the child resenting the

examination. Bowel sounds may be absent. Young infants may not have abdominal rigidity but only abdominal distension with a doughy feel and tenderness [6]. Bilious vomiting can occur due to ileus. Spontaneous bacterial peritonitis due to *Streptococcus pneumoniae* occurs in children with nephrotic syndrome or chronic liver disease. Biliary peritonitis as a result of common bile duct rupture may mimic a surgical abdomen. Surgery is needed for peritonitis secondary to perforation. Medical management includes antibiotics, intravenous fluids and bowel rest.

Intestinal Obstruction Intestinal obstruction is characterised by abdominal distension, bilious vomiting if obstruction is distal to the ampulla of Vater and cramping and colicky abdominal pain. Young infants may present with incessant crying, irritability and poor feeding. Visible intestinal peristalsis may be seen. Bowel sounds may be audible without need for auscultation (borborygmi). The child might present with signs of shock such as tachycardia.

Common causes: (1) malrotation with or without volvulus [7], (2) duodenal or ileal atresia, (3) imperforate anus, (4) Hirschsprung's disease, (5) incarcerated hernia, (6) intussusception, (7) worm obstruction (ascariasis) and (8) postsurgical adhesions. Prompt surgical management is required as delays can lead to significant morbidity and even mortality. Paralytic ileus: The child will have abdominal distension and absent or decreased bowel sounds. Child can vomit and may not pass flatus or stool. Ileus can occur as a result of sepsis, hypokalaemia, following diarrhoeal illness, surgery or trauma and sometimes due to drugs. Plain x-ray abdomen may show increased air in the small bowel, dilated bowel loops and air fluid levels.

History

A thorough history often gives a clue to the underlying aetiology (Table 15.2).

Clinical Examination The young infant is preferably examined on mother's lap if there is no life-threatening problem. A head-to-foot examination is done quickly. Facial expression may give a clue to the severity of pain. Young children cannot express pain and will cry incessantly or intermittently if there is colicky pain. Tachycardia often indicates significant intra-abdominal disease. Examination of the oropharynx, tonsils, lungs, genito-urinary system, groin for any hernia and skin for rashes (gluteal region and legs characteristic of HSP) is mandatory. In the female child, consider ovarian cysts, tumours and torsion whenever there is pain in the right or left iliac fossa. Lower abdominal pain with cervical discharge and tenderness in an adolescent girl should suggest pelvic inflammatory disease. Ectopic pregnancy or pregnancy should always be considered in post-pubertal girls.

Examination of the Abdomen: Inspection may reveal important clues to the diagnosis. A child with peritonitis prefers to lie still where as a child with visceral pain may actively move to assume the position of comfort. Oedema of the abdominal

Table 15.2 History in a child with abdominal pain

History	Probable aetiology
Site of pain	
Epigastric pain	It may be due to gastritis, peptic ulcer, hepatitis and pancreatitis
Periumbilical pain	Often nonspecific. However, in appendicitis the pain is initially localised to the periumbilical region and then to the right iliac fossa
Nature of the pain	
Visceral pain	Vague and non-localising
Somatic pain	Localising and more severe
Colicky pain	For example, renal stone, intussusception
Radiation of pain	
Loin to groin	Renal colic
Pain radiating to the back	Pancreatitis
Referred pain (pain in one organ is referred to a distant organ due to a common innervation)	In splenic rupture pain may be referred to left shoulder and in hepatic injury to right shoulder
Relation to food	Peptic ulcer
Bilious vomiting	Obstruction beyond ampulla of Vater
Abdominal distension	Intestinal obstruction
Stool – frequency, tenesmus, blood red currant jelly	Colitis
	Intussusception
Worm infestation	Intestinal obstruction
PICA	Lead poisoning
Urinary symptoms – dysuria, frequency, hematuria	UTI
Trauma	
If pain is present during sleep	Usually organic cause

wall with erythema may indicate necrotising enterocolitis. Abdominal distension with visible intestinal loops may be seen in intestinal obstruction or ileus. Look for surgical scars, hernias or other alterations in the skin. Dirty green discoloration may be seen in both flanks in pancreatitis (Turner's sign).

Palpation The examination should be gentle. Hands should be warm. Always start palpating from the quadrant opposite to the site of pain. Rebound tenderness, tenderness to percussion and guarding indicate peritonitis. Renal angle tenderness may be present in pyelonephritis or renal calculi. Murphy's sign (pain or limitation of inspiration during palpation of the right upper quadrant) may suggest cholecystitis. Rovsing's sign: Palpation in one area causes referred pain in another area indicating localised peritonitis in the area of pain [8]. Right hypochondriac tenderness may be due to hepatitis, liver abscess and congestive cardiac failure. A sausage shape mass may be palpable in the right upper quadrant in intussusception. Bladder may be palpable in the suprapubic region, and it may be the cause of pain.

Table 15.3 Investigations in a child with abdominal pain

Complete blood count: Leucocytosis and neutrophilia are seen in appendicitis. Thrombocytopenia is present in dengue
Urine microscopy and culture
Stool for occult blood testing and culture
Blood grouping and Rh typing
Blood culture
Serum amylase and lipase will be elevated in pancreatitis
AST, ALT, alkaline phosphatase (ALP), gamma glutamyl transaminase (GGT) in suspected liver pathology. If GGT is increased with mild elevation of AST and ALT, then biliary tract pathology should be suspected
Blood sugar, urine ketones to rule out DKA
Urea, creatinine, electrolytes

Percussion Liver dullness is obliterated in perforation. Tympanic note is present in gaseous distension and dull note in masses or ascites.

Auscultation Bowl sounds may be loud in intestinal obstruction (borborygmi) and absent in paralytic ileus secondary to sepsis.

Rectal examination is indicated only for situations in which occult blood, local masses or rectal lesions have to be ruled out. Examination of the groins, inguinal region and external genitalia is very important to identify hernia and torsion testes. Suspect pain of organic aetiology if age <5 years; pain during sleep; bilious vomiting; abdominal distension; guarding; rebound tenderness; hematuria; referred pain to shoulder, groin or back; jaundice; and weight loss [9].

Investigations

The investigations are listed in Table 15.3.

Imaging Studies

Plain X-ray Abdomen

Ideally a supine, upright and lateral decubitus view are needed. Air-fluid levels and dilated loops are seen in intestinal obstruction. Pneumatosis intestinalis is diagnostic of necrotising enterocolitis. Air under the diaphragm/pneumoperitoneum indicates visceral perforation. A radio-opaque renal stone may be evident. Trichobezoar and foreign bodies may be seen in mentally retarded children and children with PICA.

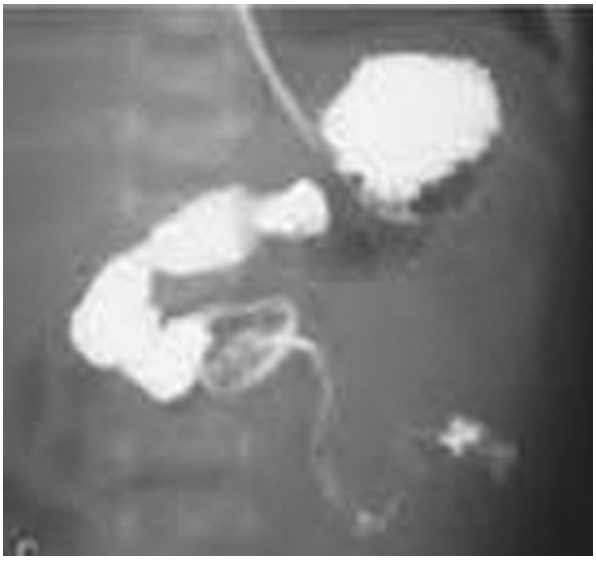


Fig. 15.3 Malrotation and volvulus on contrast study

Upper Gastrointestinal Contrast Study

It is diagnostic for malrotation of gut with near 100 % sensitivity. The normal anatomy is the duodenum lying to the left of the left pedicle of the spine, at the level of L1 (transpyloric plain). All variations of this anatomy are malrotation until proven otherwise. The corkscrew appearance on contrast imaging is that of volvulus and a surgical emergency (Fig. 15.3).

Ultrasonogram of the Abdomen

It is the initial investigation of choice as there is no exposure to radiation in the growing child. It can detect renal stones, gall stones, intussusception, ascites, liver abscess, testicular torsion, pyelonephritis, pelvic disease, acalculous cholecystitis, psoas abscess and bile duct dilatation. It helps in therapeutic drainage of pus under USG guidance. Focused assessment by sonography for trauma (FAST) is a specific tool in trauma resuscitation to confirm the presence of free fluid in the thorax, pericardium or abdomen, which guides further management.

CT Scan of the Abdomen

CT scan should be judiciously used in children as the long-term effects of irradiation are still being studied [10]. It is indicated in patients following trauma, if there are clinical signs or a mechanism suggestive of intrathoracic or abdominal injury. It

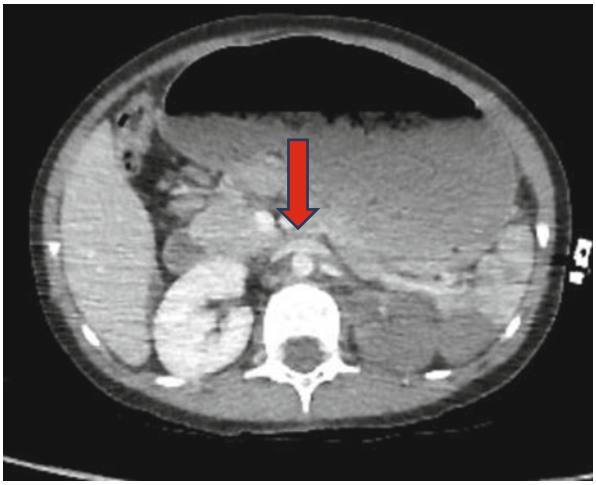


Fig. 15.4 Contrast CT scan showing left renal vascular injury with splenic bleed

can reveal pathology in intra-abdominal organs and retroperitoneal structures (Fig. 15.4). It can identify the cause of intestinal obstruction and useful in evaluating any intra-abdominal mass lesion.

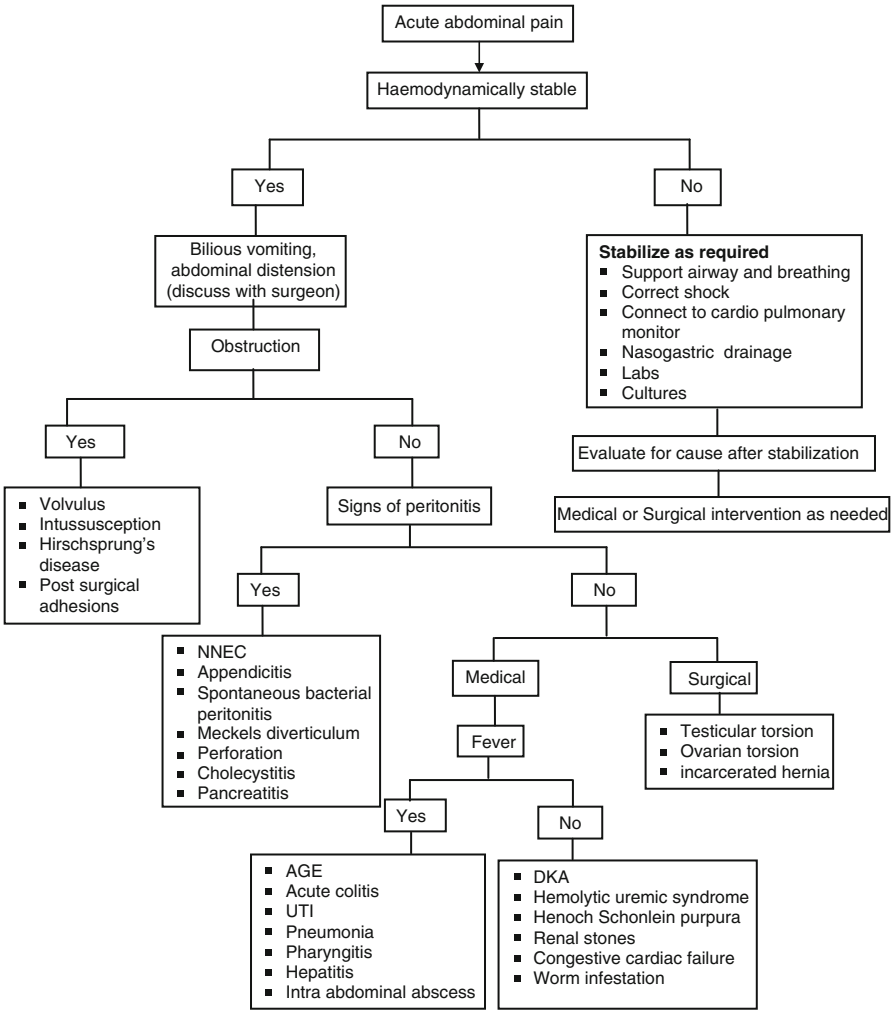
Management of Acute Abdomen

Irrespective of the aetiology, resuscitation is the most important part of the initial management. Using APLS guidelines, a systematic ABC approach should be instituted. Airway support as appropriate with supplemental oxygen should be commenced. Adequate peripheral venous access should be gained, failing which consider emergency intra-osseous access. Fluid resuscitation should be delivered with appropriate monitoring of the cardiovascular and respiratory parameters (i.e. heart rate, blood pressure, respiratory rate, oxygen saturation).

The child should be kept nil orally, and if vomiting, an age appropriate nasogastric tube is inserted to decompress the stomach. Appropriate antibiotics should be commenced, based on unit protocol and local sensitivities, if concerns of septic shock. Early surgical consultation should be sought as emergent surgery may be needed in some of the acute pathology as discussed prior.

It is not uncommon to perform an exploratory laparotomy for medical causes presenting with an acute abdomen, especially in Henoch-Schonlein purpura (HSP), as the abdominal pain may precede the rash. Severe dengue and diabetic ketoacidosis (DKA) may closely mimic surgical emergencies. It is very important to identify the underlying cause and treat it accordingly to avoid unnecessary surgical intervention which could result in morbidity.

Approach to Acute Abdominal Pain



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Part III
Psychiatry

Chapter 16
Acute Psychiatric Disorder

Ravi Pattanshetty

Key Points
- Acute psychosis has both functional and organic causes. In the emergency department, ruling out causes of organic psychosis first is crucial.
- The primary aim is to ensure that the patient does not harm himself or harm others in the department.
- Alcohol abuse is the commonest cause of organic psychosis.

Introduction

The emergency department is an inherently volatile environment, and as EM physicians, we are always trying to bring order to this chaos. The presentation of a patient with an acute psychiatric disorder causes disarray, as these patients demand additional resources and time, sometimes causing significant distress and anxiety to staff, other patients and their relatives.

Poor awareness among the general public about psychiatric disorders coupled with the growing stature of emergency departments in India as the front line for accessing medical treatment requires the EM physician to be competent in dealing with these patients should they present.

Estimated 1–5 % of the ED patients have a primary diagnosis of mental health disorder. In the Indian setting, this could vary largely depending on the area. Psychiatric patients awaiting inpatient placement remain in the ED longer than non-psychiatric patients, delaying bed turnovers. It may seem easy to cope with a busy

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Table 16.1 Organic causes of psychosis [3–7]

Drugs
I. *Drugs of abuse* – alcohol intoxication and withdrawal, opiates, amphetamines, etc.
II. *Drugs used in treatment of medical conditions* – antihypertensives, steroids, benzodiazepines, anticonvulsants, anti-parkinson drugs, cardiovascular drugs
III. *Self-harm/poisoning with chemicals* – carbon monoxide, plant toxins, heavy metals, industrial toxins
Diseases
I. *Neurological disorder*
(a) Medical conditions – stroke, Huntington's disease, lupus, temporal lobe epilepsy, encephalitis, postictal state, AIDS encephalopathy, dementia
(b) Structural problems – cerebral abscess, chronic subdural bleed, intracerebral space-occupying lesions
II. *Cardiovascular failure*
III. *Metabolic* – electrolyte disturbances, renal azotaemia, porphyria
IV. *Endocrine disturbances* – hypoglycaemia, diabetic ketoacidosis, Addison's disease, Cushing's disease, pituitary adenoma, thyroid disorders
V. *Hypoxia* [1]
VI. *Head trauma*

department full of patients with medical complaints, but it could take a single acutely psychotic patient added to this space to overwhelm the ED staff and resources [3, 4].

Pathophysiology

The pathophysiology of acute psychosis can be either psychiatric or organic disorder. For every five patients presenting to the ED, one patient will have an organic cause to the psychosis; therefore, the safest approach to such patients is to assume that there is an organic cause to the psychosis, unless proved otherwise [2].

Acute psychiatric disorders can be caused by the following:

1. *New onset psychotic episode*
2. *Acute worsening/deterioration of existing psychiatric disorder*
3. *Organic causes of psychosis* (Table 16.1)

Differential Diagnosis

Differential diagnosis of psychiatric disorders causing acute psychiatric disease:

1. *Disorder of thought*
   (a) Schizophrenia – Patient suffers from bizarre delusions, auditory hallucinations, loss of sense of reality and judgement, somatic passivity and thought insertion, broadcasting and withdrawal.

(b) Schizophreniform disorder – Features are very similar to schizophrenia but are mainly distinguished by duration; symptoms last 1–6 months (if longer, diagnosis changed to schizophrenia). Also social and occupational function impairment is not an essential criterion for diagnosis.
(c) Schizo-affective disorder – This is a disorder of the mind that affects both thoughts and emotions. Patients experience episodes that are combinations of both 'psychotic' (schizophrenic) symptoms and 'bipolar disorder' (affective) symptoms, and these must be clearly present for most of the time over a period of at least 2 weeks for diagnosis.
(d) Delusional disorder – This is characterized by the presence of one or more non-bizarre delusions (within the realm of reality but proved baseless by fact checking) persisting for more than 1 month. Here the patient doesn't have any psychotic or affective features. Social and occupational functionality is maintained [1].

2. *Disorders of mood*

(a) Mania – Patients are overtly cheerful, talkative and active. They may also take irresponsible risks, become promiscuous, indulge in gambling and spend excessively. They lose insight, have poor judgement and may suffer from mood-congruent delusions of grandiosity, exhibiting flight of ideas and hallucinations. They may be irritable, loud and violent. They have decreased need for sleep and are overtly energetic.
(b) Depression – These patients have decreased self-confidence and self-esteem, feelings of worthlessness and guilt, low mood, anhedonia, decreased appetite, sleep disturbance and memory disturbances. They have a pessimistic approach to life and self-neglect and are at a risk of self-harm and suicide [1, 18].

3. *Personality disorders* – Personality disorders are a complex set of psychological disorders with sub-classifications. Simplistically put, it affects the way one 'feels', 'thinks' and 'behaves'. These patients do not have any psychotic or affective features. They are broadly divided into Clusters A, B and C:

Cluster A – Odd and eccentric They have difficulty relating to others, have personality traits which people may find odd and have a fantasy world of their own.
Cluster B – Dramatic, emotional and erratic
They struggle to control their emotions, tend to swing dramatically between positive and negative emotions. They exhibit impulsive behaviour and are unable to maintain stable relationships.
Cluster C – Anxious and fearful
They can struggle with persistent and overwhelming feelings of fear and anxiety. They can be regarded as withdrawn and antisocial. They can be shy and socially inhibited. Obsessive-compulsive disorder falls in Cluster C.

4. *Conversion disorder* – Conversion disorder is also commonly known as functional neurological disorder. The psychological stress experienced by a person may manifest as an organic medical symptom. Sudden onset of symptoms

following stressful situations is characteristic. Common symptoms include psychogenic non-epileptic seizures, motor abnormalities or tics, pseudocyesis, paralysis, visual or auditory disturbances, amnesia, dysphagia and unresponsiveness (coma mimic). It differs from factitious disorders and malingering in a way that there is no intended or unintended benefit that the patient is seeking. In conversion disorder, patients do not know that their symptoms are not real.

5. *Delirium* – Delirium is an organically caused decline from baseline in higher mental functions.

Clinical Features

When dealing with an acutely psychotic, agitated and violent patient, it is easy to question the value of performing a clinical exam, but if done meticulously, clues could be picked up which could ultimately lead one to the cause of the illness and guide further laboratory and radiological investigations. Performing a head-to-toe exam, the following could be looked for:

1. Head – signs of trauma (lacerations, incised wounds, Battle's sign, raccoon eyes, haemotympanum)
2. Pupils – size and reactivity
3. Eyes – fundoscopy for raised ICP
4. Ears – insects and foreign bodies
5. Neck – rigidity indicating meningitis
6. Chest – use of accessory muscles, pneumonia causing confusion, pulse oximetry (indicating hypoxia), signs of cardiovascular failure
7. Abdomen – signs of cirrhosis
8. Skin – signs of liver failure, signs of IV drug use
9. Complete neurological exam [11, 14]

Investigations

All patients without known psychiatric disease should undergo the following investigations: venous blood gas analysis, full blood count, urea and electrolytes, liver function tests, serum calcium, blood alcohol, blood glucose and CRP.

Toxicology as per clinical exam.

Toxidromes can be identified and relevant investigations can be requested.

If suspicion of trauma – CT head, trauma series CT and chest and/or pelvis X-rays can be done.

Management

1. *Conducting the interview* (*the assessment*)

   (a) *The room* – Ideally, the interview should be held in a calm and cool room, away from main department with a clear egress route. The room should be devoid of sharp instruments and should only have minimum furniture. The doors should not have locks and should have a clear glass window for colleagues to keep an eye. The interviewer should position himself/herself near the door. There should be an accessible alarm bell.

   (b) *The doctor* – The interviewing doctor should think of the following before, during and after the interview and prepare accordingly:

      (i) The patient – The patient could be sad, angry, confused, withdrawn, frightened or violent.

      (ii) Colleagues/staff – The safety of your staff is most important! If patient turns violent and noisy, call security immediately and secure patient in a safe room. Keep them nearby before you start the interview if there is a pre-alert of a violent patient. Dealing with a violent and aggressive patient disturbs ED staff, and this could have a direct impact on the care of other patients.

      (iii) Other patients in ED – Unwell patients may be disturbed due to the noise. They may get neglected if all resources are pulled towards the acutely psychotic patient. Other patients and relatives will be curious and enquire about this patient; maintaining confidentiality is a challenge in such situations, but nevertheless should be strictly followed.

   (c) *The interview*

   Be positive, considerate and genuine. Do not be judgemental nor threaten the patient. Ask open-ended questions and try not to interrupt when the patient is talking. Think 'means to an end'; concentrate on your goal at all times, which is getting information. Collateral history can be obtained from relatives, police and accompanying individuals.

   Information gathered should include the following: a timeline of past medical and surgical history; drug history; social history; history of smoking, alcohol and drug abuse and drug dependence; past psychiatric history; family history for medical and psychiatric illness; developmental history; and criminal history.

   A good way to start the mental state exam is by saying, 'I want to see how well you are concentrating', or an opening statement that is unbiased, non-judgemental and definitive.

      (i) Orientation to time, place and person is paramount and must be done, most sensitive in differentiating between organic and functional.

      (ii) Mental state examination.

1. Appearance (neat/tidy or dishevelled, poorly groomed, hygiene, new/old, dirty/clean, colourful, age and gender appropriate or torn clothes) – They may have medication in their hands or pockets. They may have tattoos, needle marks on skin, wounds on head and signs of blood loss.
2. Behaviour – Observe mannerisms, gestures, eye contact and compulsions. Psychomotor retardation, tremor and ataxia are seen in organic psychosis, and repetitive activity, posturing and rocking are seen in functional psychosis.
3. Attitude – Patients may be cooperative, easily distracted or focused, evasive, suspicious, hostile, open, secretive, apathetic or defensive.
4. Level of consciousness – Reduced if medication or trauma to head is the cause for the organic psychosis. Patients in functional psychosis are usually fully conscious.
5. Orientation – Disoriented in organic and oriented in functional.
6. Speech and language – Should be assessed for rate, volume, intelligibility, quantity and quality. Speech could be slurred if patient is under the influence of drugs. Pressured speech is seen in mania and schizophrenia.
7. Mood – Patient describes his/her mood to be low or elevated.
8. Affect – Affect is the emotional state of the patient as observed by interviewer.
9. Thought process/form – Should be assessed for logic, organization, coherence, speed and relevance. This can be measured during the interview by the answers that the patient gives to the examiner's questions.
10. Thought content – Ask patient about any persistent thoughts. Withdrawal and broadcasting would be seen in schizophrenia. Does the patient experience any thought insertion, any thoughts of persecution or any fixed firm beliefs (delusions)? Visual hallucinations are reported in organic and auditory in functional psychosis.
11. Tendency towards suicide or homicide – Any thoughts of committing suicide or homicide must be specifically elicited from the patient as this can prevent further harm and loss of life.
12. Insight and judgement – Does the patient believe that he is psychiatrically unwell? Patients with functional psychosis do not have insight into their disease and refuse to believe or acknowledge that they are unwell and that they need help. When asked to solve hypothetical problems or to explain situations or proverbs, patients with functional psychosis report bizarre ideas.
13. Attention span – The patient with organic psychosis perceives surroundings occasionally and is able to focus, but this is difficult for a patient suffering from functional psychosis.

14. Memory – Recent impairment in organic and remote in functional.
15. Intellectual functioning – Impaired with organic psychosis but maintained with functional psychosis.

2. *De-escalation* ('talking down the patient') – With an agitated patient, the first approach should be to talk them down. Allowing the patient to talk and trying to understand their problem enable the patient to have trust in the interviewer. Encouraging the patient to relax and calming gestures reduce anxiety. Avoid direct and continuous eye contact; patients may take this to be a sign of aggression. Speak softly but in a firm manner; appear in control but nonthreatening. The goal is to establish a relationship with the patient, where boundaries can be negotiated and a dialogue is possible.
3. *Rapid tranquilization* (*see* Table 16.2)
4. *Physical restraint* – This is the last resort and should be done by staff who have had training in restraint methods. Hospital security and police can be called to assist with physical restraint. If physical restraint needs to be continued, then the reasons should be discussed by a multidisciplinary team consisting of emergency physician, psychiatrist, nursing staff, police or security officer. There should be a valid reason to restrain, for example, life-threatening danger to staff or other patients or the patient himself. Keeping the patient restrained to allow staff to continue with their work is not acceptable. Prolonged physical restraints can have long-term physical and psychological effects on the patient, increasing the length of stay and morbidity. Prior to initiating physical restraint, written consent must to always be taken from the patient's family member.
5. *Treatment*

   If the psychosis has an organic cause, then appropriate treatment should be commenced. Patient should be treated as an inpatient under the relevant specialty services. Intensive care input may be needed if patient is haemodynamically unstable or airway is compromised.

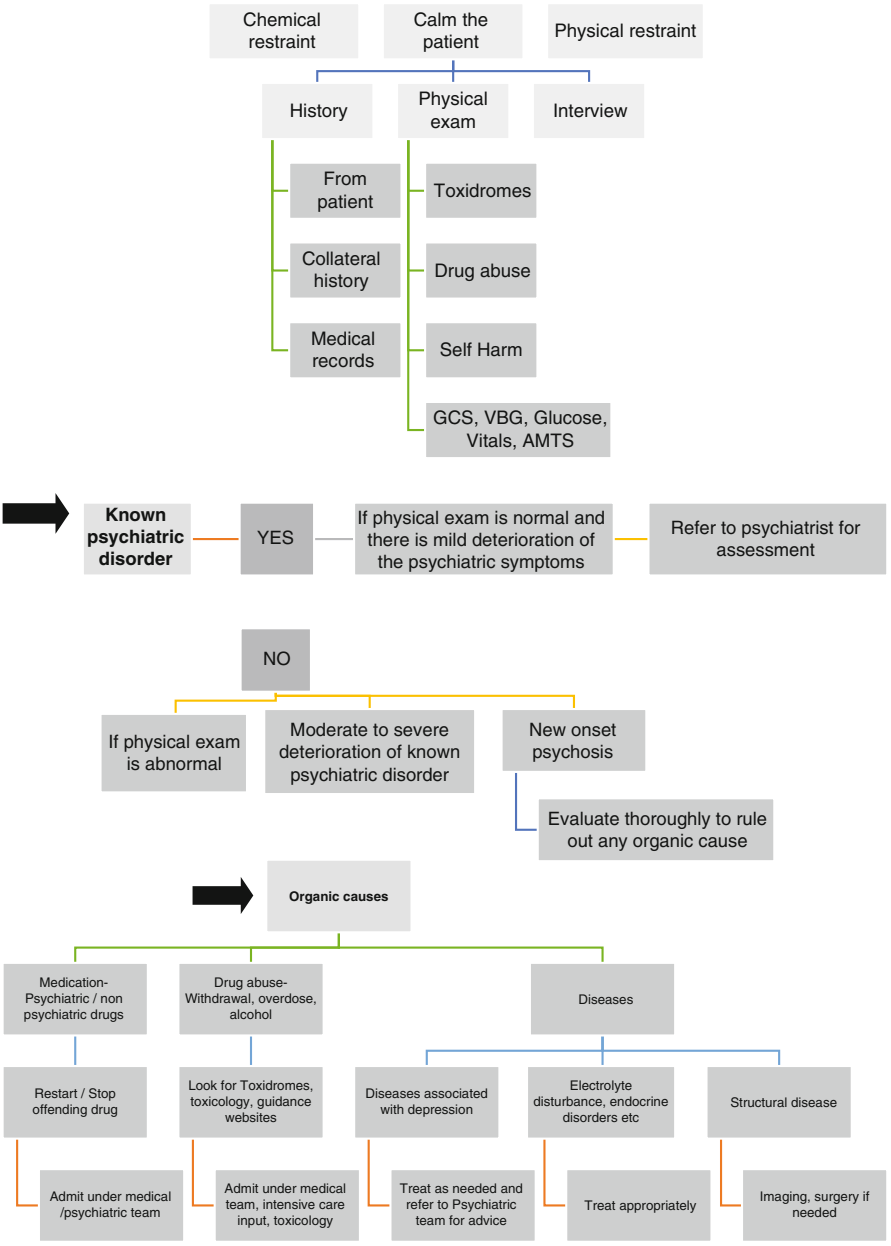
   If patient has known psychosis with mild deterioration of symptoms, an acute trigger should be identified, special emphasis should be given to drug history – any alteration or non-compliance identified – and psychiatry assessment should be requested.

   If moderate to severe deterioration of psychosis, patient should undergo thorough evaluation including laboratory testing and toxicology screen. Psychiatry consultant evaluation should be requested.

   If new psychosis is identified, then the patient could be declared medically fit and handed over to the psychiatry team only when laboratory testing, clinical exam and toxicology are within normal limits. If there is any doubt about having an organic component, then the psychiatrist should be informed and asked to continue to monitoring the patient for any symptoms or signs of physical illness [3, 4, 10, 12, 13, 19] (Flowchart 16.1).

Table 16.2 Drugs used for tranquilization [3, 8, 9, 13, 15]

Drug	Dose/route	Use/advantages	Contraindications	Complications
Haloperidol	5–10 mg IV	Acute functional psychosis to reduce tension, anxiety	Pregnant and lactating females, phencyclidine overdose, anticholinergic drug-induced psychosis. Not to be used as sole agent to manage agitation in patients with drug or alcohol withdrawal	Risk of death with high doses, hypotension and angina in severe cardiovascular disease, extrapyramidal side effects
Droperidol	2.5–5 mg IM/IV	Acute functional psychosis Compared with haloperidol, droperidol has faster onset and shorter duration of action and causes slightly more sedation		Potential association between droperidol and prolonged QT interval, torsades de pointes and sudden death
Ziprasidone	20 mg IM, every 4 h	Broader spectrum of response with fewer extrapyramidal and other side effects Oral form available		
Lorazepam	1–2 mg IM/IV Can be mixed with haloperidol 5 mg in the same syringe	With alcohol or sedative/hypnotic withdrawal, cocaine intoxication Or where a neuroleptic drug cannot be used	Allergy, sleep apnoea, respiratory insufficiency	Respiratory depression, sedation, unsteadiness



Flowchart 16.1 Approach to acute psychiatric disorder [16, 17]

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Chapter 17
Deliberate Self-Harm

Imron Subhan

Key Points
- Deliberate self-harm is usually a response to extreme personal stress.
- Treatment of associated physical condition and/or injury in DSH receives priority and must follow recommended guidelines.
- Correct assessment and management of the primary psychological condition reduces the risk of further self-harm and possible suicide.
- Patients who have self-inflicted harm may not have the mental capacity to give consent.

Introduction

Patients with self-inflicted harm are commonly seen in the emergency department (ED) and often consume substantial resources of a busy ED. Such patients are also known to walk out and leave the ED without being assessed, thereby putting themselves at further risk of injury and death.

Suicide is the 13th leading cause of death worldwide. Among those aged 15–44 years, self-inflicted injuries are the fourth leading cause of death and the sixth leading cause of ill-health and disability [1]. In India, the rate of suicides was 11/100,000 population during the year 2013 [2]. In the United States, for youth between the ages of 10 and 24, suicide is the third leading cause of death [3].

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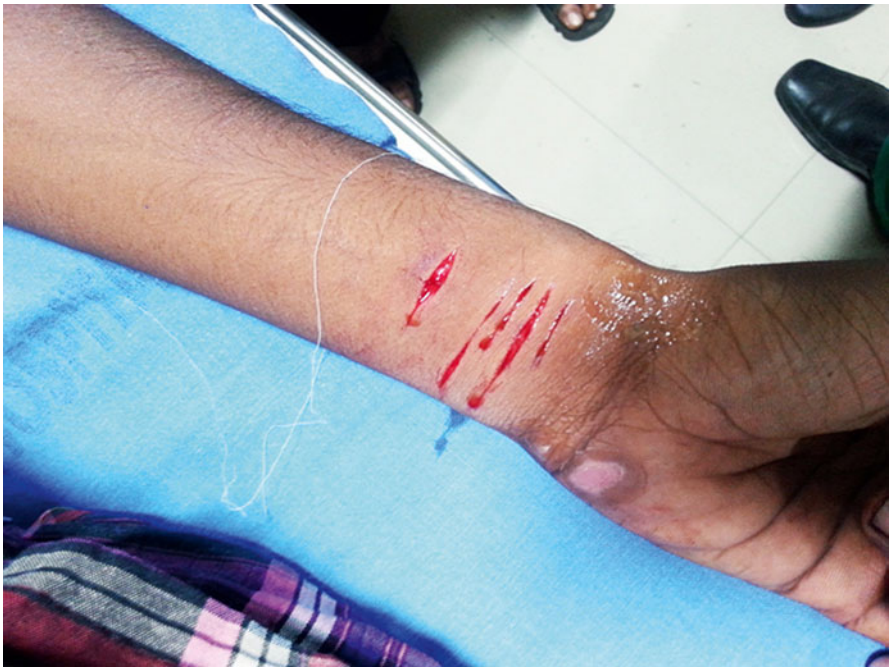


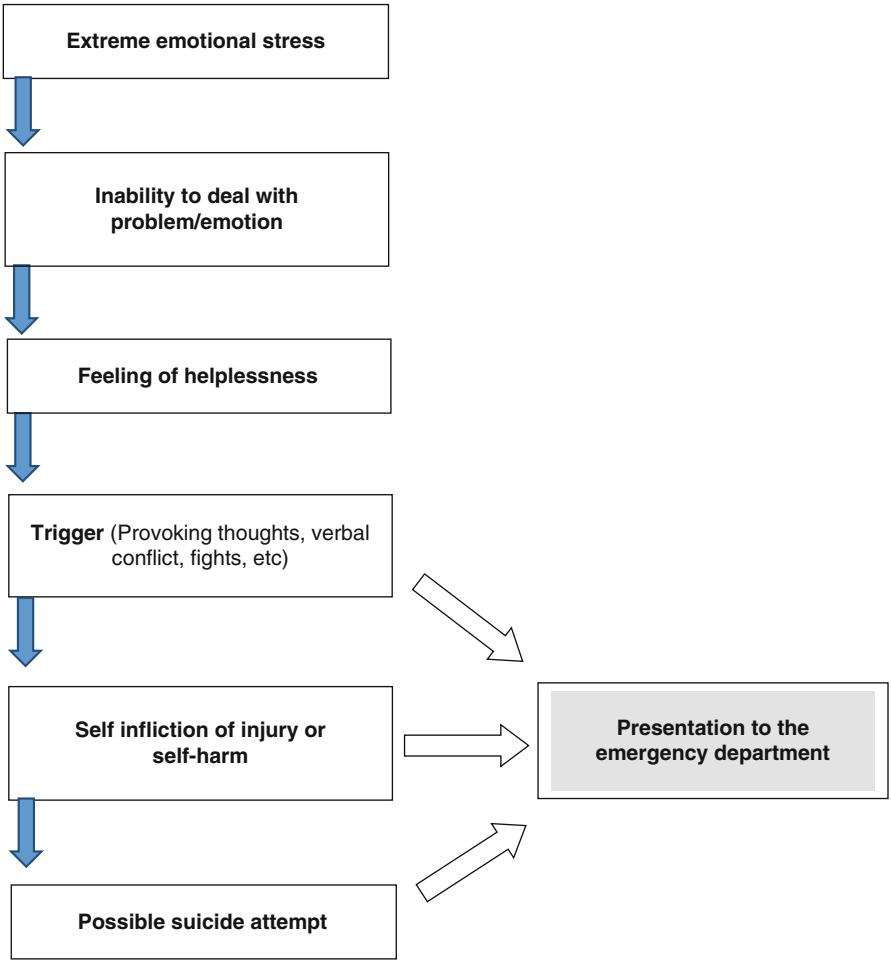
Fig. 17.1 A patient with self-inflicted lacerations over the inner aspect of the nondominant forearm demonstrating multiple "hesitation" cut marks. Hesitation cuts are characteristic of DSH (Courtesy: Dr. Krishna Prasad – Emergency Medicine, Pushpagiri Medical College, Tiruvalla -689191, India)

Patients with varying presentations can be categorized as "self-harm." These include self-inflicted cuts and lacerations; puncture wounds; hand, facial, and scalp injuries; burns and scalds; overdosage with over-the-counter (OTC), prescription, or recreational drugs; impaled foreign bodies; chemical poisoning; and even starvation (Fig. 17.1).

Deliberate self-harm (DSH) is provoked when patients face emotional or mental distress which is above their normal ability to handle. Work stress; unemployment; breakdown of relationship between friends; marital conflicts; divorce; domestic, sexual, or child abuse; financial debt; ragging or bullying; and gender or sexual discrimination are all which provoke self-harm. Preexisting mental illness like borderline schizophrenia or personality disorders tends to reduce the threshold for committing self-harm (Tables 17.1 and 17.2).

In India, family problems and illness, accounting for 24 % and 19.6 %, respectively, were the major causes of suicides [2], others being drug abuse/addiction (3.4 %), love affairs (3.3 %), bankruptcy or sudden change in economic status (2.0 %), failure in examination (1.8 %), dowry dispute (1.7 %), and unemployment

Table 17.1 Trigger pathway for deliberate self-harm



(1.6 %) [3], whereas in the United States, the top three risk factors for suicides include mood disorders, alcohol and drug abuse, and medical illnesses like cancer and chronic disease [4].

Incidents of self-harm may occur with sudden provocation or may be planned well in advance by the patient. DSH incidents should be perceived as the patient's "cry for help," and as such steps to initiate mental health assessment with appropriate counseling should be initiated in the ED itself.

Patients may sometimes walk into the ED under extreme emotional stress, before they actually harm themselves, allowing emergency physicians an opportunity to intervene.

Table 17.2 Risk stratification of patients with deliberate self-harm

Risk type	Presentations	Management
High risk	Failed at a serious suicide attempt. For example, attempted hanging, serious overdose, major injuries, deep lacerations over vascular structures, major burns, clinical condition requiring ICU admission or surgery	Inpatient admission (*restrain, if necessary*)
		Urgent psychiatric consultation
Moderate risk	Lacerations over extremities, large bruises, nonlethal overdoses, suicidal thoughts, preexisting mental illness (schizophrenia, depression, bipolar disorder, etc.), alcohol/drug abuse, clinical condition requiring ED observation for few hours	Observe in ED for 4–6 h (*restrain, if necessary*)
		Psychiatric consultation before discharge. Admit if consultation is delayed
		May require admission for psychiatric diagnosis
Low risk	Minor superficial lacerations, abrasions, or bruises, 1–2 "extra" doses of drugs, no history of alcohol/drug abuse, no suicidal thoughts, no psychiatric illness, no ongoing emotional crisis	Observe in the ED for 1–2 h
		Refer for outpatient psychiatric consultation, counseling, or de-addiction center
		Family support can help resolve crisis. Ensure family members accompany patient at discharge

Pathophysiology

A person can be motivated toward DSH by a large spectrum of social and psychological factors, which may not be related to any specific mental illness. Simple events like failure at examinations, breakdown of relationships, verbal conflicts, and any type of discrimination can trigger self-injury [5]. DSH is also associated with insomnia, fatigue, physical illness, isolation, and peer pressure. Sometimes, a trigger or reason may not be found at all. DSH patients tend to injure themselves when they are alone, and majority of them do not have any social support or immediate access to family and friends. Self-harm has been found to reduce the negative experience of severe hopelessness, nervousness, anger, and sadness. Therefore patients who face distress self-harm themselves in order to control their own response to emotions and relationship problems. DSH may be more of an attempt to regulate emotions and relief was the most common consequence [6]. It is similar to "venting out" by crumpling a paper in anger or smashing a bottle on the floor.

Usually DSH patients arriving to ED would not have any life-threatening condition despite their dramatic presentation and may not have really attempted to die. Patients who do arrive with a serious attempt at suicide may be found to have a definite psychiatric illness or a serious underlying drug/alcohol addiction problem. Many patients with drug dependence issues tend to have other personality disorders and depression or have been subjected to various types of abuse and therefore are prone to DSH. Psychiatric disorders and substance abuse commonly co-occur [7].

Substance use can heighten the mood swings of bipolar disorder; intensify the hallucinations and paranoid delusions of schizophrenia; or increase the risk of suicide, violence, and impulsive behaviors among individuals with antisocial or borderline personality disorders [8].

Clinical Features

Clinical presentation of DSH patients tends to vary with the type and degree of the self-injury inflicted by the patient. One should also be aware that it may not be possible to immediately recognize that a certain patient's condition was actually a deliberate attempt at self-harm.

A. *Trauma* – Patients commonly present with multiple linear lacerations over the skin, joints, chest, face, cartilages (ear, nose), and scalp. Such injuries will be superficial and will almost always involve the nondominant side of the body, as the patient would use his/her dominant hand to inflict the injury [9]. Many a time, such injuries may be severe underlying neurovascular structures with resulting hemorrhagic shock or neurologic deficits. Patients have been known to have cut their trachea, tendons, and even genital organs. Abdominal cuts may sometimes reach the peritoneum. Self-injury by head banging against a wall may have the head-injured patient arriving to the ED unconscious or in altered sensorium. A low GCS, anisocoria, elevated BP, and bradycardia may indicate raised intracranial pressure due to an extradural or subdural hematoma. Alcohol or drug intoxication will always complicate proper assessment of the patient. Patient may also present with hanging and neck injuries.
B. *Overdoses* – DSH patients may present in altered sensorium or unconscious due to ingestion of prescription medications in nonlethal to lethal doses. Paracetamol overdose will present early with a stable patient and in a toxic patient if presentation is delayed. It is important to remember the various presenting features of common drug overdoses so that such conditions can be identified rapidly in the ED. Patients have been known to have self-injected themselves with drugs like insulin, benzodiazepines, organophosphates [10], and induction drugs [11]. The author has personal experience of treating those who had self-administered paralytic drugs, where the outcome was fatal.

Case Example 1
A 25-year-old doctor was brought dead to the ED after self-injecting himself with 10 mg of midazolam and 100 mg of succinylcholine. He had stolen the drugs from his hospital. He was found to have been having repeated conflicts with his family and was in extreme depression. He lived alone and never did seek any professional help.

C. *Alcohol and recreational drugs* – Patients may either present with features of acute intoxication or with acute withdrawal symptoms, related to the concerned

substance abuse. It should be remembered that such patients may be conscious but experiencing hallucinations and may not be reliable with their history.

D. *Failed suicide* – DSH patients can present with a serious but failed attempt at suicide. History of fall from a large height, consumption of pesticide or acid, hanging, major burns, carbon monoxide poisoning, and deep lacerations over the anterior neck indicate a serious attempt at killing oneself. Suicide by hanging (37 %), consuming poison (29.5 %), self-immolation (8.8 %), and drowning (5.9 %) were the prominent means of committing suicides in India [2].
E. *Emotional distress* – Other patients may present with extreme distress where they can be profusely crying, sobbing, screaming, shouting, and uncooperative.

Case Example 2
A 19-year-old nurse was brought with multiple superficial lacerations over the inner aspect of the right forearm inflicted with a shaving blade. She was found to have relationship problems with her boyfriend. She had cut herself in order to get admitted to the hospital to seek sympathy. She was properly counseled after her wounds were sutured and discharged from the ED.

Differential Diagnosis

Various diagnoses must be considered in any DSH patient during clinical assessment in the ED and must take into consideration the following differentials [12, 13]:

1. Acute and chronic depression
2. Borderline personality disorder
3. Bipolar disorder
4. Schizophrenia
5. Munchausen syndrome
6. Drug/alcohol dependence or withdrawal
7. Narcotic addiction or withdrawal
8. Childhood disorders like autism, etc.
9. Bereavement, grief reaction
10. Anorexia nervosa
11. Obsessive-compulsive disorder
12. Genuine accidental injuries or overdoses

An underlying premorbid personality or history of psychiatric diagnosis is often present in patients, as described in Case Examples 3 and 4.

Case Example 3
A 40-year-old man found in a toilet lying in a pool of blood. He had cut both his wrists, both ankles, and anterior neck with a surgical blade. He was in class 4 hemorrhagic shock with bleeding from severed radial and posterior tibial arteries. The anterior part of his trachea was also punctured. He was stabilized in the ED and later found to have borderline schizophrenia. He was discharged after full recovery.

Case Example 4
A 26-year-old lady was brought to the ED with attempted suicide by jumping from the third floor of a building. She was in hemorrhagic shock with multiple rib fractures, hemothorax, splenic injury with free fluid in the peritoneum, and a femur fracture. She was stabilized in the ER and underwent emergency surgery. She was a known patient with schizophrenia on multiple drugs, with previous history of hospital admissions for drug overdoses. A discussion with her family revealed that she had developed hallucinations standing on the balcony and stepped over. She was discharged with long-term follow-up.

Investigations

Investigations are necessary to detect and rule out organic conditions:

(i) Toxicology screen
(ii) Drug screen (opiates, paracetamol, benzodiazepines, etc.)
(iii) Blood alcohol level
(iv) CT of the brain
(v) Electrolytes
(vi) Creatinine
(vii) WBC counts
(viii) Radiograms and ultrasound as required

Investigations may be also necessary prior to minor surgery:

(ix) Hb, PCV
(x) Urea/creatinine
(xi) Platelets and coagulation parameters
(xii) Chest radiograms (prior to administration of general anesthesia)

Treatment

1. *Triage* – Triage staff should identify DSH patients and place them under supervision.
   - Upgrade triage category for early physician consult.
   - Engage the patient in neutral and compassionate conversation.
   - Advise family members to let the patient remain in private to "calm down."
2. *Resuscitation* – Treatment of physical condition and/or injury receives first priority. Those with serious injuries must be managed as any other trauma patient.
   - Correct shock with crystalloids and blood. Tranexamic acid if indicated.
   - Control hemorrhage with pressure packing or temporary ligation of arteries.

3. *Suicide Risk Assessment* – All DSH patients must be assessed by the emergency physician for suicidal risk and risk of repeat self-harm.
    - Screen for risk factors for self-harm, including presence of mental illness or depression, previous attempts, drug abuse, domestic abuse, etc.
    - Assess mental capacity (ability to understand explained statements and make an informed decision).
4. *History* – Elicit the number, type, and intensity of previous episodes of self-harm. Take collateral history from a family member. Discuss with the patient regarding his/her thoughts and feelings to identify the provoking factor for the DSH incident. Review previous health records of the patient including admissions and consults.
5. *Privacy and Confidentiality* – Offer clinical and supportive care with privacy and confidentiality in mind. Many patients would be extremely anxious about their condition.
6. *Informed Consent* – All DSH patients must specifically be assessed for mental capacity and ability to give consent. Inform patient about their medical condition and plan of care. Many patients will not be in a position to understand their current health condition and will not be able to give proper consent regarding their treatment. The emergency physician may have to act in the best interests of the patient by physically or chemically restraining the patient in the ED for treatment. Consent must be taken from the patient as soon as he/she regains the mental capacity to take an informed decision.
7. *Specific Treatment* – Treatment and management of the presenting clinical condition must be rapidly started.
    - Manage airway and ensure oxygenation.
    - Correct shock, acidosis, and electrolyte abnormalities.
    - Monitor for arrhythmias.
    - Administer specific antidotes, if necessary.
    - Gastric lavage (if indicated) and supportive treatment.
    - Appropriate specialty referral.
8. *Need for Supervision* – Place DSH patients under one-to-one nursing supervision. Remove any sharp objects, wires, ropes, loose electricity panels, and barricade windows. Admission may be necessary.
9. *Psychiatrist Referral* – All DSH patients must be seen by a psychiatrist in the ED itself. Patients who continue to be in a vulnerable state are better managed by a specialist, and the possibility of a repeat attempt to self-harm can be mitigated. Referral or interhospital transfer should occur only when the patient has been stabilized and deemed suitable for vehicle transport.
10. *Risk to ED Staff* – Patients who are IV drug abusers may have blood-borne infections including HIV, HbsAg, and HCV. It would be prudent for the ED staff to have adequate personal protection, including the use of double gloves, during handling of these patients. Patients under influence of alcohol or a

hallucinogenic drug are a risk to themselves, other patients, and the ED staff. Such a patient who is armed with a gun, knife, or other weapon is a nightmare to the ED staff. It is wise to have the security staff check and disarm the patient.

Prognosis

DSH patients may injure themselves if their emotional state is not adequately addressed during their hospital stay. DSH patients are known to commit suicide within the hospital by hanging, jumping off buildings, and consuming drugs like potassium chloride syrup, etc. Some patients are only at risk during temporary phases in their life like a certain college or office, which resolves after they move out of them. Others remain at high risk for repeated self-harm incidents. Many patients commit suicide despite counseling.

Medicolegal Implications in India

Attempted suicide is a criminal offense under Section 309 of the Indian Penal Code and punishable with up to 1 year imprisonment. (As of Feb. 2015, the Indian government has started the process to delete this section and decriminalize this offense [14].)

Prevention

DSH patients should be advised to disclose their problems to their close friends, thereby seeking help from within their own trust circle, or contact depression or suicide helplines. Others can be advised to talk to their family counselor or psychiatrist.

Caretakers and family members must be educated in detecting stress and instituting simple methods to relieve stress. Objects which can cause harm like knifes, blades, prescription drugs, and poisons must be kept out of reach from such patients. Many patients improve with family support.

All patients arriving to the ED should at the minimum be screened for preexisting mental stress. Patients with a clear history of self-inflicted injury/harm should be referred to a psychiatrist on the same day. Resolution of their underlying emotional disturbance should be the goal in order to prevent repeat episodes.

Monitoring and reduction of self-harm incidents occurring in emergency department patients is one of the suicide prevention strategies followed in many communities and requires close collaboration between the emergency medicine and mental health departments.

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Chapter 18
Factitious Disorder and Malingering

Priyadarshini Marathe

Key Points

- Factitious disorders and malingering are diagnoses of exclusion only. Always rule out possibility of organic conditions. Don't be in a hurry to "label patients".
- Differentiation between factitious disorder and malingering is important. Patient with factitious disorder is mentally ill and needs treatment and support.
- Malingering patients can be challenging, as they tend to create an unsafe atmosphere in the ED and should ideally be handled by a team involving doctors, nurses, psychiatry staff and, if needed, legal support.

Introduction

A myriad of patients present to an emergency department. However, a small but significant number are not genuine emergencies. It can, at times, be a struggle trying to juggle the real and the not-so-real emergencies in the ED, where the biggest challenge of an ED physician is to triage correctly, allocate resources and invest time appropriately for each patient [1]. This chapter deals with two significant psychiatric conditions – factitious disorder and malingering. Differentiating between these two can be challenging (Table 18.1).

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Table 18.1 Differences between factitious disorder and malingering

Criteria	Factitious disorder	Malingering
Apparent external incentive	Absent	Present
Insight	Patients feel a compulsion to produce the signs and symptoms of disease, they cannot control this urge and their actions are voluntarily, but they are unaware about their inappropriate behaviour	Patients are fully aware of the reasons of feigning the illness
Cooperation with diagnostic evaluation	Present – ready to undergo painful tests and very happy to stay in the hospital for as long as it takes	Absent – they hesitate, become anxious and start to change the history or start feeling better
Trigger	Absent	Present
Chronicity	Present, allowing rationalisation of circumstances and becoming a coping mechanism	Not usually, the symptoms and signs disappear when the trigger event passes
Types	Imposed on self and imposed on another	
Past history	Enumerate in detail	Tend to focus on the new injury/illness

Section A: Factitious Disorder

Psychiatric disease and the need for psychological support are still a taboo in our society. One must always keep in mind that patients with unmet psychiatric needs (medications, counselling or social support) can present to emergency departments with physical symptoms and signs to gain attention or seek relief and support when they need a place of safety.

With factitious disorder, physical or mental illness is feigned for no apparent external incentive. The aim of these patients is to obtain sympathy and care, and they go to extreme lengths to portray symptoms and signs of illness, prolong their illness, change symptoms, produce new signs of illness and alter investigations only to be able to continue their role as a "patient". Simply assuming the role of an unwell or injured person fulfils their need as opposed to being treated or cured.

Most patients have complex and layered psychiatric issues. These patients may have personality disorders, e.g. borderline or antisocial [2]. With regard to the social/family history, these patients may have been subject to abuse during their childhood, but one must be aware that this history may be a ploy to gain sympathy. They may have had a family member with a terminal illness who was cared for resulting in their neglect and aroused feelings of abandonment. They may have had an illness themselves of which they are now cured but miss the attention that they were given at the time.

It is theorised that they are unsure of their role in the social hierarchy and being a patient not only allows them to have one role easily but also provides them with a social circle, friends and sympathetic healthcare workers [3].

It is important to understand the psyche of a patient suffering from factitious disorder. These patients are not aware that their actions are morally wrong. They do create the illness but without insight into their actions. Their need to be a "patient" is an uncontrollable urge taking over everything else, becoming a way of life where they invest time, money and energy into creating the illness, accumulating vast experience of portraying the "patient" role [4]. They are known to study other patients and behaviour of healthcare workers, research and read medical textbooks and watch online videos to be able to enact the disease and give a realistic performance just like a method actor would do.

The emergency department is an ideal target for patients with factitious disorder to gain entry into the healthcare systems. They are known to time their visit to the ED to reduce suspicion, e.g. weekends and out of hours, when the ED has a long queue of patients and short staffed with limited access to medical records and general practitioners unavailable to corroborate history. It is not unusual for patients to research the hospital and its systems before presenting to the ED, targeting new and junior residents. They indulge in doctor shopping, frequently moving from one ED to another, changing hospitals, states and even countries when their sham is exposed.

According to the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5) definitions, there are two types of factitious disorders, factitious disorder imposed on self and factitious disorder imposed on another, usually a dependent child or an adult [5]. This dependent person is unfortunately the victim here and may be undergoing neglect and/or abuse. Alerting the social services, family members or police is necessary duty of the doctor.

Factitious patients can present with physical and/or psychiatric symptoms.

Clinical Features

Gender – Seen in both males and females but common in young, unmarried women

Age – 20–40 years

Socio-economic condition – Can be from any strata of society, more likely in young girls from conservative households and well-educated and healthcare professionals

History of presenting illness – It is not unusual for these seasoned patients to use medical terminology when presenting the history of their complaints. Most common presenting illnesses are breathlessness, chest pain, unconsciousness, fainting episodes, abdominal pain, headache, renal colic and haematuria, seizures, paralysis and neurological deficits of some form, blindness and deafness.

Past medical/surgical history – Patients might give a long history of multiple illnesses, having been subjected to numerous tests and several opinions into their disease but without diagnosis. They enumerate their surgical procedures in detail but fail to produce medical records, explaining that they don't have them in their possession, in another country or destroyed in an accident. If there are records in other hospitals, they might refuse to grant permission to access them. They might

plead to the doctor to start afresh because they are tired of their illness and want answers, praising the doctor and trying to gain sympathy. Many of these patients can be very suave and manipulative.

Drug history – They may be on multiple medications which they duly take, citing elaborate details about prescriptions and insisting they cannot do without them.

On exam – They are usually calm, collected and very interested and compliant with the examination itself. It is imperative that one performs a thorough and meticulous clinical exam to rule out medical illness before subjecting the patient to unnecessary invasive tests.

They may fake breathlessness and wheezing, appearing to be in respiratory extremis.

They may give vague answers on being questioned about multiple scars on their abdomen from surgeries that were performed in failed attempts to find the cause of their pain.

They may claim having had continuous seizures before arrival but not postictal in the ED. They may also simulate a seizure while in the hospital.

There may be abscesses under the skin after having inserted foreign objects at accessible areas.

Bloods/Investigations

These patients may have underlying medical problems which may show up on routine tests performed in the ED, but most tests are otherwise normal.

These patients are aware of the tests that are used to confirm diagnoses and therefore use loopholes. They present with illnesses that do not have easy confirmatory tests, e.g. seizures, fever of unknown origin, headache or acute abdomen. They may bring with them samples of body fluids contaminated with blood.

Interaction with these patients can be frustrating for the treating doctor. They are not readily convinced about the management plan, especially if it does not involve more tests/observation period/admission to the hospital. They are quick to ask for a second opinion. They are extremely pleasant and involved in the management until they are discharged when they become aggressive and begin to question the competence of the doctor. If they become aware of the secret being discovered, they hurriedly leave the department after making a scene.

Management

Early detection will likely prevent chronic illness with regard to factitious disorder. Collaboration between primary care physicians, emergency physicians and psychiatrists is paramount. If any medical emergency has been induced, this should be

treated with the patient under supervision. Referral to psychiatry is needed but patients may not follow up if outpatient appointments are arranged. Psychiatry staff may treat this disorder with a combination of cognitive behaviour therapy and anxiolytics and/or antidepressants [5].

Section B: Malingering

Patients who are malingering do not have any organic condition or disorder, but they feign illness for a definite gain, to either avoid punishment or escape a sudden stressful life event, e.g. imprisonment, military duty, an exam and unexpected pregnancy, or to seek pleasure, e.g. seek opioid medications, aids or drugs and food and shelter for the night, obtain medicolegal documentation to support falsified injuries and claim benefits from government/hospital or claim insurance.

Malingering can be associated with antisocial or histrionic personality disorders.

Demographics – Military personnel trying to avoid duty; school-going children; politicians; criminals facing arrest or trying to get a holiday from prison; young women avoiding marriage, to avoid family or social responsibilities; immigrants facing deportation; unwed mothers, homeless looking for food and shelter on a cold night, to claim financial compensation or benefits; etc.

Gender – Both

Age – Adults, less common although seen in children and elderly

History of Presenting Illness

Patients who are malingering tend to give a detailed and flamboyant history but are usually not as thorough as factitious disorders. Their symptoms, medical and/or psychiatric, are exaggerated and inconsistent, bizarre and not conforming to known medical pathology.

They injure themselves, almost always in a non-life-threatening manner to help them tide over the situation. They may take medication or drugs of abuse to induce signs and symptoms like vomiting and diarrhoea.

The common symptoms are headache, abdominal pain, blindness, paralysis/stroke, chest pain, unconsciousness, back pain and wrist pain – carpal tunnel syndrome usually on the dominant side.

Always obtain collateral history from family members, bystanders, police, paramedics, etc. They may have been referred in a medicolegal context, for assessment by lawyers.

On exam – The general theme is that the physical exam findings are inconsistent with the history given. Patients are comfortable when they think they are not being observed.

The examination starts by carefully observing the patient looking for inconsistency from history given and performing simulation and distraction manoeuvres to obtain information to assist diagnosis [6].

Tests to Detect Malingering

Tests for Back Pain

- The 90/90 finding – In the sitting down position, the hips and knees are in 90° flexion. The ease at sitting down will be inconsistent with severe pain or resistance to movement at these joints in the supine position or inability to perform active or passive straight leg-raising test.
- In the presence of pathology at the spine, hip or knees, the patient will use his arms to support descent into and ascent from a chair. Suspect malingering if the patient did not use their arms to help with this movement but are not able to perform a squat, which is essentially the same movement.
- If the patient's sensorium is intact, gait is normal, and they are able to sit down and stand up from a sitting position, then essentially the sensory and motor systems are intact. Your findings at CNS exam of tenderness, power and reflexes should be consistent.
- Truncal rotation – Ask the patient to face away from you; hold their arms in extension and shoulder adducted, with palms facing towards the thighs. Now gently rotate the entire truck leftward and rightward. This movement occurs at the hips and should not cause pain at the lower back.
- Axial loading to detect non-physiological low back pain – Ask the patient to place their hand on the head and gently push downward. Doing this should not cause lower back pain.
- Waddell sign straight leg raise – Ask the patient to sit down and raise both legs up, extending the knees. Distract the patient by telling them that this is to examine their knees. This manoeuvre puts the spine and lower limbs in the same position as straight leg raise in the supine position, and any reports of pain should be consistent.

Tests for Stroke/Limb Weakness

- Hoover sign – With the patient supine, place your palms under the heels of the patient. In a healthy individual, while raising one leg, pressure is felt on the palm under the other leg. If malingering:
  - When asked to press down on the palm with both heels, forceful pressure will be felt on the palm under the normal limb but none under the paralysed limb.

- When the patient is asked to lift the paralysed limb, no pressure will be felt on the palm under the normal leg, because the patient is not trying to lift the leg.
- When asked to lift the normal limb, pressure will be felt on the palm under the paralysed limb.

Tests for Feigned Unconsciousness

- Do not use ammonia/strong-smelling salts – if really unconscious, this can threaten airway.
- Don't use painful stimuli if you can avoid them.
- Hand drop test – Hold one of the patient's hands over their face and drop it. If the patient is conscious they will not let their hand injure their face and will flop it down on the side. You must protect the patient's face with your other hand while you perform this test to avoid causing maxillofacial injuries if they are in fact unconscious.
- Flick the upper part of the cheek – the orbicularis oculi muscle will contract; the patient might blink or move his head away.
- Gentle brush on the eyelash causes the patient to blink. Perform this test only once.

Blindness – Refer to an ophthalmologist if there is suspicion of malingering after having ruled out systemic causes of blindness: drug abuse and posterior circulation stroke [7].

Deafness – Refer to audiometric tests alerting the technician of your suspicion for malingering.

Chest pain – All life-threatening causes must be ruled out in the ED.

Multiple Aches and Pains

- Pain drawing – Ask the patient to demarcate their symptoms on an outline of the body using a legend with signs for burning, stabbing, aching pain, pins and needles, numbness and weakness, arrows for radiation, etc. Patients exaggerating their symptoms may have the following on their pain drawing – drawing outside the body outline; implausible radiation of pain; unreal drawing; poor anatomic location, for example, involvement of an entire extremity; use of all listed modalities; use of arrows or underlying words to demarcate painful areas; writing notes; etc.

Psychiatric Complaints

- Patients may feign acute psychosis or amnesia. It is important to rule out organic causes of the same while the patient is in the emergency department. Referral to the psychiatry staff is important to establish the cause of the psychosis and amnesia where tests are available to evaluate personality characteristics and measures that are specifically designed to evaluate exaggerated cognitive impairment [8].

Bloods/investigations – It is important to rule out organic causes first. Do not withhold important life-saving investigations or tests; be judicious and prudent in using the resources available.

Interaction – If malingering is suspected, a non-confrontational approach is best. The patient may take offence if accused of lying, so allow them to save face by saying, "Let's start from the beginning, you are not telling me the whole story" or "you are a mystery, your symptoms do not add up". The patient may appear to be uncomfortable with tests, not compliant with suggested management and claim no benefit from treatment.

Management

Malingering is a diagnosis of exclusion and clinicians should not with-hold treatment. Dealing with a malingerer requires a fine balance of intervention and communication. It is good practice to ask another clinician to conduct an independent exam to confirm your diagnosis. The patient always gets the benefit of the doubt.

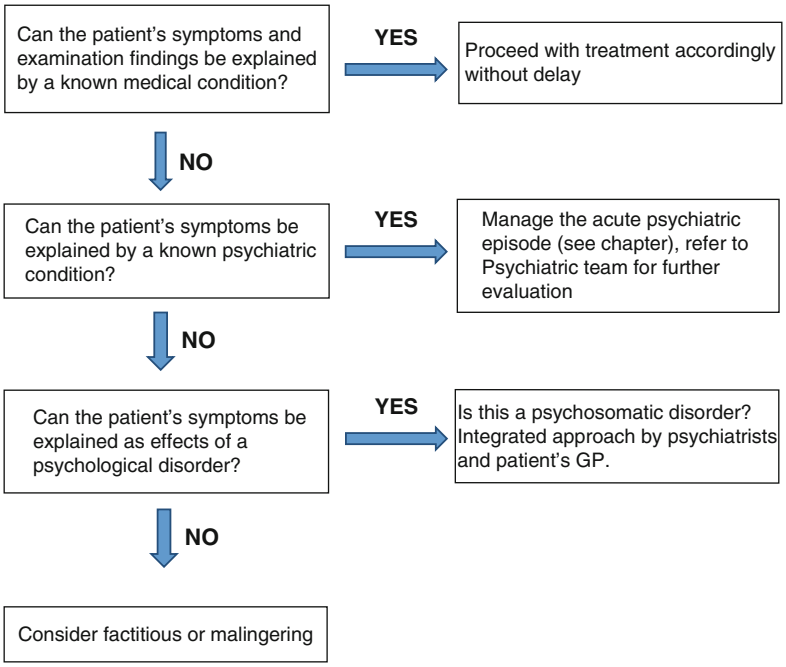
Although it is not easy to manage patients who are malingering, that is, discharging them home or treating them without incident, it is useful to identify patients who are malingering because they can then be flagged up to the admitting team. Also, if they present regularly to the ED, departmental alert policy regarding management can be drawn up by multidisciplinary teams to deal with these patients. ED physicians should refrain from giving out a medical note for leave as this may encourage malingerers. If in doubt, junior doctors should ask consultants to review patients. Patients should not be given a label!

Differential Diagnoses

Both factitious disorder and malingering are conditions where the lines between health and illness are blurred, with an added element of fiction, at times complicated by legal implications of decisions made. Therefore, it is important to move from an assumption of the diagnosis being a medical one and then to a psychiatric one and then lastly to be malingering.

It is a challenging dilemma for the ED physician to make the distinction between these similar presentations with the limited duration of interaction, and it is never really possible to rule out all medical problems, and therefore, these patients must be treated according to their symptoms.

The following simple flowchart could be used:



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Part IV
Surgery and Allied Specialties

Chapter 19
Acute Dissection of the Aorta

Neil G. Browning

Key Points
- Sudden onset of interscapular or chest pain should raise a high index of suspicion of acute dissection of the aorta, especially if myocardial infarction has been excluded.
- A CT angiogram is the best investigation to confirm the diagnosis and type of dissection.
- Acute type A dissection should be managed in a cardiothoracic unit.
- Uncomplicated type B dissection should be aggressively treated with antihypertensive regime followed by long-term blood pressure monitoring.

Introduction

Aortic dissection is a potentially life-threatening condition. Although relatively rare, it is the most common emergency affecting the thoracic aorta [1]. It typically occurs in adults between the ages of 60 and 70 years with more males than females. There is significant mortality with nearly 40 % dying prior to their arrival in the hospital. Interestingly, the first case of aortic dissection described was in the post-mortem examination of King George II of Great Britain in 1760 [2].

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Pathology

An aortic dissection starts as an intimal tear of the aorta which acts as entry point which allows blood to leak into the middle layer of the aorta (media) causing splitting off of the inner layer (intima) from the rest of the aortic wall. This leads to the progression of a sub-intimal channel resulting in the information of a true and false aortic channel (lumen) that can in turn reduce blood flow to major branches of the aorta. The false channel progresses distally and often reconnects with the true lumen via a re-entry tear. The septum separating the true and false lumen may, along its course, develop holes or fenestrations allowing the true and false lumen to communicate at multiple levels [1].

Predisposing Factors

Hypertension [3] is thought to be an important predisposing factor and in one study was present in over 80 % of patients presenting with this condition. However, dissection is also associated with increasing age and with the presence of atherosclerosis. Dissection can also occur after endovascular and/or radiological intervention (iatrogenic). Inherited connective tissue disorders such as Marfan's syndrome are strongly associated with dissections of the aorta [4]. It is unsure if an intramural haematoma (usually seen on a CT angiogram) is a potential cause of aortic dissection [4].

Causes of death following aorta dissection include rupture, organ failure and peripheral ischaemia as the dissections compromise blood flow to branches of the aorta supplying key organs (heart, brain, spinal cord, bowels, kidneys and lower limbs). The dissection is seldom linear – rather it spirals around the aorta as it progresses distally from its origin. The septum separating the true and false lumens is initially flimsy and can be manipulated by an intervention such as a stent, but once it becomes chronic, it becomes thickened and difficult to manipulate. This has important implications for treatment.

Classification

Aortic dissection can be classified according to its site (type A or type B), its effects (uncomplicated or complicated) or its duration (acute/chronic).

(a) *Site*

According to the Stanford classification [5, 6], there are type A and type B dissections (Fig. 19.1).

Type B dissection involves the descending aorta, and the entry point of the dissection is usually distal to the left subclavian artery. Type A dissection occurs proximal to this and thus involves the ascending and arch of the aorta. One can

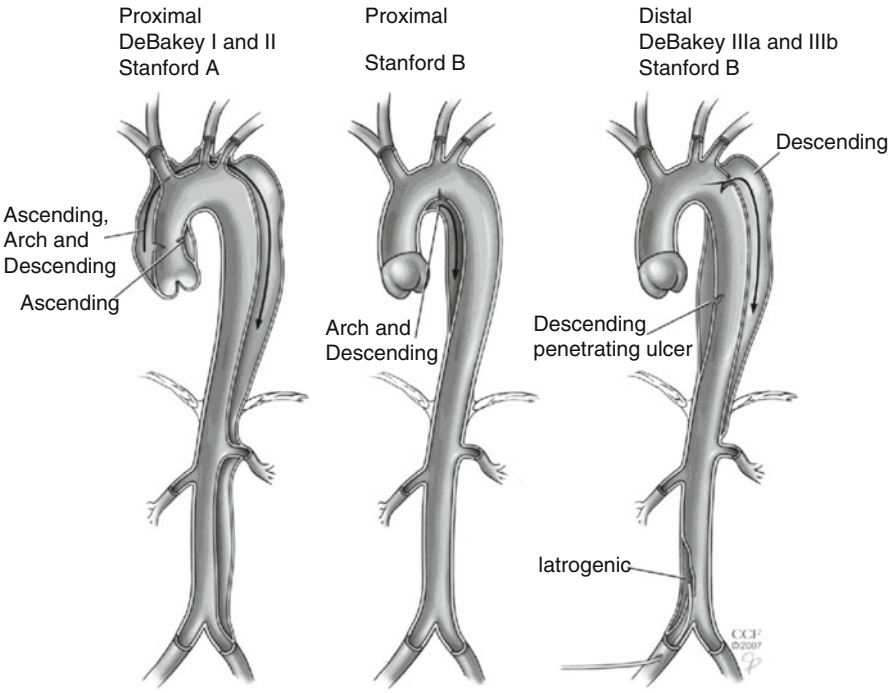

Fig. 19.1 Stanford classification of aortic aneurysms

get a dissection all the way from the aortic root down to the aortic bifurcation and beyond. The site of the dissection has significant implication for both prognoses and treatment.

(b) *Effect*
In an uncomplicated dissection, the dissection is clearly visible (on CT scan), but there is no compromise of the blood flow to branches of the aorta supplying major organs. In a complicated dissection, features include rupture (outside the wall of the aorta) or malperfusion which may present as cardiac or cerebral (type A) or renal, visceral, lower limb or spinal cord hypoperfusion (type B).

(c) *Duration*
Dissection is as classified as acute (onset 14 days or less) or chronic symptoms (present for longer than 14 days).

Clinical Presentation

This depends on whether the dissection is type A or type B and whether it is complicated or uncomplicated. The most common presenting symptom is pain which may be severe and may occur in the chest and progress to the neck or down the back.

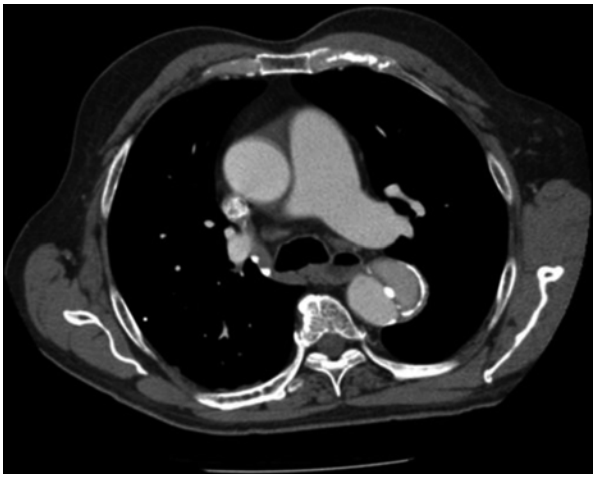

Fig. 19.2 'Slazenger sign'. Note the septum separating the true and false lumens

It may present initially as interscapular pain. In an uncomplicated dissection, this may be the only manifestation with no further sequelae.

In a complicated type A dissection, the tear may dissect into the pericardium causing a pericardial tamponade, it may dissect off the coronary arteries resulting in a myocardial infarction, it may dissect off the carotid arteries and cause a stroke or it may affect the left subclavian artery causing a loss of pulse in that arm. A complicated type B dissection may present as paraplegia due to spinal cord hypoperfusion, as an acute abdomen secondary to bowel ischaemia resulting from visceral hypoperfusion, and as renal failure or an acutely ischaemic lower limb as the dissection compromises the blood flow to the limb. What determines whether a dissection will be complicated or uncomplicated depends on how the dissection affects key branches of the aorta and also how big the false lumen is compared to the true lumen. It is not uncommon to see a dissection progressing into the coeliac, superior mesenteric, renal or iliac arteries. A large false lumen may take most of the blood flow from the normal aorta above it and compromise the true lumen so it becomes a narrow channel carrying only a fraction of the normal blood flow. Here the risk of complication is high. In uncompleted cases, one often sees a smaller true lumen, but there is enough communication between the true and false lumens through the distal re-entry post and other fenestrations in the septum to ensure adequate blood flow to the major organs whether that blood supply comes off the true or false lumen.

Investigations

In any emergency department, the usual way to confirm the clinical suspicion of an acute aortic dissection is with a CT angiogram. The axial views clearly show the aorta in cross section with a septum separating the two lumens (Fig. 19.2). This is often called the Slazenger sign because of its resemblance to a tennis ball.

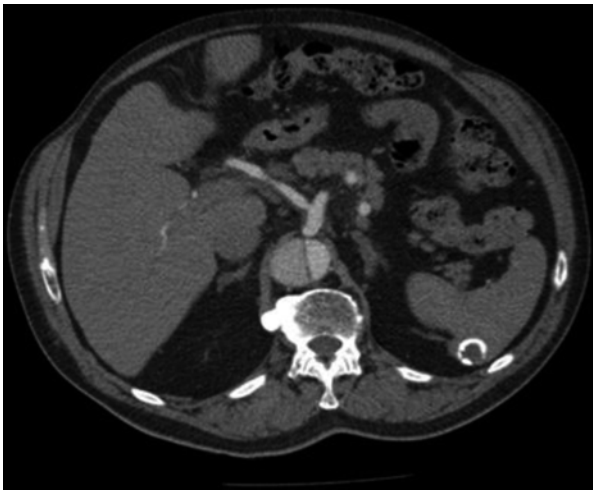

Fig. 19.3 Comparative sizes of the true and false lumens on axial view of CT scan

The CT scan also shows the size of the false lumen (usually the bigger one) compared to the true lumen (Fig. 19.3).

The true lumen often has the densest contrast in it. The CT angiogram also shows the visceral vessels that emerge from the respective lumens. Because many patients with a dissection present with chest pain, it is essential to exclude a myocardial infarction as the cause of the chest pain (ECG and troponin level).

Treatment

(a) Treatment of type A dissection. Whether this type of dissection is complicated or uncomplicated, the treatment of choice of this type of dissection is surgical, and patients should be referred to a cardiothoracic unit as a matter of urgency. The rest of this discussion will confine itself to the management to type B dissections.
(b) Treatment of uncomplicated type B dissection. The gold standard for type B aortic dissection is medical treatment, and even in an age of endovascular intervention, this remains the case at present [2].

