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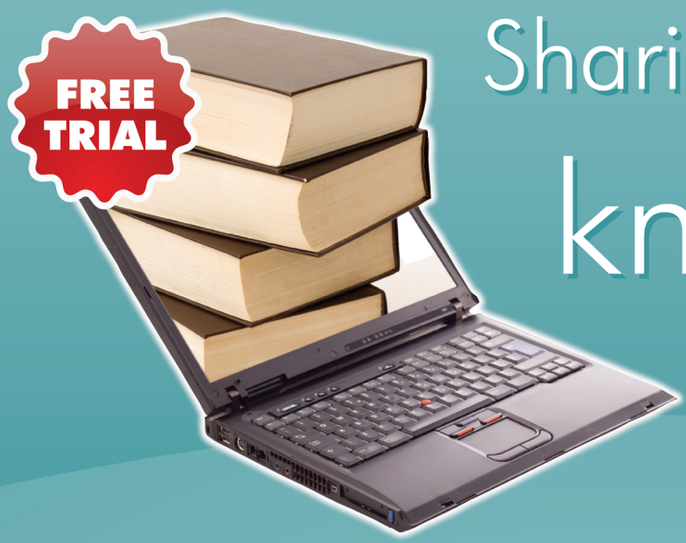

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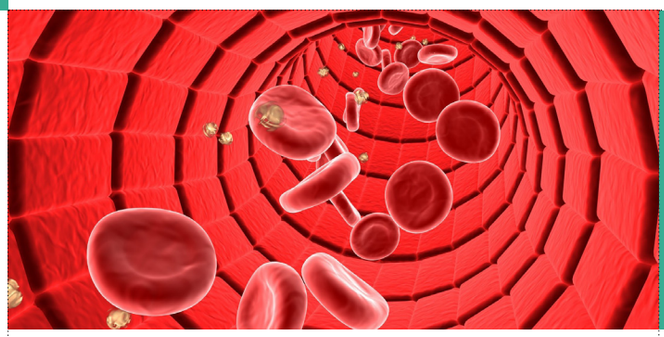
AN AUDIT INTO D-DIMER UTILISATION AT ROYAL BOURNEMOUTH HOSPITAL

J Blackman, H Cutler



An Audit Into D-Dimer Utilisation
At Royal Bournemouth Hospital
Teaching & Training

Abstract

Background

The D-Dimer antigen is a protein fragment present in blood following degradation of fibrin. Its presence is associated with venous thromboembolism (1) and an assay can be used to exclude venous thromboembolism in low risk patients (2). This audit arose from a perception that D-Dimer assays were being over-utilised at Royal Bournemouth Hospital (RBH).

Methods

A list of ordered D-Dimers from the hematology laboratory was retrieved over seven days. Case notes were retrieved and the appropriateness of the test was evaluated against three criteria. For the D-Dimer to be considered appropriate all three had to be met.

1) Pulmonary embolus considered as a diagnosis
2) Low risk patient as defined by Wells Score (<2)
3) Absence of infection

Results

39 case notes were retrieved, only 27% were deemed appropriate according to the above criteria. 51.3% of D-Dimers were ordered without PE being considered as a diagnosis. This occurred because D-Dimers were requested in the Emergency Department prior to patients being medically clerked as part of a 'battery' of standard blood tests. Interventions included discontinuing this practice and education.

A re-audit revealed a similar percentage of appropriate D-Dimers (26%) however the absolute number of D-Dimers decreased by 176 over two months saving approximately £5200 per annum.

Conclusion

D-Dimer blood tests are over-utilised at RBH, however post intervention their absolute number has dropped significantly yielding financial saving and clinical benefit / patient safety through minimising inappropriate further investigations.

Introduction

The D-Dimer antigen is a protein fragment present in blood following the degradation of fibrin. Factor VIIIa catalyses the formation of covalent bonds between D-Domains of polymerised fibrin. The D-Dimer antigen is exposed when cross-linked fibrin is degraded by plasmin (3). As such D-Dimer assays have a role in the investigation of suspected acute venous thromboembolism (4).

D-Dimer has a high sensitivity (93-95%) and low specificity (approx. 50%) (5). As such D-Dimer has proven efficacy in low risk groups for excluding PE (6) but is widely considered unhelpful in high risk populations (7). The Wells Score can be used to risk stratify the general population and when used with a D-Dimer can safely exclude venous thromboembolism (8) (See Appendix 1). Its low specificity in particular makes D-Dimer unhelpful as a screening tool.
This audit arose due to a perception that D-Dimers were being over utilised at Royal Bournemouth Hospital. Hence there were two specific aims:

1) To audit the proportion of D-Dimer assays at Royal Bournemouth Hospital which are clinically indicated.
2) To try and minimise expenditure and clinical risk associated with unnecessary investigations.

Appendix 1

Wells Score for Pulmonary Embolism	Score
Clinical signs and symptoms of DVT	3
PE is the most likely diagnosis	3
Tachycardia (>100)	1.5
Immobilisation or surgery in the previous 4 weeks	1.5
Previously diagnosed DVT / PE	1.5
Haemoptysis	1
Active malignancy	1

Score <2 = Low Risk

Score <6 = Intermediate Risk

Score <12.5 = High Risk

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J Blackman, H Cutler

Methods

A list of patients for whom D-Dimers had been performed on seven separate dates was produced. All patients on this list who were in-patients at RBH or had been discharged the same day from the Emergency Department were entered into the audit. D-Dimers ordered for hematological reasons e.g. DIC or as a work-up for deep vein thrombosis were excluded.

The case notes for these patients were retrieved and assessed for their adherence to the following criteria. Only D-Dimers meeting all 3 criteria were considered to be appropriate.

1) Pulmonary embolism documented as a differential diagnosis at any stage in the notes i.e. initial clerking, post-take ward round or entry prior to which the D-Dimer was checked.
2) Low risk patients as defined by Wells Score for PE less than or equal to 2.
3) Infection not documented as a differential diagnosis with pulmonary embolus.

The standard was for 100% of D-dimer requests to meet these criteria. If a D-Dimer is used appropriately one might also reasonably expect a positive test to be followed up with imaging to exclude pulmonary embolus. This was also examined.

The following data was gathered using a data collection form (Appendix 2).

- **DOB**
- **Hospital Number**
- **Admission Date**
- **Ward from which D-Dimer was requested (ED, Medical, Surgical, Other)**
- **Presenting Complaint**
- **Whether PE was listed in a differential diagnosis**
- **Whether infection was listed in a differential diagnosis**
- **Calculated Wells Score**
- **Probability of PE based on Wells Score**
- **Whether Wells Score was documented**
- **Indication for PE (PE, None Clear, DVT, Hematological)**
- **Date of D-Dimer**
- **D-Dimer Level and Status (Positive, Negative)**
- **Subsequent Radiological Investigation (None, VQ, CTPA)**
- **Result of Subsequent Radiological Investigation**

Exactly the same audit method was used following interventions to complete the audit loop although a two week period was used in order to compare similar numbers of D-Dimers.

Appendix 2

D-Dimer Audit Collection Sheet

Patient Details

DOB : Day__ Month__ Year____

Hospital Number _____

Admission Date : Day__ Month__ Year____

Ward : ED ☐ AAU ☐ Medical ☐ Surgical ☐

Likelihood of PE

Presenting Complaint _____

PE listed in Diff Diag? : Yes ☐ No ☐

Infection listed in Diff Diag? : Yes ☐ No ☐

Calculated Wells Score ___

Probability of PE : Low ☐ Intermediate ☐ High ☐

Wells Score Documented? Yes ☐ No ☐

D-Dimer

Indication : PE ☐ DVT ☐ Other ☐ None Clear ☐

Date of D-Dimer : Day__ Month__ Year____

Level : _____

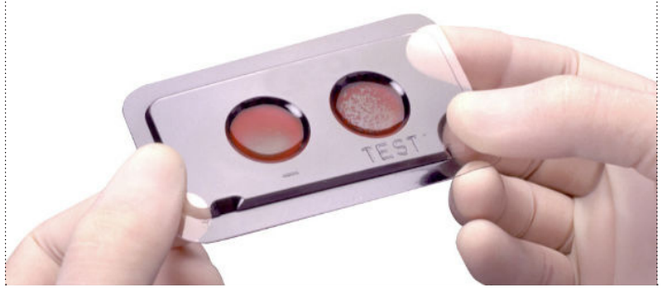
Status : Positive ☐ Negative ☐Not performed ☐

Imaging

Ix Performed : CTPA ☐ VQ ☐ None ☐

Date of Ix : Day__ Month__ Year____

Result : Positive ☐ Negative ☐ Equivocal ☐



AN AUDIT INTO D-DIMER UTILISATION AT ROYAL BOURNEMOUTH HOSPITAL

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An Audit Into D-Dimer Utilisation At Royal Bournemouth Hospital
Teaching & Training

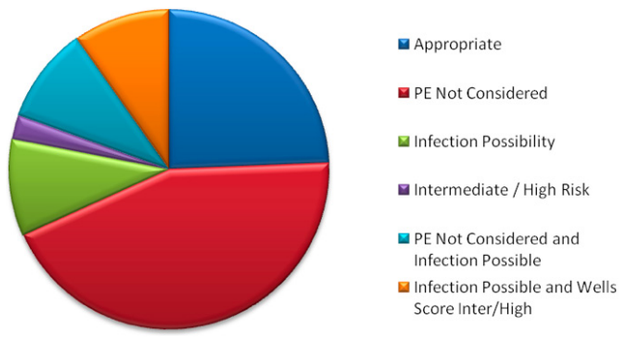
Results

A total of 39 case notes were examined over a seven day period in Phase I. Two were excluded as they were ordered for DVT and hematological reasons. Of the remaining 37, 81% of the D-Dimers were ordered from the Emergency Department, 16% from medical wards and 3% from surgical wards. The full table of results is shown in Appendix 3.

In terms of meeting the standards set out:

- **Proportion of D-Dimers ordered with pulmonary embolus considered as a diagnosis = 51.3% (19/37)**
- **Proportion of D-Dimers ordered for low risk patients i.e. Wells Score <2 = 86.4% (32/37)**
- **Proportion of D-Dimers ordered where infection was not listed as a differential = 67.5% (25/37)**
- **Proportion of D-Dimers meeting all above standards i.e. ordered appropriately = 27.0% (10/37)**
- **Proportion of positive D-Dimers followed up with radiological investigation for PE = 31.3% (5/16)**

Of the D-Dimers ordered the breakdown is shown graphically below.



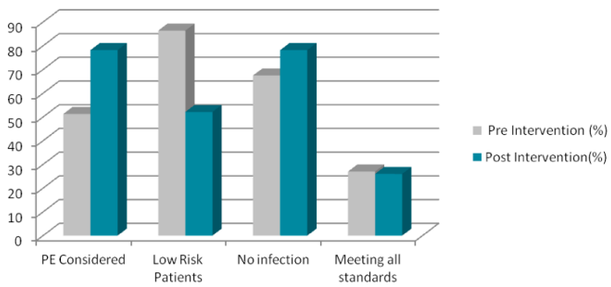
Appendix 3

For Phase II (Post Intervention), data was collected from 10/11/11 – 24/11/11. 23 case notes were examined. The proportion of D-Dimers ordered from ED dropped from 81% to 47%. The proportion from medical wards increased from 16% to 47% and the number from surgical wards increased fractionally to 6% from 3%. A full table of results is shown in Appendix 4.

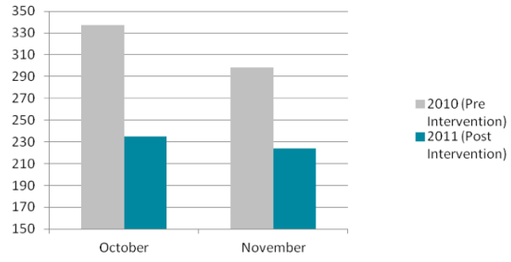
Standards pre and post intervention are tabled and graphed below.

Standard	Pre Intervention (%)	Post Intervention(%)
Proportion of D-Dimers with PE considered as diagnosis	51.3	78.2
Proportion of D-Dimers ordered for 'low risk' patients	86.4	52.1
Proportion of D-Dimers with infection not listed in differential	67.5	78.2
Meeting all 3 above standards	27.0	26.0
Proportion of positive D-Dimer followed by radiological Ix	31.3	41.0

Appendix 4



The absolute number of in-patient D-Dimers performed was compared from year to year.



AN AUDIT INTO D-DIMER UTILISATION AT ROYAL BOURNEMOUTH HOSPITAL

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Over a two month period there were 176 fewer D-Dimer requests compared with the equivalent period the previous year. Over the same period the number of GP or DVT requested D-Dimer tests remained roughly equal (244 compared with 242) suggesting that there were unlikely to be any other factors besides the interventions above affecting this result. If this trend were to be repeated throughout the year this would lead to a saving of over £5200 per annum.

Discussion

The Phase I results quite clearly showed that the majority of D-Dimers ordered at RBH were not clinically indicated and this was frequently due to pulmonary embolus not being suspected. A large proportion of these requests originated from ED where pulmonary embolus had not even been considered as a diagnosis by the clerking clinician or post take consultant.

This appeared to occur as a result of the D-Dimer being ordered as part of a 'package' of blood tests automatically triggered if the presenting complaint is shortness of breath or chest pain. Furthermore a large proportion of D-Dimers both in ED and on the wards are measured in the context of infection which helps to invalidate a positive result.

Very few D-Dimers were ordered in intermediate or high risk groups. At first glance this may appear encouraging, however this may be because on a frequent basis, pulmonary embolus was not entertained as a potential diagnosis in the first place and therefore the patient tended to score as low risk when the Wells Score was calculated.

Only 31% of positive D-Dimers resulted in further diagnostic imaging supporting the hypothesis that approximately 70% were unnecessary.
The inappropriate use of D-Dimers could be seen as problematic for two main reasons:

1) Financial

Each D-Dimer costs approximately £5 to run. Over a seven day period an average of four D-Dimers were inappropriately ordered per day. As such savings of approximately £7200 could be made per year if these were not requested.

2) Patient Safety

The presence of a positive D-Dimer could be argued by some to necessitate investigation regardless of whether it was initially clinically indicated or not. At the very least a positive result could be seen to cloud the issue and lead to the consideration of imaging to exclude a diagnosis which was not previously clinically considered. This could potentially cause harm both through exposing patients to the risk of unnecessary anti-coagulation or exposure to radiation through imaging to exclude PE. A CTPA is approximately equivalent to between 1-2 years of background radiation.

Hence in response to these results, we put in place a number of measures to maximise the proportion of appropriate D-Dimers and minimise the number of unnecessary investigations. Interventions included:

1. The removal of the D-Dimer from the pre-determined 'package' of blood tests ordered from the Emergency Department.

If the phlebotomist feels that a D-Dimer is clinically indicated, either a clinician verifies this or a coagulation sample is taken so that a D-Dimer can be added at a later time. This avoids unnecessary delay or repeated blood tests.

AN AUDIT INTO D-DIMER UTILISATION AT ROYAL BOURNEMOUTH HOSPITAL

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2. Education sessions delivered to junior doctors regarding appropriate use of the D-Dimer.

The Phase II results showed that the proportion of D-Dimers which are appropriate, according to the standards set out by this audit, have stayed approximately static after the interventions had been put in place. However the proportion of D-Dimers that were ordered without PE having even been considered as a diagnosis has fallen significantly. This has led to a significant reduction in the overall numbers of D-Dimers requested and performed.

In addition the proportion of D-Dimers requested in the context of infection has also fallen, suggesting that educational measures are helping. The proportion of D-Dimers requested in medium to high risk patients, however, has increased. This may be because having eliminated most of the D-Dimers for patients in whom PE was not a realistic possibility, the remaining share are inevitably higher risk for PE. It is this effect that led to the proportion of 'appropriate' D-Dimers remaining static at approximately 26% despite the interventions. Further interventions emphasising the benefits of D-Dimers only in low risk patient groups is required although the absolute fall in numbers is encouraging.

Finally the proportion of positive D-Dimers followed by definitive radiological investigation to exclude PE increased from 31% to 41% suggesting that more are appropriately ordered.

Overall the audit has therefore been highly successful in reducing the numbers of D-Dimers and leading to a significant cost saving (over £5000 per annum) with potential patient safety benefits as outlined above.

An Audit Into D-Dimer Utilisation At Royal Bournemouth Hospital
Teaching & Training

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Authors

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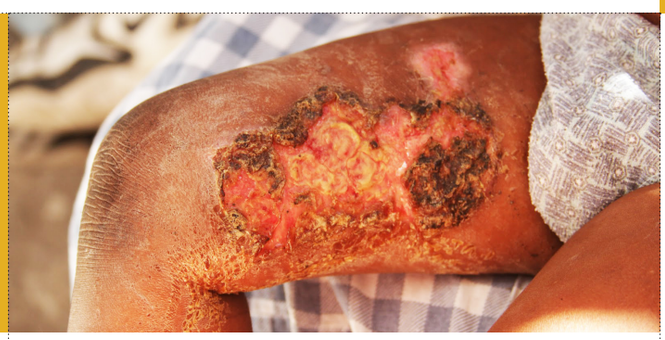
MANAGING ACCIDENTAL CHILDHOOD BURNS

S Bertaud, A Banerjee



Managing accidental childhood burns
Good Clinical Care

Abstract

We present a case based discussion of a five year old boy who attended the Accident and Emergency department with 18% surface area of burns following a household accident. It highlights the key elements of assessment in paediatric resuscitation and the principles of management of burns in children. We look at the criteria for referral to a tertiary centre and the safeguarding issues that can arise from cases of paediatric injury.

Case history

Edmund Burke, the philosopher wrote 'The first and simplest emotion which we discover in the human mind, is curiosity'. The curiosity of a child is often beautiful to behold yet potentially dangerous, as our case history reveals.

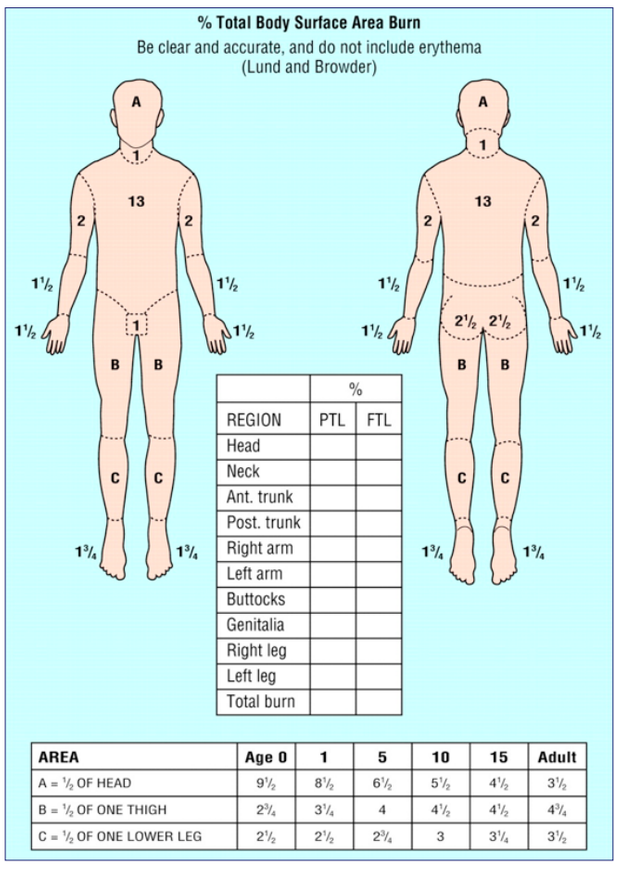
A five year old boy was brought into the Accident and Emergency department with a large area of burns to the body. He had sustained accidental flame burns while playing with a barbecue lighter that he had found in the kitchen. His mother had removed his T-shirt and put him straight into a cold shower before calling an ambulance. On arrival of the ambulance crew at the scene, the child was found to be alert, and crying, in the shower.

The patient was normally fit and well, with no other medical problems or regular medications. He was up to date with his immunisations and reaching developmental milestones without concern. Tetanus prophylaxis was subsequently not required as his immunisations were up to date. There was no family history of illness and he lived at home with his parents, his younger sister and two older half-siblings.

Examination and further investigations

The child was taken to the Resuscitation Room on arrival in the emergency department. He was weighed so as to allow appropriate fluid replacement and analgesia to be started. Initial ABC assessment revealed an alert child, with a patent airway, a clear chest, good oxygen saturation and vital signs within acceptable limits for his age. Pain relief was given with oramorph initially, followed by intravenous morphine, titrated to response. The patient was continually assessed for levels of pain and importantly the team attempted to maintain a reassuring and calm atmosphere within the resuscitation department. Intravenous access was obtained with two cannulae, and venous blood was taken for full blood count, biochemistry, group and save, and blood gas estimation. Intravenous maintenance fluids were commenced whilst initial assessment was still underway. The child remained alert throughout, albeit in distress.

The dressings applied by the ambulance service were removed and the burns were formally assessed using a Lund-Browder surface area calculation chart (See Figure 1). Findings were as follows: partial thickness burns to the front of torso of 13% of the total body surface area (TBSA), to the right arm, 3.5% of TBSA and to the left upper arm, 1.5% of TBSA. The patient was log rolled to ensure there were no other injuries.



	%	
REGION	PTL	FTL
Head		
Neck		
Ant. trunk		
Post. trunk		
Right arm		
Left arm		
Buttocks		
Genitalia		
Right leg		
Left leg		
Total burn		

AREA	Age 0	1	5	10	15	Adult
A = ½ OF HEAD	9½	8½	6½	5½	4½	3½
B = ½ OF ONE THIGH	2¾	3¼	4	4½	4½	4¾
C = ½ OF ONE LOWER LEG	2½	2½	2¾	3	3¼	3½

Figure 1

MANAGING ACCIDENTAL CHILDHOOD BURNS

S Bertaud, A Banerjee

Further management

All children with burns over 10% TBSA are to receive fluid according to the Parkland formula, in addition to maintenance fluids:

4ml/kg/% burn over 24 hours from the time of injury given ½ in the first 8 hours and ½ in the second 16 hours, given as Hartmann's fluid.

Guidelines suggest that all children with burns ≥20% TBSA should have an appropriate size urethral catheter; for burns 10-19% TBSA, catheterisation should be considered. In this case, although the patient was passing urine, the decision was made to catheterise him to optimise fluid management.

The patient went on to be referred to the local specialist burns service, who advised on dressing the wounds with clingfilm and loose circumferential bandages over the top prior to transfer. Dressing the burn reduces pain and helps to prevent infection The advantage of cling film as a dressing is that it is transparent so the burns can be re-examined in the specialist burns unit without removing the dressings and causing further pain to the child.

He was admitted to the tertiary centre for ongoing care, and made a good recovery. Referral criteria for specialised burn services are detailed in Figure 2. For children being followed up as outpatients, the parents should be informed about the possibility of toxic shock syndrome (see Figure 3) and be asked to bring back the child if the symptoms are suggestive of this rare but potentially fatal complication.

Managing accidental childhood burns
Good Clinical Care

REFERRAL CRITERIA FOR CHILDHOOD BURNS TO A SPECIALIST BURN SERVICE

- All partial thickness (PT) burns >10% Total Body Surface Area (TBSA) (Discuss if >5-9% BSA on a case by case basis)
- All deep dermal and full thickness (FT) burns
- Burns involving the face, hands, soles of feet, perineum, or major joints
- Circumferential burns of limbs
- All burns associated with smoke inhalation
- High voltage electrical injury, including lightning injury
- Severe metabolic disturbance
- Children with burn wound infection
- All children 'unwell' with a burn (see below)
- Unhealed burns after 2 weeks
- Neonatal burns of any size

Figure 2

Figure 3

UNWELL: Toxic Shock syndrome/Burns Sepsis syndrome – ANY OF:

- Temperature > 38 ^{0}C
- Rash
- Diarrhoea and vomiting
- General malaise
- Not eating and drinking
- Tachycardia/tachypnoea
- Hypotension

Figure 3

Finally, although there were no safeguarding concerns in this case, the patient was booked to be discussed at the weekly paediatric psychosocial meeting.

MANAGING ACCIDENTAL CHILDHOOD BURNS

S Bertaud, A Banerjee

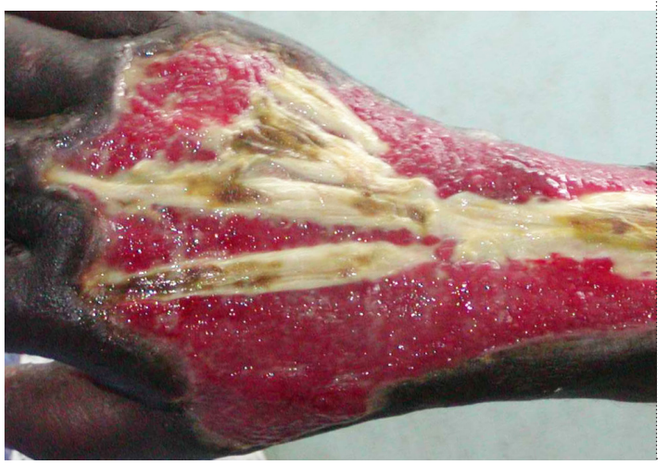
Discussion

Children under 10 years of age account for approximately 36% of burns seen in the Accident and Emergency department (1). Thermal injury is the most common mechanism of injury and scalds account for >60% of such injuries (2). Initial ABC assessment and resuscitation in line with current APLS guidelines is an essential first step in all cases of paediatric resuscitation. Accurate assessment of burn area is crucial to calculate the resuscitation formula and the Lund and Browder surface area calculation chart is a vital tool in this assessment.

