"List" = 1-3 words
"State"= short statement/ phrase/ clause

UNIVERSITY HOSPITAL, GEELONG
FELLOWSHIP WRITTEN EXAMINATION
WEEK 18– TRIAL SHORT ANSWER QUESTIONS Suggested answers

PLEASE LET TOM KNOW OF ANY ERRORS/ OTHER OPTIONS FOR ANSWERS

Please do not simply change this document - it is not the master copy !

Question 1 (16 marks)

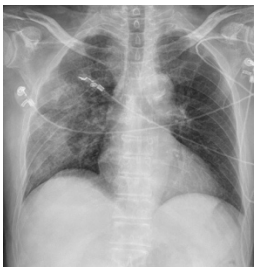
a. State two (2) features of a "single tier" trauma activation system. (2 marks)
- **Activation of full trauma team**
- **Activation based on physiological parameters, anatomical abnormalities or mechanism of injury**

b. State the major limitation of a "single tier" trauma activation system. (1 mark)
- **Over activation when mechanism of injury alone triggers activation - low specificity**(leads to inadequate utilisation of resources and increased workload)

c. State two (2) features of a "two tier" trauma activation system. (2 marks)
- **Graded response with full trauma team activation only with abnormal physiological variables or certain physical signs**
  - **May be prehospital activation or at time of ED assessment**
- **Activation of subset of the full trauma team for other criteria (usually mechanism of injury alone)**
  - **eg. Gen Sx and radiographer alerted, no anaesthetic representative required**

d. State the two (2) major effects of a "two tier" trauma activation system as compared with a one tiered system. (2 mark)
- **↓ unnecessary/ low yield full team callout (esp anaesthetics) and therefore better use resources (improve specificity)**
- **failure of full activation when necessary- with potential delays in diagnosis and/or treatment potentially life threatening injuries**

An 80 year old man is brought to your Tertiary Trauma centre emergency department after being struck by a motorcycle at high speed while walking across a road.
His observations on arrival are: BP 105/80 mmHg HR 105/min RR 36/min Oxygen saturation 85% on 15L/min via non rebreathing mask. GCS 15



e. State three (3) abnormalities shown in this X-ray. (3 marks)
- **# R ribs 2-8 laterally**
- **# in 2 places- flail segment**
- **Increased heterogenous lung opacities right lateral lung consistent with pulmonary contusions**
- **Fracture lateral third right clavicle**
- **Blurring and widening of upper mediastinum**

The patient deteriorates and requires rapid sequence induction and intubation. You have appropriate IV access, but no other management has been performed other than rapid sequence intubation.

f. State six (6) management steps that you would utilise to optimise his ventilation post intubation. (6 marks)
- **R ICC- Insertion right side 28F ICC if USS or CT evidence pneumothorax or haemothorax +/- L sided ICC depending on clinical assessment**
- **NGT- to decompress stomach**
- **Analgesia/Sedation- Ensure adequate e.g. morphine bolus 5mg then 5mg/min infusion, and midazolam 2mg bolus until sedated then 5mg/hr infusion titrated to response**
- **Paralysis e.g. vecuronium 0.1mg/kg if ongoing difficulty with ventilating**
- **Titrate ventilator settings according to physiological response in accordance with lung protective ventilator strategies e.g. tidal volume 6ml/kg, PEEP 5cmH2 increased as necessary**
- **Tilt bed/ nurse at 30° if no contraindication**

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Emergency Medicine (2003) 15, 11–17

Emergency Medicine

SYSTEMS MANAGEMENT SERIES

Trauma systems and emergency medicine

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Abstract

The impact of trauma is a major public health challenge which is likely to escalate in the early 21st century. A systematic approach to this problem is required. This review explains the conceptual framework that defines a trauma system, gives a brief historical perspective and describes some of the essential elements of the system which should make a difference to patient outcome. Emergency physicians are well placed to play a leading role in the development and implementation of trauma systems.