Ideally, patients should be admitted to an intensive care unit or high dependency unit. Treatment consists of an intravenous beta blocker (e.g. esmolol) followed by a vasodilator (e.g. nitroglycerin). The aim of treatment is threefold – to keep the systolic blood pressure less than 120 mmHg, to keep the heart rate less than 70 bmp and to control pain. If there is a poor response to beta blockers, then calcium channel blockers, ACE inhibitors, angiotensin receptor blockers and alpha blockers can also be used. Once this is achieved, patients can be switched onto oral medications. In a

series of 85 patients with complicated and uncomplicated type B acute dissections reported by Tefera et al. [3], all patients were initially treated with an antihypertensive regime. The mortality rate of the uncomplicated group was only 1.6 %. This would seem to justify an initially medical approach to treatment of this group (uncomplicated type B dissections).

The patients must be carefully observed for complications including rupture, impending rupture, and rapid dilatation of the aorta, malperfusion or uncontrollable pain. Such patients will need more aggressive intervention (see treatment of complicated dissections). It has been observed that 20–40 % of patients with an uncomplicated dissection managed medically will go on to form aneurysm dilatation of the dissected part of the aorta [7]. A recent published study has looked at early stenting of uncomplicated dissections. The idea is that if a stent can be placed over the primary tear, this will close off the inflow to the false lumen, thus reducing its size and enabling it to thrombose and allow remodelling of the aorta to occur. Hopefully, this will prevent late aneurysmal dilatation. In the INSTEAD trial [8], 140 patients were randomised to stenting plus best medical therapy (BMT) or BMT only. There was no significant difference in the all-cause mortality rate or cumulative survival rate between the two groups; however, aortic remodelling occurred in 91.3 % of the stent plus BMT group and in only 19.4 % of the BMT group alone. It seems that the stenting of the primary tear in the uncomplicated type B dissection does not confer any benefit in short-term survival, but it may have a role in preventing later development of aneurysms. Further studies are needed to decide if stenting has a role in the primary treatment of uncomplicated type B dissections.

Treatment of Complicated Type B Dissections

This applies to patients who present with an acute type B dissection with a rupture or an enlarging aorta or malperfusion of renal or visceral vessels. Traditional open repair is done through a left posterolateral thoracotomy with partial or full cardiopulmonary bypass and replacement of the thoracic aorta involved with a dissection, with a synthetic graft. The overall mortality rate of patients with complicated type B dissection treated with open surgery is around 30 % [9]. For this reason, the preferred treatment for this group of patients is endovascular. The aim of treatment is to cover the primary tear thus reducing the blood flow and pressure in the false lumen, correcting the malperfusion by reversing the true lumen collapse and promoting thrombosis of the false lumen. After aortography, a covered stent is deployed under radiological vision, covering the proximal entry tear and thus shutting off inflow into the false lumen and redirecting it to the true lumen (Fig. 19.4).

If treatment is successful, then a follow-up CT scan will show contrast in the true lumen and no contrast or very little contrast in the false lumen (Fig. 19.5).

If malperfusion persists, another stent can be deployed to the level of the coeliac artery and if necessary an uncovered stent be deployed distally in the abdominal aorta. The mortality for this procedure is 2.6–9.8 % [8]. Endovascular stenting has thus become the preferred method of treatment for complicated type B dissections.

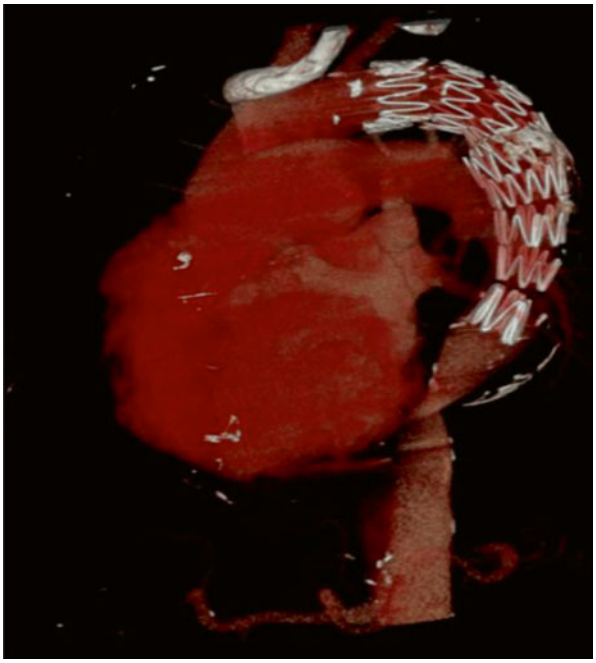

Fig. 19.4 Stent placed to cover primary tear

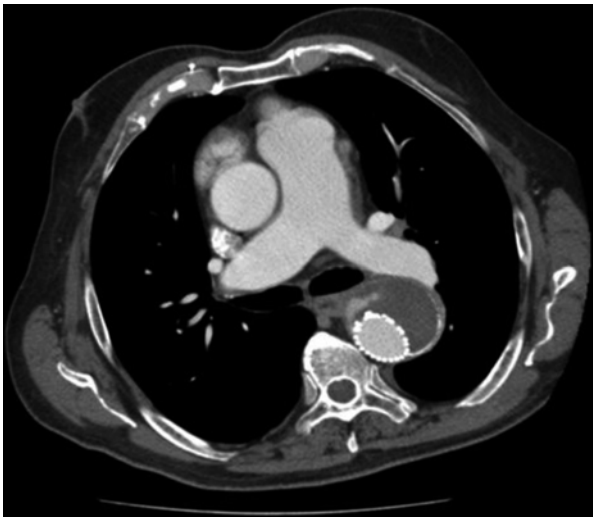

Fig. 19.5 Post-op endovascular stenting showing very little contrast in the larger false lumen

For patients presenting with an ischaemic lower limb due to the progression into the iliac artery without any other organ malperfusion, a femoro-femoral bypass provides a good solution to reverse the ischaemia.

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Chapter 20
Acute Ear Emergencies

T.L. Vasudevan and Suresh S. David

Introduction

The emergency department is often the first port of call for a variety of ear, nose and throat (ENT) disorders. Although most of these conditions are benign, there are important critical ENT disorders that must be immediately recognised and managed. This chapter, divided as three subsections, attempts to discuss salient disorders from the perspective of the emergency physician.

Applied Anatomy

- The ear canal is a tortuous tube, directed upwards and backwards in the outer half and downwards and forwards in the medial half. In children, it is almost straight. Hence, while examining the ear, the pinna should be pulled upwards and backwards in adults and downwards in children.
- There is a narrow part of external canal, the isthmus, at the junction of the outer two-third and inner one-third, which should be kept in mind, while removing foreign bodies (FB).

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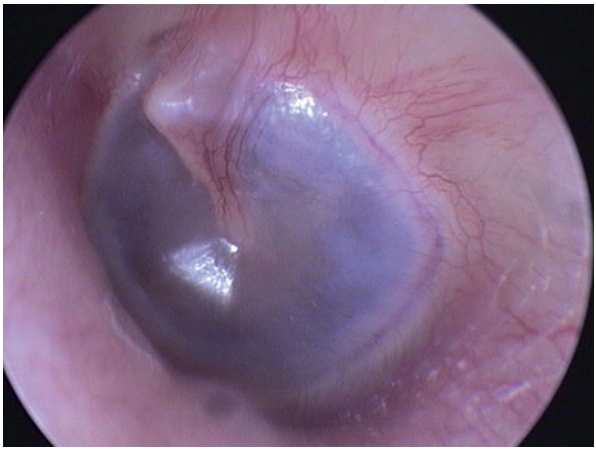


Fig. 20.1 Normal tympanic membrane

Table 20.1 Aetiology of otalgia

Trauma to the ear
Keratosis obturans
Herpes zoster oticus
External otitis
Necrotising otitis
Otalgia of children
Otalgia of air travel

- The skin is tightly draped over the canal wall which makes the furuncle very painful, and any careless handling can be very painful.
- The normal tympanic membrane is a shiny white surface with a cone of light antero-inferiorly (Fig. 20.1).
- A branch of the vagus nerve (Arnold's nerve) can get stimulated, which can result in syncope [1]. Due to the same reason, some patients develop cough on stimulating the external canal.
- The auriculo-temporal branch of the trigeminal nerve is the cause of referred otalgia of dental origin [2].
- Glossopharyngeal nerve, which presents itself as a plexus in the middle ear, is the cause of pain of the posterior one-third of the tongue and is due to tonsillar lesions [3].

Acute Ear Pain

Pain in the ear is one of the common emergencies which require urgent doctor's help in odd hours especially in children. Although there are many causes, the salient ones are enumerated in Table 20.1.

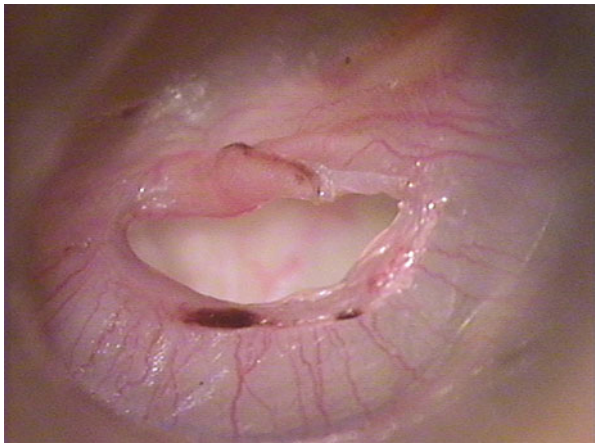


Fig. 20.2 Traumatic perforation – TM

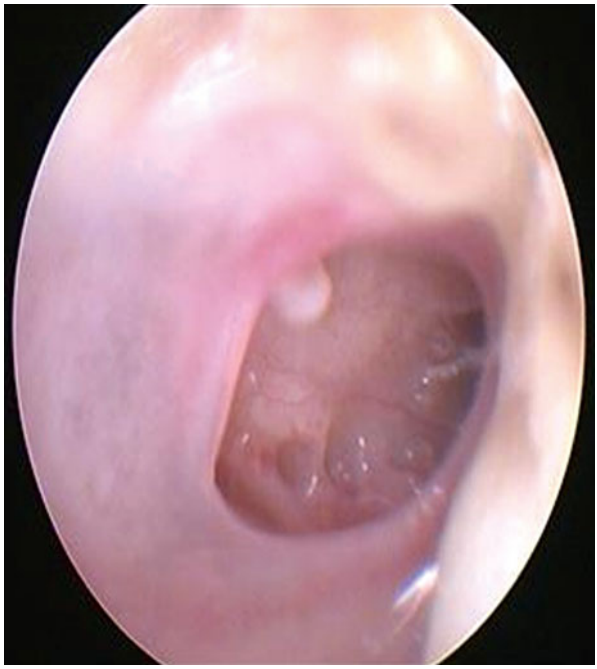


Fig. 20.3 Perforation of chronic suppurative otitis media

Trauma to the Ear

- Trauma to the ear can be trivial – nail scratch causing abrasion or haematoma of the pinna or lacerated injury. An example of occult trauma is perforation of the eardrum.
- A slap on the face/ear can result in traumatic perforation of the tympanic membrane (TM), which has a classical appearance of irregular margins (Fig. 20.2). This needs to be differentiated from the circular perforation due to suppurative otitis media (Fig. 20.3). *However*, *post-traumatic perforation will assume a circular shape*, *after a few days due to the ongoing healing process*.

- Traumatic perforation of TM has a significant chance of spontaneous healing unless impeded by application of ear drops (a common iatrogenic blunder), or use of earbuds, which have the potential to contaminate the ear canal.

Haematoma Auris

- Collection of serous fluid in the subperichondrial space in the pinna is known as pseudocyst. When it turns bloody, it is haematoma auris, and when there is infection, it is known as perichondritis or perichondral abscess.
- Haematoma auris and perichondral abscess require incision and drainage under antibiotic cover.
- The deformity which results in the healing phase of perichondritis is known as 'cauliflower ear'.

Keratosis Obturans

- This is a condition of the external ear, in which accumulation of keratotic, desquamated material presents as a sac of external canal skin. It can expand and erode the bone and can cause facial palsy [4].
- Patients present with history of pain and fullness of the ear. On examination, there is earwax of varied colour, consistency and tenderness on touching the pinna.
- Usually there will be no history of ear discharge, which is indicative of CSOM (chronic suppurative otitis media).
- There may be history of similar episode in the same ear indicating a tendency as a result of defect in epithelial migration of the ear canal skin.
- Pain relief is achieved by excision of the sac either under local anaesthesia or general anaesthesia. Use of microscope is recommended.

Necrotising Otitis Externa

- Also referred to as malignant otitis externa. However, this is not a malignant condition but can be as devastating and fatal [5].
- Progressive pseudomonas infection of the external canal and mastoid bones occurs in immune compromised, commonly in diabetics.
- Patient presents with very painful external otitis with tenderness, aural fullness and otorrhoea. The ear canal may have granulations and oedema and occasionally with lower motor neuron facial palsy.
- Pain is disproportionate to the findings inside and unrelenting, in spite of antibiotics and painkillers.

- Knowledge of this condition is necessary for early and aggressive therapy. Early ENT consultation needs to be sought.
- *Management includes s*urgical debridement, control of diabetes, opioids for pain relief and appropriate parenteral antibiotics.
- Patients with associated co-morbidities such as uncontrolled diabetes, renal impairment or peripheral vasculopathy have poor prognosis.

Herpes Zoster

- This is a painful condition of the ear, presenting with vesicular rashes on one half of face and pinna.
- Described first by Ramsay Hunt in 1907, the reactivated varicella zoster infection from dormant viral particles resident in the geniculate ganglion of the facial nerve is the cause.
- Herpes zoster oticus is the second major cause of facial palsy after Bell's palsy.
- The sequel of concern in herpes zoster oticus is the development of lower motor neuron facial palsy which is often not reversible and facial neuralgia which persists after the vesicles resolve.
- Management includes early administration of oral steroids and antiviral drugs (acyclovir) to achieve best facial nerve outcome.
- For neuralgia, carbamazepine may be necessary.

Acute Pain of Air Travel

- Acute otalgia during air travel is due to the pressure gradient between the ground level and the higher altitude, especially when there is eustachian catarrh.
- It presents as progressive increase in aural fullness and pain.
- During travel, by a manoeuvre of pinching the nose and trying to swallow the saliva, this would pop the ears open. Anti-inflammatory and decongestant antihistamine tablets can be taken. Application of nasal decongestant drops (oxymetazoline) will help immediately.
- Prevention: Treatment of upper respiratory infection prior to air travel and use of chewing gum for a few days prior to travel for anyone who has the tendency to develop ear pain during air travel.

Acute Otalgia of Infants

Incessant cry of an infant or child is often due to ear pain. Most of the ear pain is due the middle ear effusion caused by:

1. Untreated upper respiratory infection and nasal block.
2. Improper feeding positioning of breast-feeding, for example, mother lying down on side and feeding the baby.

Table 20.2 Referred otalgia

Source of pain	Cranial nerves involved
Dental caries, temporomandibular joint pathology	Facial nerve
Tonsillitis, oropharyngitis and carcinoma of oral cavity	Glossopharyngeal nerve
Pharyngolaryngeal area (infections, Ca larynx) and reflex oesophagitis	Vagus nerve (Arnold's nerve)

3. Use of feeding bottles with child lying flat on the lap of the mother. This may result in milk regurgitating into the patulous and short eustachian tube of the infant.
4. Laying down the child flat after a full feeding without burping.

Management

1. Instillation of saline nasal drops and administering decongestant antihistaminic syrup, pain-relieving syrup and antibiotic as per the associated symptoms.
2. *Do not instil ear drops*; do not try to clean the ear even if there is a discharge. Do not use cotton ear tips.

Referred Otalgia

The pain felt in the ear for the pathologies in the adjoining regions is known as referred otalgia (Table 20.2). This is due to the shared innervations of the inner ear with these regions. The following table shows the pathology and the respective nerves involved. On examination the ear appears normal. Trigeminal nerve can be a rare cause for referred otalgia.

Bleeding from the Ear

History

- Trauma
- Use of cotton ear tip
- Nasal block with pain in the ear
- Frank blood (trauma) or associated watery/purulent discharge (otitis media)
- Injury to head (watch for other signs of head injury)

- Injury to chin often causing bilateral ear bleeding due to impaction of external ear canal often misunderstood as head injury

Examination

- Pinna should be gently pulled backwards to examine the canal for laceration.
- There can be oedema of the external canal or perforation of tympanic membrane.
- If the canal is filled with wax, it should be cleaned only if soft and easy to clean by suction.
- Cleaning can be done after initial bleeding has settled.
- In children and infants, no attempt to clean should be made, since it may cause more pain and trauma.
- Do not plug the ear in suspected head injury which can cause increased pressure in the canal and can get conducted intracranially, and plugging can also promote infection.
- Examination of nasal cavity may show nasal block, which could be the cause of acute otitis media resulting in bleeding from the ear.
- Laboratory investigation for clotting profile is necessary only if there is history of bleeding diathesis or if the bleeding doesn't stop within the time of average clotting.
- Never apply ear drops.

Ear drops and ear tips: There is a tendency to use or prescribe ear drops in every ear symptom – be it pain, blocked sensation, itching and finally trauma to ear. The fact is that majority of ear problems do not require any ear drops. Ear drops applied unnecessarily wets the ear, and in traumatic perforation, the medication is potentially more harmful than the pathology. The next danger posed to the ear is the use of ear tips. This can potentially damage the eardrum, destroy the epithelial lining of external canal and also can introduce infection.

A useful algorithm which could be utilised in the evaluation of painful ear is depicted in Fig. 20.4.

Foreign Bodies in the Ear (FB) (Table 20.3)

- Syringing with warm water is the best method to remove any foreign body of the ear.
- Syringing should be done without touching the external canal skin, since it is very sensitive.
- Glycerine syringe is the ideal equipment along with suction apparatus and appropriate suction tip.

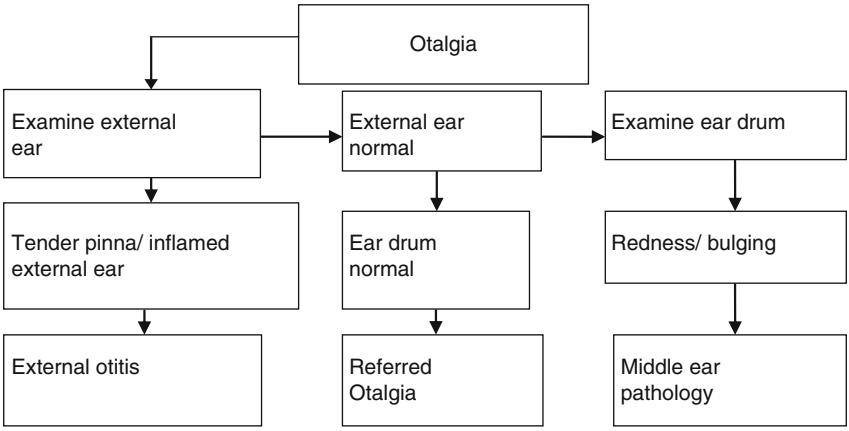


Fig. 20.4 Algorithm for otalgia

Table 20.3 Foreign bodies in the ear

Rubber pieces
Vegetable seeds
Plastic beads
Insects
Cotton tips
Metallic objects

- In case of a live insect, lignocaine is an effective agent to kill the creature quickly, which can be subsequently removed by syringing.
- Syringing is the ideal method in vegetable seeds also, with deference to the concept that vegetable may swell up with water, since the FB is going to be removed as soon as possible.
- As far as possible, the use of metallic sharp instruments should be avoided.

In case of a child, proper immobilisation of the child is essential. The classical child-holding position should be practiced. The child should be made to sit on the lap of the parent or assistant in straight posture with both the legs of the child crutched between the thighs and the left hand holding the wrist (narrowest part) of both the hands of the child together and the right palm of the examiner holding the forehead (not the chin to avoid choking) of the child firmly against chest of the assistant (Fig. 20.5).

- Occasionally when the foreign body is impacted at the isthmus or if there is an inflammation and oedema of external canal and in children, general anaesthesia may be needed for removal.
- In case of the presence of wax (cerumen), it is prudent to apply wax solvent ear drops and remove by syringing, the following day.
- Live insect FB is a true emergency.

Fig. 20.5 Positioning a child for clinical examination

Vertigo

- Vertigo, also known as dizziness and giddiness, is defined as abnormal sense of movement either rotatory or linear, if linear, back and forth or sidewards.
- Vertigo, though it is basically a symptom, can represent a large group of diseases.
- The lifetime incidence of vertigo is about 30 % for any individual, and in most occasions, the cause for vertigo is indeterminate since it is often idiopathic.

Vertigo is discussed in detail under the chapter Dizziness and Syncope, elsewhere in the book.

Facial Palsy

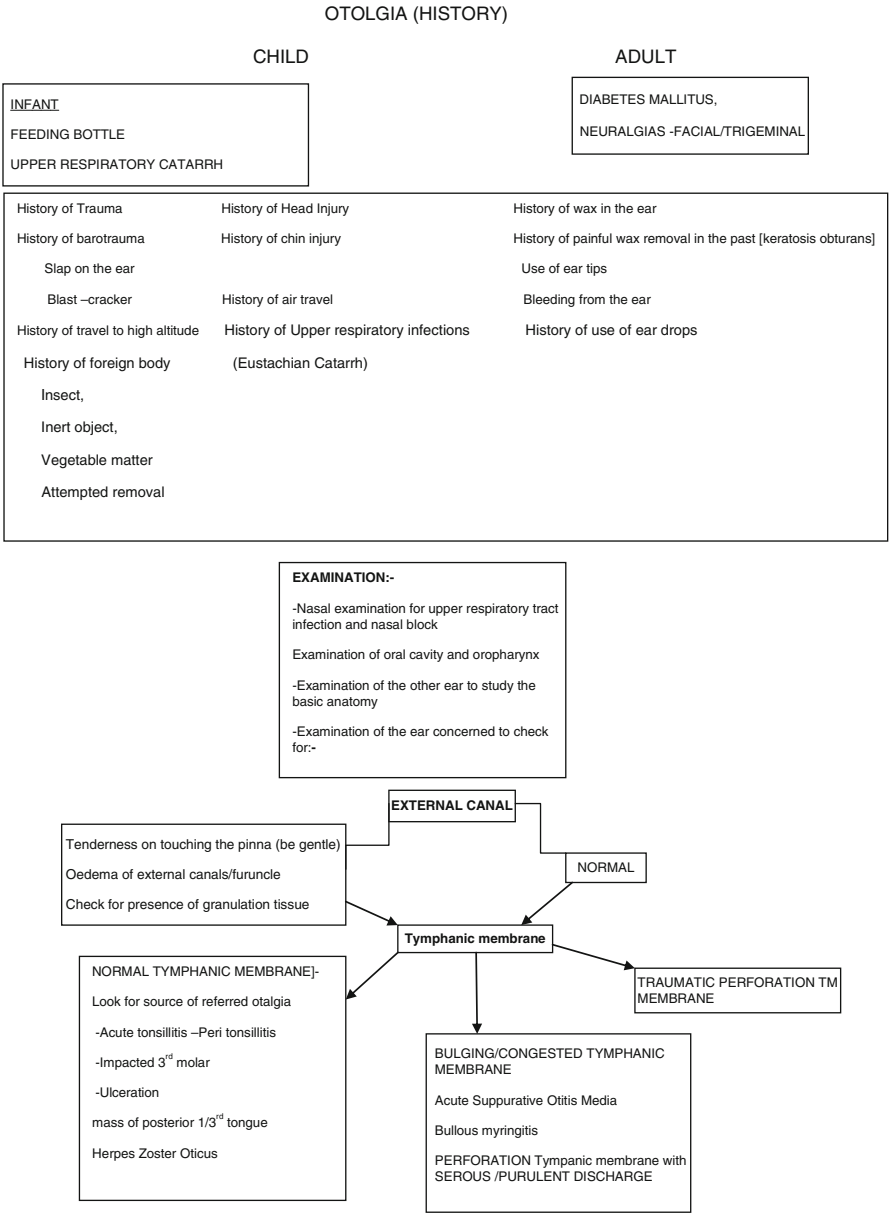
- Facial palsy causes cosmetic disfigurement, functional disability and anxiety due to uncertainty of the cause and prognosis. The commonest form of facial palsy is the Bell's palsy [6].
- Facial palsy associated with co-morbidities such as middle ear disease, trauma or vascular episodes is understood and managed with the primary disease.
- Facial palsy could be of either upper motor neuron (UMN) or lower motor neuron (LMN) origin.
- Bell's palsy is a LMN facial palsy, sudden in onset with or without pain.
- Symptoms include facial asymmetry, inability to close the eyes, inability to chew and swallow food as well as decrease in ability to taste.
- There is decrease in the amount of tears and saliva produced.
- On examination the ear canal and tympanic membrane appear healthy, unless the condition was preceded by an episode of common cold, due to which there could be signs of eustachian catarrh.
- Steroids, vasodilators and antiviral drugs are the main stay of therapy.
- Tablet prednisone for 1 mg/kg or 60 mg/day for 6 days is to be administered, which is and tapered off in 10 days.
- Tablet acyclovir is given at a dosage of 400 mg orally five times daily for 10 days.
- Physiotherapy is to be commenced early, to maintain the muscles of the face active and well exercised.
- Majority of cases recover fully. However, few cases remain with residual palsy.
- In case of signs of exposure keratitis, blepharoplasty may be required.

Sudden Hearing Loss (Idiopathic)

- Defined as a minimum of 30 dB hearing loss in three consecutive frequencies occurring within 3 days.
- Symptoms are associated with ear infections, trauma and neuro-otological conditions such as Meniere's syndrome. However, in most patients, the cause cannot be identified [7]
- Viral, vascular and autoimmune theories are postulated.
- Patient presents with sudden profound hearing loss with or without tinnitus and vertigo, more commonly unilateral. Nine out of ten people with SSHL lose hearing in only one ear.
- Obtain history of pre-existing hearing loss which the patient is ignorant of; the close relative of the patient has to be questioned. Also enquire about intake of ototoxic drugs; fever of high degree could have caused toxicity due to viral/bacterial infection.
- Requires early specialist referral.
- The prognosis is unpredictable.

Tips to Prevent Otolgia

- In a case of an infant, advise the mother not to feed the infant in a supine position, since regurgitation of milk into the short and patulous eustachian tube tends to occur.
- Both in child and in the adult, usage of nasal drops will relieve symptoms.
- Steam inhalation helps to open the eustachian tube and moisturises the respiratory mucosa.



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Chapter 21
Acute Eye Emergencies

Adam Chesters

Key Points

- Most eye emergencies present with pain, redness, altered vision or a combination of any of the three.
- The task of the emergency physician is to provide relief of pain, thoroughly examine the eye and related structures and ultimately decide whether the patient requires immediate specialist referral for a sight-threatening emergency.
- A thorough and systematic assessment using simple examination techniques will identify both the 'safe' eye and the eye that needs immediate treatment in the ED or referral to an ophthalmologist.
- The majority of presentations to the ED will be manageable with simple advice, simple medications and follow-up with their own family doctor or optician as required.

Introduction

The requirement to assess and treat ocular conditions is common to most emergency departments (ED). While some conditions can be considered minor, some even self-limiting, other conditions are a direct and immediate threat to sight. The majority of presentations to the ED will be manageable with simple advice, simple medications and follow-up with their own family doctor or optician as required [1]. Some cases are best managed by referral to a dedicated eye clinic for further assessment or

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follow-up. The purpose of this chapter is to provide emergency physicians with an approach to injuries and acute illnesses that affect the eye. By the end of the chapter, you will know which conditions must be excluded every time, the treatment approach to conditions that you will see and the emergencies that warrant waking up an ophthalmologist in the middle of the night.

Clinical Anatomy

The important external structures related to the eye are the bony confines of the orbit (including the orbital floor), the eyelids (including tear ducts) and the extraocular muscles. The anterior structures are defined as the cornea, the iris and ciliary body and the lens. The normal cornea and lens are both clear and avascular. Between the two, and produced by the ciliary body, is the aqueous that fills the anterior chamber and drains through the trabecular meshwork at the angle of the iris and cornea. The vitreous is a transparent jelly that fills the ocular cavity behind the lens. It is attached peripherally and at the optic nerve head. The retina is composed of two layers, the light-sensitive neuroretina and the outer, pigmented layer that provides nourishment to the neuroretina and shields if from excess incoming light. Both layers are adherent to the posterior rim of the globe at the ora serrata and the optic nerve head. Finally, the optic nerve head is the location where the ganglion cell axons, receiving impulses from the neuroretina, exit the eye to form the optic nerve. Otherwise known as the optic disc, this is also the point of entry for the blood vessels supplying and draining the retina.

Clinical Evaluation

The following is a nine-stage examination process [2]. The detail of how to perform each examination is beyond the scope of this chapter and can best be learned from a good teacher in the clinic, the ED or on the wards.

1. Visual acuity
   (a) Snellen chart (distance vision)
   (b) Reading small print (near vision)
   (c) Colour vision (Ishihara plates)
2. Visual field
   (a) By confrontation, one eye at a time
3. Extraocular movements
4. Eversion the lids
5. Pupils

(a) Light reflexes (direct, consensual, presence of afferent defects)
(b) Shape
(c) Accommodation

6. Cornea
   (a) External examination with a slit lamp
   (b) Fluorescein staining
7. Depth of the anterior chamber
   (a) Eclipse test (light shone from the side will illuminate all of a deep anterior chamber and only half of a shallow anterior chamber)
8. Digital assessment of tone
9. Ophthalmoscopy (with the pupil dilated)

If nothing else, the disciplined application of this examination approach will enable the emergency physician to declare that the eye is 'safe' and that there are no features of sight-threatening illness or injury that warrant immediate action. This, rather than the detailed diagnosis and management plan, is a reasonable ED goal.

Acute Disease and Common Injuries Affecting the Eye

- Most eye emergencies present with pain, redness, altered vision or a combination of any of the three.
- Visual disturbance can be partial or complete, one or both eyes can be affected and onset of symptoms can be sudden or gradual.
- Table 21.1 contains the important differential diagnoses that must be excluded for acute presentations associated with pain, redness and loss of vision.

Table 21.1 Important differential diagnoses to be excluded with presentations to the emergency department of pain, redness or acute loss of vision

Symptom	Pain	Redness (of the sclera)	Acute loss of vision
Important differential diagnoses	1. Acute angle-closure glaucoma 2. Keratitis (abrasions, ulcers, lacerations, foreign bodies) 3. Iritis 4. Optic neuritis 5. Pain around the eye (ophthalmic herpes zoster, temporal arteritis, periorbital and orbital cellulitis)	1. Keratitis 2. Iritis 3. Acute angle-closure glaucoma	1. Central retinal artery occlusion 2. Central retinal vein occlusion 3. Retinal detachment 4. Vitreous haemorrhage 5. Optic neuritis

Angle-Closure Glaucoma

Introduction

- Acute angle-closure glaucoma (AACG) is an ocular emergency that requires rapid identification, rapid treatment in the ED and immediate referral to an ophthalmologist.
- This is a sight-threatening condition.
- The condition is more common in the elderly, and in females, and is ten times more common in patients of Asian origin than white patients.

Pathophysiology

- There is reduced or obstructed outflow of the aqueous through the trabecular network at the angle of the iris and cornea.
- Continued production by the ciliary body contributes to an accumulation and subsequent increase in intraocular pressure (IOP).
- The precipitating factor is often a cause of pupil dilatation (such as dim light or emotional stress), which causes contact between the iris and the lens and closure of the drainage angle.
- Long-sighted patients with shallow anterior chambers are at greatest risk.

Clinical Features

- Angle closure is defined when at least two of the following symptoms and at least three of the following signs are present [3]:
  - Ocular pain
  - Nausea or vomiting
  - History of intermittent blurring of the vision or haloes
  - Intraocular pressure greater than 21 mmHg
  - Conjunctival injection
  - Corneal oedema
  - Mid-dilated and non-reactive pupil (Fig. 21.1)
  - Shallow anterior chamber

The patient will complain of a 'boring' pain that is associated with an ipsilateral headache.

- A recent history of similar lower-intensity symptoms may be described (intermittent blockage of the angle as the pupil dilates, but does not become stuck).
- A careful eye examination will reveal the cardinal features of the condition.

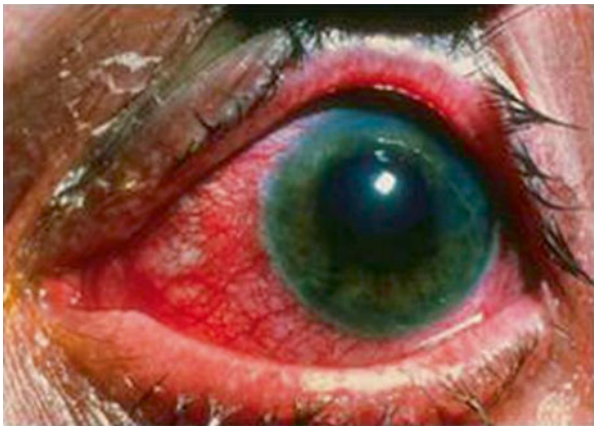


Fig. 21.1 Acute angle-closure glaucoma

- Under no circumstances should mydriatic drops be used in this group of patients (the angle closure will worsen), and fundoscopy will often be obscured by corneal oedema.
- Visual acuity may be reduced to 'counting fingers' or 'light perception'.

Investigations

- The diagnosis is a clinical one.
- Accurate intraocular pressure measurement is not required in the ED but will be performed by an ophthalmologist.

Treatment

- Reduction of IOP (through the reduction of aqueous production)
  - Intravenous and oral acetazolamide
  - Application of a topical beta-agonist (such as timolol)
- Suppression of inflammation
  - Topical steroids suppress inflammation and reduce optic nerve damage.
- Reversal of angle closure (by pupil constriction)
  - Pilocarpine is a topical miotic that should be administered 1 h after commencing the above treatment. A total of three doses are given at 15-min intervals.
  - Within the first hour of treatment, there may be a pressure-related ischaemic paralysis of the iris that renders pilocarpine ineffective. A theoretical concern exists regarding a paradoxical increase in IOP caused by induced anterior lens movement.

- Systemic complications
  - Simple analgesia and anti-emetics may help reduce anxiety and the IOP and should be used liberally.

Prognosis

- With timely and adequate treatment, vision can be recovered.
- The situation that has predisposed one eye to the condition probably exists in the other eye.
- Definitive treatment is bilateral laser peripheral iridotomy.

Orbital Cellulitis

Introduction

- Orbital cellulitis is a sight-threatening emergency requiring prompt and aggressive treatment.
- It is an infection of the soft tissues of the orbit posterior to the orbital septum.
- Differentiating the condition from pre-septal (periorbital) cellulitis is critical.

Pathophysiology

- Infection of the orbital soft tissues can be the result of direct inoculation from trauma or surgery, systemic bacteraemia with haematogenous spread or most commonly, extension of infection from adjacent structures (such as the paranasal sinuses or dental structures).
- The typical organism causing the infection is dependent on the area from which inoculation has occurred.
- In the case of penetrating trauma, a retained foreign body may act as the focus of infection.
- Rarely, fungal orbital cellulitis occurs, especially in immunocompromised patients and carries a significant mortality rate [4].

Clinical Features

- Cardinal signs of orbital cellulitis are proptosis and painful ophthalmoplegia (Fig. 21.2).
- Conjunctival chemosis and decreased vision are usually present.
- The key differentiation is between pre-septal and orbital cellulitis. Comparative features are listed in Table 21.2.

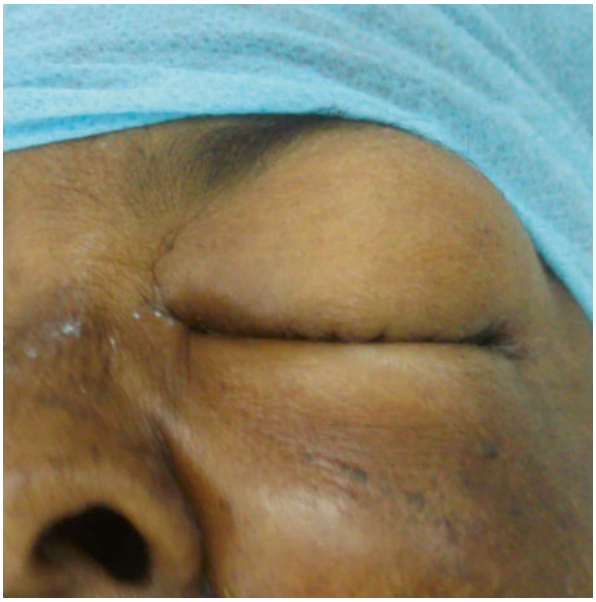


Fig. 21.2 Orbital cellulitis

- In addition to the pain, redness and disturbed vision that is common to many eye emergencies, important aspects of the history include:
  - Recent trauma or surgery to the eye (or surrounding structures)
  - History of sinusitis, dental infection or upper respiratory tract infection
  - Systemic features of infection
  - History of, or risk factors for, immune compromise

Table 21.2 Comparison of orbital and pre-septal cellulitis

	Pre-septal cellulitis	Orbital cellulitis
Aetiology	Local skin infection	Spread from sinuses or haematological spread
Clinical findings	Periorbital induration, erythema, warmth, tenderness	Proptosis, chemosis, painful ophthalmoplegia, reduced visual acuity, abnormal pupillary reaction (afferent defect)
Common bacteria	*Staphylococcus aureus*, *Streptococcus pyogenes*	*Streptococcus pneumonia*, *Haemophilus influenza*, *Staphylococcus aureus*, anaerobes
Treatment	Oral or intravenous antibiotics depending on systemic features and initial response	Intravenous antibiotics in all cases

Investigations

- In addition to inflammatory markers (CRP, ESR) and white cell count that may assist with tracing disease progression and response to treatment, blood cultures and microbiology swabs of any discharge may assist in organism identification.
- The patient should be investigated for systemic sepsis and end-organ dysfunction as the clinical situation dictates.
- The most important specific investigation is a high-resolution CT scan of the orbits and/or MRI. This will assist in the diagnosis and identify any abscesses amenable to drainage.

Treatment

- Intravenous broad-spectrum antimicrobials, started immediately based on most likely organisms and honed as necessary with results of microbiology cultures and sensitivities.
- Source control, including referral for consideration of drainage of adjacent dental or sinus abscesses or of the orbital abscess itself.
- Emergency ophthalmological surgery to drain orbital abscesses if a decrease in vision occurs, an afferent pupillary defect develops and proptosis worsens or if there is an uncomplicated abscess that does not respond to antimicrobial therapy.
- The treatment of the septic patient who requires immediate resuscitation or organ support is outside of the scope of this chapter and should proceed in line with evidence-based guidelines.

Prognosis

- Despite a reduction in mortality rate with prompt recognition and treatment, about 10 % of patients will become permanently blind in the affected eye [5].
- Other significant complications include meningitis, cavernous sinus thrombosis and intracranial abscess formation.

Conjunctivitis

Introduction

- Conjunctivitis is usually a benign, self-limiting disease [6].
- The importance to the emergency physician is to firstly identify when the painful and red eye is actually a manifestation of one of the life-threatening eye emergencies and secondly to be aware of the rare cases in which conjunctivitis can evolve to become a sight-threatening orbital infection.

Pathophysiology

- The term is a description rather than a disease itself and describes any inflammatory process affecting the conjunctiva.
- Classification is usually based on causative process, which includes bacterial, viral and fungal infections.
- Allergic conjunctivitis is associated with a local immune response that causes the inflammation.
- Neonatal conjunctivitis is a syndrome in its own right.

Clinical Features

- Regardless of the aetiology, the clinical presentation of redness, discharge and irritation is constant (Fig. 21.3).
- Often the patient will describe their eyelids sticking together on waking or a gritty sensation.
- Some may think that a foreign body is present.
- Visual acuity should be normal (although pus and exudate may blur vision initially).
- In the case of infectious conjunctivitis, a history of other family members affected or of a recent upper respiratory tract infection may be present.
- If the conjunctiva is inflamed on only one side, the aetiology is less likely to be infectious or allergic.
- By definition, the eye examination will be normal except for inflammation of the conjunctiva.
- If inflammation is present on the cornea, then kerato-conjunctivitis exists.

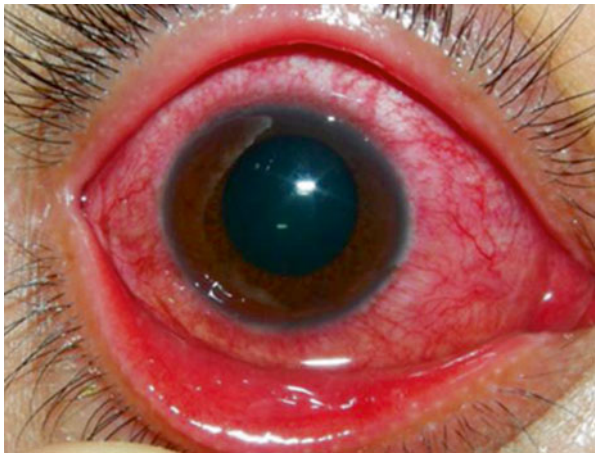


Fig. 21.3 Viral conjunctivitis

Investigations

- Conjunctivitis in adults is a clinical diagnosis and requires no specific investigations.
- A swab of any purulent discharge may isolate an organism but may not affect management.
- Neonates and patients who are immunocompromised may require additional investigations.

Treatment

- Treatment is supportive and targeted at symptom control.
- Cold compresses and artificial tears may offer some relief from the discomfort.
- Topical antibiotic treatment for infectious conjunctivitis (only 50 % of which will be bacterial) has been shown to result in early improvement of symptoms, but the duration of illness is not reduced.
- Treatment considerations should include the risk of antibiotic resistance.
- Mild allergic conjunctivitis can be treated with a simple antihistamine agent (topical or oral).
- Patients with severe conjunctivitis (evidenced by marked chemosis, lid swelling or spread to involve the cornea) or disease that is not resolving should be referred to an ophthalmologist for further assessment.
- Patients thought to have herpes simplex conjunctivitis, herpes zoster conjunctivitis or chlamydial conjunctivitis should also be referred for further assessment.

Prognosis

- Most disease is self-limiting.
- The patient should be educated regarding hand hygiene and other strategies (such as not sharing facial towels) to reduce transmission to contacts and the other eye.

Corneal Disease/Trauma

Introduction

- A breach of the corneal epithelium can be caused by a number of disease processes, but is most commonly caused by trauma, and without penetration of the globe.
- Contact lens wearers are at particular risk, and a foreign body can remain in situ.
- A red eye secondary to corneal disease or trauma is a common presentation to the ED.

Pathophysiology

- Keratitis is the term used to describe any corneal epithelial breach and occurs in any situation that causes epithelial compromise.
- Most commonly, the defect is limited to the most superficial of the five layers of the cornea.
- Severe corneal injury can involve the deeper stromal layer, in which case the term corneal ulcer is used.

Clinical Features

- Regardless of the cause, the clinical presentation will be remarkably similar:
  - Intense pain
  - Watering eye
  - Injected sclera (with ciliary injection)
  - Blurred vision (if the injury lies on the visual axis)
- A thorough history will reveal risk factors and identify potential causes. Common causes of keratitis are:
  - Injury
  - Debris
  - Extended contact lens wear
  - Foreign bodies
  - Exposure to electric arc welding, strong sunlight or tanning beds
- Rigid compliance with a standard eye examination routine will reveal all necessary signs, the cardinal of which is the diagnostic uptake of fluorescein.
- Globe penetration and a retained foreign body must be excluded in all cases.
- Dendritic ulcers are caused by herpes simplex virus and have a characteristic branched appearance.

Investigations

- Clinical examination is diagnostic.
- If there is a history of high-velocity foreign object injury, or if globe penetration is suspected, radiography of the orbits is indicated.

Treatment

- All foreign bodies should be removed.
- Simple corneal abrasions can be managed with supportive treatment.
- Simple analgesia can be advised.

- Taping the eye shut can improve symptoms and promote healing.
- The routine use of topical antibiotic ointment is not recommended, but artificial tears may provide some lubrication and symptom relief.
- All corneal ulcers (including dendritic ulcers) must be referred to an ophthalmologist for further assessment, treatment and follow-up.
- Contact lens wear should be avoided until symptoms have completely resolved (nominally, a week is a sensible minimum length of time to wait).
- Patients with reduced visual acuity should be asked to follow up with an optician to ensure resolution.
- Failure of symptoms to resolve within 48 h, decreased vision or increased pain should prompt a re-attendance for further assessment.

Prognosis

- Full recovery of vision and prompt resolution of symptoms (within 48 h) are the norm in the case of simple corneal abrasions.
- Corneal ulcers may heal with a scar, leading to a loss of vision that depends on the location of the scar in relation to the visual axis.

Haemorrhage

- Subconjunctival haemorrhage
  - Subconjunctival haemorrhage is usually unrelated to any serious underlying pathology.
  - A patient who has an undiagnosed disease of coagulation, or who is taking anticoagulant medication, is more likely to have a subconjunctival haemorrhage (Fig. 21.4).
  - Very rarely, the amount of blood in the subconjunctival space is sufficient to block the trabecular drainage angle and lead to an acute rise in IOP.
  - In the context of facial trauma, a subconjunctival haemorrhage without a posterior border should prompt a search for a zygoma fracture.
  - Uncomplicated subconjunctival haemorrhage requires no specific treatment. The patient should be reassured.
  - A routine 'clotting screen' should only be performed if there is a high index of suspicion of an underlying coagulopathy.
- Hyphaema
  - Blood in the anterior chamber is hyphaema (Fig. 21.5).
  - It is nearly universally the result of blunt trauma to the eye.
  - The blood is a result of injury to any of the ocular structures.

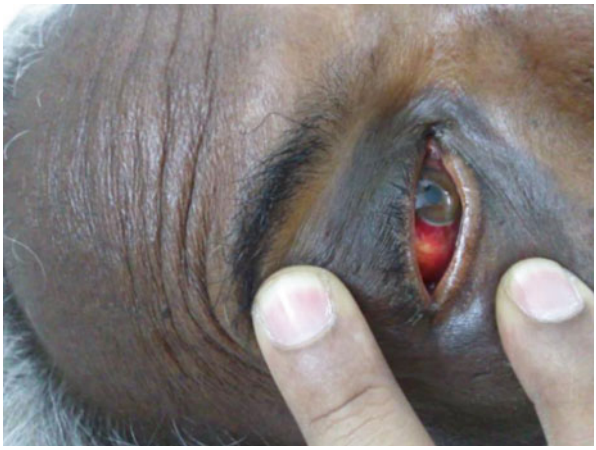


Fig. 21.4 Subconjunctival haemorrhage in a patient on warfarin

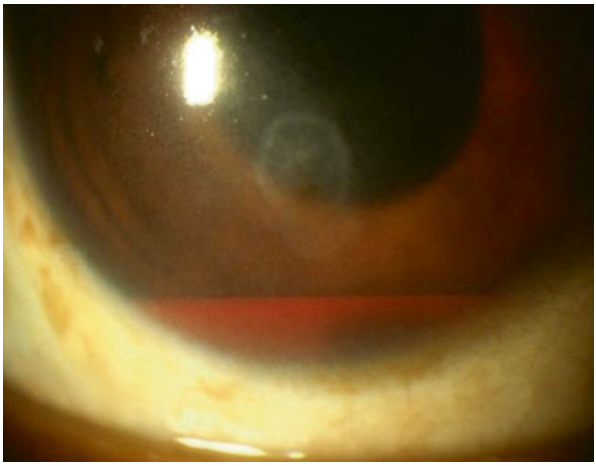


Fig. 21.5 Hyphaema

- Assessment of the upright patient will reveal a fluid level that settles at the inferior pole of the anterior chamber and is described in terms of percentage of the anterior chamber occluded.
- There is no reduction in visual acuity unless the visual axis is obscured or the intraocular pressure has become acutely elevated.
- There are three issues that make hyphaema an important diagnosis to make: thorough assessment of the ocular structures, the occurrence of secondary haemorrhage and the development of secondary glaucoma or corneal bloodstaining.
- Immediate treatment of an uncomplicated hyphaema in the ED is to protect the injured eye with a patch and provide adequate analgesia.
- The patient should be referred to ophthalmology within 24 h.

  - Around 15 % of patients have poor visual results, some as a result of associated trauma and some as direct sequelae of the hyphaema itself.

- Vitreous Haemorrhage
  - Vitreous haemorrhage presents as sudden visual loss together with flashing lights and substantial numbers of floaters.
  - It is an ophthalmology emergency requiring immediate specialist assessment.
  - Visual acuity may be significantly reduced, the pupil will react normally but fundoscopy will be obscured by the dark haemorrhage.
  - Prognosis depends on the cause but sight can be threatened.

Retinal Detachment

Introduction

- If detected early, retinal detachment can be repaired with favourable outcome [7].
- It is a time-critical eye emergency that warrants urgent ophthalmological assessment.
- The process is more common in the elderly, high myopics and those who have undergone cataract removal.
- Traumatic detachments are more common in younger patients playing contact sport.

Pathophysiology

- Retinal detachment describes the separation of the outer, pigmented layer from the underlying neuroretina.
- A tear in the neuroretina that allows vitreous to leak in between the two layers and strip them apart can cause separation.
- Alternatively, traction from inflammatory membranes, tethered to the vitreous, can initiate the process.

Clinical Features

- Flashing lights, floaters and vision loss are the cardinal features of retinal detachment.
- The vision loss is often peripheral and progressive.
- Central vision loss occurs as the macula becomes affected.
- The history may identify one of the risk factors for the condition, and the usual examination routine will pick up the salient clinical signs of peripheral field defect and an elevation of an undulating retina at fundoscopy (Fig. 21.6).

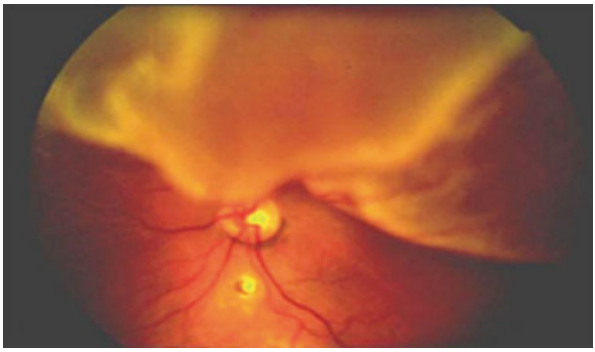


Fig. 21.6 Retinal detachment

Investigations

- This is a clinical diagnosis and no specific ED tests are required.

Treatment

- The key intervention is a referral to an ophthalmologist for consideration of reparative surgery.
- The urgency with which this occurs depends on the central visual acuity (macular involvement).
- In most cases, a careful explanation and a referral to an eye clinic within 24 h is most appropriate.
- The retinal detachment repair is usually carried out as an outpatient day-case operation.

Prognosis

- Visual function is usually restored, but the prognosis is poorer if the macula is involved.

Chemical Burns

Introduction

- Chemical burns to the eye (in this context, considered to be affecting the cornea predominately) are a cause of morbidity and permanent vision loss.
- Treatment in the ED is crucial in improving outcome and is a specific case in which the emergency physician is in the best position to directly deal with an eye emergency.

- Burns to the cornea can be classified as acid or alkali.
- The severity is dictated by the contact duration, solution quantity and the solution penetrability.

Pathophysiology

- Alkali substances penetrate more rapidly than acids and cause cell destruction and death.
- The end stage is liquefactive necrosis as the damaged cells cascade an inflammatory response.
- Alkalis penetrate the anterior chamber within 15 min, causing damage to the internal structure.
- Acids cause contact coagulation of the corneal epithelium, partially preventing penetration into the anterior chamber.
- Acid burns, therefore, tend to remain superficial (Fig. 21.7).

Clinical Features

- It is important to ascertain the exact nature of the chemical, ideally inspecting the container or information leaflet.
- The symptoms, as with any corneal injury, will be pain, abnormal vision and redness.
- In one of the only departures from the usual eye assessment, pH testing is an essential part of the routine.
- Along with visual acuity, the pH measurement is the only assessment that should delay the instigation of copious irrigation.

Investigations

- No specific investigations are required.

Treatment

- Immediate irrigation is required.
- Continuous irrigation with 0.9 % sodium chloride must continue until the pH of the eye has returned to normal (or closely matched with the other eye).
- Topical anaesthetic application will allow the patient to tolerate this unpleasant procedure.
- A number of commercially available products are available to facilitate the irrigation.

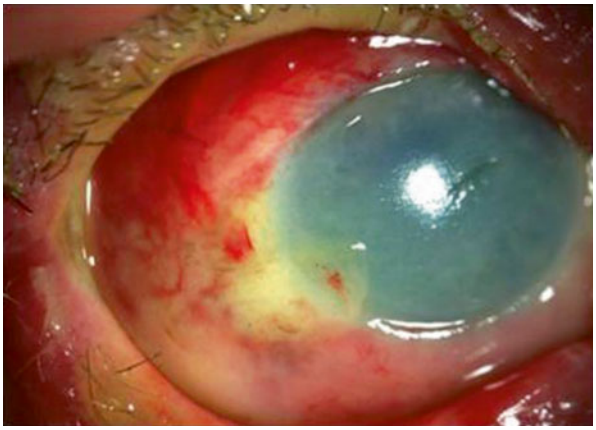


Fig. 21.7 Acid burn

Table 21.3 Roper-Hall classification of ocular surface burns

	Cornea	Conjunctiva	Prognosis
Grade 1	Corneal epithelial damage	No limbal ischaemia	Good
Grade 2	Corneal haze	<1/3 limbal ischaemia	Good
Grade 3	Total epithelial loss, unable to visualise iris	1/3–1/2 limbal ischaemia	Moderate
Grade 4	Cornea opaque, unable to visualise iris or pupil	1/2 limbal ischaemia	Poor

- Once the pH has normalised (or if the pH fails to normalise), advice from an ophthalmologist must be sought.

Prognosis

- The prognosis depends on a number of factors.
- The Roper-Hall scale (Table 21.3) classifies corneal burns according to clinical features present after irrigation [8].
- Higher grades are associated with a poorer visual prognosis.

Central Retinal Artery Occlusion

Introduction

- Central retinal artery occlusion (CRAO) causes sudden and rapidly progressive painless loss of vision in one eye.

- It is more common in patients over the age of 60, unless a specific risk factor such as valvular heart disease coexists.

Pathophysiology

- The central retinal artery is a branch of the ophthalmic artery, which supplies blood to the retina.
- Occlusion of this blood supply causes ischaemia and rapid loss of vision.
- Common causes include embolism, atherosclerosis and temporal arteritis.

Clinical Features

- Visual acuity may have reduced to light perception only.
- There may be a history of intermittent visual loss (amaurosis fugax) in the previous hours.
- The standard eye assessment may reveal an afferent pupillary defect, a pale retina at fundoscopy (the classic cherry red macula takes several hours to develop).
- Emboli may be visible in branch retinal vessels to the experienced fundoscopist.
- A general physical examination may pick up features of the underlying cause or signs of temporal arteritis (that must be excluded in order to take measures to protect the other eye).

Investigations

- The platelet count may be useful to exclude polycythaemia.
- The erythrocyte sedimentation rate (ESR) is an essential investigation if temporal arteritis is suspected.
- A number of other specialist investigations will target the underlying cause.

Treatment

- Immediate lowering of the intraocular pressure may help to dislodge the clot.
- A single intravenous or oral dose of acetazolamide is an ED treatment that can be commenced simultaneously with a request for emergency ophthalmological consultation.
- Early treatment with hyperbaric oxygen therapy may improve the prognosis.

Prognosis

- Most patients do not regain good visual acuity.
- Depending on the presence of a collateral blood supply to the macula, about 10 % of patients retain reasonable central vision.

- The presence of CRAO is an independent predictor of all-cause mortality.
- It is essential to address the underlying cause to prevent the other eye being affected.

Complex Ocular Trauma

Open Globe Injuries

- Open globe injuries are major cause of blindness [9].
- A primary, and often complex, surgical repair aims to restore the structural integrity of the eye and is often attempted regardless of the extent of the injury or the presenting visual acuity.
- Primary enucleation is a last resort for eyes that are beyond the technical capabilities of surgical repair.
- For the emergency physician, all attempts must be made to carefully document the extent of the injury, without causing any further damage.
- Associated injuries must also be dealt with.
- The assessment in the emergency department must include an objective assessment of visual acuity.
- The performance of other examination techniques is dependent on whether it is judged safe to do so.
- No digital (or other) pressure must be applied to a ruptured globe.
- Consideration should be given to plain radiographs and CT scans of the orbit to identify retained foreign bodies and define the extent of the surrounding damage.
- An immediate referral to an ophthalmologist is mandatory, and the eye must be protected with a non-adherent shield until further assessment.
- Address pain and anxiety early.

Blunt Trauma to the Globe

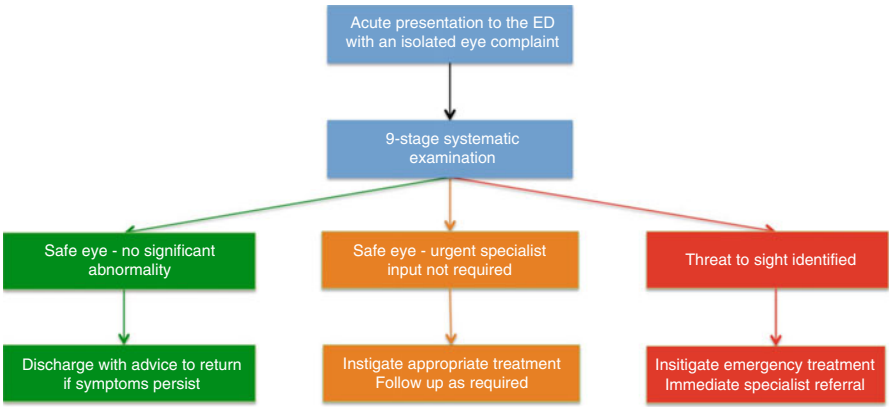
- An object that is small enough to impact the globe without fracturing the orbital rim causes orbital blowout fractures.
- The sudden spike in intra-orbital pressure buckles the relatively weak orbital floor, with potential downward displacement of the orbital contents.
- Other signs of blunt trauma to the globe (hyphaema, mydriasis, corneal injury, globe rupture, lid damage) must be sought.
- Entrapment of the extraocular muscles (inferior rectus) causes a squint and double vision.
- Plain radiographs of the facial bones may reveal fluid in the maxillary sinus and the ‘teardrop’ sign below the inferior orbital rim.
- A CT is warranted to determine the full extent of the injury.
- Urgent referral to an ophthalmologist is required in order to consider operative repair.

- Indications for conservative management include:
  - Absence of marked hypophthalmos
  - Absence of entrapped muscle
  - A fracture of less than 50 % of the orbital floor
  - Lack of diplopia
- Any blunt trauma to the globe may result in a retrobulbar haematoma, the expansion of which causes an acute orbital compartment syndrome causing pressure necrosis of the optic nerve and permanent blindness if not immediately released.
- The procedure of lateral canthotomy should be in every emergency physician's armamentarium.
- The indications for the procedure in the presence of a retrobulbar haematoma are acute visual loss and elevated intraocular pressure with proptosis.
- The problem is exaggerated in patients who are anticoagulated with warfarin or who are taking any other anticoagulant or anti-platelet agent.
- Emergency decompression may save the patient's sight.
- Irreversible vision loss can be expected with durations of severely reduced acuity longer than about 2 h.
- A lateral canthotomy is contraindicated in the presence of a ruptured globe and should also be carried out by an ophthalmologist if they are available in a timely fashion.

Summary and Algorithm

Management of the acute presentation of eye emergencies consists of:

1. A thorough, consistent and structured examination
2. Exclusion of sight-threatening emergencies
3. Instigation of emergency treatment uncommonly
4. Arranging appropriate outpatient treatment and/or follow-up usually



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Chapter 22
Acute Nose Disorders

T.L. Vasudevan and Suresh S. David

Introduction

The nose being highly vascular with communications with intracranial venous sinuses, it is highly prone for emergencies due to trauma, infections and allergy.

Applied Anatomy

- The nasal cavity is a triangular space and the anterior nares are at the antero-inferior corner. There is a tendency to assume the nose to be a pair of tubes. The posterior end of the nasal cavity – the choana – is wider; hence, an FB in the nose is predisposed to slipping backwards.
- The choana houses the adenoids, which is big in paediatric age group and is a cause for mouth breathing, epistaxis and ear pain [1].
- The antero-inferior aspect of the nasal septum is the Little's area – the junction of many blood vessels and the Kiesselbach's plexus, the commonest source of epistaxis.
- The inferior turbinate is often mistaken for a nasal polyp or foreign body.
- The inferior turbinate on either lateral wall is erectile tissues. They have reversible engorgement capability and function as protectors of the respiratory system, by

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filtering and holding large particles in the inspired air. They also control the flow of air and moisturise the inspired air.
- The dangerous area of the face is a triangle with the angles of mouth including the upper lip at the base and the apex at the root of nose [1]. Venous drainage from this region is communicating through the ophthalmic vein with the intracranial venous sinuses by veins which have no valves. Bacterial infections of this region can lead to cavernous sinus thrombosis.

Epistaxis

- Epistaxis is one of the commonest emergencies in ENT encountered by the emergency physician; this could range from trivial bleeding due to nose picking to being an expression of malignancy in the nose or nasopharynx (Table 22.1).
- Irrespective of the aetiology, the management is by standard techniques and protocols.

Management

- Keep the patient seated and provide a bowl to hold under the nose or to spit blood from the mouth.
- Establish IV access.
- Examine and attempt to locate bleeding point which is often not possible, because of active bleeding. Further, there may not be a visible pathology like granulation or aberration. The bleeding point could be hidden posteriorly.
- Pinch the nose (Hippocrates technique) – Both alae nasi are apposed against the nasal septum with index finger and thumb firmly for at least 5 min, while breathing is continued through the mouth. If available, ice cubes wrapped in cloth should be applied over the nose.

Anterior Nasal Packing

- Thudicum's nasal speculum and nasal dressing forceps are ideal for this procedure.
- Seat the patient on a bed and stand by the side.
- Use a good source of illumination, for evaluation.

Table 22.1 Aetiology for epistaxis

Children	Adult
Rhinitis	Nasal block
Trauma	Trauma
FB	Hypertension
Bleeding diathesis	Chronic sinusitis/neoplastic
Septal granulation	Tumour benign/malignant

- Ensure universal precautions – mask, gloves and eyewear.
- Have suction (with fine-tipped Frazier nozzle) available at the bedside.
- Apply a cotton pledget, soaked with oxymetazoline or phenylephrine (nasal decongestants), and apply pressure. Alternatively, the medication can be sprayed into the nostril.
- Roller gauze soaked in a combination of povidone-iodine, lignocaine and Inj. Adrenaline solution (2 % and 1:100,000, respectively) is ideal.
- Packing should commence from the posterior inferior end of the nostril, from the floor of the nose until the nostril is completely packed.
- The anterior tip of the roller pack should be accessible for later removal.
- Packing should be bilateral. The patient should be asked to breathe through the mouth.
- Maintain the pack for a maximum of 24 h.
- If available, commercial nasal packs could be utilised.
- If prior to packing, a bleeding point is identified over the Little's area, cauterisation with endoscopic bipolar cautery; trichloroacetic acid in 10 % dilution or 75 % silver nitrate may be utilised. Silver nitrate leaves behind black discoloration of the skin [2].
- While using materials for cautery, care should be taken not to touch healthy mucosa.

Postnasal Packing

- In case of bleeding from the posterior aspect of the nasal cavity, posterior nasal packing is necessary [3].
- Temporary postnasal tamponade can be achieved by the following technique:
- A pair of Foley's catheters is passed, one into each of the nostrils, until the balloon tip is visualised against the pharyngeal wall.
- Inflate the bulb with 5–15 ml normal saline.
- Retract the catheter gently, until it hitches snugly against the posterior nasal cavity.
- Apply a padded umbilical clamp across catheter, to prevent the balloon from dislodging.
- Proceed with anterior nasal clamping, as described earlier.
- Postnasal packing requires hospitalisation and antibiotic coverage and the pack is to be retained for about 24–48 h.
- Rarely, embolisation of blood vessels requires to be done.

Aftercare

1. Commence empirical antibiotic therapy. The incidence of maxillary sinusitis as a sequel is significant.
2. Admit for 12–24 h for observation of ongoing bleeding [4].

3. Instruct the patient the following:
   (a) Avoid nasal manipulation or nose blowing.
   (b) Sneeze with mouth open.
   (c) Avoid vasodilating actions – physical exertion, spicy foods and alcohol consumption.
   (d) Prevent mucosal drying by applying saline nasal sprays several times per day.
   (e) Return for review and removal of tamponade after 24 h.

Foreign Body (FB) in the Nose

- Commonly a paediatric emergency, FB can be of three types:
  - Miscellaneous objects – plastic or rubber components of toys, buttons, eraser rubber, sponge, etc.
  - Biological materials – vegetable seeds, rubber sponge, etc.
  - Chemical contents – capsules, tablets, chalk pieces, button cells, etc.

Clinical Features

- Known history of insertion of FB.
- Bleeding from the nose.
- Nasal discharge and block on one side – *Unilateral nasal discharge in children is almost always a FB unless proven otherwise*.
- Obtain history of attempted removal, since there could be:
  - Trauma to mucosa and the presence of blood clots obscuring the view of the FB
  - The FB would have relocated posteriorly and impacted.
  - The child has undergone unpleasant experience and hence would be uncooperative.
- Neglected vegetable FBs are often foul smelling due to stagnation and bacterial activity. They tend to develop granulation around them and would bleed on touch.

FB Removal

- Most of the FBs can be removed in the ED. Holding the child in proper position is important (Fig. 24.3).
- Reassure the child and explain the possibility of the FB getting pushed backwards into the oropharynx and being swallowed.
- Prepare for GA in case the FB is not able to be removed by this technique.
- Nasal endoscope is a useful tool in unusual foreign body.

Technique

- A long instrument blunt and curved at the tip is ideal. The nasal suction with curved tip, or a curved artery forceps, could be utilised. The instrument has to be passed upwards and backwards to get behind the FB and once behind adequate force can be used as traction to pull the FB.
- Similarly, a size 6 F Foley's catheter, smeared with lignocaine jelly, passed beyond the FB, inflated to fill the nasal cavity adequately and pulled forwards to bring the FB forwards, is a safe and effective alternative [5].
- Always look for more than one FB.
- After the procedure, pack the nasal cavity with a gauze roll, soaked with lignocaine to control persistent ooze, if present.

Septal Haematoma

- Septal haematoma is clearly visualised on examination, as a smooth bulge of the septum.
- There can be a history of trivial trauma or painful furuncle of the nose.
- Management is drainage by incision anteriorly and suction of the haematoma. The 2 mm incision is made vertically over the anterior part of the septal bulge; minimal dilatation of the opening done with a mosquito artery forceps and a thin nasal suction is used to suck out the serous fluid/altered blood/purulent collection [6].
- Bilateral anterior nasal packing with roller gauze is to be performed and retained for 48 h.

CSF Rhinorrhoea

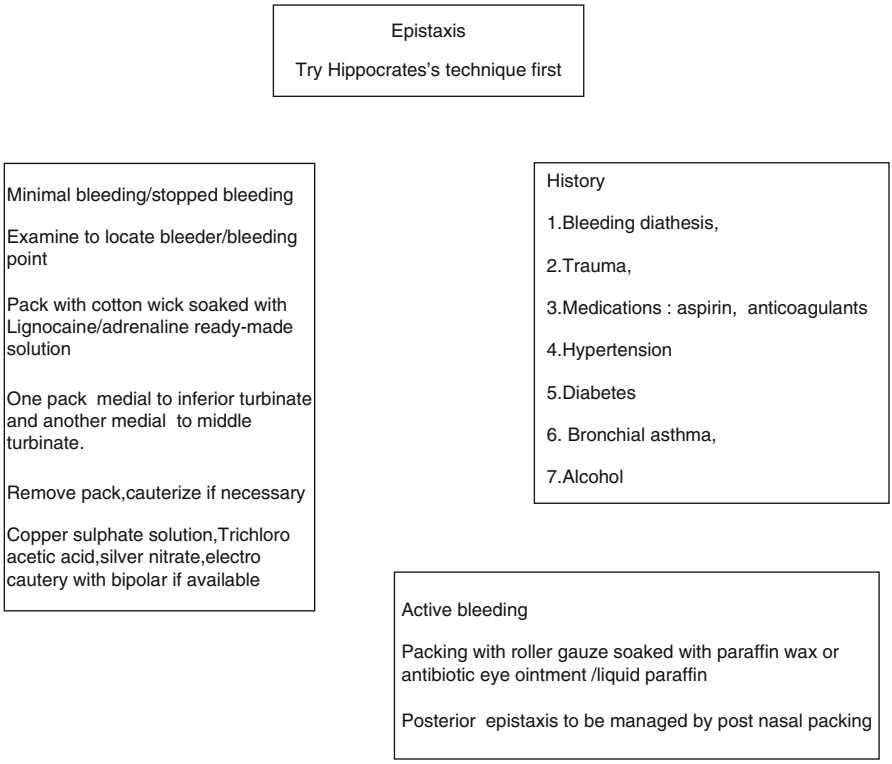
- The patient presents with clear watery nasal discharge on bending down.
- There may be a history of head injury that can be even trivial or history of nasal surgery.
- Associated symptoms can be headache, fever with chills and rigours indicating probable intracranial infections.
- There need not be a finding of rhinitis, unless there is associated sinus disease.

Filter Paper Test

- With the patient bending forwards, collect a few drops of the fluid on a blotting paper. If the drops are due to CSF leak, the filter paper would exhibit the double-ring sign – a central circle of blood and an outer clear ring of CSF [7].

- Collect the fluid directly in a sterile container, and subject it to biochemical analysis. The enzyme B2Tr is produced by neuraminidase activity of the brain and is present in CSF, perilymph and ocular aqueous humour but not in sinonasal mucous secretions and tears.
- Immunoelectrophoretic assay of beta-trace protein has been reported to have high specificity and sensitivity for CSF detection.
- Contrast enhanced CT scan to look for any lesion in the brain and the nasal cavity and to locate the leak. The leak is better demonstrated in contrast enhanced CT by injecting contrast in the spinal fluid.
- Management: Most of CSF leaks close spontaneously within 7–10 days.
- Conservative management includes absolute bed rest, medication to reduce intracranial pressure such as mannitol and broad spectrum antibiotic, if indicated.
- There is no role for empirical antibiotic therapy in CSF leak. If signs of meningitis such as fever with rigours are observed, and there is no other cause for fever, CSF analysis to detect the pathogen and antibiotic sensitivity should be carried out.
- Surgical management is by endoscopic CSF leak repair.

Management of epistaxis



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Chapter 23
Acute Throat Disorders

T.L. Vasudevan and Suresh S. David

Introduction

Sore throat is a common complaint in the emergency department and is often associated with benign conditions, such as pharyngitis. The neck and throat are regions rich in vascular and lymphatic activity. They also house the food passage and airway; hence, any pathology – allergic, infective, neoplastic as well as foreign bodies – causes significant discomfort. A high index of suspicion should be maintained to diagnose less common but serious pathology, such as epiglottitis and retropharyngeal abscess. This chapter briefly highlights the salient emergencies concerning the throat.

Foreign Body Throat

Foreign body in the throat is a true emergency and requires urgent attention.

Oropharynx

The tonsils act as pincushions in catching the sharp objects in the bolus of food; hence, FB like fish bone, sharp vegetable spicules, etc., are looked for in the tonsils and easily removed with the use of headlight, tongue depressors and a good pair of micro forceps.

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Similar FB in the posterior one-third of the tongue and the vallecula require the skill in using angled sinoscope and cooperation of the patient in the outpatient set-up.

Hypopharynx

Foreign bodies, such as pills, food materials, etc., can get trapped in the piriform fossae on either side of glottic inlet which are removed only by administering general anaesthesia.

Cricopharynx

Further impaction of food such as a mutton piece in the cricopharynx/oesophagus requires removal under general anaesthesia.

Since the above foreign bodies commonly do not cause airway obstructions, they are called semi-emergencies.

With a brisk swallowing reflux, the foreign bodies seldom enter the air passage.

A unique FB is a coin in the throat of a child [1].

A coin in the cricopharynx is seen as a circular object in the AP view of the X-ray neck.

A coin seen in the later view of the neck as circular object has to be in the trachea.

Of course any FB in the respiratory tree produces incessant cough, breathlessness and even cyanosis in case of obstruction. Hence, it is always preferable to take X-rays in both views to get an idea about the shape, location and size of a FB in the throat (Fig. 23.1).

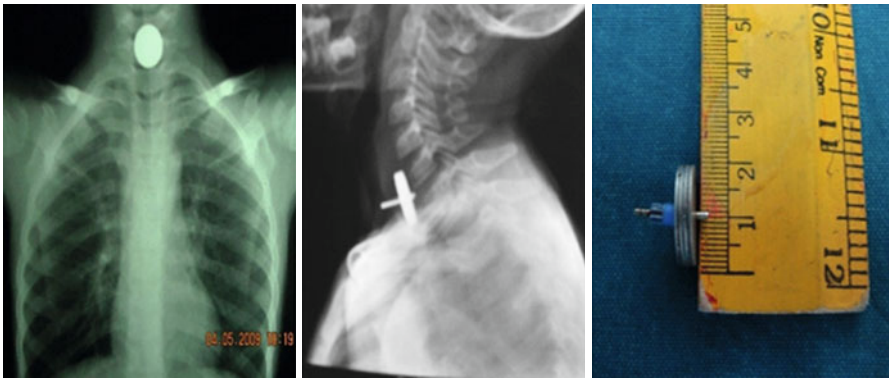


Fig. 23.1 Toy car wheel in the cricopharynx (Courtesy: Dr. S. Thangavelu, Mehta Children's Hospitals, Chetpet, Chennai – India)

Denture as a FB in the elderly is a challenging situation, because:

(a) Dentures are made of both radio-opaque and non-radio-opaque materials. It is prudent to obtain adequate description of the FB, before attempting removal.
(b) Due to the irregular shape, it can get impacted more easily.
(c) Co-morbid conditions among the elderly – therefore of serious concern particularly from the anaesthesiologist's perspective.

In children, FB in throat present with excessive salivation, refusal/difficulty for oral feeds and incessant cry. An important observation would be the presence of clarity in the voice and no breathing difficulty, thus indicating that the FB is not obstructing the airway.

FB: Obstructing the Larynx and Trachea-Bronchial Tree

The patient could be a child with history of FB in the throat presenting with incessant cough, breathing difficulty and sometimes stridor. Inability to produce a clear voice is an additional feature. This occurs when the child has ingested the FB and in the process could have inhaled the FB. On auscultation, the breath sounds could be reduced or absent.

Management

- If the FB is visible orally, finger sweep of the oral cavity can be attempted.
- Holding the child upside down and tapping the back in case of infants.
- Making the child lie down and pressing on the abdomen to force exhale.
- Tracheotomy/bronchoscopy may be indicated, if FB is suspected to be at the laryngeal inlet.
- In adults, in case of foreign body causing mild obstruction, patient can be encouraged to take slow inhalation of air and to produce powerful cough. The patient is made to lean forward and gentle and sharp blows between the shoulder blades with the heel of the palm.

In standing and leaning forward position, back blows are alternated with sharp abdominal thrust (Heimlich manoeuvre) can be attempted.

Abscesses In and Around the Neck: General Principles of Management

- Neck abscess usually presents with painful swelling in the neck, fever, dysphagia, dehydration, etc.
- General debility and airway obstruction are to be managed.

- Most common sources of infection are oropharyngeal infection and dental infections.
- Most common aerobes are streptococci viridans, followed by *K. pneumoniae* and *S. aureus*.
- Empirical antibiotic therapy, rehydration and steroid therapy are to be administered judicially. Penicillin G along with gentamicin which shows synergism with penicillin is the initial antibiotics of choice. Ceftriaxone and clindamycin combination is an appropriate alternative.
- Surgical intervention is often indicated.

Management of some of the specific and common abscesses are discussed briefly below.

Ludwig's Angina

Ludwig's angina is characterised as a rapidly progressive gangrenous cellulitis of the soft tissues of the neck and floor of the mouth [2]. The most commonly cultured organisms include *Staphylococcus*, *Streptococcus* and *Bacteroides* species. The majority of cases of Ludwig's angina occur in healthy patients with no co-morbid diseases. The majority of patients report dental pain, or a history of recent dental procedures, and neck swelling.

- Commonly, presents with firm and tender submandibular swelling.
- Occasionally, especially in adults, the presentation would be respiratory distress with dyspnoea, tachypnoea or stridor [3].
- When severe, it often results in protrusion of the tongue and airway obstruction.
- Requires prompt antibiotic therapy, often supplemented with surgical drainage.