Erythema should not be included when assessing the area of a burn. It is important to remember that burn depth is difficult to estimate and is also liable to change as resuscitation progresses. It is therefore necessary to make a quick initial sensible assessment in the acute setting and to discuss the presentation with a specialist centre at the earliest possible time. Determining burn depth involves a clinical assessment of appearance, sensation, bleeding on needle prick (best avoided in children), and blanching to pressure (3).

All children with burns will experience pain, regardless of the cause, size, or depth of the burn (4). We know that when pain is inadequately treated this can result in non-compliance with treatment and, consequently, prolonged healing. It is important to take a pre-emptive approach to pain relief and to ensure an environment where the child is as relaxed and comfortable as possible. Studies suggest that a successful approach involves frequent assessment of pain with readiness to try alternative or additional measures when pain relief seems inadequate (4).

As with all childhood injuries it is important to make an assessment of any child protection concerns you may have. This involves the use of standard indicators of risk for child abuse such as whether the child has previously been seen at the department, whether there is an inconsistent medical history, whether the findings on examination match the history and whether there was a delay in bringing the child to the department. Deliberately inflicted burns and scalds are found in 10% of physically abused children, 5% of sexually abused children and 1-16% of all children presenting to hospital with burns and scalds (5). However, this form of physical abuse is difficult to diagnose and is therefore often under-recognised.



A systematic review looking at distinguishing features of intentional and unintentional scalds found that intentional scalds were commonly immersion injuries, caused by hot tap water, affecting the extremities, buttocks or perineum or both. The scalds were symmetrical with clear upper margins, and associated with old fractures and unrelated injuries. Unintentional scalds were more commonly due to spill injuries of other hot liquids, affecting the upper body with irregular margins and depth (6). The pattern of burn injury should match the alleged history. For example, if a child reaches out and pulls at a container of hot liquid, the spill will be onto the front of the body, usually the chest, not the back, and the pattern of burn will equate with the pattern of fluid run-off.

Paediatric resuscitation ultimately requires careful and accurate assessment of the patient in a logical manner but with a willingness to adapt to the individual child to ensure adequate pain relief and minimal distress. As with all paediatric injuries, remaining vigilant to the possibility of non-accidental injury and being prepared to 'think the unthinkable' is a vital skill in safeguarding children.This case highlights the potentially devastating consequences of the curiosity of the young mind and the need for the utmost vigilance when caring for the inquisitive child.

Self-assessment

Question 1 – True or false

The following are tools for assessment of burn surface area:

a) Palmar surface of hand

b) Infrared thermography

c) Wallace's rule of nines

d) Lund and Browder chart

e) Laser Doppler imaging

Question 2 – Best of Five

You are called to assess the depth of a burn. The burn is red, dry, and blanches on pressure but regains its colour slowly. The burn is tender to touch and bleeds briskly on superficial pricking. The burn is:

a) Full thickness

b) Partial thickness

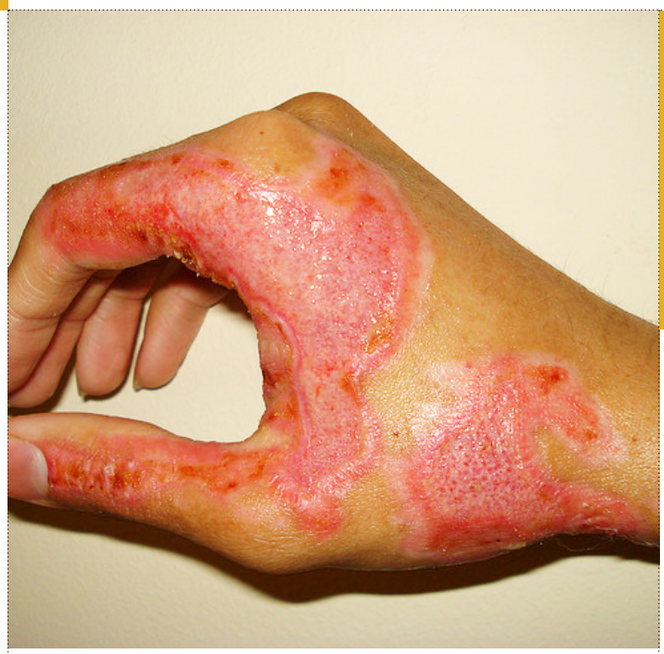
c) Deep dermal

d) Epidermal

e) Mixed depth

MANAGING ACCIDENTAL CHILDHOOD BURNS

S Bertaud, A Banerjee



Answers

Question 1

a) True
The surface area of a patient's palm is approximately 0.8% of their total body surface area. This method is good for assessing the size of relatively small burns (< 15% of total surface area) or very large burns (> 85%, when unburnt skin is counted) but is inaccurate for medium size burns.

b) False
Infrared thermography is an experimental tool for assessing burn depth.

c) True
This is a good, quick way of estimating medium to large burns in adults. The body is divided into areas of 9%, and the total burn area can be calculated. It is not accurate in children.

d) True
This body chart is the most accurate method because it compensates for the variation in body shape with age. It is therefore the most accurate assessment of the area of a burn in children.

e) False
This is another experimental tool for burn depth assessment

Question 2

c) This burn is partial thickness. Essentially, partial thickness burns encompass a spectrum of increasing severity from fixed erythema, to intact blisters, to blisters which have ruptured. Fixed erythema and intact blisters are dry because the epidermis is still in place. Ruptured blisters, if partial thickness, have a moist surface in the floor of the blister, they have a uniformly pink appearance, and they blanch on pressure.

In partial thickness burns, the dermis may be involved, but there is sparing of an underlying continuous layer of dermis from which all the elements of the skin (sweat glands, sebaceous glands, hair follicles, pigment cells, etc) can regenerate. Nerve endings are not destroyed, and these burns are very painful, and sensation is intact. Healing takes 10-14 days. There is initial scarring which fades completely over time.

Deep dermal burns are intermediate between partial thickness and full thickness burns. They arise when the burn has extended more deeply into the dermal layer, sparing the rete pegs which dip down into the subcutaneous tissues, but burning through the full thickness of some of the more superficial elements of the dermis. This gives rise to a mottled appearance.

The typical appearance of a deep dermal burn, is of ruptured blisters; the floor of these blisters is also moist, with a mottled, often pale appearance, mingling with cherry red areas caused by fixed capillary staining because the blood is fixed within damaged capillaries in the deep dermal plexus, rather than the uniform pink appearance of a partial thickness burn.

MANAGING ACCIDENTAL CHILDHOOD BURNS

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There may also be some extravasated blood. In deep dermal burns, the skin regenerates from the spared rete pegs. Deep dermal burns are slower to heal than superficial burns, and healing takes at least three weeks. Some elements of the skin will have been destroyed, and this means there is some residual scarring with loss of some of the sweat glands, sebaceous glands etc.

Full thickness burns are dry, black and charred or leathery or waxy and are often hard to touch. They extend through all skin layers into the subcutaneous tissues. They are completely insensate and if the burns are extensive, can often be mistaken for unburnt skin by their appearance.

Full thickness burns are not painful as the cutaneous nerve endings are destroyed (though in practice many burns are of mixed depth, so there may be associated areas of more superficial burns which are painful). As all the elements of the skin have been destroyed, full thickness burns heal very slowly from the edges with thick, inelastic scar tissue. They are normally excised and grafted in a burns unit so as to restore cover with functional skin.

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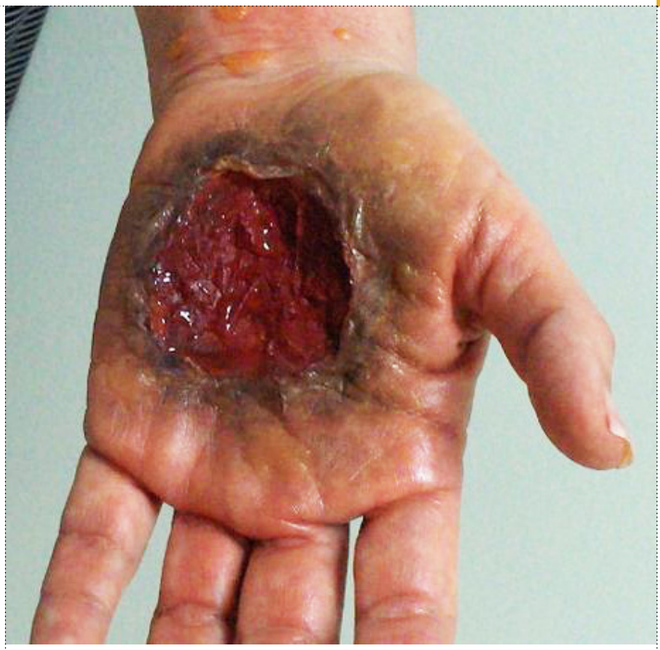

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A PENETRATING GARDEN CANE ILLUSTRATES PRINCIPLES OF WOUND CARE IN THE EMERGENCY DEPARTMENT

D Dhariwal, G George, N Jefferson, P Pretorius

A Penetrating Garden Cane Illustrates Principles Of Wound Care In The Emergency Department
Patient Management

Abstract

A seventy-nine year old female patient fell onto a garden cane which penetrated the right side of her face. The management, radiological investigation, anatomy of the injury and complications are presented as the basis for a Case Based Discussion.

Case History

We describe a seventy-nine year old, type II diabetic female patient, on warfarin for Factor V Leiden deficiency. She tripped on some rhubarb in her garden, and fell face first onto a projecting cane used to support her beans. The cane penetrated the right side of her face, overlying the right maxillary antrum. The stick broke off during the fall. Realising that hospital treatment was necessary, she made her way to her house and summoned help. She was brought to her local Emergency Department by ambulance. In the Emergency Department, it was immediately obvious that she had a circular, slightly bleeding wound lying over her right maxillary antrum. She had felt unwell since her injury. She complained of pain radiating to her right ear. She said her teeth "felt funny". She also complained of neck pain.

Examination

Her vital signs were normal, and her Glasgow Coma Score was 15/15. She had a circular wound overlying the right maxillary antrum. The cane was not seen protruding from the wound, nor was it palpable on examination, including examination from within the mouth. There was diminished sensation beside the nose in the territory of the right infra-orbital nerve. She had a global right sided facial weakness of lower motor neurone type.

Immediate Management

This patient's immediate management consisted of pain relief, and precautions against wound infection, namely local wound toilet – we were only able to clean the surface of this wound in the emergency department – anti-tetanus prophylaxis, and antibiotics.

Preliminary Investigations

Initially, an attempt was made to take X-rays of this lady's cervical spine and facial bones. However, she became acutely unwell when an attempt was made to position her on the X-ray table. She felt dizzy, and she developed amnesia for the accident. The decision was therefore made to abandon this investigation, and to proceed to an immediate CT scan of her head and cervical spine. Because the patient had given a history of a penetrating injury to the cheek, and there were signs of a lower motor neurone facial nerve palsy, the scan was extended to include the facial bones and soft tissues.

Wood cannot be seen on traditional X-rays, and it was extraordinarily difficult to see any evidence of the cane on the brain windowed source images because of the oblique trajectory of this low density cane relative to the plane of the scans (fig. 1a). However, we were able to see blood in the right maxillary antrum, indicative of local injury, and we were able to see fractures in the anterior and posterior walls of right maxillary antrum (fig. 1 a). Penetrating foreign bodies are often wedged tightly in the tissues, and we should have suspected that the cane had been retained. Indeed, a length of garden cane was clearly seen within the soft tissues of the right side of the face when the CT radiographer switched to the bone windows of the scan, where a narrow air filled cylinder is demonstrated (figs. 1b and 1c). The images confirm the cane fracturing and entering the anterior wall of the right maxillary antrum, traversing the lateral aspect of the antrum and then passing through the posterior wall of the antrum before traversing the right masticator space between the insertions of the medial and lateral pterygoid muscles where it lies 4mm medial to the ramus of the mandible (figs. 1b, 1c, and 1d).

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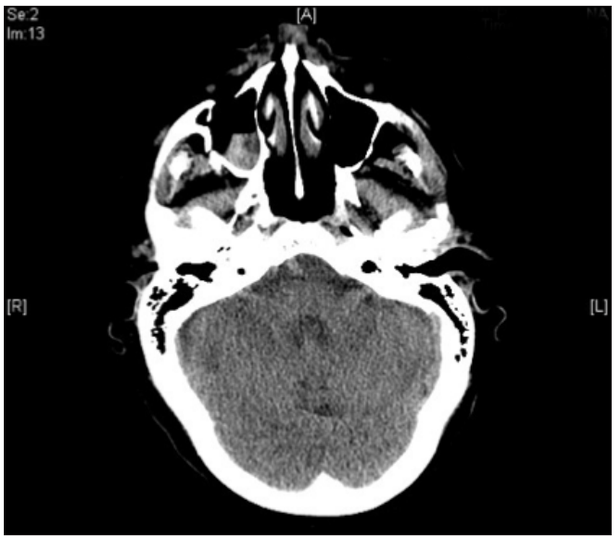

Fig 1 a: The brain windowed source image. Note the fractures in the anterior and posterior walls of the right maxillary antrum, and blood within the antrum.

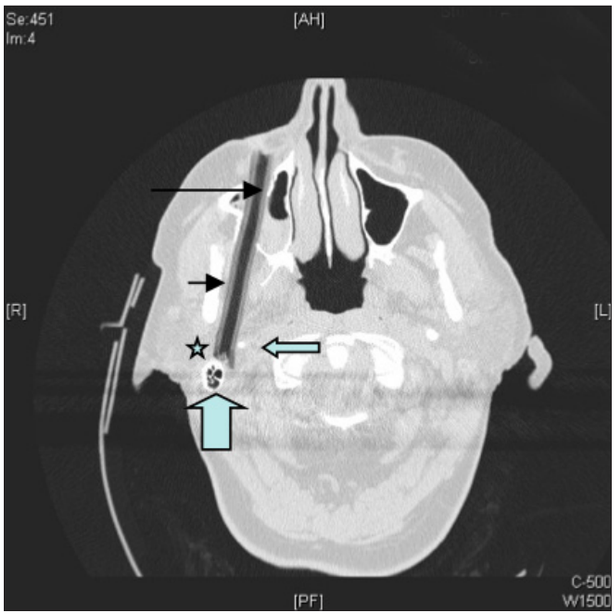

Fig 1 b: The location of the air-containing bamboo cane and its relationship to the maxillary antrum (long black arrow), mandibular ramus (short black arrow), mastoid tip (short block arrow), parotid (star), and styloid process (long block arrow) is well demonstrated on an oblique axial reconstruction of the unenhanced CT scan displayed on bone windows.

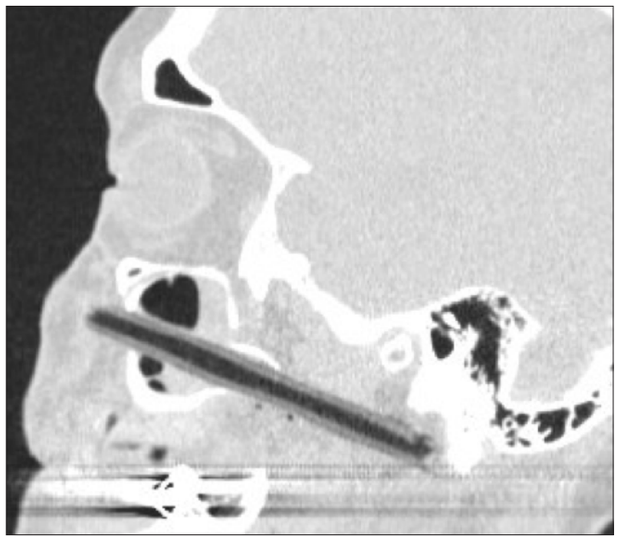

Fig 1 c: Oblique sagittal reconstruction of the unenhanced CT scan displayed on bone windows, again showing the location of the cane.

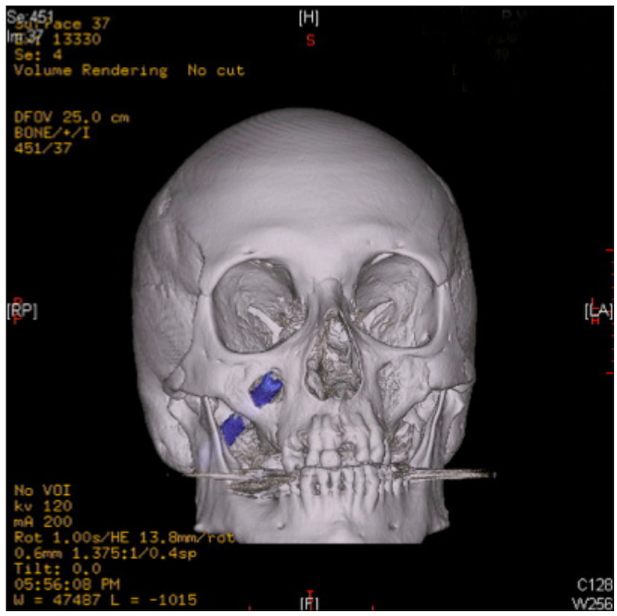

Fig 1 d: A surface shaded, 3-dimensional volume-rendered reconstruction of the CT scan demonstrates the location of the cane in a more intuitive way.

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Management Plan

The diagnosis of the cane within the face concluded the Emergency Department assessment, and we urgently referred this patient to the Oral and Maxillofacial Surgeons for definitive care, namely surgical exploration and debridement, and removal of the cane.

Case Based Discussion

Any patient with a significant wound requires an 'ABC' assessment. In this case, the patient had no airway or breathing problems, and she had no signs of circulatory shock. Therefore, she did not require any airway interventions or fluid resuscitation.

Her emergency department assessment clearly showed that she had a retained foreign body, and this had caused damage to deep structures in the face, namely her facial nerve, her maxillary antrum, infra-orbital nerve, and related structures. Therefore, surgical exploration by a specialist team was mandatory.

Before transferring her to Oral and Maxillofacial Surgery, we had to complete her initial treatment. For very painful injuries, intravenous paracetamol, or intravenous morphine titrated against pain, are suitable analgesics. Intravenous paracetamol is particularly suitable for frail, elderly patients, and also for patients who have had a head injury, because it does not cause respiratory depression or depress the conscious level. There is evidence that intravenous paracetamol is as potent as 10 mg intravenous morphine (1). Alternatively, morphine can be given intravenously and titrated against pain in small incremental doses until analgesia is achieved, demonstrated by a reduction in the pain score.

A Penetrating Garden Cane Illustrates Principles Of Wound Care In The Emergency Department
Patient Management

Any garden wound is considered to be 'dirty' because of the high risk of contamination with garden soil. This patient had a very high risk of infection for three reasons: firstly, she had a retained, potentially contaminated, wooden foreign body, and wood becomes almost always become severely infected if left; secondly the cane was traversing the maxillary antrum, resulting in compound fractures through an air sinus; thirdly, she was a type II diabetic patient. A good way of cleaning a dirty wound is to irrigate it with dilute povidone iodine in saline from a giving set, being careful to avoid splashing the eyes. (Unfortunately, in this patient, we were only able to clean the surface of the wound in the emergency department, and full toilet and debridement in theatres was therefore essential as soon as possible after the injury.)

The wound should be covered with a sterile dressing until the patient is taken to theatre. We also gave anti-tetanus prophylaxis. It is correct to give anti-tetanus hyper-immune gamma globulin, regardless of immunisation status, to any patient with a tetanus-prone wound, as in this case (2). In addition, if a patient has not completed a primary course of five immunisations, or in older patients where the longevity of prophylaxis might be in doubt, it is appropriate to give a booster dose of tetanus toxoid into the opposite arm to the gamma globulin. In addition to local wound-cleaning and anti-tetanus prophylaxis, this patient required prophylactic antibiotics because of the high risk of infection.

Not all foreign bodies are radio-opaque. Special techniques are needed to show wood in the tissues, usually ultrasound. In this case, CT on bone windows demonstrated the wooden cane because the windows were manipulated to maximise the range of radiodensities easily perceptible. This brought the air within the cylindrical cane into sharp contrast with the surrounding densities. The wood itself is much less obvious. It is worth noting that glass can be seen because it contains lead; stones and ceramics can be seen because they contain calcium; most metals can be seen, with the exception of aluminium because of its low density and low atomic number.

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The position of the cane within the face explained the neurological injuries. The infra-orbital nerve traverses the maxillary antrum, and damage to this nerve would explain the patch of numbness in infra-orbital nerve territory. Numbness in infra-orbital nerve territory is always suggestive of maxillary fracture after local trauma. The cane ended up abutting the skull base where it had crushed all branches of the facial nerve, resulting in a global facial nerve palsy of lower motor neuron type.

It is always important to follow outcomes for cases we have seen in the Emergency Department. In this case, the Oral and Maxillofacial Surgeons arranged a CT angiogram to better delineate the relationship of the cane to the vascular structures in the neck prior to surgical removal. This showed that the middle meningeal artery passes just superior to the cane as it comes off the main trunk of the external carotid artery. Distortion of branches of the external carotid artery between the ramus of the mandible and the lateral aspect of the cane was demonstrated, some of the branches lying within a millimeter of the lateral aspect of the cane. The cane is also seen to pass 10 mm lateral to the internal carotid artery, and its distal end passes through the deep lobe of the parotid gland before coming to rest against the mastoid process (fig. 2 a).

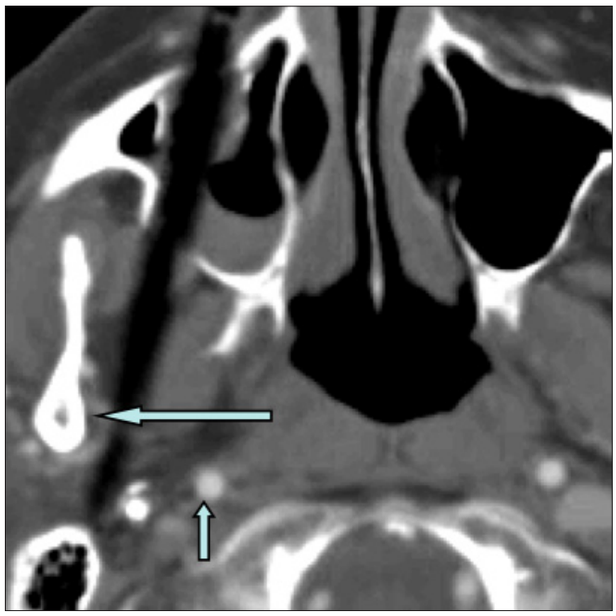

Fig 2 a: The proximity of the bamboo cane to the internal carotid artery (short arrow) and branches of the external carotid artery (long arrow) is well demonstrated on the oblique axial reconstruction of the CTA.

At operation, a right preauricular approach was performed to identify the facial nerve trunk at the stylomastoid foramen. The nerve was obscured by the deep aspect of the foreign body. A 9cm cylindrical fragment of wood (fig. 2 b) was removed from the cheek with direct vision of the deep aspect at the skull base. Once removed, the facial nerve was seen intact, and had been compressed by the wooden stick at the skull base (fig. 2 c). Facial nerve stimulation confirmed all branches were intact. The entry wound and preauricular approach were closed primarily after debridement.

Fig 2 b: The cane removed.

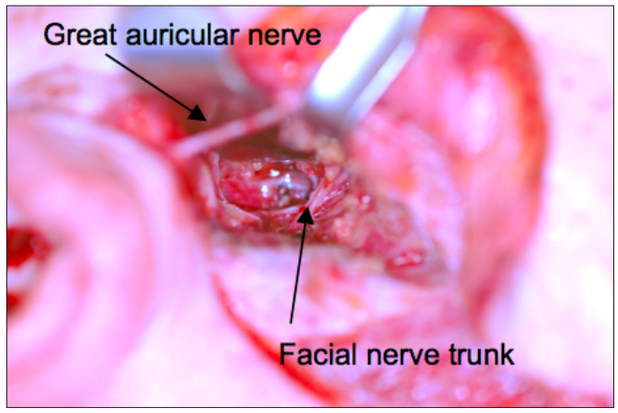

Fig 2 c: The facial nerve seen intact.

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There was no evidence of injury to the internal carotid artery and the cane was separated from the carotid sheath by the styloid process. Therefore, the period of amnesia and dizziness during positioning on the X-ray table cannot be explained on the basis of a direct cerebrovascular complication of the injury.

At three month post-operative review, there was good evidence of facial nerve recovery. This interesting case illustrates the difficulties associated with the identification of penetrating radiolucent foreign bodies (3), and it also demonstrates the risk to deep structures within the face. It was important to explain all the management decisions to the patient, allowing her time for questions and discussion.