Key words: *emergency medicine, trauma systems.*

Introduction

Trauma presents a huge, multifaceted, global, socio-economic and organizational challenge which acutely affects emergency physicians. In 1990, trauma accounted for 5.1 million deaths, or 10% of global mortality.[1] According to the Global Burden of Disease Study, projected health trends predict that by 2020 injuries from road traffic crashes alone will be the sixth leading cause of death, and that self-inflicted injuries, violence and war will occupy 10th, 14th and 15th place.[2] Injury has been estimated to account for an annual loss of 500 years of productivity per 100 000 in the USA[3] and by 2020 will be the single leading cause of global morbidity accounting for 20.1 million disability-adjusted life years.[2] Injury is also the leading cause of hospital bed day usage and of years of life lost, yet in one of the most developed countries of the world (USA) no more than 4% of National Institute of Health research funds is channelled into trauma research.[4]

Such statistics present a formidable challenge to health care providers in terms of health service research and development, political importance, capital investment and cost-effectiveness, training and evaluation. Regional trauma centres and systems have been proposed as one way forward in the USA,[5] the UK,[6,7] other European countries[8,9] and Australia[10] but such systems have not been universally implemented because of questions of need, efficacy, cost and possibly political enthusiasm. Emergency physicians with their systems-based approach to assessing and managing complex, acute, clinical problems may be in a position to drive trauma care forward especially in areas where the quantity and quality of trauma may not justify the development of highly specialized trauma services.

This review highlights some general principles in the development of trauma systems and important elements of these systems, and serves as a starting point for further discussion.

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Question 2 (12 marks)

a. List the three (3) criteria for a case definition of measles. (3 marks)
   - **Morbilliform rash**
   - **URTI symptoms including conjunctivitis or Koplics spots**
   - **Fever at the onset of the rash**

NB: case definition does not require IgM positivity

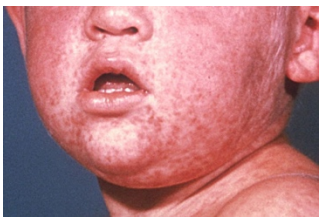
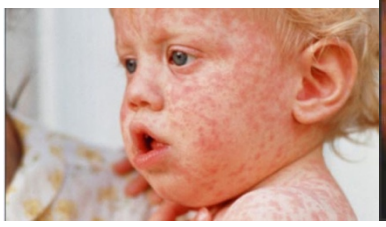
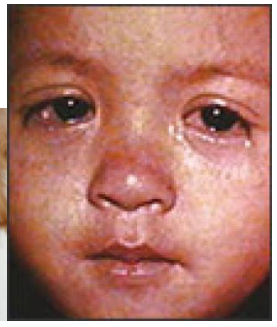
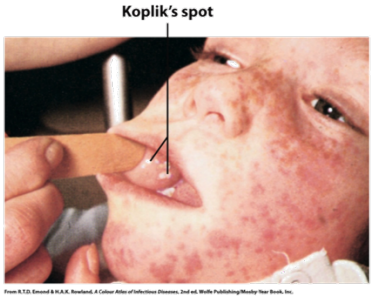
b. List four (4) features of the rash seen in association with measles. (4 marks)
   - **Fever present at onset**
   - **Appears day 3-4**
   - **Florid confluent erythematous, maculopauplar, morbilliform rash**
   - **Starts head, neck, behind ears- spreads to cover entire body- including palms and soles**
   - **Herald clinical recovery**
   - **May desquamate**

c. List four (4) groups of patients that are non susceptible to measles virus. (4 marks)
   - **Infants < 6/12 if mother immune**
   - **1-4 yr olds who have received ≥ 1 measles vaccine**
   - **2 measles vaccinations**
   - **Born < 1966 (natural immunity)**
   - **Documented evidence of laboratory confirmed measles**
   - **Measles IgG present**

You ensure adequate analgesia and hydration for an 8 month old who appears well with a case of measles in your department.