Peri-tonsillar Abscess (Quinsy)

Swelling of one of the tonsils, pushing it beyond the midline, associated with altered voice, fever, uvular oedema and a distortion of vowels informally known as 'hot-potato voice' may appear. The above symptoms indicate the formation of the peri-tonsillar abscess. It requires emergency surgical drainage, accompanied by antibiotic therapy.

Retropharyngeal Abscess

- Abscess formation is usually due to suppuration of lymph nodes. Plain X-ray shows widened prevertebral space (Fig. 23.2).

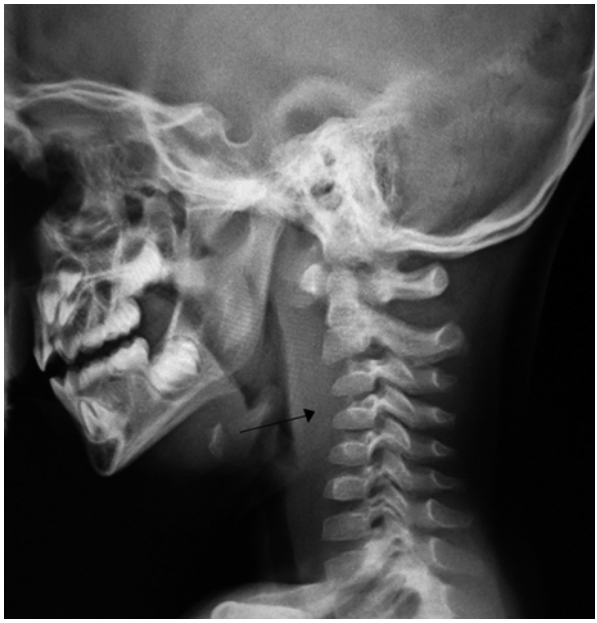


Fig. 23.2 Plain X-ray neck showing widened prevertebral space due to retropharyngeal abscess

- Presents classically in children and will require urgent surgical drainage. Intubation probably may not be possible, and therefore surgical drainage in head-down Trendelenburg position may be utilised.
- Most often it is found to be due to tuberculosis of the spine. Hence, surgical drainage by external approach followed by definitive therapy with antitubercular drugs is the usual course of therapy.

Acute Epiglottitis in Children

- This is an absolute emergency, especially in children. Interestingly, although still debated by some historians, it is believed that George Washington died from a case of acute bacterial epiglottitis [4]. The pathology is due to grossly inflamed and swollen epiglottis, most often due to an infectious aetiology. The most common bacteria identified include *H. influenzae*, beta-haemolytic *Streptococcus*, *Staphylococcus aureus* and *Streptococcus pneumoniae* [5]. Rapid airway obstruction can result from progressive inflammation and oedema of the epiglottis.
- The clinical presentation of epiglottitis in adults is different from that in children. In contrast to children, adults are less likely to present with dyspnoea, drooling, stridor or fever. Adults are more likely to report severe sore throat, odynophagia and hoarseness [6].

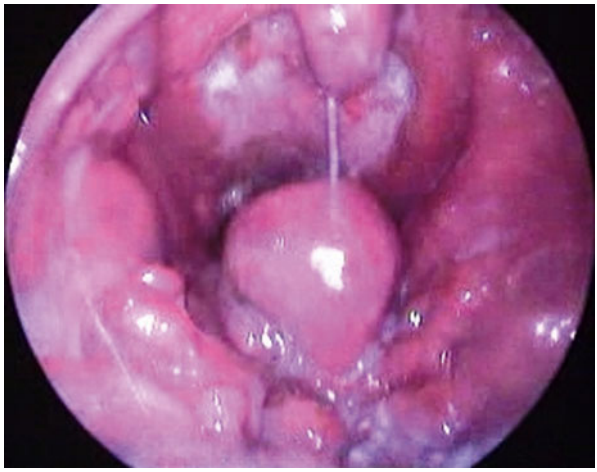


Fig. 23.3 Acute Epiglottitis as seen using 45° nasal endoscope

- Radiographic evaluation for suspected epiglottitis is being replaced by direct visualization of the epiglottis using nasopharyngoscopy/laryngoscopy as the preferred method of diagnosis. Direct visualisation is accomplished through laryngoscopy (Fig. 23.3).
- The patient presents with sudden onset of throat pain, associated with difficulty in swallowing. Plain X-ray demonstrates enlarged epiglottis on the lateral view, classically referred to as the 'thumb sign' (Fig. 23.4).
- In advanced stage, patient can breathe only in the 'tripod position'.
- Incidence of acute epiglottitis has become less common in the paediatric age group due to the universal *H. influenzae* vaccine. Hence, it is now more commonly seen in the adolescent age group.

Angioedema

Angioedema is characterised by the abrupt onset of oedema involving the lips, periorbital area, extremities, abdominal viscera and genitalia [7]. Patients with angioedema who complain of dyspnoea, hoarseness, voice changes or odynophagia or have stridor on physical exam are likely to have laryngeal involvement. This acute pathology, when it involves the larynx, can result in rapid airway obstruction.

Pathology Angioedema is caused often by mast cell-mediated mechanisms. There is degranulation of mast cells and the release of vasoactive substances, which results in altered bradykinin pathway, i.e. angiotensin-converting enzyme (ACE) inhibitor-induced angioedema and abnormalities in the complement system – hereditary angioedema [8].

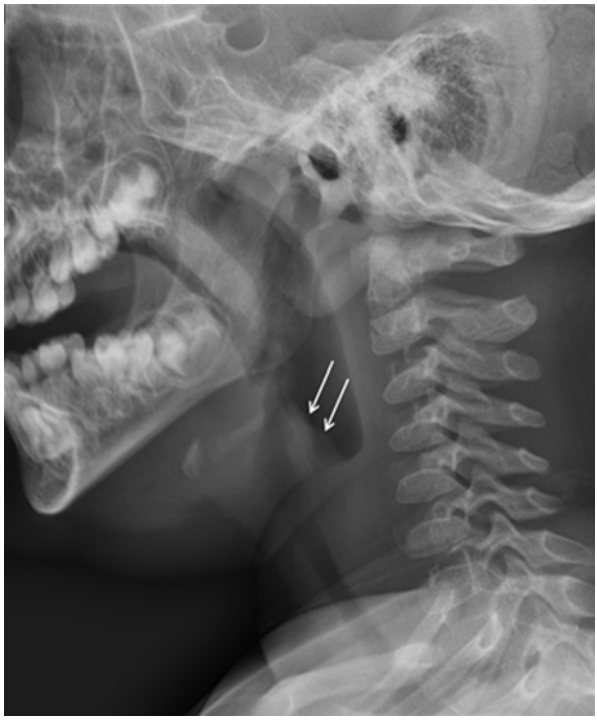


Fig. 23.4 'Thumb sign' of acute epiglottitis – lateral view plain X-ray neck

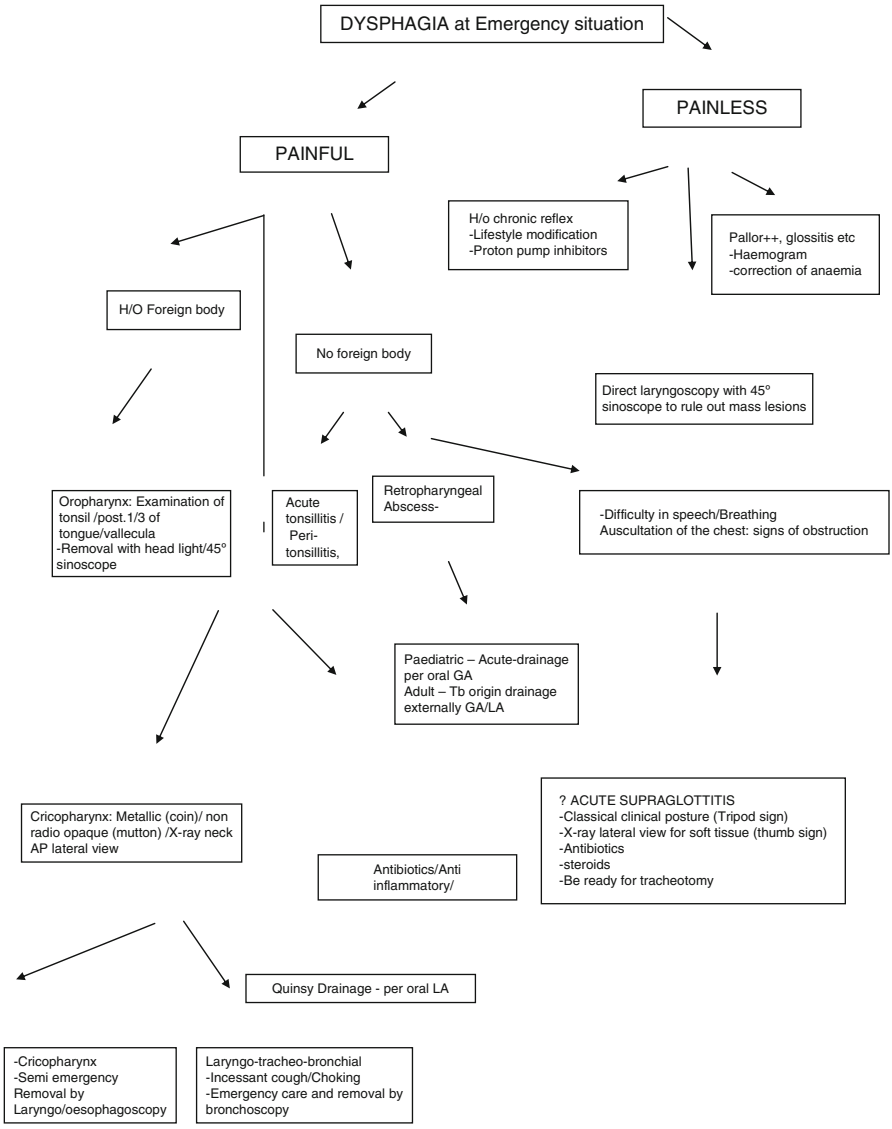
The most common causes of acute angioedema include medications, foods, infections, insect venom, contact allergens (latex) and radiocontrast material [9]. Of these, medications, foods and viral infections account for the majority of cases.

'Routine' labs (complete blood count, basic metabolic panel, liver function tests, erythrocyte sedimentation rate and urinalysis) are usually non-contributory in the evaluation of acute angioedema [10].

For patients with the combination of angioedema and urticaria, treatment may consist of epinephrine, antihistamines and corticosteroids. Patients presenting in severe respiratory distress or with marked laryngeal oedema should be given noradrenaline subcutaneously at a dose of 0.3 mg (0.3 mL of 1:1000 solution). For the majority of these patients, H1 antihistamines are the cornerstone of therapy. Corticosteroids are indicated for patients with anaphylaxis, laryngeal oedema and severe symptoms unresponsive to antihistamines [11].

Medications that have been shown to precipitate symptoms are aspirin, nonsteroidal anti-inflammatory drugs, opiates and oestrogen-containing compounds in any patient with angioedema. The majority of patients have isolated angioedema secondary to an ACE inhibitor [12]. Treatment focuses on discontinuation of the drug, airway management and supportive care. Often isolated severe laryngeal oedema is accompanied by tongue oedema and oedema of the floor of the mouth, which require intubation of the patient.

Acute Dysphagia



Plummer-Vinson Syndrome Also known as Paterson-Brown-Kelly syndrome, a common cause of dysphagia due to iron and nutritional deficiencies, genetic predisposition and autoimmune factors. This can become a cause of acute dysphagia due to impaction of either attempt to swallow a large-size pill or food items like pea. The

removal may require general anaesthesia, but patient's anaemia could pose a problem. Parenteral iron therapy to improve anaemia and dilatation of cricopharynx would be the treatment. Cricopharyngeal web formation would require dilatation using bougies.

Sudden Loss of Voice

History of sudden loss of voice can be classified according to the type of voice presented.

1. No voice produced
   (a) *Organic cause*:
   There can be absolute aphasia due to central causes such as cerebrovascular accident (CVA) involving the speech area. Absolute aphasia, which is a part of CVA, will recover either partially or fully, presents with other features of CVA and requires comprehensive management. Patients with history of hypertension, cardiovascular disease, etc., when present with aphasia should be considered as CVA, until proven otherwise.
   (b) *Functional cause*:

Occasionally patients present with history of inability to speak suddenly. It is possible, that is, of functional cause, if the following characteristics are observed:

- Have no co-morbidity, often adolescent or young adult.
- When asked to speak, the patient makes no sincere effort to speak,
- Tries to use lips only to whisper.
- Detailed history often reveals psychological conflict – marital disharmony, stress relating to schooling, etc.
- A simple test to check the voice-producing mechanism is to ask the patient to cough, in case of functional cause; often the patient is able to produce a powerful cough.
- Reassurance and psychiatric consultation is the management.

Abnormal Voice

(a) *Hoarseness of voice*: Acute viral laryngeal infection is the common cause for altered voice. Other clinical features associated are pain upon speaking, pain on swallowing, cough, fever, etc. History of voice abuse is often present.

Management: Absolute voice rest. Treatment for bacterial upper respiratory infection, including antibiotics, antihistaminic with decongestants and mucolytics and anti-inflammatory drugs, may be required. Recurrent episodes of

laryngitis are often gastro-oesophageal reflux disease (GERD), which causes reflux laryngitis.

(b) Hoarseness of voice can be an isolated phenomenon without respiratory or inflammatory symptoms, when unilateral or bilateral vocal cord palsy is to be suspected. Bilateral abductor palsy can be life-threatening and will present with stridor. The cause can be idiopathic or viral.

(c) *Spastic dysphonia*: This is another condition with functional overlay, with no hoarseness, but patient strains to produce voice.

(d) *Muffled voice* (hot-potato voice): Supraglottic pathology such as the posterior third of the tongue mass, acute epiglottis or laryngeal neoplasm is to be ruled out.

The general principle in managing voice problem is, if vitals are stable, to seek consultation with the laryngologist, preceded by advice of absolute voice rest and a course of symptomatic management.

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Chapter 24
Anorectal Disorders

Rodrick Babakhanlou

Key Points
- Understanding the anorectal anatomy is crucial in diagnosing and treating anorectal conditions.
- Common clinical presentations of anorectal disorders include pain, bleeding, discharge, pruritus and trauma to the anorectum [1].

Introduction

Anorectal disorders are common presentations to the emergency department. Pathologies can vary and include pain, bleeding, discharge, pruritus and swelling and masses.

These can be associated with various underlying gastrointestinal (inflammatory bowel diseases, malignancy) and systemic disorders (immunodeficiency, malignancy, diabetes), which can influence the complexity of the clinical presentation.

Hence, the emergency physician should have a good understanding about the anorectal anatomy and keep a wide range of differential diagnosis in mind when assessing a patient with anorectal disorders.

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Anatomy

As the anatomy of the anus and rectum is intrinsically related to its physiology and pathophysiology, its understanding is crucial in assessing pathologies of this anatomical region.

The anal canal has a length of 4 cm and is divided into an upper and lower part by the dentate (or pectinate) line [2, 3].

The dentate line is the fusion site between the embryonic entoderm and ectoderm and is the junction between two origins of arterial and nerve supply, venous and lymphatic drainage and epithelial lining [2, 3, 7].

It corresponds to a line of anal valves. Above each valve, there is a sinus, which is connected to 3–12 glands via a duct. Blockage of these ducts can result in formation of abscesses and concomitant sepsis [2, 3].

The intestine above the dentate line is innervated by the sympathetic and parasympathetic nervous system and is insensitive to pain.

Therefore, lesions (e.g. malignancies) in this region can remain asymptomatic at early stages.

The upper part of the anal canal shows a similar epithelial lining to the rectal mucosa, consisting of columnar epithelium with mucous-secreting and goblet cells draining into the anal crypts at the dentate line [2, 3].

Malignancies arising in this region are mainly adenocarcinomas.

The venous drainage above the dentate line occurs via the superior haemorrhoidal plexus and drains into the portal vein.

Internal haemorrhoids originate from this plexus.

The lower part of the anal canal is innervated by the somatic nervous system and is highly sensitive to pain, which explains why lesions in this area, such as abscesses, fissures or malignancies, are extremely painful.

The mucosa is lined with non-keratinized, stratified epithelium. Malignancies arising in this region are mainly squamous cell carcinomas.

The venous drainage occurs via the middle and inferior haemorrhoidal plexus into the internal iliac veins. External haemorrhoids originate from this plexus.

Haemorrhoids

Haemorrhoids are a cushion of vascular tissue, rich in blood vessels and muscle fibres, located within the anal canal at 3 o'clock (left lateral), 7 o'clock (right anterior) and 11 o'clock (right posterior) in lithotomy position [3, 7].

The vascular tissue consists of arteriovenous communications between branches of the superior rectal artery and the superior, middle and inferior rectal veins [6]. The muscle fibres contribute to the bulk of the cushions, which on the other hand contributes to the physiologic continence of the anal canal [1, 4, 6–8].

Table 24.1 Classification of Internal Haemorrhoids

Grades	Clinical signs
Grade I	Prominent haemorrhoidal vessels, no prolapse
Grade II	Protrusion at the time of bowel movement and spontaneous reduction after the bowel movement
Grade III	Protrusion during a bowel movement or spontaneously. Manual reduction required
Grade IV	Permanently prolapsed and irreducible haemorrhoids

Haemorrhoids located superior to the dentate line are defined as internal haemorrhoids. Covered by columnar epithelium, they lack sensory innervation [3, 7].

Internal haemorrhoids are further classified into four grades according to the degree of the prolapse [4] (Table 24.1).

Haemorrhoids located below the dentate line are defined as external haemorrhoids and are covered with stratified squamous epithelium with sensory innervation [1, 4, 7, 8].

The true incidence of haemorrhoids is not known [4, 5]. However, the prevalence of haemorrhoids in the US population has been reported to be 4.4 % in the literature with a peak between 45 and 65 years of age [4, 6].

Aetiology

The exact cause and pathogenesis of haemorrhoids remains unknown. However, several aetiologic factors have been proposed: [6, 7]

- Constipation
- Prolonged straining
- Diarrhoea
- Diet
- Occupation
- Pregnancy
- Increased intraabdominal pressure (pelvic tumours, ascites)

Clinical Features

Internal Haemorrhoids:

40% of patients with haemorrhoids remain asymptomatic [9].
Complaints arise due to enlarged and prolapsed haemorrhoids and include:

- Soiling.
- Pain.

- Bleeding: Bleeding is painless and occurs mainly during bowel motion.
- Secondary itching.

Prolapsed haemorrhoids that remain unreduced can develop ischaemia and gangrene [4–7] (Figs. 24.1 and 24.2).

External Haemorrhoids

External haemorrhoids become symptomatic only when thrombosed (Fig. 24.3).

Clinical features of thrombosed haemorrhoids are acute pain over 48–72 h, which subside after 1 week [6, 8, 13].

Diagnosis

Detailed history taking and an adequate physical examination, including inspection, digital rectal examination and anoscopy, are important steps in making the diagnosis.

The patient should be placed on his/her left lateral position with the knees and hips flexed [5, 13, 15].

Examination starts with the inspection of the anus and the surrounding skin.

The examiner should pay attention to the presence of [5, 13, 15]:

- Ulcers
- Fissures
- Excoriations
- Prolapsed haemorrhoids
- External haemorrhoids

Digital examination starts with gentle insertion of the lubricated finger into the anal canal and the rectum. A circumferential rotation of the finger should feel for masses, ulcers or painful lesions.

By asking the patient to squeeze the finger, the examiner can assess the tone of the sphincter.

After a digital examination, anoscopy should be performed to visualize the anal canal. By inserting a well-lubricated anoscope with good light conditions, a 360° rotation will provide a circumferential view of the anal canal.

By asking the patient to strain, non-prolapsing haemorrhoids can be visualized as they bulge into the lumen of the anoscope.

Differential Diagnosis [4–7]

There is a wide range of differential diagnosis. Good history taking, including the past medical history, and a thorough physical examination are the cornerstones for establishing the correct diagnosis and organize further clinical testing.

- Colorectal cancer
  Patients with anaemia, positive faecal occult blood test and a positive family history of colorectal cancer need further evaluation with colonoscopy [8].
- Anal cancer
  Any patient with complaints of pain, bleeding and a lump needs a thorough evaluation and visualization of the anal canal by anoscopy.
  Suspicious lesions detected on examination warrant biopsy for further evaluation (Fig. 24.7).
- Anal fissure
  Acute fissures are characterized by pain, exacerbated during and after defecation. Visual inspection and anoscopy may show a tear in the posterior midline of the anal canal [10, 11] (Fig. 24.4).
- Pruritus ani
- Inflammatory bowel disease and perianal Crohn's disease
  A past medical history of inflammatory bowel diseases and the presence of proctitis, discharge and systemic signs, including fever, weight loss and abdominal pain, should prompt the physician to undertake further tests, including imaging, colonoscopy and biopsies.
- Sexually transmitted diseases and condylomata acuminata
  Evaluation of the sexual history, including previous sexually transmitted diseases of the patient and his/her partner, present sores, discharge or itching, should prompt the physician to undertake further tests.

Management

Haemorrhoids can be managed operatively and non-operatively depending on their stage.

Internal Haemorrhoids

Nonoperative management

- High-fibre diet
- Adequate fluid intake
- Sitz baths
- Analgesia

This conservative approach is the mainstay of the nonoperative management of haemorrhoids grades I–III. Patients with haemorrhoids grades I–III can be discharged from the emergency department and referred to the surgeon for further evaluation and elective management [8].

Operative management

Large and prolapsed grade III haemorrhoids that do not respond to conservative treatment and grade IV haemorrhoids with a significant prolapse and incarceration require surgical intervention [8]. Hence, surgical consult should be obtained in the ED.

External Haemorrhoids

Treatment options for thrombosed haemorrhoids include either excision or conservative management.

- Surgical excision is recommended in the first 48–72 h, which results in immediate pain relief [4, 8].
- Incision and removal of the clot only are not recommended because of the high recurrence rate [4].
- Thrombosed haemorrhoids that have been present for longer than 72 h can be treated conservatively if the pain is bearable [4, 8].

Protruded internal haemorrhoids without spontaneous reduction
Manual reposition required
Permanently prolapsed and non-reducible haemorrhoids
Thrombosis of the external haemorrhoids

Anal Fissure

An anal fissure is a longitudinal cut in the squamous epithelium of the anal canal ranging from the anal verge to the dentate line [10–12] (Fig. 24.4).

- Anal fissures affect infants and adults between 30 and 50 years of age.
- Both sexes are affected equally with a reported incidence of 11 % [12–15].

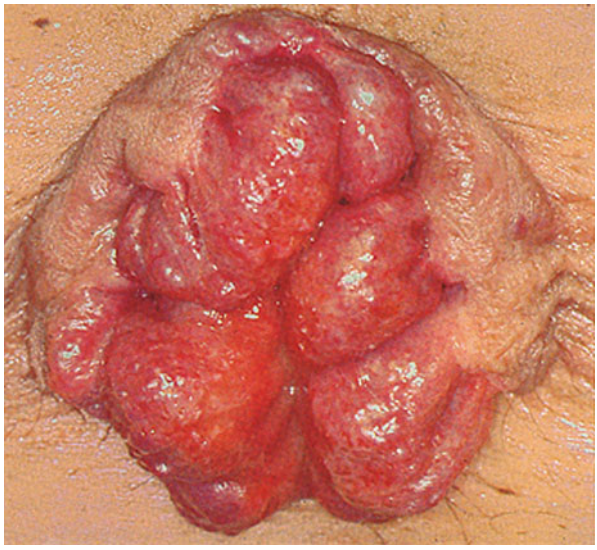

Fig. 24.1 Prolapsed haemorrhoids grade III

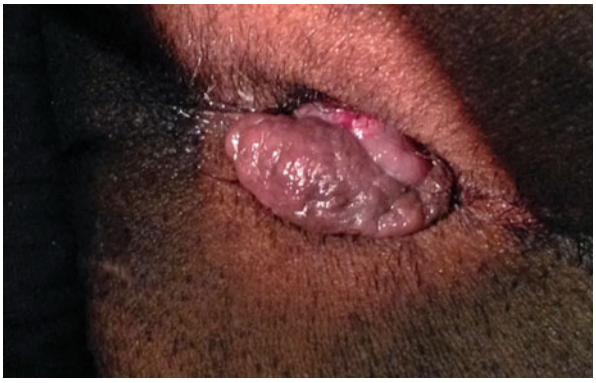

Fig. 24.2 Prolapsed haemorrhoids grade IV

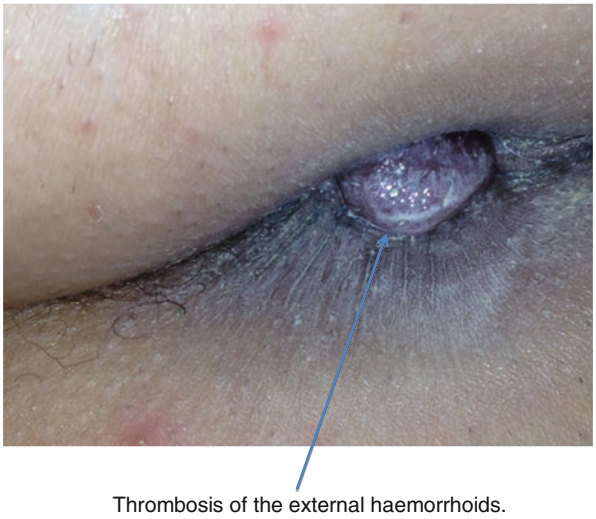

Fig. 24.3 Thrombosed haemorrhoid

- Anal fissures are most commonly located in the midline, 80 % posteriorly and 10–15 % anteriorly. Anterior fissures are more common in female patients [13].
- The presence of anal fissures in locations other than the midline should raise the suspicion of the presence of various underlying pathologies such as inflammatory bowel diseases, STDs or malignancies [10–15].

Aetiology

The exact aetiology of anal fissures is not known.

The following mechanisms play a role in the development of fissures:

- Repetitive trauma to the anal canal resulting in damage to the mucosa secondary due to constipation, hard stools and straining.
- Reduced blood flow and local ischaemia in the affected area resulting in impaired wound healing after traumatic events [13–16].
- Spasm of the internal sphincter secondary to pain has been reported to increase the resting pressure in the anal canal and contributes to further reduction of the blood flow and impaired and prolonged wound healing [15, 16].

Atypical fissures located elsewhere than in the midline can be caused by systemic pathologies, such as inflammatory bowel disease, sexually transmitted disease (HIV, syphilis) and leukaemia or after surgeries in the anal region [14–16].

Clinical Features

Clinical features of anal fissures include:

- Severe pain during defecation lasting up to minutes to hours after bowel movement.
- The pain is described as sharp and tearing during defecation followed by a dull ache with throbbing quality afterwards.
- Rectal bleeding can be present either on the stool or the toilet paper. Further clinical features include pruritus ani and constipation secondary to the fear related to pain [11–16].

Diagnosis

The diagnosis is a clinical one and can be suspected based on the history of presentation. A clinical examination, including a digital rectal examination, may not always be possible due to severe pain. However, inspection may show a tear in the mucosa located in the posterior midline (Fig. 24.4).

A complete anorectal examination, including anoscopy, should be performed under general anaesthesia in order to determine the underlying condition [11–16].

In presence of suspected lesion, biopsies and culture may be indicated.

Differential Diagnosis

- STDs (HIV, syphilis, genital herpes)
  Evaluation of the sexual history, including previous sexually transmitted diseases of the patient and his/her partner, present sores, discharge or itching, should prompt the physician to undertake further tests.
- IBD (Crohn's disease, ulcerative colitis)
  A past medical history of inflammatory bowel diseases, the presence of fissures in locations other than the midline, and systemic signs, including fever, weight loss and abdominal pain, should prompt the physician to undertake further tests, including imaging, colonoscopy and biopsies.
- Anal cancer
  Any patient with a non-healing fissure and complaints of pain, bleeding and a lump needs a thorough evaluation and visualization of the anal canal by anoscopy.
  Suspicious lesions detected on examination warrant biopsies for further evaluation (Fig. 24.7).
- Proctalgia fugax
  Proctalgia fugax is a transient pain in the rectum lasting 1–2 min due to spasm of the rectum itself without visible pathologies.
- Fistula in ano
  Compared to anal fissures, fistulae are painless and are associated with a past medical history of previous anal abscesses and fistulations.
- Intersphincteric perianal abscess
  Increased inflammatory parameters (WCC, CRP) are suggestive of an infective process, whereas in a fissure these parameters remain within normal limits.

Management

Management of anal fissures include both operative and nonoperative measures.

Most anal fissures respond to a nonoperative management, which includes both preventive measures and also medical treatment.

1. Preventive measures
   - Increased fluid intake
   - Laxatives
   - High-fibre diet
   - Sitz baths

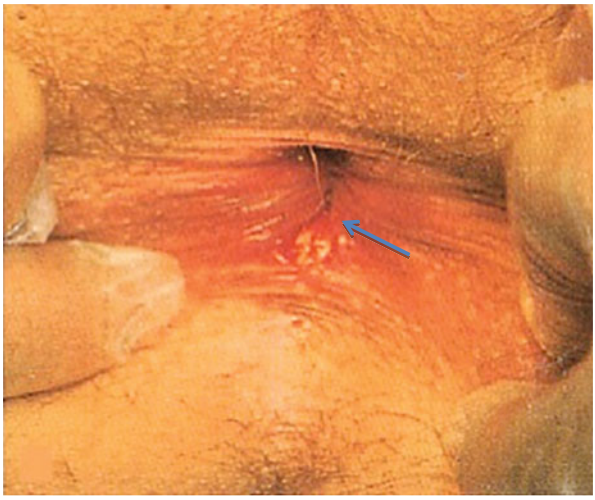

Fig. 24.4 Anal fissure

Preventive measures after every bowel motion have been shown to reduce the recurrence rate from 68 % to 16 % and are considered as the initial step of management [12, 16].

If healing fails despite these measures, the initiation of medical treatment is the second step.

2. Medical treatment [12, 16]

- Topical glyceryl trinitrate (GTN) 0.2–0.4 % three times daily for 4–6 weeks. Side effects including headaches, hypotension and nausea make this drug less favourable.
- Topical calcium channel blockers (diltiazem cream, nifedipine cream) have similar effects to GTN, but fewer side effects.
- Injection of Botulinum toxin
  Botulinum toxin is reported to reduce the release of acetylcholine and, thus, relax the sphincter muscle [17]. Injection of 20 units laterally to the fissure shows a good healing tendency, but causes incontinence as a side effect lasting for several weeks [10, 16].

Failed medical management warrants surgical intervention.

Pilonidal Disease

Pilonidal disease is a subcutaneous infection located at the upper half of the gluteal cleft.

Its incidence is estimated to be 26 per 100,000 persons and it typically presents in the second decade of life [18].

Men are more commonly affected than women with a ratio of 3:1 [18].

Aetiology

Pilonidal disease is an acquired condition. The exact pathogenesis, however, remains unclear. Pilonidal means "nest of hair," and it is believed that damaged hair, friction and negative pressure in the natal cleft result in foreign body reactions presenting in form of cavities, cysts or abscesses [18].

Risk factors include [19]:

- Obesity
- Prolonged sitting
- Family history
- Deep natal cleft
- Local trauma

Clinical Features

The clinical picture varies, ranging from [18, 19]:

- The presence of asymptomatic pilonidal cavities or sinus tracts
- Acute painful abscesses
- Pain in the intergluteal region
- Discharge of blood or pus

Diagnosis

The diagnosis is a clinical one and is based on the clinical findings, which includes the presence of pores, painless sinuses or even an acute abscess (Figs. 24.5 and 24.6).

Imaging and laboratory tests are not diagnostic.

Differential Diagnosis

Differentiating a pilonidal abscess from other inflammatory conditions can be challenging, as many cutaneous conditions are difficult to be distinguished and may even coexist.

- Perianal abscess and fistulae
  Perianal abscesses often present as painful conditions and are generally located near the anus or the anal canal.
  Fistulae are chronic manifestations of an acute inflammatory process, the abscess.
  The diagnosis of fistula

- Skin abscesses
  These lesions are located in the dermis and deeper tissues and mainly involve the buttocks. Sinus tracts are not present.
- Folliculitis
  Folliculitis is a superficial infection of hair follicles. The clinical pictures are characterized by multiple pustulous lesions located in the epidermis.
- Perianal manifestation of Crohn's disease
  Perianal manifestations of Crohn's disease can present with various pathologies, including fissures, abscesses and fistulas.
  Clinically, patients can present with pain, purulent discharge and fever.
  A past medical history of Crohn's disease makes the diagnosis more likely.
- Hidradenitis suppurativa
  This condition is a chronic follicular disease involving the intertriginous area of the axillae, the groin and inframammary and perianal regions.
  The presence of this condition in such locations makes the diagnosis more likely.

Management

- Presentations to the emergency department are related to painful acute abscesses.
- The main goal is incision and drainage of the abscess in order to provide immediate relief, although incision and drainage alone are associated with a high recurrence rate.
- Definite treatment includes surgical excision of the pilonidal sinus tracts either with primary wound closures or delayed closures [18, 19].
- Antibiotics are not indicated in the management of pilonidal disease, unless there is an associated cellulitis [18].

Perianal Abscesses

Anal abscesses and fistulae are common anorectal conditions, which in most of the cases result from a cryptoglandular infection in the anal canal [21].

Anal glands drain into the anal crypts at the dentate line via ducts. Blockage of these ducts results in retention of contents and inflammation of the glands and subsequently in abscess formation [1].

Anal abscesses represent the acute phase of manifestation of disease, whereas fistula formation is the sequelae of untreated abscesses and representative of the chronic process.

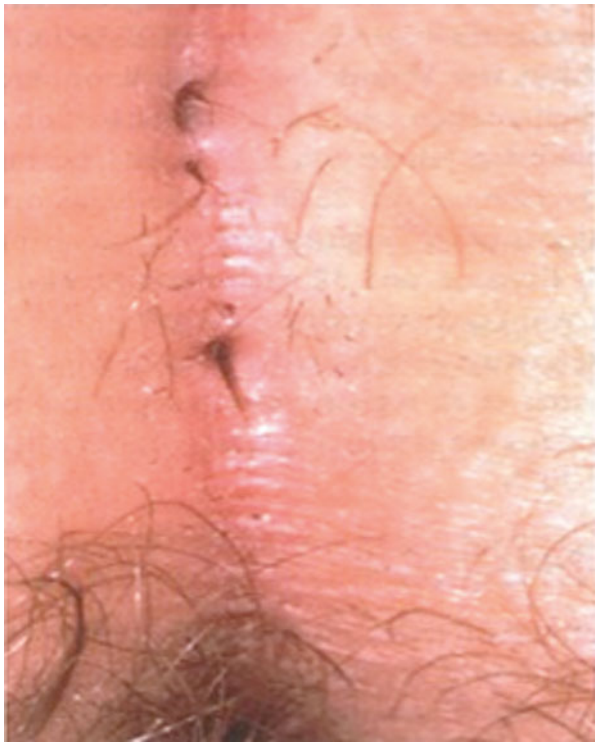

Fig. 24.5 Sinus tracts in pilonidal disease

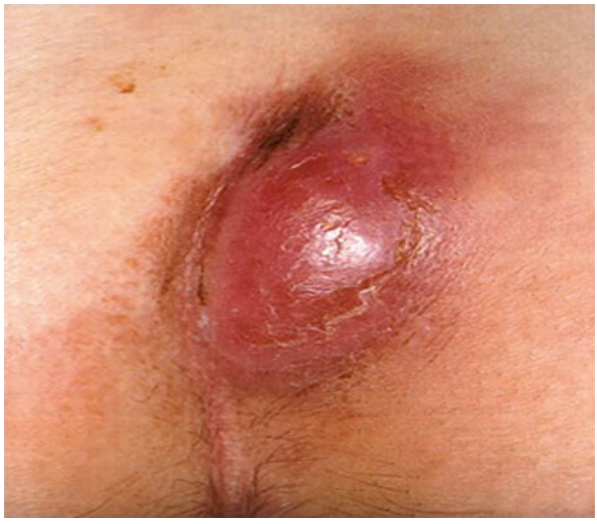

Fig. 24.6 Pilonidal abscess

The exact incidence of anal abscesses is not known. It is estimated that there are about 100,000 cases each year [22].

The disease affects mainly adults between 30 and 50 years of age and with a ratio of 3:1, men are more commonly affected than women [23].

Aetiology

Ninety percent of abscesses result from blockage of the ductus as a cryptoglandular infection.

The remaining 10 % can be caused by certain specific factors, such as [14]:

- Inflammatory bowel disease (specifically Crohn's disease)
- STDs
- Trauma
- Post-radiation
- Postoperatively
- Malignancy
- Immunosuppression

Pathogenesis and Classification

Based on their location abscesses are classified as [20]:

1. Intersphincteric abscesses:
   - Abscesses occur initially in the intersphincteric space [23].
   - If these remain in the intersphincteric space and are called *intersphincteric abscesses*.
   - A circumferential extension of an intersphincteric abscess results in the formation of a *horseshoe abscess* [14, 20, 22, 23].
2. Perianal abscesses:
   - Perianal abscess are the most common type of anal abscesses.
   - Their formation results after a downward spread of an intersphincteric abscess results.
3. Ischioanal abscesses
   - When pus penetrates the external sphincter muscle and enters the ischiorectal space, it is termed *ischioanal abscess*.
4. Supralevator abscesses
   - These types of abscesses are the least common type of abscess.
   - Their formation occurs after an upward spread of an *intersphincteric abscess* [14, 20–23].

Clinical Features [14, 20–23]

- Pain is the most common presenting feature of an abscess and can worsen during defecation or urination and in a seated position.
- Urinary retention and back pain can be present and should raise the suspicion of a supralevator abscesses.
- External cutaneous signs such as erythema or indurated masses may not always be present.
- Rectal discharge is only noted once an abscess bursts into the anal canal.

Diagnosis

Abscesses are diagnosed clinically. The presence of a swelling in the anorectal area associated with pain, discharge and fever should raise the suspicion of an anorectal abscess.

1. History
   Past medical history should include:
   - Previous abscesses
   - Anorectal surgeries
   - Previous radiation
   - Inflammatory bowel diseases
2. Examination
   - Digital rectal examination may reveal a fluctuant induration or mass, but may not be possible due to severe pain.
   - Internal examination and anoscopy under general anaesthesia may be indicated if severe pain present.
3. Imaging
   - CT
   - MRI

Imaging modalities can be helpful in evaluating the exact location and the extent of complicated abscesses [14, 20–24].

The presence of supralevator abscesses should prompt the clinician to look for intraabdominal sources, such as diverticulitis, appendicitis or pelvic inflammatory diseases [24].

Management

- Anorectal abscesses require surgical incision and drainage under general anaesthesia.
- Local anaesthetics do not achieve sufficient anaesthesia of the abscess cavity due to the acidic medium of the pus.

- Incision and drainage in the emergency department should only be reserved for small and superficial abscesses in order to provide temporary relief until timely surgical treatment can take place [23].
- Antibiotics are not routinely indicated, except in cases with surrounding cellulitis, immunosuppression and diabetes [21, 23].

Pruritus Ani

Pruritus ani is defined as a condition characterized by chronic itching in the perianal region. Its incidence ranges from 1 % to 5 % in the population and men are more affected than women with a ratio of 4:1 [25]. Patients are usually between 30 and 70 years of age [26].

Pruritus ani is classified as either primary (idiopathic) or secondary.

Idiopathic pruritus ani is responsible for 25 % of cases, whereas 75 % of patients have coexisting pathologies [25, 26].

Aetiology

Causes of anal pruritus are outlined in Table 24.2.

Clinical Features

The clinical picture can range from mild to moderate and even severe itching and burning of the perianal region [27].

As a result, vigorous washing and cleaning, scratching or application of topical substances occurs, which exacerbate the symptoms [27].

Inflammation and oedema due to destruction of the epithelium lead to increased tenderness and burning with a worsening need of scratching [27].

Diagnosis

1. History
   - Coexisting skin conditions (urticaria, atopy)
   - Allergies
   - Use of regular medication and topical substances
   - Hygiene
   - Diet
   - Use of different cloths or detergents

Table 24.2 Aetiology of anal pruritus

Infections	Bacterial
	Fungal (candida)
	Viral (STDs)
	Parasitic (pinworm)
Anorectal causes	Haemorrhoids
	Anal fissures
	Fistulas
	Malignancies (Fig. 24.7)
	Prolapse
Dermatologic causes	Psoriasis
	Atopic dermatitis
	Lichen sclerosus
	Lichen planus
	Paget's disease
Dietary factors	Coffee
	Citrus fruits
	Chocolate
	Beer
	Tomatoes
	Chilli peppers
Systemic causes	Diabetes mellitus
	Hepatic diseases (hyperbilirubinaemia)
	Leukaemia
	Aplastic anaemia
Local irritants	Poor hygiene
	Faecal soilage
	Soaps
	Deodorants
	Tight clothing
	Toilet paper

2. Examination
   - Inspection of the skin
   - Digital rectal examination
3. Investigation
   - Microbiological investigations in suspected bacterial and fungal infections (a swab should be performed prior an internal examination)
   - Biopsy of suspected lesions
   - Colonoscopy in presence of occult or visible blood

The diagnosis of primary pruritus ani should be made once potential causes of secondary pruritus ani have been excluded [27].

Management

Treatment is aimed to treat the underlying cause, especially in patients with secondary pruritus ani.

In patients with primary pruritus without an underlying cause, reassurance of the patient plays an important role.

Patients should be educated about the following treatment steps: [28]

1. Anal hygiene
2. Avoiding irritants (soap, cream, lotion)
3. Avoiding further trauma to the skin by scratching, rubbing or vigorous washing
4. Keeping the area dry and avoiding synthetic underwear
5. Maintaining regular bowel movements

The majority of patients show a good response to these conservative measures [15]. However, if these steps fail to improve the condition, a specialist referral should be considered for elective medical treatment.

Medical therapy includes application of topical steroids or topical capsaicin [16, 25, 28].

Hydrocortisone cream (1 %) can be applied two to three times a day for a maximum of 2 weeks. A longer use is not recommended due to the danger of perianal skin thinning [16, 28].

The mechanism of topical capsaicin is not completely understood. It is assumed that Capsaicin inhibits the synthesis and release of substance P, which is responsible for itching and burning pain [28].

The recommended dose of 0.006 % can be applied for a period of 4 weeks [16, 25, 28].

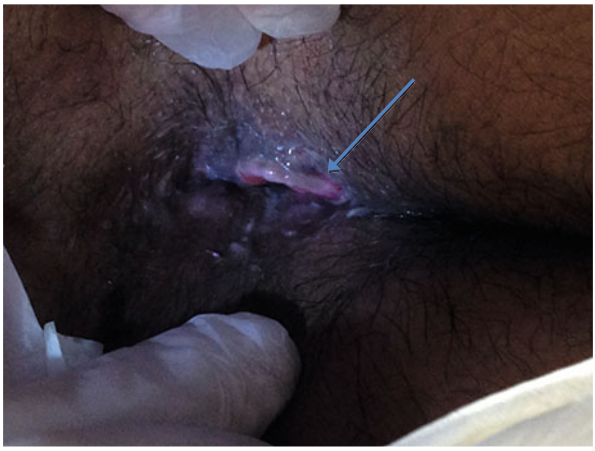

Fig. 24.7 Anal cancer

Foreign Bodies

Anorectal foreign bodies are not uncommon presentations to the emergency department. A wide range of retained anorectal foreign bodies have been reported in the literature, including sexual devices, vegetables and fruits, body sprays or deodorants [29–33] (Fig. 24.8).

Exact epidemiological data is not available. However, it has been reported in the literature that there is a male preponderance with a male to female ratio of 28:1 [29–33].

Most of the patients are between 20 and 40 years of age [29].

Aetiology

Foreign bodies can be sharp or blunt and are inserted either intentionally or non-intentionally [29–35].

Intentional insertion can occur either due to sexual practices by the patient or the partner, in psychiatric patients with mental disorders or in drug smugglers (body packers) [29–35].

Non-intentional insertion of foreign bodies can result from sexual assaults, diagnostic procedures or when a foreign body is swallowed and ends up in the rectum [29–35].

Clinical Features

In most cases, presentation to the emergency department is delayed due to embarrassment and prolonged unsuccessful attempts of removal of the foreign body.

Patients can present with [29–35]:

- Anorectal pain
- Rectal bleeding and discharge
- Pelvic pain
- Abdominal pain
- Constipation

In cases of perforation, the clinical picture can resemble that of sepsis, including hypotension, tachycardia, tachypnoea and fever. The clinical examination may reveal rectal discharge and the picture of an acute abdomen [29–33].

Diagnosis

1. History
   History taking can be difficult. Patients may not give a clear history due to embarrassment and may invent various explanations for the accident.
   History taking should include the time of insertion, the characteristic of the object (sharp vs. blunt, size) and the number of attempts to remove the FB.
2. Examination
   - Before undertaking a digital rectal examination, it is important for the examiner to exclude a sharp object in order to avoid any injuries. The digital rectal examination should assess the location of the foreign body, its size and shape and the tone of the anal sphincter [29–33].
   - An inspection should assess lacerations of the mucosa, bruising and discharge [29].
   - An abdominal examination is important in order to look for guarding and signs of perforation [29].
3. Imaging
   - Plain X-rays of the abdomen and pelvis conceal the presence of foreign bodies, their shape, size and location and also the presence of a bowel obstruction.
   - An erect chest X-ray should be performed in order to rule out a perforation.
4. Laboratory investigation
   - Routine blood tests including FBC and inflammatory parameters are performed as a routine workup.

Management

Removal of the foreign body can be challenging and depends on various aspects, such as the size, the shape and the location of the foreign body, but also the clinical condition of the patient [34, 35].

The approach includes the transanal route and the surgical route.

The transanal route should be the first choice and is recommended for low-lying foreign bodies located in the distal third of the rectum.

Reducing the spasm of the anal sphincter is recommended in order to facilitate the removal of the foreign body. Procedural sedation, premedication with benzodiazepines or a perianal block with local anaesthetics can be helpful [29, 36].

Anoscopy can help visualizing the object, which can be removed by a Kocher clamp or a forceps [29, 33–35].

If removal via the transanal route is not possible, a surgical approach is inevitable.

Predictors of failure of transanal extraction of foreign bodies have been reported to be [29]:

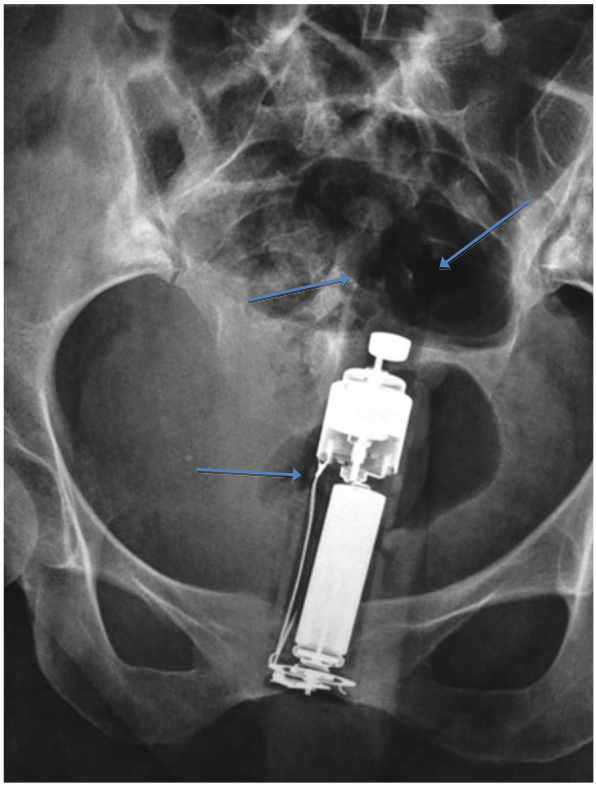

Fig. 24.8 Impacted vibrator

- Objects longer than 10 cm
- Sharp objects
- High-lying objects (proximal two third of the rectum)
- Foreign bodies that have migrated into the sigmoid colon
- Objects that have been retained >2 days

Complications that can occur after transanal removal of foreign bodies include mucosal tears, bleeding or even perforation. Therefore, flexible sigmoidoscopy is recommended for all patients, who undergo a transanal removal of foreign bodies [29–35].

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Chapter 25
Breast Emergencies

Ramya Ramakrishnan

Key Points

- A breast abscess develops as a complication of mastitis in 5–11 % of breastfeeding women. The most common causative organism is *Staphylococcus aureus*.
- Non-lactational breast abscesses form as a complication of periductal mastitis. Tobacco smoking is strongly associated with this. These abscesses are caused by mixed flora and include anaerobes.
- Appropriate antibiotics and ultrasound-guided percutaneous drainage is the treatment of choice for breast abscesses.
- Blunt trauma of the breast can cause either a crush or an avulsion injury.
- Penetrating trauma to the breast is rare. Iatrogenic injury as a result of vacuum-assisted breast biopsy is increasing.

Introduction

- Diseases or conditions of the breast that present as emergencies to the hospital are uncommon. These can be due to primary diseases of the breast or as a result of trauma to the breast. Trauma in turn can be due to blunt forces or rarely due to penetrating injury. Traumatic injury can also occur as a result of invasive procedures on the breast like core needle biopsies. The most common acute disease of the breast that brings a patient to the hospital is a breast abscess. Breast abscesses can be further classified into lactational and non-lactational.

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Lactational Breast Abscess

- Mastitis is a complication often encountered in primiparous women and develops in 1–24 % of breastfeeding women [1].
- It is most common in the first 6 weeks of breastfeeding with the highest incidence occurring during the second and third weeks. It is initially localised to one segment of the breast, but untreated can spread to affect the whole breast (Fig. 25.1). A breast abscess develops as a complication of mastitis in 5–11 % of cases [2].

Risk Factors

- The main risk factor for mastitis is breastfeeding during the early post-partum period. Milk stasis and cracked nipples may contribute to the development of mastitis, although the evidence for this is inconclusive.
- Other implicated factors include previous mastitis, maternal fatigue and primiparity.
- Reported risk factors for breast abscess include a past history of mastitis, maternal age over 30 years and gestational age greater than 41 weeks.

Microbiology

- The most common bacterium is *Staphylococcus aureus* [3]. Strains of methicillin-resistant *S. aureus* (MRSA) have been identified, particularly in hospital-acquired infections. Other organisms include streptococci and *S. epidermidis*.

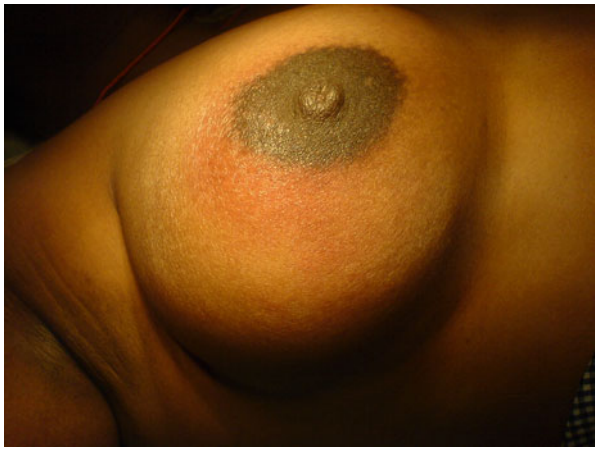


Fig. 25.1 Mastitis

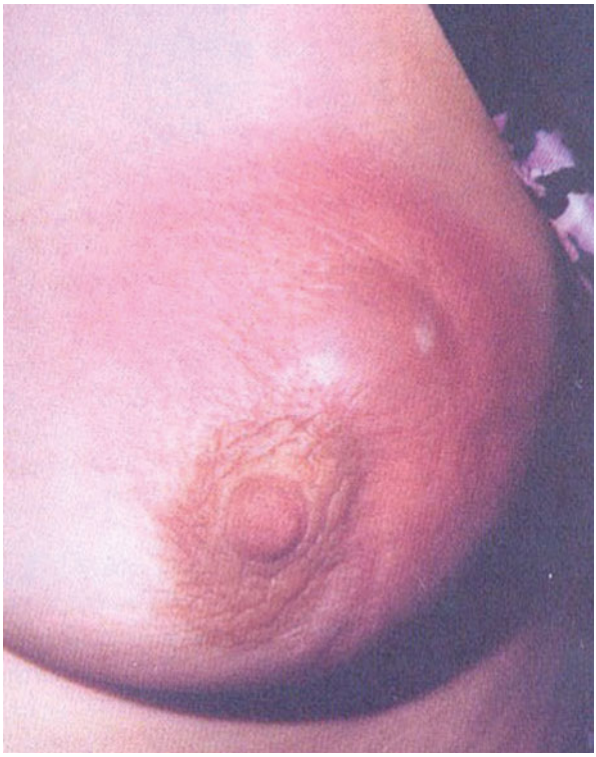


Fig. 25.2 Breast abscess

- Patients who suffer with recurrent breast abscesses have a higher incidence of mixed flora, including anaerobic organisms. On rare occasions, *Candida albicans*, not an uncommon cause of nipple pain in lactating women, can cause parenchymal infection.
- The source of the organism is usually the mouth of the infant, and the bacteria enter the breast through cracks present on the nipple.

Clinical Features

- Breast pain is the primary symptom of mastitis and breast abscess. High fever is common, along with other generalised flu-like symptoms including malaise, lethargy, myalgia, sweating, headache, nausea, vomiting and occasionally rigors.
- On examination, the patient will be febrile, and breast examination will reveal an area of induration or a tender firm mass, which may be fluctuant. There will be redness of the overlying skin. Sometimes, the skin shows patchy necrosis with or without ulceration and pus discharge. The nipple may be inverted or show evidence of cracks on the surface (Fig. 25.2).
- Lactational breast abscesses are more likely to be peripheral and more commonly in the upper outer quadrants. Axillary lymphadenopathy is uncommon.

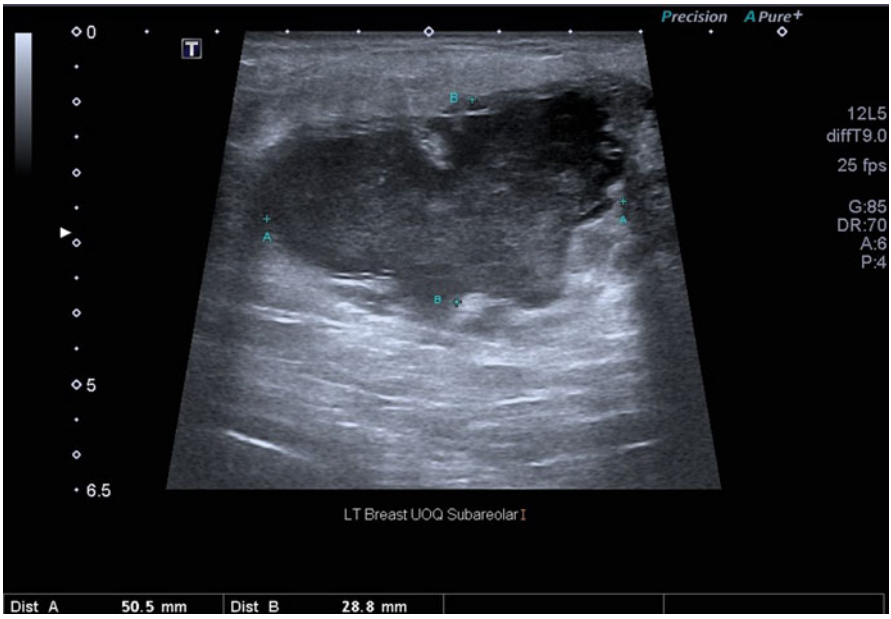


Fig. 25.3 Ultrasound of the breast showing the abscess cavity

Differential Diagnosis

Inflammatory carcinoma can mimic mastitis and breast abscess. This should be thought of when the patient has axillary lymphadenopathy or is not responding to antibiotics and if ultrasound features are different.

Investigations

- Breast abscess remains essentially a clinical diagnosis which can be differentiated from simple mastitis by doing an ultrasound examination (using a high-frequency linear transducer of 7.5 MHZ) of the affected breast.
- A localised collection of pus can be identified as a heterogenous thick-walled mass, predominantly cystic with internal echoes and septations (Fig. 25.3). Ultrasound also allows guided aspiration of any abscess providing drainage and fluid for microscopy and culture.
- A malignant lesion may mimic an inflammatory collection on ultrasound. Hence, following aspiration, if a significant lump remains or if no fluid is obtained or if the fluid is bloodstained rather than purulent, then core biopsy is recommended to exclude breast cancer.

Complications

These include cessation of breastfeeding, bacteremia and sepsis, destruction of breast tissue resulting in disfigurement, recurrent infection and scarring.

Treatment

Antibiotics

- As *S. aureus* is the common causative organism, the antibiotic of choice would be flucloxacillin or dicloxacillin in a dose of 500 mg four times a day for 5 days [4]. For patients allergic to penicillin, options include cephalexin or clindamycin.
- Tetracycline, ciprofloxacin and chloramphenicol should be avoided as they are unsafe for use in lactating women. Paracetamol should be given to manage the fever and pain.

Drainage of the Abscess

- The conventional method of management was surgical incision and drainage under general anaesthesia.
- This has largely been replaced by ultrasound-guided aspiration under local anaesthesia on an outpatient basis [5]. This can be done in the emergency room, radiology department or the breast outpatient clinic (Fig 25.4).
- Incision and drainage, preferably under local anaesthesia, should be considered only if there is overlying skin necrosis and impending rupture of the abscess.

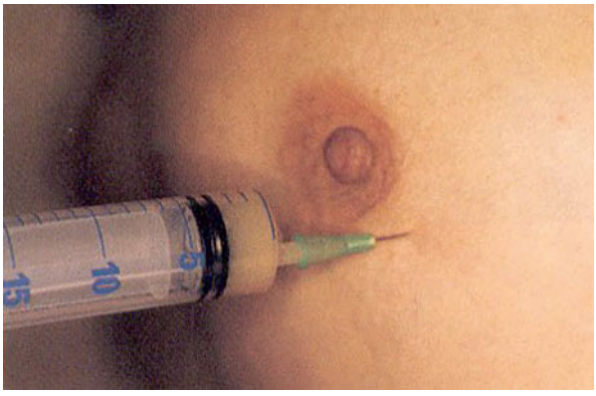


Fig. 25.4 Ultrasound guided aspiration of breast abscess

Other Supportive Treatment

The patient should be encouraged to continue breastfeeding from the affected breast. If she is unable to do so, then she should empty the breast either manually or with the help of a breast pump to avoid worsening of symptoms.

Non-lactational Breast Abscess

Non-lactational breast abscess is being increasingly seen across the globe. This is seen in women in the age group of 30–50 years.

Risk Factors

- There is a strong association of periductal mastitis and cigarette smoking with breast abscess in non-lactating women, and it may predispose to anaerobic infection and the development of mammary fistulae.
- The abscess recurs in women who are overweight, have large breasts or have poor personal hygiene [6].

Microbiology

- *Staphylococcus aureus* remains the predominant microbe isolated from breast abscesses, and there has been a recent increase in community-acquired methicillin-resistant *S. aureus* (MRSA) species. Moazzez et al. showed that 50 % of staphylococcal species and 19 % of all isolates from community-acquired breast abscess were MRSA [7].
- Other commonly isolated organisms include coagulase-negative *S. aureus*, diphtheroids, *Pseudomonas aeruginosa*, *Proteus mirabilis* and anaerobes.

Clinical Features

The clinical features are similar to lactational breast abscess except for the location of the tender mass in the breast. They are more commonly central/subareolar or seen in the lower quadrants. Peripheral non-lactational abscesses are often associated with an underlying condition such as diabetes, rheumatoid arthritis, steroid treatment, granulomatous lobar mastitis and trauma (Fig. 25.5).

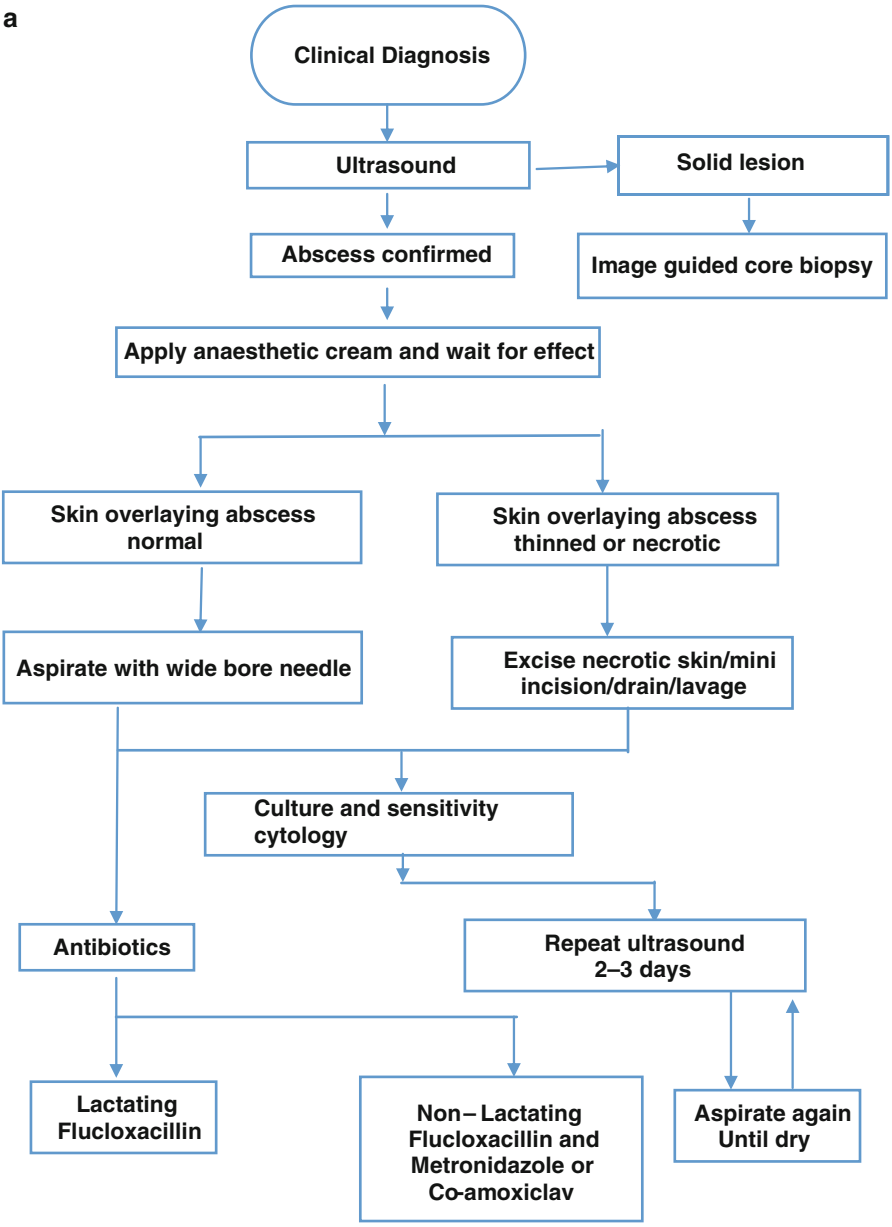
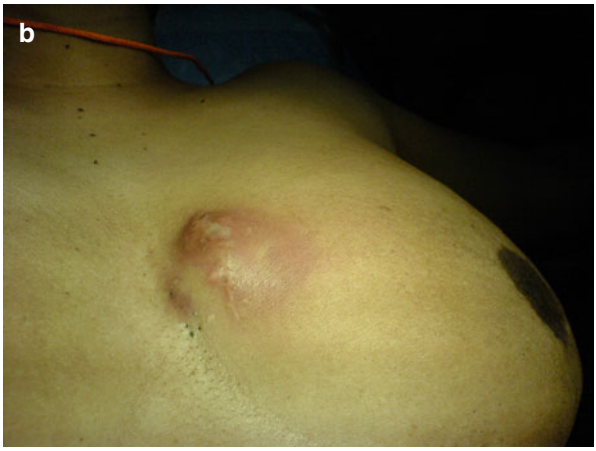


Fig. 25.5 (**a**, **b**) Non-lactational breast abscess

Fig. 25.5 (continued)



Treatment

Antibiotics

Though *S. aureus* is the most common pathogen, the occurrence of anaerobic bacteria in these patients warrants the addition of either clavulanic acid, metronidazole or clindamycin to the antibiotic of choice, i.e. flucloxacillin or dicloxacillin [8].

Drainage of the Abscess

Ultrasound-guided drainage is the procedure of choice [9].

Breast Trauma

Trauma to the breast can occur as a result of blunt or rarely penetrating force.

Blunt Trauma

Blunt trauma is usually the result of road traffic accidents, especially due to seat belt injury [10].

This can result in either a crush or an avulsion injury. Crush injury is more common resulting in a haematoma.

Clinical Features

- Patient will have pain and bruising over the affected breast. A diagonal bruise across the chest would result from a seat belt injury.
- Underlying rib fractures and lung injury may produce severe pain and breathlessness. A painful mass within the breast would be due to a haematoma.
- Avulsion injuries can cause an expanding haematoma under the breast and may require surgical intervention.

Investigations

Appropriate history and clinical findings should be substantiated by imaging. Ultrasound of the breast will reveal the exact nature of the pathology. A chest x-ray is mandatory to rule out lung injuries and their consequences.

Treatment

Most blunt injuries can be managed with conservative treatment, i.e. analgesics and appropriate dressings.

Complications

Haematoma can get infected leading to an abscess. Traumatic fat necrosis can occur as a long-term complication of breast injury. This mimics a breast carcinoma on imaging and may have to be surgically excised in order to confirm the true nature of the lesion.

Penetrating Trauma

- Penetrating injury to the breast is quite rare. It can occur as a result of stab injuries or road traffic accidents.
- With more number of women undergoing core needle biopsies for suspicious breast lesions, iatrogenic injury to the breast is increasing now. Uncontrolled bleeding is seen in about 5–7 % of patients after vacuum-assisted breast biopsy (VABB). Haematomas are more common following core biopsies [11].

Investigations

A chest x-ray and ultrasound breast would reveal the nature of the underlying injury. Rarely an angiogram is required in the presence of ongoing bleed following a breast biopsy [12].

Treatment

Lacerations should be sutured appropriately. Haematomas should be managed with compression dressings. Ongoing bleed or an expanding haematoma may require therapeutic embolisation.

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Chapter 26
Burns

Narendra Nath Jena, K.N. Vallirajan, and Binita Jena

Key Points
- Burn injuries cause a significant mortality and morbidity resulting in social isolation and stigma.
- Assessing the depth of burns is important in planning wound care and predicting functional and cosmetic outcome.
- Immediate management of major burns is a well-organised protocol which needs to be implemented for best outcome.
- Appropriate wound care reduces morbidity and facilitates early healing.

Introduction

Burn is an injury to the skin caused by thermal, chemical, radiation or electrical energy. Respiratory damage from smoke inhalation is also considered as burn-related injuries. The term burn is also used to include scalds which are caused by contact with hot liquid or steam. Globally, an estimated 195,000 deaths occur each year from fire-related burns, and it ranks among the 15 leading causes of deaths in children and young adults aged 5–29 years [1]. Over 95 % of fire-related deaths occur in low- and middle-income countries [1]. In addition to the fire-related mortalities,

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millions are left with lifelong disfigurement and disabilities which often result in social stigma and rejection. Hence, burns are a serious public health problem.

Assessment of Burns

Risk Factors

Young children, adults aged >60 years and males are at increased risk of sustaining burns [2, 3]. The increased risk in children and adults aged >60 years may be due to lack of co-ordination, dependency and poorly developed self-protective mechanisms. The increased risk in males may be due to increased risk-taking behaviour and contact with flame and high voltage at employment.

Classification of Burns

There are several ways of classifying burns. Burns can be classified by their mechanism or cause, depth or degree of burns and the extent of the body surface area burned.

Classification of Burns by Mechanism

- Thermal burns can present as:
  - Flame burns – caused by flames started by stoves, lamps, candles, lighted cigarettes, etc.
  - Contact burns – caused by hot solid items such as hot pressing iron, hot utensils, cigarette ends, etc.
- Chemical burns – caused by chemical exposure such as strong acids or alkali
- Electrical burns – caused by passage of electrical current from an electrical outlet, cord and appliance through the body
- *Inhalational burns*
  - Caused by breathing in superheated gases, steam, hot liquids or noxious products of incomplete combustion. They cause thermal and chemical injury to the lungs and airway.

Classification by Depth or Degree of Burns

- Burns are classified according to the depth of injury to the skin which helps in planning for wound care and predicting functional and cosmetic outcome.

- In the past, burns were classified as first-, second- or third-degree burns based on the clinical appearance of the skin after injury. This old system of classification is replaced by a new system which classifies burns as superficial and partial- or full-thickness burns.
- Burn wounds are dynamic and need reassessment in the first 24–72 h. Hence, this classification is more important once patients are in a burns centre and less important in the emergency department as burn wounds change in the first 3–5 days post injury.
- Assessing the percentage of full-thickness burns that definitely require surgery is one of the major determinants of survival following burn injury.

First-Degree or Superficial Burns

Superficial (first-degree) burns are burns to the epidermis. The surface appears fiery red and very painful, but no blister. Superficial burns are caused by exposure of the skin to solar radiation (sunburn) or brief exposure of the skin to hot substances, liquids or flames. Superficial burns heal within a week with no permanent change in skin colour, texture or thickness (Fig. 26.1).

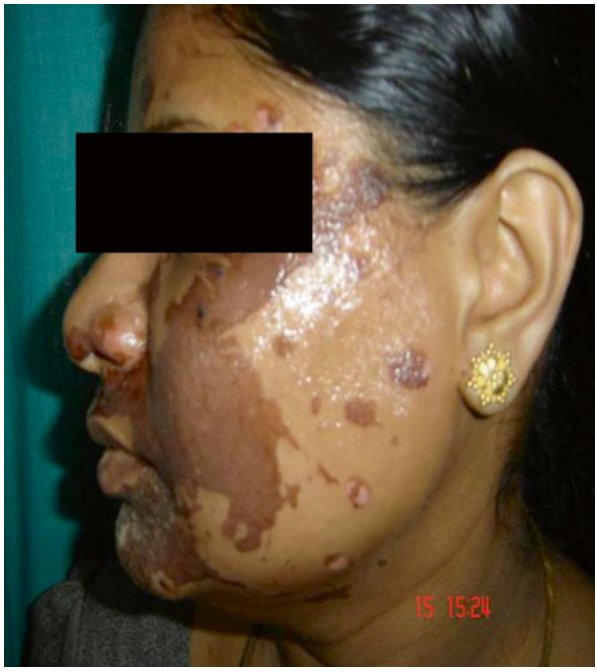


Fig. 26.1 Superficial burns – healing phase of superficial burn (5th day); note the brownish discolouration of initial erythematous skin

Second-Degree or Partial-Thickness Burns

In second-degree burns, the damage to the skin extends beneath the epidermis into the dermis and is characterised by a red or mottled appearance with associated swelling and blister formation. The surface has a wet, weeping appearance and is extremely painful and hypersensitive even to air current.

- Superficial second-degree burns: take less than 3 weeks to heal (Figs. 26.2 and 26.3).

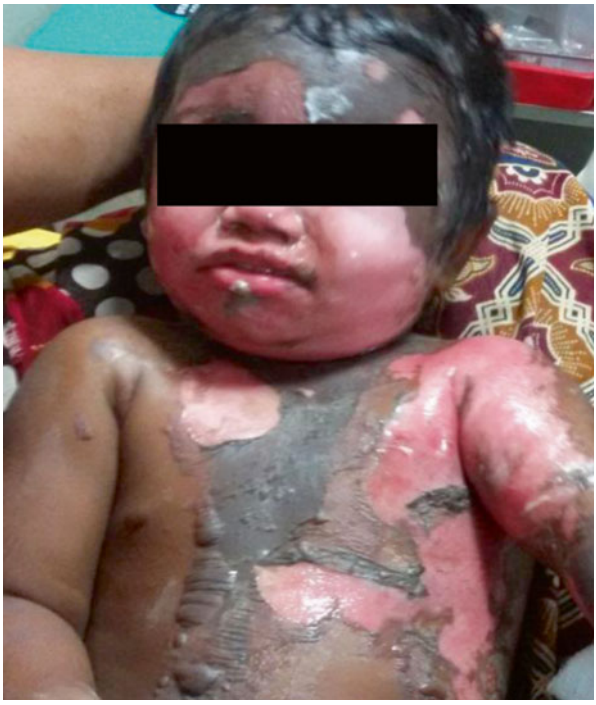


Fig. 26.2 Second-degree superficial burns – with blister formation and wet weeping appearance

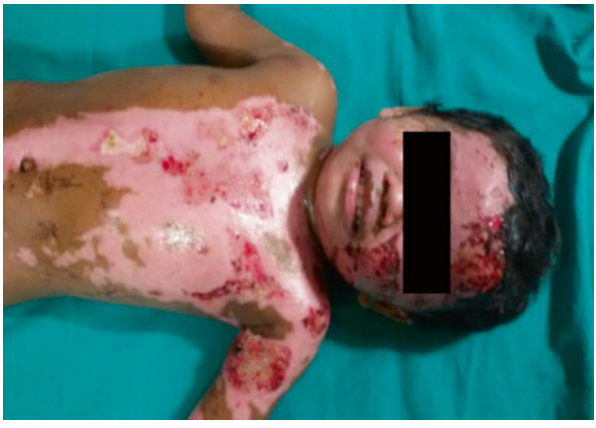


Fig. 26.3 Second-degree superficial burns – the above child 8 days after wound dressing

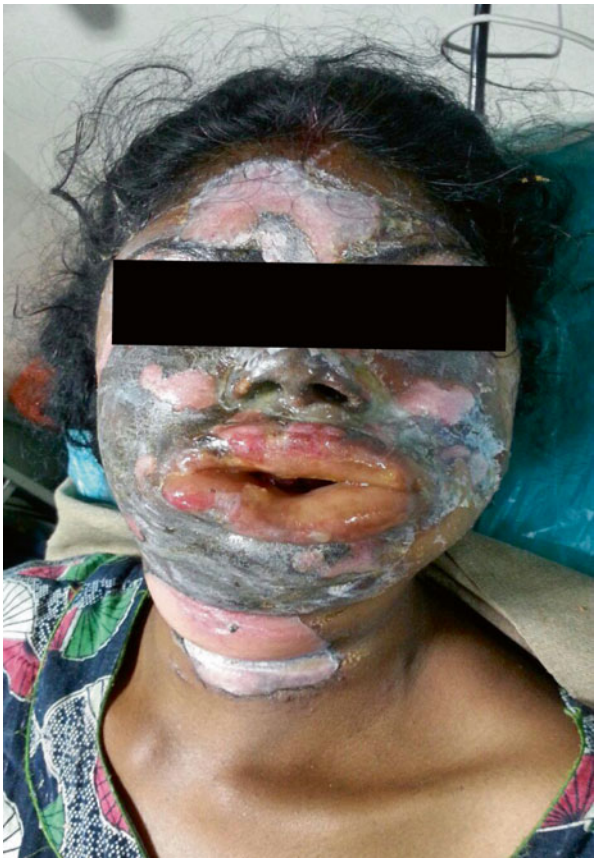


Fig. 26.4 Second-degree deep burns

- Deep second-degree burns: take more than 3 weeks to heal and are likely to form hypertrophic scars (Fig. 26.4).