Test Yourself Questions

1. Which of the following penetrating foreign bodies can be identified on X-ray?

a. A glass shard from a milk bottle
b. A broken tooth in a bite wound
c. A piece of denim clothing driven into a wound
d. A piece of blackthorn
e. A bullet from a gunshot wound

2. A shepherd is kicked by a sheep during shearing. This injury crushes the tissues at the level of the distal third of his shin and exposes the tibia. Which of the following statements are true?

a. This wound is tetanus-prone
b. This patient should receive anti-tetanus hyper-immune gamma globulin
c. This patient does not need any anti-tetanus prophylaxis if he has completed a course of five anti-tetanus immunisations
d. The anterior tibial nerve is at risk of injury
e. Pre-tibial wounds heal well

Answers

1. a, b, e.
Glass contains lead, a tooth contains calcium, and a bullet is made from lead: all of these are radio-opaque.

2. a, b, d.
Sheep stand on soil and faeces in the field, and therefore a kick wound will be contaminated. Furthermore, it will cause a blunt crush injury. Clostridium tetani lives in the gut of sheep, cattle and horses, and the spores survive in soil. Clostridium tetani is anaerobic, and flourishes in crushed devitalised tissue. Therefore, this is a tetanus-prone injury. Careful wound toilet and debridement are paramount, as is effective anti-tetanus prophylaxis. The anterior tibial nerve is superficial to the distal tibia and is at risk or injury. Pre-tibial wounds overlying bone heal very poorly.

Acknowledgements

We have written consent from our patient for publication, and we are grateful to her for allowing us to submit this interesting case. We should like to thank Bryan Todd for assistance in preparing the image file. All the authors had clinical responsibility for the patient, and they jointly wrote the manuscript. There are no competing interests.

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MANAGEMENT OF CHEST TRAUMA IN THE EMERGENCY DEPARTMENT: A CASE BASED DISCUSSION

C Ginnis, S Rymaruk, R Dalton

Management of Chest Trauma in the Emergency Department: A Case Based Discussion
Good Clinical Care

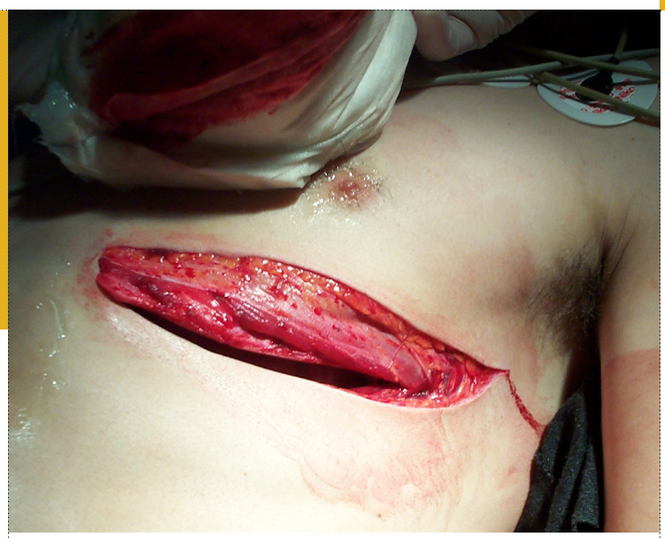
The Case

A 48 year old man was brought to the Emergency Department by air-ambulance after colliding into a cliff face whilst paragliding; he had then fallen a further 5 feet to the ground. The pre-alert call to the Emergency Department suggested he had sustained significant thoracic injuries.

On primary survey, his airway was self-maintained and cervical spine fully immobilised. High flow supplementary oxygen was being delivered via a non-rebreath mask and intravenous (IV) access had already been obtained by the paramedics.

Chest examination revealed marked reduction in chest expansion on the right side, with dullness to percussion and absent air entry also on the right side. Tracheal position was central. The patient was tachypnoeic with a respiratory rate of 20 and saturations of 94% on high flow oxygen - an arterial blood gas with FiO_2 of 0.85 showed pO_2 10.1kPa and pCO_2 4.2 kPa with a metabolic acidosis and lactate of 3.7. There was tenderness across the whole anterior chest, the right clavicle and the posterior right chest wall but there was no evidence of penetrating injury or flail segment. A right sided haemothorax was suspected, and whilst other members of the team continued the primary survey and fluid resuscitation, a 38 Fr chest drain was inserted at the 5th intercostal space in the mid axillary line. 1700ml of blood drained rapidly.

The patient was shocked; he had a blood pressure of 74/49 and a heart rate of 123. His capillary refill time was 4 seconds. Examination of the abdomen, pelvis and long bones were unremarkable. Further large bore IV access was secured and blood was sent for FBC, U&E, clotting screen, amylase and cross match for 6 units of blood (2 units of type specific blood and 4 units of fully cross matched). Fluid resuscitation was commenced immediately with a 500ml bolus of warmed Hartmanns solution and the patient was catheterised to monitor urine output. Reassessments were made after each intervention.



The patient had a GCS of 15, equally reactive pupils, normal peripheral neurology and normal blood glucose. His temperature was 35.9°C and following full exposure, no other injuries requiring immediate intervention were identified. Titrated IV morphine boluses were given to provide analgesia.

A portable chest x-ray done in the resuscitation room showed a right sided haemothorax and multiple rib fractures. His pelvic x-ray was normal and a FAST scan was negative for intra-peritoneal fluid.

The patient's heart rate and blood pressure had not improved following the initial fluid bolus. Following 2 litres of warmed crystalloid, the patient was given 2 units of type specific blood and 3 units of fresh frozen plasma all within one hour of admission. The timing of this was crucial as the rapid drainage of blood into the chest drain indicated that significant blood loss had occurred necessitating prompt replacement of blood products. 1 gram of intravenous transexamic acid was given as a bolus and a further gram was given over the next 8 hours. 90 minutes after arrival in the ED the patient's blood pressure was 105/60 and heart rate was 94. A repeat arterial blood gas showed a pO_2 of 19.8 kPa and pCO_2 of 4.0 kPa with a FiO_2 of 0.85, and the pH and lactate were within normal limits.

As the patient's condition continued to stabilise without on-going need for iv fluid or blood products, a CT scan of the head, neck, chest, abdomen and pelvis ('pan-scan') was performed. It showed normal intracranial and intra-abdominal appearances, comminuted right scapular and clavicle fractures, right sided 1st and 3rd – 9th rib fractures, bilateral pulmonary contusions (more extensive on the right side) and a small right sided pneumothorax (Figure 1). There was no evidence of disruption to the major vessels or heart.

Although the initial output from the chest drain was 1700ml of blood, over the next 2 hours only a further 110ml drained and the patient's blood pressure remained stable. The decision was made not to take the patient for emergency thoracotomy and the cardiothoracic team planned to transfer him to the cardiac intensive care unit for optimisation of his physiology and for close observation.

MANAGEMENT OF CHEST TRAUMA IN THE EMERGENCY DEPARTMENT: A CASE BASED DISCUSSION

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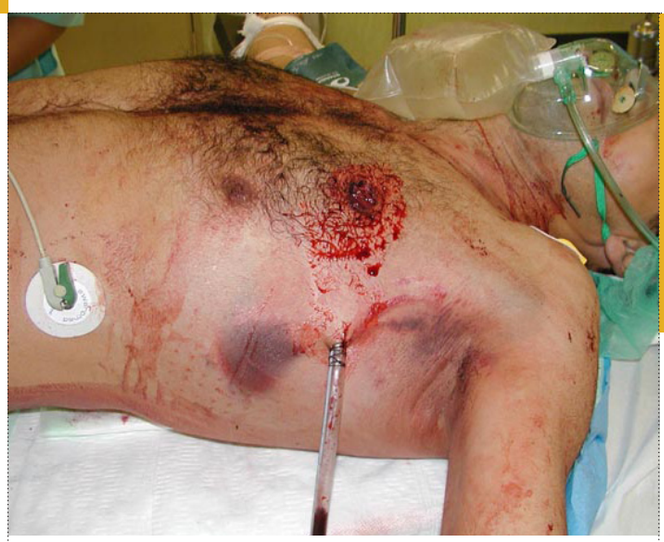

Over the subsequent 24 hours the patient required continuous positive airway pressure (CPAP) to meet his increasing oxygen demand. This was due to developing pulmonary contusions and acute respiratory distress syndrome (ARDS) and a chest x-ray taken at 2 days shows the progression in radiological features (Figure 2). Orthopaedic fixation of the clavicle fracture was delayed until the patient was well enough.

After the initial period of worsening hypoxia on the critical care unit, the patient's oxygenation gradually improved and his FiO_2 demand decreased. The patient was managed with CPAP for a further 5 days, and was discharged to the ward after 9 days. He made an uneventful recovery on the ward and went home 6 days later after a stay in hospital of 15 days in total.

At a clinic review 3 months later he was progressing well with near normal spirometry results. However, there was evidence of fracture malunion of ribs (4, 5 and 6) and the patient is currently considering surgical intervention for this.

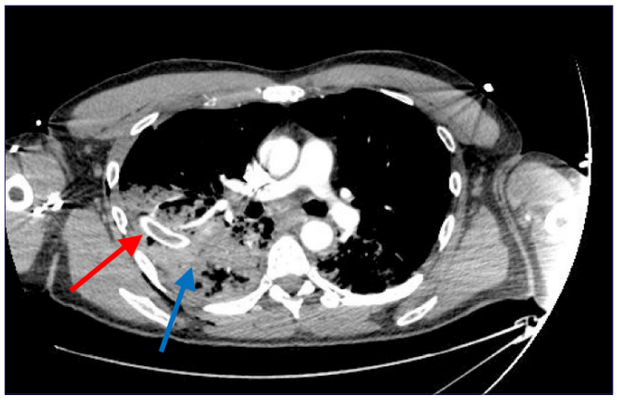

Figure 1: This axial slice of the thoracic CT scan performed shows evidence of extensive pulmonary contusion (blue arrow) within the right lung field. The chest drain is also visible (red arrow).

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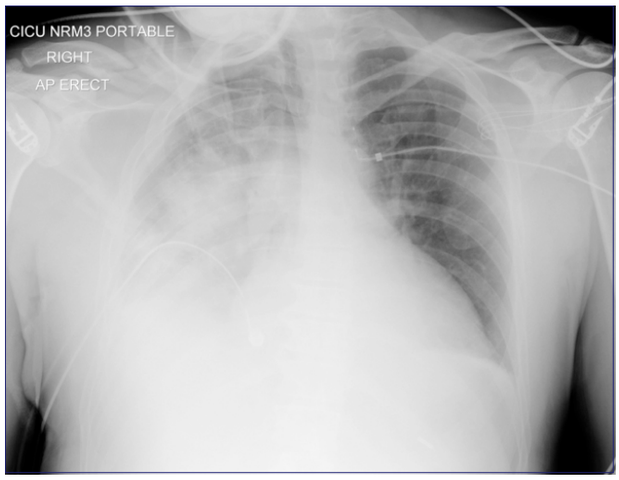

Figure 2: This chest radiograph shows extensive opacification in the right lung field consistent with a pulmonary contusion. There are multiple rib fractures and a right sided chest drain in situ.

Discussion

How do I identify significant chest trauma?

The first clue in identifying chest trauma comes from the history - it is always important to find out the mechanism of injury so that you can judge whether significant trauma is likely. A primary survey should be carried out on all patients with major trauma and life threatening chest injuries that should be identified at this stage are tension pneumothorax, open pneumothorax, massive haemothorax, cardiac tamponade and flail chest (see table 1). These all need immediate treatment by senior clinicians.

The patient in this case study had signs consistent with a massive haemothorax identified during the primary survey, as well as evidence of respiratory and cardiovascular instability. He was managed acutely with high flow oxygen, resuscitation with IV fluid and blood products, carefully titrated morphine and insertion of a right sided chest drain.

MANAGEMENT OF CHEST TRAUMA IN THE EMERGENCY DEPARTMENT: A CASE BASED DISCUSSION

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Condition	Examination findings on affected side	Treatment
Common to all injuries	External signs of injury to chest wall (although significant internal injuries can be present with few external signs) Look for evidence of: • ventilatory failure • cardiovascular instability	Immediate management with: • high flow oxygen • large bore IV access, fluid & blood products as required • analgesia – usually a titrated IV opiate acutely • intubation & ventilation if required
Massive haemothorax	Absent air entry Dullness to percussion Reduced chest expansion	Large bore chest drain insertion (see later)
Tension pneumothorax	Absent air entry Hyper-resonance Reduced chest expansion Trachea deviated away from the affected side	Immediate needle thoracocentesis 2nd intercostal space midclavicular line, followed by large bore chest drain insertion
Open pneumothorax	Open chest wound, 'sucking' Absent air entry Hyper-resonance Reduced chest expansion Trachea in central position	Flutter valve dressing / 3 sided dressing (allows air to exit but not enter the pleural cavity), followed by chest drain insertion
Flail chest & pulmonary contusion	Palpable chest wall trauma Paradoxical chest movement (some of the chest wall is indrawing on expiration and moving outwards on inspiration)	Good quality analgesia, consider thoracic / high lumbar epidurals and intercostal nerve blocks (by specialist)
Cardiac tamponade	Muffled heart sounds Raised JVP / distended neck veins Hypotension Usually penetrating trauma to the chest	Pericardiocentesis using echo guidance to drain blood from the pericardial sac

Table 1: Life threatening chest injuries.

When should I insert a chest drain?

If a traumatic haemothorax or pneumothorax is suspected on examination and the patient is compromised, insertion of a chest drain is indicated before obtaining a chest x-ray. A large bore chest drain (32-38Fr) is used to allow drainage of large volumes and to reduce the risk of drain occlusion. The drain is inserted using the open thoracostomy technique in the triangle of safety – between the borders of latissmus dorsi and pectoralis major at the level of the nipple (4th - 5th intercostal space) (1). Large volumes of blood in the pleural space can cause a tamponade effect on the bleeding source, and when the blood is drained the haemorrhage can actually increase. For this reason it is important to establish IV access before chest drain insertion so that fluid and blood product resuscitation can be administered.

In patients that do not have clinical evidence of life threatening chest injury it is usually reasonable to obtain a chest x-ray first to help guide the decision about chest drain insertion. It is important to note however that supine portable chest films will not show classical features of haemothorax or pneumothorax and should be interpreted in conjunction with clinical findings by the resuscitation team. The patient in this case study showed signs of cardiorespiratory compromise and had significant examination findings indicative of a right sided haemothorax: immediate insertion of a chest drain was indicated.

Does the patient need a thoracotomy?

Massive haemothorax is defined as immediate drainage of more than 1500mls of blood or more than one third of the patient's blood volume (2). It is more common in penetrating injury although it can happen in blunt trauma and is usually associated with cardiac damage or disruption of large systemic or hilar vessels – these patients, especially with penetrating injury, are highly likely to need a thoracotomy to identify and control the source of haemorrhage. Even in patients with less than 1500ml of initial drainage, continuing blood loss from the drain of >200ml/hour for 2 to 4 hours may indicate significant persistent haemorrhage. It is the physiological state of the patient however, rather than the measured amount of blood loss that will determine the need for thoracotomy and on-going need for blood product transfusion is a strong indication.

In the significant majority of blunt chest injuries however, where the bleeding is likely to be from intercostal vessels and lung parenchyma, the bleeding source often thromboses spontaneously and fewer than 10% of these patients require a thoracotomy to control the haemorrhage (3). In this case study the patient had a very large output from the chest drain initially (1700ml), but the subsequent drainage was low and the patient remained clinical stable: the patient was monitored closely in a critical care setting and did not require a thoracotomy in either the acute phase or later in his clinical course.

Should I arrange a CT scan?

A CT scan is the investigation of choice in the stable patient with significant mechanism of injury for chest trauma (4). The chest x-ray in these cases can be difficult to interpret and important pathology can easily be missed, especially on supine films where haemothorax and pneumothorax can be more difficult to identify. Even on an erect chest x-ray it takes 300-500 ml of blood to obscure the costophrenic angle so a haemothorax could still be missed (3). It is common for patients with a normal chest x-ray to have thoracic injuries seen on CT scanning. CT scanning in the multiply injured patient has been shown to identify pathology that is not yet clinically apparent and is indicated in all patients with multiple sites of injury.

Ultrasound scanning of the chest may be useful to help identify fluid or air the pleural space, however there is significant potential for misinterpretation and so CT scanning is favoured (5).

Which specialties should I involve?

The trauma team consists of emergency physicians, anaesthetists, general and orthopaedic surgeons, as well as nurses and radiographers. With chest trauma it is likely that specialist cardiothoracic input will be needed - in this case they were on-site but in many situations a patient may need transfer to a specialist centre if they are stable enough. It is easy to see that with so many specialties involved, the management of a trauma patient in the ED can become confused and fraught. This is where communication and team-working skills are essential, and the senior ED doctor will take the lead in coordinating the different aspects of the patient's care.

MANAGEMENT OF CHEST TRAUMA IN THE EMERGENCY DEPARTMENT: A CASE BASED DISCUSSION

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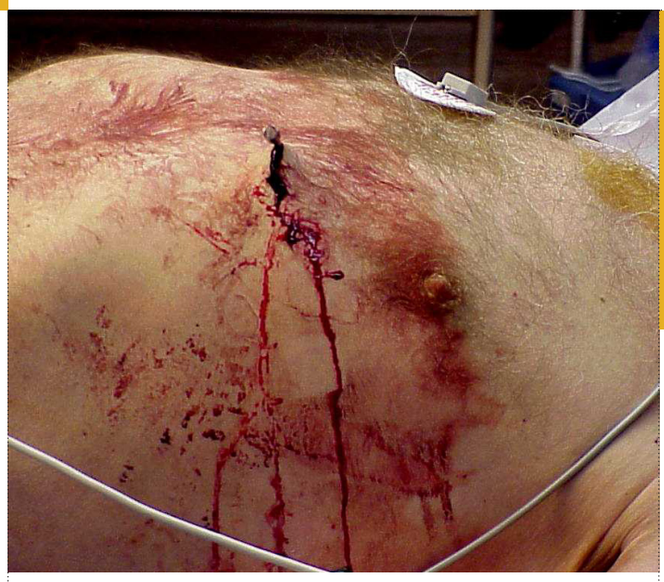

What happens when the patient leaves the ED?

Management on a critical care unit is essential for patients with severe injuries. As well as the direct consequences of injury to thoracic structures, patients with significant chest trauma are at risk of developing ARDS where mediator-driven inflammatory processes lead to impaired gas exchange and may induce multi-organ failure (6). The patient in this case study developed an increasing oxygen demand in the immediate days after his injury and was already in the appropriate critical care setting to identify and manage this problem; this highlights the importance of anticipating the potential for deterioration and need for early specialist input.

Questions

1. Which of the following is NOT a landmark used when inserting a chest drain?

(a) Pectoralis Major muscle
(b) Latissmus Dorsi muscle
(c) Serratus anterior muscle
(d) 4th intercostal space
(e) Mid-axillary line

2. All of the following can be found on examination of a patient with a tension pneumothorax EXCEPT?

(a) Paradoxical movement of the chest
(b) Respiratory distress
(c) Hyper-reasonance on percussion
(d) Tracheal shift away from the affected side
(e) Distended neck veins

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3. Which of the following in NOT a cause of a 'white out' on chest radiograph?

(a) ARDS
(b) Haemothorax
(c) Massive pleural effusion
(d) Pulmonary contusion
(e) Lobar pneumonia

4. Which of the following is NOT true of cardiac tamponade?

(a) It usually arises from penetrating thoracic injuries
(b) Beck's triad may occur
(c) Kussmaul's sign may be detected on examination
(d) Pericardiocentesis is the definitive management
(e) Cardiac tamponade and tension pneumothorax present with similar clinical signs

5. Which of the following is NOT true regarding traumatic cardiac arrest?

(a) It usually has a successful outcome
(b) Patients should have bilateral chest drains inserted
(c) Emergency thoracotomy may have a role in the management of some traumatic arrests
(d) The cause of arrest is usually hypoxia or hypovolaemia
(e) It is usually a result of a penetrating injury

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Answers

1. Answer C

When inserting a chest drain, the 'triangle of safety' should be used to prevent injury to neighbouring structures. The 'triangle of safety' is bound by the Latissmus Dorsi muscle (posteriorly), Pectoralis Major muscle (anteriorly), and the 4-5th intercostal space (inferiorly). The drain should be inserted just anterior to the mid-axillary line.

2. Answer A

All of the above are associated with a tension pneumothorax with the exception of paradoxical movement of the chest wall. Paradoxical movement of the chest suggests evidence of a flail chest due to two or more rib fractures in two or more places. This results in depression of the segment on inspiration due to a fall in intrathoracic pressure. This segment cannot contribute to ventilation and there is likely to be significant underlying pulmonary contusion.

3. Answer E

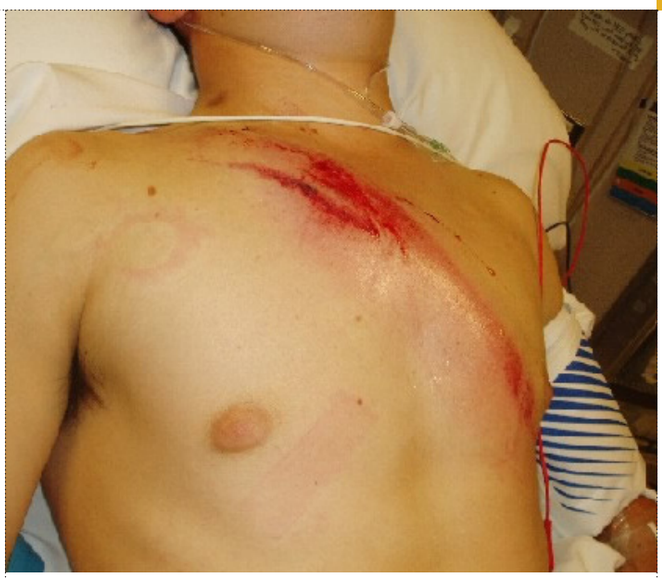
All of the above can cause complete opacification of the hemithorax with the exception of lobar pneumonia which will be confined to the affected segment of lung. Complete consolidation of a hemithorax may occur if there is extensive infection, which will also lead to a 'white out' appearance but not a simple lobar pneumonia. In ARDS, opacification tends to be bilateral.

4. Answer D

Cardiac tamponade is usually the result of a penetrating injury rather than blunt. It is classically described as being associated with Beck's triad of hypotension, distended neck veins and muffled heart sounds. The JVP may be seen to rise with inspiration (Kussmaul's sign) due to increased intrathoracic pressure. Pericardiocentesis is an emergency and potentially life-saving procedure in the Emergency Department, but surgical intervention is definitive. The signs of cardiac tamponade and tension pneumothorax both may present with shock and distended necks veins, since both impede venous return due to raised intrathoracic pressure.

5. Answer A

Traumatic cardiac arrest carries a poor prognosis with a survival rate of less than 1%. It is usually secondary to hypoxia and/or hypovolaemia. Chest drains should be inserted into both hemithoraces as part of the management of primary survey chest injuries such as haemothorax or tension pneumothorax. Emergency thoracotomy may have a role in the management of patients who have had a traumatic arrest such as those with penetrating injuries who have had a witnessed arrest in the Emergency Department, although it is rarely successful.



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MANAGEMENT OF HEAD INJURED PATIENTS WITHIN THE EMERGENCY DEPARTMENT

R Farquharson, L Brock

Management of head injured patients within the Emergency Department
Good Medical Pratice

Introduction

Patients with head injury form a large part of the workload within an Emergency Department (ED). Around 700,000 people present to EDs across England and Wales with head injuries each year. The majority of these, around 90% will be minor injuries and the overall death rate from head injury is low at around 0.2%. Some 40-50% of head injury attendances will be in children (1).

Head injuries can be classified in various ways. Classification using mechanism and severity are described below.

Mechanism	
- Blunt	High velocity (e.g. automobile collision) Low velocity (e.g. fall, assault)
- Penetrating	Gunshot wounds Other penetrating injuries
Severity	
- Minor	GCS 13-15
- Moderate	GCS 9-12
- Severe	GCS 3-8

Reference 3

Traditionally, the investigation and management of this substantial group of patients was at the discretion of individual departments and clinicians and as such varied significantly. More recently, guidelines for the management of head injured patients presenting to the ED have been developed by the National Institute for Clinical Excellence (NICE) and were published first in 2003 and then revised in 2007. These provide a reference for the use of imaging in this group of patients and for referral to secondary care.

Initial approach

The initial approach to all injured patients in an ED is the same. Firstly you need to ensure your own safety – personal protective equipment is mandatory. Then an assessment is made in a recognised A<C>BCDE approach (see table 1). This approach allows a rapid but thorough assessment of the patient to be made and can be applied to all patient groups.

A Airway with Cervical spine control and Control of any major haemorrhage
B Breathing
C Circulation
D Disability
E Exposure

Table 1: Initial approach to the injured patient.

If the patient has multiple injuries this approach leads to detection and management of injuries in a logical way with minimal opportunity to miss something crucial. In isolated head injured patients it decreases the risk of missing other associated and sometimes unexpected injuries. It also ensures reduction in secondary brain injury from hypoxia and hypovolaemia.

In an ambulant patient with an isolated head injury, the initial assessment may simply be a brief conversation establishing events and orientation, an assessment for cervical spine injury and a set of observations (pulse, blood pressure, respiratory rate, oxygen saturations, GCS and papillary responses).

History taking

It is important during history taking to establish a clear picture of the events which led to the injury taking place. This will allow you to make a judgement of the magnitude and direction of the forces involved and to use this information in your overall patient assessment. It will also allow you to consider other potential associated injuries that may have occurred in conjunction with the head injury.