d. List three (3) other key management steps for this patient. (3 marks)
   - **Notification to DHS- REQUIRED**
   - **Place in isolation room** (ideally negative pressure)
   - **Full personal protective equipment for staff** e.g. face mask
   - **Vitamin A (recommended for children < 12 months by WHO)**
   - **Notification to hospital infection control**
   - **Manage at home unless poor oral intake, respiratory compromise or CNS complications**
   - **Advice re- exclude from day care for > 4/7 from appearance of rash**
   - **Inform of potential complications-** Otitis Media, pneumonia watch for drowsiness (Acute post measles encephalitis) **and explain ED return criteria verbal and written**
   - **Full infection clean of room and leave vacant for > 30 min**
   - **Contact tracing with MMR +/- immunoglobulin for susceptible individuals**

The measles rash: NB: No perinasal or perioral sparing. The eyes/nose/lungs "run" continuously (Brassy cough)

Question 3 (13 marks)

It is 1000 hrs in your tertiary, mixed emergency department. You assess 12 month old twins who present with three days of vomiting and diarrhoea. They have both vomited 3 times and had 3 loose bowel actions today. You diagnose viral gastroenteritis for both twins.

a. List three (3) key examination findings that you would use to assess the level of dehydration. (3 marks)
   - **Recent wt change- Bare weight if weight from last 2 weeks available- MANDATORY**
   - **Decreased skin elasticity**
   - **CRT >2 sec/ mottled skin**
   - **Absent tears**
   - **Abnormal respiration- e.g. tachypnoea, deep acidotic breathing**
   - **Dry mucous membranes**
   - **Sunken eyes**
   - **Tissue turgor**
   - **Abnormal radial pulse**
   - **Tachycardia (>150)**
   - **Decreased UO**

Twin 1:.You estimate fluid losses to be about 5% body weight. You estimate his weight to be 10kg.

b. State your approach to management of this child for the first 1 hour. (4 marks)
   - **Attempt oral rehydration with ORS at 200mls (10-20mls/kg) over 1 hour in frequent (5 minutely)** small amounts by cup, ice pole, or syringe, if not tolerated due to flavour dilute juice or lemonade acceptable (although less preferable to ORS)
   - **If ongoing GI losses attempt rapid nasogastric rehydration 250mls/hr for 4 hours** via enteral infusion pump (25ml/kg/hr)
   - **Ondansetron PO 2mg stat dose if ongoing significant vomit and/or slow rate NGT infusion** (occasional vomit acceptable)
   - **Regular antipyretic if ongoing fever leading to lethargy/apathy**
   - **Parenteral education-** (need to qualify, not just "parental education- the Q asked for "state") **-encourage return to breastfeeding/ normal age appropriate diet as soon as able (**no need to exclude/ dilute any food/fluids)

c. Justify your choice for this regime. State two points in you answer. (2 marks)
   - **"Moderate" dehydration- rehydration via enteral route is as effective as the IV route**
   - **Enteral route has:**
     - **fewer complications**
     - **decreased admission rates**
     - **shorter hospital stay**
     - **faster return to normal diet and fluids**
     - **improves symptoms of nausea**
     - **more cost effective when compared to IV rehydration**

NB: Try to focus on clinical efficacy/benefit in a "justification" answer rather than "quick/easy/familiar" etc

Twin 2: You estimate fluid losses to be > 15% body weight You estimate his weight to be 10 kg.