Third-Degree or Full-Thickness Burns

In full-thickness burns, the damage involves the epidermis, dermis, subcutaneous tissue and deep hair follicles. The surface may appear translucent, mottled or waxy white. These burns are painless and dry; it may appear red, but do not blanch on pressure. Full-thickness burns require skin grafting (Fig. 26.5)

Fourth-Degree Burns

Fourth-degree burns involve subcutaneous tissue, muscles and bone; it requires reconstruction, often amputation (Fig. 26.6)

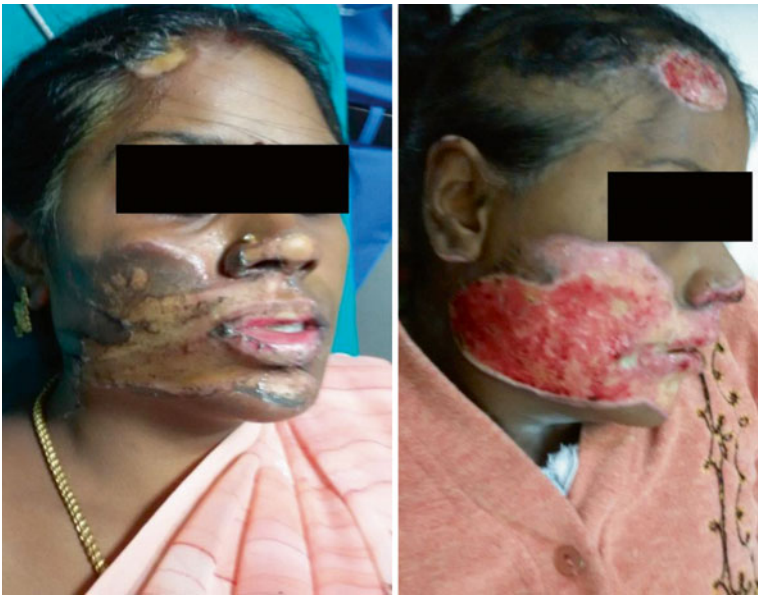


Fig. 26.5 Third degree burn-Before and after debridement

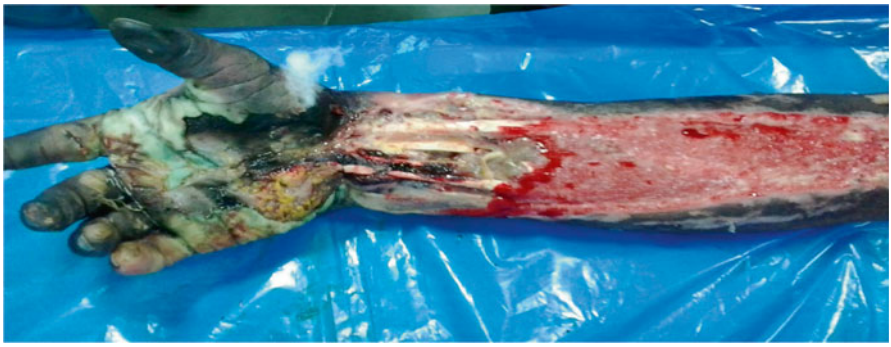


Fig. 26.6 Fourth-degree burn

Classification by Extent of Burn

The extent of burns refers to the proportion of total body surface area burned. The most commonly used method to determine the extent of burns is the "rule of nines". This method assigns 9 % to the head and neck region, 9 % to each arm (including the hand), 18 % to each leg (including the foot) and 18 % to each side of the trunk and 1 % for perineum. The "rule of nines" method is used for adults and children older than 10 years. For children younger than 10 years, the Lund and Browder chart is used (Fig. 26.7).

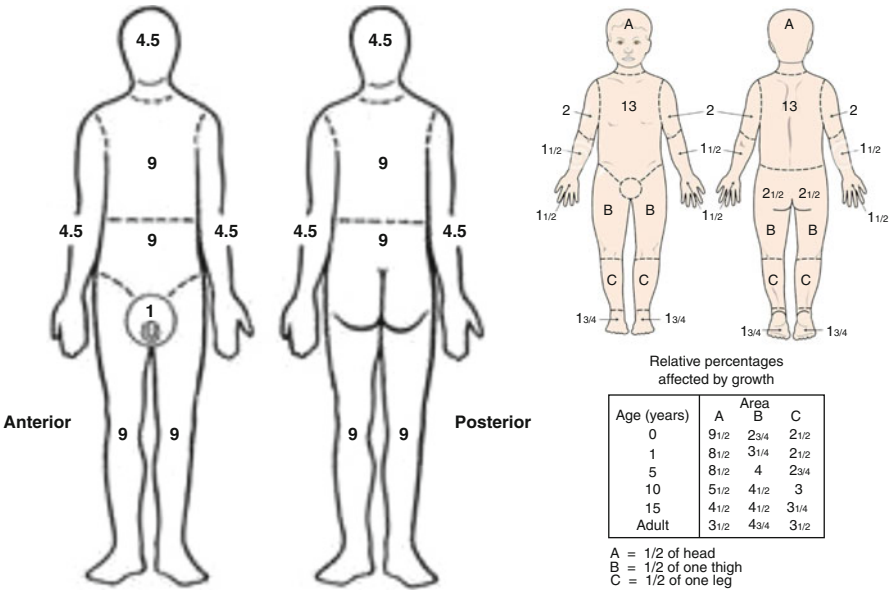


Fig. 26.7 Percentage of burns

Baseline Investigations for Major Burn Patient

- Blood: CBC, type and crossmatch, carboxyhaemoglobin, serum glucose, electrolytes, pregnancy test in all females of child-bearing age and arterial blood gas analysis.
- CXR and other X-rays if associated with injuries.
- Cardiac monitoring: dysrhythmias may be the first sign of hypoxia or electrolyte imbalance.
- Circulation: major burn patients may be in hypovolaemic shock. Blood pressure measurement may be difficult or unreliable. Hourly urine measurement reliably assesses the circulating blood volume; hence, indwelling urinary catheter must be inserted.

Management of Minor Burns [4]

- Minor burns should be cleaned with soap and water or a diluted water-based disinfectant.
- All blisters should be left intact to reduce the risk of infection. Large blisters should be aspirated by sterile technique.
- Nonadhesive dressing with gauze padding is usually effective.
- Wound should be re-examined at 48 h to assess the depth of the burns.

- If wound infection occurs, daily wound inspection and dressing will be needed. Antibiotics should be prescribed.
- Tetanus prophylaxis and adequate analgesia should be ensured.

Management of Major Burns

Immediate Management

Airway: Inhalational injury can cause upper airway obstruction and oedema. Inhalation of carbon particles and toxic fumes can cause chemical tracheobronchitis and pneumonia. Clinical presentation of inhalational injury may be subtle and may not appear in the first 24 h [5]. Carbon monoxide poisoning should be suspected in patients who were burned in enclosed areas. Inhalational injury should be suspected in:

- Face and/or neck burns
- Singeing of eyebrows and around the nose
- Carbon particles in the oropharynx or sputum
- Hoarseness of voice
- Impaired awareness, e.g. due to alcohol or head injury and/or confinement in a burning environment
- Carboxyhaemoglobin >10 %
- Explosion with burns involving the head and torso

Management of acute inhalational injury needs early endotracheal intubation and ventilation [6]. Stridor is an indication for immediate endotracheal intubation.

Breathing:

- Arterial blood gas analysis should be obtained for all major burn patients. But PaO2 does not reliably predict carbon monoxide poisoning. Therefore, carboxyhaemoglobin levels should be obtained. One hundred percent oxygen should be supplemented.
- Carbon monoxide has a greater affinity for haemoglobin; hence, it displaces oxygen. CO dissociates very slowly, but this dissociation is increased with 100 % oxygen supplementation via a non-rebreather mask. Patients with CO levels <20 % usually do not have symptoms; higher levels can cause confusion, headache, coma and death.
- The head and torso should be elevated by 20–30° to reduce neck and chest wall oedema. If full-thickness burns to the chest wall are causing severe restriction of the chest wall movements, then chest wall escharotomy (burn incised through subcutaneous fat and underlying soft tissue; no anaesthetic is required) may be needed.

Circulation: Adults with burns >15 % body surface area and child with burns >10 % body surface area require fluid resuscitation. Large-bore IV access should be obtained in all major burn patients.

- Fluid needs to be titrated to maintain adequate urine output and special care needs to be taken when myoglobinuria is present.
- Lactated ringer solution without dextrose is the fluid of choice except in children under 2 years, who should receive 5 % Dextrose ringer lactate.

- The time of fluid resuscitation is to be calculated from the time of burn and not from the time of initial therapy.
- In children, provision for the daily maintenance requirements on top of the calculated amount.
- Any fluid already given should be detected from the calculated reuirement.
- The resuscitation formula most commonly used for fluid calculation are Parkland formula and modified Brookes formula.
- Resuscitation formula (Adults) for first 24 h

Formula	Crystalloid	Colloid	Daily needs
Parkland	4 ml/kg/% TBSA	None	None
Brook (modified)	2 ml/kg/% TBSA	None	2 l

- The calculated volume is given at different rates:

  First 8 h – Half of the total to be given.
  Next 16 h – Half of the total to be given.
  Next 24 h – Half of the total given on first day.

- The end point to aim for is a urine output of 0.5 ml to 1 ml/kg/h in adult and 1–1.5 ml/kg/h in children.

Management of Burns

- All clothing and jewellery should be removed. Adherent clothing and tar should be cooled by water and removed by formal debridement.
- Dry chemical powders should be carefully brushed from wounds.
- Burns should be irrigated with running cool tap water for 20 min, taking care to avoid hypothermia especially in children. Chemical burns may need longer periods of irrigation.
- Dressings can help to relieve pain and keep the wound clean, but avoid circumferential dressing as it can cause constriction.
- *Adequate analgesia*: with strong opiates

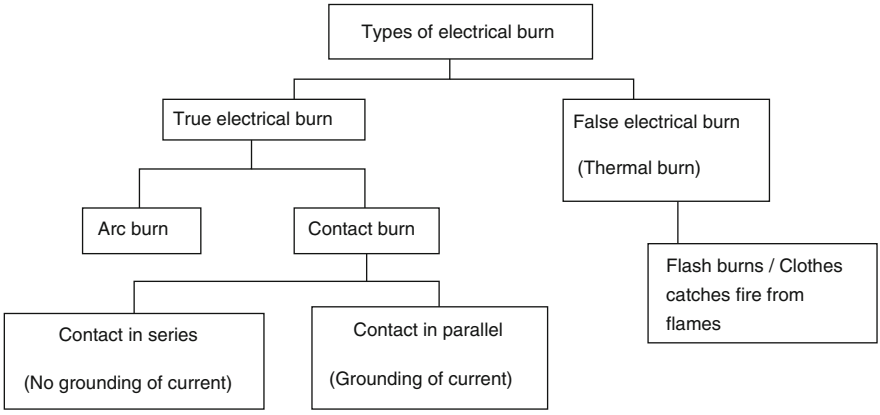
Ensure Tetanus Prophylaxis

- In patients who have not received primary tetanus immunisation, 0.5 ml tetanus toxoid IM should be given at presentation, followed by two further injections at 4 weeks' interval and a booster dose at 6 months to 1 year. These patients should also receive 500 units of tetanus immunoglobulin IM at presentation.
- For all burn patients who have had primary tetanus immunisation series and a booster dose, 0.5 ml of tetanus toxoid should be given IM at presentation.
- If the last booster is more than 10 years or if there is a delay between time of burning and treatment, patients should receive 500 units of human tetanus immunoglobulin IM in addition to tetanus toxoid injection, loaded in a different syringe and given at a different site.

Electrical Burns

Electrical burns occur when a source of electrical power comes in contact with a person's body. Electrical current causes damage by the following mechanisms [7]:

- Conversion of electrical energy into thermal energy causing massive tissue destruction and coagulative necrosis. Difference in the rates of heat loss in superficial and deep tissues can result in relatively normal overlying skin with deep muscle necrosis. Hence, electrical injuries are more serious than they appear on the body surface.
- Secondary injuries from fall and violent muscle contractions.

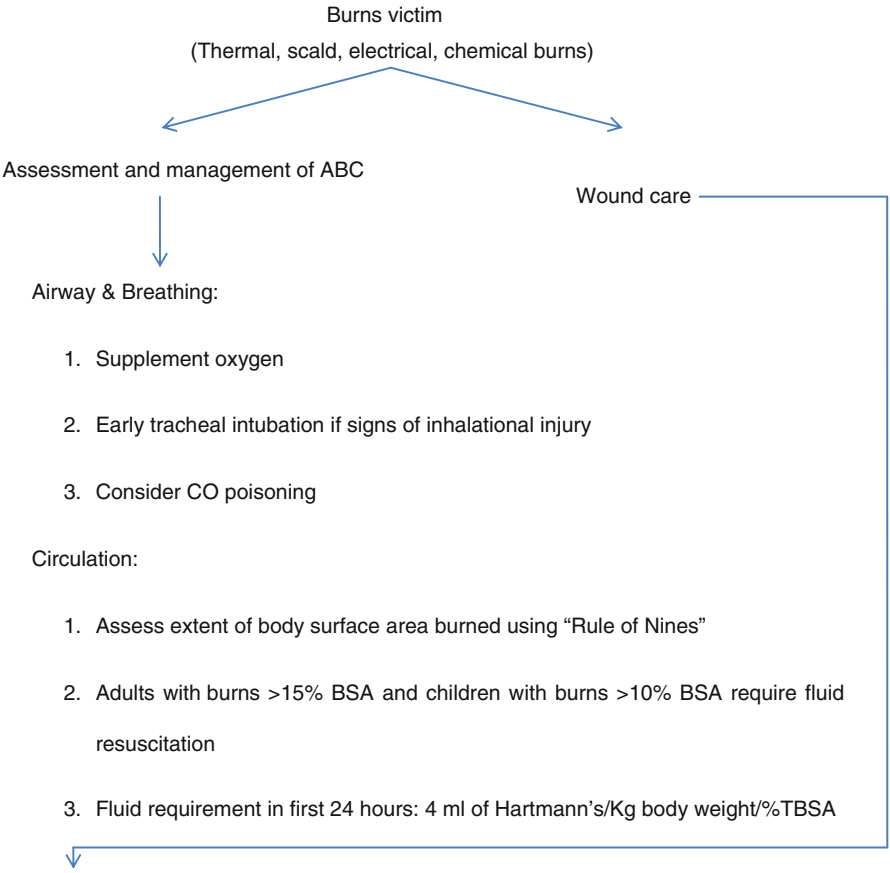


Electrical injury can result in rhabdomyolysis causing myoglobinuria and acute renal failure. In rhabdomyolysis fluid resuscitation should be increased 9 ml×%TBSA×B.Wt to ensure a urine output of 1.5 to 2 ml/h. If urine appears dark, myoglobinuria should be assumed, and fluid therapy should be initiated even before laboratory confirmation. If the pigment doesn't clear by increased fluid administration, 25 g of mannitol should be given, and 12.5 g of mannitol should be added to subsequent litres of fluid to maintain diuresis. Metabolic acidosis should be corrected by maintaining adequate perfusion and by adding sodium bicarbonate to alkalise the urine.

Chemical Burns

Chemical burns can result from acidic, alkaline or petroleum products. Alkali burns are deeper and more serious than acid burns. Chemical burns should be washed with copious amount of water for 20–30 min and for longer periods with alkali burns. Alkali burns to the eye will require continuous irrigation to the eye for the first 8 h. Dry chemical powders should be carefully brushed away before irrigation.

Burns Algorithm



Conclusion

Burns are a major health concern in low-resource countries due to the lack of health and safety laws as well as preventive measures. Burn injuries cause a significant mortality and lifelong disability resulting in social taboo and rejection. Estimation of percentage of full-thickness burns needing definite surgery is a major predictor of survival in burn victims. Management of major burns involves assessment of airway, estimation of carboxyhaemoglobin, fluid resuscitation, dressing of burns, assessing the need for escharotomy/fasciotomy, adequate analgesia and tetanus prophylaxis.

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Chapter 27
Penile and Scrotal Pain

Gopalakrishnan Nurani Sundareswaran

Key Points

- Genital pain has physical as well as psychological components. A sensitive approach to investigate and resolve both mentioned above is deemed as good medical practice.
- Physical examination is incomplete without examination of all related anatomical structures.
- Delay in detecting or referring certain conditions for definitive care can have disastrous consequences with potential loss of life or organ.

Introduction

Patients presenting with genital pain may pose a considerable challenge to the emergency physician, due to the vague and evasive behaviour commonly noted among this group of patients. Personal embarrassment and social inhibitions are often the most common reasons for a delay in seeking attention for these medically significant conditions. A clinician who adopts an empathetic attitude in addition to providing prompt and adequate analgesia will be rewarded with the opportunity to conduct thorough history taking and complete physical exam, on a cooperative and confident patient.

The main objectives of the clinical encounter are:

- To provide prompt and adequate analgesia
- To formulate a list of differential diagnoses, to request appropriate investigations and to arrange prompt referral to other specialists if needed

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- To educate patients about the preventive aspects of their problem
- To initiate contact tracing for certain transmissible conditions

Pathophysiology

Genital pain is a common symptom associated with a wide variety of medical conditions. Some conditions are benign and self-limiting, whereas others, if left undiagnosed or untreated, rapidly threaten life or organ. Importantly, the severity of pain has no correlation with the morbidity or mortality associated with the underlying disorder.

Tables 27.1 and 27.2 list the important causes of penile and scrotal pain, respectively.

Epididymitis

This is an inflammation of the epididymis. When the inflammation involves the testis as well, it is called epididymo-orchitis. This is the most common diagnosis for scrotal pain in all age groups. In the young male, the possibility of sexually transmitted infections must be considered.

Table 27.1 Causes of penile pain

Aetiology	Examples
Trauma	Fracture of penile shaft
	Zipper injury
	Torn fraenulum
	Urethral tear/stricture
Infection	Fournier's gangrene, balanitis, balanoposthitis, cystitis, UTI
STDs	Herpes simplex, human papillomavirus, chlamydia, gonorrhoea, syphilis, trichomoniasis
Infestations	Lice, scabies
Vascular	Sickle cell crisis
Allergic	Contact dermatitis related to latex, chemicals, metal piercings, tattoos, lubricants
Inflammation	Psoriasis, Reiter's disease
Mechanical	Peyronie's disease, priapism, paraphimosis
Malignancy	Penile tumours, urethral tumours
Referred pain	Prostatitis, urinary retention
	Genitourinary calculus

Table 27.2 Causes of scrotal pain

Aetiology	Examples
Trauma	Testicular rupture, intratesticular haematoma, haematocele
	Spontaneous haematomas with anticoagulants
Infection	Fournier's gangrene
	Scrotal abscess, sebaceous cyst with secondary infection, orchitis, epididymitis, epididymo-orchitis
Vascular	Varicocele, sickle cell crisis
Mechanical	Torsion of the testis, torsion of the epididymis, torsion of the appendix testis, torsion of the appendix of the epididymis, incarcerated or strangulated inguinal hernia
	Hydrocele, spermatocele
Malignancy	Testicular tumours with secondary changes like infection, rupture, infarction, haemorrhage
Inflammation	Henoch-Schonlein vasculitis, fat necrosis of scrotal wall, contact dermatitis
	Post-viral orchitis
Referred pain	Abdominal aortic aneurysm, UTI, genitourinary calculi, urine retention, acute appendicitis

Causes

- Infections – Bacteria (usually *Coliforms*), *Chlamydia*, *Neisseria* sp., viruses, fungi, mycobacteria
- Drug induced – Occasionally associated with amiodarone
- Chemical – Urine reflux
- Autoimmune

Clinical Features

- May present acutely with sudden onset symptoms or may have a slowly progressive chronic course.
- The pain develops gradually either unilaterally or bilaterally.
- Systemic symptoms like fever and rigors.
- Dysuria, haematuria or a discharge from the urethra.

Investigations

- Blood counts, urea and electrolytes, liver function tests
- Urine microscopy

- Midstream urine for culture
- Urine for Chlamydial polymerase chain reaction test
- Urethral discharge swabs
- Ultrasound of the scrotum

Treatment

- Aim is to identify and correct the underlying cause.
- Oral NSAIDS for analgesia.
- In the prepubertal age group, viruses are the most common pathogen. Treatment is mainly supportive and symptomatic. Antibiotics are indicated only for the few patients who have pyuria or a positive urine culture [1].
- In the below-40 age group, sexually transmitted pathogens must be considered.
- For confirmed chlamydial or gonococcal infections, ceftriaxone 500 mg intramuscular, followed by oral doxycycline 100 mg twice daily for at least 2 weeks must be used. Ofloxacin 200 mg twice daily for 14 days is the second-line treatment [2].
- In the above-40 age group, *Coliforms* are the usual pathogens. Oral ofloxacin 200 mg BID for 14 days or oral ciprofloxacin 500 mg BID for 10 days may be prescribed.
- Antibiotic use may be modified once definitive identification of the causative pathogen is made.

Prognosis

This condition generally responds well to early identification of the cause and definitive treatment along with supportive anti-inflammatory drugs.

In the extremes of age or in the immunocompromised, contiguous spread of the infection may lead to coexisting orchitis, or haematogenous seeding may lead to septicaemia.

Prevention

- Early vaccination against sexually transmitted disease, the advocacy of monogamy and the use of condoms all reduce the incidence of this condition.
- Patients having sexually transmitted epididymitis must be encouraged to seek treatment for all their sexual contacts.

Orchitis

This is the inflammation of the testes and may be unilateral or bilateral.

It may occur in isolation or as a part of epididymo-orchitis.

Pathophysiology

Viral infection is the most common cause of orchitis. Infections such as mumps, Coxsackie virus, infectious mononucleosis and varicella have all been commonly noted to precede an episode of orchitis.

The same bacteria implicated in epididymitis may result in orchitis as a result of either direct or haematogenous spread. The possibility of *Chlamydia* and *Gonococci* must be considered in appropriate age groups.

Autoimmune orchitis may be primarily due to anti-sperm antibodies or occur as part of a systemic vasculitis.

Clinical Features

- Unilateral or bilateral tender, enlarged and painful testes.
- Scrotal skin over the affected testes may appear erythematous and shiny with loss of the normal scrotal folds.
- Fever, rigors and nausea may occur occasionally.
- Testicular torsion must always be ruled out as a possibility in any case presumed to be epididymo-orchitis [3].

Investigations

- Blood counts, urea and electrolytes, liver function tests.
- Urine microscopy, culture and urethral swabs.
- Ultrasonographic examination may be requested in cases of suspected hydrocele or pyocele.

Treatment

- For bacterial infections, follow the same antibiotic guidelines as for epididymitis.
- NSAIDS for analgesia.
- Use supportive scrotal slings or intermittent cold pack applications.

Prognosis

- Recovery is good if the condition is diagnosed appropriately and treated early.
- A few cases may be complicated by the development of hydrocele or pyocele.
- Rarely, the affected testes undergo atrophy and contribute to subfertility if only one testis is involved or infertility if both testes are involved.
- Immunocompromised individuals are at particular risk of rapid spread of microbes locally or systemically and may present as fulminant gangrene or septicaemia.

Prevention

The presentation of young patients with sexually transmitted diseases must be used as an opportunity to educate them about safe sexual practices and hygiene.

Paraphimosis

Pathophysiology

This is a progressive and painful condition which occurs when a narrow and retracted foreskin cannot be protracted and forms a constricting band around the glans impeding lymphatic and venous return, setting off a vicious cycle of events.

Patients with pre-existing phimosis are naturally more prone to developing this condition.

Chronic balanoposthitis, metal piercings of the genitalia and iatrogenic causes, such as a persistent retracted prepuce, following urinary catheterisation can predispose to this condition.

Clinical Features

- Doughnut-shaped swelling around the anterior aspect of penis with varying degrees of glanular oedema.
- Delayed cases may present with discoloured or frankly gangrenous glans or preputial skin.

Treatment

- Under penile dorsal nerve block and liberal lubrication, attempts are made to protract the foreskin back over the glans [4].
- Various techniques exist to reduce the oedema over the glans prior to manipulation including application of sugar or hypertonic saline gauze and multiple needle punctures of oedematous tissues.
- If digital manipulation fails, a temporising dorsal slit may be made on the foreskin to relieve the constricting pressure of the ring.
- Consider referral to urologist for a planned circumcision even after successful initial treatment.

Zipper Injury

Introduction

This is the most common genital trauma in prepubertal boys.

Pathophysiology

May be sustained while zipping up or down and has no correlation to wearing undergarments.

The zipper actuator or the teeth can entrap the penile skin, glans or both Fig. 27.1.

Treatment

- Various techniques exist to release the trapped skin, all of which focus on mechanically breaking the zipper apparatus apart using wire cutters, bone cutters or a screwdriver after adequate penile local anaesthesia or under general anaesthesia [5, 6].
- An effective way is to cut the median bar, with wire cutter, and the zipper falls apart.
- The two plates of the zipper actuator may also be prised apart using two needle holders to relieve the pressure on the tissues [7].

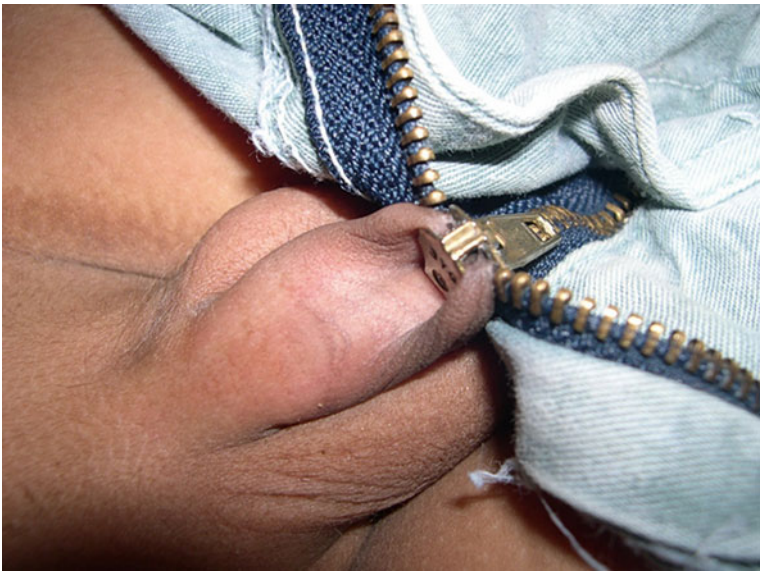

Fig. 27.1 Zippered penis (Courtesy: Prof. Suresh S. David. Pushpagiri Medical College Hospital-Tiruvalla 689101-Kerala, India)

- The zip may be cut transversely through the cloth and teeth, and pulled at both ends to release trapped tissues [8].
- Repair of any lacerations is done with absorbable sutures. A secondary survey for urethral injuries is mandatory.

Urethral Trauma

Pathophysiology

Urethral trauma may be caused by blunt or penetrating trauma and can involve the anterior (bulbar or penile part) or posterior urethra (at or above membranous part).

May lead to various degrees of injury including partial or complete tears or crushing of the tissues.

Clinical Features

- Inability to urinate
- Visible perineal or shaft ecchymosis
- Presence of blood at the urethral meatus

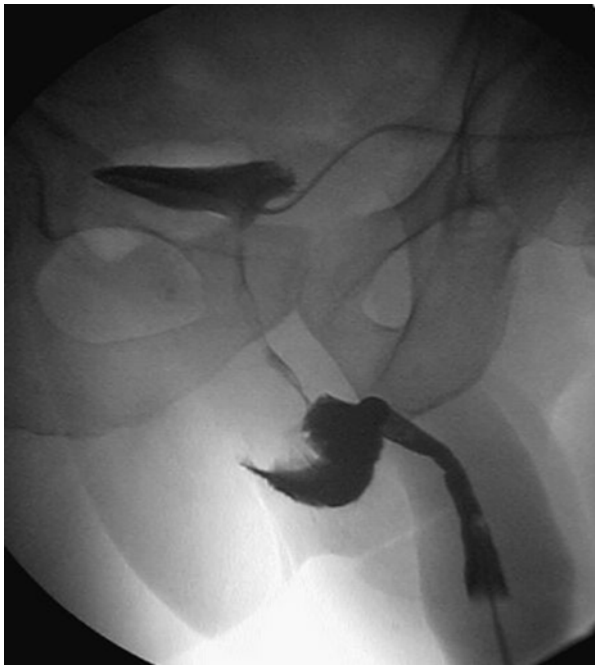

Fig. 27.2 Fluoroscopy showing extravasation of contrast in urethral trauma (Courtesy: Prof. Suresh S David. Pushpagiri Medical College Hospital-Tiruvalla 689101-Kerala, India)

Diagnosis

- May be reliably made by history and clinical features.
- An ascending urethrogram is mandatory before attempting catheterisation. Screening under fluoroscopy would demonstrate extra-urethral presence of the contrast (Fig. 27.2).
- Suprapubic or endoscopic urethral catheterisation is an initial temporising measure.
- Definitive treatment involves referral to urology for early surgical repair in case of anterior tears and evaluation for delayed repairs in case of posterior tears [9].

Fournier's Gangrene

Pathophysiology

This condition is characterised by fulminant, aggressive, necrotising infection of the fascial layers of external genitalia, perineum and perianal regions (Figs. 27.3 and 27.4).

Suppurative bacterial infection results in micro-thrombosis of the small subcutaneous vessels leading to the development of gangrene of the overlying skin. The causative organisms are commonly *Staphylococci*, *Streptococci*, *Enterococci*,

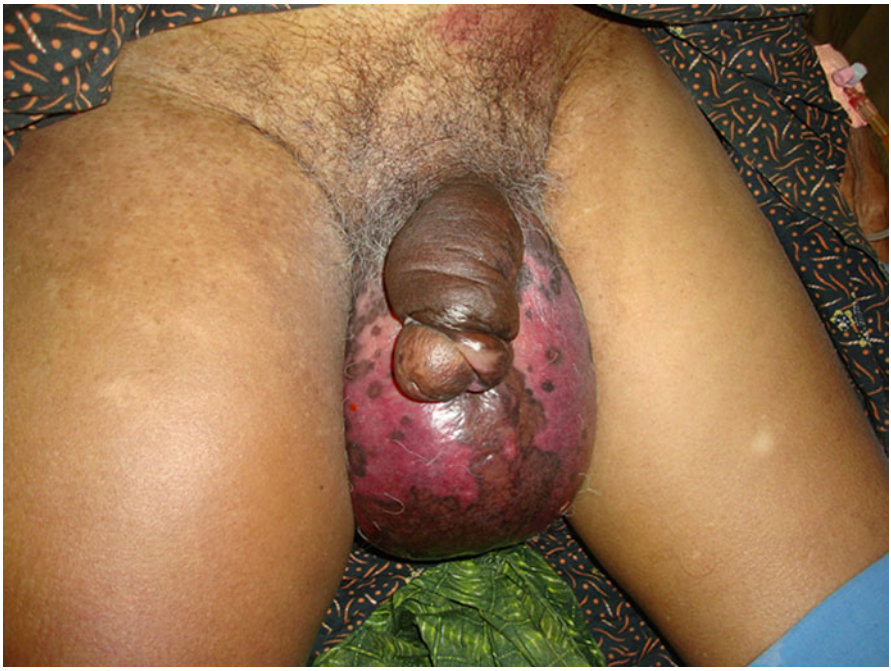

Fig. 27.3 Fournier's gangrene (Courtesy: Prof. Suresh S David. Pushpagiri Medical College Hospital-Tiruvalla 689101-Kerala, India)

Bacteroides and *Corynebacteria* although fungi like *Candida* and *Lactobacilli* have also been implicated [10]. Polymicrobial synergism leads to rapid tissue destruction and end-artery obliteration to set up conditions ideal for extremely rapid spread of the process [11].

Many of the patients affected are immunocompromised due to pre-existing conditions like diabetes mellitus, chronic alcoholism, malignancy, steroid use, chemotherapy, malnutrition, lymphoproliferative conditions or HIV infection.

Clinical Features

- Fever, chills, visible swelling or cellulitis in the genital or perineal area are early signs.
- Necrotic patches commence to appear over the skin and progress to extensive necrosis.
- Pain is present but a very unreliable symptom, since it has no correlation to the extent of the infection.
- There may be a palpable crepitus near the perineum due to colonisation of gas-forming organisms.
- Significant systemic signs, out of proportion to the local symptoms of the disease, are a hallmark of this condition.

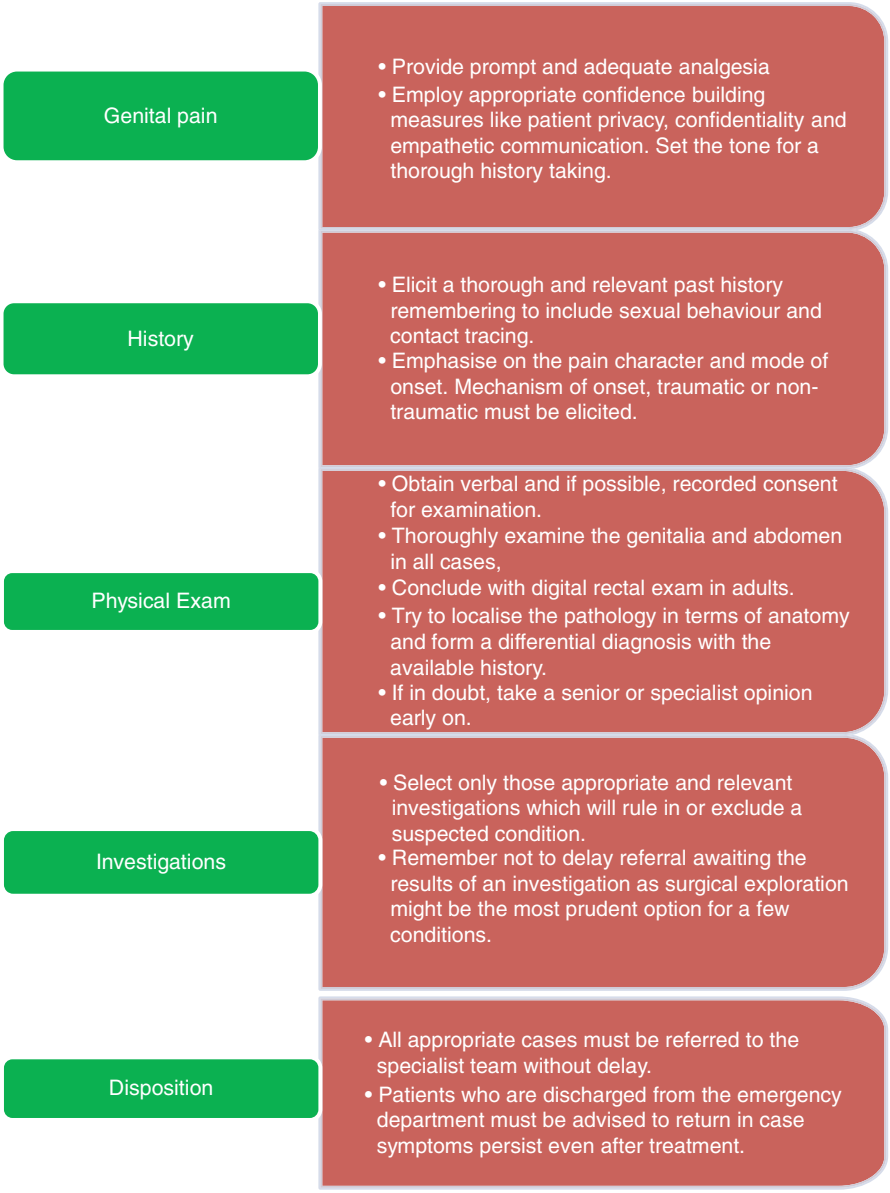

Fig. 27.4 Management algorithm of genital pain

Investigations

- Diagnosis may be made by the clinical presentation alone.
- Laboratory tests aid in detecting organ failure secondary to septicaemia. Wound swabs and blood cultures help to guide antimicrobial therapy.

- Treatment must be initiated aggressively and involves antimicrobial therapy to cover for Gram-positive, Gram-negative and anaerobic organisms along with fluid resuscitation.
- Current guidelines advocate the use of triple antibiotic therapy with third-generation cephalosporins, penicillins and metronidazole.
- Emerging evidence also favours the use of carbapenems like meropenem or alternatively piperacillin and tazobactam [12].
- Antifungal treatment is with intravenous fluconazole.
- Urgent referral to urology for early surgical debridement of necrotised tissues is mandatory as the advance of necrosis has been reported to be as high as 2–3 cm every hour.

Prognosis

This is a true urological emergency and one which has a mortality rate of 20–30 % even after initiation of appropriate and aggressive treatment.

A very high index of suspicion is necessary to prevent the high morbidity and mortality.

Fracture of Penile Shaft

Pathophysiology

- Defined as a break in the tunica albuginea which covers each corpus cavernosum individually, due to mechanical forces.
- May be caused by forceful coitus, masturbation or trauma to an erect penis.

Clinical Features

- Sudden pain, bruising, deformity and sudden loss of erection usually after trauma. It is not unusual for patients to report an audible crack during the process.
- Blood at the urethral meatus, difficulty in passing urine or the finding of a high riding prostate on digital rectal exam all raise a suspicion of an associated urethral injury.
- The fracture is usually transverse and may involve one or both corpora cavernosa.

Investigations

- History and clinical features are occasionally sufficient to make a reliable diagnosis.
- An ultrasound is only an adjunct to confirm the diagnosis.
- An ascending urethrogram is indicated for suspected urethral trauma [13, 14].

Treatment

- Early referral for surgical treatment is mandatory and is associated with a low incidence of delayed complications [15].

Differential Diagnosis

- Isolated rupture of dorsal vein or dorsal artery of penis

Balanitis and Balanoposthitis

Introduction

Balanitis is an inflammation of the glans penis. When the inflammation involves the prepucial skin as well, it is termed balanoposthitis.

Pathophysiology

These conditions are more common in uncircumcised males. Diabetics are more prone to developing candidal infections.

The inflammation may be due to infection by bacteria, viruses or fungi. Atopy and vigorous washing are also known predisposing factors [16]. Drugs like tetracyclines, salicylates, phenacetin and antihypnotics are causally associated.

Investigations

- A good history is essential to detect the aetiology and help in directing appropriate treatment.
- Swabs and cultures reliably identify the causative organism in cases of infection.
- Skin biopsy must be considered in cases refractory to medical management.

Treatment

- This is directed at the underlying cause.
- A gentle daily penile wash with warm water after retracting the foreskin, followed by drying and avoidance of physical irritants like detergents and spermicides, ensures good recovery.
- Diabetics must be advised for tight glycaemia control.
- Current recommendations include first-generation cephalosporins for bacterial infections and clotrimazole cream or oral fluconazole for fungal infections. Steroids are reserved for advanced conditions and used sparingly after antimicrobial therapy has been initiated.

Prognosis

Appropriate therapy ensures a good recovery. Refractory cases not responding to initial therapy must be considered for skin biopsy.

Vaso-Occlusive Crisis

Introduction

Various haematological conditions commonly present with genital pain secondary to vaso-occlusion [17]. Sickle cell disease is the most common condition presenting in this manner. Other conditions include haemoglobin C and thalassemia.

Pathophysiology

These events have a definite precipitating factor which in most cases is infection or dehydration, and the common pathway is mechanical obstruction of penile microcirculation by abnormally shaped RBCs.

Clinical Features

- Most common presentation is with pain and priapism which may be intermittent.

Treatment

- Adequate analgesia
- Oxygen supplementation
- Hydration
- Intravenous administration of alkalinisation agents
- Infection control
- Admission to monitor response to treatment

Priapism

Introduction

This is a painful and prolonged erection of the penis which persists for more than 4 h and is unrelated to sexual stimulation or activity.

Pathophysiology

It involves engorgement of the corpora cavernosa with blood.

Trauma, vaso-occlusion and drug injections into the corpora cavernosa are the commonest causes.

There are two distinct types which have different treatments [18].

- *Ischaemic or low-flow type* is a true emergency and must be treated as a compartment syndrome affecting the penile shaft.
- Presents with a painful and rigid penis. Diagnosis is confirmed by cavernosal aspiration of blood which is hypoxic and acidic. Other diagnostic tests like cavernosal Doppler ultrasound add little and only delay treatment.

Treatment

- Is urgent and does not depend on the precipitating factor.
- Cavernosal and saline irrigation provides partial relief of symptoms.

- Intra-cavernosal phenylephrine in a dose of 500 mcg to 1 mg every 5 min up to a maximum of 1 h is recommended under full haemodynamic monitoring.
- If medical treatment fails after 48 h, surgical management is indicated and commonly involves creating cavernoglanular shunts to divert stagnated blood.

Non-ischaemic or high-flow priapism is usually secondary to trauma but can also spontaneously occur due to fistula formation between the cavernous arterioles and corpora cavernosa.

Diagnosis

- The patient presents with a semi-rigid and often painless priapism.
- Blood aspirated from the corpus cavernosum is neither hypoxic nor acidic.
- Doppler ultrasound adds little by way of additional diagnoses and delays treatment.

Treatment

- Most cases of non-ischaemic priapism respond well to conservative measures like analgesia, application of ice packs and monitoring for resolution. Cases that do not resolve might need evaluation for angiography-assisted embolisation of the offending arterioles or surgical repair of fistula.
- All cases of priapism must be referred to urology for treatment and monitoring.
- Treatment for low-flow priapism is time sensitive and any delay can result in serious structural and functional deficits for the patient.

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Chapter 28
Procedural Sedation

Susan Tharian

Key Points
- All procedures that potentially induce pain – diagnostic or therapeutic, should be performed in the ED under procedural sedation.
- Procedural sedation carries all the risk of general anaesthesia; therefore, assessment of the patient, management of airway, monitoring, management of complications and fasting guidelines should be addressed.
- Paediatric group is more vulnerable for complications.

Introduction

Minor procedures are carried out in the emergency medicine department as part of patient care. These procedures can be painful, distressing or unpleasant. Procedural sedation is harnessed in such situations to reduce discomfort, apprehension and potential unpleasant memories associated with such procedure and facilitate performance of the procedure with ease.

Procedural sedation in ED is indicated in the following:

- Reduction of fractures or dislocations
- Suturing of wounds

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- Incision and drainage of abscess
- Electrical cardio-version
- Endotracheal intubation and other short invasive procedures
- In non-invasive ventilation
- Foreign body removal

The American College of Emergency Physicians (ACEP) defines "procedural sedation as a technique of administering sedative or dissociative agents with or without analgesia to induce a state that allows the patient to tolerate unpleasant procedures while maintain cardiorespiratory function" [1]. Procedural sedation carries all the risks of general anaesthesia. So it is mandatory that emergency physician should have an adequate knowledge of the drugs used for sedation and analgesia, their side effects and associated complications. Moderate to deep sedation is required to do these procedures in the emergency department.

Levels of sedation as described by the American Society of Anaesthesiologist [2]:

- Minimal sedation – essentially mild anxiolysis or pain control. Patients respond normally to verbal commands, e.g. – charge of burn dressing.
- Moderate sedation and analgesia – (formerly known as "conscious sedation"). Patients are sleepy but arousable by voice or light touch. Although their respiration is self-maintained, this may be suppressed with deeper levels of sedation, e.g. direct current cardio-version.
- Deep sedation and analgesia – require painful stimulus to evoke a purposeful response. Airway or ventilation support may be needed, e.g. major joint reduction.
- General anaesthesia – patient has no purposeful response to even repeated painful stimuli. Airway and ventilation support is usually required. Cardiovascular function may also be impaired. Not appropriate for general use in the emergency department.

Drugs commonly used for sedation in ED are showed in the Table 28.1.

Opioids

- Offer analgesia with minimal sedation in usual doses.
- Morphine and fentanyl are the commonly used opioids in the ED.

Morphine

- Is the most frequently used analgesic in the ED
- Provides potent analgesia, sedation and euphoria

Table 28.1 Dosage of drugs commonly used in adults for procedural sedation in the emergency department

Drug	Dose	Onset of action	Duration of action
Morphine [3]	0.1 mg/kg IV	5–10 min	120–160 min
Fentanyl	1-mcg/kg IV over 1–2 min repeat dose after 30 min	1–2 min	30–60 min
Midazolam	0.02–0.1 mg/kg IV If further sedation required, you may repeat with 25 % of initial dose after 3–5 min	1–2 min	30–60 min
Propofol [4]	0.5–1 mg. kg IV loading dose may repeat by 0.5 mg/kg increments in every 3–5 min 1 mcg/kg IV over 10 min	<1 min	3–10 min
Ketamine	1–2 mg/kg IV	1–2 min	5–10 min
Dexmedetomidine	1–2 mcg/kg IV over 10 min Maintenance infusion 0.3 mcg/kg/hr –0.6 mc/kg/hr	15 min	4 h
Etomidate	0.2 mg/kg IV	<1 min	3–5 min
Ketofol	(1:3) 10 % ketamine 0.5 ml + 1 % propofol 15 ml–0.05 ml/kg followed by 0.025 ml/kg for subsequent doses	1–2 min	10 min

Advantage

Morphine is useful in acute and chronic pain. Prolonged duration of action useful in procedures where pain is expected to remain after procedural sedation (PS).

Side Effects

- Respiratory depression
- Bradycardia – (increased activity over the vagal nerves)
- Hypotension – (due to histamine release)
- Nausea and vomiting
- Pruritus
- Urinary retention

Dosage: 0.1 mg/kg IV

Caveat: Select small dosage for infants and elderly as they are more prone for respiratory depression.

Fentanyl

- Semi-synthetic opioid, phenylpiperidine derivative
- Has few advantages over morphine – rapid onset of action, shorter duration of action and minimal cardiovascular effects

Dosage: 1–2 mcg/kg IV

Benzodiazepines

Ideal agent for minimal sedation. This group of drugs has anxiolytic, sedative, amnesic and anticonvulsant properties and also produces some degree of muscle relaxation. The disadvantage is that they have no analgesic effect. In painful procedures an analgesic drug should be used along with benzodiazepines.

Midazolam is the most commonly used benzodiazepine for PS.

Advantages

- Faster onset of action
- Provides complete amnesia
- Less pain on injection
- Faster recovery
- Safe in day-care procedures

Side Effects

Produces dose-dependent decrease in ventilation, decrease in blood pressure and increase in heart rate. COPD patients have greater midazolam-induced depression of ventilation.

Dosage: 0.05–0.1 mg/kg IV

Propofol

It is a substituted isopropyl phenol compound. 1 % solution in an aqueous solution of 10 % soya bean, 2.25 % glycerol and 1.25 % egg phosphatide. A general anaesthetic agent which produces dose-dependent sedation. It has also antiemetic, antipruritic and anticonvulsant properties [13].

Advantages

- Rapid onset and rapid recovery
- Suppresses the presser response in intubation and other invasive procedures

Disadvantages

- It has no analgesic property and should not be used as a single agent in painful procedures.
- It produces severe or excruciating pain with injection [14, 15]. Pain can be reduced by selecting a larger vein or by prior administration of 1 % lidocaine or a potent short-acting opioid into the vein [16].

Side Effects

- Produces dose-dependent depression of ventilation
- Hypotension
- Allergic reactions
- Strongly supports bacterial growth of *Escherichia coli* and *Pseudomonas aeruginosa*. It is recommended that:

  (a) An aseptic technique should be used in handling the drug.
  (b) The contents of the ampoule containing propofol should be withdrawn into a sterile syringe immediately after opening.
  (c) The contents of the opened ampoule should be discarded after 6 h of opening the ampoule.

Dosage: 1–2 mg/kg IV

Caveat: not advocated in shock patients, especially trauma patients. Adequate hydration before IV administration of propofol is recommended to prevent hypotension.

Ketamine

It is a phencyclidine derivative, NMDA (N-*methyl*-d-*aspirate*) receptor antagonist.

It produces:

- Anxiolysis
- Amnesia
- Profound analgesia

Side Effects

- Produce dissociative anaesthesia which resembles a cataleptic state in which the eye remains open with a slow nystagmic gaze. The patient is non-communicative, although wakefulness may be present. Varying degree of hyper-tonus and purposeful skeletal muscle movements occur independent of surgical stimulation.
- Increased production of oral secretions. This can be reduced by administration of anti-cholinergic drugs.
- Hypertension and tachycardia.

Advantages

- Can be used safely in hypotensive patients.
- Maintains pharyngolaryngeal reflexes.

Disadvantages: Emergence Delirium [11, 12]

Emergence from ketamine anaesthesia may be associated with visual, auditory and proprioceptive illusions which may progress to delirium. Dreams and hallucination can occur up to 24 h after administration of ketamine. The emergence phenomenon can be prevented by prior administration of IV benzodiazepines about 5 min before administrating IV ketamine.

Caveat: It should not be used in hypertensive and psychiatric patients.

Dosage: 1–2 mg/kg IV

Dexmedetomidine is a highly selective α2 adrenergic agonist [6].

It provides sedation, anxiolysis, analgesia and sympatholysis. Sedation produced by α2 agonist is unique in the sense that patients can be easily aroused during the procedure, can respond to verbal commands and then can return to sleep like state when not stimulated. Sedation is dose dependent.

Advantages

- Rapid onset of action
- No respiratory depression
- Attenuates haemodynamic response to tracheal intubation

Side Effects

- Hypotension
- Bradycardia

- Sinus arrest
- Dry mouth

Caveat: Use with caution in patients with advanced heart block or severe ventricular dysfunction and in patients getting other vasodilators or negative chronotropic agents. Care should be taken in elderly patients and in patients with impaired renal and hepatic functions.

Dose

Loading dose 1 mcg/kg IV over 10 min followed by maintenance infusion 0.3 mcg/kg/h IV – 0.6 mcg/kg/h IV [7]

Etomidate

It *is* an ultrashort-acting non-barbiturate hypnotic used for anaesthesia [18].

Advantages

- Rapid onset of action fast
- Minimal cardiovascular respiratory effects
- No histamine release

Disadvantages

- No analgesic property
- Pain on injection

Side Effects

- Nausea
- Vomiting
- Myoclonus

Dosage: 0.2 mg/kg IV

Ketofol – combination of ketamine and propofol [8, 9]

Studies have shown combination of these two agents has several benefits – haemodynamic stability, lack of respiratory depression, good recovery and potent post-procedural sedation. Ketofol has been used in different ratios (1:1, 1:2, 1:3 and 1:4), and it was found that ketofol in a ratio of 1:3 is ideal for bolus administration and it has shortest emergence time and long antiemetic duration [10]. To prepare ketofol 1:3, add 10 % ketamine 0.5 ml to 1 % propofol 15 ml in a 20 ml syringe.

Dose

Children – 0.1 ml/kg initially followed by 0.05 ml/kg at 2 min then 0.025 ml/kg for subsequent doses

Adult – 0.05 ml/kg followed by 0.025 ml/kg for subsequent doses

Nitrous Oxide: is an inhalation agent used along with oxygen in a ratio of 50:50. Used mainly in dentistry and labour analgesia. To get pain relief, a local anaesthetic will be administered in combination with nitrous oxide [5].

Caveats

- In painful procedures combine an analgesic agent to the sedative agent.
- When two drugs are combined, the dose of the individual drugs should be reduced.
- Combination of drugs provides better procedural sedation with minimal side effects.
- In the event of adverse reaction, use the reversal agent to antagonise the side effects.

Reversal Agents – antagonises the side effects of the drugs (Table 28.2).

Table 28.2 Reversal agents

Drug	Antagonist drug	Dose adult (mg)	Repeat dose	Dose children	Duration of action (minutes)
Opioids	Naloxone	0.2–0.4 IV	0.2 mg	IV or IM 0.1 mg/kg dose 2 mg	IV 20–40
					IM 30–60
Benzodiazepines	Flumazenil	0.2 IV	0.1 mg/min	0.02 mg/kg IV 1 mg	IV 30–60

Procedural Sedation in Paediatric Patients

Procedural sedation is very useful in children as they are very scared and apprehensive. Even examination may be difficult, procedural sedation can be provided in such situation and in performing diagnostic procedures like lumbar puncture and change of burn dressing.

- The selection of sedative and analgesic agents differs slightly when these agents are used in children. Chloral hydrate, barbiturates, benzodiazepines and propofol are used in children. Since none of these agents provide analgesia, for painful procedures an opioid analgesics or ketamine may be used.

- Midazolam can be administered via various routes, e.g. oral, intranasal, rectal, intramuscular and intravenous so this carries an advantage in paediatric patients.
- Age – appropriate equipment for measuring blood pressure and oxygen saturation.
- A crash cart with age appropriate drugs and equipment.

Dosage Guidelines for Paediatrics: Table 28.3

Table 28.3 Dosage guidelines for paediatrics

Drug	Paediatric dose	Comments
Midazolam	Intravenous IV 0.05–0.1 mg/kg. Not to exceed, i.e. total cumulative dose of 0.4 mg/kg or 6 mg	Reduces 30–50 % if combined with opioid analgesia younger children <5 year may require high dose up to 0.6 mg/kg/dose
	Intramuscular 0.1–0.2 mg/kg, 30–40 min before procedure	
	Oral 0.25–0.5 mg/kg, 30–45 min before procedure Intranasal 0.1–0.6 mg/kg/dose, inhaled intranasal 10 min before procedure Rectal 0.5 mg/kg/dose administered PR 30–45 min, before procedure	
Fentanyl	1 mcg/kg/dose I/v If needed, you may repeat by 1 mcg/kg not to exceed total cumulative dose of 4 mcg/kg	Provides analgesia for painful procedures increased risk of respiratory depression when combined with sedatives
Ketamine	Intravenous 1–2 mg/kg 0.25–1 mg/kg every 10–15 min administer slowly not to exceed 0.5 mg/kg/min Intramuscular 2–5 mg/kg/dose Oral 6–10 mg/kg/dose mixed in some drink – 30 min before procedure	Provides excellent sedation and analgesia Increases salivary secretions Increases heart rate and blood pressure, intracranial pressure, emergence hallucinations severe in older children
Propofol	Use in paediatric data limited 1–1.5 mg/kg/iv loading dose 0.25–0.5 mg/ kg iv every 3–5 min	Provides rapid sedation
Chloral hydrate	25–75 mg/kg/dose – PO/PR Administer 30 min before procedure	

Guidelines for Performing Procedural Sedation and Analgesia

1. Obtain proper history:
   - Co-morbidities
   - Drug allergies
   - Current medications
   - Time and description of last meal
2. Physical examination with special attention to airway evaluation.
3. Check the equipment:
   - Suction apparatus
   - Oxygen source
   - Airway management devices of appropriate size
   - Resuscitation equipment
4. Availability of drugs:
   - Sedatives and analgesics
   - Resuscitation drugs
   - Reversal agents for opioid and benzodiazepines
5. Intravenous access.
6. Monitoring:
   Continuous monitoring of the following parameters during and after procedural sedation is mandatory. Monitor the following:
   - ECG
   - Pulse rate
   - NIBP
   - Saturation
   - Respiratory rate
   - Oxygen saturation
   - Capnography (end tidal CO2) [17]
7. Personnel:
   - Emergency physician – administers the drug and performs the procedure.
   - Qualified nurse – continuously monitors the patient and assists the provider in case of an adverse event.
   - Emergency physicians should coordinate procedures requiring procedural sedation and analgesia with the ED staff.
8. Counselling and consent.
   Explain the procedure to the patient and relatives and obtain an informed written consent for the procedural sedation.
9. Documentation.
   All the details should be documented properly, i.e. patient's ID, name of the drug dose, route of administration, vitals, time and administration complications if any etc.

Discharge criteria

After procedural sedation – if the patient's condition enables him to get discharged, the following criteria should be fulfilled before discharge from ED:

- Awake and alert
- Able to walk without difficulty
- Normal vital signs
- No bleeding, no complication related to the procedure
- Adequate analgesia
- Post-procedure investigations done (chest x-ray after reduction in case of a dislocation of a joint)
- Able to tolerate oral feed

On Discharge

- Send the patient home with a responsible person.
- Give instructions – not to involve in dangerous activities like driving, operating machinery.
- Written discharge summary.

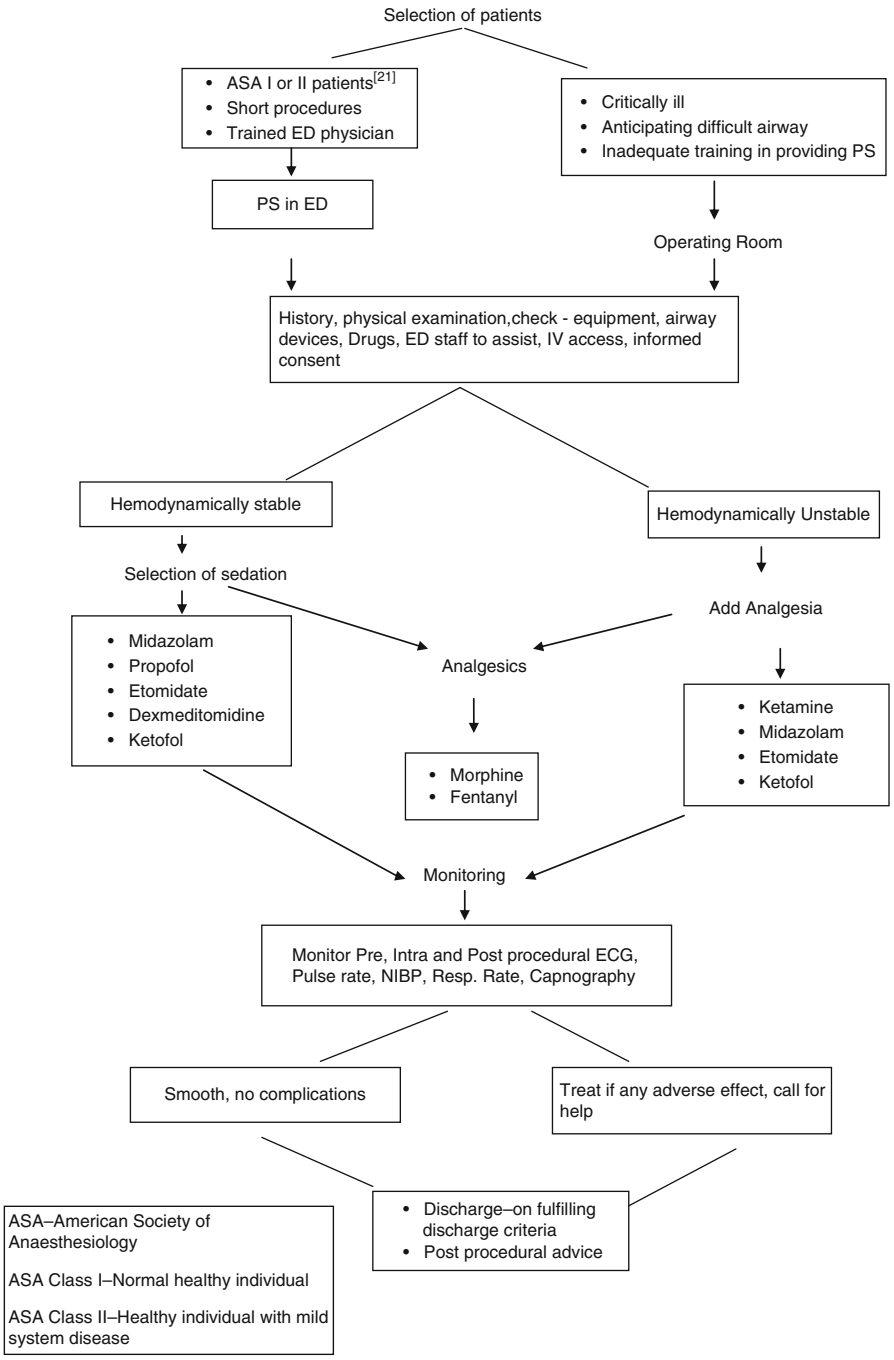
Fasting Guidelines for PS [19]

- The risk of aspiration during PS in the ED is low as the patients are put in a moderate level of sedation and are able to maintain the protective reflexes.
- The time of fasting recommended for elective procedures is 3 h, whereas in emergency procedures PS may be performed regardless of fasting time.
- The timing and size of the last meal should guide drug choice and depth of sedation.
- If the ED physician anticipates any concerns regarding the management of airway in special situations, they should discuss with the consultants and such procedures can be done under general anaesthesia in the operation theatre.

Prerequisites for emergency physicians to provide procedural sedation and analgesia:

- Appropriate and adequate training and skills in providing procedural sedation
- Adequate knowledge of the dosage, effects and side effects of the drugs used
- Able to maintain airway if there is a need arise
- Able to perform CPR in case of adverse events

Algorithm for procedural sedation in adults



Conclusion

Procedural sedation in the ED is safe, provides pain relief and is beneficial. While providing PS emergency, the physician should be vigilant and detect the adverse effects and treat them urgently.

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Chapter 29
The Diabetic Foot

Magdy Moawad

Key Points
- Patients with diabetic foot may present as an emergency with sepsis (with or without ischaemia); they may also present with tissue loss.
- A prompt diagnosis, clear pathway, management plan as well as urgent surgical intervention will cut back complications and reduce the risk of amputation.
- Multidisciplinary team approach is optimal, to ensure holistic management.

Epidemiology

The global prevalence of diabetes mellitus is 5.1 % and is anticipated to be 7.7 % by 2030 [1]. About 15–25 % of diabetic patients are expected to develop diabetic foot infection and ulceration. Whilst 60–80 % of them will eventually heal, 5–24 % will end with amputation.

Pathogenesis of Foot Problems

Neuropathy

Factors that predominantly contribute to the development of diabetic foot are peripheral neuropathy and peripheral vascular disease. More than 60 % of foot ulcers are due to primary neuropathy, which affects all components of the nervous system: sensory and motor fibres and the autonomic system.

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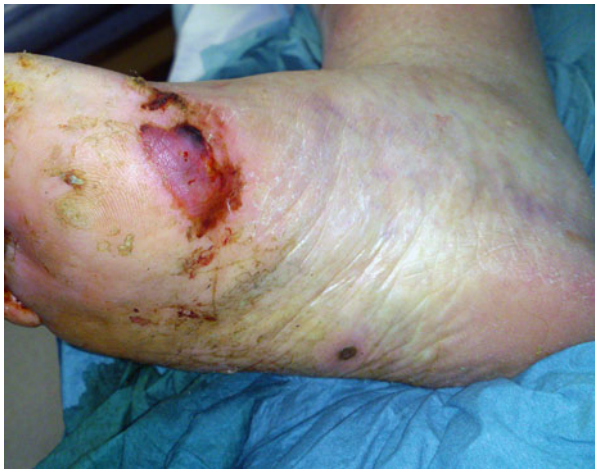

Fig. 29.1 Cellulitic foot with purplish discolouration on the planter surface of the first metatarsal head indicating subcutaneous necrosis. Note the presence of an ulcer with a halo of erythema on the lateral margin of the foot that is also the portal of entry for infection

(a) *Sensory neuropathy* affects both type A myelin fibres which are responsible for proprioception and pressure sensation and type C sensory fibres which are responsible for pain. This loss of protective sensation increases the risk of foot ulceration. Skin damage following any minor trauma will lead to foot infection and abscess formation that eventually leads to ulceration (Fig. 29.1).
(b) *Motor neuropathy* results in atrophy of interosseous and lumbricalis muscles that leads to claw deformity of the toes and arch of foot. These deformities lead to areas of high pressure at the head of the metatarsal bones that may not be noticed by patients as they usually have sensory loss.
(c) *Autonomic neuropathy* leads to altered blood flow regulation and diminished sweating which is responsible for dry skin and fissures. Consequently, the feet are rendered prone to infection and ulcers.

Arterial Insufficiency

Although diabetic patients have warm swollen feet, they have reduced capillary flow as a result of the microangiopathy, which causes arteriovenous shunts. Atherosclerosis in diabetic patients affects the crural vessels rather than the proximal vessels. The compromised blood flow to the feet may result in an ischaemic ulcer or gangrene.

Management

Evaluation

Problems of the diabetic foot can be serious, resulting in limb loss and – in the worst-case scenario – loss of life. Patients may present with symptoms and signs limited to the foot or with systemic problems. Patients could present with sepsis

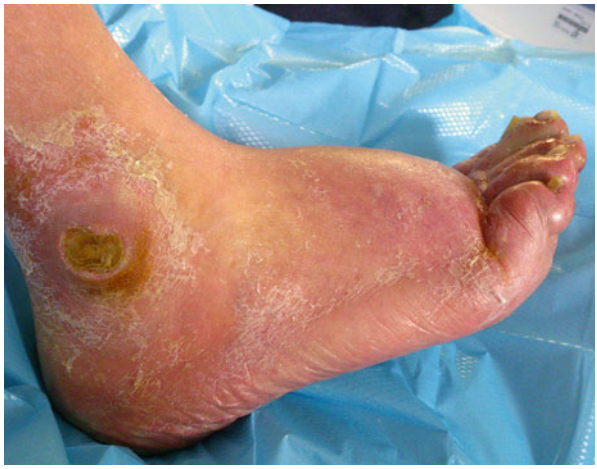

Fig. 29.2 An infected neuropathic ulcer over a lateral malleolus seen on the right foot

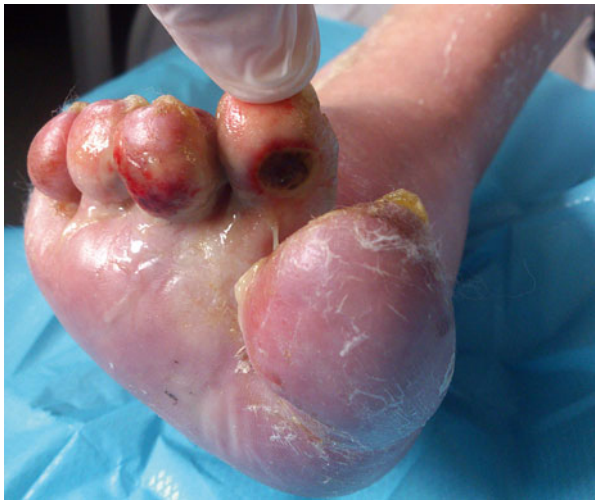

Fig. 29.3 A black neuro-ischaemic ulcer on the medial side of the right second toe caused by tight shoes. Careful inspection of the areas between the toes is essential

and/or tissue loss. A detailed history especially of recent trauma or systemic disease such as renal or cardiovascular problems should be obtained.

Clinical Features

Patients with foot infection may complain of pain, swelling, discharge and offensive odour (Fig. 29.2). They may also have ulceration or tissue loss. In septic cases other systemic symptoms and signs of infection such as poorly controlled hyperglycaemia, nausea, vomiting, fever, etc. will exist.

A thorough lower limb examination is mandatory (Fig. 29.3). The examination should include full vascular and neurological assessment as well as assessment of

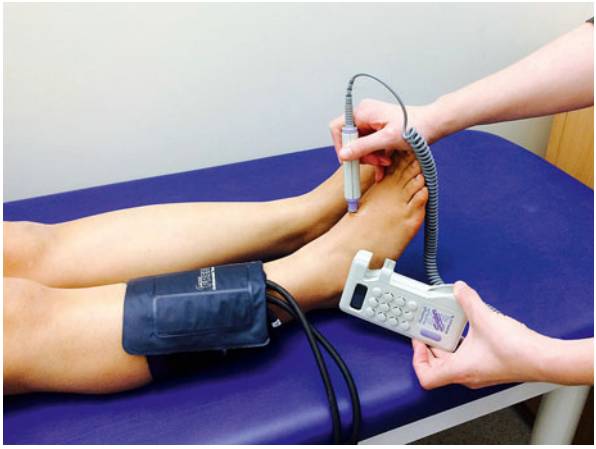

Fig. 29.4 Measurement of the dorsalis pedis systolic blood pressure. A blood pressure cuff is placed around the calf, and the blood pressure is measured with a Doppler probe over the dorsalis pedis artery. Note the Doppler probe angle of approximately 60°

the foot anatomy for deformities. They could have lower limb ischaemia or, at times, normal blood flow. If dorsalis pedis and posterior tibial pulses are not palpable, ankle-brachial pressure index (ABPI) should be calculated using a Doppler probe (Fig. 29.4). The systolic blood pressure is measured in the brachial, posterior tibial and dorsalis pedis arteries; then, the highest ankle pressure is divided by the brachial pressure. This ratio is called the ABPI (normal 0.9–1.3). The ABPI might be high if the vessels are calcified and noncompressible. Transcutaneous oximetry and toe pressure will be helpful in these circumstances.

Investigations

Baseline blood tests including full blood count, blood glucose level, C-reactive protein, erythrocyte sedimentation rate, electrolytes, renal function tests and blood culture are to be initiated.

Imaging in the Diabetic Foot

Many modalities are optional for imaging the diabetic foot. The aim of imaging is to detect early changes of osteomyelitis and infection. Although a plain X-ray is useful in detection of any foreign body or gas in the soft tissue, it has lower sensitivity (40–60 %) and specificity rates (60–90 %) in early detection of osteomyelitis [2]. Ultrasonography is useful in detection of any fluid collection to differentiate between reactive collection and collection resulting due to infection. It also plays an important role in guided aspiration procedures for culture and sensitivity [3]. Currently, magnetic resonance imaging is the modality of choice in detecting osteomyelitis (sensitivity and specificity >80 %) in diabetic patients with foot swelling

[4, 5]. Duplex scanning of the arterial system will provide valuable information about the blood flow and patency of the vessels. Although angiography is the gold standard for investigating the ischaemia, it carries risk of contrast media nephropathy, especially in diabetic patients. On the other hand, MR angiography is useful for assessing occlusive disease and will help in planning for further management of the lower limb ischaemia.

Treatment

Multidisciplinary team approach is optimal to address the multidimensional complications of diabetes. The principles of management include antibiotic treatment and surgical debridement or amputation in conjunction with medical stabilisation. If the patient has underlying lower limb ischaemia, management of the foot infection will take priority over any revascularisation procedure.

Antibiotic Treatment

Although the choice of antibiotic treatment should be based on the culture and sensitivity results, early empirical broad-spectrum parenteral antibiotic treatment is recommended. The antibiotic of choice is usually based on severity of the infection and the expected pathogen [6]. Mild infection is often due to gram-positive *Staphylococcus aureus* bacteria only [7, 8]. On the contrary, severe infection is usually caused by polymicrobial pathogens (mixed gram-positive, gram-negative and anaerobic bacteria) [9, 10]. Methicillin-resistant *Staphylococcus aureus* (MRSA) infection is isolated in 30–40 % of patients [11]. The presence of resistant bacteria species is associated with treatment failure and, consequently, high risk of amputation.

Surgical Management

Surgical management of diabetic foot complications is dependent on the viability of the foot and leg. The aim of early debridement is to assess the extent of infection and bone destruction as well as the amount of bleeding from the wound edges [12–14]. Drainage of any collection, opening of all the infected tracks and excision of all non-viable and slough tissue are crucial. Intraoperative bone and deep tissue cultures should be sent for microbiology and histopathology. It is not uncommon that patients may require multiple sessions of surgical debridement to ensure satisfactory control of infection. Trans-metatarsal amputation is essential, if there is extensive planter infection or forefoot gangrene. Below-knee amputation is recommended in patients with extensive bone destruction and infected non-healing ulcers, as a result of extensive neuroarthropathy (Charcot foot).

Wound healing after surgical debridement is quite challenging. Numerous studies illustrated the value of negative-pressure wound therapy in assisting the wound healing of the diabetic foot [15, 16]. After wound stabilisation skin graft, flaps or reconstruction may also be required to achieve satisfactory functional lower limb [17].

Revascularisation

In patients with ischaemia, prompt revascularisation is imperative to improve clinical outcome and limb salvage rate. Most diabetic patients have coexisting comorbidities; therefore, they are at high perioperative risk [18]. Percutaneous transluminal angioplasty is currently the procedure of choice for the treatment of any significant stenotic or occlusive atherosclerotic lesions [19].

Aftercare and Prevention

Following complete wound healing and recovery from foot surgery, patients are likely to develop foot deformity. Appropriate footwear is essential to avoid high-pressure areas and developing recurrence of foot ulceration and consequent complications. Patients should be reviewed on a regular basis by a multidisciplinary team to avoid risk of amputation.

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Chapter 30
Wound Care

Abraham Mathew

Key Points
- Classification of wounds helps to understand the management of them.
- Proper debridement and dressings in the initial phase of treatment help to hasten wound healing.
- Various wound dressing techniques can be adopted depending on the type of wound.

Introduction

Wounds are one among the common reasons for a patient coming to the emergency department. The management of wounds has been as old as humanity. There are many causes of wounds, trauma being the commonest. The wounds can be acute or chronic. This chapter will highlight on the different aspects mainly pertaining to the acute wound as well as touch upon briefly about chronic wound.

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Classification of Wounds

There are many ways to classify wounds. The easiest would be the Rank and Wakefield classification [1] into tidy and untidy wounds. Tidy wounds are wounds caused by a sharp instrument, thereby having less devitalised tissue and minimal tissue loss. On the other hand, untidy wounds are caused by various forces like crushing, tearing, etc. resulting in a lot of devitalised tissue and tissue loss; also there will be contamination. The other types of classification [2] include:

1. Based on aetiology – trauma (sharp/blunt), iatrogenic, burns, bites and pressure ulcer
2. Based on length of time – acute (<6 weeks) and chronic (>6 weeks)
3. Based on depth – superficial (above dermis) and deep (traverses sub dermis)
4. Based on integrity of integument – closed wounds (contusion, haematoma) and open wounds (incised, lacerated, puncture, avulsed, burn)

Physiology of Wound Healing [3]

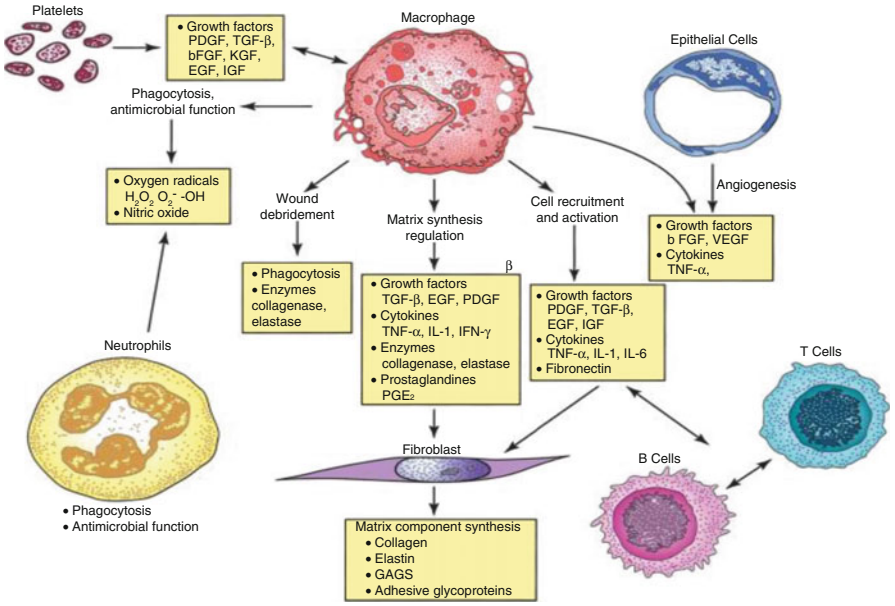
There are three phases of wound healing after the haemostasis: inflammation, proliferation and maturation.

Haemostasis is not strictly a phase of healing but is the initial event. The fibrinous clot is formed which helps to preserve the integrity of the circulatory system and limit blood loss. Platelets play a major role in initiating the coagulation cascade. Fibrinolysis then sets in to dissolve the clot for allowing cell migration into wound space which initiates the next phase. The release of cytokines by platelets, viz. PDGF, TGFβ and platelet factor IV attracts inflammatory cells, i.e. the neutrophil and macrophage. Simultaneously, vasoactive amines are released from injured tissue and platelets which increase vascular permeability. These are histamine, serotonin and prostaglandins.

Inflammation – In this phase, there is destruction of bacteria and removal of devitalised tissue by neutrophils and macrophages. Pro-inflammatory cytokines that are released by the neutrophils are TNFα, IL1, IL6, IL8 and INF α. This promotes further neutrophil and macrophage inflow. There are anti-inflammatory cytokines released subsequently once the inciting factors are removed. These are IL4 and IL10. In the latter half of this phase, macrophages play an important role. They phagocytose bacteria, release MMP (matrix metalloproteinases) and release growth factors. The MMP help dissolve the damaged

extracellular matrix and aid cell movement and thus help to stimulate fibroblasts, endothelial cells and keratinocytes.

Diagram – the key players in the process of wound healing [4]



Proliferative phase – In this phase, a scaffolding is laid for the repair of the wound through angiogenesis, fibroplasias and epithelialisation. Granulation tissue formation is characteristic of this phase. This is composed of a capillary bed, fibroblasts, macrophages and a loose arrangement of type III collagen, fibronectin and hyaluronic acid. The onset of wound contraction marks the final part of proliferation.

Maturation phase is also called remodelling phase as there is a maturation of collagen type I collagen is formed replacing type III collagen until a ratio 4:1 is achieved. There is decreased wound vascularity and wound contraction due to fibroblast and myofibroblast activity.

Hence, normal wound healing is a dynamic process with complex interactions between the extracellular matrix, controlled angiogenesis, epidermal and dermal cells and soluble mediators. The understanding of this will be useful in applying various treatment options to promote its healing.

The Concept of Wound Bed Preparation and the TIME Model [5, 6]

Wound bed preparation is a holistic approach to wound care by detecting the phase of wound healing which has been affected in the wound and to treat accordingly hence creating an optimal wound healing environment. The TIME model was an algorithmic approach to this effect.

1. Tissue management, i.e. debridement.
2. Inflammation and infection control.
3. Moisture balance – Moist environment promotes wound healing, i.e. by wet dressings.
4. Epithelial (edge) advancement. – Skin cover techniques promote epithelialisation.

In general, wound bed preparation is more important in the chronic wound management.

The Paradigm of Biofilm in Wound Care

There is evidence to a formation of a biofilm over the raw area in wounds which predisposes to infection and chronicity of the wound [7]. A biofilm is a collection of microbial cells that are attached to a surface and embedded in a self-produced extracellular polymeric substance. In acute wounds, they are seen to organise around blood vessels, and in chronic wounds, they form in the extracellular matrix of the wound bed itself. Debridement removes the biofilm but it reaccumulates and reestablishes gradually. They are highly resistant to environmental stress and host immunity and mechanically prevent wound contracture. *Pseudomonas aeruginosa* and gram-positive cocci colonies have been seen in biofilms. Research shows that the repeated debridement and removal of biofilms and topical antibiotics may be useful to promote healing [8].