Head injury may occur as a result of a collapse from an associated medical condition and so this needs to be considered when the history is being established.

Events following the head injury then need to be explored – presence and duration of loss of consciousness, vomiting, seizures, amnesia of events.

A look at current medications is essential. Certain mediations – particularly Warfarin – are known risk factors for bleeding following a head injury. Tetanus status needs to be ascertained in the presence of a scalp wound.

Finally is consideration of social circumstances. Any person being discharged home following a head injury needs to be going home with a responsible person who can continue their observation.

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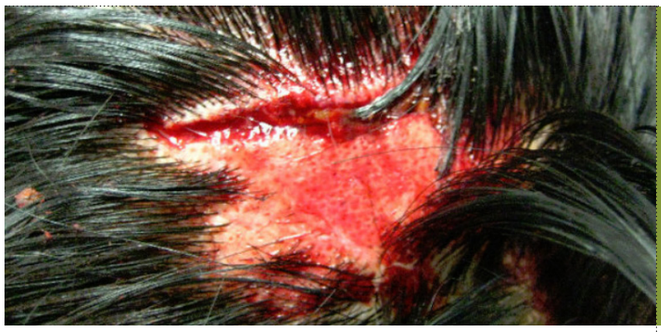

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Examination

Following an initial assessment, a more focused examination of the head injured patient needs to take place.

An assessment of Glasgow Coma Score using the Glasgow Coma Scale needs to be accurately made and clearly documented. This allows repeated calculations of the score and an assessment of progress to be made. See table 2.

Eyes:	
4	Open spontaneously
3	Open to verbal stimulus
2	Open to painful stimulus
1	No opening
Motor:	
6	Obey commands
5	Localises to a painful stimulus
4	Withdraws from a painful stimulus
3	Flexes in response to a painful stimulus
2	Extends in response to a painful stimulus
1	No response
Verbal:	
5	Normal, orientated conversation
4	Confused speech
3	Inappropriate words
2	Grunts. Incomprehensible sounds
1	No verbal response

Table 2: Calculation of Glasgow Coma Score.

A full neurological assessment then needs to be performed that includes cranial nerves with examination of the fundus, and the peripheral nervous system. Cranial nerves III, IV, V, VII and VIII are particularly affected by a basilar fracture. Dysfunction can present immediately or a few days after the initial injury.

Specifics to be sought and excluded include – any sign of a base of skull fracture – haemotypanum (blood behind the ear drum), Battle's sign (bruising behind the ears), 'panda eyes' (bilateral bruising to the eyes), lateral subconjunctival haemorrhage and evidence of leakage of CSF (cerebrospinal fluid) from the nose or ear. The basilar skull fracture may provide an entrance for bacteria, therefore antibiotics are often considered. In reality, most CSF leaks resolve spontaneously without complications within a week, thus prophylactic antibiotics are generally not required. If a patient with a known CSF leak presents with fever, the diagnosis of meningitis should be strongly suspected and managed appropriately.

An assessment of any wound needs to be made to determine any suggestion of an open or depressed skull fracture. Depressed skull fractures may be felt beneath a scalp laceration. The examination should be performed cautiously to avoid driving the bone fragment further into the cranial tissue. You should bear in mind that a depressed fragment may lie a few centimetres from a wound and overlying swelling may disguise the palpable depression.

Always perform a BM (bedside blood glucose) in any patient with GCS <15. The cervical spine should be assessed in all patients with a head injury. Immobilisation using a triple immobilisation system (collar, head supports and tape) should be employed in patients where there is concern – see table 3 (4). Immobilisation should be employed at the earliest opportunity when concern is raised, but can also be applied at any time during the patient journey should a concern become apparent. It should be noted that the cervical spine cannot be safely cleared in certain situations; those with distracting injuries, under the influence of alcohol or drugs or displaying a reduced GCS should be treated as having a C-spine injury until proven otherwise.

- GCS <15 on initial assessment
- Neck pain or tenderness
- Focal neurological deficit
- Paraesthesia in the extremities
- Clinical suspicion of cervical spine injury

Table 3: Risk factors for cervical spine injury requiring triple immobilistaion (4).

A check for occult, concurrent injuries should always be made. Haemodynamically unstable trauma patients should be assumed to have a bleeding source other than their head injury. A relatively small amount of intracranial bleeding can cause significant brain injury due to limited space inside the cranium, but is very unlikely to upset the haemodynamic status of a trauma patient. The relationship between pressure and volume within the skull vault is described by the Monro-Kellie hypothesis.

The Monro-Kellie hypothesis states that the cranial compartment is incompressible, and the volume inside the cranium is a fixed volume. The cranium and its constituents (blood, CSF, and brain tissue) create a state of volume equilibrium, such that any increase in volume of one of the cranial constituents must be compensated by a decrease in volume of another.

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The principal buffers for increased volumes include both CSF and, to a lesser extent, blood volume. These buffers respond to increases in volume of the remaining intracranial constituents. For example, an increase in lesion volume (e.g. epidural hematoma) will be compensated by the downward displacement of CSF and venous blood. These compensatory mechanisms are able to maintain a normal intracranial pressure (ICP) for any change in volume less than approximately 100–120 mL. Changes in volume over 120ml begin to increase ICP, subsequently decreasing cerebral perfusion pressure (CPP). The physiological response to a fall in CPP is to raise systemic blood pressure, dilating cerebral blood vessels, increasing cerebral blood flow and continuing the vicious cycle. As CPP continues to fall, brain ischaemia and infarction occur.

Suggestions that ICP is raised include complaints of headache, vomiting without nausea and visual disturbance. In such patients' pupillary dilatation, papilloedema and the Cushings triad (increased systolic BP, widened pulse pressure and bradycardia) are signs of increased ICP.

In infants and small children, the effects of ICP differ because their cranial sutures have not closed. In infants, the fontanelles bulge when ICP gets too high. Bradycardia is especially suggestive of a raised ICP in children.

Investigation and Imaging

Head trauma may include fractures, contusions, haematomas and diffuse injuries. Such injuries can be described by their morphology or type of intracranial lesion, as shown below.

Morphology	
- Skull fractures	
Vault	Linear vs. stellate Depressed vs. non depressed Open vs. closed
Basilar	With/without CSF leak With/without seventh nerve palsy
Intracranial Lesions	
- Focal	Epidural Subdural Intracerebral
- Diffuse	Concussion Multiple contusions Hypoxic/ischaemic injury

Reference 3

Skull fractures are local injuries caused by direct impact to the skull. They do not, necessarily represent underlying brain injury, however, the force required to fracture the skull is substantial, thus an underlying traumatic brain injury should be purposefully excluded.

- Linear fractures; *a single fracture that goes through the entire thickness of the skull. Significant injuries include those that cross the middle meningeal groove or major venous dural sinuses. A linear vault fracture substantially increases the risk of an intracranial injury.*

- Stellate fractures; *a fracture in which there are numerous fissures radiating from the central point of impact or injury throughout surrounding bone tissue.*

- Depressed fractues; *occur when traumatic impact drives the bone piece below the plane of the skull. The edges of the depressed portion of skull may become lodged underneath the adjacent intact bone and fail to rebound into their anatomical position. Penetration of tissue and laceration of the dura can occur as a result.*

- Open/compound fractures; *occur when a scalp laceration overlies a fracture. If the dura has been torn, direct communication between the laceration and cerebral surface is present.*

- Basilar fractures; *defined as linear fractures at the base of the skull. These fractures are the result of considerable force and underlying brain involvement must be ruled out.*

Intracranial lesions can be classified as diffuse or focal, although these two forms frequently co exist.

- Diffuse Brain Injuries; *pathological process where axons are stretched and twisted by shear and tensile biomechanical forces. Range from mild concussions, with normal CT scans, to severe hypoxic ischaemic injuries with irreversible structural and physiological damage.*

- Epidural Haematomas; *blood clots formed between the inner table of the skull and the dura. These lesions are relatively uncommon (0.5 – 1% of all patients with a TBI2), primarily a disease of the young and often result from a tear of the middle meningeal artery. Delay in clinical manifestation of an EDH may result from disruption of a major venous sinus.*

- Subdural Haematomas; *account for approximately 30% of severe brain injuries[3]. Often develop from the shearing of small surface or bridging blood vessels of the cerebral cortex, resulting in the formation of blood clots between the dura and the brain. These haematomas are common in patients with brain atrophy e.g elderly/ alcoholics, with the slow venous bleeding delaying the development of clinical signs. The haematoma compresses the underlying brain tissue for prolonged periods, subsequently causing significant tissue ischaemia and damage.*

- Intracerebral Haematomas; *formed deep within the brain tissue as the brain is propelled against irregular surfaces in the cranial vault. Resultant small haemorrhages coalesce to form ICH's or coalescent contusions, which, alongside cerebral oedema can produce substantial mass effects and precipitate a herniation syndrome.*

The mode of imaging employed in head injured patients is CT. Plain x-rays of the skull are very rarely indicated and largely this investigation should be considered inappropriate in this group of patients. The only times when plain skull x-ray should be employed is for a penetrating head injury or a child with a boggy swelling.

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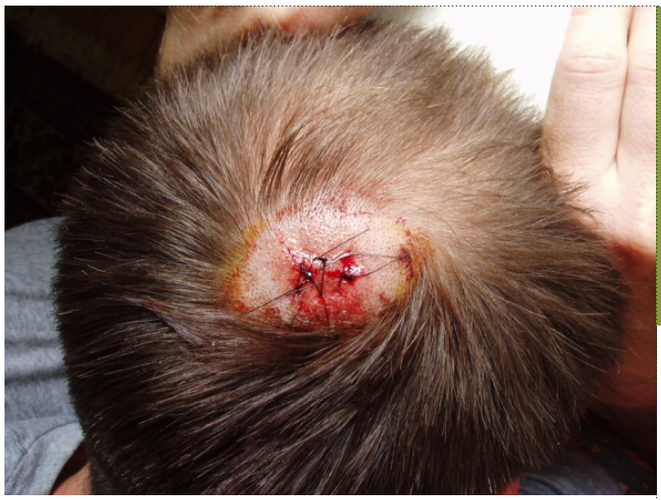

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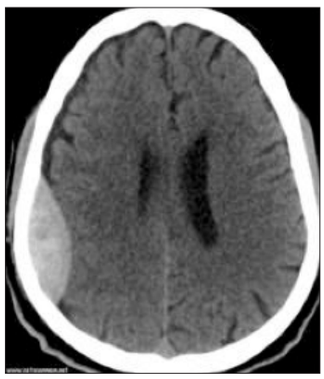
The indications for a CT head scan are listed in table 4.4.

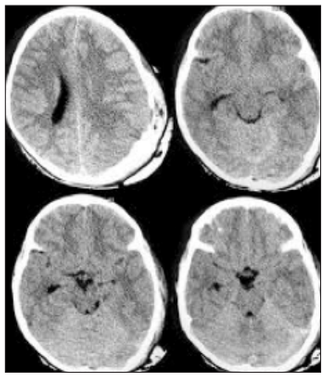
- GCS <13 on initial assessment in the ED
- GCS <15 at 2 hrs after injury on assessment in the ED
- Suspected open or depressed skull fracture
- Any signs of base of skull fracture (see examination)
- Post-traumatic seizure
- Focal neurological deficit
- >1 episode of vomiting
- Amnesia for events >30 minutes before impact
- Any loss of consciousness or amnesia with
  -Age >65
  -Coagulopathy (history of bleeding, clotting disorder, Warfarin treatment)
  -Dangerous mechanism of injury (pedestrian or cyclist struck by motor vehicle, occupant ejected from motor vehicle, fall from greater 1m or 5 stairs)

Table 4: Indications for CT head.

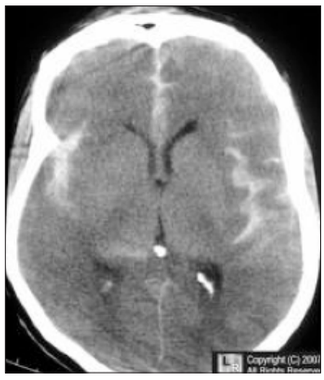
CT imaging shows characteristic patterns depending on the injuries sustained.



Typical biconvex/lenticular shape of an epidural haematoma as it pushes the dura away from the inner table of the skull.
medicinemcqsonline.com



Subdural haematoma; In contrast to an epidural haematoma, these injuries appear to conform to the contours of the brain. Subdural haematomas may grow to cover the entire surface of the hemisphere.
hawaii.edu



A well-defined, hyperdense, homogenous area of haemorrhage representing an intracerebral haematoma. These often become well demarcated over time.
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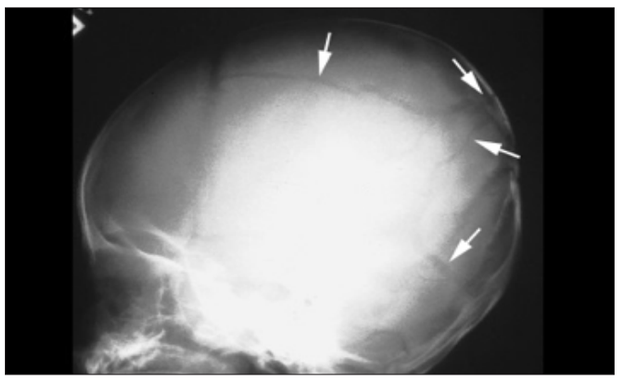
Children with head injuries

Specific factors from the history that allow a judgement to be made relating to the seriousness of the head injury, may not be obtainable directly from the child themselves and as such parents or witnesses to the injury may need to be consulted.

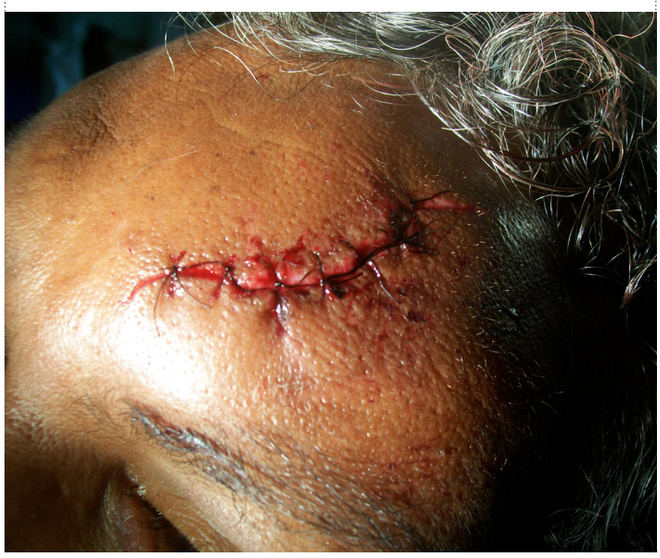
Until the cranial sutures close, children's skulls are more distendable than adults. Consequently, young children often sustain less TBI after head trauma than adults with comparable mechanisms of injury. However, there does appear to be a age-dependent brain vulnerability. Very young children have a higher rate of mortality than older children with the same severity of injury.

Another important factor to consider in children is the potential for non-accidental injury. In head injured children younger than 1 year of age, as many as 66% of all injuries and 95% of severe head injuries may be non-accidental (2).



An example of a linear skull fracture in a child. Injuries such as this could represent NAI.
http://www.childabuseconsulting.com/child-abuse-fractures.html

The criteria for CT scan imaging in children is listed in table 5 (4).



- **Loss of consciousness lasting >5 minutes (witnessed)**
- **Amnesia (anterograde or retrograde) lasting >5 minutes**
- **Abnormal drowsiness**
- **3 or more disctrete episodes of vomiting**
- **Clinical suspicion of non-accidental injury**
- **Post traumatic seizure with no history of epilepsy**
- **GCS <14 or for baby under 1 year GCS (paediatric) <15 on assessment in ED**
- **Any sign of base of skull fracture (see examination)**
- **Suspicion of open or depressed skull fracture or tense fontanelle**
- **Focal neurological deficit**
- **If <1year, presence of bruise, swelling or laceration >5cm on head**
- **Dangerous mechanism of injury (high speed road traffic accident either as pedestrian, cyclist or vehicle occupant; fall from height >3m; high speed injury from projectile or object)**

Table 5: Criteria for request of CT head in children.

Alcohol/ substance use

The ingestion of alcohol or other substances at any time prior or following a head injury can make assessment of the patient difficult. These substances may effect patient co-operation with history taking and examination. They may also have a direct effect on the patient's GCS.

If there is any concern relating to the head injured patient who is under the influence of a substance, it should not be assumed that the substance is the cause and the patient should be investigated and managed as having a significant head injury until proven otherwise.

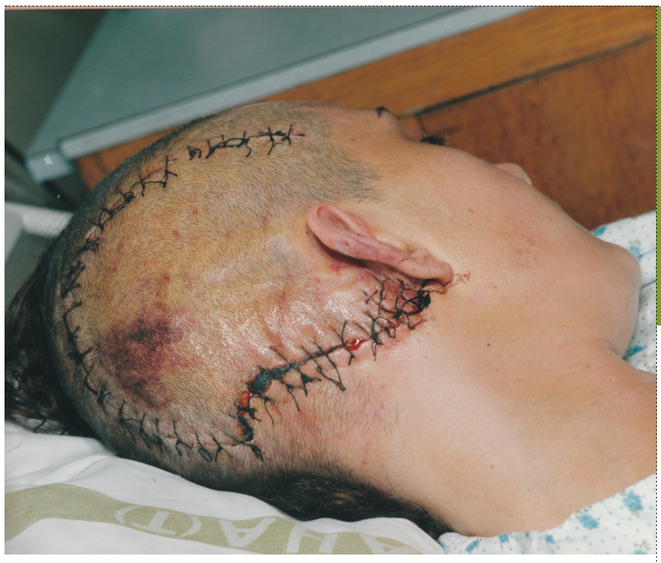
If there is any doubt surrounding these patients, they should be admitted to hospital for observation until a further examination of the patient without the effect of drugs or alcohol can be made.

The aggressive patient

Also of note is that in certain cases we are at risk from the patients directly – aggression often accompanies this group of patients. The source of this aggression may be varied and may relate to fear of the situation or be caused by other substances such as alcohol and drugs. But most importantly is the agitation and aggression which can be a symptom of brain injury itself and so it is vital that a full assessment is made in these difficult circumstances. Trained security personnel are available in the ED and can assist if required.

MANAGEMENT OF HEAD INJURED PATIENTS WITHIN THE EMERGENCY DEPARTMENT

R Farquharson, L Brock



Management of head injured patients within the Emergency Department
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Patients requiring admission

As a general rule, any patient who has sustained a head injury and about whom you have clinical concern – whatever that may be – should be admitted for a period of observation.

If there is any abnormality on CT scanning or if the head injury is significant eg GCS <8, the case should be discussed with the local neurosurgical unit and transfer of the patient considered for on-going management with or without active neurosurgical intervention.

All observation must include specific neurological observation – monitoring of GCS, pupils and limb movements to ensure that any deterioration is identified as early as possible and can be managed appropriately.

A guideline for criteria that suggest admission is necessary had been produced as part of the NICE guidance – see table 6 (2).

• Patients with new, clinically significant abnormalities on imaging
• Patients who have not returned to GCS 15 after imaging, regardless of imaging result
• When a patient fulfils the criteria for CT scanning but this cannot be done within the appropriate period, either because CT is not available or because the patient is not sufficiently cooperative to allow scanning
• Continuing worrying signs (eg persistent vomiting, severe headaches) of concern to the clinician
• Other sources of concern to the clinician (eg drug or alcohol intoxication, other injuries, shock, suspected non-accidental injury, meningism, cerebrospinal fluid leak)

Table 6: Criteria for admission

Discharge home

Any patient being discharged home following a head injury must be discharged to the care of a responsible person who can supervise them. Verbal and written advice should be provided that both the patient and their carer can understand. They should be advised of when to seek any further medical advice and how this can be achieved. Patients should also be advised of the risk of ongoing/delayed symptoms following a head injury and should be advised to seek community or primary care advice in this circumstance

Conclusion

Patients presenting to the ED with head injury are common in every age group and represent a significant workload for Emergency staff. It is vital that each patient is carefully considered and examined and that guidelines relating to head injury management are employed when investigation and management decisions are being made.

Head injured patients may be challenging, in terms of behaviour and additional substances being involved, but it is vital that a complete and accurate assessment is made. If the clinician feels that this cannot be done at the time of presentation, the patient should be admitted to hospital and observed until a full assessment can be made.

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LOCAL ANAESTHETIC TECHNIQUES: A PRACTICAL GUIDE

S Smith

Local Anaesthetic techniques: a practical guide
Good Medical Pratice

Abstract

Local anaesthetics are used extensively by practitioners in almost every field of medicine; however all therapeutic procedures have risks, and local anaesthetics are no exception.

This paper is a practical guide to the use of local anaesthetics, for analgesia when painful procedures are undertaken. It covers the basic sciences relevant to local anaesthetic use, and the common techniques in current use. The techniques commonly in use by Foundation Years doctors are described in detail, together with the necessary safety measures. There is also discussion on how to decide on which technique to use, and the necessary prerequisites of informed consent and management of adverse reactions.

Local anaesthetics: an introduction

Local anaesthetics (LA) are commonly used in the emergency setting. However, as with all therapeutic measures, they are not without risk. This article is design to review the basic science and principles of LA use, before describing the use of LA.

When using LA, the following should be considered. Firstly, what is the best method of achieving local anaesthesia, and secondly, how can the risks to the patient be reduced or mitigated.

Local anaesthesia can be achieved through a variety of methods: topically, local infiltration, or regional/ nerve blocks. These, however, should not be considered as absolute and exclusive of other methods of pain relief. The combinations of analgesia with anaesthesia will go a long way to help with the persistent issue of under-recognition and under-treatment of pain (1, 2).

It is also important to consider adjuvant techniques (especially in paediatric patients), such as distraction and anxiolysis (through psychological or chemical means), or supplementary analgesia.

Mechanism of action: what you need to know

Local anaesthesia (LA) act through a reversible, receptor mediated blocking of the axonal sodium channels, preventing depolarisation of the cutaneous pain fibres. The lipid soluble molecule passes across the axonal membrane (non-ionised), and in its ionised form blocks sodium channels. Therefore, tissue pH affects ionised to non-ionised ratio, and this in turn affects speed of penetration across axon membrane (and speed of onset).

Higher lipid solubility increases potency, and binding to sodium channels affects duration of action. LAs are more able to cross un/ lightly-myelinated nerves, and hence are more effective at blocking pain sensation than motor function. Most LA are vasodilators, which tends to reduce duration of action, hence the addition of adrenaline to some preparations. The combination of altered tissue pH and increase in vasodilation in infection explains the relative ineffectiveness of LA in infected tissues (such as abscesses) (3).

The chemical structure is of an aromatic and amine group linked by either an ester bond (e.g. procaine, tetracaine), or amide chain (e.g. lidocaine, bupivicaine). Amides are metabolised by the liver, esters by plasma cholinesterase.

What patients need to know: keeping the patient informed

For patient consent to be valid, it is important to ensure that the patient is informed of the risks of the procedure (see table 1 and below), and how you intent to mitigate these risks (4). The patient also needs to be aware of the therapeutic alternatives and adjuncts, and the rationale for advocating the particular LA technique the clinician is selecting. The clinician performing the technique should obtain the consent (preferably written consent for the more invasive blocks), after discussion with the patient.

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Risk	Clinical Features	Prevention	Treatment	Notes
Pain of injection		Use fine bore needle, inject slowly, warm LA to ambient temperature, buffer LA (1:10 ratio with 8.4% bicarbonate)		Subdermal injection less painful than intradermal. Injection through wound rather than through skin.
LA systemic toxicity	Vaughn-Williams Class 1 action Neurological toxicity includes peri-orbital tingling, drowsiness, abnormal sensation, lightheadedness, twitches, proceeding to fits, coma	Do not exceed safe maximum doses Care when injecting into vascular areas (e.g. intercostals blocks), aspirate before injection,	Antidote is Intralipid® Use benzodiazepines for seizures Avoid lidocaine for dysrhythmias, and phenytoin for fits (both have class 1 action)	
Failure of anaesthesia		Use longer acting LA for longer procedure Test sensation before procedure Ensure adequate time for onset of LA before procedure		
Allergic reaction	Variable	Always ask an allergy history Allergy to LA specific to group (ester or amide)	See anaphylaxis algorithm of Resuscitation Council [13]	True allergy to amide group uncommon [5], allergic reaction is often to paraben additives
Damage to local structures	Variable	Do not use adrenaline containing LA on structures with no collateral circulation (e.g. digits, penis, nose) Use nerve stimulator / ultrasound guidance [14] Check (and document) neurovascular status before and after injection		Sudden increase in pain when nerve directly in contact with needle

Table 1: Risks of LA.

The patent also needs to be aware of the practical elements the individual technique will involve (such as how many injections (and where these will be), approximate onset and offset times and what this will entail (e.g. what will the patient be unable to do, and for how long). It is important to inform the patient that the block may result in absence of pain sensation, while leaving other sensory modalities intact (due to the smaller diameter of the pain fibres), so the patient may feel what you are doing, but not any pain.