d. State your approach to management of this child for the first hour. Provide four (4) points in your answer. (4 marks)
   - **IV 200ml (20ml/kg) bolus normal saline repeated up to 600 ml (60 ml/kg) intravenous, or until shock is corrected** (if no hepatomegaly or heart disease)
   - **Measure blood glucose and treat hypoglycaemia with 5ml/kg of 10% glucose.**
   - **Once shock corrected, standard intravenous rehydration with 0.9% normal saline + 5% dextrose deficit (1000mls) + maintenance (960mls) at 80mls/hr for 24hours** (in addition to any fluid boluses given to treat shock)
   - **Check UEC, if K < 3 add 20mmol/L KCl, slow rate rehydration of 48hrs if hyper or hyponatraemia**

e. Justify your choice for this regime. (2 marks) State two points in you answer. (2 marks)
   - **"shock" by definition** (> 10% weight loss)
   - **After adequate circulating blood volume has been re-established, ongoing rehydration and maintenance fluids is required with correction of any electrolyte abnormality**

Gastroenteritis- From RCH clinical practice guidelines

This guideline has been adapted for statewide use with the support of the Victorian Paediatric Clinical Network

- Infectious gastroenteritis causes diarrhoea with or without vomiting (non-bilious) or cramping abdominal pain.
- Many cases can be managed effectively with oral rehydration.
- Enteral rehydration is preferable to intravenous hydration.
- Shocked children require urgent resuscitation with 20 mls/kg boluses of IV Normal Saline.
- **Children on fortified formulas need to have their fortification ceased during acute illness.**

Assessment:

Is the diagnosis of gastroenteritis correct?:

Consider important differential diagnoses:

- UTI
- Appendicitis
- Other infections
- Surgical causes of acute abdomen

Consider the diagnosis carefully if there is

- Abdominal pain
- Isolated Vomiting

Are there significant comorbidities /risk factors?

Red flags

The following features may occur in gastroenteritis, but should prompt careful consideration of differential diagnoses and review by a senior doctor:

- severe abdominal pain or abdominal signs
- persistent diarrhoea (> 10 days)
- blood in stool
- very unwell appearance
- bilious (green) vomit
- vomiting without diarrhoea

Children with the following features should be discussed with a senior doctor:

- short gut syndrome
- ileostomy
- complex/cyanotic congenital heart disease
- renal transplant or renal insufficiency
- very young (<6 months)
- poor growth
- use of fortified feeds (concentrated feeds or caloric additives)
- recent use of potentially hypertonic fluids (eg Lucozade)
- other chronic disease
- repeated presentations for same/similar symptoms

Degree of dehydration

Assess on clinical signs and documented recent loss of weight (NB: Bare weight on same scales is most accurate). Weigh bare child and compare with any recent (within 2 weeks) weight recordings. Precise calculation of water deficit due to dehydration using clinical signs is usually inaccurate. The best method relies on the difference between the current body weight and the immediate pre-morbid weight. Unfortunately this is often not available.

Clinical signs of dehydration give only an approximation of the deficit.

Patients with mild (<4%) dehydration have no clinical signs. They may have increased thirst.

Moderate dehydration (4-6%)

- Delayed CRT (Central Capillary Refill Time) > 2 secs
- Increased respiratory rate
- Mild decreased tissue turgor

Severe dehydration (>/= 7%)

- Very delayed CRT > 3 secs, mottled skin
- Other signs of shock (tachycardia, irritable or reduced conscious level, hypotension)
- Deep, acidotic breathing
- Decreased tissue turgor

Other 'signs of dehydration' (such as sunken eyes, lethargy & dry mucous membranes) may be considered in the assessment of dehydration, although their significance has not been validated in studies, and they are less reliable than the signs listed above.

Unless an accurate & recent loss of weight is available as a guide, calculating percentage weight loss by clinical signs is only an estimation.

Deficit

A child's water deficit in mls can be calculated following an estimation of the degree of dehydration expressed as % of body weight. (e.g. a 10kg child who is 5% dehydrated has a water deficit of 500mls). The deficit is replaced over a time period that varies according to the child's condition. Precise calculations (eg 4.5%) are not necessary. The rate of rehydration should be adjusted with ongoing assessment of the child.

Replacement of deficit

Replacement may be rapid in most cases of gastroenteritis (best achieved by oral or nasogastric fluids), but should be slower in diabetic ketoacidosis and meningitis, and much slower in states of hypernatraemia (aim to rehydrate over 48 hours, the serum sodium should not fall by >1mmol/litre/hour).