Management of the Acute Wound

The primary survey is done as per the ATLS protocol. Following this, a careful history and a meticulous examination with documentation are the initial step in wound care management. It helps to suspect the presence of foreign bodies and to get an idea of deeper penetration and the amount of contamination to expect.

Administration of Tetanus Prophylaxis

If the patient is fully immunised in the past but has not received booster for 5 years, then he/she should receive 0.5 ml of tetanus toxoid IM. Any patient who has not received full course of immunisation should not only receive tetanus toxoid but also receive passive immunisation with 250 units of human immune globulin IM or 4 units/kg body weight in paediatric population.

Wound Cleansing

Cleansing of the wound with copious saline irrigation/pulsed irrigation helps to remove much of the particulate contaminants as well as expose hidden areas. Various solutions have been recommended for irrigation though normal saline is conventionally favoured. Randomised control trials have shown that there is no evidence to show that using potable tap water to cleanse acute wound increases infection [9].

Irrigation with antibacterial preparations like iodine, hydrogen peroxide, etc. have been shown to impair wound healing and hence avoided. Ingrained dirt that does not come out with irrigation will have to be removed by debridement.

Local anaesthetic infiltration, i.e. lignocaine (0.5–1 %) combined with $1:10^5$–$1:2\times10^5$ dilution of adrenaline, can be done in the wound edge to facilitate further exploration.

If there is a chance of deeper penetration close to vascular structures, the wound may be occluded with a sterile pad and further exploration done only in the operation theatre.

After cleansing, irrigation and anaesthetising the wound, the area surrounding the wound should be prepared by hair clipping, cleaned with povidone iodine and draped with sterile towels.

Haemostasis – It is the next step in wound care. Irrigation usually opens up collapsed or constricted cut ends of vessels, and the surgeon can see the bleeding and control it.

The three main techniques for haemostasis are compression, ligation and thermal coagulation.

Packing a bleeding cavity and compression by applying pressure for 5 min promotes normal haemostasis. Small- and medium-sized vessels can be controlled by causing thermal coagulation with unipolar or bipolar diathermy. Application of a tourniquet maybe helpful if the bleeding is fast and not receding. Larger vessel cut ends can be caught with haemostat and ligated or clipped. Main vessel injury has to be compressed with packing and referred to the vascular surgeon.

Debridement [10] (Surgical Debridement or Sharp Debridement)

It is the excision of all grossly dead and damaged tissues with the help of sharp dissection. If it is difficult to assess the viability, the wound is left open and inspected at intervals before closure. In late and infected wounds, debridement is done and wound is left open.

In debridement, first the ragged skin margins are excised followed by the surface of the wound. All intermuscular planes are inspected for possible contaminants. Under a tourniquet, healthy tissue is bright and homogenous. If muscles are avulsed proximally and hanging from the tendon, mostly it has to be excised. Vessels and nerve are safeguarded and carefully debrided, if they have adjacent unhealthy tissue. Avulsion of skin can lead to degloving injury – here it is difficult to assess the viability. If the margin does not bleed on incising, then mostly the skin won't survive.

Disrupted tendons can be sutured or fixed with a suture to the surrounding tissue for a second-stage reconstruction. Exposed bones are irrigated and mechanically cleansed with bone rongeurs. Nerves and main vessels are not excised but are skeletonised from the damaged tissues. While performing under the tourniquet, it is important to release it after debridement and secure haemostasis. Avulsed skin can be reassess 48 h to confirm viability.

Other Methods of Debridement

- Mechanical debridement: This uses wet dressings over which dry dressings are used. On removal of the dressing, the slough gets stuck to the gauze and hence it is removed. This is however a painful process.
- Hydro-surgical debridement (pulsed lavage treatment) [11]:
  In patients with a large amount of slough in the wound, a pulsed lavage and suction device is used. A jet of sterile saline under high pressure is made to strike tangentially to the tissues. Protection from the mist of the spray has to be done for both patient and doctor.
- Enzymatic debridement:
  Proteolytics, fibrinolytics and collagenase are some enzymes that can be used to digest necrotic tissue – the application is usually in chronic wounds with a tough eschar. Here the eschar has to be scored or crosshatched to facilitate its entry.
- Autolytic debridement:
  In this method, an occlusive dressing is applied to keep wound watertight. This creates a moist wound bed environment helping the body's enzymes to break down necrotic tissue. The materials used for this are hydrogel, hydrocolloid and collagen.
  In comparison to enzymatic debridement, autolytic debridement has been shown to be more expensive [12].

- Maggot debridement therapy (MDT, biotherapy or larval therapy) [13]:
  There is medical evidence now demonstrating the safety and efficacy of maggot therapy for a variety of problematic wounds. This is also relevant when the antibiotic therapy is increasing becoming ineffective due to the rise of multidrug resistant bacteria. Ironically, the benefit of maggots in wounds was first noted in the wounds of soldiers abandoned in World War I. Today as it stands, MDT is the application of a safe and effective species of maggots to cause a controlled therapeutic myiasis, i.e. infestation on a live host. They are placed in a special dressing in contact with the wound for 48–72 h and then removed when they are satiated and full. The treatment has been reported to have 40–50 % success in salvaging limbs from amputation [14].

Infection Control

Prophylactic antibiotics have a role in wound contaminated with intestinal contents, e.g. penetrating abdominal wounds. In late wounds, parenteral antibiotic therapy is useful especially if the wound is closed primarily after debridement. The selection of antibiotic is based on the organisms suspected to be found within the wound. Multiple agents can be used when there is enteric contamination or when the patient has a poor immune status due to co-morbidities.

Dressings

A landmark research by Winter on plastic dressings done in pigs [15] proved that moist dressings increased wound healing two to three times. This led to the concept of moist dressings in contrast to the classical gauze dressings. A moist environment has been shown to promote epithelialisation from the periphery to the central aspects of the wound in a faster pace. Gauze dressing on the other hand causes desiccation and scab formation, and hence epithelialisation occurs at a slow pace as cells have to migrate under the scab searching for a moist area to migrate. Moist dressings have been seen to promote the 'autolytic' debridement where inflammatory cytokines help to destroy bacteria.

Examples of moist dressing materials are transparent films like Opsite™, a polyurethane membrane, hydrocolloid hydrogel and foam dressings.

Negative Pressure Wound Treatment (NPWT) or Vacuum-Assisted Dressings

This is becoming increasing popular in acute wound care scenario. Earlier it was done for chronic wounds where it has proven to be very effective. NPWT acts by sucking the excess fluid at wound site and helps improve microcirculation and reduce bacterial

load. At the microscopic level, it promotes collagen synthesis, angiogenesis and granulation tissue. It is not useful when wounds are very contaminated or infected [16].

Topical Agents in Wound Care

1. Antimicrobials
   These can be antiseptics and antibiotics.
   The common antiseptics are 10 % povidone iodine, sodium hypochlorite solution (Edinburgh University solution of lime, EUSOL) and acetic acid. These solutions are best used for surgical site cleansing of intact skin. They are detrimental to fibroblasts and epithelium. If used, they have to be diluted at least 1:10 times in saline. Antiseptics act by contact destruction of the pathogenic organisms.
   The topical antibiotics recommended for use are mupirocin, neomycin, bacitracin and topical silver agents.
2. Growth factors
   There are two classes of growth factors clinically available for wound care.
   Recombinant human platelet-derived growth factor and epidermal growth factor. They are mainly used for chronic wounds which have no slough in them. There have been concerns of the risk of malignancies with these agents.
3. Matrix-forming agents
   Pure collagen, hybrid collagen products and collagen-containing dressings are the examples. They are based on the fact that collagen forms an important content if the connective tissue hence favours the deposition of new tissue and attracts cells for healing.
4. Enzymes
   These are used again in chronic wounds and are useful for enzymatic debridement of the dead and necrotic tissue. Examples are collagenase and papain-urea combination.
5. Medical honey [17]
   Current evidences are showing the benefits of gamma-irradiated sterile honey. Its antibacterial action is due to its low pH, anti-protease activity and its hygroscopic action. It also prevents malodour from cancer-related wounds and shows increased healing in multidrug resistant bacteria like MRSA. It is also useful in neonatal wounds.

Pain Management in Wound Care

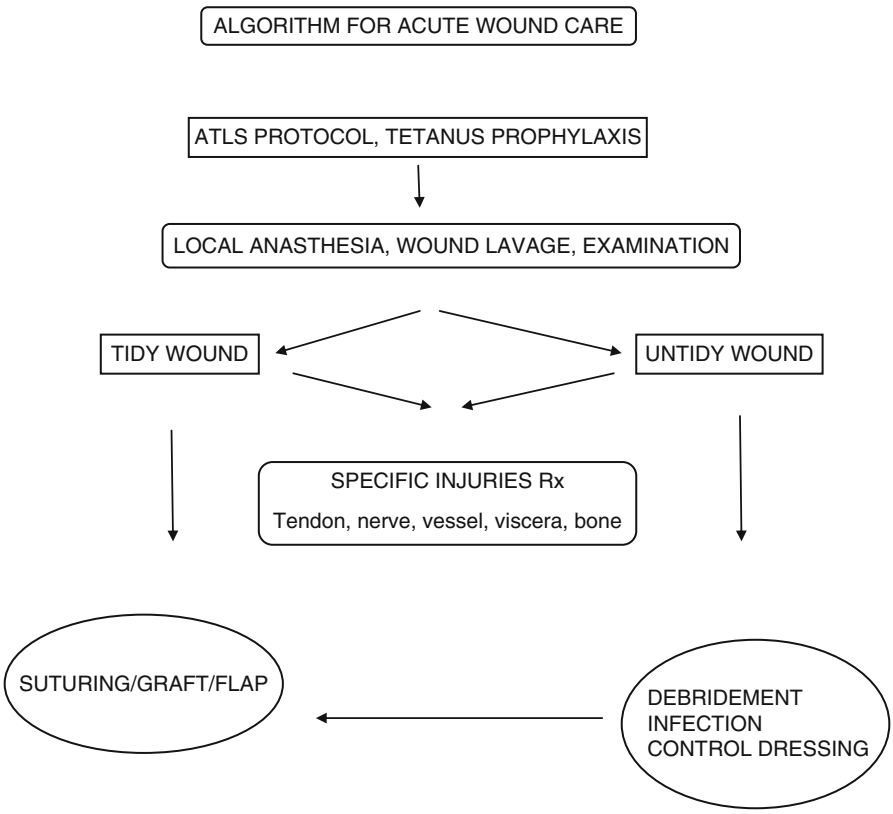
Irrespective of whether a wound is acute or chronic, pain is a major problem in wound management. A large prospective study of the prevalence of pain in wound care, the MAPP study, showed that the maximum pain is during dressing removal. There was a 95 % reduction in pain in acute wounds when the dressing material was changed to a non-adherent hydrocolloid dressing [18].

Wound Closure Techniques

A tidy wound can be closed primarily with sutures after the initial treatment and when there is minimal skin loss. For untidy wounds presenting late, we have to consider the 'TIME' model giving due weightage to debridement and dressing. Injured deeper structures like tendon, nerve, vessels and viscera have to be treated specifically. Once the wound starts to granulate and contract, skin grafting or flap cover techniques can be utilised. In specific wounds following burns, early excision and grafting are beneficial. Punctured wounds have to be treated more carefully because of the risk of deep-seated contamination.

Conclusion

Hence, the success behind managing wounds includes various aspects, principled around wound healing, local and general therapy, proper and repeated debridement, selection of appropriate dressing and wound cover techniques.



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Part V
Toxicology

Chapter 31
Approach to Acute Poisoning

S. Senthilkumaran and P. Thirumalaikolandusubramanian

Key Points
- History is notoriously unreliable in patients with intentional (suicidal) overdose.
- Some ACLS drugs may be dangerous in patients with poison-induced cardiac disorders [1].
- Intentional overdose in a child or adolescent should raise the possibility of physical or sexual abuse.
- Pregnancy test to be done in any young woman with drug overdose or poisoning.
- Consult a regional poison center or toxicologist if not completely familiar with the exposure agent.

Introduction

- Poisoning is a common emergency department presentation.
- Patients with poison exposure and toxicity may present with a wide spectrum of clinical presentations and problems [2].

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- General principles can be applied as a framework in a logical way to treat most poisonings, so that potential morbidity and mortality can be prevented.
- Only less than 5 % of poisonings requires the use of specific antidotes [3]; meticulous general supportive care is the most important approach in caring for most poisoned patients.
- For effective management of an acutely poisoned victim, five structured, systematic steps are required. These are:
  - *Resuscitation and initial stabilization*
  - *Risk assessment of poison*
  - *Reducing absorption* and *removal of toxin*
  - *Antidotes*
  - *Supportive care*

Resuscitation and Initial Stabilization

- A poisoned victim often represents an acute emergency with a broad spectrum of multi-organ system involvement. So poisoning must be viewed as a multiple chemical trauma and is a dynamic medical illness; furthermore, patients may deteriorate within a few minutes to hours of presentation [4].
- The initial phase or "primary survey" addresses supporting vital functions and identifies the toxic agent wherever possible.
- On arrival of a patient with poisoning, the initial priorities are the maintenance of airway, breathing, and circulation.
- Hypoxia and airway compromise are recognized to be significant contributing factors for deaths in intoxicated patients. Hypoxia rapidly results in end-organ damage and cardiac arrest, whereas hypercarbia precipitates cerebral vasodilatation, respiratory acidosis, and a reduced level of consciousness.
- If the patient has an altered level of consciousness, his cervical spine must be immobilized unless trauma has been excluded.
- The clinician must pay special attention in patients who are not able to maintain a patent airway due to impaired airway protective reflexes as these patients are at increased risk of aspiration because of toxin-induced vomiting, unprotected gastric lavage, or depressed sensorium.
- Endotracheal intubation is needed if respiratory insufficiency is present. Cyanosis and apnea are usually late findings. Therefore, assessment of early respiratory insufficiency by arterial blood gas (ABG) analysis is critical.
- A good vascular access should be obtained as early as possible.
- Depending on the poison, patients may present with hypotension or hypertension and bradyarrhythmias or tachyarrhythmias.

Table 31.1 Agents that cause seizures (OTIS CAMPBELL)

Organophosphates
Tricyclic antidepressants
Isoniazid, insulin
Sympathomimetics
Camphor, cocaine
Amphetamines, anticholinergics
Methylxanthines (theophylline, caffeine)
Phencyclidine (PCP)
Benzodiazepine withdrawal, botanicals (water hemlock), GHB
Ethanol withdrawal
Lithium, lidocaine
Lead, lindane

- The pathogenesis of hypotension varies and may consist of hypovolemia, myocardial depression, cardiac arrhythmias, and toxin-induced vasodilatation [5]. Treatment should be tailored, but initially, a bolus of crystalloid fluids is indicated in most instances.
- Awareness of specific poison and the mechanism producing hypotension may assist in choosing the vasopressor in refractory shock.
- After initial resuscitation, all patients with altered sensorium should receive a *cocktail* of dextrose, naloxone, and thiamine. However, this strategy is not well supported by the literature, and empiric administration of coma cocktail has been questioned [6].
- Many toxins and withdrawal syndromes may result in seizures (Table 31.1). For the toxin-induced seizure, benzodiazepine agents are the first drug of choice. If benzodiazepines are ineffective, barbiturate may be used.
- Phenytoin is generally not recommended in the severely poisoned patient as it is often ineffective and may worsen the overall toxicity of some agents [7].
- It is reported that in high doses, propofol acts as anticonvulsant by depressing both the cortex and subcortex, but in low doses, it may act as proconvulsant by inhibiting the inhibitory subcortex, which results in the "release" of normal hyperexcitability in the cortex [8].
- Occasionally, pyridoxine is required for seizures induced by isoniazid or gyromitrin mushroom poisoning.
- Investigation of other potential causes of seizure disorder such as intracranial hemorrhage or infarct through brain imaging should also be considered.

Risk Assessment of Poison

- A brief and focused evaluation should be done as soon as the life-support phase has been accomplished. The primary goal is to determine the potential severity of toxic exposure.

History

When evaluating a poisoned patient, a good approach is to:

- Identify the reason of exposure (i.e., intentional, unintentional, misadventure).
- The type of substance involved (i.e., prescription, over-the-counter, herbal, illicit drug).
- The formulation (i.e., immediate vs. sustained release).
- The dose of the substance, the amount of substance involved.
- The modes of exposure (i.e., ingestion, inhalation, topical, ocular, and others (injection, rectal, vaginal, and transplacental).
- The time of exposure (hours since exposure, acute vs. chronic).
- Any potential co-ingestion and the severity of exposure.
  - Although the history is important, it may be unreliable or incomplete particularly those who have ingested poison with a suicidal intent; hence, other people such as relatives, colleagues, friends, prehospital personnel, or previous medical records may provide crucial information to aid in management.
  - Occupational history and hobbies should also be inquired as these can result in chemical exposures.

Examination

- A thorough head-to-toe examination must be performed after the initial stabilization of the patient.
- As most deaths due to poisoning are a result of respiratory compromise; special attention should be given to the evaluation of the respiratory system.
- Based on the examination findings, it may be possible to identify the type of poison involved [9]. Recognition of a specific toxidrome (a constellation of signs and symptoms associated with a specific poisoning) is useful in managing patients with exposure to unknown substances (Table 31.2) but may be misleading in certain circumstances such as exposure to more than one poisonous substance or adulterated with another toxidrome-causing substance.
- In the absence of a classic presentation or toxidrome, separating patients with suspected poisoning into broad categories based on alteration of vital signs (Table 31.3), ocular findings (Table 31.4), and sensorium level (Table 31.5) can be extremely helpful in narrowing down potential etiologies and determine drug or toxin class.

Table 31.2 Toxidromes

Toxidrome	Site of action	Signs and symptoms
Opioid	Opioid receptor	Sedation, miosis, decreased bowel sounds, decreased respirations
Anticholinergic (hot as a hare, dry as a bone, red as a beet, mad as a hatter)	Muscarinic acetylcholine receptors	Altered mental status, sedation, hallucinations, mydriasis, dry skin, dry mucous membranes, decreased bowel sounds, urinary retention
Sedative-hypnotics	Gamma-aminobutyric acid receptors	Sedation, normal pupils, decreased respirations
Sympathomimetic	Alpha- and beta-adrenergic receptors	Agitation, mydriasis, tachycardia, hypertension, hyperthermia, diaphoresis
Cholinergic (DUMBELS)	Nicotinic and muscarinic acetylcholine receptors	Altered mental status, seizures, miosis, lacrimation, diaphoresis, bronchospasm, bronchorrhea, vomiting, diarrhea, bradycardia
Serotonin syndrome	Serotonin receptors	Altered mental status, tachycardia, hypertension, hyperreflexia, clonus, hyperthermia

Table 31.3 Diagnosing toxicity from vital signs

Bradycardia (PACED)
Propranolol or other beta-blockers, poppies (opiates), propafenone, phenylpropanolamine, anticholinesterase drugs
Clonidine, calcium channel blockers
Ethanol or other alcohols
Digoxin
Tachycardia (FAST)
Free base or other forms of cocaine
Anticholinergics, antihistamines, amphetamines
Sympathomimetics (cocaine, amphetamines), solvent abuse
Theophylline
Hypothermia (COOLS)
Carbon monoxide
Opiates
Oral hypoglycemics, insulin
Liquor
Sedative-hypnotics

(Continued)

Table 31.3 (Continued)

Hyperthermia (NASA)
Neuroleptic malignant syndrome, nicotine
Antihistamines
Salicylates, sympathomimetics
Anticholinergics, antidepressant
Hypotension (CRASH)
Clonidine, calcium channel blockers
Reserpine or other antihypertensive agents
Antidepressants, aminophylline
Sedative-hypnotics
Heroin or other opiates
Hypertension (CT SCAN)
Cocaine
Thyroid supplements
Sympathomimetics
Caffeine
Anticholinergics, amphetamines
Nicotine
Tachypnea (PANT)
PCP, paraquat, pneumonitis (chemical)
ASA and other salicylates
Non-cardiogenic pulmonary edema
Toxin-induced metabolic acidosis
Bradypnea (SLOW)
Sedative-hypnotics (including GHB)
Liquor
Opiates
Weed (marijuana)

Table 31.4 Agents that affect pupil size

Miosis (COPS)
Cholinergics, clonidine
Opiates, organophosphates
Phenothiazines, pilocarpine
Sedative-hypnotics
Mydriasis (AAAS)
Antihistamines
Antidepressants
Atropine and other anticholinergics
Sympathomimetics

Table 31.5 Agents that alter the sensorium

Antidepressants
Antihistamines
Antipsychotics
Atropine
Barbiturates
Benzodiazepines
Cyanide
Carbon monoxide
Ethanol
Organophosphates
Narcotics
Lithium

Toxicology Laboratory

- When a specific toxin (acetaminophen, salicylate iron, theophylline, and lithium) or even class of toxins (toxic alcohol) is suspected, requesting qualitative or quantitative levels may be appropriate if deemed necessary for diagnosis and treatment.
- In the patient whose history is generally unreliable or in the unresponsive patient where no history is available, the clinician may gain further clues as to the etiology of a poisoning by toxicology screening which may confirm toxin exposure but rarely alters management.
- The usefulness of a toxicology laboratory is further limited by a prolonged turnaround time and high costs involved to run such a specialized service.
- In most cases of poisoning, standard investigations (e.g., serum electrolytes, glucose, ABG, ECG, etc.) are often more rewarding for diagnostic purposes than a toxicology screen.
- Three important gaps in toxicology are the *anion gap*, *the osmolal gap*, *and the oxygen saturation gap* [10].
- The normal range of anion gap may vary from 3 to 12 mEq/L. An increase in anion gap greater than 20 mEq/L suggests lactic acidosis, uremia, ketoacidosis, or selected intoxications (Table 31.6).
- The anion gap may not be elevated in the setting of an organic acidosis if a concurrent condition that lowers the anion gap exists (e.g., hypoalbuminemia – for every 1 g/L decrease in the plasma albumin, the anion gap falls by 2.5 mEq/L).
- Many toxins do not increase the anion gap, whereas others (e.g., lithium) decrease the anion gap.
- Low molecular weight drugs and toxins increase the discrepancy between measured and calculated plasma osmolality (Table 31.7). Normal plasma osmolality is 285–295 mOsm.

Table 31.6 Agents causing an elevated anion gap (METALACID GAP)

Methanol, metformin
Ethylene glycol
Toluene
Alcoholic ketoacidosis
Lactic acidosis
Aminoglycosides, other uremic agents
Cyanide, carbon monoxide
Isoniazid, iron
Diabetic ketoacidosis
Generalized seizure-producing toxins
ASA or other salicylates
Paraldehyde, phenformin

Table 31.7 Agents that increase the osmolar gap (ME DIE)

Methanol
Ethylene glycol
Diuretics (osmotic diuretics like mannitol)
Isopropyl alcohol
Ethanol

- The serum osmol gap is a common laboratory test that may be useful when evaluating poisoned patients. This test is most often discussed in the context of evaluating the patient suspected of toxic alcohol (e.g., ethylene glycol, methanol, and isopropanol) intoxication. Though this test may have utility in such situations, it has many pitfalls and limitations which limit its effectiveness.
- The "oxygen saturation gap" is the difference between the calculated oxygen saturation from a standard blood gas machine and the reading from a pulse oximeter. If it is greater than 5 %, the patient's hemoglobin may be abnormal, representing carbon monoxide poisoning, methemoglobinemia, or sulfhemoglobinemia.
- Simple bedside diagnostic tests for methemoglobinemia are white filter paper and cooking test [11].
- Abnormally high venous oxygen content (arterialization of venous blood) is a characteristic of cyanide and hydrogen sulfide poisoning.
- Serum creatine phosphokinase may be obtained when a patient exhibits signs of altered temperature regulation and muscle tone such as in the serotonin and neuroleptic malignant syndromes or the sympathomimetic toxidrome. Additionally, many common drugs have the potential to cause rhabdomyolysis.
- Radiologic studies should be ordered as needed to evaluate for complications related to the exposure like aspiration or some drugs such as opioids and salicylates that can cause non-cardiogenic pulmonary edema.
- A chest X-ray is necessary in those with respiratory complaints or compromise.
- Abdominal X-ray may be useful in diagnosing certain radiopaque toxins [12] which can be remembered by the mnemonic *CHIPES*: chloral hydrate, heavy metals, iron, phenothiazines, (body) packers, enteric-coated drugs, and salicylates (Fig. 31.1).

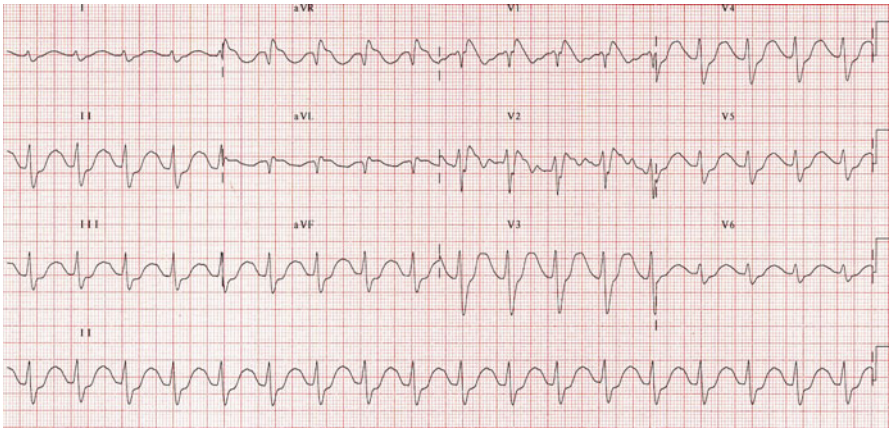

Fig. 31.1 ECG showing Sinus tachycardia, broad QRS complexes and positive R wave > 3 mm with R/S ratio > 0.7 in aVR consistent with Tricyclic antidepressants overdose

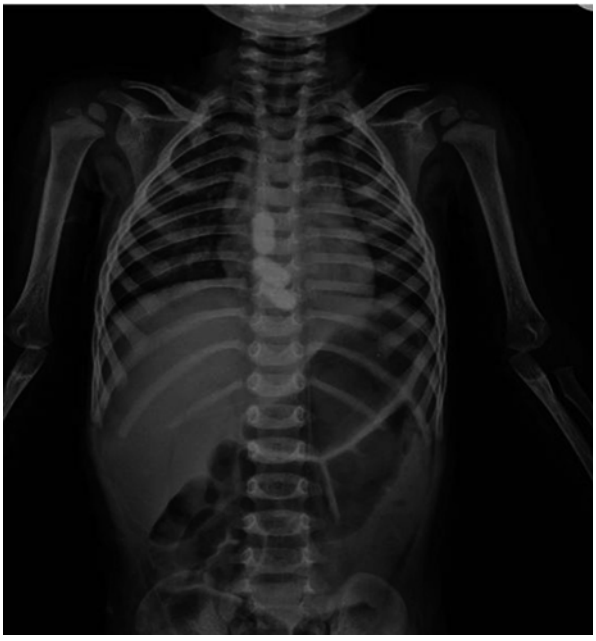

Fig. 31.2 Radio-opaque iron tablets visible in the esophagus of a child after ingestion

- An ECG should be obtained in most poisoned patients. Bradycardia and tachycardia can suggest potential toxicologic etiologies.
- The triad of pseudo right bundle branch block consisting of R wave in aVR, S wave in lead I, and S wave in aVL are highly sensitive indicators of sodium channel blockade resulting from tricyclic antidepressant exposure [13] (Fig. 31.2).

Reducing Absorption and Removal of Toxin

- The initial decontamination consists of removal of all the clothing of the patient and putting them in a plastic bag. While doing so, the physician must protect himself.
- After disrobing, the patient should be washed with soap and copious amounts of water.
- The use of neutralizing agents is strongly contraindicated.

Gastric Decontamination

- Multiple methods of removal of unabsorbed poison from the gut are available to reduce the bioavailability of an ingested toxin including induction of emesis, gastric lavage, and use of activated charcoal and cathartics.
- Although controversial, clinicians must always determine whether the benefits outweigh the associated risks. Before performing a procedure for gastric emptying, it is important to consider:
  - Whether the ingestion is potentially lethal.
  - Can the procedure remove a significant amount of toxin.
  - Risk of toxicity would be decreased.
- Gastric decontamination [14] is unnecessary if the patient has ingested a nontoxic agent or less dose of a toxic agent, if the patient had prior repeated vomiting or patient presents late after ingestion, the toxin is rapidly absorbed, or if he is free of symptoms despite passage of time during which the toxin is known to produce features of toxicity.
- Nevertheless, gastric emptying is indicated even if he is asymptomatic if the patient has ingested a high-risk toxin (cyanide, paracetamol). However, some toxins such as (antidepressants, phenothiazines, salicylates, opioids, phenobarbital, and anticholinergics) may delay gastric emptying.
- Gastric emptying is also delayed in comatose patients. It is also delayed if the toxin forms a mass in the stomach. In these situations, a delayed gastric emptying may be performed though there is no evidence to support this.

Syrup of Ipecac

- Emesis, which was once a favored means of gastric emptying, is now limited to first aid in home settings.
- Current recommendations [15] discourage routine use of ipecac due to lack of evidence for improved outcomes and risks including delayed administration of oral antidotes and other decontamination products, aspiration, and complications from prolonged emesis and retching.

Gastric Lavage

- The term "gastric lavage" means orogastric lavage with a large bore tube. It allows direct irrigation and removal of unabsorbed toxin from the stomach.
- Before doing gastric lavage, the gag reflex of the patient must be checked. If gag reflex is absent or patient is unconscious, endotracheal intubation should be done before passing the lavage tube to protect the airway.
- For inserting an orogastric lavage tube, the patient should be placed in left-lateral position with the headend lowered.
- Gastric contents should be aspirated before any lavage fluid is introduced. The lavage is then performed by using fluid aliquots of 3–4 ml/kg. The lavage is continued till the return is clear. The lavage return should approximate the amount of fluid given to avoid fluid or electrolyte abnormalities.
- It is contraindicated in patients with an unprotected airway, after ingestion of corrosive substances or substances with a high aspiration potential (such as hydrocarbons) and those with premorbid conditions where a risk of gastrointestinal tract bleeding is present.

Cathartics

- Cathartics have been used for many years with the belief of increasing the elimination of the toxins from the gut.
- While a single dose of a cathartic is usually well tolerated, repetitive dosing can lead to serious complications includes electrolyte imbalance, dehydration, and abdominal distension. Cathartics are contraindicated in the presence of ileus, intestinal obstruction, renal failure, hypotension, severe diarrhea, and abdominal trauma.
- Commonly used cathartics are magnesium sulfate (30 g for adults and 250 mg/kg in children), magnesium citrate (4 ml/kg up to a maximum of 300 ml), and sorbitol (1 g/kg as 70 % solution).

Whole Bowel Irrigation (WBI)

- Whole bowel irrigation refers to the mechanical cleansing of the entire GI tract by the instillation of large volumes.
- The solution used most often is a polyethylene glycol specifically formulated not to cause electrolyte abnormalities.
- This therapy has been found to be useful in the overdose of iron, heavy metal, sustained-release or enteric-coated xenobiotics, theophylline, cocaine, or heroin body stuffers and packers. The WBI solution is run at 25–40 ml/kg/h, either orally or by way of nasogastric tube for 4–6 h or until the rectal effluent becomes clear.
- It should not be used if there is bowel obstruction, perforation, or ileus.

Activated Charcoal (AC)

- The use of activated charcoal has revolutionized the treatment of poisoning and has become the first-line treatment for patients who have ingested a potentially toxic amount of drug [16].
- It appears to be the most efficacious and safe decontamination method when the ingested substance is unidentified. However, routine administration in nontoxic ingestions is not indicated.
- The usual dose is 1 g/kg body weight. The tablet form of AC is not recommended. Activated charcoal should be given as premixed slurry with or without a cathartic.
- Activated charcoal is contraindicated in patients with ileus or mechanical obstruction and caustic ingestion.
- It is not effective in caustics, corrosives, heavy metals, alcohol, rapid onset – cyanide, chlorine, others (iron), aliphatic hydrocarbon and lithium can be recalled by the mnemonic *CHARCOAL*.
- The complications are rare but include aspiration into the lungs, hypokalemia, constipation, and intestinal obstruction.

Multiple Doses of Activated Charcoal

- Multiple doses of activated charcoal (MDAC) have been recommended in treating certain poisonings.
- Free charcoal which is available in the intestines can bind any toxin and also reduce the half-life of several drugs by interrupting enterohepatic or enteroenteric recirculation.
- Additionally, free toxin in the blood is likely to diffuse out of the blood into the intestines where it binds the charcoal, thereby maintaining the concentration of free toxin in the intestines near zero. This is termed "gut dialysis."
- The optimal dosage regimen has not yet been clearly established. Depending upon the severity of poisoning, the doses are 0.5–1 g/kg body weight every 1–4 h.
- Expert opinion suggests that MDAC should only be considered in patients presenting with a potentially lethal dose with a long half-life, toxin has a significant enterohepatic circulation (carbamazepine, phenobarbitone, theophylline, digoxin, dapsone, quinine, and plant poisoning or bezoar formation).
- The use of MDAC can cause aspiration, constipation, impaction, and obstruction.

Enhancing Excretion

- Once the absorption of a toxin has been reduced by various methods, the next rational step is to enhance the elimination of already absorbed toxin from the body.
- Important methods for this purpose are alkaline diuresis with alteration in urinary pH, hemodialysis, hemoperfusion, hemofiltration, and exchange transfusion.

- Urine alkalinization [17] is a method of enhancing elimination of weakly acidic toxins by trapping them in an alkaline urine compartment. This is only recommended as first line for the treatment of moderately severe salicylate poisoning and as second line in the ingestion of fluoride, methotrexate, phenobarbital, 2,4-dichlorophenoxyacetic acid, and mecoprop.
- The goal is to maintain the urine pH to 7.5–8.0 with IV sodium bicarbonate.
- In this process, it is essential to check the potassium, as the hypokalemic patient will not develop alkaline urine without adequate potassium stores.
- Compromised renal function and significant preexisting heart disease are contraindicated for urine alkalinization.
- Although alkalinization significantly lowers the serum half-life of phenobarbital, whether it actually improves clinical outcome and decreases the length of hospital stay is unclear.

Extracorporeal Elimination

- The removal of toxins by extracorporeal techniques such as hemodialysis, hemofiltration, and hemoperfusion is the most invasive and expensive method of enhancing elimination of toxins [18].
- It is indicated for agents with low protein binding, low volume of distribution, high water solubility, and low molecular weight [19].
- Toxins accessible to hemodialysis can be remembered by the mnemonic *I STUMBLE* (Table 31.8).
- Charcoal hemoperfusion is useful for barbiturate, carbamazepine, theophylline overdose, and parquet poisoning.

Specific Therapy

- It is important not to waste time in tracing an antidote because the antidotes are available only for a few poisons, and treatment of most cases of poisoning is mainly supportive. If the toxin can be identified, specific therapy using antidotes should be administered.

Table 31.8 Toxins accessible to hemodialysis (I STUMBLE)

Isopropanol
Salicylates
Theophylline (caffeine)
Uremia
Methanol
Barbiturates, beta-blockers (water soluble, such as atenolol)
Lithium
Ethylene glycol

Table 31.9 Chemical antidotes

Poison	Antidote
Acetaminophen	NAC (N-acetylcysteine)
Anticholinergics	Physostigmine
Anticoagulants (warfarin/coumadin, heparin)	Vitamin K1, protamine
Arsenic	Dimercaprol
Benzodiazepines	Flumazenil
Botulism	Botulinum antitoxin
Beta-blockers	Glucagon
Calcium channel blockers	Calcium, glucagon
Cholinergics	Atropine
Carbon monoxide	Oxygen, hyperbaric oxygen
Caffeine, theophylline	Propranolol
Cyanide	Amyl nitrate, sodium nitrate, sodium thiosulfate, hydroxycobalamin
Cocaine, amphetamine	Labetalol, or phentolamine with esmolol, metoprolol, or other cardioselective beta-blocker
Digoxin	Digoxin Fab antibodies
Iron	Deferoxamine
Isoniazid	Pyridoxine
Lead	BAL, EDTA, DMSA
Mercury	Dimercaprol, DMSA, penicillamine
Methanol	Ethanol and fomepizole
Methemoglobin inducers	Methylene blue
Opioids	Naloxone
Oral hypoglycemic agent	50 % Dextrose, octride
Organophosphates and carbamates	Atropine and pralidoxime
SSRI	Cyproheptadine or chlorpromazine
Tricyclic antidepressants	Sodium bicarbonate
Toxic alcohols	Ethanol drip, dialysis

- The clinician should be aware of the specific indications and contraindications associated with the administration of the antidote [20].
- Important antidotes, which are available in India, have been listed (Table 31.9).

Supportive Therapy

- The aim of the supportive treatment is to preserve the vital organ functions till poison is eliminated from the body and the patient resumes normal physiological functions.
- The fluid, electrolyte, and acid-base status should be closely monitored in all patients.
- An important rule in toxicology is to *treat the patient, not the poison.*

Table 31.10 Criteria for admission of the poisoned patient to the ICU

Emergency intubation
Respiratory depression (e.g., PaCO2>45 mmHg)
Cardiac arrhythmia
Second- or third-degree atrioventricular block
Hypotension
Unresponsiveness to verbal stimuli
Glasgow Coma Scale score that is less than 12
Toxin-induced seizures
Increasing metabolic acidosis
Pulmonary edema induced by toxins (including inhalation) or drugs
Hypothermia or hyperthermia
Tricyclic or phenothiazine overdose that manifests anticholinergic signs, neurologic abnormalities, QRS duration of greater than 0.12 s, or QT that is greater than 0.5 s
Body packers and stuffers
Emergency surgical intervention
Antivenin administration
Need for continuous infusion of naloxone
Need for emergency dialysis

Disposition

- After evaluation, treatment, and an appropriate observation period, asymptomatic patients may be discharged from the emergency department.
- The disposition of symptomatic patients depends on the actual or predicted severity and the need or potential need for therapeutic interventions.
- Routine admission of the poisoned patient to the intensive care unit (ICU) is not necessary. Admit in ICU if any of the following is present (Table 31.10).

Pearls and Pitfalls in Poisoning

- High-flow oxygen can induce more free radical injury in paraquate poisoning.
- Using succinylcholine as a muscle relaxant can cause prolonged paralysis in organophosphorous poisoning.
- Insulin can act as a potent vasopressor especially in calcium channel blocker and beta-blocker overdose.
- Transcutaneous pacing may be very useful in digoxin or oleander-induced bradycardia.
- Using calcium gluconate to correct hyperkalemia associated with digoxin overdose or oleander seed poisoning can result in asystole.
- Atropine should be titrated until secretions are dried.
- Hypoxic insult or co-ingestion can alter the pupil size which may be misleading.

- Attributing an altered mental status to alcohol because of its odor on a patient's breath is potentially dangerous and misleading.
- Beware of delayed complications from slow absorption of medications.
- All suicidal patients require psychiatric assessment before discharge.

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Chapter 32
Bites and Stings

Christeine Ariaranee Gnanathasan and Subramanian Senthilkumaran

Key Points
- Antivenom is the mainstay of therapy for poisonous snakebite and there is no role for test dose.
- The shorter the interval between envenomation and the onset of symptoms, the more severe is the morbidity and mortality.
- Provide prescriptions for EpiPen to patients discharged after presenting with life-threatening reactions to bee stings.

Introduction

- There are about 3,000 species of snakes that are found in the world and of them about 600 are venomous, mainly distributed in warm tropical regions [1].
- Global burden of snakebite is estimated to be about 421,000 envenomations and about 20,000 deaths each year [2]. About 50,000 people die of snakebite every year in India [3].

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Types of Snakes

Venomous snakes belong to two families:

1. Family Elapidae (cobras, kraits, and coral snakes) and a subfamily Hydrophiidae (sea snakes)
2. Family Viperidae – the family of Viperidae has two subfamilies:
   - Viperidae (Russell's viper, saw-scaled viper)
   - Crotalinae (pit vipers, hump-nosed vipers, and green pit vipers)

Toxic Effects of Snake Venom

- Snake venom is not a single component but is a highly complex mixture of a variety of components, enzymatic and nonenzymatic proteins and toxins with varying actions, which can work together having different toxicities: cytotoxic, neurotoxic, hemorrhagic, procoagulant and anticoagulant, nephrotoxicity and myotoxicity, etc. [4, 5].
- Variation of venom composition of the same species may be due to geographical, seasonal, or snake-specific reasons (e.g., Russell's viper).
- Quantity of venom injected at a bite is very variable, depending on the species and size of the snake and the mechanical efficiency of the bite.
- For whatever the reason, a proportion of bites by venomous snakes does not result in envenoming, called dry bites.

Pathophysiology

- Elapidae venom has predominately neurotoxins which cause paralysis of muscles by blocking the neuromuscular junction [6]. These could be either postsynaptic (Elapidae) or presynaptic (Elapidae and some Viperidae).
- Some components of the neurotoxins have either direct or indirect action on the brain causing alteration of the level of consciousness [7].
- Viperidae venom is rich in procoagulant enzymes and leads to coagulopathy. Hemorrhagins (zinc metalloproteinases) damage the endothelial lining of blood vessels causing spontaneous bleeding and direct red cell damage that produces intravascular hemolysis.
- Hemolytic and myolytic phospholipase A2 produces rhabdomyolysis and myoglobinuria causing secondary renal failure and red cell membrane damage leading to hemolysis.
- Local tissue injury occurs at the site of bite due to proteases, hyaluronidases, and other enzymatic effects of the venom.

Clinical Features

Russell's Viper

- Local swelling
- Neurotoxicity – ptosis, external ophthalmoplegia, dysphagia, paralysis, etc.
- Coagulopathy – incoagulable blood, hematuria, hematemesis, gum bleeding (Fig. 32.1), etc.
- Myotoxicity – myalgia and rhabdomyolysis
- Acute kidney injury

Cobra

- Local swelling, blistering, and tissue necrosis (Fig. 32.2)
- Neurotoxicity – ptosis, external ophthalmoplegia, dysphagia, respiratory paralysis, limb and muscle weakness (Fig. 32.3), etc.
- No coagulopathy

Krait

- Neurotoxicity – ptosis, external ophthalmoplegia, dysphagia, respiratory paralysis, limb muscle weakness, etc.
- No local effects/envenoming
- No coagulopathy

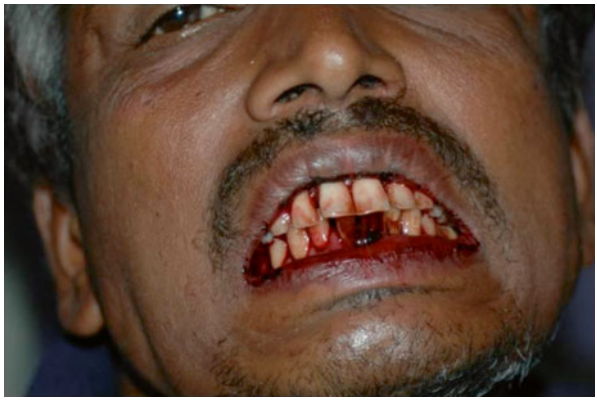


Fig. 32.1 Gum bleeding due to Coagulopathy

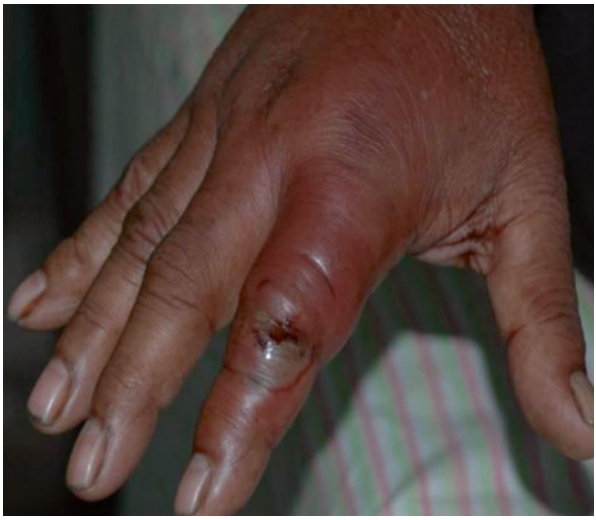


Fig. 32.2 Local swelling, blistering, and tissue necrosis due to Cobra bite

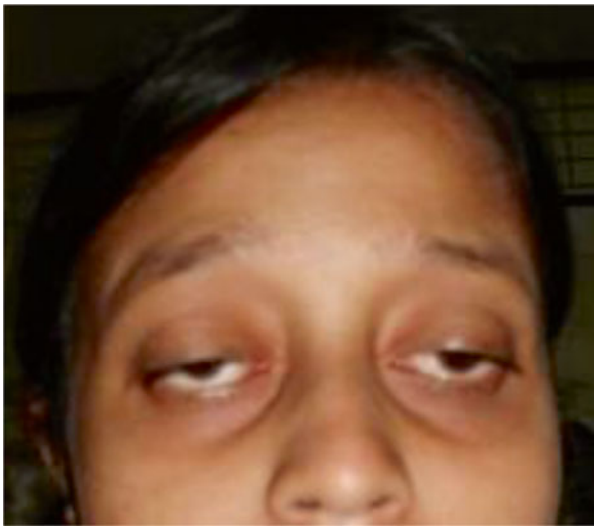


Fig. 32.3 Ptosis in a patient with snake envenomation

Saw-Scaled Viper

- Local bleeding and swelling
- Coagulopathy – incoagulable blood and gum bleeding, etc. (Table 32.1)

Hump-Nosed Viper

- Local swelling, hemorrhagic blisters, and necrosis
- Coagulopathy – incoagulable blood and gum bleeding, red urine, etc.
- Acute kidney injury

Table 32.1 Clinical aspects and therapeutic response

Feature	Cobras	Kraits	Russell's viper	Saw-scaled viper	Hump-nosed viper
Local pain/tissue damage	Yes	No	Yes	Yes	Yes
Ptosis/neurological signs	Yes	Yes	Yes	No	No
Hemostatic abnormalities	No	No	Yes	Yes	Yes
Renal complications	No	No	Yes	No	Yes
Response to neostigmine	Yes	No	No	No	No
Response to ASV	Yes	Yes	Yes	Yes	No

Sea Snakes

- Acute rhabdomyolysis, trismus, myoglobinuria, hyperkalemia, and acute kidney injury
- Neuromuscular paralysis and respiratory failure

Green Pit Viper

- Gross swelling and pain in the bitten limb and painful lymphadenopathy

Diagnosis

- If the dead/live snake is brought, it can be identified.
- Otherwise, the species responsible can be inferred from the clinical syndrome of symptoms and signs. Since we do not have any venom detection kits at the point of care so, the syndromic approach is useful [8].
- Circumstantial evidence also helps in determining a krait bite – sleeping on the floor in a rural house and presenting with abdominal pain and neurotoxicity [9].
- It is supported by evidence of incoagulable blood detected by 20 min whole blood clotting test (20 WBCT) – this is a very useful and informative bedside tests that require only a new, clean, dry, glass tube or bottle.
- Point-of-care INR testing devices should not be used for snakebite cases to diagnose venom-induced consumption coagulopathy [10].

Investigations

Twenty Minute Whole Blood Clotting Test (20 WBCT)

- This is a very useful bedside test to find out any evidence of coagulopathy.
- In Southeast Asian region, incoagulable blood is a diagnostic of a viper bite and rules out an elapid bite.

- If there is any doubt, repeat the test in duplicate, including a "control" (blood from a healthy person).

Warning! If the tube or bottle used for the test is not made of ordinary glass, or if it has been used before and cleaned with detergent, its wall may not stimulate clotting of the blood sample in the usual way and test will be invalid.

- Depending on the availability, other investigations should include a full blood count with platelet, coagulation studies including d-dimer, FDP, PT, APTT, and biochemical tests including creatine kinase. A urine analysis is helpful for detecting blood or myoglobin.

Management of Snakebite

First-Aid Treatment

- The four main steps involved in sequence in the correct first aid can be remembered by the mnemonic; do it *right*.
- *R = reassure the patient*.
  (75 % of snakebites are from a venomous species and that even if it is a venomous snakebite, on average, only 50 % of such bites actually envenomate; the rest is called "dry" bites.)
- *I = immobilize without compression*.
  (Immobilize in the same way as a fractured limb. Do not allow the victim to walk but should be carried. Do not apply any compression in the form of tight ligatures; their use can increase necrosis.)
- *GH = get to the hospital fast*.
  (Traditional remedies have no proven benefit in treating snakebite.)
- *T = tell the doctor.*
  (Any systemic symptoms such as drooping eyelids, double vision, dribbling, and any taste of blood in the victim's mouth or developing bruising at the bite site that manifests on the way to the hospital.)

Most traditional first-aid methods should be discouraged; they do more harm than good!

- Analgesia.
- Anti-snake venom.
- Anticholinesterase therapy.
- Tetanus prophylaxis if needed.
- Local wound care.
- Antibiotic should be considered in case of cellulitis or necrosis.

Anti-snake Venom Treatment

- Antivenom is the only specific antidote to snake venom.
- The most important decision in the management of a snakebite victim is whether or not to give antivenom.
- Antivenom treatment carries the risk of severe adverse reactions, and in most countries, it is costly and may be in limited supply.
- Polyvalent antivenin manufactured in India will not neutralize venom of pit vipers [11].

Indication for Antivenom

Antivenom treatment is recommended if and when a patient with proven or suspected snakebite develops one or more of signs of systemic envenoming (Table 32.2).

Table 32.2 Indication for antivenom

Systemic envenoming
Hemostatic abnormalities:
Spontaneous systemic bleeding (*clinical*)
Coagulopathy or thrombocytopenia (*laboratory*)
Neurotoxic signs:
Ptosis
External ophthalmoplegia
Paralysis (*clinical*)
Cardiovascular abnormalities:
Hypotension
Shock
Cardiac arrhythmia (*clinical*)
Abnormal ECG
Acute renal failure:
Oliguria/anuria (*clinical*)
Rising blood creatinine/urea (*laboratory*)
Hemoglobinuria/myoglobinuria:
Dark-brown urine (*clinical*)
Urine dipsticks, hyperkalemia (*laboratory*)
Local envenoming
Local swelling involving more than half of the bitten limb (in the absence of a tourniquet)
Swelling after bites on the digits (toes and especially fingers)
Rapid extension of swelling (e.g., beyond the wrist or ankle within a few hours of bites on the hands or feet)
Development of an enlarged tender lymph node draining the bitten limb

- Skin/conjunctival hypersensitivity testing does not reliably predict early- or late-antivenom reactions and is not recommended.
- There is no absolute contraindication to antivenom treatment, but patients who have reacted to equine serum in the past and those with a strong history of atopic diseases should be given antivenom only if they have signs of systemic envenoming.
- It may be a monospecific or polyspecific antivenom that should be given by the intravenous route only.
- Intravenous infusion: reconstituted freeze, dried, or liquid antivenom is diluted in approximately 200–300 ml of isotonic saline and infused at a constant rate over a period of 1 h and repeated as needed.
- Snakes inject the same dose of venom into children and adults. *Children must therefore be given the same dose of antivenin as adults. But*, *volume of the infusion should be adjusted according to the body weight and underlying diseases. Correct dose of antivenin*, *given promptly*, *is needed to save children*.
- Saving the mother's life is the priority in pregnancy. In this regard the mother should not be deprived of any treatment that is indicated.

Adverse Reaction to Antivenom

Anaphylaxis and anaphylactoid reaction is a major obstacle for the treatment of the antivenom and may be life threatening. In case of anaphylaxis, then:

- *Stop antivenom*.
- 0.5 mg of 1:1000 adrenalines will be given IM into lateral thigh for adults. Children are given 0.01 mg/kg body weight of adrenaline IM.
- For long-term protection against anaphylactoid reaction, hydrocortisone and H1 and H2 blockers will be administered by IV.
- A second dose of 0.5 mg of adrenalin 1:1000 IM is given after 10–15 min if the patient's condition has not improved or is worsening.
- This can be repeated for a third and final occasion, but in the vast majority of reactions, two doses of adrenaline will be sufficient.
- If there is hypotension or hemodynamic instability, IV fluids should be given.
- The ASV can be restarted slowly for 10–15 min after the patient had improved, under close monitoring. Then the normal drip rate should be resumed.

End Point for Antivenin

- Spontaneous systemic bleeding usually stops within 15–30 min.
- Neurotoxic envenoming of the postsynaptic type will begin to improve as early as 30 min after antivenom but usually takes several hours.

Criteria for Giving More Antivenom

- If the blood remains incoagulable as measured by 20WBCT 6 h after the initial dose of antivenom, the same dose has to be repeated. It is based on the time taken for the liver to restore coagulable levels of fibrinogen and other clotting factors are three to nine, mean 6 h.
- In patients who continue to bleed briskly, the dose of antivenom should be repeated within 1–2 h.
- In case of deteriorating neurotoxicity or cardiovascular signs, the initial dose of antivenom should be repeated after 1–2 h.

Caution on Neurtoxic envenomation:
- Snake venom-induced dysautonomia can cause severe hypertension [9]
- Dysrhythmia can occur in susceptible patients. Hence, cardiac monitoring is needed [10]
- Snake envenomation with both internal and external ophthalmoplegia, quadriparesis and anarthria will mimic brain death [11]

Management in Special Situations

- *When no antivenom is available*: conservative approach/plasmapheresis.
- *Neurotoxic envenoming with respiratory paralysis*: assisted ventilation.
- *Hemostatic abnormalities*: transfusion of clotting factors and platelets, fresh frozen plasma.
- *Renal failure*: dialysis.
- *Dark-brown urine* (*myoglobinuria or hemoglobinuria*): correct hypovolemia and acidosis and consider a single early infusion of mannitol.
- *Severe local envenoming*: local necrosis, intracompartmental syndromes – surgical intervention may be needed, but the risks of surgery in a patient with consumption coagulopathy, thrombocytopenia, and enhanced fibrinolysis must be balanced against the life-threatening complications of local envenoming.

Disposition

- Patients without signs of envenomation should still be admitted for observation for 24 h due to the potential delay in onset of findings of venom poisoning.
- Any snakebite victim with signs or symptoms of envenomation should be admitted to the hospital.

Rehabilitation

- Physical and psychological are needed for patients who have had complications and recovered following a long hospital stay.

Scorpion Stings

- Scorpion stings are acute life-threatening medical emergency which are common in rural areas of many tropical and subtropical regions of the world.

Epidemiology

- Out of 1500 scorpion species known to exist, about 30 are of medical importance.
- In India, 86 species of scorpions have been identified; *Mesobuthus tamulus* and *Palamneus gravimanus* are of medical importance [12].
- Even though millions of scorpion stings occur annually, most cases are minor, with localized pain and minimal systemic involvement. However, severe envenomation is the most important health issue in South Asia, Middle East, and North Africa.
- While the incidence of scorpion stings is higher in adults, the severity of envenomation is considerably greater in children, in whom the mortality is up to ten times higher than in adults [13].

Toxicity of Venom

- Venom is deposited deep to subcutaneous tissue after sting and it is almost completely absorbed from sting site within 7–8 h (70 % of the maximum concentration of venom in the blood reached within 15 min of sting).
- Several toxins have been identified in scorpion venoms, most of which are small peptide toxins that target voltage-dependent ion channels and toxins alter these channels, leading to prolonged depolarization and, hence, neuronal excitation [14].
- The toxins that have the greatest medical significance are the scorpion α-toxins which cause massive endogenous release of the catecholamines as well as other vasoactive peptide hormones.

Pathophysiology

- Scorpion venom delays the closing of neuronal sodium channels, resulting in "autonomic storm" owing to sudden outpouring of endogenous catecholamines into the circulation.

- Parasympathetic effects are less severe compared to sympathetic effects.
- The combination of sympathetic excitation and the release of catecholamine in plasma cause the majority of the severe systemic effects.
- The severity of scorpion sting depends on the victim's age, the season, and the time between sting and treatment.

Clinical Manifestations

- Despite the variety of different scorpion species that exist, majority of them produce similar neurotoxic excitation. However, *Centruroides* and *Parabuthus* scorpions are associated primarily with neuromuscular toxicity, whereas severe envenomation from *Androctonus*, *Buthus*, and *Mesobuthus* scorpions is associated with cardiovascular toxicity.

Local Manifestations

- Severe excruciating local pain.
- Swelling, redness, local increase in temperature, and regional lymph node involvement.
- Local edema, urticaria, fasciculation, and spasm of underlying muscles are rarely seen due to serotonin.
- Positive tap test is present (on tapping increase in paresthesia occurs) in some patients.
- The stings from the *H. lepturus* scorpion of Iran do not cause immediate severe pain. The majority of cases are characterized by erythema, purpuric, and bullous lesions that resolve, but in about 20 % of cases, there is delayed localized necrosis that develops over hours or days. The syndrome appears to be similar to that associated with bites by loxosceles spiders.

Systemic Manifestations

- Systemic symptoms may develop within minutes, but may be delayed as much as 24 h. Features of autonomic nervous system excitation are transient cholinergic and prolonged adrenergic stimulation.
- Hypertension is common and occurs early in response to sympathetic stimulation.
- Prolonged massive release of catecholamines causes restlessness, piloerection, marked tachycardia, mydriasis, hyperglycemia, hypertension, toxic myocarditis, cardiac failure, and pulmonary edema.
- All forms of electrocardiogram abnormalities are documented.

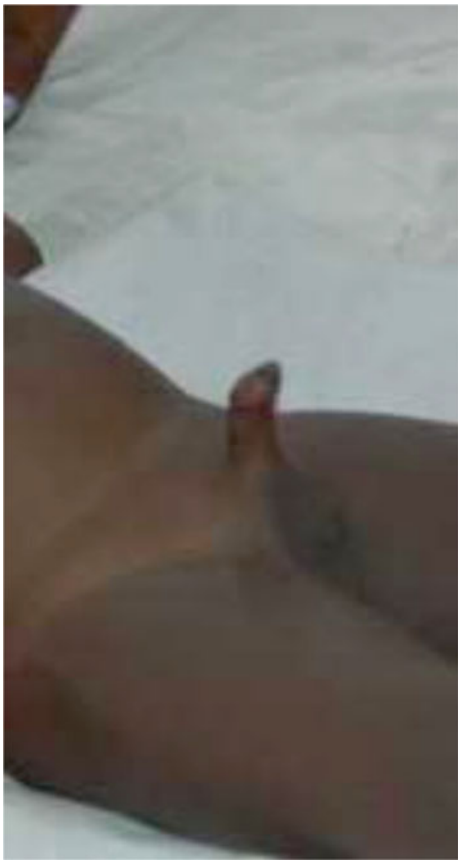


Fig. 32.4 Priapism

- Priapism (Fig. 32.4) may occur secondary to cholinergic stimulation.
- Vomiting and abdominal pain are common after scorpion stings.
- Hemiplegia and other neurological lesions have been attributed to fibrin deposition resulting from disseminated intravascular coagulation.
- Acute pancreatitis has been reported with *Leiurus quinquestriatus* and *Tityus* species.
- Systematic features of *H. lepturus* scorpion sting are direct hemolysis with hemoglobinuria and acute kidney injury that often warrants for dialysis.

On the basis of clinical manifestations, scorpion envenomation is graded into four grades [15]. Ideally, clinical grading should be a direct treatment.

- Grade I: local pain and paresthesia without any systemic manifestation
- Grade II: patient with pain and paresthesia distant from the site of sting with or without tachycardia and without cardio respiratory signs
- Grade III: patients with peripheral circulatory failure, cardiovascular, and respiratory manifestations
- Grade IV: patient with central nervous system manifestation and multisystem involvement

Differential Diagnosis

- Tachycardia, excessive secretions, wheezing, and respiratory distress accompanying serious scorpion stings may be mistaken for asthma, airway obstruction from a foreign body, or poisoning with cholinergic agents such as organophosphate insecticides.

Investigations

- There are no specific diagnostic investigations recommended for scorpion stings. Investigations should focus on potential complications of scorpion envenomation.

Treatment

- Mild pain can be alleviated by application of ice packs over the site of sting and with oral analgesics.
- Severe excruciating local pain that does not respond to oral analgesia can be transiently relieved by ring block using combination of lignocaine and bupivacaine which can give prolonged relief from pain.
- Prazosin is a pharmacological and physiological antidote to scorpion venom actions. It totally reverses the metabolic and hormonal effects of alpha-receptor stimulation [16].
- Prazosin (plain tablet not sustained release form) is administered orally as 1 mg in adults (children 30 μg/kg).
- Repeat prazosin in the same dose after 3 h depending on the clinical response and later every 6 h (not exceeding 5 mg total in a day) till the extremities are warm and dry and the peripheral veins are visible easily.
- Prazosin can be given irrespective of blood pressure provided there is no hypovolemia.
- Dobutamine is indicated for hypotension due to cardiogenic shock.
- Nitroglycerin is used to treat pulmonary edema, whereas other vasodilators like hydralazine, captopril, nifedipine, sodium nitroprusside, and clonidine are not recommended because of potential adverse effects like sympathetic stimulation and reflex tachycardia.
- Atropine used to reverse severe bradycardia associated with hypotension and excessive sweating or salivation can cause an autonomic storm with transient cholinergic stimulation followed by sustained adrenergic hyperactivity; hence it is contraindicated.
- Neuromuscular incoordination, sympathetic agitation, and seizures are alleviated with the administration of benzodiazepine.
- The use of antivenom for scorpion stings remains controversial.

- Commercially prepared antivenins are available in several countries for some of the most dangerous species. The dose is 5–25 ml of antivenom diluted in two to three volumes of isotonic saline to be given intravenously over an hour.
- If there is no significant improvement, further doses of antivenom can be given [17] (total dose of antivenom required is 30–100 ml in severe envenomation).

Disposition

- Grade I and II envenomations after a short observation period (3–4 h after sting occurred) for progression of symptoms.
- Grade III and IV envenomations require admission to ICU if they have not received antivenom or have any evidence of a complication.
- Encourage patient to return for progression of symptoms.
- Patients suspected of severe envenomation should be hospitalized for at least 24 h and closely observed for cardiovascular and neurological sequelae.

Hymenoptera Stings

- Hymenoptera stings pose great hazards in tropics and Hymenoptera are the most important venomous insects known to humans.
- More fatalities result from stings by these insects than by stings or bites by any other arthropod.

Entomology

- The Hymenoptera families of medical interest include Apidae (honeybees), Bombidae (bumblebees), Vespidae (wasps, hornets, and yellow jackets), and Formicidae (ants).
- The Bombidae and Vespidae have stingers that remain functionally intact after a sting, resulting in their ability to inflict multiple stings.
- Yellow jackets may attack without provocation and are the most common cause of insect-induced anaphylactic reactions.

Toxicity of Venom

- Hymenoptera venom contains several components such as proteins, enzymes, and vasoactive amines.
- Melittin is the major component of honeybee venom that can cause degranulation of basophils and mast cells.

- Fire-ant venom is composed primarily of a transpiperidine alkaloid that causes tissue necrosis.
- Honeybee venom is immunochemically distinct. However, yellow jacket and hornet venoms have a high degree of cross-reactivity.
- The dose of venom delivered per sting may vary from none to the entire contents of the venom gland. It has been estimated that about 1500 stings would be required to deliver a lethal dose of hymenoptera venom for a nonallergic adult who weighs 70 kg [18].

Pathophysiology

- Hymenoptera venom contains different peptide antigens that all may trigger allergic reactions.
- The most well-known allergic reaction is the Type I anaphylactic or immediate hypersensitivity reaction, and this is similar to IgE-mediated allergic reactions.
- Delayed reactions develop several days to a week after the sting and these reactions are non-IgE mediated.
- Although most deaths result from immunologic mechanisms, some are from direct toxicity.

Clinical Manifestations

- The patient may present with local or systemic signs of envenomation.

Local Manifestations

- Local reactions are common, resulting in swelling, erythema, and a burning sensation at the sting site, vesiculation and blisters, itching, and a sensation of warmth.
- These reactions are benign, resolve within hours to days, and are not predictive of systemic reactions to subsequent stings.

Systemic Manifestations

- There is no correlation between systemic reaction and the number of stings.
- Vomiting, diarrhea, hypotension, syncope, angioedema, bronchospasm, laryngospasm, rhabdomyolysis, coagulopathy, and death.
- The complications associated with severe Hymenoptera envenomation include cerebral and myocardial infarction.

Delayed Reaction

- It is believed to be immune complex mediated that consists of serum sickness-like features appearing 5–14 days after a sting.

Differential Diagnosis

- Extensive local reactions must be differentiated from an infectious cellulitis.
- Anaphylactic reactions may be due to variety of drugs, foods, and environmental allergens.

Investigations

- There are no specific diagnostic investigations recommended.
- CPK, coagulation profile, ECG, and renal function should be checked in severe cases of multiple stings.

Treatment

- The single bee sting injury does not require any treatment.
- The bee stingers often remain embedded in the patient's skin, and these have to be removed as soon as possible.
- Rapid removal of the stinger by any means (the method is unimportant) is most effective in minimizing envenomation [19].
- Large local reactions usually respond well to a short course of antihistamines, analgesics, H2 blocker, and steroids.
- Nebulized salbutamol may be considered for treatment of bronchospasm.
- Renal function should be closely monitored and symptomatic treatment instituted [20].
- Provide tetanus prophylaxis if appropriate.
- In cases of multiple wasp stings, secondary infections should be anticipated and appropriate antibiotic prophylaxis to cover skin flora.
- Establish airway, breathing, and circulation to provide adequate airway, ventilation, and perfusion.
- Envenomated patients suffering anaphylaxis should receive an intramuscular epinephrine.

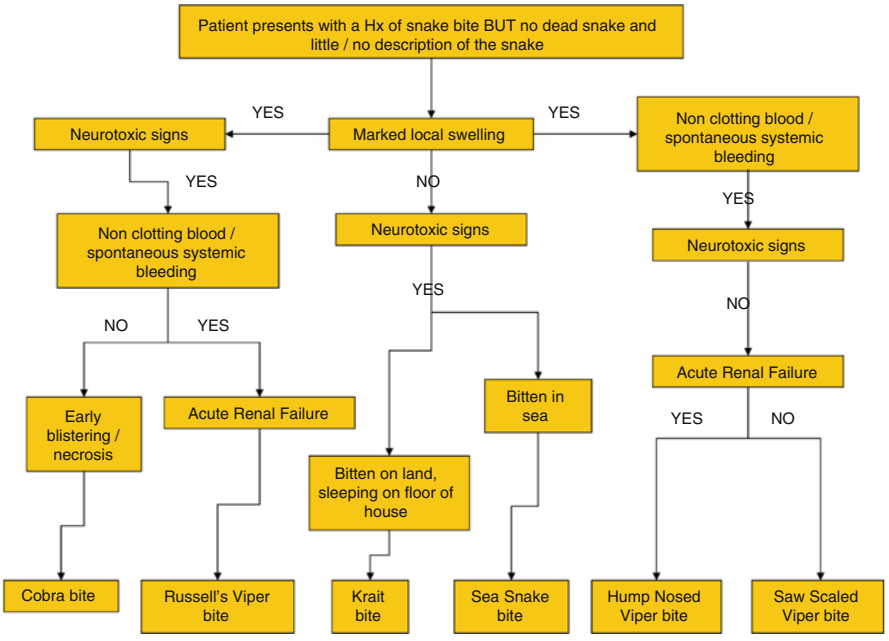
Disposition

- Minimal isolated local reaction can be discharged.
- Life-threatening reaction requires 24 h of observation.
- ICU admission is required if there is worsening of symptoms, airway compromise, and hemodynamic instability.

Pearls and Pitfalls

- Differentiating between the biting species either as venomous or nonvenomous based on the bite marks is useless.
- If the captured or killed snake be brought to the ED with the victim, identification of the species should be carried out carefully as there is always an inherent danger in this practice as they can even envenomate when they are dead.
- If patient received antivenom, monitor for delayed serum sickness.
- Rely on the absence of a visible lesion at the site of the sting to rule out scorpion envenomation.
- Patients experiencing a systemic reaction to wasp or bee stings should be referred to an allergy specialist.
- In patient on beta-blockers who is resistant to epinephrine, glucagon can be administered.

Clinical pathway – snakebite management



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Chapter 33
Household Poisoning

Fazle Rabbi Chowdhury and Abdul Mumith Ruhan

Key Points
- Any attempt at gastric emptying or dilution of compound is contraindicated in corrosive poisoning.
- Alkali is more dangerous.
- Secure airway as soon as possible as airway compromise from oedema may progress rapidly.
- Immediately remove batteries lodged in the oesophagus.

Introduction

- Among the thousands of harmless products available for household, very few are hazardous. Even then, poisoning with these substances is one of the common modes of poisoning all around.
- Meta-analysis of global data on household poisoning is still missing, though there are a considerable number of regional evidences.
- Patil et al. found household products as the most common means of poisoning agents in an urban setting of India [1].
- Baseline survey in Bangladesh showed household products are responsible for around 20.6 % acute poisoning cases [2].

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- Studies done in New Zealand and South Africa also revealed it as one of the commonest agents of poisoning [3, 4].
- Although it is commonly accidental, a number of suicidal attempts are not negligible. The tough challenge for clinicians and toxicologists is to identify the few exceptional life-threatening situations where immediate interventions are needed.

Classification

According to the type of products, household chemicals can be classified as denoted in Box 34.1.

Box 34.1 Household Chemical Preparations

(A) Bleach

- Sodium hypochlorite
- Hydrogen peroxide

(B) Cosmetics and toiletries

- Shaving gel/foam
- After shave lotion
- Hair remover
- Hair colour
- Shampoo
- Perfume
- Creams and lotions
- Nail polish
- Nail polish remover
- Mouthwash
- Shower gel
- Talcum powder

(C) Detergents

- Washing-up liquid
- Fabric conditioner
- Soap
- Automatic washing/dish washing machine liquid

(D) Disinfectants

- Toilet cleaner
- Phenol
- Antiseptic solution
- Sterilizing tablet

(E) Petroleum distillates

- Kerosene
- Petrol
- Paint thinner
- Paintbrush cleaner
- Turpentine substitute
- Furniture and floor polish

(F) Food products

- Vinegar
- Food colours

(G) Others

- Lyme
- Mothballs
- Button battery
- Glue

Household agents can be also classified according to their chemical natures as acid, alkaline, volatile substances and others.

Acid	Alkaline	Volatile substances	Others
Toilet cleaner	Sodium hypochlorite	Perfume	Nail polish
Hair colour	Hydrogen peroxide	Kerosene	Nail polish remover
Phenol	Shaving gel/foam	Petrol	Talcum powder
Antiseptic solution	After shave lotion	Paint thinner	Button battery
Sterilizing tablet	Hair remover	Paintbrush cleaner	Glue
Vinegar	Shampoo	Turpentine substitute	
Food colours	Mouthwash	Furniture and floor polish	
	Shower gel	Mothballs	
	Washing-up liquid		
	Fabric conditioner		
	Soap		
	Automatic washing/dish washing machine liquid		
	Lyme		

Pathophysiology

- Almost all household products described above are corrosive in nature. While describing the pathophysiology, it will be more convenient to discuss as a unified entity.

- A wide range of lesions may occur after ingestion of corrosive substances including mild epithelial injury to whole thickness necrosis of the involved organs [5, 6].
- The pathology depends on the route of exposure or the route of absorption of the poison into the body. Most cases of household poisoning occur through oral ingestion leading to corrosive effects in the alimentary system along with systemic toxicity.
- Direct contact to the skin or eye may cause damage to the contact area.
- The poisons pass more quickly through warm, wet, sweaty skin than cold, dry skin. Poisons can also be injected through the skin from a syringe, or a pressure gun, or during tattooing, or damaged skin area.
- Poison injected under the skin or into the muscle has to pass through several layers of the tissue and through lymphatics before reaching the blood vessels, so it acts more slowly.
- Several factors influence the pattern of lesions, including the nature of the corrosive, the duration of contact, the amount of the substance encountering the tissue, etc. [7]
- Lesions may vary from superficial to deep [8]. Moderately strong acids produce coagulation necrosis of the gut mucosa, and formation of eschar, limiting the damage up to the superficial layers [5].
- Moderately strong alkali cause liquifactive necrosis of the mucosa with saponification and continued deeper penetration into the underlying tissues causing extensive damage.
- In both strong acids and alkali, tissues immediately become corroded, soft and sometimes dissolved, disintegrated and perforated, and thus, the whole or a part of it turns into a small soft pulpy mass. In the acute stage bleeding, perforation and necrosis may occur.
- Long-term complications include the formation of the oesophageal stricture, antral stenosis and even the development of the oesophageal carcinoma [9].
- Stricture formation is more common in patients with second- and third-degree burns [10]. Dishwasher detergent ingestion is more prone to cause severe injury to the upper gastrointestinal tract and oropharynx [11].
- Direct contact to the skin may result in sloughing out of the affected tissue, ulceration and necrosis. Inflammatory changes in the skin occur if contact is prolonged [12].
- Ocular contamination can cause various degrees of damages to the eyes.
- Nail polish and nail polish remover contain solvents including acetone. Acetone can cause acidosis.
- Button batteries smaller in size can pass the gastrointestinal tract, but batteries larger than 20 mm may stick in the oesophagus causing obstruction with the risk of breaking and releasing their contents resulting in localized tissue damage and oesophageal perforation.

- The negative battery pole identified as narrowest side on lateral X-ray causes most severe necrotic injury. Batteries lodged in the oesophagus may cause serious burns within 2 h.
- Even on the gut wall, local corrosive effects with subsequent perforation may occur due to leakage of ingredients and local electrical discharge.

Clinical Features

All household compounds produce nearly the same pathology and thereby the same clinical features. The most common symptoms are related to the local irritative/corrosive effects on the aerodigestive tract [13].

(A) *Features of acid burn*:

- Lip, buccal mucosa and palate injuries may be associated with mucosal sloughs or ulcers producing mild to severe pain and/or bleeding.
- Involvement of the pharynx and/or larynx can produce localized throat pain, burning sensation, difficulty in swallowing, hoarseness, stridor and respiratory distress along with the development of mucosal oedema [14].
- Perforation of the oesophagus and/or stomach may occur, which can lead to chemical peritonitis exhibited by the features, e.g. severe abdominal pain and tenderness, vomiting, fever, muscle guard and rigidity. Gastrointestinal upset, abdominal pain, vomiting, diarrhoea and haematemesis occur in more severe cases.
- Systemic effect includes circulatory collapse, metabolic acidosis, hypoxia, respiratory failure, intravascular coagulation and haemolysis [15].
- Renal failure can occur as the end result of acute tubular necrosis. Later effects of acid ingestion include stricture formation, pyloric stenosis and achlorhydria.

(B) *Features of alkali burn*:

- Ingestion of acid often causes severe damage to the stomach, whereas ingestion of alkali usually injures the oesophagus sparing the stomach. Alkali damage to the oesophagus may produce diffuse ulceration and sloughing, with petechial haemorrhagic spotting throughout the whole mucosa and its underlying tissues.
- Oesophageal perforation can develop along with its sequel, e.g. mediastinitis, pneumonitis, cardiac injury and tracheoesophageal fistula.
- Metabolic acidosis may develop in patients with severe gastrointestinal bleeding.
- Renal failure is a rare complication, may be accompanied by GI bleeding and shock.

- Inhalation of alkaline vapour may cause hoarseness, stridor, upper airway oedema, wheezing, pneumonitis and respiratory failure.
- Ocular exposure may produce severe conjunctival irritation, chemosis, corneal epithelial defects and limbal ischaemia, and permanent visual loss may occur in severe cases.
- Dermal contact with alkaline corrosives may produce redness, irritation, pain and sometimes full thickness burns of the skin.