Local Anaesthetic techniques: a practical guide
Good Medical Pratice

Keeping the patient safe: side effects and toxicity of LA

The risks of LA use are detailed in table 1. The most clinically important issues to remember are those of allergy (uncommon(5)), and systemic toxicity. Systemic toxicity is often mis-diagnosed as hyperventilation or anxiety, due to the nature of the symptoms: never assume symptoms after LA use are benign (6). A standard management protocol for LA toxicity exists (5).

It is important to calculate the dose being given, and to ensure it is within the safe maximum dose. To calculate dose the easiest formula is: Dose in milligrams = volume of LA x percentage solution x 10. Safe maximum does are lidocaine 3mg/kg (plain), 7mg/kg (with adrenaline); bupivicaine 2mg/kg (8). These doses are not additive: a maximum dose of lidocaine cannot be given at the same time as another LA. When combining LA, therefore, the maximum safe doses need to be proportionately reduced (e.g. in a 50:50 mixture of lidocaine/bupivicaine use 1.5mg/kg and 1mg/kg as maximum safe doses). There is also a need for consideration of the volume required for the individual block (see table 2).

Name of block	Indication	Anatomical localisation	Volume of LA injected	Risks	Notes
Upper limb blocks					
Digital nerve block Often termed 'ring block' (incorrectly)	Finger/ toe blocks, distal to MCPJ/MTPJ.	Dorsal approach is less painful. Aim for digital nerve in web space, angle to midline until needle palpable at palmar surface. Dorsal s.c.† injection in band across MCPJ.	c.1 ml laterally, 1 ml dorsally.	Torniquet effect of injections. Aspirate before injection. Use 23G needle.	Digital nerve runs at level of dorsal end of IPJ creases/ at skin colour change.
Intrathecal block	Finger/ toe blocks, including MCPJ/MTPJ.	Either distal palmar, or proximal digital crease, in midline.	23G needle, down to bone, inject while withdrawing. Sudden decrease in pressure signals in tendon sheath, inject 2mls.		Only 1 injection, but less effective for little finger and thumb. Distal digital pressure increases efficacy.
Metacarpal block	Finger/ toe blocks, including MCPJ/MTPJ.	Distal palmar crease, palmar aspect, either side flexor tendons (2 injections for one digit)	3-4 mls.	Dorsal approach less painful, but deeper injection, and higher risk local damage.	Longer onset time than digital nerve block.
Blocks at the wrist	To block hand for minor surgery/ wound care				Remember nerve sensory distributions.
Radial nerve block		Infiltrate s.c. from FCR[1] to radio-ulnar joint.	5mls.	23G needle. Higher risk i.v. injection.	
Median Nerve block		At proximal wrist crease, between PL and FCR. 1cm depth.	Max. 5mls. Inject 2 mls as withdrawing to block palmar cutaneous branch.	Risk of nerve damage; see notes in text. 23G needle.	
Ulnar nerve Block		At proximal wrist crease, between ulnar artery and FCU. 1cm depth.	Max. 5mls. Inject 3mls s.c. from FCU to ulnar border.	Risk of nerve damage; see notes in text. High risk intravascular injection. 23G needle.	

Table 2: Commonly used nerve blocks.

LOCAL ANAESTHETIC TECHNIQUES: A PRACTICAL GUIDE

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Name of block	Indication	Anatomical localisation	Volume of LA injected	Risks	Notes
Lower limb blocks					
Femoral nerve block	Usually for analgesia in fracture neck of femur, shaft of femur fracture	3.5cm depth, 1cm lateral to artery, just below inguinal ligament.	10mls (use 40mls to achieve triple nerve block)	Risk of nerve damage; see notes in text. High risk intravascular injection. 23G needle.	May mask compartment syndrome. Digital pressure distal to injection to ensure triple nerve block.
FIB block	Usually for analgesia in fracture neck of femur, shaft of femur fracture	1cm below junction outer 1/3 and inner 2/3 inguinal ligament. Depth judged by second of two 'pops' as pass through fascia (lata then iliaca)		Used blunt (filling) needle to pierce fascia.	Inguinal ligament is on the line joining anterior superior iliac spine and pubic tubercle. Digital pressure distal to injection to ensure triple nerve block.
Ankle blocks	For procedures on the foot, such as wound repair, foreign body localization and removal				Sensory distribution variable, therefore often need to use multiple blocks. Total of five nerves to be block to anaesthetize foot.
Posterior tibial (PT) n.		Patient prone Between tendo-achilles and upper border of medial malleolus. Lateral to PT artery.	5-10 mls, depth: almost touching tibia.	23G needle	Blocks most of sole of foot.
Sural n.		Patient prone Between tendo-achilles and lateral malleolus, 1cm above tip.	Aim for s.c. injection.	23g needle.	Anaesthetizes heel of foot.
Saphenous n.		Supine patient. S.c. injection from medial malleolus (1cm above tip) to extensor hallucis longus (EHL)	Additional deeper injection at medial side EHL.	23G needle	Medial aspect ankle.
Deep peroneal n.		Midpoint anteriorly between malleoli, maximum 1cm depth	c.4ml.		Blocks between 1st/2nd toes. Largely motor nerve, lies between EHL and tibialis anterior tendons.
Superficial peroneal n.		Supine patient. S.c. injection from lateral malleolus (1cm above tip) to anterior tibia	5-10ml.		Blocks volar aspect foot.
Blocks of the head/face					
Forehead blocks	Block of forehead and scalp	s.c. injection from midline, immediately above eyebrow, to lateral aspect eyebrow.	5-10mls	Danger of intra-orbital injection.	Suprorbital nerve leaves through notch supraorbital crease (1/3 way along from medial end eyebrow). Supratrochlear nerve exits medial/superior orbit
Ear blocks	Procedures of the ear (e.g. wound care, removal of piercing)	A: 1cm below earlobe from SCM to angle mandible B: s.c. infiltration behind ear C: infiltrate anterior to meatus.		High risk of intravascular injection.	A: greater auricular nerve B: lesser occipital nerve C: auriculotemporal nerve
Dental blocks	Infraorbital nerve.	Buccogingival fold between 1st-2nd premolar, aiming towards infra-orbital foramen	1-2mls.	High risk of intravascular injection. Use dental syringes and adrenaline.	Blocks cheek and upper lip. Mental n. supplies lower lip, similar technique on mandible.
Scalp blocks	Greater occipital n.	At level of external occipital protuberance, infiltrate s.c. between trapezii.			Combine with forehead and auriculTemporal n. blocks to anaesthetize entire scalp

Table 2: Commonly used nerve blocks.

† IPJ: interphalangeal joints. MCPJ: metacarpophalageal joints. s.c: subcutaneous. i.v.: intravenous

‡ FCR: flexor carpi radialis FCU: flexor carpi ulnaris PL: Palmaris longus SCM: sternocleidomastoid

The use of adrenaline increases the onset and offset time, but also may increase infection rate, and preferably avoided except in situations where vasoconstriction is required (e.g. bleeding scalp lacerations).

From a pragmatic perspective, the most important issues are pain of injection and failure to achieve adequate anaesthesia. It is important to remember to inform the patient that LA may not remove all sensation, but should ablate pain.

Lastly, it is important to remember that administering LA is an invasive procedure and therefore adherence to infection control measures is important: this should be a sterile procedure.

Selection of technique

The techniques at your disposal are:

1: Local infiltration. *The main problem is the need for multiple injections, and the tissue distension (which is painful, and affects the wound edges, possibly reducing cosmetic outcome by making accurate opposition difficult). Haematoma block and intrapleural blocks are variations on this technique.*

2: Topical application. *Commonly used for corneal anaesthesia (the eye will need to be patched until the return of the corneal reflex). This is a useful technique for painful procedures in children (e.g. EMLA for venepuncture/ cannulation, LAT gel for wound closure).*

3: Field block. *A zone of anaesthetised tissue is obtained by injecting anaesthesia around the operative field. This is a technique could be employed for abscess incision and drainage (see caveats above), or for removal of foreign body.*

4: Nerve block: *Injection of LA around a nerve to block the sensory distribution of the nerve(s). This technique has a higher risk of damage to local structures, especially the neuro-vascular bundle (see table 1). A nerve stimulator or ultrasound guidance is advised for the larger blocks to reduce this risk, but is an expensive resource requiring expertise (nerve stimulators can be painful if used incorrectly/ poorly). Clinical identification of direct contact/ injection into nerve is through a sudden increase in pain.*

5: Intravenous regional anaesthesia (Bier's block). *This involves the injection of local anaesthetic intravenously, in a limb which has a tourniquet applied (pneumatic cuff) at above systolic blood pressure. The use of a double cuff enables inflation of the lower cuff over anaesthetised tissue, and deflation of upper cuff after anaesthesia achieved (as the cuff is often painful).*

6: Central neural blockade (will not be discussed in this article).

'How to choose' and 'how to do' guide to some commonly used techniques. A great deal of debate surrounds the selection technique.

The techniques at your disposal are for distal radial fracture manipulation there is controversy over the use of both Bier's and haematoma blocks (9), the two commonly used techniques; there is currently a trial comparing triple nerve block with fascia iliaca block in fractured neck of femur patients (10). The common indications are listed in table 2.

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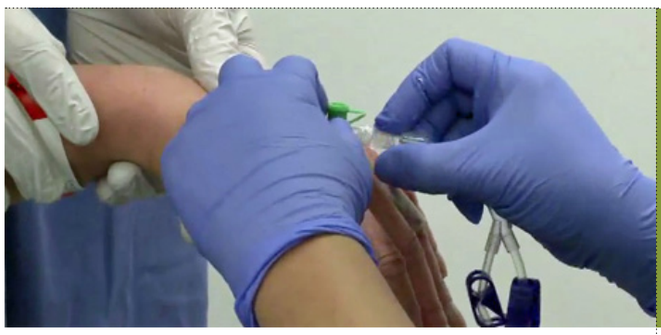
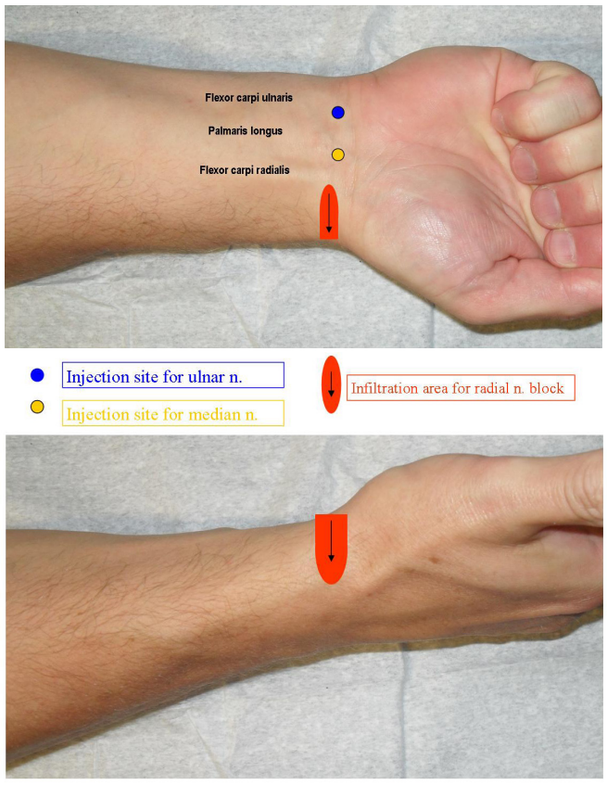
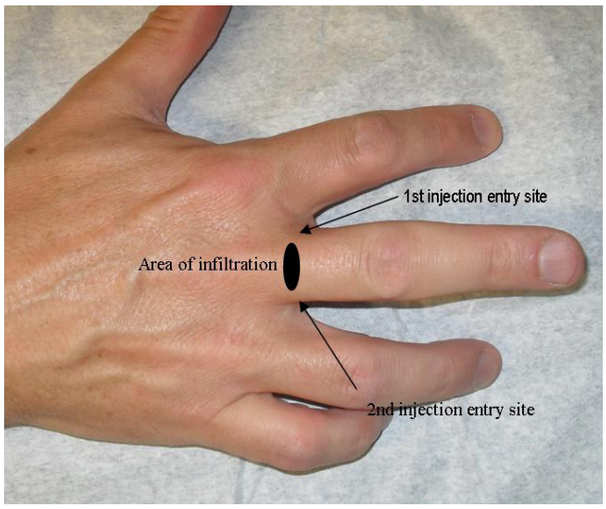


It is also important to emphasise that the techniques of particular nerve blocks is also sometimes a matter of debate, as a number of approaches to the nerve to be blocked may exist (for example, volar and dorsal approaches for digital blocks), and the descriptions in this text represent the authors' preference. The selection of both technique and approach should be based on a combination of patient preference and operator skill (and familiarity) with the technique together with a decision on appropriateness of technique for achieving anaesthesia.

The commonly used blocks are described in table 2, and some are illustrated in pictures 1 and 2. Haematoma blocks and field blocks are illustrated in picture 3.

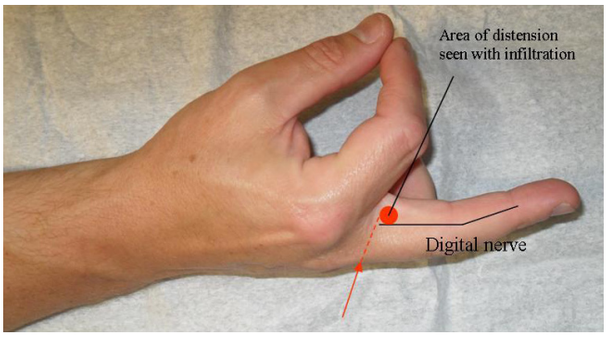


Pitcure 1: Blocks At The Wrist



Direction of needle

Path of needle



Pitcure 2: Digital Nerve Block

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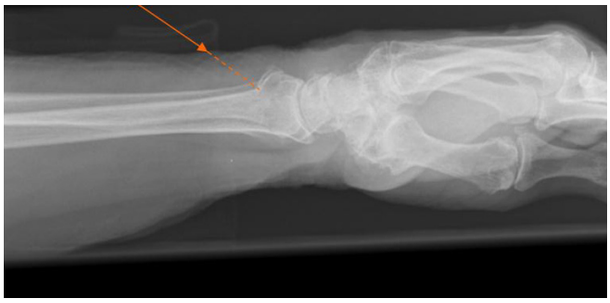
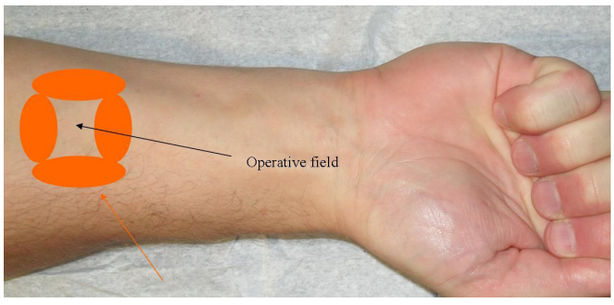
Haematoma Block



Direction and path of injection. With haematoma blocks, aspiration of blood is evidence of correct placement (is contrast to nerve blocks). 10mls sufficient for Colles' facture, volume depends of fracture site.

Field Block



Areas of subcutaneous infiltration.

Picture 3: Haematoma & Field Blocks

The main contraindications to the use of these techniques are patients who are unable to cooperate with the anaesthesia and procedure (e.g. for regional blocks: uncooperative patients, children) and allergy to LA. Patients who are unable to report side effects (e.g. pain of nerve damage, signs of toxicity) should also not receive regional anaesthesia (11). Additional contraindications include infection at injection site, coagulopathy, and (obviously) patient refusal. If anatomical landmarks are difficult to identify e.g. due to injury or previous surgery then local infiltration or topical techniques should be considered.

These techniques should only be performed when the operator has demonstrated competency. All the techniques should be performed with consideration for infection control standards: generally these should be considered as sterile procedures (12).

Summary

This article has discussed the commonly used local anaesthetic drugs, and described their use to achieve anaesthesia for emergency procedures. The important points that a practitioner must remember are to ensure patient gives informed consent; that the practitioner is aware of the risks of LA and regional anaesthesia, and knows management of potential adverse events.

Best of 5 self-assessment questions

1. You are injecting local anaesthetic into the forehead of a patient with a laceration. A few minutes after starting the supraorbital block, the patient complains of feeling faint, and of swelling and itchiness around the lips. The patient is known to have C1 esterase inhibitor deficiency. What is your management (after you have stopped procedure, checked observations and put into Trendelenburg position, and moved patient to a monitored bed)?

A: Continue to observe, this is probably a vasovagal episode
B: Give 1.5mg/kg Intralipid® intravenously to reverse toxic effects of lidocaine
C: Give 0.5mg adrenaline intramuscularly, as this is an allergic reaction
D: Give chlorpheniramine 10mg intravenously and hydrocortisone 200mg intravenously
E: Give bolus of 500 IU of C1 esterase inhibitor

2. You are preparing the local anaesthetic injection for a triple nerve block, for a patient with a fractured neck of femur. You elect to use a 50/50 mixture of lidocaine 1% and bupivicaine 0.25%, with a total volume of 30mls. How much of each drug does this equate to? The patient weighs 90kg.

A: 200mg lidocaine, 50mg bupivicaine
B: 150mg lidocaine, 37.5mg bupivicaine
C: 1500mg lidocaine, 25mg bupivicaine
D: 200mg lidocaine, 12.5mg bupivicaine
E: 400mg lidocaine, 125mg bupivicaine

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Answers

1. Answer: C

Although true allergy to local anaesthetics (LA) is often said to be rare, allergies do occur, to both classes of LA, and to the additives. Exacerbation of C1 esterase inhibitor deficiency does not give pruritic rash. Toxicity to LA does not cause angio-oedema, the symptoms relate to neurological and cardiac toxicity. Antihistamines and steroids are commonly given in allergic reactions, but the first line treatment is i.m. adrenaline

2. Answer: B

Local anaesthetic doses are easiest calculated by using the formula: VOLUME multiplied by PERCENTAGE multiplied by 10 equals DOSE IN MILLIGRAMS. It is important to remember the safe maximum doses, and that you cannot add the maximum dose of different LAs, as toxicity will be more likely.

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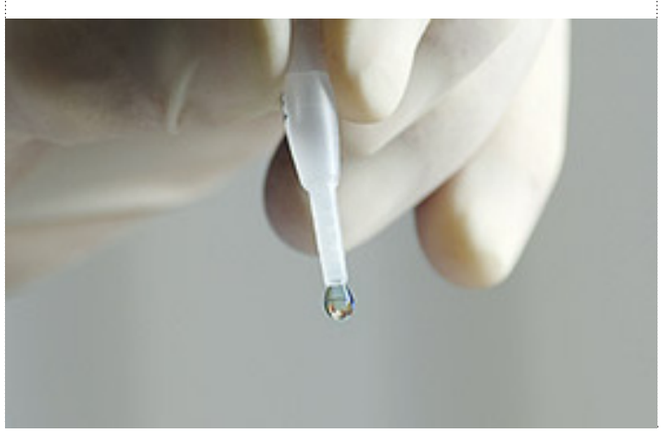
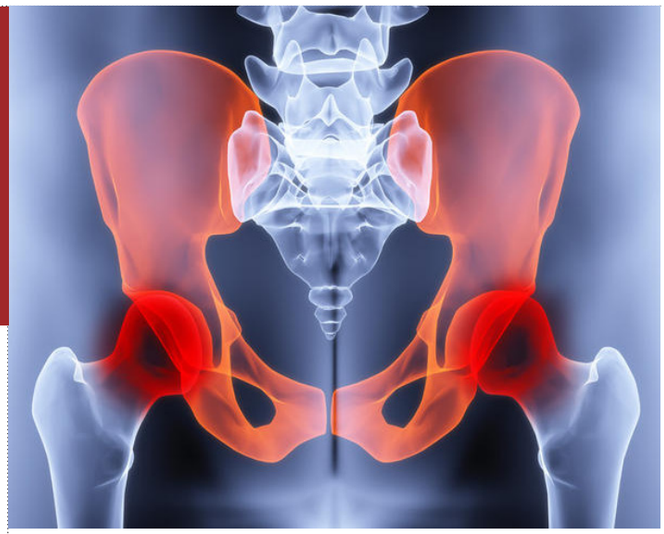


MANAGING PELVIC FRACTURES

N Frew, A Acornley



Managing Pelvic Fractures
Patient Management

Abstract

Following a high speed road traffic accident a patient is airlifted to hospital where the trauma team are assembled. The case is used to illustrate the management of high energy pelvic injuries, and the management of major haemorrhage in this context. Low energy pelvic injures are not considered.

Case History

A 55 year-old man was involved in a high speed frontal collision with another vehicle (Figure 1). The combined closing speed was estimated at over 60mph. The other driver was dead at the scene. The patient was trapped by the steering column and was extracted from the vehicle by fire crew and taken by air ambulance to the nearest Level 1 trauma centre where a trauma team comprising senior A&E, surgical, orthopaedic and anaesthetic staff were waiting.

Figure 1: The accident scene.

The paramedics report there was no loss of consciousness and no blood at the scene. On arrival the patient's vital signs were:

- Heart rate 130 beats/min
- Respiratory rate 24/min
- Blood pressure 90/50 mmHg
- Oxygen saturation 99%, on 10L oxygen
- GCS 14/15.

The patient appears drowsy and complains of pain in his lower abdomen and right hip.

Initial Management

This patient has been involved in high energy trauma and may have sustained multiple injuries. He is assessed and treated using an Advanced Trauma Life Support (ATLS) approach (1).

Primary survey

The patient is talking, maintaining his own airway and there does not appear to be any respiratory compromise. Care was taken to immobilise the cervical spine. Chest examination is normal. The extremities are cool with delayed capillary refill.

These findings are consistent with Class 3 shock.
Think about the following:

- What are the possible sites of blood loss?
- How would you decide which site is responsible?
- What do you do next?

Two large bore cannula are sited and warmed crystalloid solution is infused and blood requested. The lower abdomen is bruised and tender. Chest (CXR) and Pelvis X-rays (PXR) are obtained as part of the primary survey. The CXR shows some posterior rib fractures but is otherwise normal. The PXR shows an unstable pelvic fracture and a binder is applied (Figures 2&3). Focused Assessment with Sonography for Trauma (FAST scan) was negative and a pelvic source of bleeding is assumed.

So far the patient has received 2000ml of warmed crystalloid solution and 1 unit of packed red cells. A pelvic binder is in place. Blood pressure is 110/60 mmHg and pulse 100bpm. He is fully alert and a thorough secondary survey is performed looking for associated injuries. Blood is noted at the urethral meatus therefore the patient is not catheterised and a urological opinion sought. Rectal examination reveals a normal prostate. A trauma CT (head, spine, chest, abdomen and pelvis) is organised.

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Managing Pelvic Fractures
Patient Management

Discussion

Pelvic fractures should be suspected in any patient involved in high energy trauma and in any patient with serious abdominal or lower limb injuries. There may be swelling or bruising of the lower abdomen, thighs or perineum and these areas should be carefully inspected.

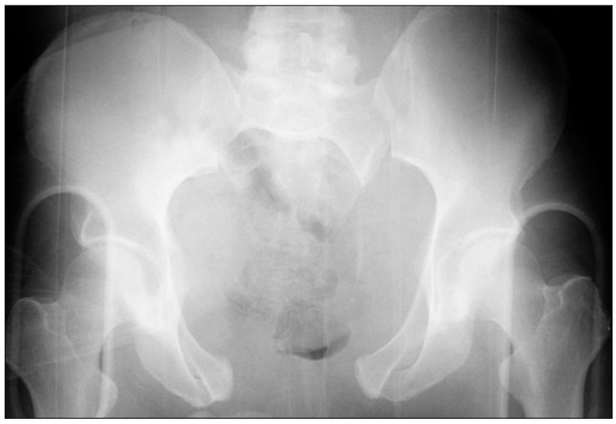

Figure 2a: Initial X-ray showing 'open book' type pelvic fracture (AP compression type 2).

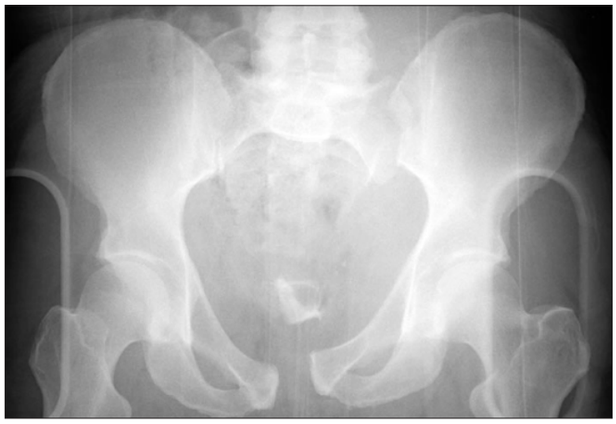

Figure 2b: After application of pelvic binder.

Lateral Compression	AP Compression	Vertical Shear
Type I Sacral compression on the side of impact associated with transverse pubic rami fractures	Type I Pubic diastasis <2.5 cm, either at the symphysis or through sagittal ramal fractures. The posterior sacroiliac ligaments remain intact	Vertical pubic rami fractures, sacroiliac joint disruption +/- adjacent fractures
Type II Transverse rami fractures and ipsilateral posterior iliac wing fracture or SI joint disruption	Type II Symphysis diastasis > 2.5cm and anterior sacroiliac joint disruption. The posterior sacroiliac ligaments remain intact	
Type III In addition there is external rotation of contralateral hemipelvis and sacroiliac joint injury	Type III There is complete disruption of the pubic symphysis and the sacroiliac joint	

Table 1

In this case the initial management is the same for any patient involved in major trauma and treatment follows ATLS protocols. Rapidly assess ABCDEs, remove all clothing and take measures to prevent hypothermia.