Ongoing losses (eg from drains, ileostomy, profuse diarrhoea)

These are best measured and replaced - calculations may be based on each previous hour, or each 4 hour period depending on the situation. (eg. 200ml loss over previous 4 hours becomes replacement of 50ml/hr for the next 4 hours.)

Replacement of ongoing losses

Normal (0.9%) saline may be sufficient, or 5% albumin may be used if sufficient protein is being lost to lower the serum albumin. See Burns guideline for additional losses from burns.

Investigations:

In most children with gastroenteritis **no investigations** are required.

Faecal samples may be collected for bacterial culture if the child has significant associated abdominal pain or blood in the faeces, as a bacterial cause of gastroenteritis is more likely. However, these results usually don't alter treatment. Extensive testing for viral and bacterial causes is expensive and usually does not influence treatment.

Some viruses (e.g. enteroviruses) and other organisms (e.g. *Dientamoeba fragilis* and *Blastocystis hominis*) can be found in the stools of healthy individuals and their detection does not change the management. Testing is usually not indicated. Consider stool microbiological investigations if:

- the diarrhoea has not improved by day 7, particularly if the child has recently been abroad
- you suspect septicaemia
- there is blood and/or mucus in the stool, particularly if protracted or the child is systemically unwell
- the child is immunocompromised.

Blood tests (electrolytes, glucose) are not necessary in simple gastroenteritis but are required for children with:

- severe dehydration
- renal disease or diuretic use
- altered conscious state
- 'doughy' skin (suggesting hypernatraemia)
- home therapy with excessively hypertonic fluids (eg homemade solutions with added salt) or excessively hypotonic solutions (eg prolonged plain water or diluted formula)
- profuse or prolonged losses
- ileostomy

Acute management:

Ondansetron

- Not recommended for children < 6 months old or < 8kg
- Should only be administered once in this setting.

Anti-diarrhoeals are not recommended.

Oral rehydration

- **Lemonade, homemade oral rehydration solutions (ORS) and sports drinks are not appropriate fluids for rehydration**
- Stop any feed fortifications (such as extra scoops of formula or Polyjoule)
- Encourage parents to find methods to help children drink. Eg: cup, icypole or syringe, aiming for small amounts of fluid often.
- Continue breastfeeding.
- Suggest ORS eg. GastrolyteTM, HYDRAlyteTM, PedialyteTM
- Early feeding (as soon as rehydrated) reduces stool output, and aids gastrointestinal tract recovery.
- Recommend usual diet once rehydrated.
- If diarrhoea worsens in setting of formula feeding, consider the temporary (2 weeks) use of lactose free formula.

Trial of oral fluids in the emergency department:

- Most children with mild/no dehydration can be discharged without a trial of fluids after appropriate advice and follow-up arranged.
- **Aim for 10-20 mls/kg fluid over 1 hour of ORS;** give frequent small amounts.
- Significant ongoing GI losses (frequent vomiting or profuse diarrhoea) minimises the chance of success at home.

Consider early NGT rehydration in these children.

Nasogastric Rehydration (NGTR)

- Nasogastric rehydration is a safe and effective way of rehydrating most children with moderate dehydration, even if the child is vomiting. It is preferred over the IV route.
- **Most children stop vomiting after NGT fluids are started.** If vomiting continues, consider ondansetron and slow NG fluids temporarily.
- Use ORS eg. GastrolyteTM, HYDRAlyteTM, PedialyteTM .
- **This is not applicable to children with dehydration from respiratory illnesses eg bronchiolitis or with hypernatremia who require a tailored rehydration plan** [insert link to bronchiolitis, hypernatremia guideline]

Rapid nasogastric rehydration:

- 25ml/kg/hr for 4 hours
- Suitable for the majority of patients with gastroenteritis and **moderate dehydration** (see indications for 'slower' NGR and indications for IV rehydration below)
- To calculate hourly rate see table 2:

Slower rehydration is preferred for the following patients:

- Infants < 6 months
- Comorbidities present.
- Children with significant abdominal pain.