(C) *Features of volatile substance*:

- Ingestion of kerosene immediately produces burning sensation in the mouth and pharynx, nausea, vomiting, abdominal pain and diarrhoea by increasing peristalsis.
- Kerosene odour may be present in the breath and vomitus.
- Aspiration of poisonous chemical to the lungs can cause chemical pneumonitis. More soluble agents have the tendency to involve the upper respiratory tract, whereas less soluble agents reach the distal airways, resulting in pulmonary oedema.
- Secondary pneumonia may develop immediately, and in the long run, there may be bronchiectasis, bronchiolitis obliterans and lung destruction.
- The victim may present with fever, cough with/without sputum production, wheeze, respiratory distress, crackles in the lungs, chest pain, feature of respiratory failure, e.g. severe breathlessness, tachypnoea, cyanosis, worm extremities, tremor, unconsciousness, etc. [16].
- Following direct inhalation of volatile substances, respiratory manifestation may occur within 30 min producing symptoms of drowsiness, dizziness, agitation, anorexia, nausea, vomiting, stomach ache, sore throat, cough, vagal inhibition, respiratory depression and/or failure, tachypnoea, cyanosis, nasal flaring as well as supraclavicular, intercostal retraction and chest indrawing, haemorrhagic pneumonitis, pneumatocele, intravascular haemolysis and cardiac arrhythmia.
- Cardiac arrhythmia due to sensitization of the heart to adrenaline is the most common and well documented in experimental conditions. Deaths from cardiac arrhythmia during or soon after volatile substance ingestion are unpredictable and unpreventable, and resuscitation is rarely successful. In rare cases, myocarditis may develop.
- CNS excitation is followed by lethargy, impaired consciousness, convulsion and coma [17, 18].
- Substances containing acetone, e.g. nail polish and nail polish remover, can cause features of acidosis, e.g. headache, confusion, fatigue, sleepiness, rapid and shallow breathing, hypoxia, lack of appetite and sweet and fruity smells in the breaths; even the patients may be comatose.
- Ocular exposure can cause severe conjunctival irritation, chemosis, corneal epithelial defects, limbal ischaemia and periorbital oedema. Permanent visual loss may occur in severe cases [19].

- Direct contact of the corrosives to the skin may result in varying degrees of burn, pain, irritation, redness, blistering, ulceration, lacerating necrosis and defatting of the skin with severe scarring after weeks.

Investigations

Following poisoning by household products, corrosive injury to the oesophago-gastro-duodenal tract requires immediate diagnostic and therapeutic approach.

- Upper gastrointestinal endoscopy should be performed early (within 24 h) to define the extent of injury, i.e. perforation or stricture, and guide appropriate therapy especially in addressing the need for emergency surgery [20]. The extent of injury can be graded according to endoscopy findings.

  Grading system for corrosive burns of the alimentary tract with endoscopy: [5]

Grade	Features
1	Erythema and oedema only
2a	Localized, superficial friability, blisters or ulceration
2b	Features as for grade 2a, but with circumferential ulceration
3	Multiple deep ulcers, area of necrosis

- Radiology helps in detection of perforation and other corrosive injuries to the stomach. Perforation in the stomach can be noted by plain X-ray of abdomen taken in erect posture showing crescentic gas shadows under both domes of the diaphragm [20]. Other radiological features of corrosive damage to the stomach include gastric atony, thickened mucosal folds and mural filling defects representing mucosal oedema, mural haematoma and necrosis. Similar changes, although to a lesser extent, can be found in the duodenum. Within a few weeks following ingestion, there is rapid progression to antropyloric stenosis, leading to gastric outlet obstruction [20].
- After lung ingestion or inhalation of toxic households, the initial chest radiograph can be normal up to 48 h; therefore, delayed radiographs are important. The most common radiographic pattern is pulmonary oedema; some other patterns of radiographic opacities can be seen [16].
- Biochemical examination of the blood like CBC, serum electrolyte, arterial blood gas (ABG) analysis, etc. may reveal features of infection, acidosis, respiratory failure, septicaemia, etc.
- If damage to any specific organ occurs, e.g. the kidney and liver, the respective biochemical markers are significantly changed.

Management

General management of poisoning is common focusing on airway, breathing and circulation. That's why in this chapter, specific management is described according to the nature of the agent.

1. *Treatment of acid exposure*:

   - Emesis should not be induced. It is contraindicated.
   - Attempt at gastric lavage is contraindicated due to danger of perforation.
   - Dilution and/or neutralization are also contraindicated.
   - Steroids confer no benefit and may mask abdominal sign of perforation.
   - Have early endoscopy as soon as possible.
   - Give analgesic for pain.
   - Give soluble calcium tablet for hydrofluoric acid ingestion if patient is able to swallow followed by intravenous infusion of 10 % calcium gluconate 10 ml in saline.
   - Administer oxygen inhalation and ventilation if necessary.
   - For external burn and eye injury, wash immediately with water or saline for 10–30 min. Then use silver sulphadiazine ointment for the skin and chloramphenicol ointment for the eye.
   - For the oedema of the glottis, tracheostomy should be done.
   - For oesophageal stricture and gastric outlet obstruction, surgery is usually done after 4–6 weeks of ingestion.
   - Manage complications (e.g. peritonitis) if any.

2. *Treatment of alkali exposure*:

   - Dilution or neutralization, induce emesis, gastric aspiration and lavage are contraindicated.
   - Irrigate exposed eyes with sterile cold water or saline at least for 20 min and continue until the P^H returns to normal.
   - Demulcents – egg white, olive oil, butter and cold milk – should be avoided.
   - Clear airway and support with O_2 inhalation and artificial respiration and, if necessary, intensive care unit support including ventilator.
   - Administer IV fluid for maintaining nutrition and electrolyte balance.
   - As there is no specific antidote for alkaline corrosives, symptomatic treatments are to be done. Neutralization with alkali nowadays is not done.
   - Surgical treatment must be considered for any patient with grade II or III oesophageal injury.
   - Analgesic, including narcotics, may be given to relieve pain.
   - Diagnostic endoscopy should be performed within 12–24 h of alkali ingestion.
   - Corticosteroids have no role in the management of a case and complication. It is rather harmful.

3. *Treatment of volatile agents*:

   (A) *Asymptomatic cases*: Admit for observation and discharge after 24 h if no symptom develops.

   (B) *Symptomatic cases*: Symptomatic patients should be managed according to the following guidelines:

   - Evaluate and maintain the ventilatory status of the patients. Administer O_2 to all patients with respiratory symptoms.
   - Some patients (respiratory failure) may require ventilatory support.
   - If the skin has been exposed to volatile agents, remove all contaminated clothing and wash the skin with copious amounts of water.
   - Routine antibiotics are not recommended, the occurrence of secondary infection of the affected lung can usually be readily detected by reappearance of fever on third to fifth day after ingestion.
   - Provide other supportive treatments including nutritional support.
   - Corticosteroid, activated charcoal, cathartics, mineral oil and olive oil have no beneficial effect.

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Chapter 34
Pesticide Poisoning

Denis Traore, Tyler B. Draeger, and P. Thirumalaikolandusubramanian

Key Points
1. Emergency management may be initiated following directions on packaging or material data safety sheets (MSDS) for the pesticide if available.
2. Pesticide formulations are usually mixed with other substances, necessitating consultation with poison centre or medical toxicologist.
3. Poisoned patients should always receive full mental health evaluation to determine intent and should be assessed for potential towards repetition.

Introduction

Pesticides are chemical compounds used commercially, in agriculture and in households worldwide. They aim at controlling or eliminating insects, animals, fungi and other microorganisms. This chapter introduces the major agents within each category of pesticides—insecticides, rodenticides and avicides, fungicides and herbicides. Each year, approximately seven million people worldwide experience pesticide poisoning—353,000 of which die [1].

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Insecticides

Insecticides are chemicals, naturally occurring or synthesised, which are used to kill insects.

Acetylcholinesterase Inhibitors (ACI) (Organophosphates and Carbamates)

- Widely used as insecticides.
- Organophosphates: ester derivatives of phosphoric acid—OP(OR).
- Carbamates: ester derivatives of carbamic acid—$HOC(O)NH_2$.
- Annual worldwide exposure of humans is an estimated three million—10 % of which die.

Mechanism of Toxicity

- Inhibit acetylcholine esterase (AChE) and neuropathy target esterases (NTE).
- Impairment of synaptic transmission.
- Symptoms are secondary to choline excess.
- Inhibition by organophosphates is irreversible.
- Carbamates transiently inhibit AChE.

Clinical Features

- Cholinergic manifestations:
  - A common mnemonic is DUMBELS:

    Defecation (diarrhoea)
    Urination
    Miosis
    Bradycardia, bronchorrhoea and bronchospasm
    Emesis
    Lacrimation
    Salivation

Patients may present with abdominal cramps, wheezing, diaphoresis and hypotension and cardiac arrhythmias

- Nicotinic manifestations:
  - Muscle weakness
  - Fasciculation
  - Tremor
  - Muscle paralysis

Further signs are tachycardia, hypertension, pallor and mydriasis [1].

- Central nervous system manifestations:
  - Seizures
  - Altered mental status, possibly leading to coma
  - Respiratory failure
  - Organophosphate-induced delayed neuropathy—polyneuropathy 1–3 weeks post-exposure

    Believed to result from NTE inhibition
    Characterised by muscle dysfunction and paraesthesias
    May become a chronic condition
  - Intermediate neurologic syndrome—24–96 h post-exposure in up to 40 % of patients

    Depressed deep tendon reflexes, weak neck flexion, respiratory insufficiency, weakness of proximal muscles and cranial nerve symptoms

Investigations and Diagnosis [1]

- RBC AChE level: marker of exposure correlates well with severity.
- FBC, LFTs and U&Es.
- ECG (rule out arrhythmias).
- CXR (rule out aspiration secondary to bronchorrhoea).

Treatment

- Atropine—1–3 mg IV in adults and 0.02 mg/kg in children to resolve the muscarinic symptoms.
  - If no improvements within the first five minutes, atropine dose is to be doubled.
  - Severe cases may require hundreds of milligrams for atropinisation.
- Slow infusion of oxime loading dose over 20 min.
  - Oximes reactivate cholinesterase.
  - For organophosphate poisoning.
  - Limited use in carbamate poisoning.
  - Pralidoxime: loading dose—30 mg/kg IV.
  - Maintenance dose—8 mg/kg/h.
  - Obidoxime: loading dose—4 mg/kg IV.
    Maintenance dose—0.5 mg/kg/h.
- Benzodiazepines for seizures.
- Gastric lavage or activated charcoal within 2 h of ingestion.

Organochlorides

- Previously widely used in developed countries and still used extensively in developing nations.
- Chlorinated organic hydrocarbons (R-Cl).
- Most well known is probably DDT (dichlorodiphenyltrichloroethane).

Mechanism of Toxicity

- Antagonise chloride transport in GABAergic neurons through inhibition of Ca^{2+}/Mg^{2+}ATPase within the CNS resulting in overstimulation [2]

Clinical Features

- Characterised by CNS overstimulation:
  - Seizures; abdominal discomfort; nausea and vomiting; headaches; hyperaesthesia particularly in the extremities, face and tongue; hyperactivity; discoordination and dizziness; confusion; and myoclonus
  - Convulsions
  - Toxic to the liver, kidneys and lungs
  - Rhabdomyolysis with high doses [2]

Investigations and Diagnosis

- Gas chromatography may be used to detect organochlorides but may be impractical.
- Full neurological examination is necessary.
- Organ-specific markers such as liver transaminases to detect end-organ damage.

Treatment

- Prevent further absorption and manage the clinical manifestations [2].
  - Cholestyramine and sucrose polyester to increase faecal excretion [3].
  - Bowel irrigation at the discretion of a medical toxicologist.
  - Activated charcoal may prevent binding, but not adequately studied [2].
- Aggressive therapy with benzodiazepines or barbiturates for seizures (phenytoin generally not effective) [2].
- Haemoperfusion is shown effective in removing organochlorides from the blood [2].

Pyrethroid Compounds

- Derivatives of (1R,3R)-trans-chrysanthemic acid
- Extensively used worldwide
- Frequently mixed with other pesticides to increase effective spectrum
- Currently used in many common household brands

Mechanism of Toxicity

- Delayed closing of sodium channels during repolarisation resulting in an inward sodium current lowering action potential threshold [4]
- At very high levels, acts on GABAergic neurons
- May further act on protein kinases resulting in additional calcium and neurotransmitter release
- Directly neurotoxic, particularly in the sciatic and posterior tibial nerves resulting in damage resembling Wallerian degeneration

Clinical Features

- Dermal paraesthesias, muscular fasciculation, listlessness, dizziness, fatigue, headache and loss of appetite [5].
- Altered consciousness, seizures and comas at high doses.
- Dysphagia, sore throat, mouth ulcerations, epigastric pain and vomiting when ingested.
  - Aspiration pneumonitis and pulmonary oedema may occur [6].
  - Hepatic and renal dysfunction also seen [6].
- Cardiotoxicity rare, but intermittent to persistent sinus arrest with escape junctional rhythm observed [7].
- Leukocytosis may be seen [6].

Investigations and Diagnosis

- Gas chromatography confirmatory only, may not be practical
- Careful neurological testing required
- ABG to detect anion gap metabolic acidosis
- ECG, renal function and liver function testing for end-organ damage
- Blood count for leucocytosis [6]

Treatment

- Paraesthesia treated with irrigation vitamin E containing creams or local anaesthetics [8]
- Chloride channel agonists for seizures
  - Phenobarbitone effective at 25 % anaesthetic dose
  - Higher doses not effective against seizures [9]

Rodenticides and Avicides

Employed against rodents and birds in households and agriculture

Warfarin and Warfarin Derivatives (Superwarfarins)

- Also known as coumarins or colloquially as "rat poisoning"
- Possess anticoagulative properties
- Molecular formula: $C_{19}H_{16}O_4$

Mechanism of Toxicity [10]

- Vitamin K activates the coagulation factors II, VII, IX and X and anticoagulant proteins C and S.
- Warfarins inhibit vitamin K_1-2,3 reductase, required for vitamin K reactivation.
- Symptoms occur once active vitamin K-dependent factors have become depleted.

Clinical Features [10]

- Anticoagulant effects within 12–48 h after ingestion.
- Superwarfarins are more potent and cause worse symptomatology.
- Epistaxis, flank pain +/− haematuria, intracranial bleeding, gastrointestinal bleeding, haemoptysis, gingival bleeding, menorrhagia and ecchymosis.

Investigations and Diagnosis

- FBC, LFT, PT/PTT/INR, type and cross
- Levels of vitamin K, factors II, VII, IX and X and PIVKA II (protein-induced in vitamin K absence)
- Acetaminophen and toxicology screen

- Non-contrast head CT
- Mixing study—distinguishes coagulation factor deficiency from a circulating anticoagulant

Treatment

- If asymptomatic: observation and INR taken after 48 h
- If elevated INR without bleeding signs: oral vitamin K_1 (1–5 mg per day in children and 20 mg per day in adults initially, monitor INR for long-term dose determination)
- If actively bleeding patient: vitamin K_1IV (<1 mg/min) and FFP (15–30 mg/kg)
  - May also consider prothrombin complex concentrate (PCC) or recombinant factor VIIa
  - Haematology and toxicology consult

Calciferol (Vitamin D)

- High quantity required for human toxicity
- Important role in calcium and phosphate homeostasis
- Molecular formula: $C_{27}H_{44}O$

Mechanism of Toxicity

- Calcium and phosphate absorption from the small intestine
- Causes bone to secrete calcium into the blood and stimulates renal tubular reabsorption of phosphate
- Toxicity exerted through hypercalcaemic action

Clinical Features

- Symptom onset is gradual.
- Hypercalcaemia, confusion, renal insufficiency, anorexia, abdominal pain, nausea and vomiting, arrhythmias, systemic metastatic calcifications, osteomalacia, lethargy and constipation.

Investigations and Diagnosis

- FBC, U&E and ECG

Treatment

- Management targets hypercalcaemia.
- Mild to moderate hypercalcaemia(<3–3.5 mmol/L)—no initial treatment.
- Severe hypercalcaemia (>3.5 mmol/L)—normal saline infusion (200–300 ml/h or to maintain urine output of 100–150 ml/h), furosemide, steroids, calcitonin and bisphosphonates as needed.

Sodium Fluoroacetate

- Highly toxic agent.
- Lethal dose for humans is 2–10 mg/kg [11].
- Molecular formula: ($C_2H_2FNaO_2$).

Mechanism of Toxicity [11]

- Impairs a host of metabolic pathways
- Mimics acetyl-CoA to enter and halt the tricarboxylic acid (TCA) cycle
- Hinders metabolic pathways of fatty acids, urea and glucose

Clinical Features [11]

- Symptom onset may be delayed up to 6 h.
- Hypocalcaemia, lactic acidosis, nausea and vomiting, abdominal pain, diaphoresis, altered mental status, seizure, coma, reversible renal failure, hypotension and respiratory depression.
- Cardiac manifestations: dysrhythmias.
- ECG changes—nonspecific ST segment and T wave changes or QTc prolongation.

Investigations and Diagnosis

- FBC and U&E—specifically, repeated calcium levels
- Repeated arterial or venous blood gases and ECG

Treatment

- No antidote exists.
- Supportive management.
- Any asymptomatic patient ought to be monitored for 24 h.

Chloralose

- Used for control of nuisance birds and rodents
- Chemical formula: (R)-1,2-O-(2,2,2-trichloroethylidene)-α-D-glucofuranose

Mechanism of Toxicity

- Binds allosteric sites in GABAergic neurons
- Potentiates affinity for the $GABA_A$ receptor resulting in CNS depression

Clinical Features

- Coma, extremity myoclonus, generalised convulsions and tracheobronchial hypersecretion.
- Liver injury and rhabdomyolysis may be observed.
- May result in acute heart failure with hypokinesia, ST depression and elevated troponin.
- Pulmonary oedema also observed [12].

Investigations and Diagnosis

- Confirmed by gas chromatography-mass spectroscopy (GC-MS) or ^{1}H-nuclear magnetic resonance (^{1}H-NMR)
- Electrocardiogram, echocardiogram and cardiac blood markers to assess cardiac function
- Chest x-ray to assess lungs for pulmonary oedema
- Liver function testing and measurement of creatine phosphokinase MM (CK-MM) levels to assess liver and monitor for rhabdomyolysis [12]

Treatment

- Gastric lavage and activated charcoal within first 2 h [12].
- Nitrates and diuretics for pulmonary oedema [12].
- Atropine if no cardiac contraindications may be used with caution for tracheobronchial hypersecretions.
- Benzodiazepines for extremity myoclonus and convulsions.

Thallium Rodenticide

- Elemental metal (Tl), historically used extensively as a rodenticide
- Continued use as rodenticide in developing countries
- Still used extensively in manufacturing

Mechanism of Toxicity

- Substituted in membrane transport proteins and intracellular processes for potassium due to similar diameter
  - Binds Na-K ATPase with 10× affinity of potassium disrupting Na-K homeostasis
- Absorbed rapidly and efficiently through mucous membranes
- Excreted renally
- Thought to damage mitochondrial membrane by disrupting the sulphhydryl groups [13]
- May cause insoluble riboflavin complex formation producing riboflavin deficiency via sequestration
- Associated with axonal nerve damage

Clinical Features

- Initially patients may experience abdominal pain, nausea and vomiting, diarrhoea or constipation, sialorrhea and increased thirst [14].
- Subsequently, patients typically manifest neurological symptoms.
  - Paraesthesias, hyperaesthesia, hyperalgesia and progressive weakness originating in distal extremities (particularly great toe) [14]
  - Diplopia, nystagmus, changes in colour perception and blindness due to optic neuritis frequently observed [14]
- Delirium, restlessness, hallucinations and delusions are common, especially in children [14].
- Convulsions in severe cases.
- Late (≈2 weeks) manifestations include yellow acneiform lesions of the face and ichthyotic lesions on the dorsum of the hands and feet and hyperkeratotic lesions of the soles of the feet and palms of the hands.
- Alopecia is a characteristic late manifestation [14].
- Thallium is hepatotoxic, nephrotoxic and cardiotoxic [14].

Investigations and Diagnosis

- Unexplained neuropathy in the presence of alopecia or skin manifestations.
- Urine or plasma analysis for presence of thallium is confirmatory.

- Anaemia, leucocytosis, thrombocytopenia, elevated creatinine, elevated LDH, hypokalaemia and elevated liver transaminases are frequently seen [14].

Treatment

- Gastric lavage with activated charcoal within the first 4 h of ingestion.
- Oral administration of Prussian blue 250 mg/kg daily divided into three to four doses [13].
  - Combine with 5–10 % mannitol for paralytic ileus [13].
- Charcoal haemoperfusion during first 48 h.
- Haemodialysis with potassium supplementation for later stages of intoxication.
- *Dithiocarbamate and forced potassium dieresis may exacerbate neurological symptoms* [13].

Fungicides

Fungicides inhibit fungal growth and development. Agriculture makes significant use of these compounds.

Pentachlorophenol

- Used in wood, leather, toilet paper, agriculture and oil plant products as a preservative and fungicide
- Empiric formula: C_6HCl_5O, also available as a sodium salt (C_6Cl_5NaO) and ester
- Used throughout the world

Mechanism of Toxicity

- Uncoupler of the electron transport chain.
- Prevent phosphate uptake by mitochondria during oxidation of alpha-ketoglutarate.

Clinical Features

- Pyrexia along with profuse sweating, tachypnoea, weakness, tachycardia, irritability and lethargy.
- Coma and convulsions with severe intoxication [15].
- Epigastric pain with ingestion [16].
- Renal failure, liver failure and cardiac failure may occur [15].

Investigations and Diagnosis

- One-dimensional thin-layer chromatography of serum is confirmatory.
- Progressive metabolic acidosis, proteinuria and elevated plasma urea.
- Chest x-ray similar to bronchiolitis or pneumonia [15].

Treatment

- Management of pyrexia, fluid resuscitation and correction of electrolyte disturbances
- Exchange transfusion [15] and forced diuresis with furosemide and mannitol [16] shown effective

Thiocarbamates

- Widely used internationally
- Exist in two isomeric forms: S-thiocarbamates:$RSC(=O)NR_2$ and O-thiocarbamates: $ROC(=S)NR_2$
- Used in preservation of seeds and seedlings, turfgrass, fruits and vegetables and ornamental plants

Mechanism of Toxicity

- Competes with nicotinamide adenine dinucleotide (NAD+) binding, preventing oxidation of acetaldehyde to acetate [17]
- Influences vesicular transport within cells causing glutamate release and contributing to basal ganglia lesions [17]
- Highly toxic to dopaminergic neurons and shown to induce accumulation of copper within the hippocampus and cerebellum, resulting in neurotoxicity [18]
  - Mitochondrial uncoupling leading to perturbed respiration in dopaminergic and GABAergic neurons
  - Cellular destruction via the formation of reactive oxygen species

Clinical Features

- Irritation of the mucosal surfaces (eyes, nose, throat, etc.).
- Coughing and dyspnoea.
- Nausea, vomiting and headache similar to migraines.

- Exposure to thiocarbamate solution can cause erythema and burns at higher concentrations, and some patients may experience urticarial reaction.
- Epigastric pain with ingestion.

Investigations and Diagnosis

- Detected by measuring carbon disulphide levels in patients breath

Treatment

- Supportive care with adequate hydration

Organomercury Compounds

- One of the most toxic pesticides
- Major agents—methyl mercury (CH_3Hg^+), methoxyethyl mercury ($C_3H_7HgO^+$) and phenyl mercuric acetate ($C_8H_8HgO_2$)

Mechanism of Toxicity

- Absorbed through the skin, lungs or gut
- Predilection to accumulate in red blood cells and the nervous system
- Long half-life in the human body (45–56 days)

Clinical Features

- Metallic taste, numbness and tingling in the hands and face, tremor, headache, fatigue, altered mental status, incoordination, slurred speech, loss of proprioception, hearing loss, impaired vision and muscle spasticity or rigidity

Investigations and Diagnosis

- FBC and U&Es
- Mercury blood and tissue levels
  - >5 μg/dL is diagnostic for acute exposure.

Treatment

- Acute poisoning—succimer (DMSA) proves most effective.
- *Dimercaprol* (*BAL*) *exacerbates the neurologic manifestations.*
- EDTA is inefficacious.

Organotin Compounds

- Used to control blights
- Major agents—triphenyltin ($C_{18}H_{15}Sn$), fentin hydroxide ($C_{18}H_{16}OSn$), fentin chloride [$(C_6H_5)_3SnCl$] and fentin acetate ($C_{20}H_{18}O_2Sn$)

Mechanism of Toxicity

- Limited skin and gastrointestinal absorption
- Irritates the skin, eyes and respiratory tract
- Toxic to the central nervous system

Clinical Features

- Headache, epigastric pain, nausea, vomiting, dizziness, convulsions, loss of consciousness, photophobia, mental disturbances and glycosuria secondary to blood sugar elevations

Investigations and Diagnosis

- Diagnosis relies heavily on clinical history with congruent clinical manifestations.
- FBC and U&E.

Treatment

- No antidote exists.
- Chelating agents show no efficacy.
- Supportive management only.

Herbicides

Employed in agriculture; herbicides curtail pest plant species and are frequently formulated with toxic components.

2,4-Dichlorophenoxyacetic Acid

- 2,4-Dichlorophenoxyacetic acid (2,4-D) used extensively for broadleaf weed control in various plant species
- Most used herbicide in many developed countries
- Molecular formula: $C_8H_6Cl_2O_3$

Mechanism of Toxicity

- Causes disturbance to the hydrophobic phospholipid bilayer in cell membranes resulting in cellular dysfunction and the development of echinocyte formation in erythrocytes.
- Causes disorganisation of the Golgi apparatus, perturbation in the function of microtubules and inhibition of synthesis of complex gangliosides.
- Acts as a false neurotransmitter mimicking acetylcholine.
- Strongly inhibits cytochrome c reductase and succinate dehydrogenase and uncouples the electron transport chain in hepatic mitochondria resulting in cellular damage [19].

Clinical Features

- Nausea and vomiting, confusion, aggression and tachypnoea are common [20].
- Loss of deep tendon reflex, tachycardia and pyrexia with diaphoresis, vasodilatation and coma are common later signs [20].
- Correlation between exposure and chloracne.

Investigations and Diagnosis

- Gas chromatography of plasma and urine samples is confirmatory [20].
- ST depression on ECG with prolonged QT interval and T wave inversion raises suspicion in conjunction with elevations in creatinine phosphokinase [20].
- Isolated elevation in urea; elevated ALT and AST levels are frequent.

Treatment

- Primarily supportive.
- Alkaline diuresis is shown effective [20].

Glyphosate

- One of the most widely used herbicides in the developed world
- Used in conjunction with plants engineered to be resistant to the chemical
- Molecular formula: $C_3H_8NO_5P$

Mechanism of Toxicity

- Engineered to be minimally toxic to humans and other animals
- Primary deleterious effect from surfactant [21]:
  - Polyethoxylated tallow amine (POETA) is a common surfactant.
  - Causes membrane disruption and inhibits cellular respiration leading to cellular necrosis [22].

Clinical Features

- Poorly absorbed through the skin
- Hypotension, mental deterioration, abdominal pain and diarrhoea, nausea and vomiting are common.
- Respiratory failure, acute kidney injury, severe acidosis and cardiac arrhythmias including junctional rhythms with premature complexes, ventricular and supraventricular tachycardias, bradyarrhythmias and atrial fibrillation are also seen [21, 23].
- Erosion of the gastrointestinal tract, dysphasia and pharyngeal pain and gastrointestinal haemorrhage in ingestion [23].

Investigations and Diagnosis

- Acute kidney injury, hyperkalaemia, pulmonary oedema and metabolic acidosis are indicative of poor prognosis [24].
- Gas and high-performance liquid chromatography to identify the presence and quantity of glyphosate in the plasma is only useful for confirmation [24].

Treatment

- No specific antidote.
- Observe patients for a minimum of 24 h.

- It is controversial whether haemodialysis is useful [24].
- Consider gastric lavage and administration of activated charcoal within the first hour of ingestion (limited evidence) [23].
- Refractory hypotension may be treated with intravenous fat emulsion therapy [23].

Paraquats and Diquats

- Class: bipyridyl herbicides
- Sole pesticide to cause severe mucosal burns
- Molecular formula: paraquat ($C_{12}H_{14}N_2^{2+}$) and diquat ($C_{12}H_{12}N_2$)

Mechanism of Toxicity

- Corrosive agents
- Rapidly absorbed once ingested.
- Reach most organs through the blood circulation—the lungs, heart, kidney and liver are most vulnerable.
- Create reactive oxygen species, destroying tissues via lipid peroxidation.
- Paraquat accumulates in alveolar cells—necrosis and pulmonary fibrosis ensue.

Clinical Features

- Symptom onset within 6–12 h
- Topical exposure: skin ulceration, erythema and blistering; eye irritation and ulceration
- Inhalation exposure: throat burns, lung damage and epistaxis
- Oral ingestion:
  - Small to medium dose (<15 ml 20 % solution)—symptoms within days to weeks: pulmonary fibrosis, nausea, vomiting, diarrhoea and failure of the liver, heart and kidneys.
  - Large dose (>15 ml 20 % solution) presents acutely: shock, seizure, acute kidney injury (AKI), confusion and coma, respiratory failure, perforation of viscera, haematemesis, abdominal pain and dysphagia.
- Complaints of "burning" skin carry an increased risk of lethality.

Investigations and Diagnosis

- Urine or serum paraquat/diquat levels
- FBC, LFTs, U&E, serial ABG, coagulation profile and blood sugar levels—every 6–12 h
- Chest x-ray, ECG and potentially OGD

Treatment

- No antidote exists.
- Supportive management only.

General prognostic guidelines for paraquat ingestion:

- <20 mg/kg—none to moderate symptoms, complete recovery
- 20–40 mg/kg—death secondary to pulmonary fibrosis within weeks
- >40–50 mg/kg—death within 1–5 days secondary to multi-organ failure, cardiogenic shock and corrosion of gastrointestinal tract.

Cross-Classified Pesticides

These pesticides possess characteristics of two or more categories of pesticide.

Chloropicrin

- Chemical formula: CCl_3NO_2
- Used widely against plant diseases, insects and nematodes protecting vegetable, citrus and field crops and ornamental plants
- Also used in the treatment of wood from pests, such as termites
- Historically used as a rodenticide

Mechanism of Toxicity

- Inhibition of pyruvate dehydrogenase and succinate dehydrogenase
- Activates endoplasmic mechanisms and chaperone proteins involved with protein misfolding
  - Misfolding of sulphhydryl-containing proteins [25]
- Shown to cause oxidative damage due to reactive oxygen species [26]
- Irritation to epithelial surfaces on gross scale
  - Inhalation results in pulmonary oedema

Clinical Features

- Eye irritation, sore throat and headache, shortness of breath and cough.
- Other findings include hallucinations, tachycardia and persistent chest wall pain with increased plasma creatinine phosphokinase MM (CK-MM).

- Acute pulmonary oedema may be observed in severe cases of inhalation.
- Ingestion of chloropicrin is extraordinarily rare.

Investigations and Diagnosis

- Gas-liquid chromatography and GC-MS can be used to detect chloropicrin in exposed tissues, but may not be practical.
- Chest radiography indicated to assess for pulmonary oedema.
- CK-MM levels and cardiac function testing to assess for muscle and cardiac damage [26].

Treatment

- N-acetylcysteine may be beneficial [26].
- Continuous irrigation to exposed areas for minimum of 15 min.
- Ensure airway patency and treat potential bronchospasms with bronchodilators.
- Observe for a minimum of 24–48 h for possible delayed onset pulmonary oedema.

Arsenic

- Inorganic forms—arsenite (AsO_3^{3-}) and arsenate (AsO_4^{3-}) highly toxic [27].
- Chronic exposure to arsenic via deep-water wells poses a public health problem in many countries.
- Ingestion of 100–300 mg—high risk of mortality.

Mechanism of Toxicity [27]

- Binds to red blood cells, which distribute arsenic to multiple organ systems
- Interferes with several enzymes
- Inhibition of gluconeogenesis, glutathione metabolism, glucose uptake and fatty acid oxidation
- Crosses placenta

Clinical Features

- Acute toxicity (symptoms within minutes to hours):
  - Nausea, vomiting, cholera-like diarrhoea, haemorrhagic gastroenteritis, hypotension, tachycardia, dysrhythmias and myocardial ischaemia.

  - Shock, pulmonary oedema, acute renal failure, encephalopathy, seizure and dysaesthesia.
  - Inhalation causes acute haemolytic anaemia, jaundice, abdominal pain and acute renal failure.
- Subacute toxicity (symptoms within 1–3 weeks):
  - Headache, altered mental status, sensory and motor neuropathy, QTc prolongation, pancytopenia, cough, alveolar infiltrates, rash, alopecia, periorbital oedema and Mees lines.
- Chronic toxicity (defined as continued low-level exposure):
  - Hyper- or hypopigmented keratosis, Bowen disease and squamous and basal cell carcinoma
  - Hypertension, fatigue, malaise, leucopenia, anaemia, peripheral arterial disease, non-cirrhotic portal hypertension, diabetes mellitus and lung cancer

Investigations and Diagnosis

- FBC, U&E, abdominal x-ray, ECG and 24-h urine arsenic levels (collected in a metal-free container)
- Nerve conduction studies
- Urine hCG in premenopausal females

Treatment [23]

- Chelation agents:
  1. Unithiol IV (first line)—slow infusion of 3–5 mg/kg for 20 min every 4 h
  2. Dimercaprol (alternatively)—3–5 mg/kg IM every 4–6 h
- Once patient is stable—switch to an oral chelating agent, either unithiol (4–8 mg/kg every 6 h) or succimer (7.5 mg/kg every 6 h or 10 mg/kg every 8 h).
- Continuous monitoring of the heart.

Metal Phosphides

- Include zinc, aluminium and magnesium phosphide (Zn_3P_2, AlP and Mg_3P_2, respectively).
- Magnesium and aluminium phosphide are used to kill insects.
- Zinc phosphide is used to control rodents.

Mechanism of Toxicity

- Phosphine produced
- Directly inhibits mitochondrial respiration by disturbing the mitochondrial membrane potential and inhibiting complex IV of the electron transport chain [28].
- Alters mitochondrial morphology reducing its function, thus inhibiting aerobic respiration [28].

Clinical Features

- Typical symptoms include shortness of breath, respiratory pain, headache, lightheadedness, nausea, chest tightness or pain, dizziness, eye irritation, sore throat and cough, nausea and vomiting, abdominal pain, decreased level of consciousness and cyanosis.
- Other manifestations include blurred vision, dizziness, dyspnoea, apnoea and seizures and cardiac arrhythmias (sinus tachycardia, bundle branch block and ST elevation in a Brugada pattern) and left ventricular failure resulting in cardiogenic shock [29].

Investigations and Diagnosis

- Electrolyte imbalances, leucocytosis and hyperglycaemia.
  - Acid-base imbalance (metabolic acidosis or mixed acid-base disturbances).
  - Hypomagnesaemia and hypernatraemia.
  - Other findings include derangements in liver function tests consistent with hepatotoxicity.
- Abdominal x-ray is useful in identifying radiopaque zinc phosphide ingestion.
- ECG abnormalities are shown to be poor prognostic factors.
- Hyperglycaemia useful in assessment of severity of intoxication.

Treatment

- Gastric lavage may be performed using activated charcoal or potassium permanganate.
- A mixture of coconut oil and sodium bicarbonate has also been reported to be successfully used for gastric lavage.
- Liquid paraffin is used to accelerate the excretion of phosphine from the GI tract.
- Treatment of hypomagnesaemia with magnesium sulphate [29].

Prevention

- Though most poisonings resulting in hospitalisations are a result of suicide attempts, majority of non-intentional poisonings result from occupational exposure [30].
- Farmers often mishandle pesticides:
  - In developing countries, containers often written in unfamiliar languages.
  - Many farmers are illiterate.
- In some areas pesticide poisoning results in more deaths than infectious diseases [30].
- About 99 % of deaths from pesticide exposure occur in developing countries [31].
- May decrease poisonings by reducing the use of pesticides worldwide [30].
- Pamphlets or leaflets written in local languages with easily recognisable figures may enhance proper handling.
- Control of sale of pesticides and expenditures on farmer training may further reduce poisonings [30].
- A third of all suicides worldwide committed by means of pesticide poisoning.
- Pilot programmes in Sri Lanka and China to curtail pesticide poisoning suicide attempts:
  - Storage of pesticides by farmers in boxes requiring two keys held by two different individuals to open
- Underlying factors responsible for individuals attempting or intending to attempt suicide should be addressed.
  - All pesticide poisoning patients are to be given a thorough mental health evaluation.

Quick Reference Chart

Pesticide category	Pesticide class	Predominant manifestations	Specific antidote	Treatment
Insecticides	Organophosphates and carbamates	Muscarinic (DUMBELS) Nicotinic CNS	Atropine and oximes	Give atropine—1–3 mg IV in adults and 0.02 mg/ kg in children. Within the first 5 min post infusion, the atropine dose is to be doubled Slowly infuse a loading dose of oximes over a 20-min period Pralidoxime: loading dose—30 mg/kg IV maintenance dose—8 mg/kg/h Obidoxime: loading dose—4 mg/kg IV; maintenance dose—0.5 mg/kg/h
	Organochlorides	GABAergic overstimulation Hepatotoxic, nephrotoxic, pulmotoxic	None	Seizures should be controlled with aggressive therapy with benzodiazepines or barbiturates. Activated charcoal may be administered. Cholestyramine and sucrose polyester has also shown to increase faecal excretion. Haemoperfusion has been shown to be effective. Bowel irrigation may be considered
	Pyrethroid compounds	Neurological overstimulation and neurotoxicity	None	Skin paraesthesia may be treated with washing affected area and using vitamin E containing cream or using local anaesthetics. Seizures may be treated with phenobarbitone at 25 % anaesthetic dose

Pesticide category	Pesticide class	Predominant manifestations	Specific antidote	Treatment
Rodenticides and avicides	Warfarin and derivatives	Coagulopathy	None	Gastric lavage with activated charcoal if within first hour of ingestion Patients with relatively normal INR and no evidence of acute bleeding may be given vitamin K_1 1–5 mg per day in children and 20 mg per day in adults. Monitor INR every 4 h during the first 24 h and then daily thereafter In patients with acute bleeding, give 15–30 mg/kg FFP in addition to vitamin K_1 With severe, ongoing haemorrhage, slowly administer vitamin K_1 intravenously at less than 1 mg/min in conjunction with FFP and prothrombin complex concentrate (PCC) [alternatively, recombinant factor VIIa in place of PCC may also be considered]
	Calciferol	Hypercalcaemia	None	Treatment of hypercalcaemia Mild to moderate hypercalcaemia (calcium level <3–3.5 mmol/L), no initial treatment is necessary Severe hypercalcaemia (calcium level >3.5 mmol/L) initially infuse normal saline at 200–300 ml/h and titrate to maintain a urine output of 100–150 ml/h Give furosemide, steroids, calcitonin and bisphosphonates as needed
	Sodium fluoroacetate	Metabolic acidosis Hypocalcaemia	None	Gastric lavage with activated charcoal within the first hour of ingestion
	Chloralose	CNS depressant	None	Gastric lavage with activated charcoal within the first 2 h. Treatment of pulmonary oedema with nitrates and diuretics. Extremity myoclonus and convulsions may be treated with benzodiazepines. Atropine may be used with caution for tracheobronchial hypersecretions
	Thallium	Substitution of potassium in cellular processes causing: Neurotoxicity, hepatotoxicity, nephrotoxicity, cardiotoxicity Alopecia and dermatological manifestations	Prussian blue	Gastric lavage with activated charcoal within the first 4 h. Oral administration of Prussian blue 250 mg/kg daily divided into 3–4 doses. Prussian blue should be combined with 5–10 % mannitol for paralytic ileus. Charcoal haemoperfusion if still within 48 h Haemodialysis with potassium supplementation in later stages of intoxication

Fungicides	Pentachlorophenol	Hepatotoxic, nephrotoxic and cardiotoxic	None	Exchange transfusion and diuresis with furosemide and mannitol have been shown to be effective
	Thiocarbamates	Neurotoxic, mitochondrial uncoupling	None	Supportive therapy
	Organomercury compounds	Metallic taste Neurotoxic	Succimer (DMSA)	Irrigation of contaminated areas and supportive therapy and succimer (DMSA) Note: Dimercaprol (BAL) exacerbates the neurologic manifestations and is consequently contraindicated
	Organotin compounds	Neurotoxic	None	Decontamination of affected areas and supportive therapy
Herbicides	2,4-Dichlorophen-oxyacetic acid	Cholinergic overstimulation and electron transport chain uncoupling Neurotoxic and hepatotoxic	None	Alkaline diuresis
	Glyphosate/ POETA	Inhibition of mitochondrial respiration: Cardiotoxicity and nephrotoxicity	None	Supportive therapy. Gastric lavage and administration of activated charcoal within the first hour of ingestion. Intravenous fat emulsion therapy for refractory hypotension
	Paraquats and diquats	Pulmonary fibrosis (paraquat)	None	Irrigation of exposed skin for a minimum of 15 min with soap and water and eyes with isotonic saline for 30 min. Analgesia for pain management Gastric lavage within the first hour with activated charcoal (1–2 g/kg) or fuller's earth (1–2 g/kg in 15 % aqueous solution) Supplemental oxygen must be avoided so to prevent the creation of further reactive oxygen species leading to more tissue damage (if the patient is severely hypoxic, only enough oxygen is to be given to maintain a PaO_2 of 60 mmHg)
Cross-classified pesticides	Chloropicrin	Mucosal irritant	N-acety-lcysteine	Irrigation of affected areas. N-acetylcysteine treatment. Bronchodilators for bronchospasms
	Arsenic	Varies by target organs	Chelating agents: Unithiol, dimercaprol, succimer	Irrigation of contaminated areas Unithiol slow IV infusion of 3–5 mg/kg for 20 min every 4 h or dimercaprol 3–5 mg/kg IM every 4–6 h Once stabilised, switch to an oral chelating agent; unithiol (4–8 mg/kg every 6 h) or succimer (7.5 mg/kg every 6 h or 10 mg/kg every 8 h)
	Metal phosphides	Inhibition of mitochondrial respiration resulting in neurotoxicity, hepatotoxicity, nephrotoxicity, cardiotoxicity	None	Gastric lavage using activated charcoal or potassium permanganate. Alternately, coconut oil and sodium bicarbonate may be used for gastric lavage. Liquid paraffin to accelerate excretion of phosphine. Treatment of hypomagnesaemia with magnesium sulphate

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Part VI
Trauma

Chapter 35
Abdominal Trauma

G.V. Oosthuizen

Key Points
- Blunt trauma and penetrating trauma are two distinct entities, each needing a specific approach to evaluation and management.
- Missed abdominal injury is a major contributor to morbidity and mortality.
- Early involvement of the general surgeon or trauma surgeon is advisable in the management of abdominal trauma.

Blunt Trauma

Introduction

Blunt trauma to the abdomen may present in isolation (e.g. a kick to the abdomen) or in the setting of polytrauma (e.g. pedestrian vs vehicle with multiple fractures and abdominal injuries).

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Clinical Features

The clinical features will be governed by:

- Nature of the intra-abdominal injuries
- Surrounding musculoskeletal injuries
- Time delay from injury to presentation
- Level of consciousness
- Level of systemic analgesia

Signs and symptoms suggestive of abdominal injury include:

- Abdominal pain.
- Physical signs of injury (e.g. bruising [1], contusions, low rib fractures).
- Abdominal tenderness with or without distension.
- In unconscious patients, imaging is critical because abdominal findings are unreliable.

Investigations

The first critical decision with blunt abdominal trauma (provided they are well oxygenated) is whether they are haemodynamically stable.

- Haemodynamically *unstable* patients: rapid and active search for blood loss, external and internal (chest, pelvis and long bones).
- Focused assessment with sonography for trauma (FAST) [2] or, if not available, diagnostic peritoneal lavage (DPL) [3] should follow.
- Haemodynamically *stable* polytrauma patients: single-contrast CT of the abdomen [4, 5] (IV contrast only) [6], unless duodenal or rectal injury possible, then oral or rectal contrast added (see Fig. 35.1).
- Fully cooperative patients with isolated abdominal trauma: serial abdominal examination.
- If bladder injury is possible (pelvic fracture with or without haematuria), cystogram should be performed [7].

Treatment

- Positive FAST or DPL with haemodynamically unstable patient: emergency laparotomy

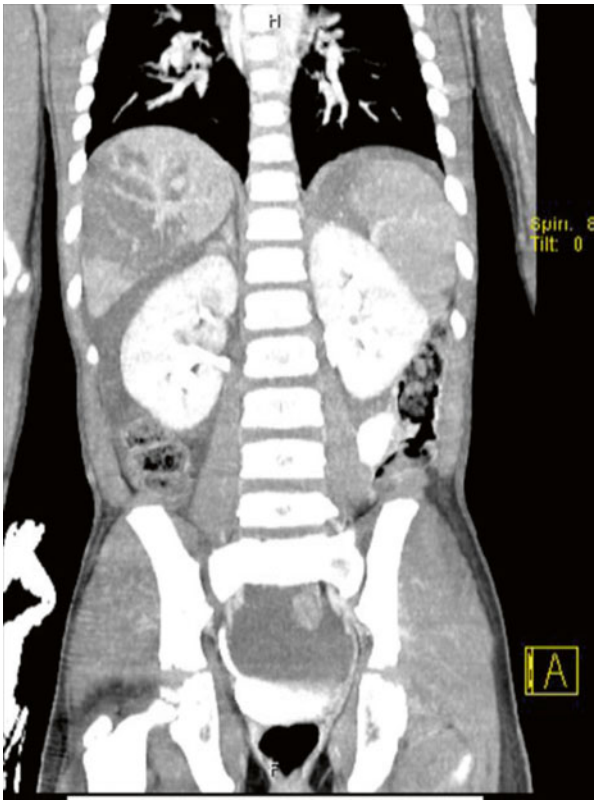
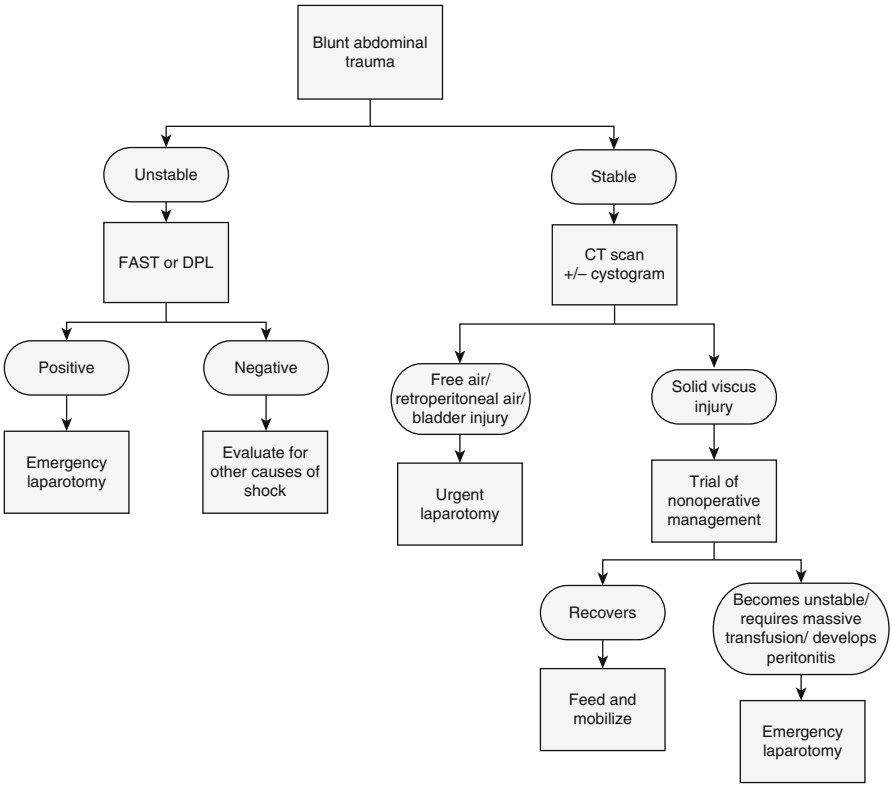


Fig. 35.1 CT abdomen demonstrating liver injury

- CT findings that mandate emergency laparotomy: free air in the peritoneal cavity; retroperitoneal air in the region of the duodenum, right or left colon or rectum and devascularizing injuries of the liver or kidneys.
- Solid visceral damage, even high-grade injuries, may be managed nonoperatively in a high-care setting, as long as the clinical condition remains stable [8, 9] (urine output, improving lactate levels/base deficit and no ongoing blood transfusion to maintain Hb>10 g/dl).
- Arterial blush on CT: consider catheter-directed angiography, with embolization of the bleeder, especially if initially presented with hypotension [10].

Algorithm of management



Penetrating Trauma

Introduction

Stab wounds and gunshot wounds are separate entities.

- Haemodynamically *unstable*, or with established *peritonitis*, and/or *free air* under the diaphragm on erect chest X-ray *requires emergency laparotomy*.
- The discussion that follows applies to patients that are haemodynamically stable and do not have established peritonitis or free air on chest X-ray and are being managed in an appropriately resourced setting.

Clinical Features

Careful examination must identify all wounds to the abdominal region (below the nipples anteriorly/below the tips of the scapulae posteriorly, extending to the pelvic brim). The anatomical region of stab wounds and the trajectory of gunshot wounds bear heavily on their management.

Investigations

Stab Wounds

- Erect chest X-ray (or supine lateral shoot through of the abdomen) for free air under the diaphragm.
- Plain abdominal X-ray is useless unless the knife is impaled.
- If vascular injury is suspected (e.g. stab left iliac fossa with decreased distal pulses), CT angiogram is warranted.
- For suspected bladder injury (suprapubic stab wound with/without haematuria), a cystogram should be considered.
- For suspected rectal injury (pelvic stab in the region of the rectum): rigid sigmoidoscopy, or radiographic contrast enema, or CT with rectal contrast.

Gunshot Wounds

- All of the above also applies to gunshot wounds.
- With gunshot wounds, it is important to try and establish the trajectory of the bullets, as they are predictive of the injuries present.
- Plain abdominal X-ray should be done with markers at the site of each wound (for instance, a paperclip can be taped to each anterior gunshot wound and opened up paperclip can be taped to each posterior gunshot wound [11]).
- With gunshot wounds there should be an even number of holes or holes plus bullets found. If the total number is uneven, further X-rays should be done of other body regions, looking for a missing bullet.
- A bullet that has travelled to another body region may be indicative of a tract with serious injuries.
- Single-contrast CT of the abdomen is warranted for *stable* patients with an isolated gunshot to the right upper quadrant (entry and exit in the right upper quadrant) [12]. If CT shows isolated injury to the liver (or liver and kidney),

such an injury may be managed nonoperatively, as described above, for blunt injuries to solid viscera.
- The obese patient with a gunshot tract suspected as tangential (i.e. traversed only extra-abdominal adipose tissue) may also undergo CT if available. If CT demonstrates an extra-abdominal tract, the patient may be safely discharged. If CT is not available for such a patient, they should be admitted and observed as described below, for stab wounds.

Treatment

Stab Wounds

- No uniform approach to the management of stab wounds of the abdomen.
- First world centres, especially where penetrating abdominal trauma is uncommon, CT and/or laparoscopy or laparotomy threshold may be very low.
- Selective nonoperative (SNOM) approaches apply to busy centres that cannot afford to image and/or operate on all penetrating abdominal injuries and are evidence-based.

Anterior Stab Wounds

Patients with a stab wound to the anterior abdomen are generally tender to palpation in the area of the stab wound, but the rest of the abdomen is soft and non-tender (Table 35.1) [13].

- If tenderness spreads further away from the stab site involving the rest of the abdomen, hollow viscus injury is highly likely and urgent laparotomy must follow.
- If after 24 h there has been no clinical change, the patient may be fed and discharged.
- Exception: left upper quadrant stab wounds; stomach content may take much longer to lead to peritonitis (compared with injuries to the small bowel or colon). Longer observation after the test feed is advocated.
- Evisceration of omentum: clean, reduce and close the abdomen. Then be managed as above.

Table 35.1 Management of anterior stab wounds

Admission
Analgesia (IVI morphine acceptable)
Nil by mouth
Serial clinical review – by the same clinician, 2 hourly

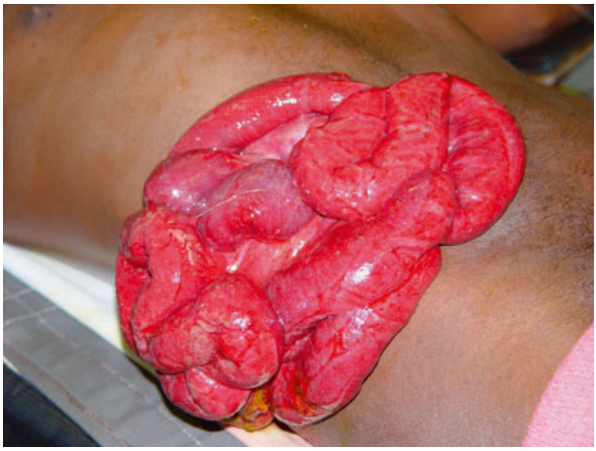


Fig. 35.2 Eviscerated small bowel

- Eviscerated bowel, likelihood of injury is high, urgent laparotomy (Fig. 35.2):
  - If the patient requires transfer, reduce the bowel and close the skin.
  - Small abdominal wall defect and the protruding bowel are strangulated but viable; enlarge the defect and reduce the bowel.
  - Not reducing strangulated bowel may lead to significant dead bowel by the time of laparotomy.
- Stab wounds to the left lower ribcage (below the level of the nipple anteriorly or the tip of the scapula posteriorly) have about a 30 % likelihood of an injury to the diaphragm [14].
  - Chest X-ray should be inspected for signs of herniation of hollow viscera through the diaphragm.
  - Absence thereof does not rule out a diaphragmatic defect.
  - Admit and observe as described above and if remains stable, semi-elective laparoscopy to inspect the left diaphragm is done. If a defect is found, it is repaired laparoscopically or open.

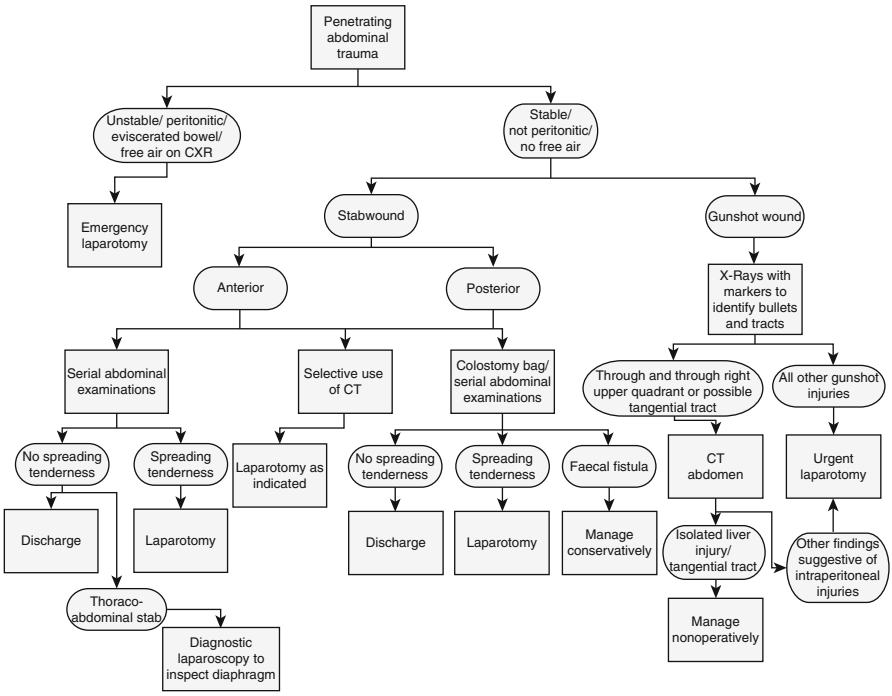
Posterior Stab Wounds

- A selective nonoperative approach [15] is advocated.
- Do not suture posterior wounds: a retroperitoneal hollow viscus injury can progress to necrotizing fasciitis. Place a colostomy bag over the stab site.
- The patient should be admitted and observed (including serial abdominal examinations). Most patients will have no injury requiring laparotomy.
- Delayed onset of a colonic fistula generally settles on conservative management.
- Patients with haematuria must be imaged: single-contrast CT including CT-IVP phase. Management as per the chapter on urological injury.

Gunshot Wounds

- High likelihood of hollow visceral injury warranting urgent laparotomy.
- Exceptions include isolated liver injuries and tangential injuries as discussed above. Recent literature supports SNOM in selected patients [16].

Management Algorithm



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Chapter 36
Early Management of Trauma

Timothy Craig Hardcastle

Key Points
- Trauma is the second leading cause of mortality in all ages and the leading cause of mortality under age 44 years.
- Undertake evaluation and resuscitation in a systematic and methodical manner – to identify and treat according to priority.
- Treat injuries that are life-threatening as they are found and not at the end of the evaluation.
- Newer resuscitation strategies are associated with better outcome.
- Surgical intervention may take priority over completing the entire assessment – intervene and return to the assessment later.

Introduction

Trauma remains the major killer of persons in the age groups 15–44 years of age in the majority of lower- and middle-income countries, with many factors influencing the causes and consequences, and the second biggest killer, after infective diseases, in children. Systems of prehospital care, early adequate resuscitation and timely

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definitive care result in improved survival, with the need for rehabilitation to re-integrate the survivors into gainful societal activity [1–3].

Pathophysiology and Mechanism of Injury [4]

There are basically two mechanisms of injury in any trauma situation, although they may co-exist, with the actual clinical features primarily governed by the organ injury, be it solid organ, hollow viscus or soft tissue and bone:

Blunt trauma such as:

- Motor vehicle and other transport-related incidents
- Assault with a blunt object
- Fall onto a blunt object
- Fall from a height

Penetrating trauma:

- Broken glass penetration
- Knife wound
- Low-energy bullet wound (velocity has become less important)
- High-energy bullet wound

Combined:

- Bomb blast
- Some motor vehicle accidents with impalement of body parts

The pathophysiology of injury is twofold, namely, that caused by the injurious event and that resulting from the inflammatory reaction mounted by the body in response to the injury. Both of these processes may make the patient prone to sepsis or multiple organ dysfunctions [5].

Injury patterns may be predictable from knowing the site of impact and the forces generated. A good history from emergency medical service (EMS) staff is helpful here.

Approach to Trauma Evaluation and Resuscitation

Initial Approach

Severe trauma does not allow for the opportunity to follow the normal pattern of medical evaluation, namely, that of history, followed by examination, side room tests and followed by imaging and laboratory investigations. One must focus on saving the patient from death.

Table 36.1 Components of a trauma team

Team member	Role [7]
Doctor (1 up to 3)	Performs medical assessment and procedures, FAST scan
Nurse (1 up to 3)	Performs vital signs assessment and nursing procedures
Scribe	Documentation (can be performed by a nurse)
Porter	Patient movement, take bloods to laboratory, assists with log-roll
Radiographer	Bedside imaging
Technician	Point-of-care laboratory (can be done by Dr or nurse too)
Surgeon/emergency specialist	Team captain/decision-maker (role can be performed by any medical doctor if specialist is not available

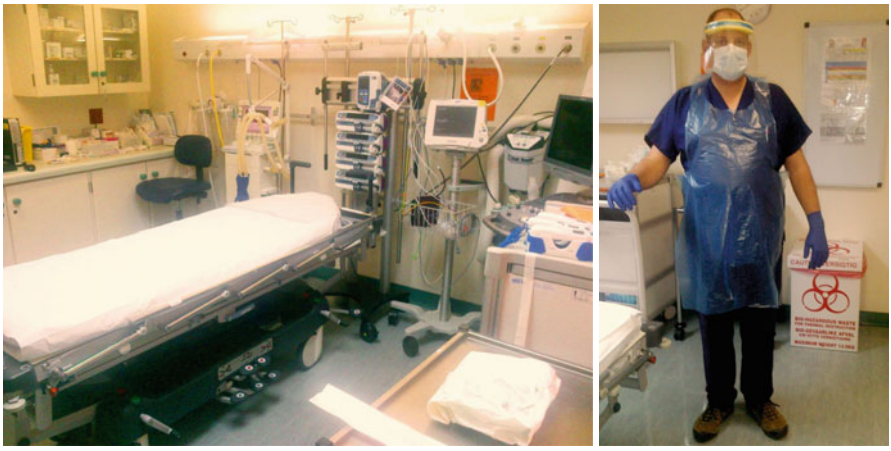

Fig. 36.1 The correct environment and personal protective equipment is essential

Trauma is best managed by a team of committed providers, with no proof that smaller teams fair worse than larger teams, as long as all members of the team understand and perform their role [6] (Table 36.1).

Standard precautions should be followed by all members of the trauma team including wearing: mask and visor, gloves, apron or scrub gown and overshoes or boots.

It is essential that any medical equipment in the emergency department, drugs and fluids are checked regularly for functional and expiry issues. This should be performed at least daily by both nurses and doctors to avoid delays to care when the patient arrives. If pre-notification is received prior to the patient's arrival, equipment should be made ready and blood bank placed on standby for urgent transfusion supplies if the history is suggestive (Fig. 36.1).

There are a number of systematic trauma evaluation programmes, such as the International Trauma Life Support® or Prehospital Trauma Life Support® (for EMS personnel), National Trauma Management Course (IATSIC) or Primary Trauma Care (Oxford University), and the traditional gold standard has been the Advanced Trauma Life Support (ATLS®) for doctors (or the Trauma Nurse Core Course) approach as it is simple and ONE safe way: the ATLS® has become progressively more evidence-based over time [8–14].

Handover

The use of an acronym type handover system such as the De-MIST (demographics, mechanism, injuries, signs, treatment) is useful to impart essential knowledge before patient assessment without undue delay.

- Demographics (age, gender, where from, etc.)
- Mechanism of injury (what happened)
- Injuries identified prehospital/at referring facility
- Signs and symptoms (and any results from another facility)
- Treatment given till the patient arrived at your facility

Patient Care Sequence

The aim of the approach to the treatment of the injured is to attempt to identify life-threatening injuries, intervene to treat these as they are found and then progress to the next part of the resuscitation. The traditional approach is based on so-called primary survey, followed by secondary survey and eventually a tertiary survey in the admitting service ward or ICU.

- Primary survey – A, B, C, D and E – is the resuscitation phase.
- History (AMPLE) and baseline special exams are performed after life-saving intervention.
- Secondary survey follows: the head-to-toe review of all injuries – aim to avoid missed injury.
- Definitive care traditionally follows after stabilisation with disposition to a trauma surgeon to coordinate care.

What has become clear over the past 10 years is that this approach works well for the patient that does not need urgent surgical intervention; however, the newer literature espouses the need to undertake life-saving surgery during the primary survey if needed and to continue thereafter with further assessment. An example of this would be immediate sternotomy or thoracotomy to relieve a pericardial tamponade or laparotomy for control of intra-abdominal bleeding. An even more recent addition is urgent extraperitoneal pelvic packing for exsanguinating pelvic fracture haemorrhage [15].

A: Airway and C-Spinal Control [16]

There is always the assumption with blunt trauma that there is an injury to the C-spine and, therefore, to the cord, so absolute care is required. This is much less true for penetrating trauma, where airway control must not be delayed or impeded by spinal motion restriction.

Assess the airway for:

- Patency
- Maintenance: Is it maintained or is intervention and support needed?
- Identification of potentially threatened airway – intervene early to reduce complications.
- Airway-specific injuries.

DO NOT use head-tilt chin lift: JAW THRUST is the basic airway manoeuvre of choice (Tables 36.2, 36.3, and 36.4).

The main aim of airway management is a DEFINITIVE airway, which is usually defined as a cuffed endotracheal tube below the vocal cords with the cuff inflated, placed either transoral, transnasal or surgical (cricothyroidotomy in emergency/tracheostomy in elective) (Fig. 36.2).

If two attempts by two operators are unsuccessful, then proceed to RESCUE OPTION, currently using a laryngeal mask airway (LMA) is the procedure of choice as this allows time to prepare for elective tracheostomy, while not a definitive airway, it offers more protection than an oropharyngeal tube. Alternatively if there is an inadequate seal, one can proceed to needle cricothyroidotomy as a temporary solution – using jet-ventilation will allow about 20 min to obtain further assistance.

Table 36.2 Airway challenges

Airway structure	Common injuries/cause for airway compromise
Lumen	Lacerations of the lips, mouth/tongue/soft palate Foreign bodies
Wall	Soft tissue swelling/haematoma under tongue/in face Mandible, maxillary or other facial fractures Hyoid bone, laryngeal or cricoid fracture, tracheal punctures
Extra-luminal	Local compressive neck swelling (haematoma) displacing trachea Central injuries causing lower level of consciousness and airway loss

Table 36.3 Indications for intubation

A. Airway not maintained or is threatened by swelling or foreign body
B. Need for ventilation and non-invasive ventilation unlikely to succeed, hypoxia present
C. Shocked patient, who will need systemic support
D. Comatose patient (Glasgow coma scale <9/15)

Table 36.4 Steps in trauma airway management

Pre-oxygenate – for about 2–3 min, aiming to have saturation >95 %
Drug assisted (rapid sequence intubation) is best:
Etomidate or ketamine are equivalent
Succinylcholine or rocuronium for paralysis
Remove the C-collar if present – manual in-line support of the spine
Cricoid pressure (BURP) is recommended unless there is active vomiting
Use bimanual manipulation to obtain best view
Assess Cormack-Lehane and Mallampati gradings
Consider use of a bougie or video laryngoscope if available
Consider two-handed laryngoscope
Place endotracheal tube through cords under vision = best way to know it is in
Secure the tube to the mouth (do not tie in the OPA, only the ETT) at around 23 cm at the teeth for most adults
Check the placement with end-tidal CO_2 or colour change monitor and confirm correct depth with chest X-ray

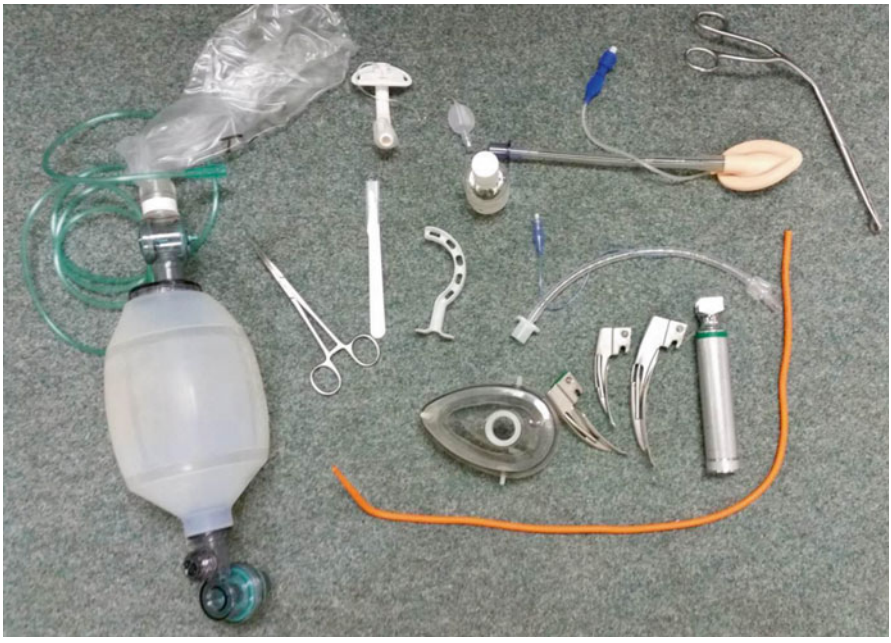

Fig. 36.2 Equipment for trauma airway management

Should one face the dreaded "cannot intubate-cannot ventilate" scenario, then one is forced to move towards the surgical definitive airway, namely, cricothyroidotomy. So-called slash tracheostomy is not recommended. Only once the airway is secured, proceed to breathing.

B: Breathing and Ventilation

Assessment of breathing must be independent of airway. If the patient is breathing, the assessment of rate, mechanism (e.g. flail chest/diaphragmatic breathing) and oxygenation will guide the need for either chest tubes or ventilation. The apnoeic patient requires not only airway management but full ventilation support.

The primary survey goal of breathing assessment serves to exclude life-threatening chest injuries: treatment is determined by clinical findings (Table 36.5).

Indications for ventilation:

- The patient with apnoea
- Adult RR >30 or <10/min
- Poor mechanism with severe flail chest and contusion >25 % lung volume on CXR/CT
- Blood gas: pO_2 <8 kPa/60 mmHg or pCO_2 >6,5 kPa/50 mmHg
- GCS <9/15

Placement of a chest tube:

- Fourth to fifth ICS, anterior to mid-axillary line ("safe triangle").
- Clean and drape. Give plenty of local anaesthetic – give it time to work!
- Incise skin and underlying fat with the scalpel only.

Table 36.5 Life-threatening chest injury

Pathology	Signs/symptoms	Initial treatment	Further treatment
Tension pneumothorax	Decreased air entry Hypotensive Hyperresonance	Needle decompression	Intercostal chest tube drain
Open pneumothorax	Sucking chest wound	Occlusive 3-way dressing	Intercostal chest tube drain and close wound
Massive haemothorax: >1,000 ml stat or 400 ml/h *X*2-4 h	Dull to percussion Hypotension Pallor Collapsed neck veins	Insert intercostal chest tube drain with autotransfusion option ideal	Consider urgent thoracotomy for surgical haemorrhage control
Lung contusion/flail chest/rib fractures	Hypoxia, tachypnoea, crepitations bilateral, diffuse patchy white out on CXR – get worse over next 24 h	Check for occult pneumo- and haemothorax. Ensure oxygenation and analgesia. Consider CPAP/non-invasive ventilation	Intubation and early ventilation with recruitment (8 ml/kg) manoeuvres and PEEP, progressing to lung-protective ventilation
Major airway rupture	Bilateral surgical emphysema and persistent pneumothorax with "fallen lung sign" on X-ray	Chest decompression with >1 chest tube drain, intubation and ventilation, consider single lung ventilation	Surgical correction if ventilation unable to oxygenate

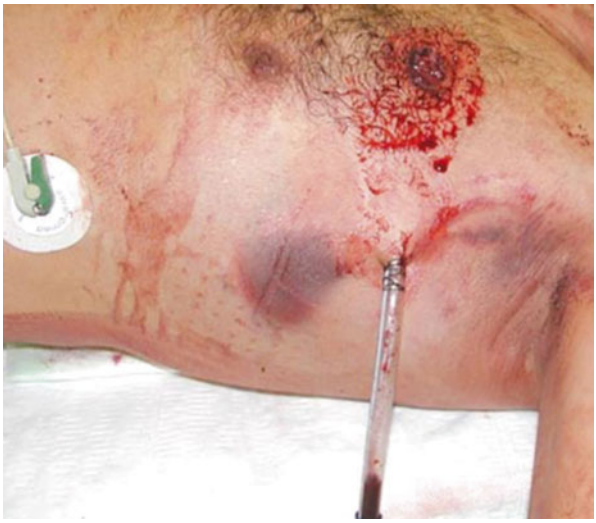

Fig. 36.3 Well-placed chest tube after gunshot chest

- Take a blunt-tipped spreadable forceps and run it over the underling fifth rib until you feel the pleural "pop" and then open the clamp and pull back while open, spreading the intercostal muscles off the underlying bone – make a hole big enough for a finger to check for intrapleural adhesions – cause no further harm!
- DO NOT USE ANY SHARP-POINTED TROCHARS – remove these devices if they are in the drain tube; preferably use a clear PVC-type Argyle catheter with at least two drain holes, less chance of blockage.
- Place the tip of the drain on the blunt spreadable forceps and direct it into the chest aiming always apico-posterior, so that blood can drain from the supine patient and air will collect at the apex.
- Fix the drain to the chest wall with sutures and tape, and connect it to a valve-based [17] or water-seal chest drain set (the advantage of the former: allows for autotransfusion). Use saline in the drain to allow for autotransfuse the blood that drains (Fig. 36.3).

C: Circulation and Haemorrhage Control

The best indicator that a patient is in hypovolaemic shock is the pulse rate: the patient with a tachycardia (adult >120/min) [18] is shocked till proven otherwise. Importantly it is more about perfusion than pressure!

The treatment of bleeding is to STOP the bleeding. It is pointless to resuscitate the patient with fluids, and they bleed onto the floor. Control the bleeding with DIRECT pressure. It may be necessary in C to stop what you are doing, take the patient to the operation room and surgically control the bleeding if it is in the chest, abdomen, pelvis or long bones!!! FAST (focussed assessment sonar in trauma) is a

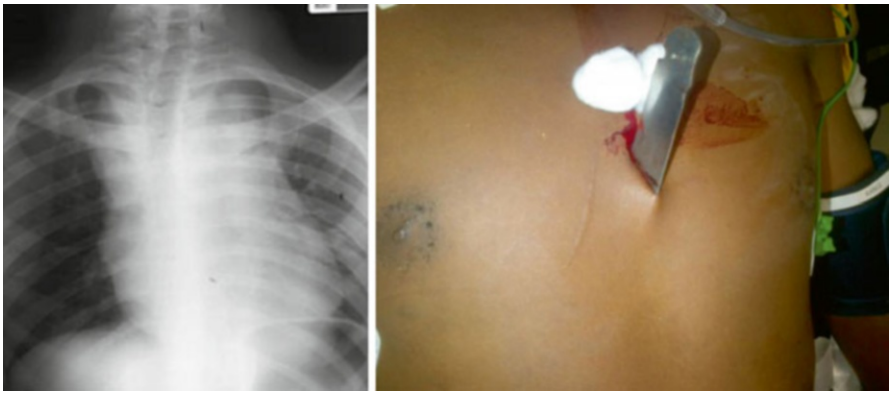

Fig. 36.4 Life-threatening cardiovascular injury: CXR blunt trauma – wide mediastinum and knife wound causing pericardial tamponade

Table 36.6 Traditional classification of shock (Adapted from ATLS® manual)

Percentage blood loss	0–15 %	15–30 %	30–40 %	>40 %
Pulse	Normal	Tachycardia	Severe tachycardia	Slowing to bradycardia
Pulse pressure	Normal	Narrowed	Widened	N/A
BP	Normal	Raised diastolic	Systolic/diastolic drop	Severe hypotension
Urine output	More than 1 ml/kg/h	0,5–1 ml/kg/h	less than 0,5 ml/kg/h	Anuria
Consciousness	Normal	Agitated	Stuporous	Comatose
Resp rate	Normal	20–30	30–40	Slowing

recommended extension of the clinician's armamentarium to identify intra-abdominal haemorrhage in the acutely unstable trauma patient. Laparotomy is the default operation unless the chest is the clear bleeding source [15].

The routine use of procoagulants (rVIIa and tranexamic acid) is currently still controversial outside of specific time frames and guidance from coagulation tests such as ROTEM/TEG.

Apart from major bleeding, one must identify end-organ ischaemia (usually a mark of a peripheral or cervical vascular injury), cardiac tamponade (mainly with penetrating trauma) and possible blunt aortic arch rupture (usually acceleration-deceleration injury) (Fig. 36.4, Table 36.6).

Fluid in resuscitation: traditionally crystalloid fluid in high volumes has been used in fluid resuscitation; however, it is now recognised [19] that it is far better to use blood and blood products in a balanced product ratio giving the patient what they have lost. An initial bolus of up to 2 l of Ringer's lactate (20 ml/kg in children) is acceptable to differentiate responders from non-responders. Consider permissive hypotension, which means keeping the SBP around 90 mmHg until surgical control of bleeding and

then resuscitate fully for obvious noncompressible bleeding in the context of rapid access to surgical facilities. Also consider autotransfusion if capabilities exist. Blood should be given in the 1:1:1 ratio of *red cells*, *to plasma*, *to platelets* – this is a goal, not an absolute target; about 1:2:1 is probably optimal [20, 21]. While restrictive bolus therapy is appropriate for non-responders who can access surgery, large-bore central access (such as dialysis or "sheath introducer" large-bore catheters) and the use of high-capacity giving sets are advised intraoperatively (Fig. 36.5).

The best gauge of whether or not you are gaining the upper hand in the resuscitation is the perfusion of vital organs, such as the heart, brain and kidneys: so urine output is an objective assessment of the degree of your success. Place a urine catheter early to assess this, after doing a rectal exam on the male patient with pelvic trauma to exclude a urethral rupture. Blood gas lactate is the best marker! [22] Aim for lactate <2,5 mmol/l. Base deficit of less than 4 is a good surrogate.

Exclude the life-threatening circulation injuries:

- Cardiac tamponade (Beck's triad): muffled heart sounds and pulsus paradoxus with hypotension and distended neck veins. Treatment is OPEN the chest in the operation room.
- Thoracic aorta rupture with a pseudoaneurysm – good upper limb pulses, weaker lower limb pulses and radio-femoral delay but may have no signs and only be suspected on CXR with a widened mediastinum (>8 cm at T4 in adults). Most die on scene!

Also quickly check the limbs for distal pulses and reduce long bone and pelvic fractures to decrease both pain and fluid loss. Exclude compartment syndromes! Once the obvious bleeding is controlled, look for hidden losses such as the chest, abdomen and pelvis.

Vascular injures to the peripheral circulation may result in limb-threatening situations, and this must be detected early. Early involvement of the SURGEON is essential.