The leading cause of death from pelvic fracture is haemorrhage and as the patient has circulatory compromise the pelvic fracture is dealt with as part of 'C' in the primary survey. Increasingly pelvic binders are being applied at the scene by the paramedic team when a pelvic fracture is suspected.

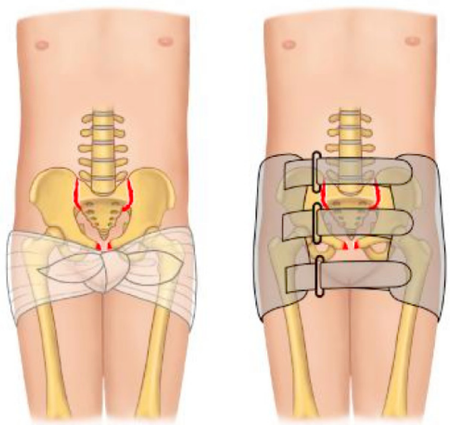

Figure 3: Demonstrating the proper positioning of a pelvic binder centred over the greater trochanter. A tightly applied sheet may be equally effective.

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The potential sources of blood loss are chest, abdomen, pelvis/retroperitoneum, and limbs. Don't forget blood loss that may have occurred at the scene (remember 'blood on the floor and 4 more' (1)).

Quickly the potential sites of bleeding are narrowed down to the abdomen/pelvis on the basis of examination and initial X-rays.

Three investigations are commonly used to differentiate between abdominal and pelvic bleeding:

- FAST scan
- Diagnostic peritoneal lavage (DPL)
- CT scan

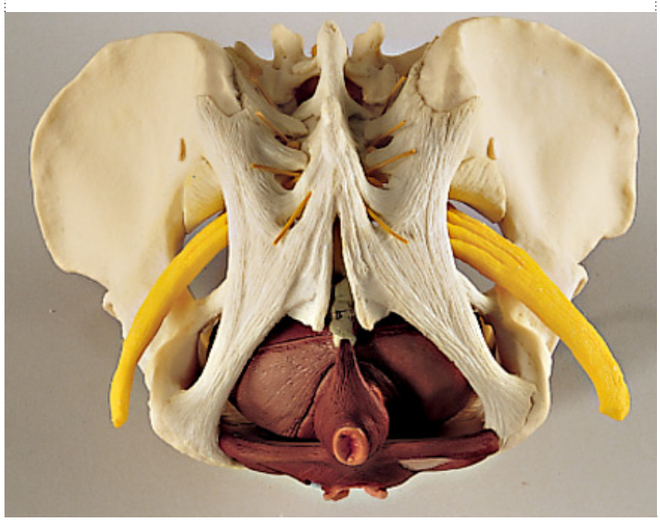
ATLS guidelines suggest CT is contraindicated in haemodynamically unstable patients however some authors challenge this view[2]. In this case FAST was performed during the primary survey as it is quick and non-invasive.

Manual testing of pelvic stability was not performed as this has a low sensitivity for detecting pelvic fractures and carries the risk of dislodging primary blood clots leading to further bleeding. The patient should be log rolled once to allow examination of the back and rectal examination which is mandatory. A high riding prostate may signify urethral rupture. Care must be taken to minimise mobility at the fracture site by keeping the number of examinations to a minimum.

Management of bleeding

Haemorrhage is the leading cause of death in patients with a pelvic fracture and where there is haemodynamic instability, mortality rates may as high as 35% (3).

The goal of resuscitation is to restore organ perfusion which is accomplished by the use of resuscitation fluids to replace lost intravascular volume. Raising blood pressure too rapidly can lead to increased bleeding if the source of haemorrhage has not been controlled.



This leads to consumption of clotting factors and in turn further bleeding. The current trend is for 'balanced' or 'controlled resuscitation', balancing the goal of organ perfusion with the risks of re-bleeding by accepting a lower than normal blood pressure, allowing time for definitive control of bleeding (1).

Patient Update

Trauma CT is performed 40 minutes after arrival. This demonstrates an AP Compression type 2 injury (See Figure 4/Table 1 - pelvic fracture classification). Contrast extravasation on both arterial and portal venous phase imaging suggests arterial bleeding is likely. No intra-abdominal source of bleeding is identified.

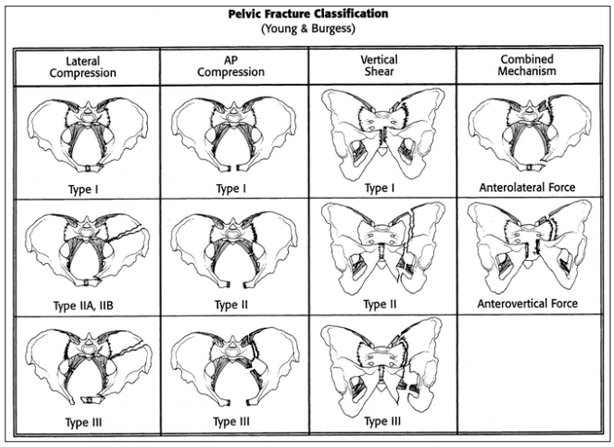


Figure 4. This is the most widely used classification system which classifies injuries according to four mechanisms.

On transfer back to the emergency department the patient again becomes drowsy and hypotensive. The primary survey is repeated:

Heart rate is 125bpm and blood pressure 80/40mmHg. Initial haemoglobin level was 6.8 g/dL and haematocrit 21.4%.

Despite transfusion of 3 further units of crossmatched packed red blood cells and 2 units of fresh frozen plasma he remains haemodynamically unstable. The patient is taken to the angiography suite where the interventional radiologist performs pelvic angiography. There was substantial bleeding from the left internal pudendal artery which was selectively cannulated and embolised (Figure 5). The patient was transferred to intensive care for further monitoring.

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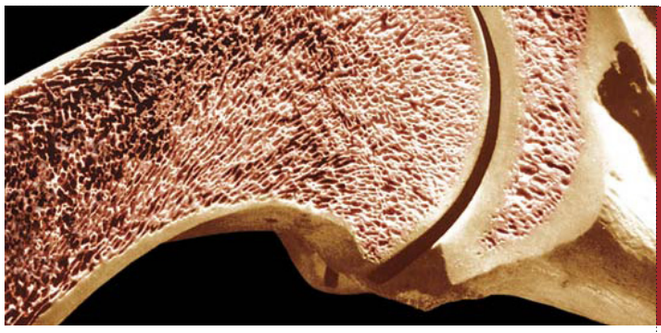

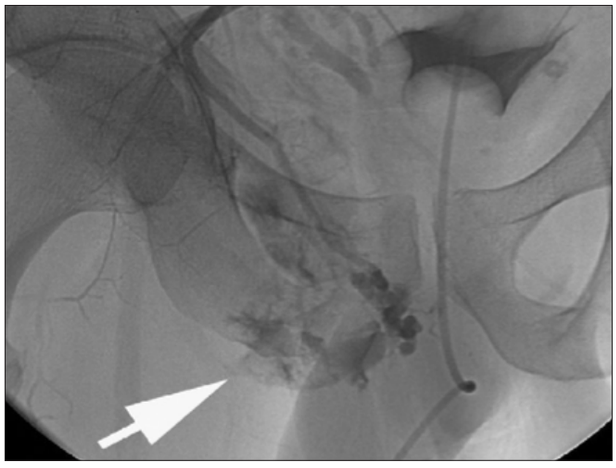

Figure 5: Angiography prior to embolisation of the bleeding vessel.

Methods of haemorrhage control

Three sources of bleeding are recognised in pelvic fractures: arterial, venous and bleeding from cancellous bone. Venous and cancellous bleeding is generally managed by initial stabilisation of the fracture to facilitate tamponade. This can be achieved by the use of a pelvic binder or external fixation with the aim of reducing pelvic volume and minimising bleeding from the fracture site. In this case the pelvic binder was felt to be effective and external fixation would provide no additional advantage.

Arterial injuries comprise 10% to 15% of cases of hemorrhagic pelvic fractures (4) and their management is more controversial and will differ depending on local protocols and expertise.

Traditionally external fixation and pelvic packing was the treatment of choice for suspected arterial haemorrhage. Surgical control of haemorrhage is now less favoured as this decompresses the haematoma with loss of tamponade. It is only contemplated if laparotomy is indicated for other intra-abdominal injures.

Angiographic embolisation of bleeding vessels is being increasingly used with reported success rates of 85–100% (5-8). This should be considered in any patient whose source of bleeding is predominately due to the pelvic fracture and who remains haemodynamically unstable following initial resuscitation.

Anatomy

The pelvis transmits weight from the trunk to the lower limbs and provides protection for pelvic viscera, vessels and nerves.

The pelvic ring consists of the two innominate bones (each comprising ilium, ischium, and pubis) and the sacrum, articulating anteriorly at the symphysis pubis. The stability of this ring is dependent not only of the bony parts but also on the integrity of the strong ligaments which bind the segments together.

The sacroiliac joints can be divided into anterior and posterior and are held together by the anterior and posterior sacroiliac ligaments. The posterior sacroiliac ligaments are the strongest in the body and are most important in maintaining pelvic stability. The pubic symphysis is weaker and more easily ruptured.

Urological injury

This occurs in about 10% of patients and should be strongly suspected where there is significant displacement of the pubic symphysis or pubic rami. Most bladder ruptures are extra-peritoneal and generally require repair (9).

Urethral injury is more common in men and catheterisation should not be attempted as this may convert a partial tear to a complete tear. In this case a retrograde urethrogram is performed demonstrating a partial tear and a suprapubic catheter is inserted. Complete tears may require more complex reconstructive surgery (9).

Patient Update

The patient remained haemodynamically stable with no evidence of recurrent bleeding and four days later was taken to theatre for open reduction and internal fixation of the pelvis with a plate across the pubic symphysis and screws posteriorly across the sacroiliac joint (Figure 6). He was discharged home two weeks later and after six months had recovered well except for some difficulty passing urine due to a urethral stricture.

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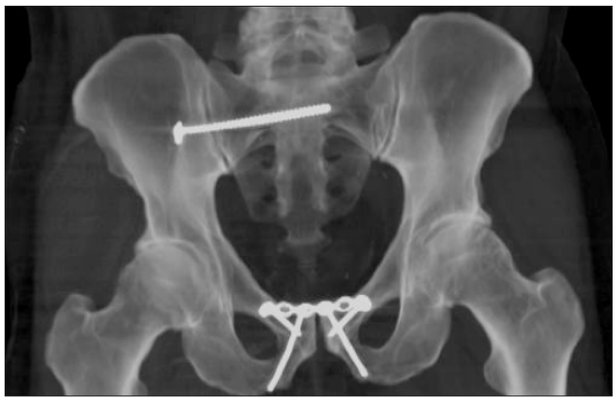

Figure 6: Follow-up x-ray after internal fixation.

MCQs

1) A 25 year old female is knocked down by a car while crossing the road. There was a brief period of loss of consciousness. On arrival in A&E there is marked bruising over the lower abdomen, right flank and right hip. Observations were: BP 90/50. HR 138, RR 28, Oxygen Saturations 96%, GCS 13/15. The following are all acceptable investigations during initial assessment EXCEPT:

A) Chest X-ray
B) Diagnostic peritoneal lavage (DPL)
C) Trauma CT scan (head, spine, chest, abdo, pelvis)
D) Pelvis X-ray
E) FAST Scan (Focused Assessment by Sonography in Trauma)

2) The following are all accepted approaches for controlling bleeding associated with unstable pelvic fractures EXCEPT:

A) Pelvic wrap
B) Balanced fluid resuscitation
C) Pelvic external fixation
D) Minimising movement of the pelvis
E) Angiography and embolisation

Answers

1. Answer = C

The initial assessment of any patient with a suspected high energy pelvic fracture is always the same and follows ATLS protocols. Chest and pelvic x-rays are included as standard in the primary survey in this situation. Either DPL or FAST scanning may be used depending on local policy to look for intra-abdominal bleeding however neither will demonstrate the source of the bleeding and nor can they assess the retroperitoneum or pelvic musculature. Some authors (2) advocate that CT scanning should be elevated to the primary survey alongside circulation in haemodynamically unstable patients citing the following advantages:

- the primary source of bleeding is identified more quickly allowing earlier and more focused angioembolisation
- other organ injuries are identified early
- the number of unnecessary laparotomies is reduced
- the number of units of transfused blood is reduced

This is however not standard practice and current ATLS guidelines state that CT is contraindicated in haemodynamically unstable patients.

2. Answer = B

Fluid resuscitation is an important component in the initial management and stabilisation of the patient with an unstable pelvic fracture however it is only a temporising measure and is not a substitute for definitive control of bleeding. This may be achieved by the use of a pelvic wrap or binder which acts to oppose the bleeding cancellous bony surfaces and reduce pelvic volume to tamponade bleeding. Unnecessary movement and manual testing of pelvic stability carries the risk of dislodging the primary (and best) blood clot which can lead to further bleeding and consumption of clotting factors. Angioembolistion is an effective method of controlling arterial bleeding.

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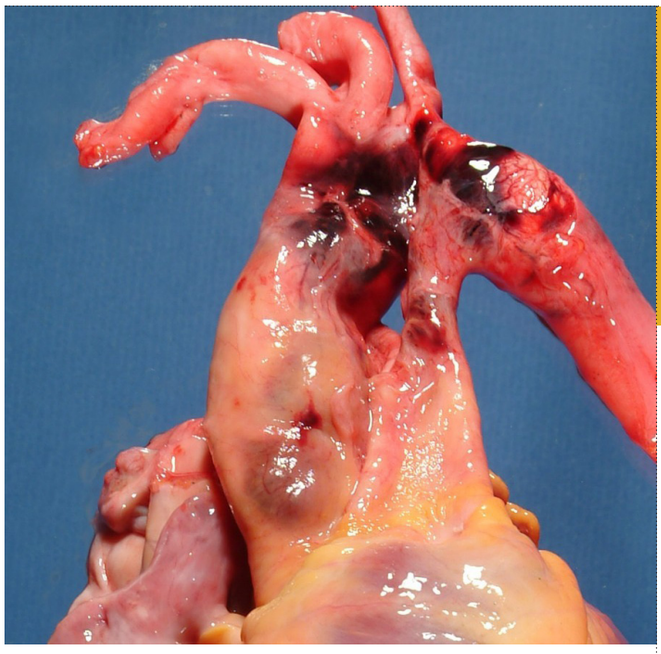
THE DISSECTING AORTA

A Spiteri, R Brown



Abstract

Aortic dissection is a rare vascular emergency with very high mortality if left untreated, thus needing rapid assessment and resuscitation with early involvement of high level care teams. Patients are usually acutely unwell at presentation, but a proportion of patients have vague and atypical symptoms and signs, making diagnosis difficult and liable to error. Aortic dissection should be part of the differential diagnosis of any patient with chest, abdominal and back pain. It may also present in an atypical manner such as with syncope, abnormal neurological signs, congestive heart failure and respiratory or gastrointestinal bleeding. The structured diagnostic approach and immediate management for a patient presenting with chest pain is discussed.

Case History

A 58 year old man presented to our emergency department with sudden onset of chest pain around 90 minutes prior to arrival. Whilst standing talking to a colleague he complained of sudden central chest pain radiating through to the back and epigastrium. The pain was immediately severe with a pain score of 10/10. He felt continuous pain since onset that was worsening on inspiration and associated with mild shortness of breath. He could not recollect a similar episode in the past. The paramedics gave sublingual glyceryl trinitrate with no relief. There was no associated nausea, vomiting, sweating, palpitations, dizziness or collapse. After 15mg of morphine the pain score severity was 8/10.

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Good Clinical Care

On further questioning the patient described a past history of hypertension, hypercholesterolaemia and had undergone a metallic aortic valve replacement in 2005 following the diagnosis of Marfan's syndrome. His medications included warfarin, atenolol, ramipril and simvastatin. He was a social drinker and non-smoker.

On examination the patient looked unwell and in pain with relief after further morphine use. His recorded observations on arrival were a heart rate of 76 beats per minute, blood pressure of 193/87 mmHg in the left arm and 173/81 mmHg in the right arm, a respiratory rate of 18 breaths per minute, temperature of 36.2°C and oxygen saturations of 99% at room air. Cardiorespiratory examination revealed only a metallic second heart sound and scattered inspiratory rales in the base of the left lung. There was mild epigastric tenderness on palpation but no palpable expansile masses or organomegaly. Limb examination showed synchronous radio-radial pulse. His right lower limb was noted to be pulseless with delayed capillary refill time but normal sensation.

His ECG on admission suggested ischaemia with T wave inversion in III, AVF and V3 to V6 (figure 1), whilst laboratory tests were essentially normal including arterial blood gases and troponin. The INR was raised as expected in a patient on warfarin at 2.7. Immediate management with aspirin 300mg and clopidogrel 300mg for presumed coronary insufficiency was given together with glyceryl trinitrate infusion at 4mg per hour. The nature and severity of the pain with distal arterial insufficiency was thought to be suggestive of an aortic dissection and a wide mediastinum on chest x-ray (figure 2) supported this provisional diagnosis.

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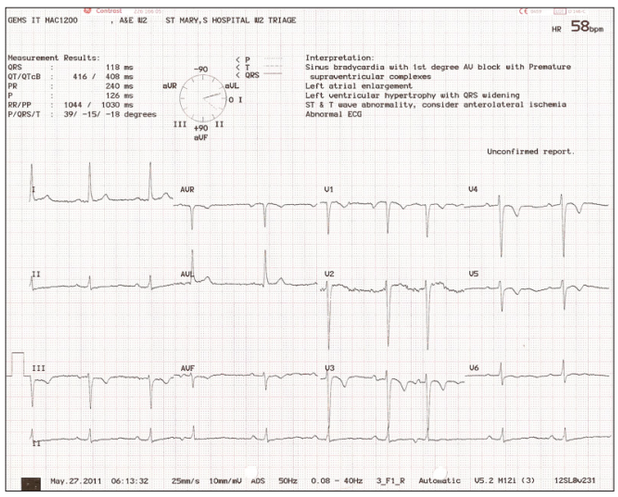

Figure 1: ECG showing sinus bradycardia at 58bpm with 1st degree heart block and T wave inversion in V3-V6, III and AVF.

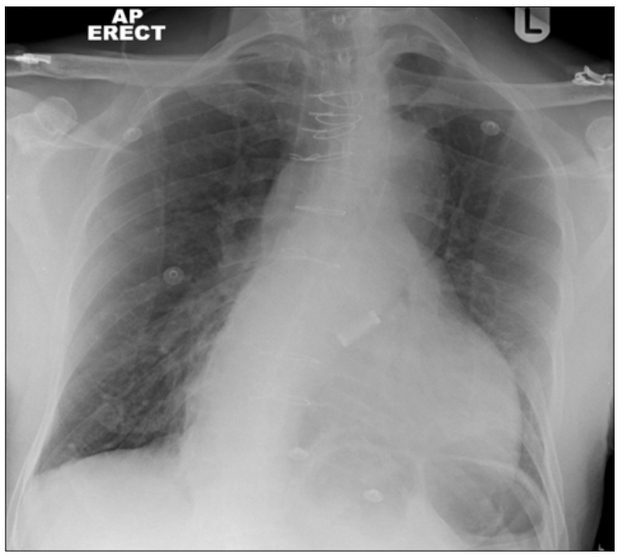

Figure 2: Chest Xray showing a widened mediastinum, prominent aorta and a metallic aortic valve.

A contrast enhanced computerised tomography of the chest showed a type B aortic dissection extending from the aortic arch (just distal to the origin of the left subclavian artery) to the right external iliac artery (figure 3-6). The thoracic aorta measured 40mm in diameter and the true lumen was narrowed down to 11mm. The abdominal aorta measured 31mm in diameter with a true lumen measuring only 12 mm.

The coeliac axis, superior mesenteric artery and renal arteries originated from the true lumen of the abdominal aorta and were well opacified with contrast. Both kidneys showed good cortical enhancement. The inferior mesenteric artery originated from the false lumen. Severe stenosis of the right common iliac artery and proximal external iliac artery was noted.

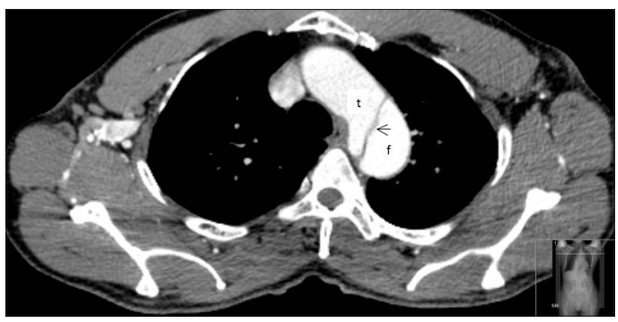

Figure 3: Aortic dissection originating from aortic arch just distal to left subclavian artery showing the intimal flap (arrowed) with the true (t) and false (f) lumen on each side.

After confirmation of the dissection, a labetolol infusion was commenced (after loading dose of 50mg) in addition to the glyceryl trinitrate infusion to control the blood pressure and heart rate. The patient was catheterised to aid fluid management and admitted to the vascular High Dependency Unit. Further imaging included a transthoracic echocardiogram which showed an extensive aortic dissection flap that started proximal to the left common carotid artery. However a second contrast enhanced CT reconfirmed that the dissection only involved the descending aorta.

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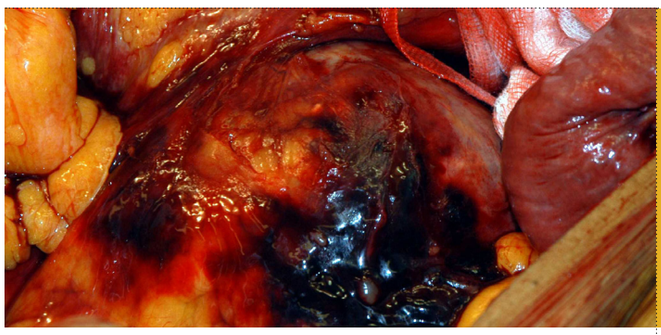

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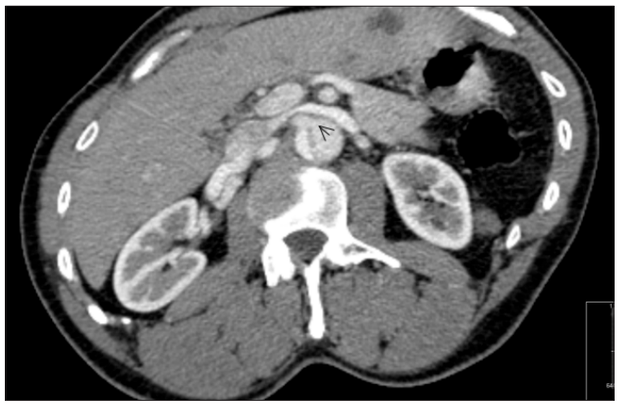

Figure 4: Note the origin of the right renal artery from the true lumen (arrow).

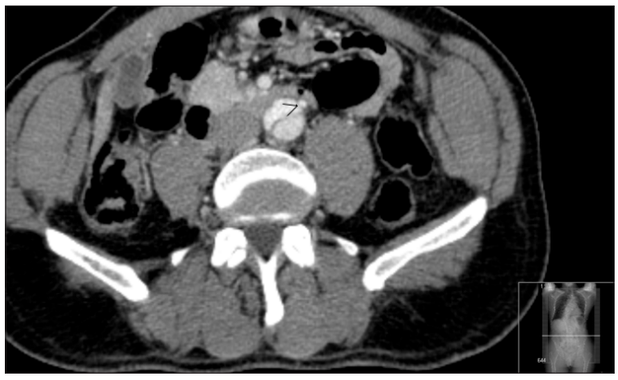

Figure 5: The inferior mesenteric artery is originating from the false lumen (arrow).

The patient was treated conservatively with strict blood pressure control over a period of 18 days. The warfarin was initially stopped and a heparin infusion started. The vascular state of the right lower limb improved with no sign of ischaemia. Prior to discharge he was on oral antihypertensive medications and re-warfarinised. A CT scan on day 4 showed mild enlargement of the aneurysmal sack but no extension of the dissection. A final CT scan prior to discharge showed no new change.

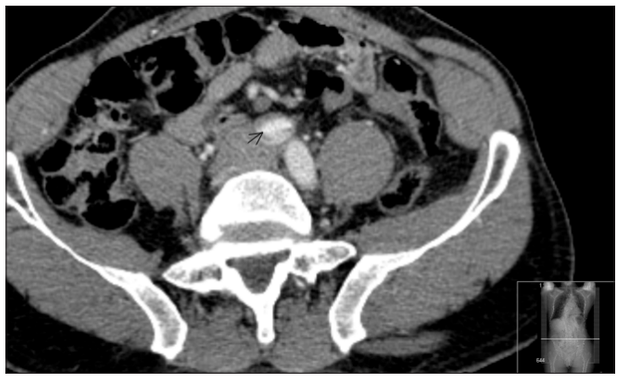

Figure 6: The dissection extends into the right common iliac artery (arrow) beyond the aortic bifurcation.