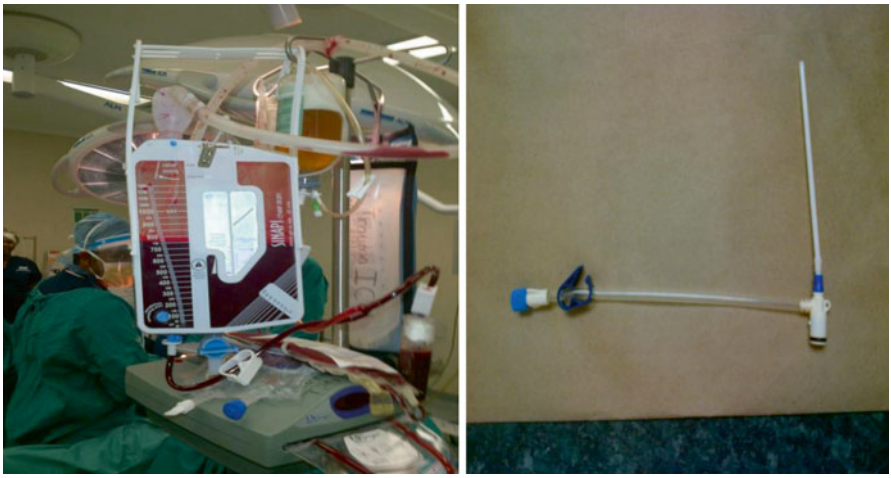

Fig. 36.5 Autotransfusion with chest drain device [17] and large-bore central catheter

D: Disability and Neurological Impairment

During the primary survey there are only THREE things of importance:

- Pupil reactions
- Any obvious neurological deficit: Localising signs
- Glasgow coma score: Eye opening, speech and motor function/15

Any abnormality detected here requires that the patient be supported and further harm prevented by avoiding hypoxia, hypotension and hypothermia. Good A, B and C impact outcome of abnormal D. The additional requirement is for neuroimaging, either a CT scan of ideally the head and spine (Fig. 36.6) [23].

E: Expose the Patient, but Maintain Environmental Control

- Undress them completely – ideally cut off the clothing!
- Keep warm with light blanket, warm fluids and warm room (recommended at least 24 °C) [24].
- Log-roll now and examine the back – get them off the spine-board onto a padded but firm stretcher (5 cm padding adequate).

After addressing the life-threatening injuries, proceed to detailed history and other baseline special examinations.

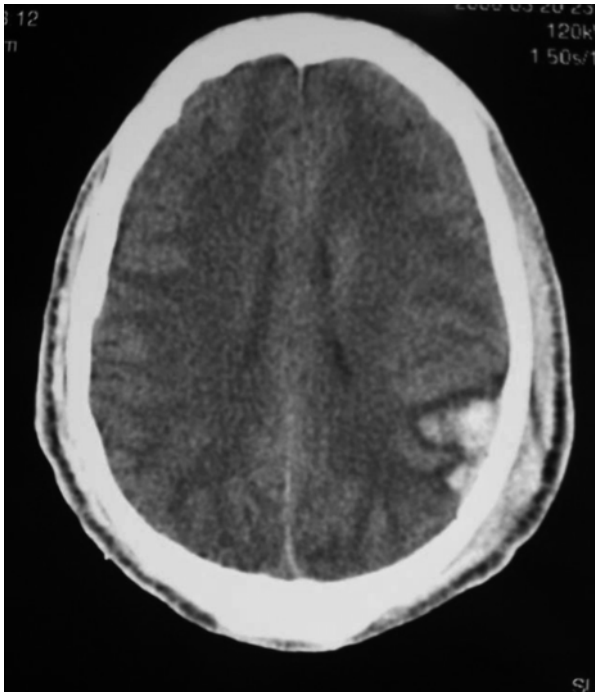

Fig. 36.6 CT head – intracerebral haemorrhage

History

- *A = Allergies*
- *M = Medications used*
- *P = Previous medical/surgical history*
- *L* = Time of the *last meal* before the time of the trauma
- *E = Events surrounding the current admission*

The patient, if conscious, family, onlookers or the EMS staff may provide information. Ask pertinent questions related to the type of incident.

Baseline investigations:

- AP chest and pelvis X-ray are ESSENTIAL (neck X-ray less important if spinal motion restriction maintained and is unnecessary for most penetrating trauma).
- Blood gas, FBC, U&E, crossmatch and urine dipstick, toxicology screen and pregnancy tests are routine as appropriate. TEG/ROTEM coagulation studies are gaining importance (Figs. 36.7 and 36.8).

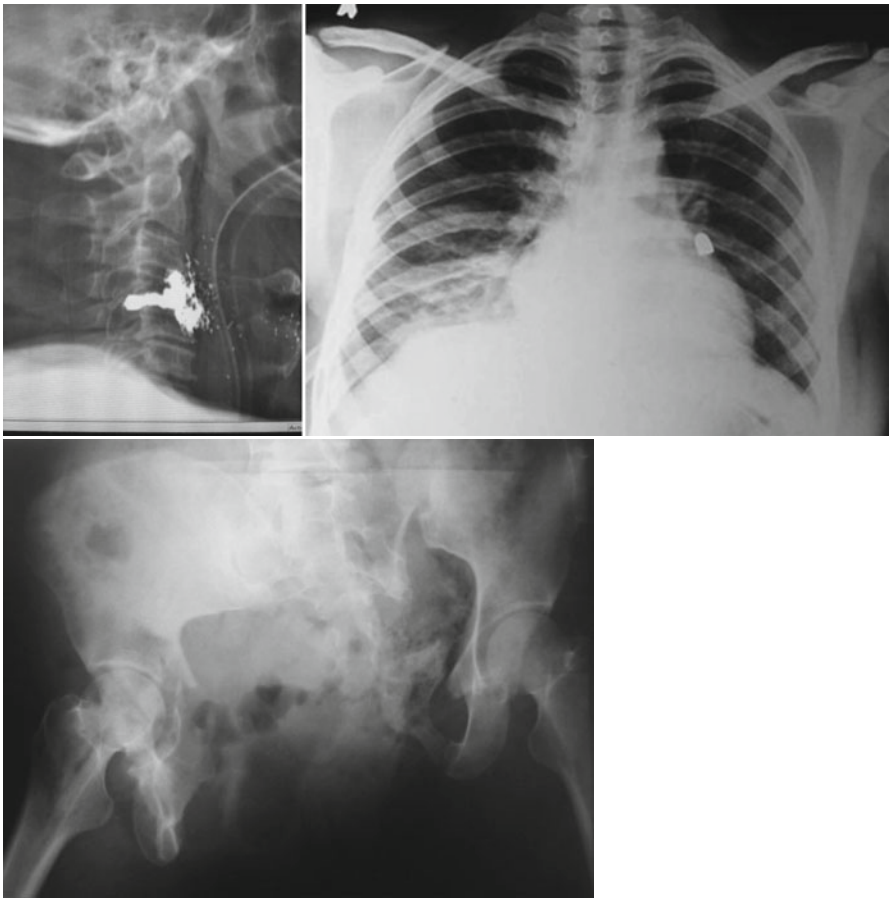

Fig. 36.7 CXR after gunshot, NXR after gunshot, PXR – open book with lateral shear injury

Fig. 36.8 Point-of-care blood gas and coagulation analysers should be part of a well-equipped resuscitation area

Table 36.7 Items to focus on during secondary survey

Body region	Common injury	Precautions/management
Head	Skull base fracture – CSF leak	Head up, exclude C-spine, consider brain injury
Face	Facial bone injury	Airway risk, bleeding
Neck	Vascular – bruits/deficits Pharyngo-oesophageal	Vascular imaging/repair Contrast study/repair
Chest	Simple pneumothorax, small haemothorax, rib fractures, blunt cardiac injury	Treat as found, adequate analgesia
Abdomen	Solid organ injury Hollow viscus injury Perineal injury	CXR or lateral abdominal shoot-thru to exclude free air Must have GCS >12/15 to evaluate clinical tenderness
Limbs	Fractures of the hands, feet and compartment syndromes Soft tissue injury to ligaments and muscle	Imaging and referral Creatine kinase level Venous bicarb

While this is being processed, one can now begin the SECONDARY SURVEY: This is head-to-toe examination of the entire patient with “fingers or probes into every orifice”, so as to not miss any injury, including a “log-roll” if not yet performed (Table 36.7).

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Chapter 37
Face and Neck Trauma

Vivesh Rughubar and Timothy Hardcastle

Key Points
- Management of the airway may be complex.
- Haemorrhage control is essential and often challenging.
- Neck wounds must be adequately evaluated to prevent later complications.
- Blunt neck trauma is an often under-evaluated cause of vascular injury.

The maxillofacial region is divided into:

- Upper face – frontal bone and frontal sinus
- Midface – this is further divided into:
  - Upper midface which includes the zygomatic bones, nasal bones and ethmoids
  - Lower midface comprising the alveolar bone, teeth and palate
- Lower face – the mandible [1]

The management of the patient with maxillofacial or neck injury requires a team effort which includes the trauma surgeon, maxillofacial surgeon, anaesthesiologist, otolaryngologist and plastic surgeon [1]. Different approaches apply to the

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patient with blunt neck or facial injury versus those with penetrating neck trauma in particular. Cervical spine trauma is common and is also addressed in this chapter.

The following systems should be evaluated:

Vitals	Senses	Head	Neck
Airway	Hearing	Scalp	Wounds
Breathing	Vision (including ocular movement)	Subcutaneous tissue	Aerodigestive tract
Perfusion	Smell, eating and talking	Skull	Vascular structures
Glasgow score			Spinal cord

Injury Classification

Le Fort fractures (involve the pterygo-palatine plates)[a]	*Naso-ethmoid fractures*	*Tripod fracture*
Le Fort 1 – hard palate	Nasal bone fracture	Maxilla
Le Fort 2 – midface	Ethmoid sinus fracture	Zygoma
Le Fort 3 – full face		Orbital ridge
Le Fort 4 includes frontal sinus involvement		
Mandible fracture	*Orbital blow-out fracture*	
Body	Inferior orbital wall	
Ramus	Muscle entrapment risk	
Articular surface		

[a]Unilateral or partial injury possible (recent additional classification of LF4)

Management Approach

Maxillofacial injuries rarely require immediate repair. Associated injuries may require intervention. Tuckett et al. have proposed an algorithm adapted from Cogbill et al. for the management of maxillofacial trauma patients as seen in the flow chart (Fig. 37.1).

Stable patients require imaging with preferably CT scan and three-dimensional reconstruction to plan definitive surgical management. An example of how the various injury complexes can all be present in one patient is presented in Fig. 37.2.

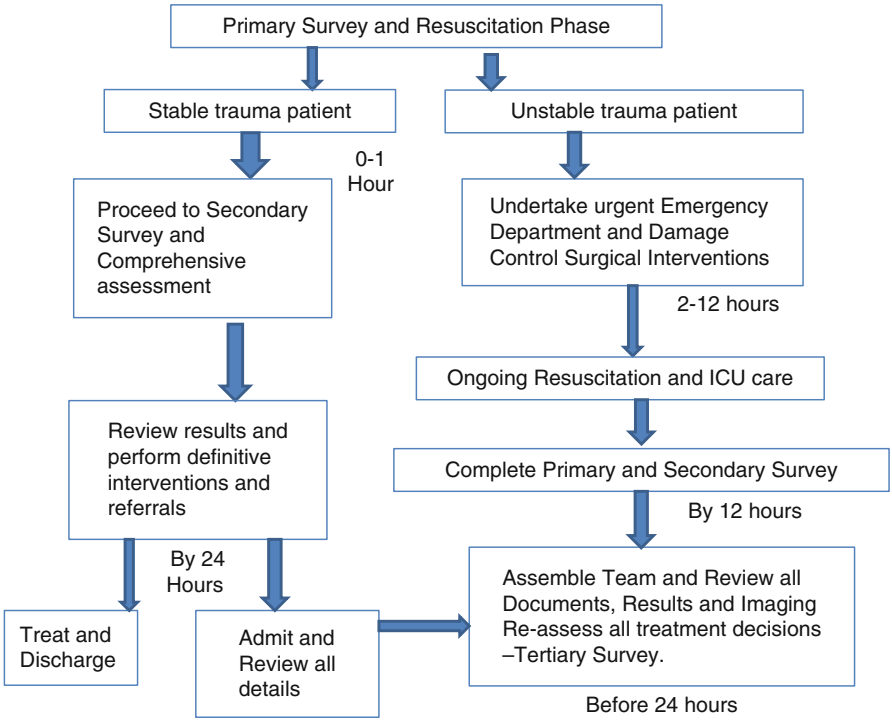

Fig. 37.1 Algorithm for management of maxillofacial trauma

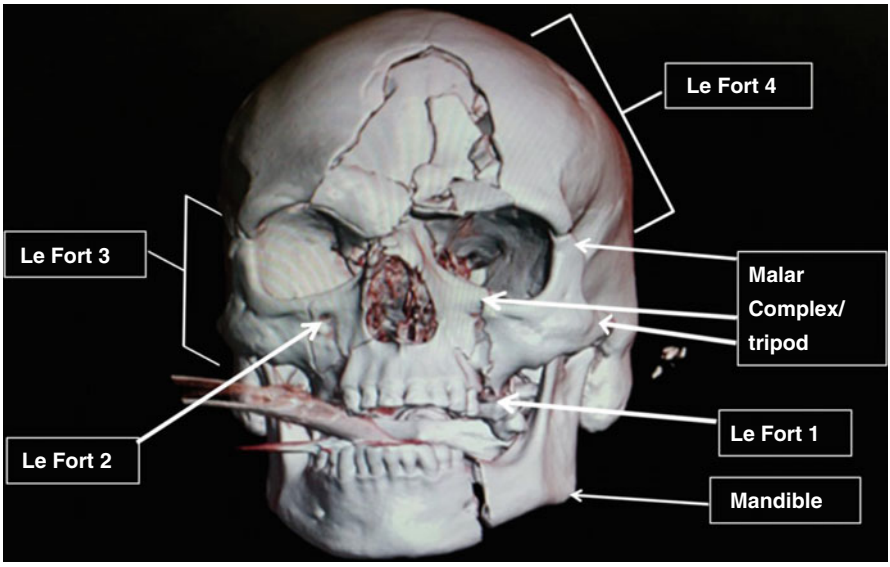

Fig. 37.2 Demonstrates mandible fracture, Le Fort 1, 2, 3 and 4 as well as a left malar-complex (tripod) fracture

Airway Issues

Any injury to the facial skeleton has the potential to place the airway at risk.
Other factors include [1, 2]:

- Level of consciousness.
- Alcohol intoxication.
- Loss of airway reflexes.
- Vomiting [3].
- Supine position or is restrained (Table 37.1).
- Allow the patient to sit up if conscious while preparing for awake intubation. This may not be possible in a patient with a spinal injury [1].
- Endotracheal intubation is the gold standard but results in the loss of verbal communication in a conscious patient and impacts on history taking and clinical assessments. Nasotracheal intubations are generally *not* appropriate.
- Skull base fractures should be considered in all midface fractures especially if there are signs of periorbital ecchymosis, retro-auricular ecchymosis, CSF leaks or seventh nerve palsy [2].
- The laryngeal mask airway is the bale-out option, but consideration of a surgical airway (cricothyroidotomy) is mandatory when an endotracheal intubation has failed [1]. It is generally not recommended to perform a tracheostomy in such emergency scenarios.
- Lowering the head of the bed can allow gravity pooling of blood in the pharynx followed by suctioning preventing aspiration. This may not be tolerated in a conscious patient and may lead to increased intracranial pressure if there is concomitant head injury.

Facial Bleeding

Maxillofacial haemorrhage may be life threatening and requires immediate attention. Bleeding from the maxillofacial region may appear to be excessive, but seldom accounts for hypotension and other causes for blood loss and shock must be investigated.

Table 37.1 Causes of airway obstruction

Bleeding
Fracture displacement
Soft tissue oedema
Foreign bodies (teeth and dentures)
Bilateral mandible fractures impair ability to swallow
Sublingual haematoma
Postero-inferior displacement of the maxilla (manual/digital reduction improves the airway)

- C-spine collars may obscure lacerations of the posterior aspect of the scalp.
- Panfacial and midface fractures tend to bleed excessively due to several anastomoses from bilateral internal and external carotid arteries.
- Swallowed blood can result in vomiting – early orogastric tube placement is essential (Table 37.2)

Figure 37.3 shows a patient with posterior and anterior nasal packing with Foley catheters and bismuth-iodine-paraffin paste gauze for major facial bleeding.

Cervical Spine

In all patients who have sustained maxillofacial injuries, it is important to assume that there is an associated cervical spine injury until clinically or radiologically excluded.

- Between 4 % and 6 % of adults with blunt poly-trauma have a cervical spine injury [8, 9].

Table 37.2 Measures to stop maxillofacial bleeding [4–7]

Direct pressure
Sutures or staples
Oral packing
Anterior nasal packing (Fig. 37.3)
Foley catheter bilateral posterior nasal packing (commercial devices are available) (Fig. 37.3)
Open reduction and internal fixation
Transcatheter angio-embolisation with coils or gelfoam

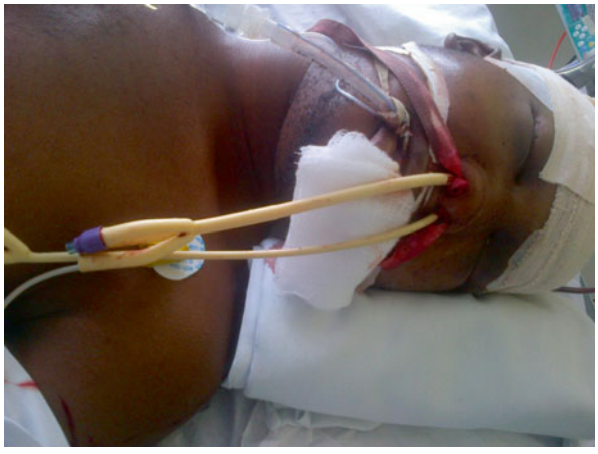

Fig. 37.3 Bilateral Foley posterior nasal packing and anterior packing with "BIPP" gauze

- With facial fractures, up to 10 % have associated cervical spine injuries [10–12].
- Cervical spine motion restriction with a rigid or semi-rigid cervical collar [1].
- Incorrect application could result in airway obstruction especially in patients with anterior neck injuries and mandibular factures.
- It is also recommended to apply a head block with straps in these situations as it allows greater access to the fracture. Placement of spinal collars may result in an increase in intracranial pressure as maxillofacial injuries may be associated with an intracranial injury.
- Exclude spinal injuries clinically if possible. The Canadian C-Spine Rule (CCR) [13] and the National Emergency X-Radiography Utilization Study (NEXUS) [14] are two clinical decision tools that can be used in this situation. Distracting injuries, an altered Glasgow Coma Scale (GCS) and drug and alcohol ingestion could make exclusion difficult if not impossible.
- Computed tomography (CT) is the imaging modality of choice when clinical exclusion of the cervical spine injury is not possible [15–17].
- Midface injuries are commonly associated with C5–C7 disruption, while C1–C4 injury is associated with lower face injuries [18, 19]. The latter is also associated with blunt carotid artery injury.

Eyes

- Globe and orbit injuries occur commonly (up to 13 % and vision-threatening injuries occurring in 0.8 % [20]).
- Related to fractures involving mainly the frontal bones and midface skeleton [21, 22].
- Ocular injuries are not easily identified at initial examination and may often be missed.
- The eyes and related structures should be evaluated after primary survey [21].
  - Assess pupillary reflexes, size and shape. If the pupils are dilated due to pharmacological agents, the dose and time of administration must be documented.
  - Test perception of light.
  - Review the conjunctiva and cornea
  - Look for foreign bodies and hyphaema.
  - Contact lenses should be removed [22].
  - Visual acuity if conscious (count fingers/hand movement/light perception) [14, 23, 24]
  - Oedema, ecchymosis, ptosis, hypertelorism, exophthalmos and enophthalmos must be noted [23].

Proptosis occurs in 3 % of all head injuries [25] and may be easily missed during the examination. Retrobulbar haemorrhage is a reversible cause of proptosis that may occur following orbital injuries [23, 26–28] (Table 37.3).

Table 37.3 Signs of retrobulbar haemorrhage

Proptosis
Diffuse subconjunctival haemorrhage
Elevated intra ocular pressure
If progressive, refer for surgical decompression as it can result in irreversible ischemia in a short time period

Table 37.4 Clinical features suggestive of blunt neck injury

Ligature marks
"Seat-belt sign"
Decreased level of consciousness
Neurological signs of paraplegia or quadriplegia
Respiratory difficulty
Neck haematoma

Table 37.5 Indications for vascular imaging in blunt neck trauma

Fracture of C1–C4
Visible neck wounds or evidence of seat-belt sign
Haematoma present
Signs of TIA/stroke
Bruit
Facial bone fractures

Blow-out fracture of the floor of the orbit may lead to entrapment of periorbital tissue causing diplopia and enophthalmos and may be associated with the stimulation of an occulo-cardiac reflex.

Blunt Neck Injury [29]

Blunt neck injury is mainly twofold: vascular injury with intimal flaps and later occlusion (with subsequent stroke) and crush injury of the laryngo-tracheal tissues. Digestive tract injury is rare. Fractures with cord injury can also occur.

Evaluation of blunt neck trauma depends on mechanism of injury and associated injury profiles. Airway is best controlled by endotracheal intubation and cricothyroidotomy should be avoided in cases of suspected laryngeal or tracheal crush injury (Table 37.4).

The diagnostic approach is as follows:

- Plain radiology must show C1–T1 and AP, and lateral views are needed.
- Open mouth view is done, if not intubated.
- CT scan and 3-D reconstruction is the gold standard.
- CT angiogram of the carotid and vertebral arteries should be performed for any patient exhibiting the signs shown in Table 37.5).

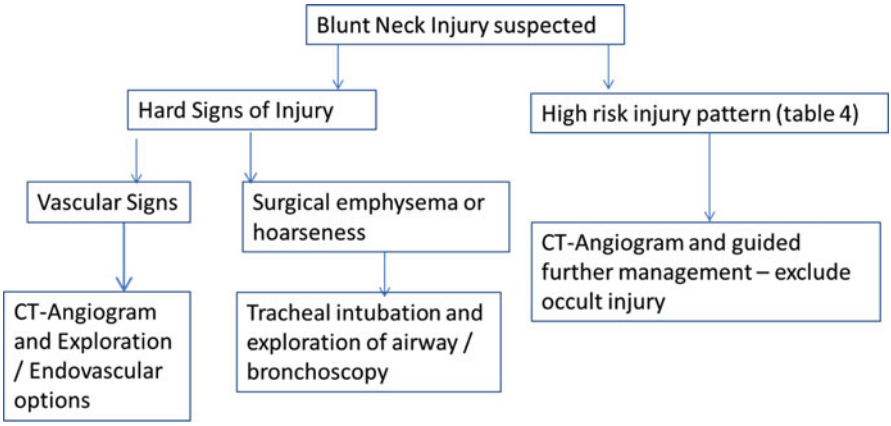

Fig. 37.4 Blunt neck trauma algorithm

Treatment is based on the injuries identified in conjunction with a trauma, ENT or vascular surgeon and early referral is recommended. An algorithm for approaching blunt neck trauma is attached as Fig. 37.4.

Penetrating Neck Trauma

Penetrating injuries to the neck are far more likely to cause injury to the vessels or aerodigestive structures and consequently require a more aggressive approach. Approximately 10 % of all penetrating trauma includes a neck wound.

There are two approaches and these depend on haemodynamic status:

- Unstable or actively bleeding patients require urgent surgery.
  - Airway control should be obtained early.

    Placing an endotracheal tube bulb below the injury may occlude a tracheal injury and enable ventilation.
  - Bleeding may be temporized with Foley catheters [30]. Figure 37.5 demonstrates Foley catheter haemostasis prior to surgery for a bleeding neck wound.
- Delay can lead to neurological injury (stroke).
- Stable patients (normotensive and cooperative) may undergo examination and imaging.

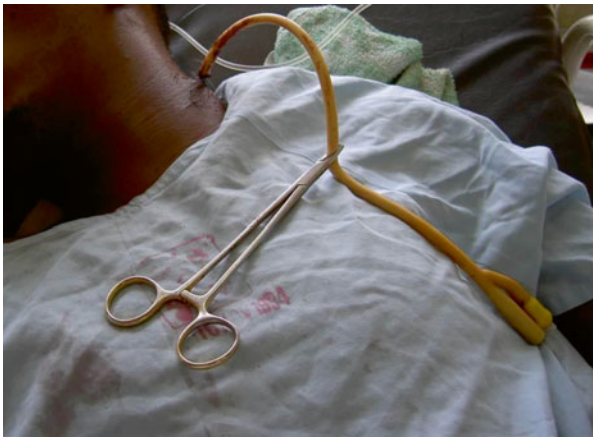

Fig. 37.5 Foley catheter placed into neck wound to compress the bleeding vessel with the inflated catheter balloon

- Two approaches are used:

  Traditional "neck-zone" approach
  Millennial "no-zone" approach [31]

- Hard signs of vascular injury demand imaging and usually need intervention.
- Liberal use of digestive tract imaging (iso-oncotic water-soluble contrast swallowed by the patient who is able to cooperate) [32] is recommended, since missed injury, or delayed oesophageal injury identification, leads to increased morbidity and mortality.
- Signs of aerodigestive injury include:

  Surgical emphysema
  Ipsilateral haemo-pneumothorax
  Saliva draining from the wound
  Dysphonia or dys-/odynophagia

- Endoscopy (oesophageal and bronchoscopic) are used selectively.
- Treatment is directed toward the injury identified if involving the arteries or the oesophagus, while venous injury or airway injury is managed on the basis of selective nonoperative approaches. The surgical techniques are outside the scope of this book. Figure 37.6 shows an example of an angiogram with a carotid artery pseudoaneurysm while Fig. 37.7 shows an example of a contrast swallow.

An algorithm for the assessment and management of penetrating injury is shown in Fig. 37.8.

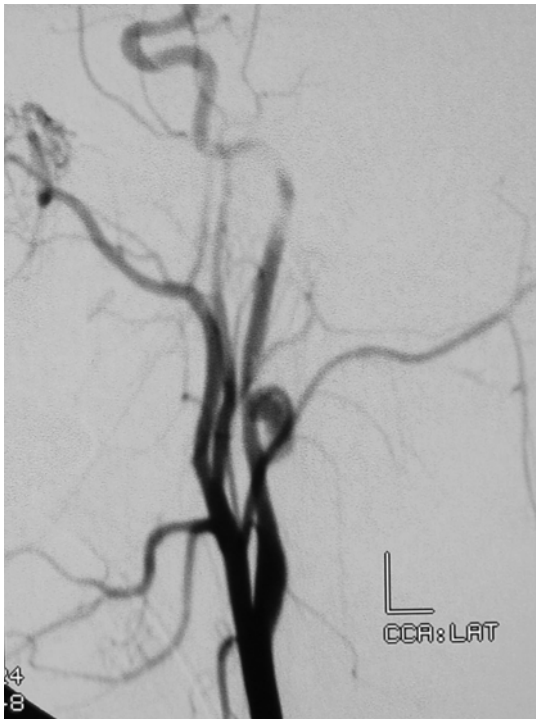

Fig. 37.6 Internal carotid pseudoaneurysm on angiogram after stab wound of neck

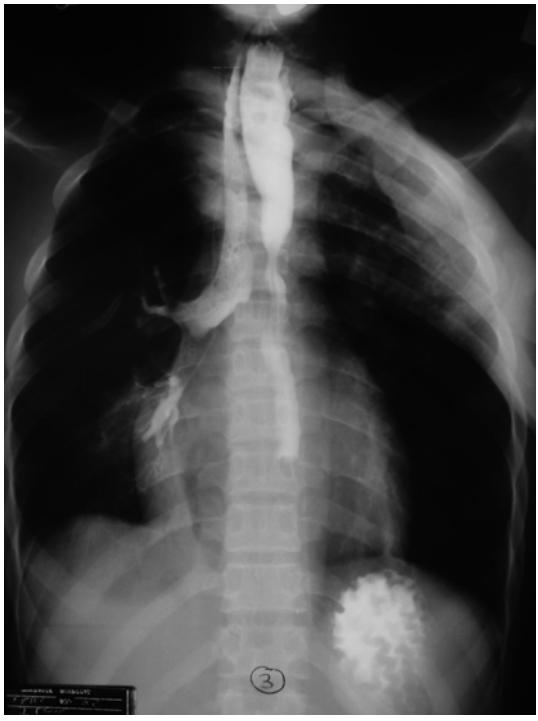

Fig. 37.7 Traumatic tracheaoesophageal fistula after penetrating neck wound

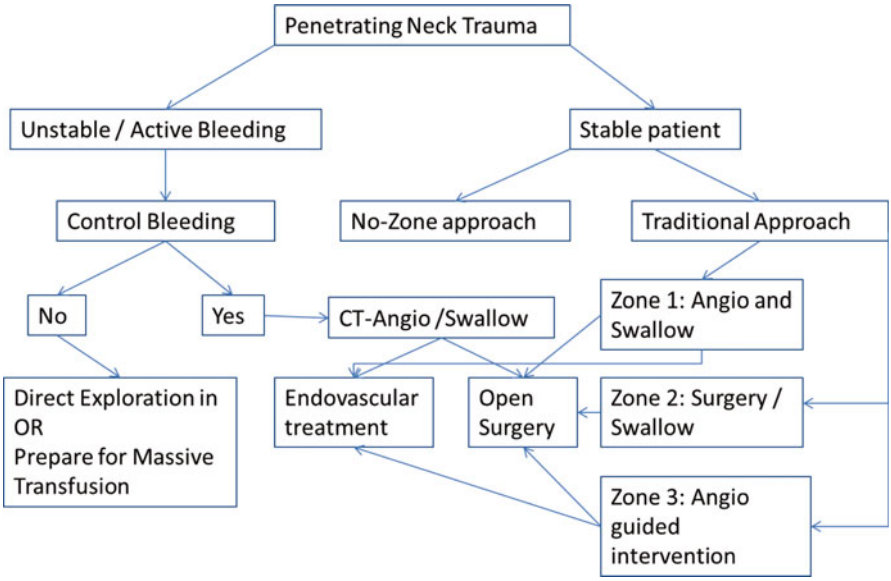

Fig. 37.8 Approach to penetrating neck injury: stable versus unstable

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Chapter 38
Head Injuries

Prashanth Maharaj

Key Points

- Assessment and management of traumatic brain injury according to evidence-based contemporary guidelines are essential in preventing secondary brain injury.
- Prevention of secondary brain injury is the major concern prior to definitive neurosurgical management.
- Utilising evidence-based contemporary guidelines such as Glasgow Coma Scale and The Brain Trauma Foundation guidelines are essential for optimal assessment, in guiding management as well as for prognostic value.
- Computed tomography (CT) scanning is the investigation of choice in traumatic brain injury.

Introduction

Head injuries are a leading cause of morbidity and mortality around the world [1].

The concepts of head injury (HI) and traumatic brain injury (TBI) require clarification.

- Head injury is a non-specific and antiquated term, which includes clinically evident external injuries to face, scalp and calvarium, such as lacerations, contusions, abrasions and fractures, and may or may not be associated with traumatic brain injury.

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- Traumatic brain injury is well defined as an alteration in brain function manifest as confusion, altered level of consciousness, seizure, coma or focal neurological deficit resulting from blunt or penetrating forces to the head [2].

TBI has a preponderance for young males (male to female ratio 4:1) attributed to interpersonal violence and motor vehicle accidents in the "testosterone years" [2].

Mechanisms of injury include road traffic accidents, falls and assaults with significant variation in different populations.

Pathophysiology [3]

Traditionally, TBI has been divided into primary and secondary brain injury.

- Primary brain injury is damage at the time of initial trauma to the brain, is mechanically irreversible and includes brain lacerations, contusions, haemorrhages and avulsions.
- Secondary brain injury is derived from complications initiated by the primary brain injury and includes potentially avoidable entities such as hypoxic-ischaemic injury, cerebral oedema, metabolic dysfunction, alterations in vascular permeability, diminished blood flow, inflammation, diffuse axonal injury and consequences of intracranial hypertension.

Both primary and secondary brain injury can be additionally categorised as focal or diffuse, which is traditionally derived from the presence or absence of radiographic mass lesions on computed tomography. However, the modern understanding of TBI shows that any attempt to conclusively classify brain injury remains a difficult task due to the fact that most TBI consist of a heterogeneous admixture of focal and diffuse damage (Table 38.1).

Table 38.1 Factors affecting secondary brain injury

Intrinsic	Extrinsic
Elevated ICP	Inadequate resuscitation of circulatory shock
Low CBF/CPP	Inadequate oxygen delivery
Systemic hypotension/shock	Aggressive hyperventilation
Reperfusion injury	Anaesthetic agents, alcohol, other drugs
Hypoventilation/hypoxaemia	Nosocomial infections
Brain oedema and mass effect	
Brain herniation	
Brain haemorrhage	
Cerebral arterial vasospasm	
Inflammation	
Hyperthermia	
Chronic systemic illness	

ICP intracranial pressure, *CBF* cerebral blood flow, *CPP* cerebral perfusion pressure

Exogenous insults [4]:

- *Hypotension*: A single episode following TBI has been shown to nearly double risk of death. The duration and number of hypotensive episodes are associated with higher mortality.
- *Hypoxia*: SpO2 <90 % or PaO2 <60 mmHg has been associated with poor outcomes and avoidance hereof advocated in The Brain Trauma Foundation guidelines [5, 8, 9].
- *Hyperglycaemia* may occur following TBI due to sympathetic and hormonal responses to trauma. Blood glucose >11.1 mmol/L correlates with poor outcomes in terms of mortality and long-term outcome.
- *Hypercapnia* and *hypocapnia*: Hypercapnia causes increased cerebral blood flow through vasodilation. In the setting of TBI, reduced intracranial compliance, this vasodilation may increase intracranial pressure significantly and thus reduce cerebral perfusion pressure. Hypocapnia (<30 mmHg) due to hyperventilation, in the first few hours, has demonstrated worse outcomes due to additional underperfusion and ischaemia.
- *Intracranial inflammation* is subject to the Monro-Kellie doctrine which states that the intracranial pressure is a balance of blood-CSF and the brain. The inflammatory response initiated by brain injury by release of cytokines, free radicals and other inflammatory mediators results in alterations in blood-brain barrier permeability, glial swelling and regional as well as global cerebral blood flow changes with resultant brain oedema, and thus the blood flow is either reduced leading to further ischaemia or the CSF is displaced with the risk of herniation and poor outcome.

Cerebral blood flow following trauma undergoes three distinct phases:

1. Hypoperfusion: first 24 h – regional or global which may result in ischaemia due to active muscular vasoconstriction
2. Hyperaemia: 24–72 h – increased cerebral blood flow which results in increased intracranial pressure
3. Vasospasm: days 4–14 – reduced cerebral blood flow with risk of ischaemic cerebral deficits

Initial Management [5]

All head-injured patients should be managed per evidence-based contemporary protocols. The Advanced Trauma Life Support (ATLS) guidelines are favoured in most countries around the world. Specific principles relevant to traumatic brain injury during initial resuscitation include the following:

- Early oral tracheal intubation is required in severe head injuries, GCS <8, loss of laryngeal reflexes, maxillofacial injuries and ventilatory insufficiency (clinical).
- Cervical spine motion restriction: due to a 5–15 % risk of concomitant injury.

- Ventilation should be to avoided hyper- and hypocarbia: goal of 30–35 mmHg as well as ensure adequate oxygenation, i.e. oxygenation saturations >90 % or PaO2 >60 mmHg.
- Hypotension from associated injuries should be actively managed as it is an independent predictor of morbidity and mortality. Blood pressure should be maintained and systolic pressures below 90 mmHg should be avoided.
- Cerebral perfusion pressures of between 50 and 70 mmHg are recommended
- Assessment of level of consciousness (Glasgow Coma Scale), pupil reactivity and motor function are pivotal to further resuscitation, investigation and management.
- Assessment of extracranial injuries per ATLS is vital so as to prevent secondary brain injury.

Brain-Specific Resuscitation Strategies [5]

1. Hyperventilation reduces PaCO2 thus reducing cerebral blood flow and as a result intracranial pressure. If attempted in the initial phase of hypo-perfusion, cerebral hypoxia and cytotoxic oedema may result and worsen. Thus the current recommendation is to aim for PaCO2 to be 30–35 mmHg.
2. Osmotherapy: Osmotically active agents which increase plasma osmolality and increase plasma oncotic pressure thus cause efflux of fluid from traumatised brain tissue.
   (a) Mannitol is effective for control of raised intracranial pressure (0.25–1 g/kg) but the circumstances of use are limited to short-term administration if signs of transtentorial herniation or progressive neurological deterioration are present – whilst awaiting diagnostic investigations (CT scan) and interventions (evacuation of intracranial mass lesions). There is little evidence to advocate its use in any other circumstances. Its use is limited by its diuretic effect and propensity to cause intravascular volume depletion with repeated usage, leading to nephrotoxicity [6].
   (b) Hypertonic saline (3 %) may benefit patients (poly-traumatised) with haemorrhagic shock (small-volume resuscitation) with concomitant traumatic brain injury. It is thus preferred to mannitol in the setting of hypotension in the severely injured patient in reducing intracranial pressure [5, 8, 9].
3. Prophylactic hypothermia: Current literature suggests that its use is not significantly associated with reduced mortality. Hence it cannot at this point be widely advocated [10].
4. Infection prophylaxis is advocated for procedures (including intubation/tracheostomy) as it may reduce incidence of pneumonia (but has no effect on mortality/length of hospital stay). Early tracheostomy may reduce days of mechanical ventilation but does not affect mortality or incidence or nosocomial pneumonia.

Early Outcome Sustaining Interventions

- Once the C-spine has been cleared, elevation of the bedhead to 30–45° head up is advised to improve venous drainage and collar removal is advocated.
- Early seizures are prevented by routine seizure prophylaxis for 1 week, using phenytoin in adults and valproate in children in doses approximating 20 mg/kg. There is no effect on late seizures.
- Early enteral feeding reduces the stress-ulcer risk along with the use of topical mucosal protectants such as sucralphate.
- TBI patients have a high risk of thromboembolic complications and early calf-compression pumps followed by chemoprophylaxis with low-molecular-weight heparin after 48 h is advised.

Classification

Head injuries may be classified based on mechanism of action, severity as well as if injury is focal or diffuse.

Head injuries per ATLS are based on Glasgow Coma Scale [7].

Severity	Glasgow Coma Scale
Minor	13–15
Moderate	9–12
Severe	3–8

Mechanisms may be classified as blunt or penetrating: Penetrating injuries may be classified into high and low velocity. These injuries may be caused by projectiles (bullets) or knives.

Blunt injuries are commonly due to motor or pedestrian vehicle collisions, falls, assault and sports-related injury.

The above mechanisms may produce *focal or diffuse* injuries: Diffuse injuries may range from concussion to diffuse axonal injury. Focal injuries include skull fractures (linear, depressed or compound) and intracranial haematomas.

Investigations [3]

During primary survey:

- Monitoring of blood pressure (systolic >90 mmHg), pulse oximetry (>90 %) and end-tidal CO2 (30–35 mmHg) may be used so as to prevent secondary brain injury.

During secondary survey:

- Excluding injuries that will result in hypoxia or hypotension are pivotal, viz. pneumothorax, haemothorax, tension pneumothorax, shock and abdominal compartment syndrome.

During tertiary survey:

- Imaging: Skull radiographs may be used as an adjunct in a setting where CT scan is not available; however computed tomography of the brain (preferably with C-spine inclusion) is currently the norm in assessing traumatic brain injury.

Indications for an immediate CT of the head in an adult include:

- GCS <13 initially
- GCS <15 at 2 h after injury on assessment in the emergency department
- Suspected depressed or open skull fracture
- Signs of base of skull fracture (periorbital, ecchymosis, post-auricular ecchymosis, subconjunctival haemorrhage, CSF rhinorrhoea or otorrhoea)
- Focal neurological deficit including fixed unresponsive pupil
- More than one episode of vomiting

In children:

- Loss of consciousness or amnesia for more than 5 min
- Amnesia (anterograde/retrograde) for more than 5 min
- Three or more episodes of vomiting
- Post-traumatic seizure (without history of epilepsy)
- Clinical suspicion of nonaccidental injury
- GCS <14, suspicion of skull fracture or tense anterior fontanelle

Diffuse injuries: [8] (Table 38.2)
Focal injuries: [3] (Tables 38.3 and 38.4, Figs. 38.1, 38.2 and 38.3)

Prognosis [9]

Multiple scales/scores have been used to predict outcomes following traumatic brain injury (Table 38.5).

Table 38.2 Marshall grading – CT abnormalities in brain trauma

I. No visible intracranial pathology on CT scan
II. Cisterns patent with midline shift of 0–5 mm and/or lesions densities present; no high- or mixed-density lesion >25 cm [3] may include bone fragments and foreign bodies
III. Cisterns compressed or absent with midline shift of 0–5 mm; no high- or mixed-density lesion >25 cm [3]
IV. Midline shift >5 mm; no high- or mixed-density lesion >25 cm [3]
V. Any lesion surgically evacuated[a]
VI. High- or mixed-density lesion >25 cm [3]; not surgically evacuated

[a]See later regarding criteria surgical management

Table 38.3 Comparison of focal brain haemorrhages

Extradural	Subdural	Intracerebral	Subarachnoid
Associated fracture Blood outside dura Arterial bleed (middle meningeal) Venous bleed (temporal or venous sinus) Temporal region common "Walk-talk-drop" in 30 % Findings on CT scan involve biconvex hyperdense lesion	Acceleration-deceleration force Clot under dural over brain Usually parenchymal brain injury Torn bridging veins More common than EDH Acute (3–21 days) and chronic (>21 days) based on density on CT	Direct focal injury Injury of parenchymal vessels Underlying depressed skull fractures and along the tract of penetrating injuries Commonly temporal and frontal basal regions in blunt injury	Intraventricular and subarachnoid similar Result in obstructive hydrocephalus and raised intracranial pressure Often seen with other injuries as part of a blunt trauma spectrum or with penetrating vessel injury

Table 38.4 Management of specific injuries [8]

Surgical intervention	Medical intervention	Other interventions
Large EDH (>15 mm/5 mm shift) Most acute SDH, with or without bone flap and/or craniectomy if: Clot thickness >10 mm Midline shift >5 mm Deteriorating GCS Pupillary abnormalities ICP >20 mmHg ICP monitor placement for all GCS <9 Parenchymal lesions are at the discretion of the treating neurosurgeon, usually >50 cm [2] volume or >5 mm shift or if ICP >20 mmHg Open or significantly depressed skull fracture	Small EDH – repeated CT and neurological observation Small SDH Small parenchymal lesions provided ICP monitoring or neurological observation and repeat CT at 24 h are possible All open skull fractures need therapeutic antibiotics	Early feeding and use of topical mucosal protective agents prevent stress ulcers Regular turning and pressure ulcer protection are essential Steroids are not recommended as this increases mortality

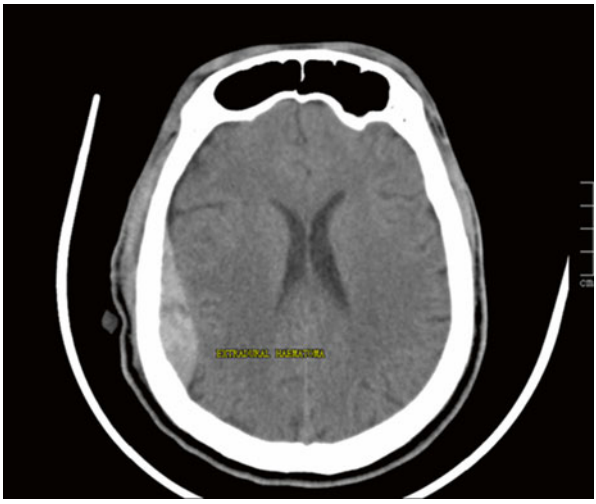

Fig. 38.1 Shows an acute extradural haematoma

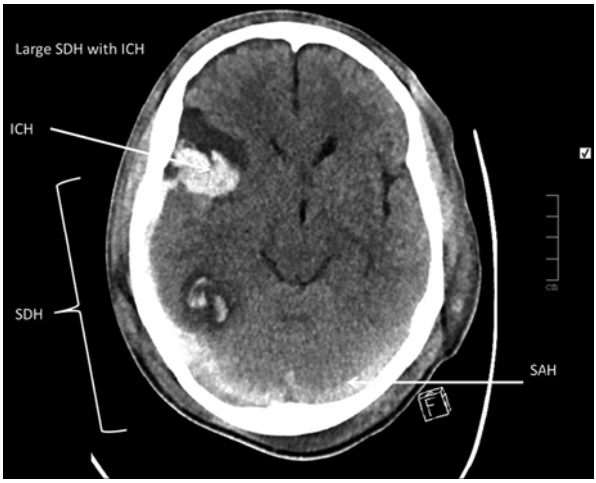

Fig. 38.2 Shows subdural, intracerebral and subarachnoidal

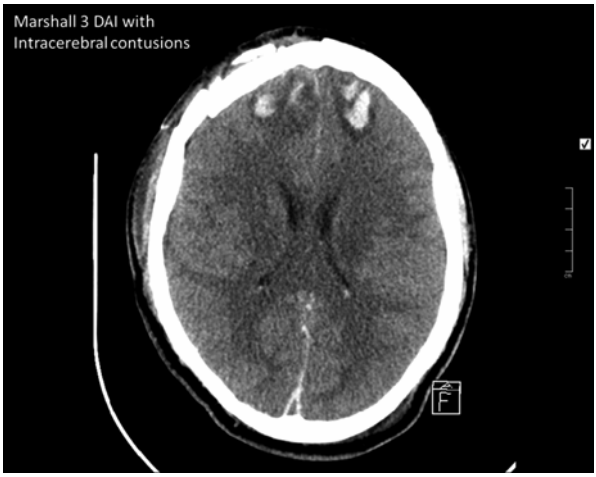

Fig. 38.3 Shows intracerebral contusions and Marshall grade 3 DAI

Table 38.5 Glasgow Outcome Scale (GOS)

Level	Term	Definition
1	Dead	No life
2	Vegetative state	Unaware of self and environment
3	Severe disability	Unable to live independently
4	Moderate disability	Able to live independently
5	Mild disability	Able to return to work/school

GOS of 4 or 5 is considered a favourable outcome.
Predictors of outcome:

- Age: Severe TBI older patients have worse outcomes than young adults, and young children still have neuronal plasticity.
- Glasgow Coma Scale (GCS): Lower total score is associated with poor outcome; the motor score component has the highest prognostic rank in predicting outcome. GCS is best established ("resuscitated GCS") after hypotension and hypoxia have been corrected. Intoxication can also hamper outcome prediction.
- Pupillary response and eye movement: Unilateral dilated pupil suggests a mass lesion and abnormally dilated and non-reactive pupils are strongly associated with poor neurological outcome. The absences of oculocephalic and oculovestibular responses are poor prognostic signs.
- Type of brain lesion on imaging studies: Marshall classification has prognostic significance with progressive mortality increasing from I to IV and good to moderate outcomes in 62 % of Grade I and 6 % in Grade IV.

Acknowledgement Images are the property of Dr T C Hardcastle and the IALCH Trauma Service – copyright held by IALCH.

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Chapter 39
Paediatric Trauma: Resuscitation and Early Management

Daan Den Hollander

Introduction

Trauma is the second most important cause of death amongst children between 5 and 14 years. A quarter of all trauma deaths occur in children with 950,000 deaths worldwide in 2004. In low- and middle-income countries (LMICs), the group most commonly affected by trauma is vulnerable road users, including pedestrians, and – in Asia – children carried on motorized two-wheelers [1].

Differences Between Children and Adults

Children are not small adults – this is as true in trauma as elsewhere; children differ in:

- Structure: they differ in their airways, abdominal organs are more exposed and bones are more resilient
- Physiology: they are more prone to heat loss and hypoglycaemia; they have better cardiovascular compensatory responses. When their blood pressure drops, they are usually close to arrest. Their overall reserves are usually less than those of adults, though.
- Psychologically: children do not appreciate and thus do not avoid danger and may not remove themselves from harmful situations.

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Pathophysiology of Paediatric Trauma

Deterioration in a paediatric trauma can be described in four stages. These will be described separately for respiratory and for circulatory failure. These stages form the categories into which the deteriorating child is categorized (see Fig. 39.1).

Respiratory Failure

Examples are tension pneumothorax, haemothorax and lung contusion.

- Stable patient: the deterioration is minimal and causes no physiological changes.
- The compensated state, respiratory distress: physiological changes are the result of recruitment of compensatory mechanisms that are increased respiratory rate and use of accessory muscle (increased effort).
- The decompensated state: the compensatory mechanisms fail to maintain oxygenation of the blood, which is reduced oxygen saturations and end-organ failure (brain: irritability, confusion, decreased level of consciousness).
- Respiratory arrest: there is no spontaneous respiratory effort.

Circulatory Shock

Examples are ruptured spleen (haemorrhagic shock), cardiac contusion (cardiogenic shock), tension pneumothorax (obstructive shock) and spinal cord injury (distributive shock).

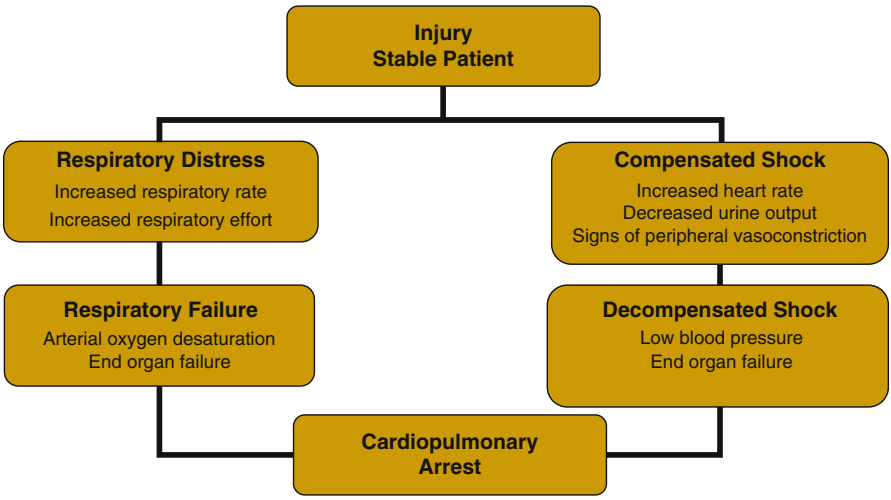

Fig. 39.1 Deterioration stages in paediatric trauma

- Stable patient
- Compensated shock: increased heart rate, decreased urine output and peripheral vasoconstriction (blue, mottled extremities; decreased capillary refill; weak peripheral pulses with strong central pulses)
- Decompensated shock: low BP for age (<70 + age × 2); end-organ failure
- Circulatory arrest

The Trauma Team

Trauma patients greatly benefit by being received by a dedicated trauma team, with well-defined and practiced roles and protocols [2]. Such teams typically consist of:

- A team leader, who evaluates the patient, decides on investigations and treatment and is in overall charge.
- If more doctors or advanced life support paramedics are available, these manage the airway/breathing and the circulation. When managing children, paediatric doctors may be included as they have extensive skills and experience in resuscitating critically ill children.
- Two or three nurses assist the doctors, take and record vital signs and record any interventions done and treatment given, with the times. Each nurse is attached to one of the doctors.

The team is ideally forewarned of the arrival of a trauma patient. During the trauma resuscitation, the team is not involved with any other patient in the emergency department. The roles of the respective team members are defined and the team regularly practices 'trauma codes' so they get used to their roles in the trauma resuscitation and to each other. Team skills, such as 'closed loop communication', are practiced.

Resuscitation

Resuscitation in children follows ATLS and PALS principles. These can be described as three ACTs (where ACT stands for assessment, categorize and treatment):

ACT 1
When approaching the patient, *assess* ABC (appearance, breathing and colour), *categorize* in any of the categories mentioned above and *treat* accordingly (e.g. assess for respiratory arrest, give supplemental oxygen). The vital parameters in a child are tabulated in Table 39.1.

ACT 2
The assessment in this stage is the ATLS Primary Trauma Assessment. It is important to carry this out in a hierarchical manner, treating each abnormality when it is

Table 39.1 Physiological range of vital observations in a child

Age	BP	HR	RR
1 month	80/45	140	40–80
1–2 years	95/55	110	20–40
3–4 years	100/65	100	20–40
5–8 years	110/60	95	15–25
Adult	120/80	70	16

encountered before proceeding to the next stage. The reader is referred to Chap. 36 on adult trauma resuscitation. Here only the salient points of paediatric trauma resuscitation are emphasized.

Airway

- The paediatric airway is relatively shorter and more anterior than the adult one. The narrowest point is at the cricoid cartilage. The cricothyroid membrane lies above the chin fold in children younger than six [3].
- Turbulence in the airway as a result of crying significantly increases airway resistance. Adequate pain control and a reassuring environment are essential.
- Indications for intubation in the paediatric trauma patient are enumerated in (Fig. 39.2). It needs to be emphasized that paediatric intubation is more difficult than in the adult. An experienced intubator, optimal lightning, a rapid sequence technique using suxamethonium (0.6 mg/kg) and etomidate (0.3 mg/kg) and a plan for the failed-intubation scenario are essential elements of safe paediatric airway management. Never have more than two attempts, each of which should last <30 s [4]. If not successful either refer back to bag-mask-valve ventilation, find a more experienced intubator or proceed to a surgical airway.
- Endotracheal tube sizes are given by the formula (age)/4 + 4 for uncuffed tubes and (age)/4 + 3,5 for cuffed tubes. Although traditionally uncuffed tubes have been recommended for children under the age of 6, cuffed tubes are increasingly used in this population with a high risk of aspiration and expected need for ventilation of poorly compliant lungs [5].
- If a surgical airway is necessary, a cricothyroidotomy is contraindicated in the younger child: it would place a tracheal tube above the narrowest part of the airway and is associated with a high incidence of misplacement of the tube and of later tracheal stenosis. A needle tracheostomy followed by a formal tracheostomy as soon as the child is stable is the surgical airway of choice.
- Intubation in head-injured children in the prehospital setting is controversial. Although in adults intubation prevents secondary brain damage, there is no evidence that this is the case in children [6, 7]. Episodes of hypoxia during intubation attempts by less experienced providers and hyperventilation of the intubated patient may outweigh the benefits. Intubation attempts should never last more than 30 s, and after intubation ventilation should not exceed one breath per 6–8 s.

- Inability to ventilate the child using a bag-valve-mask (including maxillofacial trauma)
- Need for prolonged control of the airway (transport)
- GCS ≤ 8
- Respiratory failure as indicated by hypoxemia or hyperventilation
- Respiratory distress, indicated by a respiratory rate < 15 or > 40
- Decompensated shock resistant to initial fluid administration
- Loss of protective airway reflexes

Fig. 39.2 Indications for intubation in the paediatric trauma patient

- Protection of the cervical spine is an essential part of airway management, initially by in-line manual stabilization and subsequently by immobilization on a spinal board using a rigid cervical collar and head blocks. Younger children have a relatively large head and may require a folded 5 cm thick blanket under the torso: aim to align the ears and the shoulders in the same horizontal plain.
- Because of the high risk of aspiration, all traumatized children should also undergo gastric tube insertion.

Breathing

- Because of the close proximity of the trachea to the anterior chest wall, it is easy to mistake tracheal air movement for breath sounds. In young children, always listen in the axillae.
- Tension pneumothorax is a potentially lethal injury that is easily treatable by the primary care physician and should never be missed. It may not be obvious when the patient is first seen and repeating the primary survey regularly: every 30 min (and after every time the patient was moved). The clinical signs and management of a pneumothorax in children are similar to that of an adult. It is, however, recommended to tunnel a thoracostomy tube by making the skin incision one intercostal space lower than where the tube will enter the pleural space to avoid it sucking in air on removal.

Circulation

Because of their excellent vasomotor response, children maintain their blood pressure for a long time. A high pulse rate is therefore the most important early sign of shock and a drop in blood pressure signifies advanced shock. Children will often display signs of intense vasoconstriction (see above). The algorithm for assessment of circulatory status and the appropriate intervention is depicted in Fig. 39.3.

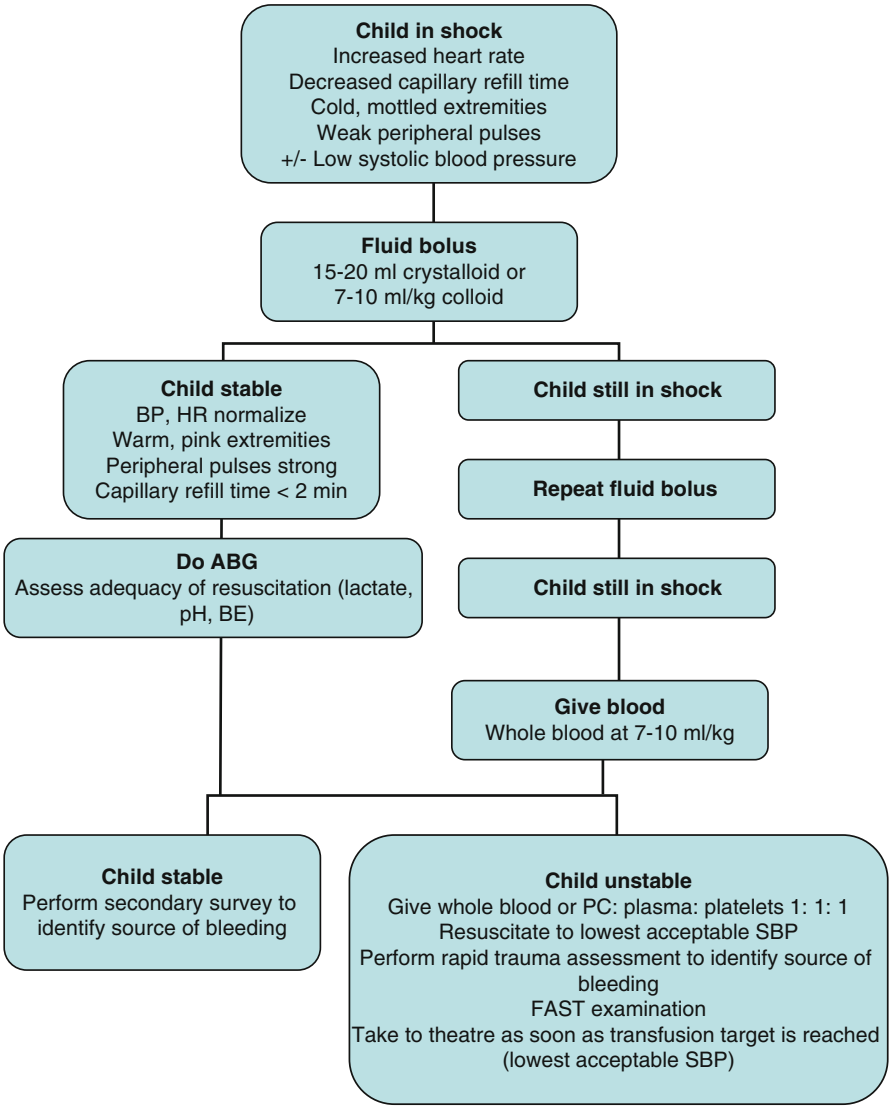

Fig. 39.3 Management flow process for circulatory abnormality in paediatric trauma

- Peripheral access can be difficult but should be attempted first. In a patient with advanced stages of shock, alternative access should be sought early, i.e. after no more than three attempts or not more than 2 min. An intra-osseous line is easiest, but a femoral venous catheter allows more rapid fluid resuscitation.
- Children are resuscitated by fluid boluses of 15–20 ml/kg of a crystalloid solution or 7–10 ml/kg of a colloid solution. These are drawn up in a 50 ml syringe in injected directly in the bloodstream. If the bolus fails to restore the vital signs to normal, they may be repeated.

- Common causes of significant bleeding in children are injuries to the solid organs of the abdomen (the spleen, liver, kidney), scalp injuries and pelvic or chest injuries. A focused abdominal sonography for trauma (FAST) or peritoneal lavage may alert the emergency physician to the presence of blood in the peritoneal cavity.
- Cardiac contusions are rare but may occur after relatively trivial trauma such as being hit by a cricket ball in the chest. The condition is characterized by otherwise unexplained hypotension with high central venous pressures and is managed by fluid optimization and inotropic support. A haemo- or tension pneumothorax is another cause of unexplained hypotension

Disability

Two scales are in use to assess disability in the trauma patient:

- The AVPU score is a simple score (alert, reaction to verbal stimulation, reacting to pain and unresponsive). It is used in a number of emergency department scores
- The Glasgow Come Score should be used in all children with a decreased level of consciousness. The motor component can be used on its own [8]. Clinical deterioration is present when a child drops two points on the overall scale or one point on the motor component. Adjustments must be made for preverbal children (see Table 39.2).

Table 39.2 Paediatric Glasgow coma scale

Eye opening	*1 year*	*<1 year*	Normal aggregate scores
4	Spontaneously	Spontaneously	<6 months 6–12 months: 12 1–2 years: 13 2–5 years: 14 >5 years: 14
3	To verbal command	To shout	
2	To pain	To pain	
1	No response	No response	
Best motor response			
5	Obeys commands		
4	Localizes pain	Localizes pain	
3	Flexion to pain	Flexion to pain	
2	Extension to pain	Extension to pain	
1	No response	No response	
Best verbal response	*5 years*	*2–5 years*	*0–2 years*
5	Orientated and converses	Appr. words and phrases	Coos and/or babbles
4as	Disorientated, converses	Inappropriate words	Cries irritably
3	Inappropriate words	Cries to painful stimuli	Cries to pain
2	Incomprehensible sounds	Moans and grunts to pain	Grunts to pain
1	No response	No response	No response

Exposure and Control of the Environment [9]

- Exposure in a patient with (possible) serious trauma should be by cutting the clothes to avoid undue movements of the spine. The entire patient should be exposed to avoid missed injuries.
- Children cool off quickly and steps should be taken to prevent unnecessary heat loss. The chances of survival drop significantly when the core temperature drops below 35 °C and survival is unlikely below 32 °C. Hypothermia is associated with coagulopathy and acidosis.
- External methods of heat control are setting the room temperature (to at least 24 °C) 9, warm blankets and external heat sources such as Bair Hugger. They are most effective in maintaining core temperature.
- Internal heating by infusing warm fluids and filling the bladder or stomach with warm fluids are more effective in warming a hypothermic patient.

ACT 3: AMPLE History and Secondary Survey

These are essentially similar to adult trauma evaluation. A few paediatric peculiarities will be addressed here.

The Head and Neck

Significant head injuries may occur in children without associated skull fractures. Routine CT skulls in neurologically intact children have, however, not proven to be beneficial and unnecessarily increase radiation-associated long-term risk [10].

A scalp hematoma in a child under the age of 2 is often associated with intracranial bleeding and is an indication to perform a CT scan [11].

The Chest

Apart from pneumothoraces, the only other common significant chest injuries are rib fractures and lung contusions. Because of their pliable bones, rib fractures always signify a significant injury in children. Lung contusion that cannot be seen on a plain CXR is rarely clinically significant [12]. Lung contusions associated with respiratory embarrassment require early intubation and ventilation with positive end-expiratory airway pressures set to maintain oxygen saturations above 94 %.

The Abdomen

Blunt force trauma to the solid organs of the upper abdomen is the most common injury mechanism in children. Injuries to hollow viscera are rare.

An exception is the injury complex associated with an ill-fitting lap belt: ecchymosis of the anterior abdominal wall, transverse fractures of the lumbar spine often with spinal cord transection and hollow viscus and vascular injuries of the abdomen.

Extremity Injuries

The immature skeleton is prone to a number of unique injuries during childhood:

- Greenstick fractures of the long bones.
- Torus fractures.
- Growth-plate injuries. These may or may not result in late deformity if not treated properly. Significant growth-plate injuries require anatomical reduction, which usually requires surgery. They should all be evaluated by an orthopaedic surgeon.
- Epiphyseal avulsions occur because in children epiphyseal growth plates are weaker than the ligaments spanning the associated joints. Injuries that in adults would result in a dislocation cause an epiphyseal avulsion in a child. Any child with pain and swelling around a joint should have the area x-rayed, and if an avulsion injury is found, referred to orthopaedic service.

Tubes and Investigations

- All children with significant trauma must have a gastric tube to keep the stomach empty and avoid aspiration of its contents.
- Unless the child has a urethral injury (blood on the meatus, boggy swelling in the perineum, high-riding prostate), urinary catheterization must be performed to allow hourly monitoring of the urine output.
- Blood must be taken for a full blood count and clotting profile, urea and electrolytes, liver function and amylase and – where available – an arterial blood gas analysis. A crossmatch must be sent, but unless the child is haemodynamically unstable, it is usually not necessary to order blood.
- A trauma series of x-rays (anteroposterior chest, pelvis and lateral neck) should be requested, preferably as a bedside procedure. If the child is sent to the radiology department, somebody with skills to recognize and manage deterioration in a child must accompany every seriously injured child, even if it seems stable.

- Children with a decreased level of consciousness in whom the abdomen cannot be assessed and those with a severe mechanism of injury in whom no indication for immediate surgery exists should undergo a CT angiography on admission.
- If a child under the age of 7 undergoes a CT scan for evaluation of traumatic brain injury, the C-spine must be included as C-spine injuries at this age often involve the upper three vertebrae which are difficult to see on plain x-rays.

Child Abuse

Practitioners looking after acutely injured children should always be on the lookout for child maltreatment. Tell-tale signs are [13]:

- An inappropriate delay in seeking medical help for serious injuries.
- Discrepancies in the medical history, especially early on in the admission.
- Single mother, social isolation and substance abuse. Young boy with a disability or mental disorder such as ADHD.
- Certain injuries have a high risk of being nonaccidental:
- Subdural hematomas in infants and toddlers
- Rib fractures in children 0–3 years old, especially if multiple and in the absence of a history of major trauma [14].
- Sudden death in a child with rib fractures (paediatric basic life support rarely causes rib fractures)
- Hollow viscus perforations in the absence of traffic injury
- Spiral fractures of the long bones
- Any of the following burns: stock and glove pattern of the extremities, donut burn of the buttocks and contact burns including cigarette burns

Practitioners suspecting child abuse have an obligation to admit the child (place of safety) and inform the relevant authorities. Documentation is essential, as is avoiding leading questions.

Documentation and Disposal

Documentation is essential, particularly if the child will be transferred to another health-care provider, whether in the same hospital or ‘elsewhere’. Mechanism and time of injury, findings on primary and secondary survey and details of observations and management are essential. When the patient is referred to a trauma centre higher up in the chain (the common indications are listed in Table 39.3), the following procedure is recommended:

- The intention to refer and the reasons therefore must be discussed with the child’s family. Consent for the transport (verbal) and for investigations (CT scan) and procedures (surgery, blood transfusion – written) must be obtained.

Table 39.3 Indications for referral of an injured child to a specialist paediatric trauma unit

All seriously injured children below the age of 5
All children with a life-threatening injury, i.e. who are haemodynamically unstable or were so on arrival in the emergency department
Children with injuries to two or more body compartments (e.g. head and chest or chest and abdomen)
Children involved in accidents with one or more of the following characteristics
Pedestrians thrown out of their shoes or run over
Falls from more than 8 m
MVAs with ejection from a vehicle, death in the same passenger compartment, prolonged extrication time, rollover of the vehicle, major vehicle deformity and intrusion in the passenger compartment
All children with suspected intra-abdominal pathology due to either blunt or penetrating trauma, whether they qualify for surgery or not
Children needing subspecialty care (e.g. neurosurgery)

- The patient must be discussed with the receiving trauma team. The following information is usually required: mechanism and time of the injury, injuries found and/or suspected, vital signs and urine output on admission and at the time of the referral and investigations and management until the referral. Any advice given by the trauma team must be followed.
- If the patient is accepted, an appropriate ambulance must be organized. While waiting for the ambulance to arrive, perform regular 'clinical reviews' and react accordingly. Do not hesitate to rediscuss the patient with the trauma team when the situation changes.
- Send all relevant information with the patient, including (copies of) observation and treatment charts, laboratory results and any x-ray investigations.
- Anticipate any problems during the transport (in many LMICs transport times may be several hours): make sure that drips, drains and tubes are secure when the ambulance leaves. If the child will be transported by air, consider whether he or she needs a chest drain for a subclinical pneumothorax.
- A courtesy call to the receiving team on departure of the child with the estimated time of arrival is usually much appreciated (Table 39.3).

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Chapter 40
Tertiary Survey and Avoiding Missed Injury

Timothy Craig Hardcastle

Key Points
- In severely injured patients, there is a distinct risk of missed or delayed diagnosis of injury.
- Missed injury is most often due to incomplete history or examination or the non-performance of essential imaging and laboratory testing.
- Systematic errors and multifactorial error avoidance techniques decrease the risk of missed injury.
- Tertiary Survey is a one method shown to work in the acute trauma patient.

Introduction

Trauma is prevalent in the society, yet despite the regularity of injury, the problem of missed injury and delayed diagnosis persists. Missed injury is poorly defined and various publications use varied definitions; however the most common is an injury detected either after primary and secondary survey is complete or after 24 h, whichever comes first [1, 2]. In most of the published series, the incidence varies between 2 % and 4 %, but there are reports of over 30 % missed injury, depending on how the classification is used [1]. Most missed injuries have minor overall consequences in terms of survival of the patient; however there are certain injuries that may be

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devastating if missed due to functional outcome impairment or even death, although these appear to generally be a minority. In a recent meta-analysis of 17 studies [3], it is stated that there was an equal mix of minor and major missed injuries, with a very low (1–4 %) incidence of life-threatening complications. Minor missed injury may delay return to previous state of social interaction, and eventual daily activity may be compromised, suggesting there is a need for more robust systems of review to avoid such incidents [1–3].

Underlying Causes of Missed Injury

Missed injuries occur with a remarkable degree of regularity in recognisable patterns, with the same or similar patterns of injuries being missed repeatedly in most reported series from around the globe. Most commonly there is a human factor involved in the process of the error.

The underlying factors increasing the risk of missing an injury in the trauma patient include:

- Life-threatening injury that changes priority of care
- Altered mental state or chemical neurological impairment
- Distracting major injury that distracts the focus of the treating team
- Need for emergency surgery precluding the completion of the primary and secondary survey ('damage control')
- Inexperience and stress of the medical and nursing team
- Inappropriate, or inadequate, access to or performance of imaging or laboratory examinations

These include patient factors, technological factors, the health worker's physical and emotional state, ambient climatic and working conditions, the physical structure of the facility, social, legal and cultural influences and finally the quality of the hospital organisation [1].

Clarke and co-workers [4] used Reasons' Swiss cheese model [5] to identify the causes of missed injury and determine a classification of the types and causes of these injuries (Table 40.1 is a simplified version of the classification with one added category).

Clinical Features: What Injuries Are Commonly Missed and Why? [6]

- Cervical spine: common and often minor
- Other spinal injury – due to focus on C-spine and inadequate imaging
- Chest: progression of blunt chest trauma with lung contusion – underestimated severity, aortic injury missed on supine CXR
- Abdomen: missed (particularly blunt) bowel injury or retroperitoneal injury

Table 40.1 Classification of errors leading to missed injury

Type	Content
Latent failure	Management, staffing or resource issues
Active failure	Direct failure in patient care
Subtype of failure	
Cognitive lockout	Ignoring obvious clinical picture
Slip	Inattention to detail (e.g. wrong side drain)
Lapse	Simply forgetting to do/examine an issue
Rule-based error	Wrong plan based on repeated usual action (e.g. sending unstable patient to CT before resuscitation)
Knowledge-based error	The pathology is outside the clinical experience of the clinician ('seniority'/novel situation)
Method of failure	
Act of omission	Failure to perform a needed action
Act of commission	Unnecessary or wrong action
Violation	Deviation from standard of care
Error of execution	The correct action, yet incorrect result (e.g. false-negative test)
Error of judgement	Wrong decision due to lack of consultation (linked to lapses)

Adapted from Clark et al. [4]

- Ureteric injury often missed with penetrating trauma to the back or flanks
- Rectal injury – extra-peritoneal if there is no blood on rectal exam
- Small bones of the hands and feet, to ligaments (particularly knee dislocation)
- Facial bones (especially if undisplaced)
- Blunt vascular injury (intimal flap with late occlusion), peripheral and carotid
  - Common with seatbelt sign and knee dislocation or fractures
- Myoglobinopathy and acute kidney injury (especially the aerobic type) [7]
- Foreign bodies or the effects of underlying non-traumatic pathology
- Mis-triage in the very young and the elderly due to false security or underappreciation of the physiological differences
- Repeated handovers of care between providers with inadequate documentation and lack of a coordinating clinician ('ship captain' – ideally the 'trauma surgeon')

Investigations: Relevant Investigations and Their Evidence-Based Rationale

It is important to link certain commonly missed injury patterns and devise methods of error avoidance. An important aspect of this relates particularly to the use of imaging modalities in an appropriate manner, led by the clinical picture of the patient. In particular, blunt trauma has higher missed injury rates and as such demands more liberal use of advanced imaging compared with penetrating trauma. Table 40.2 highlights some of the common injury patterns and how to best assess these.

Table 40.2 Common missed injury patterns and how to identify these

Injury	Common associated injury	Avoidance imaging
Head – traumatic brain injury	Cervical spine injury	If CT – brain, include neck
Facial bone fractures	Blunt carotid/vertebral injury	CT face and CT angiogram
Flail chest	Blunt cardiac or aortic injury, missed lung contusion on CXR	CT angio and lung windows troponin I – level [8]
First/second rib/ clavicle fracture	Subclavian intimal injury	CT angiogram
Blunt chest trauma	Thoracic spinal injury	CT chest including spine

Tricks to Avoid Missing Injury

The use of systematic evaluation techniques and constant re-evaluation are the most important methods to reduce missing injury. The use of protocols and checklists also assists in this process. Croskerry advises that reducing dependence on memory by written protocols, regular review of the patient in context (called 'metacognition') and ensuring the optimal clinical environment can decrease error [9]. The use of clinical decision nodes and feedback loops further enhances this process.

One example pertinent to the trauma patient is known as the Tertiary Trauma Survey, first described by Enderson [10], which aims to reduce the type I missed injury (missed in primary and secondary survey). The Tertiary Survey method has been reviewed and shown to only have a 1.5 % 'missed injury' rate after this has been performed (type II) compared to a rate almost twice that prior to instituting the Tertiary Survey concept [2]. On average this meta-analysis described a need for surgical intervention in only 15 % of all the missed injuries identified, indicating that most are minor injuries.

A third, underreported, missed injury group, the type III (detected after hospital discharge) [2] requires further study, but may be an additional area for improvement and could possibly be further reduced through the performance of an additional 'pre-discharge' tertiary survey.

Prevention [1]

The Tertiary Survey: The way to reduce missed injury – contemporary and evidence based.

1. *Why do the Tertiary Survey*?
   - Many delayed diagnoses are related to laboratory results or imaging only available after secondary survey is complete.
   - Some of these (up to 15 % [2]) will require intervention.
2. *What does the Tertiary Survey consist of*?
   - Complete clinical review (primary and secondary survey)
   - Complete radiological and laboratory review

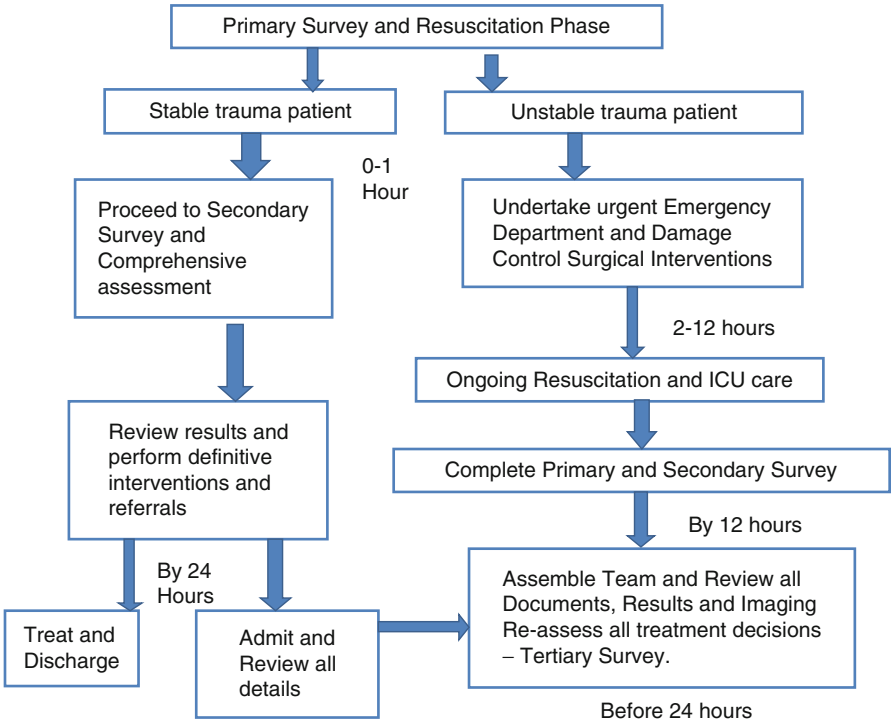

Fig. 40.1 Algorithm for the Tertiary Survey

- Review of the phases of care: resuscitation and surgical intervention to date
- Formulation of further care plan

3. *Who should undergo Tertiary Survey*?

- All trauma patients requiring admission.
- All trauma patients undergoing damage control or intensive care.
- Outpatient rates of missed injury are low (<1 %) and may be excluded.

4. *When should Tertiary Survey take place*?

- On admission to the ICU
- After 24 h post-resuscitation
- On readmission after discharge if the patient presents with ongoing complaints

5. *What sequence works best for Tertiary Survey*?

- Team-based review.
- Fresh eyes present (e.g. previously uninvolved senior clinician).
- Present the initial management, imaging and laboratory results and review decisions taken at each stage. Present surgical procedures. Critique and advice based on entire picture, the further plan, either to exclude additional injuries or to intervene on new injuries noted. Actively look for subtle missed injury that may require intervention (Fig. 40.1).

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Chapter 41
Trauma in Pregnancy

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Key Points

- All women between the ages of 10 and 50 are considered to be of childbearing age and should have a pregnancy detection test as part of the trauma resuscitation.
- Good understanding of the alterations in the physiology and anatomy of a pregnant woman is critical during trauma resuscitation.
- Initial assessment and resuscitation in pregnant patients is similar to resuscitation in non-pregnant patients.
- The foetus is physiologically dependant on the mother and is likely to have a more favourable outcome, if resuscitation is focussed on restoring normal physiological status in the mother.
- Managing trauma in pregnancy should involve a multidisciplinary team approach including the emergency physician, trauma surgeon, obstetrician and anaesthetist.

Introduction

Managing trauma in pregnancy requires sound understanding of the physiological changes in the maternal physiology and anatomy. Initial management should be aimed at optimal resuscitation of the pregnant mother as the foetus is fully

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dependant on adequate uterine blood flow for survival. Further management should include a multidisciplinary approach to monitor and manage both the pregnant woman and the foetus. Injuries should be managed in a similar way as any other trauma patients, but special attention should be paid to uterine involvement. Preterm labour, abruptio placentae, foeto-maternal haemorrhage and uterine rupture are common pregnancy-related injuries that could lead to both maternal and foetal mortality. Optimal and aggressive resuscitation with proper foetal monitoring in the emergency department is paramount to ensure good maternal and foetal outcome.

Trauma in pregnancy can be divided into blunt, penetrating, burns, electrocution and all other minor injuries.

Epidemiology

Trauma is the leading non-obstetric cause of maternal morbidity and mortality in the world [1]. It complicates approximately 7 % of pregnancies worldwide [2]. In the United States of America, trauma was the cause of 46 % of all maternal deaths [3]. The leading causes of trauma in pregnancy are motor vehicle accidents (MVA), falls, assault, gunshot wounds and burns [4].

Young patients, drug abuse, alcohol abuse and domestic violence have been identified as risk factors for obstetric trauma [2].

In pregnant trauma patients, the risk for foetal mortality is higher than maternal mortality, and the risk factors have been identified as injury severity score (ISS) >12, severe blood loss and maternal shock, abruptio placentae and disseminated intravascular coagulation (DIC) [5]. Life-threatening injuries with a high injury severity score carry a 40–50 % risk of foetal death and make this the most significant risk factor for foetal mortality [6].

Physiological and Anatomical Changes in Pregnant Woman (Table 41.1)

Uterus

The uterus is an intra-pelvic organ that gradually increases in size during pregnancy. Up to a gestation of 12 weeks, it remains intra-pelvic and then extends into the abdomen until it reaches the umbilicus around 25 weeks and the epigastrium at 40 weeks. The intra-abdominal organs are consequently displaced superior and posterior making them less likely to be injured. In the first trimester the uterus has a thick wall which decreases in diameter as gestation progresses. The foetus is relatively well protected by the pelvis in the first and second trimester due to more amniotic fluid and mobility as well as the bony protection of the pelvis. In the last trimester the uterus fills the whole abdominal cavity, and the wall is thin and lies

Table 41.1 Most important anatomical and physiological alterations during pregnancy and effects during trauma

System	Changes	Implication	Action
Respiratory	PaCO2 35–40 mmHg	Poor compensation for acidosis	Aggressive fluid management
			Early haemorrhage control
	Mucosal oedema, increased body fat, enlarged breasts	Difficult endotracheal intubation and ventilation	Use smaller-size ET tube
Cardiovascular	30 % increase in cardiac output	More fluid loss before symptomatic	Early and aggressive resuscitation
	Pulse rate increased 15–20 BPM	Difficult to recognise shock	High index of suspicion
Haematological	Increase in plasma volume of 1,500 ml	More fluid loss before symptomatic	
Urinary	Increase in GFR	Reduced urea and creatinine	Early fluid resuscitation to prevent acute renal injury
Uterus	Large and thin walled, obtains most of the abdomen	Protects organs, but more vulnerable to injury and foetal injury	Good foetal monitoring, early ultrasound

directly posterior to the abdominal wall, providing little protection from blunt abdominal trauma. In a supine position the gravid uterus could compress the inferior vena cava and aorta leading to mechanical obstruction, poor venous return and reduced cardiac output.

Respiratory System

Pregnancy causes general weight gain, increased breast size and oedema of the pharynx and larynx. These changes could complicate airway management and make rapid sequence intubation difficult [7, 8]. Increased intra-abdominal volume affects the movement of the diaphragm and causes a reduction in the functional residual volume (FRV). Increased levels of progesterone in pregnancy lead to an increase in inspiratory capacity. This increases the tidal volume and minute ventilation. Respiratory rate also increases due to higher oxygen demand. The increase in minute ventilation causes hypocapnia at rest and reduces the efficacy of respiratory compensation for metabolic acidosis. Pregnant women are thus more susceptible to acidosis and can already be acidotic despite a normal PaCO2 [9]. Total vital capacity remains relatively similar.

Blood Volume and Composition

Plasma volume is increased and reaches a maximum at 34 weeks gestation. The haematocrit is reduced and less than 35 % could be normal. Pregnant women can thus loose more than a litre of fluid before showing any signs of shock. Blood is also hypercoagulable which can lead to an increased risk of thromboembolism after trauma. White cell count is also raised.

Cardiovascular System

Cardiac output is increased up to 1,500 ml during the third trimester. This can be explained by the increase in plasma volume and general vasodilation during pregnancy. Stroke volume increases by about 20 %. Maternal heart rate increases by 15–20 beats per minute [10]. Systolic blood pressure remains stable whilst there could be a slight drop in diastolic pressure. About 20 % of the maternal cardiac output is allocated to the gravid uterus.

Urinary System

Glomerular filtration rate increases by 40–50 % and leads to reduction in plasma urea and creatinine to almost half the normal value [9]. The kidneys are very sensitive to reduction in cardiac output and are not a priority in the physiology of pregnancy which could cause renal tubular necrosis if the patient is not resuscitated aggressively.

Gastrointestinal System

The gravid uterus causes displacement of the intra-abdominal organs which leads to high risk of regurgitation and aspiration. Gastric emptying is also reduced, increasing the risk of vomiting after trauma.

Musculoskeletal System

The symphysis pubis is widened and the sacroiliac joint spaces are increased on x-ray.

Management

Primary Survey

The primary survey for pregnant women should be performed in the same sequence as in other patients in trauma resuscitation. Attention should be paid to the physiological alterations of pregnancy (Table 41.1). During this stage resuscitation and monitoring of the mother should be the priority, and foetal assessment should be postponed until after the primary survey.

Airway

It should be anticipated that the patient will have a difficult intubation due to oedema of the respiratory mucosa and extra fatty tissue around the neck. Have a smaller than usual ET tube ready as well as other special airway equipment such as a gum elastic bougie, introducer and laryngeal mask airway (see Chap. 107).

Breathing

Manage all pathology as soon as possible. Apply monitors: blood pressure, pulse oximetry, ECG and capnography.

Circulation

If the patient is in her third trimester, she should be asked to lie in the left decubitus position by inserting a wedge under the right flank or manually displacing the gravid uterus to the left to prevent caval obstruction. Remember that due to the increase in plasma volume and cardiac output in pregnant woman, the signs and symptoms of shock will be delayed, and foetal distress could be present despite normal vitals. The goal in fluid resuscitation should therefore be to maintain the physiological hypervolaemia so that the gravid uterus is adequately perfused. Inadequate resuscitation can lead to Sheehan syndrome.

The abdominal examination is important as it contains the gravid uterus and needs to be assessed for abruptio placentae, uterine rupture and premature labour. Special attention should be paid to vaginal bleeding, presence of amniotic fluid and contractions of the uterus. At this stage a focussed assessment with sonography in trauma (FAST) scan can be considered to detect free fluid in the chest or abdomen.

Disability

Assess the level of consciousness. If the GCS is less than 8 out of 15, or she only responds to pain, RSI should be performed to protect the airway [3]. Consider pregnancy-related causes of decreased consciousness such as pre-eclampsia or eclampsia if signs are present that do not fit the normal picture.

Adjuncts to Primary Survey

- Arterial blood gas analysis. Remember that PaCO2 is low and bicarbonate is reduced in normal pregnant woman.
- Urinary catheter.
- Nasogastric tube to drain gastric content and relief traumatic gastric paresis. This will reduce the risk of aspiration and improve respiration.
- Do a chest and pelvic x-ray. If chest and pelvic injury is expected, x-rays should be done despite the radiation risk to the foetus. Try to shield the foetus when possible.
- Do a FAST scan.
- Obtain urgent obstetric consultation and start foetal monitoring. Foetal heart tones should be assessed and can be heard after 20 weeks gestation [11]. Doppler ultrasound can be used from 10 weeks and continuous monitoring by cardiotocography is recommended from 24 weeks gestation. Cardiotocography for a minimum of 6 h is additionally recommended [9].

Secondary Survey

In addition to the standard secondary survey, factors related to the pregnancy should be included and prioritised in order to improve foetal outcome. The patient should also be examined in the left decubitus position with manual displacement of the uterus to the left (Fig. 41.1).

Clinical Examination

Special attention should be paid to the abdominal examination. Expose and palpate the abdomen looking for signs of uterine injury and premature labour (Table 41.2). Uterine size and foetal position should be assessed and clinical gestational age determined. A sterile speculum examination should be performed to detect vaginal bleeding and presence of membranes or amniotic fluid. Placentae previa or any foetal parts may also be observed.

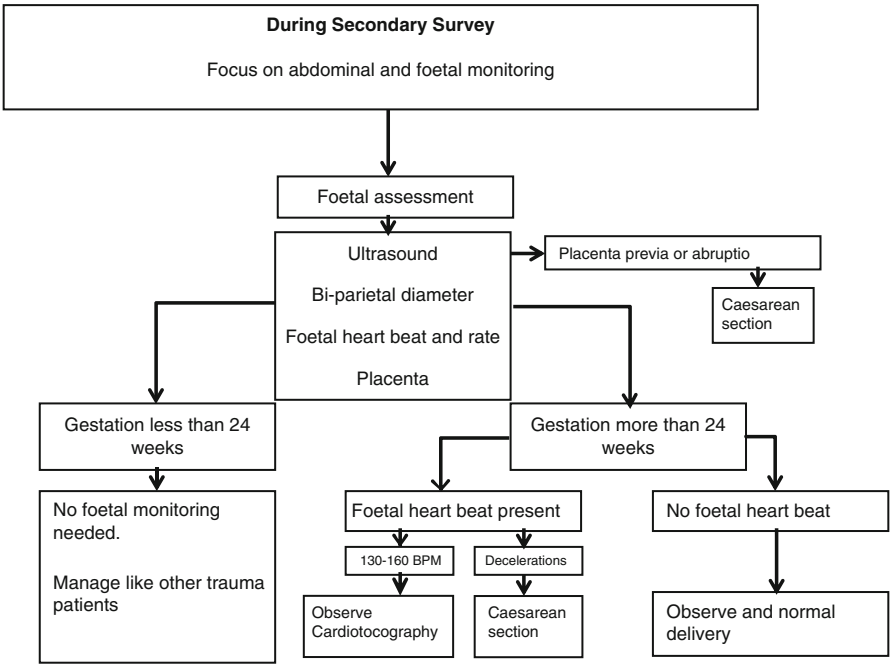

Fig. 41.1 Algorithm for managing the pregnant patient in trauma

Table 41.2 Specific pregnancy-related injuries and clinical signs [11]

Injury	Pathology	Signs
All trauma including minor	Preterm labour and delivery	PV haemorrhage
		Uterine contractions
		PV amniotic fluid
		Dilated cervix
		Abdominal tenderness
Blunt abdominal trauma	Uterine rupture	Abdominal tenderness
Seatbelt		PV haemorrhage
		Abnormal foetal position
		Absent fundus of uterus
		Maternal shock
		Free peritoneal fluid
Blunt abdominal trauma	Abruptio placentae	Severe abdominal tenderness
Deceleration		PV haemorrhage
Blunt abdominal trauma	Foeto-maternal haemorrhage	Suspect in all Rh patients
		No specific signs

Bimanual vaginal examination should also be performed to determine uterine contractions, foetal position and dilatation of the cervix. If vaginal bleeding is noted, manual examination is contraindicated until the cause of bleeding is determined. Perform manual rectal examination to exclude intestinal haemorrhage and assess anal tone.

Ultrasound

Ultrasound in pregnant trauma patients is helpful as it is available at the bedside and does not pose any risk to the foetus. The FAST examination can be used to determine the presence of free fluid in the abdomen. Even though the anatomy is altered in pregnancy, FAST is as accurate in pregnant patients as in non-pregnant patients during all trimesters. A study showed specificity of 100 % and sensitivity of 83 % for detecting free peritoneal fluid in pregnant patients with blunt abdominal trauma [12]. Detection of organ specific pathology is less accurate, needs more experienced ultrasound practitioners and can easily be misinterpreted. If intra-abdominal extra-uterine injury is suspected, CT scan is still the gold standard.

Ultrasound can also be used to assess the foetus. Ultrasound Doppler or M-mode can be used to detect the foetal heart and determine the rate. It is recommended that the standard ultrasound protocol should include the following: foetal heart, foetal heart rate, placental assessment for abruptio, gestational estimation by biparietal diameter and brief amniotic fluid survey [13]. Specificity for detecting abruptio is as high as 100 %. Sensitivity for detecting abruptio is between 24 % and 50 % which means that one cannot rule out abruptio accurately if it was not detected by ultrasound [14].

CT Scan

The American College of Obstetricians and Gynaecologists has recommended that CT scans could be performed on pregnant trauma patients if indicated and that the risk to the foetus is minimal [15]. The CT scan is equally accurate in detecting abdominal injuries in pregnant patients. It is very accurate in detecting abruptio placentae with a sensitivity and specificity of 86 % and 98 %, respectively [16]. Care should be taken to use as little ionising radiation as possible and CT should only be done if there is a clear cut indication.

Rhesus Isoimmunisation

Up to 40 % of pregnant trauma patients with blunt abdominal trauma will have foeto-maternal haemorrhage and are at risk of Rh isoimmunisation. It is recommended that all Rhesus-negative mothers should have a Kleihauer-Betke (KB) test and should receive 300 ug Rh immunoglobulin intramuscularly [17].

Definitive Management

Indications for emergency surgery are the same as for non-pregnant patients. Care should be taken when administering drugs for anaesthesia and analgesia as it may be harmful in pregnancy. Surgery could also increase the risk for preterm labour. The obstetrician should be involved in the definitive care of the patient. Betamethasone will assist in preparing the foetal lungs for delivery [18–20].

Caesarean Section [19]

Indications for caesarean section:

- Foetal distress with viable foetus
- Abruptio placentae
- Uterine rupture
- Penetrating uterine trauma with viable foetus

Perimortem Caesarean Section [18]

Perimortem caesarean section is a surgical delivery of the foetus after the mother had a cardiac arrest and cardiopulmonary resuscitation is underway. This procedure should be performed to save both the foetus and the mother. Delivery of the foetus will immediately improve venous return and increase maternal perfusion. At the same time the foetus can be resuscitated separately. Even though no clinical trials have been performed, the outcome described in case reports is good [19].

Perimortem caesarean section should be performed by a skilled clinician and within 4 min after cardiac arrest and CPR is ongoing. CPR should continue after delivery and further surgical intervention be performed after the patient is stabilised.

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Chapter 42
Trauma to the Urogenital Tract

Emmanuel Owusu Sekyere

Key Points
- Trauma to the urogenital tract should be actively sought for as it presents with non-specific signs and symptoms.
- Management depends on the presence of other life-threatening injuries, and the advanced trauma life support approach is used as in any trauma situation.
- Management is largely conservative for the majority of injuries but some require surgery, and a prompt decision should be made to avoid complications.
- Delayed diagnosis is associated with increased morbidity and mortality.

Introduction

Urogenital injuries occur in 5–10 % of polytrauma patients, of which the kidney is the most commonly injured of the urogenital organs [1] followed by the bladder and urethra [2]. Blunt kidney injury is the third most common solid organ injury following splenic and hepatic injury and often associated with other abdominal organ injuries. Injury to the urogenital tract could be caused by either blunt or penetrating forces. The mechanism of injury, vital signs, physical examination findings and

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other investigations are utilised to derive a diagnosis. This chapter will provide a practical approach with regard to assessment, diagnosis management and complications of urogenital injuries.

Initial Assessment

The initial assessment of patient with suspected urogenital trauma should preferably follow contemporary evidence-based guidelines. This serves to address life-threatening conditions first. Fortunately, the vast majority of urogenital trauma is usually not life-threatening with non-specific signs and symptoms, thus risking a delay in diagnosis. However, the presence of other organ injuries might give a clue to a possible urogenital organ injury, for example, pelvic fracture, with the associated risk of bladder or urethral injury.

Therefore urogenital trauma should be ruled out in a polytrauma patient. It is emphasised that the presence of life-threatening injuries takes precedence over further urogenital injury evaluation and investigation, until the patient is stabilised.

Renal Injuries

By virtue of the anatomical location of the kidneys, significant force is required to cause an injury which tends to also cause associated injuries. The male to female ratio of renal kidney injury is 3:1 and more than 70 % of patients are under the age of 40 years [1]. Conservative management is increasingly the management modality of choice; however, imaging is needed to accurately grade the injury and rule out pedicle involvement or uretero-pelvic junction transection.

Mechanism of Injury

Acceleration-deceleration (fall, high-speed motor vehicle accident) or crush injury will result in contusion, laceration or total rupture of the renal parenchyma. Blunt injuries account for over 90 % of renal injuries (including those in children) and are commonly due to motor vehicle accidents, falls, contact sports and assaults [1].

Gunshot and stab injuries are the main cause of penetrating injuries which represent about 10 % of renal injuries. Iatrogenic injuries such as renal biopsies and percutaneous nephrostomies represent other causes of penetrating injuries [2]. History of any pre-existing organ dysfunction and anatomic abnormality such as solitary kidney, hydronephrosis from any cause, renal cyst and tumour is very important, as even a minor injury can easily become complicated.

Table 42.1 Clinical findings suggestive of renal involvement

Haematuria
Flank pain
Fractured ribs
Flank ecchymoses
Abdominal distension
Abdominal masses

Signs and Symptoms

Physical examination may reveal obvious penetrating trauma, for example, a stab wound to the flank. Clinical findings suggestive of renal involvement are depicted in Table 42.1:

Haematuria is present in 80–94 % of renal trauma cases and the extent of haematuria does not correlate with the degree of renal injury. Uretero-pelvic junction injuries and renal pedicle injury can occur without haematuria in 25–50 % of patients [2–4]. Haematuria is normally the hallmark of urogenital injuries but is neither sensitive nor specific for differentiating minor from major injuries [3, 5]. Unstable patients either have an episode of hypotension (systolic blood pressure <90 mmHg) or evidence of ongoing haemorrhage and lactic acidosis.

Investigation

- Full blood count.
- Urea and creatinine.
- Urine dipstick (rapid and reliable way of assessing haematuria).
- Urine myoglobulin to rule out crush syndrome, which has an impact on the kidney.
- Pregnancy test in female reproductive age patients.
- β2 microglobulin appears to be a marker of renal injury of high value and credibility, contributing to early diagnosis and reliable monitoring of patients [6].

Imaging

The indications for imaging are based on the mechanism of injury and clinical findings are enumerated in Table 42.2:

A haemodynamically stable patient with blunt injury and only microscopic haematuria could be exempted from further imaging studies.

Penetrating trauma:

- Imaging is indicated regardless the degree of haematuria.
- Unstable patients with ongoing haemorrhage: definitive haemorrhage control takes precedence.

Table 42.2 Indications for imaging in blunt renal trauma

Gross haematuria
Microscopic haematuria and shock
Presence of major associated injuries
History of rapid deceleration injury with clinical indicators of renal trauma (3–5)

- The urological tract is thoroughly examined intra-operatively, as part of damage control surgery.
- Plain abdominal films may give indication of a urogenital organ injury such as the presence of rib fractures and pelvic fracture, asymmetric soft tissue overlying the flank and asymmetric psoas shadow.
- Standard intravenous pyelography is no longer the imaging of choice for renal trauma [5] but can still be very useful in situation where it is the only available investigation. It is useful in determining the presence or absence of kidneys and defining renal parenchyma and the collecting system. Extravasation of contrast indicates a major renal injury and should prompt immediate attention. For unstable patients undergoing exploratory laparotomy, single-shot IVP on the table may be performed to further evaluate urological injuries although this is not essential today.
- Focused assessment with sonography in trauma (FAST) has become the preferred initial imaging study for unstable patients with blunt abdominal trauma as part of trauma assessment [7]. In a retrospective review of emergency ultrasound conducted in a level 1 trauma centre between January 1995 and January 2001, the sensitivity of ultrasound for all urological injuries was 67 %, and specificity was 99.8 %. For isolated urological injuries, sensitivity and specificity were 55.6 and 99.8 %, respectively [7]. Ultrasound is the most accurate in the detection of higher grades of renal injuries (grade III and above). The disadvantage is user dependency and low sensitivity for lower-grade renal injuries and other associated injuries that may be present and need urgent attention.
- The gold standard for accurately detecting and grading renal injuries is the contrast-enhanced computer tomography (CT) [3, 4, 8], performed in *stable* patients. It has the advantage of grading the extent of injury and also offers visualisation of the entire retroperitoneum, abdomen and pelvis (Fig. 42.1). The absence of contrast enhancement of an injured kidney is the hallmark of renal pedicle injury.
- Magnetic resonance imaging (MRI) is not used often in renal trauma but can be useful in patient with iodine sensitivity and also where CT facilities are not available, while angiography is useful to specifically locate the source of bleeding prior to embolisation treatment or for the evaluation of a delayed bleed.

Treatment of Renal Injuries

From the initial assessment, and arterial blood gas, the patient is categorised as haemodynamically stable or unstable. Unstable patients require surgery. Most renal trauma (AAST grades 1 and 2 and most grade 3) [3] is managed non-surgically.

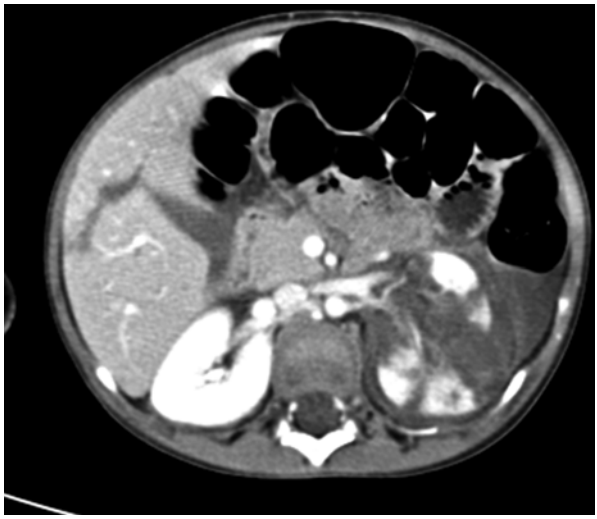

Fig. 42.1 Blunt left renal injury – AAST grade 4, which shows that there is parenchymal and collecting system injury with a surrounding haematoma

However controversy exists with the management of higher grades of injuries with urinary extravasation and devitalised tissue, but an increasing trend towards a conservative approach despite an increased risk of complications is noted.

Nonoperative Management

Indications for a nonoperative approach include stable patients with blunt or penetrating renal injury with grade I-III injury. These patients are managed either by a urology or trauma surgeon as inpatients. The components of conservative management include:

- Initial bed rest till macroscopic haematuria clears followed by minimal mobilisation for 3 weeks.
- Hydration and antibiotics
- Monitoring of haematuria, abdominal signs and clinical vitals and serial haemoglobin level 12 hourly for the first 3–5 days.
- Repeat imaging 2–4 days after trauma minimises the risk of missed complications, especially in grade 3–5 blunt injuries; however the usefulness of frequent CT should be weighed against the risk of increased radiation exposure.
- Repeat CT scans should always be performed on patients with fever, unexplained decreasing haematocrit or significant flank pain.
- Therapy is aborted if patients decompensate in which case patient could be offered angioembolisation treatment or open surgery.

Stable patients with grade III or above injury should have a formal angiography followed by a transcatheter embolisation if active bleeding is identified [2].

Operative Management

The indications for renal exploration are:

- Haemodynamic instability due to renal haemorrhage [2].
- An expanding or pulsatile perirenal haematoma identified at exploratory laparotomy indicates vascular injury or severe parenchymal injury.
- Grade V vascular injury

Paediatric Renal Trauma

Blunt injuries account for more than 90 % of renal injuries. This is due to increased mobility of the kidney, which is proportionally larger in relation to the rest of the body. In addition, the kidneys lie lower in the abdomen, thus less protected by the ribs and abdominal musculature. A clinical approach similar to that of adults is used in evaluating. However, hypotension is an unreliable sign, since blood pressure may be maintained in the presence of significant injury [4]. Renal ultrasound continues to be ideal for initial screening, but there has to be a lower threshold for contrast CT scan.

Ureteric Injuries

The ureters are well protected by the psoas muscle and bony pelvis; hence injuries are rare, accounting for about 1 % of urogenital trauma [5].

- Blunt trauma is mainly PUJ avulsions.
- Penetrating trauma is far more common – any site can be injured.
- Diagnosis is difficult: haematuria may be absent [6], and therefore a high index of suspicion based on the mechanism of injury or penetrating wound tract is required.
- Late presentation, due to missed injuries, is associated with sepsis, urinoma, upper urinary tract obstruction or urinary fistula [9].
- Bilateral injuries are rare.
- Management at the time of ureteric injury is essential in reducing morbidity and mortality [10].
- Flank pain, anuria, peritonitis, ileus, secondary hypertension and urinary leakage are suggestive of a ureteric injury.

Imaging

Extravasation of contrast on delayed films (CT or IVP) is the hallmark of ureteric injury, or on x-ray 30 min after intravenous contrast may confirm the diagnosis of ureteric injury. One shot (high-dose) IVP is often not a diagnostic and as such

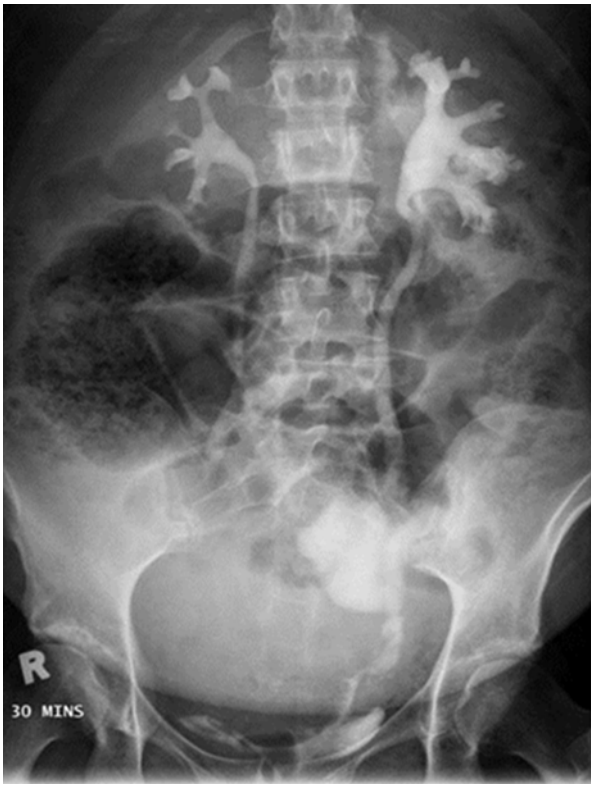

Fig. 42.2 An intravenous urography showing extravasation of contrast left distal ureter

unreliable in detecting ureteric injuries [10]. Retrograde contrast ureterogram can be performed when antegrade studies are inconclusive and the patient is haemodynamically normal (Fig. 42.2).

Management

- Early referral to a trauma surgeon or urologist.
- Partial tears may be managed by insertion of a ureteric stent for a period of about 6 weeks with drainage of the bladder for few days to prevent reflux.
- Complete tears are best managed surgically [10].

Nephrectomy is the last option with failed multiple attempts at repair [4]. In damage control the ureters may be exteriorised by means of ureterostomy or can be ligated and subsequent percutaneous nephrostomy performed. Complications of ureteric injury could be as a result of delayed diagnosis or from surgical management which include ureteral strictures and urinary leakage.

Bladder Injury

- Majority due to blunt trauma.
- Sixty to 90 % of patients with bladder injuries have associated pelvic fractures [11]. There is also occasional perforation by bony fragments.
- Bladder injuries may be classified as intraperitoneal and extraperitoneal.
- Full bladder is a risk factor for intraperitoneal rupture at the dome of the bladder, the weakest and most mobile part of the bladder.
- Pelvic fractures are associated with bladder injuries in 3.6 % of cases (almost always extraperitoneal).
- Fifteen per cent of bladder injuries have an associated urethral injury.

Symptoms and Signs

- Gross haematuria is present in 95 % of cases.
- Patient might complain of inability to void.
- Abdominal distention.
- Bruising at the suprapubic region or associated pelvic fracture.
- Blood at the meatus and retention of urine more likely urethral involvement.

Imaging

- Cystogram and CT cystography have comparable sensitivity of 90–95 %.
- CT has an advantage of assessing for other injuries.
- Fill the bladder with at least 350 ml of diluted contrast (Fig. 42.3).
  - Plain pelvic x-ray with complete filling.
  - Post-drainage phase is taken.
- The disadvantage of CT is the fact that it does not guarantee a full bladder, so catheter clamping is recommended.
- Excretory urography has low accuracy in detecting bladder injuries.

Treatment

Most extraperitoneal bladder injuries are managed conservatively with catheter drainage for about 10 days even in the presence of extensive retroperitoneal and scrotal extravasation (Table 42.3).

All intraperitoneal bladder injuries require immediate repair along with all penetrating injuries. Continuous bladder drainage post-operatively is performed for 7–14 days to allow for healing of the repair. Cystogram prior to catheter removal is recommended.

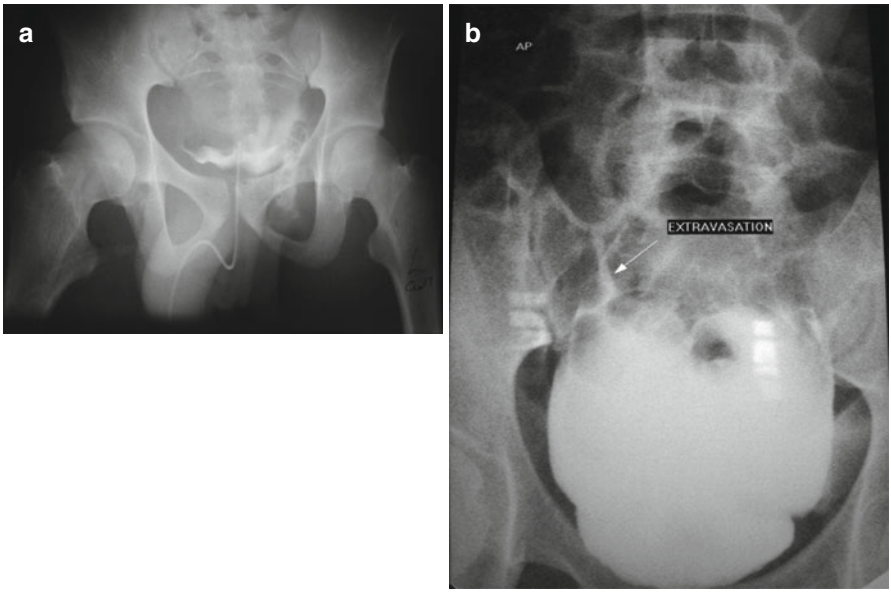

Fig. 42.3 (**a**) Extraperitoneal bladder rupture, and (**b**) Intraperitoneal bladder rupture

Table 42.3 Indications for surgical repair of extraperitoneal bladder injury

1. Bladder neck or ureteric orifice involvement
2. Presence of foreign body such as a bony fragment in the bladder wall
3. Associated organ injuries such as the rectum
4. Entrapment of the bladder wall
5. Surgical treatment for other related injuries such as open reduction and fixation of a pelvic fracture

Urethral Injury

Urethral injuries are more common in men commonly due to a blunt injury

Causes

- Anterior urethra: fall astride/straddle injury.
- Anterior urethra: direct blow to the perineum.
- Posterior urethra: due to pelvic fractures 4–6 % of cases.
- Some penile fractures are also associated with anterior urethral injuries [12].

Symptoms and Signs [5]

- Symptoms include dysuria or inability to void with a distended bladder.
- Blood at meatus or introitus.

- Bruises/haematoma/ecchymosis on the perineum.
- High riding or displaced prostate on rectal examination.
- Urethral instrumentation is avoided till extent of injury is identified by way of imaging.
- Rectal injuries must be actively ruled out.

Imaging

- Retrograde urethrogram is the gold standard in evaluating urethral injuries.
- Combine with an antegrade urethrogram to define the extent of urethral defect.
- CT and magnetic resonance imaging (MRI) may be valuable in assessing the distorted pelvic anatomy.
- Cystoscopy for direct visualisation of the injury without radiation exposure enables differentiation between complete and partial rupture and can be done at the bedside with a flexible cystoscope. It is the investigation of choice in penile fracture where there is a high false-negative rate with urethrogram.
- In females with suspected urethral injuries, a urethroscopy and vaginoscopy are performed.

Treatment

- Acute: urinary diversion via the suprapubic route for a partial and complete rupture.
- If possible to insert a urethral catheter for a partial rupture, this acts as a stent.
- A urologist will be needed to manage concomitant injuries such as penile fracture and also for delayed management of urethral injuries which could be done either via an open approach or endoscopically.
- Penetrating injuries should be explored, debrided and repaired early [13].
- Complications include urine leak, sepsis (necrotising fasciitis) and stricture formation.

Penile and Scrotal Injury

- Penile trauma is rare and can affect the anterior urethra and the corpora cavernosa of the penis [14].
- Penile and scrotal injuries accounted for 1.5 % of urological trauma in a recent report from Scotland of which 20 % resulted from damage to external genitalia [15], but range is 3–11 % of urogenital trauma [12]. The most common cause is motor vehicle collision (68 %) followed by gunshot (17 %) in one Nigerian study [15].
- Other mechanisms of injury include a kick to the perineum and penile fracture.
- Clinical findings are usually obvious and include haematoma, bruising laceration, degloving and avulsion presentation.

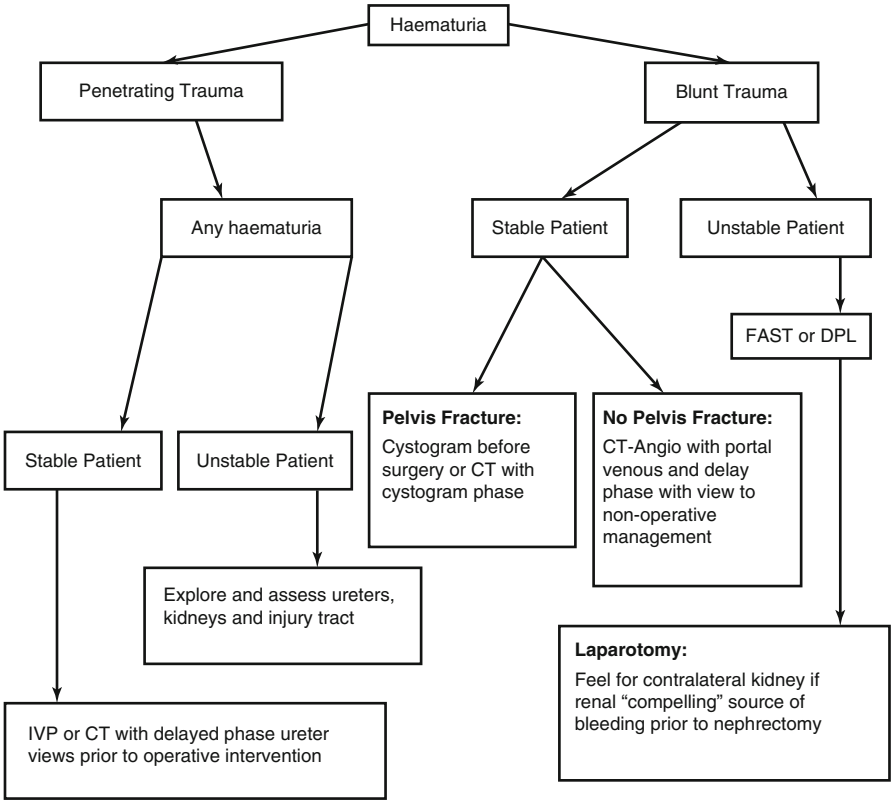

Fig. 42.4 The management of traumatic haematuria

- The primary goal of management is cosmesis and sexual or urinary function (erect position in males).
  - Penetrating injuries: early surgical exploration, debridement and primary repair are recommended [16].
  - Scrotal haematomas should be explored to eliminate testicular torsion.
  - Superficial injuries can be managed conservatively.
  - Complications include urethral strictures, erectile dysfunction and impotence.

Conclusion

A multidisciplinary approach including trauma surgeon, urologist, vascular surgeon and plastic surgeon is required for effective management not forgetting the psychological and psychosocial implications of delayed diagnosis with its associated complications. A generic approach to assessment of the patient with haematuria is found in Fig. 42.4 summarising the approach outlined in this chapter.

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Chapter 43
Thoracic Trauma

David J.J. Muckart

Key Points
- The majority of thoracic injuries involve peripheral lung damage, are diagnosed clinically and with plain chest radiology and can be managed non-operatively.
- CT angiography is the gold standard for assessing blunt thoracic trauma.
- Displaced rib fractures, flail chest and sternal fractures are associated with major intrathoracic injuries.
- Central injuries are associated with a substantial increase in the need for surgery and confer greater morbidity and mortality.
- Emergency room thoracotomy should only be performed for penetrating trauma.

Introduction

Although thoracic trauma accounts for only 20 % of trauma-related deaths [1] when this results in hypoxia or major haemorrhage and is combined with traumatic brain injury, chest trauma is the contributing cause in 75 % of mortalities [2]. The majority of thoracic injuries are peripheral causing damage to lung tissue resulting in

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pneumothoraces or haemothoraces which, at most, require intercostal drainage alone for resolution. Surgical intervention for the lung injury is uncommon, but blunt chest trauma involving the lower thorax may result in damage to solid abdominal viscera in approximately 30 % of patients and penetrating trauma may involve the diaphragm or hollow abdominal viscera. For these reasons it is prudent to consider the torso rather than the chest alone. Central thoracic injuries which involve the heart, major vasculature or airway structures require operative intervention more frequently and carry a high mortality rate.

Clinical Assessment and Imaging

Careful clinical assessment will establish the diagnosis in the vast majority of patients:

- Erect postero-anterior chest x-ray, preferably taken in full inspiration, confirms most pathologies.
- Due to patient discomfort or the presence of extra-thoracic injuries, most images are supine position anteroposterior films.
  - Pneumothorax will be unchanged, but in the supine position a haemothorax appears as a hazy opacity in the affected hemithorax (veiled lung) and a fluid level will not be apparent (Fig. 43.1).
  - The scapulae are within the field and the medial border may be confused with a pneumothorax.
  - Supine magnification gives the central structures a false impression of a widened mediastinum.

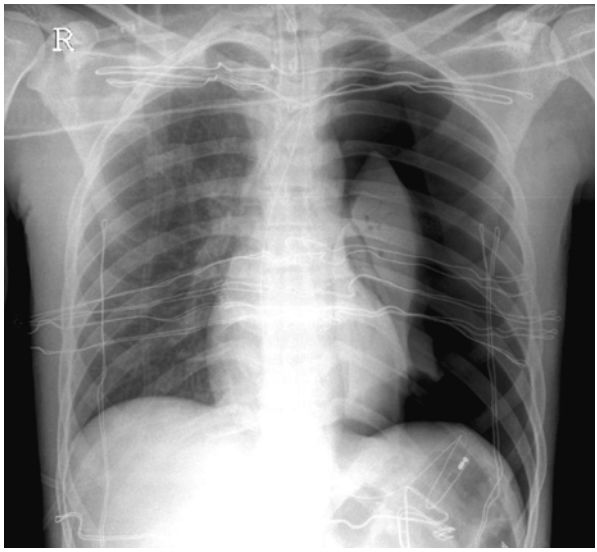


Fig. 43.1 Supine chest x-ray with large simple pneumothorax on the left and veiled lung on the right

These variations must always be considered when interpreting x-rays. For blunt thoracic trauma, CT scanning is the gold diagnostic standard [3], but, in addition to the standard angiogram phase, images must be viewed in the lung window setting to reveal pneumothoraces and contusions. A contrast-enhanced CT is essential to exclude aortic injuries.

Blunt Thoracic Trauma

Injury to the Chest Wall

Rib Fractures

With the exception of children, who may sustain major intrathoracic damage without disruption of the bony thoracic skeleton, rib fractures are invariable sequelae of blunt chest injury in adults. In those without fracture displacement, 50 % of rib fractures will not be evident on chest x-ray. Unless there is a significant underlying lung injury, the major problem is pain which is best managed by rib block and non-steroidal analgesics if there are two or less rib fractures [4]. Rib fractures with displacement signify major force to the bony skeleton and are a warning sign for underlying lung injury (Fig. 43.2).

Specific areas of rib fractures require special attention to exclude associated injuries.

- Fractures of ribs 1 and 2 (especially if combined with displaced clavicular fractures) risk injury to the subclavian vessels. The supposed association with aortic injury has been disproved [5].

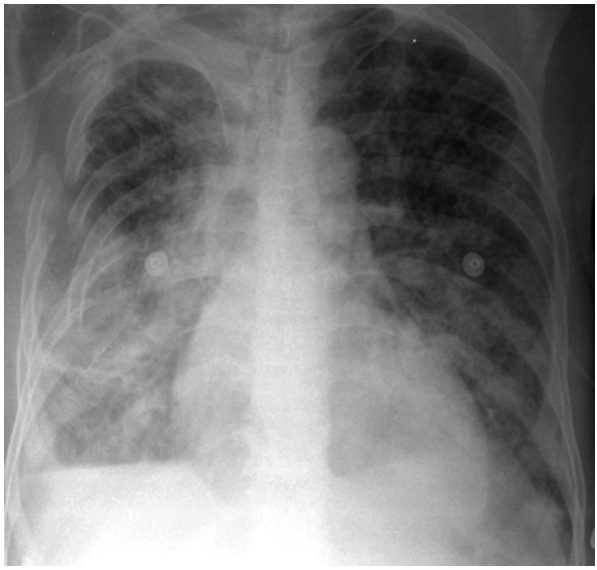


Fig. 43.2 Erect chest x-ray with obvious deformed rib fractures in the right mid-thorax, an underlying pulmonary contusion and fluid level in the right hemithorax

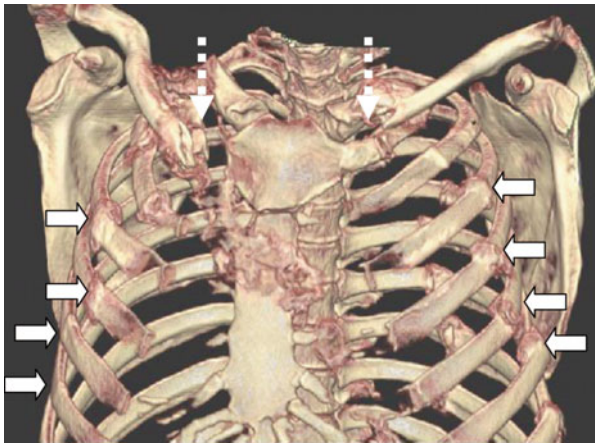


Fig. 43.3 Severe bony thoracic disruption with bilateral anterior rib fractures (*solid arrows*) and bilateral clavicular fractures (*dashed arrows*). A *row* of posterior fractures is visible on the left hemithorax. The patient had combined anterior and lateral flail segments

- Mid-thoracic rib fractures: pulmonary contusions.
- Liver or splenic trauma coexists with lower rib fractures in one-third of patients.

Sternal Fractures

These follow direct injury to the anterior chest, most commonly in unrestrained drivers. Disruption of the costo-chondral junction is common and results in an anterior flail chest. There is a high association with blunt cardiac injury especially with displaced fractures [6].

Flail Chest

By definition this occurs when there are multiple fractures of >2 adjacent ribs ipsilaterally (lateral flail), fractures of >2 ribs bilaterally (flail sternum: anterior flail), or lateral rib fractures with medial costo-chondral disruption (Fig. 43.3). Flail chest is a clinical and not a radiological diagnosis since with costo-chondral disruption the cartilaginous component is radiolucent.

It is extremely important to establish the diagnosis as there is a strong association with major intrathoracic injuries [1]. The affected segment moves in a paradoxical fashion and is usually associated with severe pain, alteration of chest wall mechanics and major underlying lung damage.

The management is based on the severity of the lung injury, the extent of ventilation-perfusion (V:Q) mismatch and the presence of associated injuries.

- Unilateral injuries without significant pulmonary dysfunction:
  - Regional anaesthesia (epidural or paravertebral block)
  - Non-invasive positive pressure ventilation

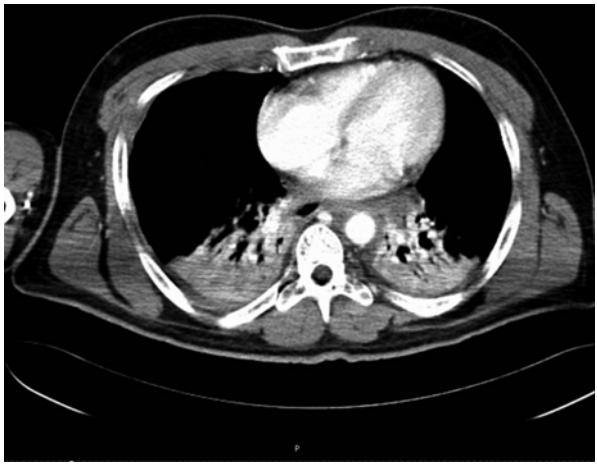


Fig. 43.4 Bilateral lung contusion, notably posterior due to shearing against the spinal column

- Bilateral injuries or unilateral in association with a significant lung contusion:
  - Intubation and mechanical ventilation.
  - Systemic analgesia and not regional anaesthesia is required.

Fluid restriction in the hope of preventing worsening lung function is obsolete and resuscitation should be undertaken along conventional guidelines. There is considerable debate concerning the benefit of rib fixation. While of undoubted benefit in those with major chest wall deformity for both mechanical and aesthetic purposes, its role in flail chest or other rib fractures is uncertain [7].

Pulmonary Contusion

Contusion is a misnomer, and the pathology is not a bruise but a laceration within the lung parenchyma most commonly in the posterior thorax as a result of the lung shearing across the vertebral column (Fig. 43.4). This results in intra-alveolar haemorrhage with surrounding oedema [1]. If the tear extends beyond the visceral pleura, a haemopneumothorax will be evident, but many of these injuries are confined to within the lung parenchyma.

The severity of lung damage can be deceptive, not readily visible on plain chest x-ray, and invariably progresses over the following 24–48 h with worsening hypoxia as a result of V:Q mismatch, the inflammatory response in the injured lung and spill over of blood and secretions into the unaffected lung.

- CT scanning is the diagnostic modality of choice.
- Positive-pressure ventilation is essential, and although pressure support using non-invasive ventilation may suffice for minor contusions, intubation and invasive ventilation should be instituted early for major contusions.

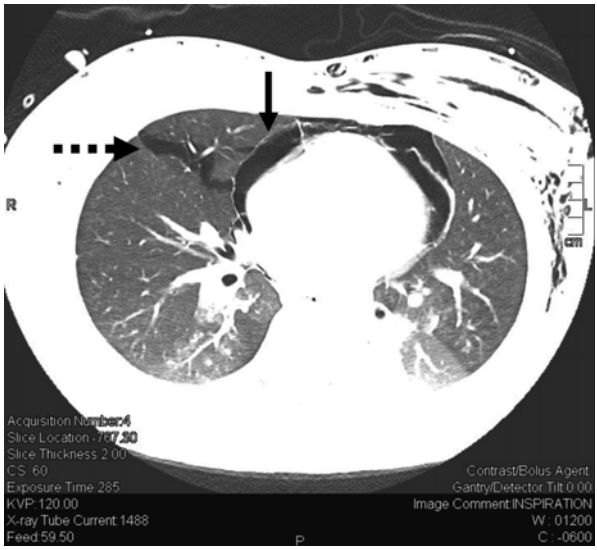


Fig. 43.5 The Macklin effect. Pneumomediastinum and pneumopericardium (*solid arrow*) following blunt thoracic trauma. Air can be seen tracking along the pulmonary vasculature (*broken arrow*), and subcutaneous emphysema is present in the left chest wall

- Initial management: Recruitment is possible using 8–10 ml/kg tidal volumes. Protective lung ventilation (6 ml/kg) has limited initial applicability.
- Recruited alveoli should be maintained using positive end-expiratory pressure (PEEP), and thereafter a more protective ventilatory strategy may be adopted.

Pneumomediastinum and Pneumopericardium

Gas in the mediastinum and pericardium occurs in about 15 % of major blunt thoracic injuries. Unlike its penetrating counterpart where this may signify an oesophageal injury, this is a fairly insignificant finding and arises as a result of alveolar rupture with air tracking along the pulmonary vasculature to be released into the mediastinum or pericardial sac (Fig. 43.5) and named the Macklin effect [8]. On rare occasion sufficient gas may become trapped in the pericardium to cause tamponade.

Aortic Rupture

This accounts for the majority of deaths from exsanguination and 80 % of victims never reach the hospital [9].

- This may occur at any point in the aorta.
- Commonest site is the isthmus (60–80 %) just distal to the origin of the left subclavian artery.
  - Postulated mechanisms which include rapid stretching with intimal tearing
  - Shearing of the aortic wall by twisting

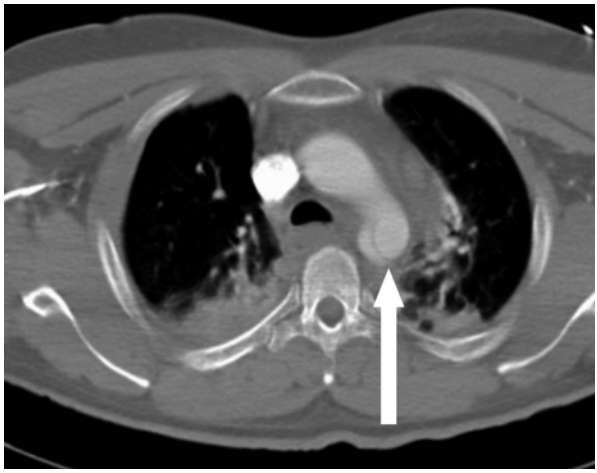


Fig. 43.6 Blunt aortic injury. The intimal tear can be seen as a radio-opaque crescent within the lumen of the descending aorta (*arrow*). The mediastinum is widened and bilateral pulmonary contusions are evident posteriorly

  - Water hammer effect where the diaphragmatic crura obstruct flow through the distal thoracic aorta with a substantial rise in aortic pressure and subsequent tearing

The intima is non-elastic and stretching results in intimal fracture and an intimal flap. Should the lesion progress to involve the elastic media, the aorta becomes acutely aneurysmal. Full-thickness lesions involving the intima, media and adventitia result in death. Intimal lesions may not become aneurysmal acutely, and patients present with pseudo-coarctation with upper limb hypertension and lower limb hypotension.

All patients with significant blunt thoracic injury should have:

- Blood pressure compared in the upper and lower limbs. A discrepancy of >15 mmHg in systolic pressure warrants imaging of the aorta.
- Multiple injuries are the rule and hypotension may arise from acute blood loss from other sites. There are a number of classic findings on plain chest radiology:
  - Wide mediastinum
  - Other salient features being a left pleural cap where blood has tracked extrapleurally from the mediastinum to sit atop the left lung
  - More vertical position of the left main bronchus which is pushed downwards as it sits underneath the aneurysmal aortic arch
  - Loss of the aortic knob
  - Deviation of the oesophagus to the right (abnormal position of a nasogastric tube)
  - Loss of the pulmonary aortic window

As would be expected, there are almost invariably multiple rib fractures and other extra-thoracic injuries. CT angiography is the recommended screening tool in patients fit enough for transportation to radiology (Figs. 43.6 and 43.7).

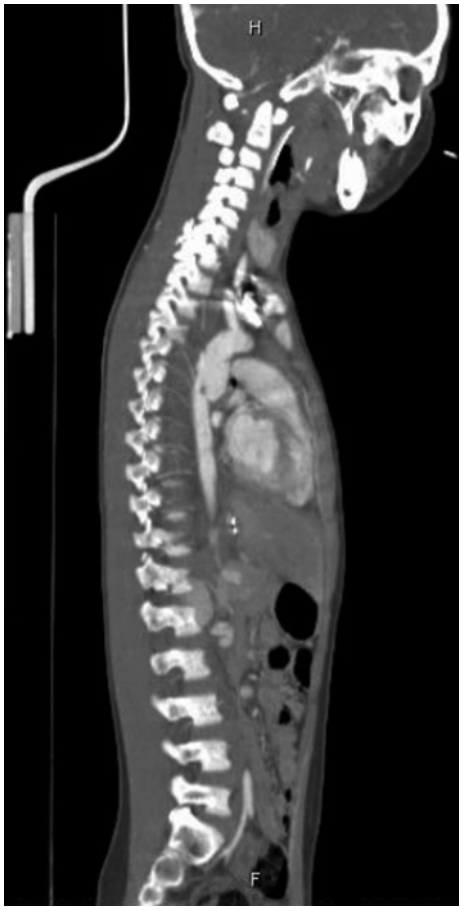


Fig. 43.7 Lateral view of patient in Fig. 43.4. The aneurysmal aorta is clearly visible distal to the left subclavian artery

Acute non-thoracic life-threatening injuries assume priority and thereafter the aortic injury needs to be addressed. Virtually all patients require mechanical ventilation for polytrauma, and initial medical management consists of sedation, analgesia and avoiding hypertension and tachycardias by means of beta blockade with or without vasodilators. Surgical management consists of open repair on or off cardiac bypass or endovascular stenting (Figs. 43.8 and 43.9). Due to the much better outcomes, the latter is now the preferred method in patients with suitable aortic anatomy [10].

Tracheo-Bronchial Injury

This is an exceptionally uncommon injury, occurs in only 1–3 % of major thoracic trauma and has an extremely high mortality rate. As with other major central injuries, the mechanism is usually due to rapid deceleration. The left main bronchus is

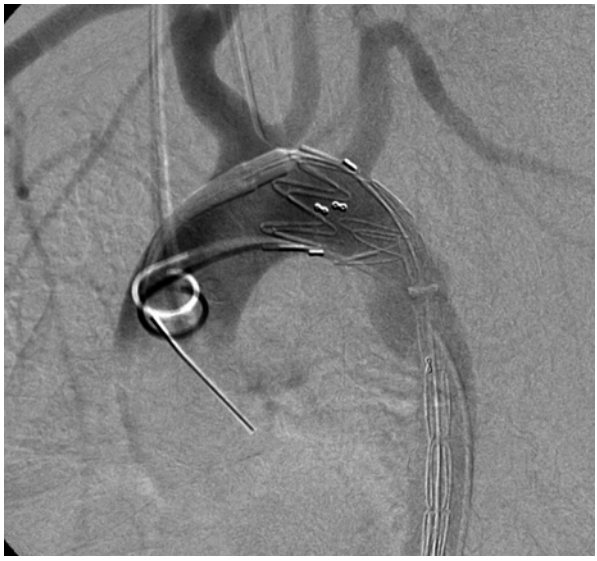


Fig. 43.8 Thoracic endovascular stent being deployed across a traumatic aortic aneurysm. There is a bovine abnormality of the origins of the innominate and left common carotid arteries

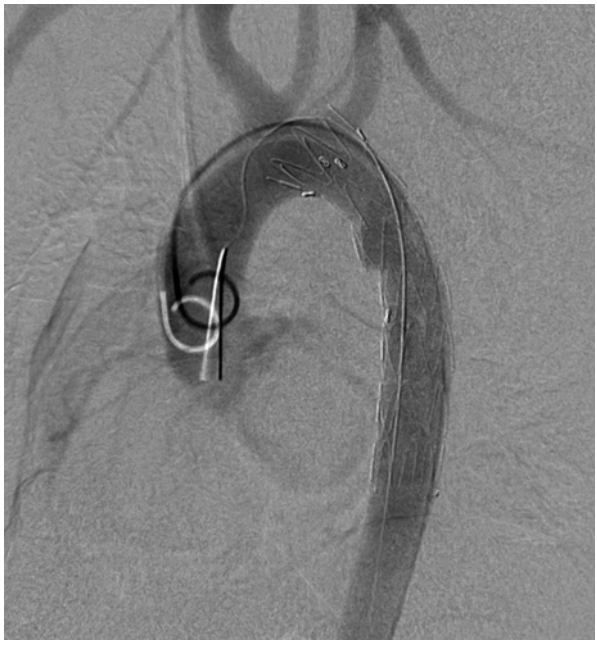


Fig. 43.9 Post deployment angiogram showing endovascular stent in position. Although close to the origin of the left subclavian artery, there is no occlusion of the orifice

protected by the aortic arch and the right main bronchus is the commonest site of injury usually 2 cm from the carina. Patients present with severe respiratory distress and massive subcutaneous emphysema.

Chest x-ray features:

- Large pneumothorax, mediastinal gas.
- Lung lies on the diaphragm (the fallen lung sign).

Placement of an intercostal drain usually worsens the hypoxia as inhaled gas follows the line of least resistance, exiting via the chest drain and resulting in hypoventilation of the good lung. Management entails selective intubation of the unaffected lung under bronchoscopic guidance and repair of the bronchus.

Blunt Cardiac Injury

This arises following rapid deceleration and a direct blow to the chest.

There is a spectrum of damage:

- Minor concussion through contusion
- Infarction
- Rupture of the septum, valves and chordae
- Complete wall disruption

The signs and symptoms depend on the extent of damage and location on the myocardium and range from minor disturbances in rhythm and contractility to major mechanical disruption and death. Unless the coronary arteries are directly involved, myocardial infarction is uncommon. The diagnosis is confirmed by elevated troponin I [11], and the management is as for any non-traumatic dysrhythmia or cardiac dysfunction. Echocardiography is recommended to ascertain any wall motion abnormalities, valve damage or septal defects.

Diaphragmatic Rupture

The right diaphragm is protected by the liver and the left diaphragm is more frequently involved (20 % versus 80 %). Unlike other injuries, this is associated with the use of restraints where the sudden pressure of the lap strap during rapid deceleration produces an acute rise in intra-abdominal pressure and diaphragmatic rupture. The commonest lesion is a radial tear, but on occasion there may be rupture of the central tendon into the pericardial sac or a circumferential tear close to the lateral insertion of the diaphragm. On the right side liver herniation occurs (Fig. 43.10), but on the left bowel herniation occurs acutely in less than 30 % of patients but should it occur gastric herniation may mimic a pneumothorax on x-ray. Before inserting a chest drain, all views should be carefully scrutinized (Fig. 43.11a, b).

Although rarely it may occur in isolation, multiple injuries are the rule and laparotomy is often required for other reasons at which time the diaphragm is repaired using non-absorbable interrupted sutures.

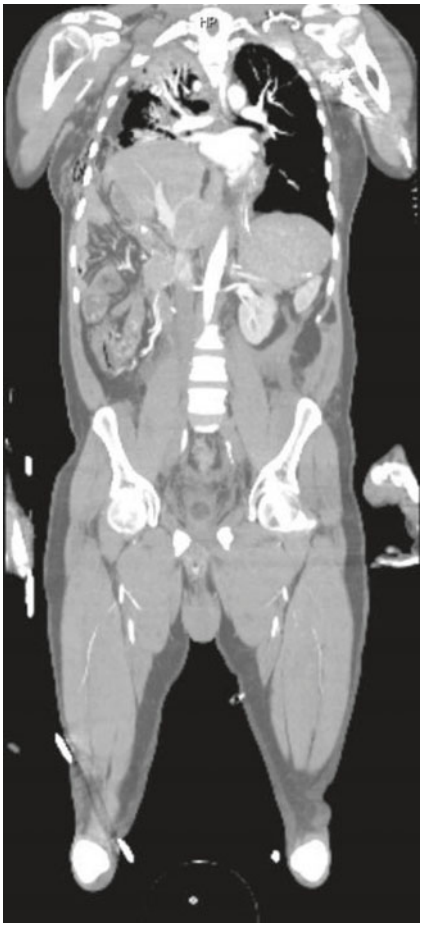


Fig. 43.10 Rupture of the right hemidiaphragm with herniation of the liver into the right hemithorax

Penetrating Thoracic Trauma

Pneumothorax and Haemothorax

As with blunt trauma, peripheral injury involves lung parenchyma only and at most all that is necessary is an intercostal drain for pneumothoraces or haemothoraces. Simple pneumothorax is the rule which in the absence of respiratory distress or signs of a tension pneumothorax can be confirmed radiologically before the insertion of an intercostal drain. The diagnosis of a tension pneumothorax is clinical and management must not be delayed by requesting a confirmatory chest x-ray.

Tension pneumothorax presents with:

- Severe respiratory distress
- Tracheal deviation away from the side of the pneumothorax

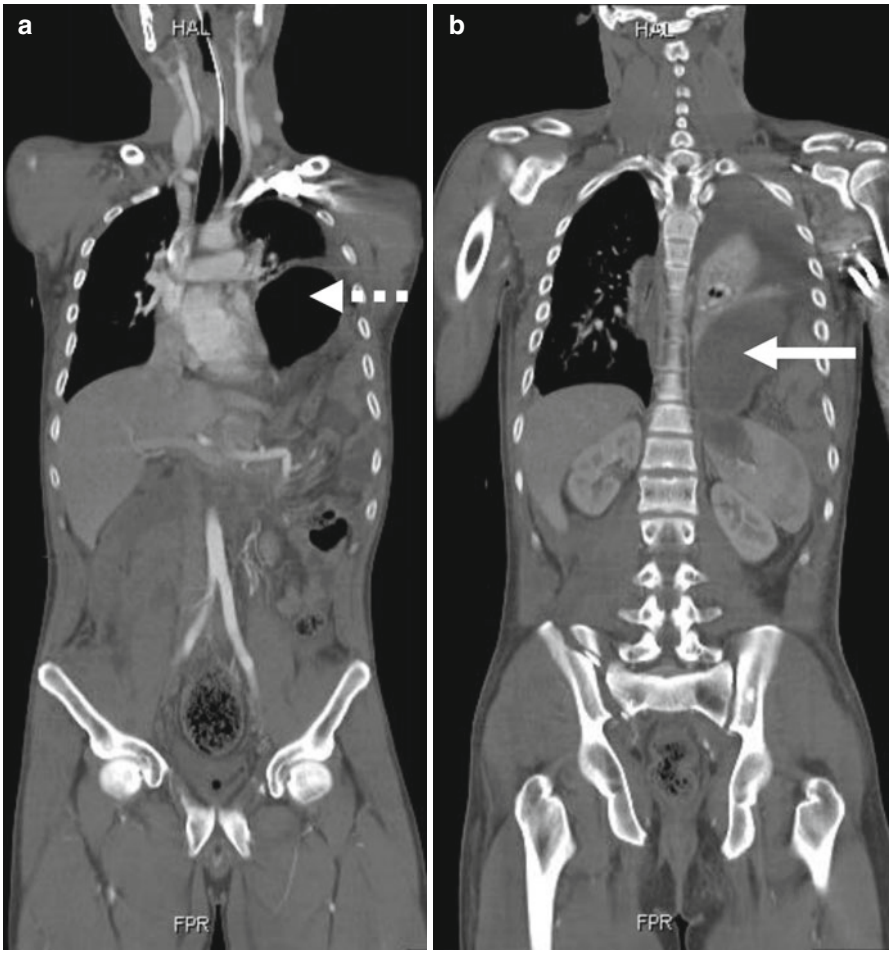


Fig. 43.11 (**a**) Rupture of the left hemidiaphragm with gastric herniation (*broken arrow*). (**b**) More posterior slice showing fluid-filled stomach within the left hemithorax (*solid arrow*). There is a large left haemothorax with collapse of the left lung

- Distended neck veins
- Hypotension
- Hyper-resonant ipsilateral percussion note
- Absent breath sounds on auscultation

Initial management should be by needle thoracocentesis through the fifth intercostal space in the mid-axillary line. The traditional method via the second intercostal space in the mid-clavicular line is now obsolete [12]. Once decompression has been successful with needle thoracocentesis, an intercostal drain should be placed in the anterior or mid-axillary line in the fifth interspace. Immediate intercostal drain insertion before a thoracocentesis has been performed should be avoided

because in the presence of a high pressure air collection within the pleural cavity rapid re-expansion of the lung may allow gas to enter the pulmonary circulation resulting in air embolism.

Ongoing air leaks through an intercostal drain are rarely due to a true bronchopleural fistula as nearly all injuries are peripheral. Management is expectant and most will heal spontaneously. Insertion of a second intercostal drain for persistent pneumothoraces may be required with or without high-volume low-pressure suction.

Surgery for peripheral lesions is rare but may be required for persistent haemorrhage most commonly arising from an intercostal vessel, occasionally from a lung laceration.

Mediastinal Trauma

Gunshot wounds to the mediastinal structures are associated with high prehospital mortality rates, and the majority of injuries arise from stab wounds to the heart or great vessels.

Cardiac Injury

The classic presentation of penetrating cardiac trauma is with tamponade which results in distended neck veins, muffled heart sounds and hypotension, the eponymous Beck's triad described in 1912. The only differential diagnosis is a tension pneumothorax which is distinguished by the hyper-resonant percussion note and absent breath sounds on the affected hemithorax. Cardiac injuries rarely present with diminished air entry for if the wound involves both pleura and heart exsanguination is the norm. Although all cardiac chambers are at risk, the commonest chamber to be injured is the misnamed right ventricle which sits anteriorly. The surgical approach may be via median sternotomy which gives optimal access or left anterior thoracotomy extended transversely across the sternum if further access is necessary. The pericardium is divided in the midline avoiding the more lateral phrenic nerves. In the face of major haemorrhage (which is uncommon), control may be obtained by digitally squeezing the edges of the wound together or inserting a Foley catheter into the laceration. Great care must be taken with this latter technique as it has the potential to enlarge the laceration if the inflated balloon pulls through the wound during repair which is by non-absorbable sutures. With lesions in close proximity to the coronary vessels, a mattress suture must be inserted lateral to the laceration, passed deep to the vessel, out of the myocardium and then returned to the point of insertion. In the absence of myocardial swelling, the pericardium may be closed leaving a small window inferiorly for drainage. Should closure not be possible, the pericardium should be left open. Pericardial and pleural drains are inserted as necessary.

Injury to Mediastinal Vessels

Injury to the great vessels may result in partial or complete occlusion or an arteriovenous fistula. As with cardiac trauma, only those with contained injuries reach the hospital, and time is usually available for angiography to determine the nature of the lesion. Where amenable, stenting has now become the procedure of choice.

Oesophageal Trauma

Blunt injury to the thoracic oesophagus is virtually non-existent and almost all arise from penetrating trauma [13]. Undiagnosed this injury is highly lethal, death arising from uncontrolled mediastinal sepsis. The diagnosis is suspected by a history of dysphagia and odynophagia, vomiting and haematemesis, crepitus in the root of the neck and mediastinal gas and hydrothorax on plain chest x-ray. Speed is of the essence in confirming or excluding the injury, the gold standard being a contrast swallow using water soluble iso-osmolar, nonionic contrast medium [14]. Although barium is the most accurate medium, this should be avoided in the initial investigation for leakage through a perforation which may result in mediastinal inflammation. Gastrografin should also be avoided if there is a risk of aspiration as this causes severe pneumonitis. If the initial swallow is normal using the preferred contrast medium, a dilute barium swallow may be performed as this has a higher diagnostic yield.

Surgery is the mainstay of management, the procedure depending on the time between injury and diagnosis. As soon as a perforation is confirmed, the patient should be commenced on appropriate antimicrobials. The injury must be exposed by opening the mediastinal parietal pleura widely, followed by debridement, irrigation and drainage of the mediastinum. The proximal two-thirds are approached via a right thoracotomy, the distal third from the left hand side. If surgery is undertaken early, the perforation may be closed using interrupted sutures with or without buttressing using pleural or intercostal muscle flaps, but beyond that time frame, suturing is likely to fail and wide free drainage and systemic antimicrobials are the recommended options. At the time of surgery, a nasogastric tube must be placed as enteral feeding is still possible postoperatively.

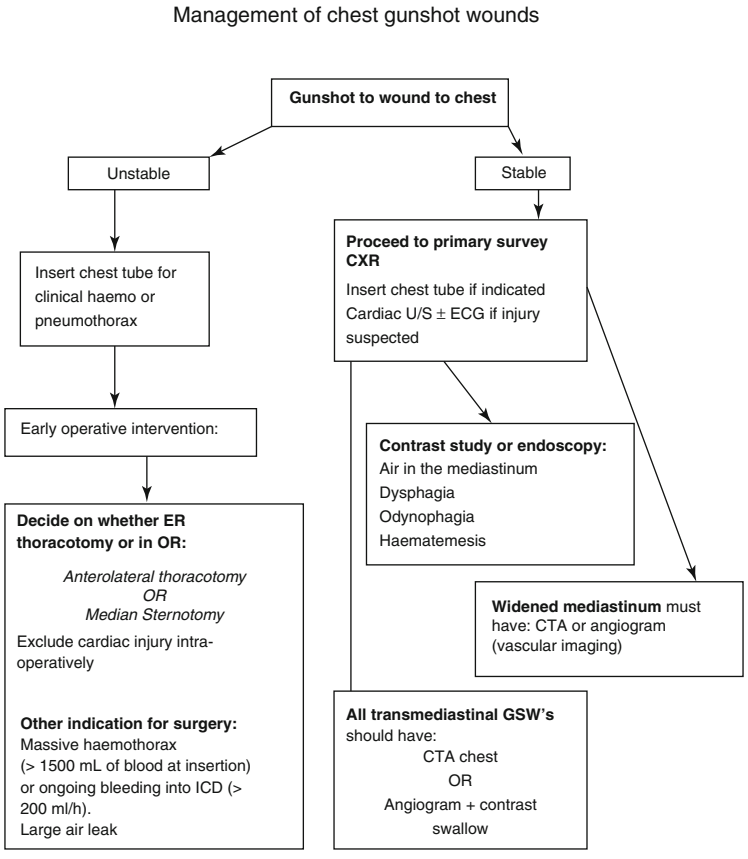
Diaphragmatic Injury

These most commonly occur following a left lower chest stab wound and unless there is a coexistent hollow visceral abdominal injury may go unrecognized. Although some may heal spontaneously, there is a risk of subsequent diaphragmatic herniation days, weeks, months or even years later with significant morbidity and mortality rates. As a result there has been a trend to confirm or exclude these injuries by means of thoracoscopy or laparoscopy during which the lesion may be repaired [15].

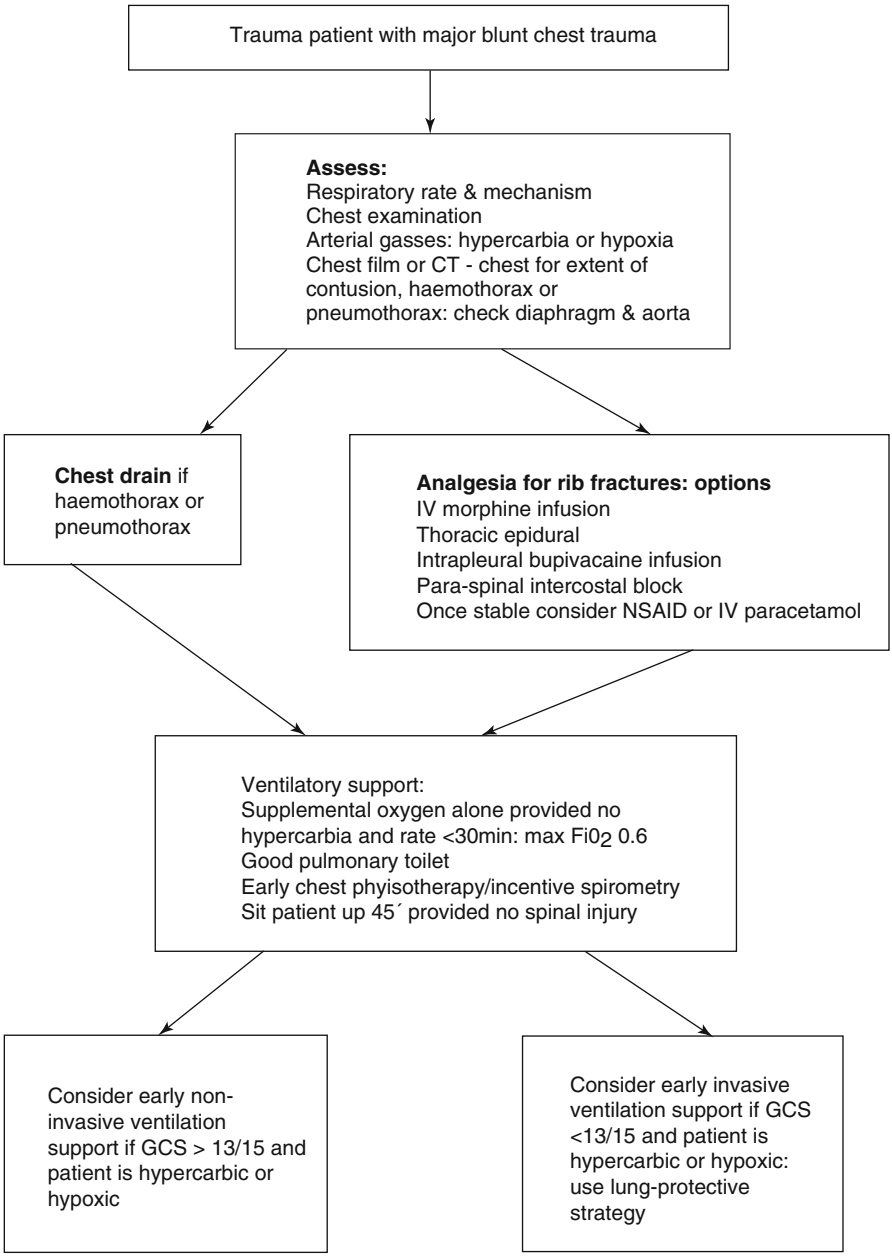
Emergency Room Thoracotomy

The commonest indications for thoracotomy outside of the operating room are in patients with penetrating wounds who are in extremis following severe intrathoracic or intra-abdominal haemorrhage or pericardial tamponade. In the first two the procedure is for direct control of haemorrhage or release of tamponade; the last mentioned is termed a resuscitative thoracotomy whereby the distal thoracic aorta is cross-clamped to minimize further abdominal haemorrhage until a laparotomy can be performed. This is a heroic attempt to salvage a dire situation and patients must be carefully selected [16]. The physiological indications are witnessed cardiac arrest for less than 5 min with CPR in progress; electrical activity on the electrocardiogram with or without a palpable pulse and evidence of papillary, corneal or gag reflexes. There are considerable risks to emergency department staff due to the rapid use of sharp instruments and inevitable exposure to uncontrolled bleeding. The outcome is generally poor with overall survival rates of <10 % although this is substantially better for cardiac tamponade compared to thoracic or abdominal haemorrhage [17]. Emergency thoracotomy for blunt thoracic trauma is rarely, if at all indicated.

Flowchart for penetrating chest trauma (Trauma Society of SA Consensus Document, used as Shareware)



Flowchart for blunt chest trauma (Trauma Society of SA Consensus Document [Shareware])



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