Discussion

The reported incidence of thoracic aortic dissection is estimated to be 3-4 per 100,000 persons per year. Such an incidence is likely to be an underestimation as, unless the appropriate imaging is undertaken, patients may remain undiagnosed. (1) This is further compounded by the fact that the untreated mortality in the first 24-48 hours is up to 1-2% an hour, usually as a result of ischaemia to vital organs or rupture of the sac. (1,2)

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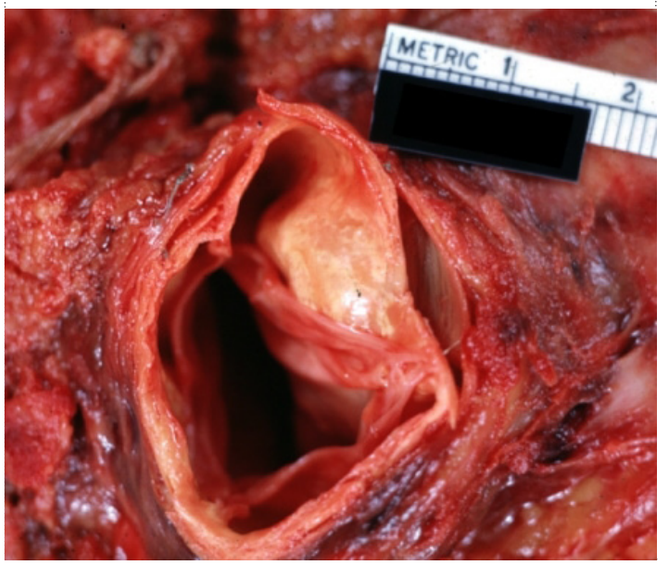
The diagnosis must be considered if there is an unusual presentation of a common condition, or when there are recognised 'red-flag' risk factors as shown in table 1 (2,3) Our patient suffered from Marfan's syndrome, chronic hypertension, had a history of aortic valve pathology and surgery all of which made the dissection diagnosis more possible. Early consideration of the diagnosis will avoid the otherwise relatively high mortality in patients with Marfan's syndrome who present at a relatively younger age. (2,4)

Past medical history 'red flag' findings prompting consideration of aortic dissection
Chronic hypertension* – exacerbated by smoking and cocaine use
Connective tissue disorder – Marfan's syndrome, Ehlers-Danlos syndrome
Iatrogenic – vascular or cardiothoracic surgery (e.g. valvular repair), catheterisation
Hereditary vascular diseases – bicuspid aortic valve, aortic coarctation
Family history of aortic dissection, aneurysm or sudden death
Vasculitis – giant cell arteritis, syphylis, Takayasu arteritis, Behcet's disease
Other – Turner's syndrome, pregnancy, tuberculosis, traumatic
*Long standing hypertension is the most common predisposing factor.

Table 1: Risk factors of aortic dissection (2,4)

The Stanford classification of aortic dissection into type A and B is illustrated in figure 7. Type A aortic dissection involves the ascending aorta, whilst type B does not. One of the diagnostic challenges is to weigh up the relevance of any single symptom or sign. Characteristically the pain is described as being the worst ever and this detail must be sought explicitly.

Anterior chest pain is associated with type A dissections involving the ascending aorta, whilst interscapular pain is associated with those involving the descending aorta (type A and B). Type A dissections are also associated with aortic regurgitation, myocardial ischaemia or infarction and cardiac tamponade as the dissection extends from the ascending aorta to the aortic root. Table 2 shows the main presenting symptoms and signs of acute aortic dissection as reported in the International Registry of Acute Aortic Dissection.(5)



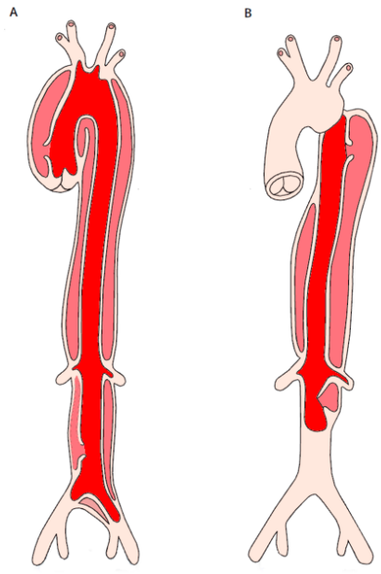


Figure 7: Stanford classifications of aortic dissection. Type A but not type B dissection affects the ascending aorta.

Type A dissections affect the ascending aorta and can cause malperfusion of brain, coronary arteries, aortic regurgitation and cardiac tamponade. Type A and B dissections involving the descending aorta can cause malperfusion of the spinal cord, liver, bowel, kidneys and legs.

	Type A (%)	Type B (%)	Overall (%)
Symptoms and signs			
Chest or back pain	85	86	85
Severe or worst-ever pain	90	90	90
Abrupt onset of pain	91	89	90
Migrating Pain	85	90	19
Pain presenting within 6h of symptoms	79	-	-
Abdominal pain	22	43	30
Any focal neurological symptoms	17	5	12
Syncope	13	4	9
Hypotension*, shock, or tamponade	27	69	49
Hypertension* at presentation	36	69	49
Any pulse deficit	31	21	27
Aortic regurgitation	44	12	32
Cerebrovascular accident	6	2	5
Congestive heart failure	9	3	7
Chest radiography			
Widened mediastinum	63	56	60
Abnormal aortic contour	47	49	48
Normal	11	21	16
Electrocardiography			
Normal	30	31	30
Left ventricular hypertrophy	23	32	26
Myocardial ischaemia or infarction	24	10	17
Percentages represent total number for whom data were available. *Systolic blood pressure ≥150mmHg or <100mmHg			

Table 2: Risk factors of acute aortic dissection from the International Registry of Acute Aortic Dissection (5,6)

THE DISSECTING AORTA

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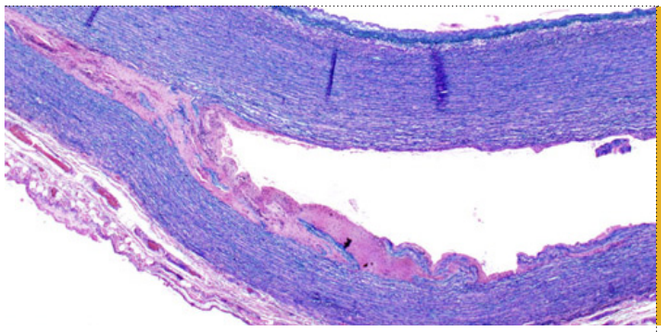

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Since up to 10% of patients present without pain, there must be a thorough examination of the patient to identify unusual or unexplained physical signs. Conventionally, aortic catastrophes are thought to be associated with hypotension, but, as in our patient, hypertension is more common in type B dissection. (5) In aortic dissection (as opposed to aortic aneurysmal rupture), it is unusual to palpate a dilated aorta in the abdomen.

The absence of a pulse in the right limb but with no apparent source of potential embolism or previous peripheral vascular disease also strongly suggests dissection. As in our case the loss of limb pulses is typically painless. (6) In any patient with signs or symptoms of vascular compromise that is not easily explained, the diagnosis of dissection must be considered. This includes signs of end-organ ischaemia such as stroke, syncope, paralysis and anuria. Pressure on nearby structures from the sac (e.g. oesophagus causing dysphagia) as well as rupture (e.g. into the pericardium or gastrointestinal tract resulting in tamponade and bleeding respectively) can further add to the numerous presenting complaints (5,7).

Chest pain accounts for about 6% of visits to the emergency department in the UK. (8) Many patients with aortic dissection present with chest pain alone. The diagnostic approach to the patient with chest pain should attempt to rule out the life threatening diagnoses with the minimum number of investigations. Easily obtained investigations such as chest radiography, ECG and cardiac enzymes, helpful in cardiac ischemia, are not sensitive or specific enough to rule out or rule in aortic dissection (5,9,10); in particular the chest x-ray is normal in around 16% of patients. (5) In our patient an abnormal ECG in the presence of chest pain may easily be diagnosed as a cardiac event and this may further be supported by the presence of new cardiac murmurs. However other clinical signs (the absent peripheral pulse) and pain that is out of proportion to the ECG changes and unrelieved by glyceryl trinitrate is strongly suggestive of an alternative or more sinister diagnosis.

Once dissection has been considered as a potential diagnosis further imaging is a must. In our case we organised an immediate contrast enhanced CT of the chest, abdomen and pelvis. Abdominal ultrasound is reliable in determining the diameter of the aorta but is less reliable in identifying the presence of a flap. The more accurate transoesophageal ECHO (TOE) is better suited for unstable patients but not readily obtainable in the emergency department. In our case transthoracic ECHO (TTE) was used to further delineate the aortic flap through a suprasternal window approach. TTE also aided in assessing for a haemopericardium, abnormal cardiac wall motion, valvular and aortic root pathology.

A non-contrast CT will not provide the images required to identify the origin of the dissection and thus contrast is required. The CT scanning suite is often remote from the ED or HDU and transfer with monitoring must be carefully planned to minimise the risk. Modern scanners are able to complete the examination rapidly and help to make the process safer. Clear anatomical delineation of the flap is essential to determine the need and nature of intervention. Angiography and MRI/MRA are alternative modes of diagnostic imaging but may be less appropriate particularly if the patient is unwell. (2,6,11)/

The key aim of the imaging is to identify the potential threat to the end organs and to identify which arteries are compromised by the dissection. Stanford type A dissections require urgent surgery whilst type B, as in our patient, are managed medically unless complicated by a leak, rupture or organ blood flow compromise. Thus accurate differentiation between the two is very important. Any suggestion that the gut or renal circulation may be compromised by the dissection would be an indication for vascular replacement to restore the arterial supply. An alternative to open operation and vascular bypass nowadays includes radiologically placed fenestrated stents, purposely designed for individual patients to provide appropriate vascular supply (12,13). In our patient, although the inferior mesenteric artery originated from the false lumen, the perfusion was maintained and no further intervention was required.

While the hypotensive patient requires resuscitation to maintain perfusion of vital organs pending intervention, the hypertensive patient, as in our case, requires blood pressure control to minimise the risk of extension of the flap or rupture. The goal would be to maintain the systolic blood pressure at less than 110 mmHg or mean arterial pressure of 70-80 mmHg and a heart rate of 60-70 beats per minute. Reducing the blood pressure must be finely balanced with maintaining perfusion and a combination of drugs is used to both lower the force of contraction and dilate the arterioles (combined alpha and beta blocker such as labetalol) and to venodilate and hence reduce the preload of the ventricle (nitrates - preferably sodium nitroprusside which also has an arteriolar dilatory effect) (5,6). Beta blockers have the most desirable effect of reducing the force of left ventricular ejection resulting in a rapid lowering of the change in pressure over time (dP/dT). Using a beta blocker as the initial treatment will also offset the reflex tachycardia caused by subsequent nitrate infusion thereby minimising aortic wall stress (6). If beta blockers are contra-indicated calcium channel blockers can be used (6).

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Aortic dissection is a vascular emergency and must be considered in the differential diagnosis of a wide spectrum of presenting conditions in view of the high mortality if otherwise undiagnosed. Prompt diagnosis and emergency treatment in a resuscitation area is critical. Immediate involvement of senior clinicians and high level care teams is needed. If vascular services are not available locally, urgent transfer must not be delayed. Accurate history taking, with emphasis on 'red flag' risk factors, along with examination of the patient is crucial in order to provide early management of such a perilous illness.

Test Yourself

Q1. Aortic Dissection can present with:

a) Paraplegia
b) Abdominal pain
c) Syncope / stroke
d) Myocardial ischaemia / infarction
e) All of the above

Q2. All of the following are risk factors for aortic dissection except:

a) Hypertension
b) Marfan's syndrome / Connective tissue disorder
c) Cocaine
d) Diabetes
e) Family history of aortic dissection

Q3. What percentage of patients with aortic dissection has an alternative diagnosis made, with possible life-threatening consequence?

a) 5%
b) 10%
c) 30%
d) 50%
e) 80%

Q4. Chest radiography changes indicative of aortic dissection include all of the following except:

a) Left pleural effusion
b) Widened mediastinum
c) Pulmonary venous congestion
d) Deviation of trachea, bronchus or oesophagus
e) Left apical pleural cap

Q5. All of the following are false in acute aortic dissection except:

a) Men are more commonly affected than women, but the latter have worse prognosis
b) Absence of pulse deficit excludes the diagnosis of aortic dissection
c) Nitrate therapy can be used as initial or sole therapy to lower the blood pressure
d) A negative D-Dimer has an excellent sensitivity and is a useful rule-out test
e) A normal examination, ECG and chest X-ray rule out the diagnosis of aortic dissection

Answers

Q1: e
Aortic dissection can present according to which aortic branch it affects. Thus any branching artery can be affected including coronaries, carotids, subclavians, spinal artery, coeliac, mesenteric, renal and iliac arteries.

Q2: d
Diabetes has been shown to be negatively associated and possibly protective for aortic dissection. The mechanism is uncertain.1 In contrast chronic hypertension, connective tissue disorders, family history and cocaine use are associated with increased risk (1).

Q3: c
30% of patients with aortic dissections are misdiagnosed on initial presentation, often leading to fatal consequences (2).

Q4: c
Pulmonary venous congestion is not a typical feature of aortic dissection. Widened mediastinum is typical of aortic dissection. Other features include deviation of mediastinal structures, obliteration of the aorto-pulmonary window, indistinct aortic contour, displaced intimal calcification, left sided pleural fluid and apical cap.

Q5: a
Men are more commonly affected by aortic dissection by a ratio of 2:1.1 Women tend to present at a later age and have a worse prognosis. Examination including pulse deficit, ECG and chest X-Ray are not sensitive enough to exclude aortic dissection. Nitrates, such as sodium nitroprusside, if used as initial or sole treatment can result in reflex tachycardia exacerbating the dissection further. D-Dimer is not reliable to exclude dissection with sensitivities varying between 88 and 100% (9,10).

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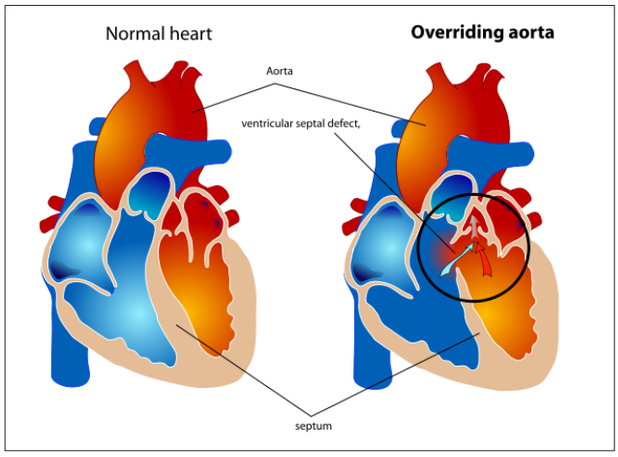

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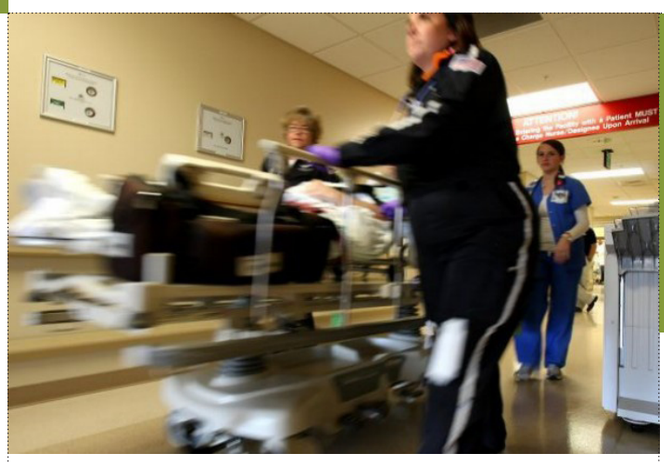
TRAUMA CARE IN THE UNITED KINGDOM - HOW CAN WE BRIDGE A GAP IN THE EXISTING MEDICAL EDUCATION?

J Kaczynski



Introduction

The main focus of this article is to explore the current trauma care training opportunities for the foundation and core surgical trainees in the United Kingdom, in particular for those who are interested in the surgical or emergency care medicine career.

Trauma is the leading cause of death and disability in the first four decades of life[1]. This causes more "life-years" lost than cardiovascular disease and cancer combined, resulting in significant social and economic costs (1,2). Trauma can be extremely complex and unpredictable; therefore early diagnosis and rapid treatment by the skilled doctors are essential for patient survival.

The National Confidential Enquiry into Patient Outcome and Death (NCEPOD) revealed that 60% of trauma patients in England and Wales have received suboptimal perioperative trauma care (3). This has led to a major reconfiguration of the trauma services, which is currently taking place across the United Kingdom (UK). The new regional trauma system is based on the excellent results achieved by the London Trauma Project, which showed a significant reduction in mortality from severe injuries (4). The regional trauma system model aims to reduce the disability and death following major trauma by utilising the "chain of survival" concept (5-8). In such approach, the trauma victim receives the best possible care during each phase of the "trauma disease" in order to optimise clinical outcome. In practice, this means that the trauma care starts immediately after injury at the scene and subsequently is delivered in the dedicated trauma and rehabilitation centres.

The Intercollegiate Group On Trauma Standards developed the guidance for the Commissioners on delivery the best trauma care across the UK (6). The document describes the principles of the regional trauma system concept, which aims to deliver the best trauma care to a defined population within a geographical region by the right specialist (6,7). In effect all Strategic Health Authorities (SHAs') in England and Local Health Boards (LHBs') in Wales, excluding Scotland (Scottish Government rejected trauma reorganisation plans), have defined the geographical areas with Major Trauma Centres (MTC) and Trauma Units (TU) (3,6).

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Within a geographical area, all acute hospitals are part of the regional trauma system, and the MTC or TU status is based on the capabilities and resources of a particular hospital. The role of the MTC is to treat the most seriously injured patients, which make for about 15% of the injured population (5,6). The TU trauma units manage patients with less serious injuries (Injury Severity Score< 15) from a local area and ensure that the MTC is not overloaded with "moderate trauma" patients who would very likely be de-prioritised over the major trauma cases (5,6).

Several criteria have to be met by a particular hospital in order to receive the MTC designation. These major criteria include (6,7): the 24 hour Consultant led trauma team, ability to perform emergency CT within 30 minutes of arrival, fresh frozen plasma available within 15 minutes of request, spinal assessment completed within four hours of injury and mortality from haemorrhagic shock <30%. The vast majority of the regions will have one MTC supported by the several TU. The exception is the London trauma system, which provides 4 MTC, 25 TU and 12 TU outside London's area (7).

However, some regions may not be able to provide the MTC trauma care due to remote geography, access and high costs. In such circumstances, the trauma networks should be established which allow for the coordination of the trauma care within the neighbouring regions. One of the examples of such a network is the delivery of an emergency neurosurgery aiming at performing the craniotomy within four hours of injury in Wales. In such a scenario, the trauma victim following an immediate Computed Tomography (CT) is transferred from the TU to the MTC by an air ambulance. These demographical differences are challenging and further work is required in order to meet the trauma care requirements. The guidance on the development of the regional trauma systems is the first and most comprehensive document which sets out the plans for the trauma system development in the UK.

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Until regional trauma system is fully implemented, which will provide the continuum of care for the injured, the majority of the trauma patients are still going to be managed in the local Emergency Departments by a "trauma team" consisting of junior clinicians often with limited trauma experience. The 21st century trauma systems require not only a resuscitation area and easy access to Computerised Tomography (CT)/Angiography and theatre suite at the same time, but full integration of rehabilitation services, education, research and performance monitoring (5).

To provide a high quality service for trauma victims, we need trainees who would with confidence and up to the highest levels apply algorithms in management of the trauma patients. Working knowledge of basic resuscitation techniques is essential to all doctors, but especially for those wishing to embark on a surgical or emergency medicine career. Unfortunately, at present in the United Kingdom trauma surgery doesn't exist as a separate subspecialty. This may eventually change in the future, but even if we had the new training in place, it would take at least another decade for these newly qualified surgeons to be ready to provide trauma care.

Therefore, the Emergency Medicine (EM) physicians play a central role within new trauma system. They are currently and for many years onwards the only available trauma specialists in the UK. The EM Consultants will be leading the trauma team 24 hours a day being supported by the allied specialities such as general surgery, trauma and orthopaedic, neurosurgery and cardiothoracics.

Trauma education plays a vital role within the new trauma system. Unfortunately, currently there is no formal trauma teaching available within the undergraduate or postgraduate curriculum. So how can you as a trainee maximise trauma experience? The list below provides examples of the potential opportunities in the United Kingdom.

1. Courses provided by the Royal College of Surgeons of England and Edinburgh (9,10) include:

- Advanced Trauma Life Support (ATLS)

- Specialty Skills in Emergency Surgery and Trauma

- Pre-Hospital and Emergency Department Resuscitative Thoracotomy

- Emergency Abdominal and Thoracic Surgery for the General Surgeon

- Definite Surgical Trauma Skills for the General Surgeon

Both Colleges provide a detailed description about each of the courses including prices, dates and location under the section "courses" on their websites (Figure 1).

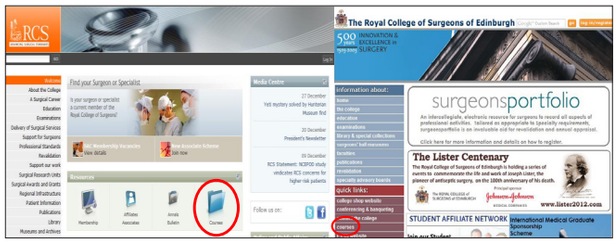

Figure 1: The Royal College of Surgeons of England and Edinburgh websites.

2. Two main websites focused on the trauma care contain articles, images, moulage scenarios, case presentations and information about upcoming conferences (11,12) (Figure 2):

http://www.trauma.myzen.co.uk/
http://www.trauma.org/

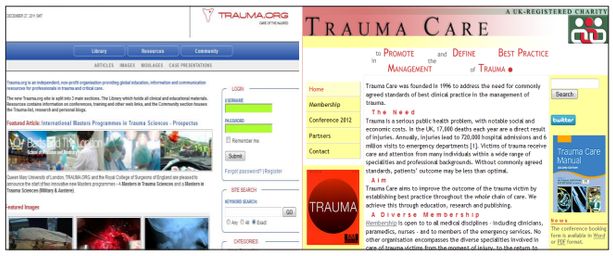

Figure 2. Trauma websites.

3. There are two Masters programmes, which are the only formal trauma studies available in the United Kingdom (13,14):

- MSc Trauma Surgery at the Swansea University: a 2 ½ year part-time study, which covers all aspects of Trauma Surgery, from initial resuscitation to final recovery. During the first year, students attend one week blocks at the School of Medicine- Morriston Hospital, Swansea. Second year provides an opportunity to develop an independent research question related to trauma surgery culminating in a dissertation. http://www.swan.ac.uk/pgcourses/medicine/msctraumasurgery/

- International Masters Programmes in Trauma Sciences – Prospectus: a two year, part time distance-learning course providing comprehensive and critical understanding of the most up-to-date science and practice of trauma care. It is a result of collaboration of Barts and the London School of Medicine, Queen Mary University of London; trauma.org and the Royal College of Surgeons of England. http://www.trauma.org/index.php/main/article/1285/

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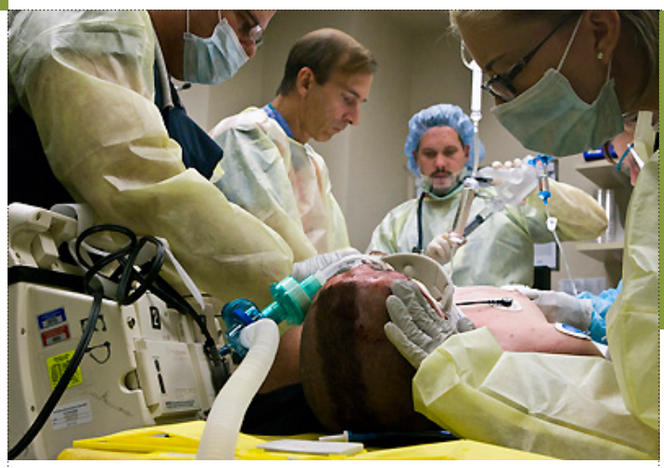

4. Elective experience in trauma units abroad provides an invaluable exposure and opportunity to gain insight into the challenges related to a front line trauma patient care (15). An article written by the Oliver Fuge describing the Groote Schuur Hospital trauma unit in the Cape Town (South Africa) can be found at Surgeons news, January 2011, p68-69.

5. National annual Student Trauma Conference will take place in March 2013 in Edinburgh (16). The conference is aimed primarily at foundation year trainees. It provides information on the principles of trauma management, and equips a junior doctor in practical skills required to work in a trauma department.https://ubis.rcsed.ac.uk/courses/coursedetails.aspx?diaryId=966

Conclusion

I strongly recommend to any foundation year trainee interested in trauma care who wishes to embark on future surgical or emergency medicine career to use as many opportunities as possible to build up and expand on the trauma skills.

All above proposed activities provide a platform for the foundation and core trainees to gain an understanding of the trauma as a "disease" in order to understand all consequences of an injury.

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TRIAGE IN THE EMERGENCY DEPARTMENT: SEPSIS

TE Kaier, CB Narshi

Triage in the Emergency Department: Sepsis
Patient Management

Abstract

Triage in the Accident and Emergency (A&E) department is the first step for any patient admitted to a hospital. Baseline observations help to identify critically ill patients and facilitate an urgent assessment with treatment via calculation of the Modified Early Warning Score (MEWS). The higher the score, the more urgent attention is required from a doctor. In this article we outline the Surviving Sepsis Campaign 2008 (SSC) guidelines.

Additionally, the key steps in management of the patient presenting with sepsis are summarised from the current literature. These steps include an oxygen administration, resuscitation with intravenous fluids and infusion of intravenous antibiotics. Also basic definitions of sepsis with its subsequent stages are provided. Therefore, the reader will appreciate why a streamlined process in assessing patients in the emergency department is used, as well as have a thorough understanding of the importance of rapid and aggressive treatment of patients with sepsis.

Case study

A 62-year-old woman presented to the A&E department with a 4 day history of dysuria and urinary frequency associated with an abdominal pain. Additionally, the patient complained of lethargy, nausea and rigors. Her co-morbidities comprised hypertension and hypothyroidism.

During triage, baseline observations were recorded by an A&E staff nurse and these revealed: temperature 38.5°C, heart rate 115 bpm, blood pressure 88/59 mmHg, oxygen saturations 97% on air and respiratory rate 20/min. The MEWS score of 3 (see table 2) has been calculated from the above haemodynamic parameters indicating a critically ill patient. Subsequently, the A&E nurse established peripheral venous access, sent routine bloods and blood cultures. The nurse has highlighted the high MEWS score to an A&E FY2 trainee who promptly assessed the patient using the ALERT approach (please explain what does the ALERT stands for).

On examination, the airway was patent, with a borderline tachypnea, circulation was compromised with a low blood pressure, tachycardia, and cold peripheries. Disability assessment revealed a pale but alert patient with a Glasgow Coma Scale of 15 and capillary blood glucose of 6.4 mmols. On Exposure the FY2 demonstrated soft abdomen with tenderness in the right flank.

A urine dip was positive for leucocytes ++ and nitrites +. The patient's blood counts were reported as below: Haemoglobin 13.5 g/dL, White Cell Count 17000 µL-1, C-Reactive Protein 320 mg/L, Sodium 138, K 4.2, Creatinine 120 µmol/L, Urea 10.5 mmol/L.

In view of the above history and examination findings an immediate intravenous fluid bolus of 250mls 0.9% isotonic saline was administered. Additionally, the FY2 trainee started the patient on intravenous antibiotics according to the local microbiology guidelines and continued further intravenous fluids infusion in order to maintain a systolic blood pressure above 90 mmHg. The patient responded well to this initial resuscitation. Her blood pressure improved, tachycardia and fever settled with further IV (intravenous) fluids administration.

The patient was then referred to the medical team for further management with the working diagnosis of urinary sepsis. During the hospitalisation, an abdominal ultrasound was performed and excluded a perinephric abscess and obstruction of the urinary tract. Blood cultures confirmed growth of Escherichia coli sensitive to the antimicrobial treatment used. Subsequently, the antibiotics were changed to a suitable oral preparation when the patient was afebrile for 24 hours, allowing for a safe discharge at the earliest opportunity.

This case highlights that simple measures can save patients' lives. These include:

Early triage by nursing staff in the emergency department, recording necessary observations and calculating an early warning score.

Blood cultures should be obtained at the earliest opportunity

Highlighting a patient with a high MEWS to a doctor, ensures a prompt antibiotic treatment, resuscitation with oxygen and intravenous fluids. The aforementioned key recommendations are outlined in great detail in the Surviving Sepsis Campaign (SSC) (1).

TRIAGE IN THE EMERGENCY DEPARTMENT: SEPSIS

TE Kaier, CB Narshi

Triage in the Emergency Department
Patient Management

Discussion

In the presented case the patient was tachycardic, hypotensive and febrile on admission, with a clear source of sepsis. The subsequent management of this patient's case illustrates implementation and importance of a number of guidelines. Before discussing some of these guidelines it is important to define some basics - what is sepsis?

Sepsis is one of the commonest reasons for admission to hospital in general and intensive care units in particular (2), for which the American College of Chest physicians and the Society of Critical Care Medicine introduced diagnostic criteria in 1992. The guidelines published in the Surviving Sepsis Campaign (1) divided the clinical response to a nonspecific insult, of either infectious or non-infectious origin, into the following subgroups:

Systemic inflammatory response syndrome (SIRS) - a response that can be triggered by a variety of conditions; this can occur in the absence of infection and is defined by the presence of more than one of the clinical manifestations outlined in table 1 (3). Noninfectious pathologic causes include pancreatitis, ischaemia, trauma and tissue injury, haemorrhagic shock and immune-mediated organ injury (4).

Here are some basic definitions:

Sepsis – SIRS and documented source of infection (5)

Severe Sepsis – confirmed infection and evidence of an altered organ perfusion

Sepsis syndrome – an altered organ perfusion however no confirmed infection

Septic shock – an infection associated with hypotension (defined as a systolic arterial pressure below 90 mm Hg) refractory to IV fluids

Multiple organ dysfunction syndrome (MODS) – describes the development of a progressive and potentially reversible physiological dysfunction in 2 or more organs (3, 6)

Sepsis and its consequences

Sepsis is defined as infection plus more than one systemic manifestations of infection (1). Systemic manifestations are any of the criteria outlined in Table 1. In the illustrated case the patient fulfilled several of these criteria including fever, heart rate > 90 min-1, arterial hypotension, leucocytosis, elevated CRP. So why is it so important to recognise these patients as early as possible? Substantial evidence from the literature (1, 7, 8, 9) demonstrated significant improvements in both mortality and morbidity following early sepsis recognition and prompt treatment. A comprehensive How-to Guide - The Surviving Sepsis Campaign (SSC).

General variables
Fever (> 38.3°C)
Hypothermia (core temperature <36°C)
Heart rate >90 min^{-1} or >2 SD above the normal value for age
Tachypnea
Altered mental status
Significant edema or positive fluid balance (>20 mL/kg over 24 hrs
Hyperglycemia (plasma glucose >140 mg/dL or 7.7 mmol/L) in the absence of diabetes
Inflammatory variables
Leucocytosis (WBC count >12,000 μL^{-1})
Leucopenia (WBC count <4000 μL^{-1})
Normal WBC count with >10% immature forms
Plasma C-reactive protein >2 SD above the normal value
Plasma procalcitonin >2 SD above the normal value
Hemodynamic variables
Arterial hypotension (SBP <90 mm Hg; MAP <70 mm Hg; or an SBP decrease <40 mm Hg in adults or <2 SD below normal for age)
Organ dysfunction variables
Arterial hypoxemia (PaO2/FIO2 <300)
Acute oliguria (urine output <0.5 mL/Kg hr or 45 mmol/L for at least 2 hrs, despite adequate fluid resuscitation)
Creatinine increase >0.5 mg/dL or 44.2 µmol/L
Coagulation abnormalities (INR >1.5 or a PTT >60 secs)
Ileus (absent bowel sounds)
Thrombocytopenia (platelet count <100,000 μL^{-1})
Hyperbilirubinemia (plasma total bilirubin >4 mg/dL or 70 µmol/L)
Tissue perfusion variables
Hyperlactatemia (> upper limit of lab normal)
Decreased capillary refill or mottling

WBC, white blood cell; SBP, systolic blood pressure; MAP, mean arterial blood pressure; INR, international normalized ratio; aPTT, activated partial thromboplastin time.
adjusted from Dellinger et al. (1)

Table 1: SIRS criteria.

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A stepwise process of managing sepsis comprises of the two care bundles: the first one focuses on resuscitation and aims at the patient optimisation within the first six hours of sepsis. Whereas, the second bundle consists of number of recommendations for the following 24 hours. We summarised the key aspects of these two bundles below:

1. Sepsis Resuscitation bundle – the following interventions should be accomplished as soon as possible and scored over the first 6 hours (9):

1.1. Initial resuscitation should begin immediately if there is any evidence of hypotension or an elevated serum lactate > 4 mmol/L. As there is no evidence supporting one specific type of fluid for resuscitation, either colloids or crystalloids may be used. A fluid challenge technique should be applied and suspected hypovolaemia should at first be treated with ≥ 1000 mL of crystalloids or 300-500 mL colloids (1) respectively.

1.2. Blood cultures prior to antibiotic administration: Appropriate cultures should be obtained whenever possible without causing a significant delay in the administration of antibiotics (e.g. blood cultures, urine, cerebrospinal fluid, wounds, respiratory secretions or other body fluids).

1.3. Broad spectrum antibiotics: Intravenous antibiotics should be started as early as possible but certainly within three hours of presentation to an emergency department. Where necessary, an empirical therapy covering majority of the most common pathogens is indicated. This should then be assessed on a daily basis and adjusted as per microbiology results and clinical course. Combination therapy is advocated in anybody with suspected pseudomonas infection or neutropaenia, with the aim of de-escalation to the most appropriate agent (1).

Typical duration of therapy would comprise of a 7-10 days course but should be stopped immediately if the presentation is thought to be due to a noninfectious cause. Administering antibiotics at the earliest opportunity appears to be most beneficial compared to any other intervention in the treatment of a septic shock. Kumar et al. demonstrated that reducing time to effective antimicrobial treatment was the most important factor in improving overall survival, compared to early fluid resuscitation, single versus multiple drug class antimicrobial therapy and the initiation of inotropic support.

An effective antimicrobial administration within the first hour of documented hypotension was associated with increased survival to hospital discharge in adult patients (79.9%) but each hour of delay over the ensuing 6 hours led to an average decrease in survival of 7.6% (Figure 1). This number declined to 42% if treatment was delayed until the sixth hour.

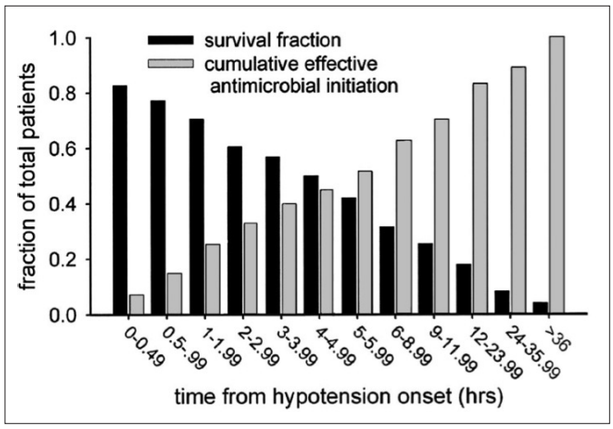

Figure 1: Cumulative effective antimicrobial initiation following onset of septic shock-associated hypotension and associated survival. The x-axis represents time (hrs) following first documentation of septic shock-associated hypotension. Black bars represent the fraction of patients surviving to hospital discharge for effective therapy initiated within the given time interval. The gray bars represent the cumulative fraction of patients having received effective antimicrobials at any given time point. Reproduced with kind permission of Anand Kumar, MD; from (7)

The sepsis source

Where there is presence of a focus of infection, e.g. an abscess, infected necrotic tissue, or generalized peritonitis, this should be evaluated for urgent and adequate source control measures. This includes radiological (i.e. percutaneous drainage) as well as surgical measures in the forms of radical excision and debridement of infected tissue, incision and drainage of an abscess, resection of an involved organ or an amputation (in the case of an unsalvageable limb).

1.4. Tissue hypoperfusion (defined as hypotension despite initial fluid challenge or blood lactate ≥ 4 mmol/L) should be managed with a goal focused approach: infusion of initial minimum of 20 ml/kg of crystalloid (or colloid equivalent) and vasopressors for refractory hypotension to maintain mean arterial pressure (MAP) ≥ 65 mm Hg. Rivers et al. (8) compared a standard-therapy approach (receiving treatment at the clinicians' discretion according to a protocol for haemodynamic support) in the treatment of severe sepsis to an early goal-directed therapy (EGDT).

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Those assigned to the EGDT followed a protocol including frequent boluses of crystalloid to achieve adequate blood pressure and, failing this, vasopressors, with an aim to correct tissue hypoperfusion. These patients also received red blood cell transfusions to achieve a reasonable haematocrit (30%) Patients in the EGDT received a significant higher amount of intravenous fluids within the first 6 hours after the start of therapy (mean of 4981ml compared to 3499ml), as well as red-cell transfusion (64% vs. 18.5%) and inotropic support. This reduced in-hospital mortality in the goal-directed therapy group from 59% to 38%.

1.5. If the patient **remains hypotensive** despite fluid resuscitation (and is therefore in septic shock) a central venous pressure (CV) of > 8mm Hg and central venous oxygen saturation ($ScvO_2$) of > 70% should be achieved.

2. Sepsis management bundle – adds the following recommendations to the goals highlighted above within the first sepsis resuscitation bundle:

2.1. General therapeutic considerations:

2.1.1. Aim Urine output ≥ 0.5 $mL \cdot kg^{-1} \cdot hr^{-1}$: if this cannot be achieved by infusion of crystalloids or colloids, transfusion of packed red blood cells and/or administration of a dobutamine infusion is recommended. Red blood cell transfusion should occur when haemoglobin levels fall below 7.0 g/dL to a target of 7.0-9.0 g/dL. In the absence of bleeding, fresh frozen plasma should not be used to correct laboratory clotting abnormalities. Platelets however should be transfused if the platelet count falls below 5 x 10^9/L regardless of an apparent bleeding.

2.1.2. Vasopressors & Inotropes: A mean arterial pressure (MAP) of above 65 mm Hg should be maintained; recommended vasopressors are norepinephrine or dopamine, an inotropic agent of choice is dobutamine, however as this management is reserved for higher level care in a HDU/ITU (High Dependency Unit / Intensive Treatment Unit) setting we would refer the interested reader to the original guidelines (8).

2.1.3. Stress ulcer prophylaxis: This can be achieved by administration of a H_2 (Histamin H_2 receptor) blocker or a proton pump inhibitor to prevent an upper gastrointestinal bleed.

2.1.4. Deep vein thrombosis prophylaxis: any critically ill patient should receive unfractionated heparin (UFH) or low-molecular-weight heparin (LWMH) unless contraindicated – in that case, mechanical devices (compression stockings, intermittent compression devices) can be used as an alternative.

2.2. Specific therapies and their evidence base:

2.2.1. Low dose steroids: Annane et al (11) postulated that septic shock may be associated with relative adrenal insufficiency – in a randomized controlled trial a 7-day treatment with low doses of hydrocortisone and fludrocortisone significantly reduced the risk of death in patients with septic shock and relative adrenal insufficiency (from 63% in the placebo group to 53% in the group receiving corticosteroids).

2.2.2. Recombinant human activated protein C (Drotrecogin alfa (activated)): Bernard et al (12) published a multicenter trial with human activated protein C that has antithrombotic, anti-inflammatory and profibrinolytic properties. Primary end point being death from any cause, the mortality rate was reduced at 24.7 percent (vs 30.8 percent in placebo group), resulting in a relative risk reduction of 19.4 percent. However the incidence of bleeding was higher in the treatment arm (3.5 vs 2.0 percent, P=0.06).

2.2.3. Glucose control: Van den Berghe et al (13) investigated the effects of intensive compared to standard insulin therapy in critically ill patients on a surgical intensive care unit. Stress induced hyperglycaemia or relative insulin deficiency was previously associated (14, 15) with a higher rate of complications affecting both mortality and morbidity. The authors achieved a reduction in mortality from 8 to 4.6 percent by using an insulin infusion in any patient with a blood glucose level > 6.1 mmol/L, adjusting the infusion to maintain normoglycaemia at 4.4 to 6.1 mmol/L.

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We appreciate that not all of these targets can be met easily, e.g. the initial resuscitation phase involving measurements of CVP (Central Venous Pressure) or central venous oxygen saturations require central access. However, all of the above highlights that *early involvement of critical care* is key element in improving the outcome in severe sepsis with shock. Several of these goals can only be achieved on an intensive care unit and a patient with signs of multi-organ failure ought to be managed in such an environment, ideally incorporating early goal-directed therapy recommendations.

Results from the SSC 2008 were published in 2010 - while some (9) might interpret results regarding certain interventions (e.g. attainment of a central venous pressure, measurement of blood lactate) as not having improved the outcome, Levy et al. (10) state clearly that the Surviving Sepsis Campaign ought to be a performance improvement process rather than a 'dedicated scientific evaluation of the impact of the guidelines on clinical outcome'.

The Importance of Triage

Why is all this so important for triaging in the Emergency Department? Sepsis is one of the commonest reasons for admission to hospital and we can rarely influence patient outcomes as significantly as in this presentation. The majority of patients are admitted to a hospital via an A&E department.

During triage the patient is allocated to a particular care group, based on the haemodynamic parameters and calculated MEWS score. This allocation to the care group along with the MEWS score addresses the potential urgency of a medical assessment (e.g. chest pain, abdominal pain, minor injuries). The early warning score is just one of the early "smoke detectors" at the patient's first stop. As mentioned before a high MEWS triggers a prompt assessment of the patient by a clinician who with an appropriate use of fluids, investigations and early antibiotics ensures patients' safety during their hospitalisation. Furthermore, this may well prevent the patients from eventually requiring the critical care interventions described above, with a poor prognosis due to unnecessary delays in recognition and treatment.

The use of above mentioned care groups in combination with a scoring system ensures that critically ill patients are assessed rapidly. However, all these systems are just triggers to aid triage and identification of the critically ill patients. Constant vigilance and rapid assessment of any patient presenting to an emergency department remains key. This is usually the time point when a physician first encounters the patient and the crucial countdown begins that might well determine the survival.

Score	3	2	1	0	1	2	3
Systolic BP	<70	71-80	81-100	101-180	181-200	201-220	>220
Heart rate (bpm)	<40	—	40-50	51-100	101-110	111-130	>130
Respiratory rate (RR/min)	<8	—	8-11	12-20	21-25	26-30	>30
Oxygen Saturations (%)	<85	>85	>90	>94	—	—	—
Respiratory Support	CPAP BiPAP	>60%	O2	Nil	—	—	—
AVPU	—	—	—	Alert	Voice	Pain	Unconscious
Urine Output (ml/hr)	<80	80-119	120-200	>200	>800	—	—
Pain Score	Severe	Moderate	Mild	None	—	—	—

Table 2: Modified Early Warning Score (MEWS) as per Leeds Teaching Hospitals Trust Guidelines 2011.

Test yourself

1. How do you define sepsis (according to the Surviving Sepsis Campaign)?

1. SIRS and documented source of infection.
2. Infection with refractory hypotension.
3. A condition that can occur in the absence of infection.
4. A patient with a temperature of 38°C plus cough.
5. A high white blood cell count.

2. Which 5 SIRS (Systemic inflammatory response syndrome) criteria are included in the Surviving Sepsis Criteria?

1. Tachypnea, altered mental status, arterial hypertension, thrombocytosis, high serum lactate.
2. Thrombocytopenia, Creatinine increase >0.5 mg/dL or 44.2 µmol/L, plasma CRP >2 SD above the normal value, altered mental status, hypothermia.
3. Hyperglycaemia, leukocytosis, arterial hypotension, acute polyuria, coagulation abnormalities.
4. High serum lactate, increased capillary refill, ileus, hyperbilirubinemia, leucopenia.
5. Significant edema, altered mental status, tachypnea, heart rate < 50 min^{-1}, normal white blood cell count.

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3. Please calculate the Modified Early Warning Score (MEWS) from the following observations: heart rate 105 bpm, blood pressure 95/60 mmHg, respiratory rate 20 RR/min, oxygen saturations 92%, no respiratory support.

1. MEWS = 3
2. MEWS = 4
3. MEWS = 2
4. MEWS = 8
5. A MEWS cannot be calculated as pain score, urine output and AVPU are not given.

4. Your patient is alert with a blood pressure of 85/52 mm Hg, a heart rate of 130 bpm, temperature of 38.8°C, oxygen saturations of 97% (on room air) and a respiratory rate of 18 RR/min. His blood counts show a haemoglobin of 7.8 g/dL and white blood cell count of 19,000 µL-1. His urine dip is positive for leucocytes and nitrites. What is the best course of action?

1. Discuss the case with your registrar.
2. Give a fluid challenge of 500 mL crystalloid and oral antibiotics for a presumed UTI.
3. Give a fluid challenge of 500 mL colloid, IV antibiotics as per trust guidelines for urinary sepsis and crossmatch 2 units to transfuse at the earliest opportunity. Discuss the case with your registrar and consider referring to the critical care outreach team.
4. Start the patient on vasopressors and refer to ITU.
5. Ensure that all possible cultures have been taken, give the patient 750 mL crystalloid and wait for the chest XR until you start antibiotics.

5. What is the overall mortality rate in adult patients with septic shock (infective episode with hypotension)?

1. 34%
2. 45%
3. 52%
4. 56%
5. 68%

Answers

1. Answer: 1
Teaching notes: Sepsis is defined as infection, documented or suspected, and more than one SIRS criteria. SIRS stands for Systemic inflammatory response syndrome and can occur in absence of infection, but the more SIRS criteria present the higher the likelihood of infection. SIRS criteria were defined by Dellinger et al. (2008) and include:

- general variables, e.g. fever, tachycardia, altered mental status
- inflammatory variables, e.g. leukocytosis, high CRP
- haemodynamic values, e.g. hypotension
- organ dysfunction variables, e.g. rise in serum creatinine, hypoxaemia, acute oliguria
- tissue perfusion variables, e.g. high serum lactate, decreased capillary refill

Option 2 is more in keeping with septic shock or sepsis syndrome, depending on whether the infection is confirmed or not. Option 3 is only partly true. Options 4 and 5 are not limited to sepsis, and the differential diagnosis in both scenarios is broad.

2. Answer: 2
Teaching notes: Systemic inflammatory response syndrome criteria are shown in Table 1. Also see: Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008. Critical Care Medicine. 2008 Jan.;36(1):296–327.

3. Answer: 1
Teaching notes: Please refer to the MEWS scoring system – Table 2.

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4. Answer: 3

Teaching notes: The Surviving Sepsis Campaign guidelines, updated 2008, are clear in the fact that intravenous antibiotic therapy should be started as early as possible. An empirical anti-infective therapy is appropriate if the source of sepsis unclear - however, in the case above there is no evidence of a respiratory compromise and one can argue that the positive urine dip is sufficient to start IV antibiotics for presumed urinary sepsis while awaiting further investigations. The patient should receive appropriate fluid resuscitation with either 300-500 mL colloid or ≥ 1000 mL crystalloid as well as blood product administration if haemoglobin levels fall below 7.0 g/dL.

See Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008. Critical Care Medicine. 2008 Jan.;36(1):296–327.

5. Answer: 4

The overall mortality rate in adult patients with septic shock is 56.2%. Administration of antibiotics within the first hour of hypotension was associated with a survival rate or 79.9%, but each hour of delay over the ensuing 6 hours was associated with an average decrease in survival of 7.6%. Also see: Kumar et al. Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock*. Critical Care Medicine. 2006 Jun.;34(6):1589–1596.

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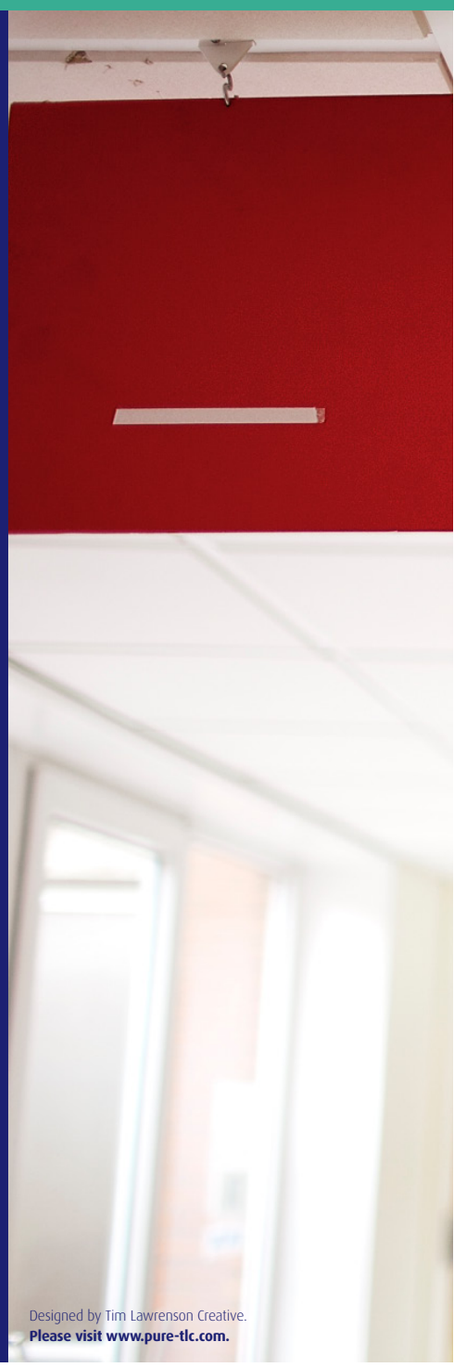

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