Replace deficit over first 6 hours and then give daily maintenance over the next 18 hours
The calculated amounts do not need to be modified for exact degree of dehydration and should be used for patients with moderate or severe dehydration based on clinical signs.
* RCH enteral pumps deliver a maximum of 300 ml/hr;
** ie residual maintenance delivered over shorter time course

Ongoing profuse losses during NGT rehydration:

- If vomiting continues consider ondansetron and slow NG fluids temporarily.
- For patients who continue to have **significant vomiting** (2 large vomits in 1 hour) or **significant abdominal pain** during NGTR, re-examine the patient to exclude differential diagnoses including development of ileus. If satisfied with examination, then halve rate of NGT fluids.
- If vomiting continues despite halved rate or **profuse ongoing diarrhoea**, consider
  - Slower NGTR
  - IV fluids

Intravenous rehydration

Indications:

- Current evidence suggests NGTR is safer and more effective but IV rehydration is indicated for severe dehydration and if NGTR fails (eg. ongoing profuse losses or abdominal pain).
- Also suitable for children who already have an IV insitu.
- Certain comorbidities, particularly GIT conditions (eg. short gut or previous gut surgery) - discuss these patients with senior staff.

IV Fluids see guideline:

- **If shocked: 20ml/kg 0.9% sodium chlodride (normal saline)** boluses, repeated until shock is corrected. **If > 40 ml/kg boluses required, see shock guideline {link}**
- Measure blood glucose and **treat hypoglycaemia with 5ml/kg of 10% glucose**.
- **Rapid IV Rehydration:** In older children > 4 years with moderate dehydration with no comorbidities, no electrolyte disturbance and no significant abdominal pain, consider **10 ml/kg/hr (up to 1000ml/hr) for 4 hours of Plasma-Lyte 148 and 5% Glucose OR 0.9% sodium chloride (normal saline) and 5% Glucose, then reassess.**
- **Standard IV Rehydration:** Otherwise, rehydrate at the rates in Table 4 below for the first 24 hours.
- **Use Plasma-Lyte 148 and 5% Glucose OR 0.9% sodium chloride (normal saline) and 5% Glucose** for rehydration after any required boluses. If serum K < 3mmol/L, add KCl 20mmol/L, or give oral supplements.
- **Measure Na, K and glucose** at the outset and at least 24 hourly from then on (more frequent testing is indicated for patients with comorbidities or if more unwell). **Venous blood gases** provide rapid results. It is not necessary to send an electrolyte tube to the lab unless measurement of urea or creatinine is clinically indicated.
- Consider septic work-up or surgical consult in severely unwell patients with gastroenteritis.

After 1st 24 hours, if needed, use Standard Intravenous Fluids unless abnormal ongoing losses or electrolyte disturbance.

Sodium abnormalities

- If serum sodium is taken and is <135mmol/l or >145mmol/l see Hypernatremia guideline or Hyponatremia guideline.

Monitoring of rehydration

- Bare weigh patient 6 hourly in moderate and severe dehydration, who are receiving NGTR or iv fluids.
- Carefully reassess after 4-6 hours, then 8 hourly to guide ongoing fluid therapy. Look particularly for:
  - weight change
  - clinical signs of dehydration
  - urine output
  - ongoing losses
  - signs of fluid overload, such as puffy face and extremities.

Discharge after RAPID nasogastric rehydration:
Medical review before discharge required if:

- < 4% wt gain
- Signs of dehydration or otherwise unwell
- ≥ 3 large stools during rehydration
- Abdominal pain worsening
- Advice and Gastroenteritis Fact Sheet should be given to parents before discharge. Encourage review the next day with the GP.

Consider consultation with local paediatric team when:

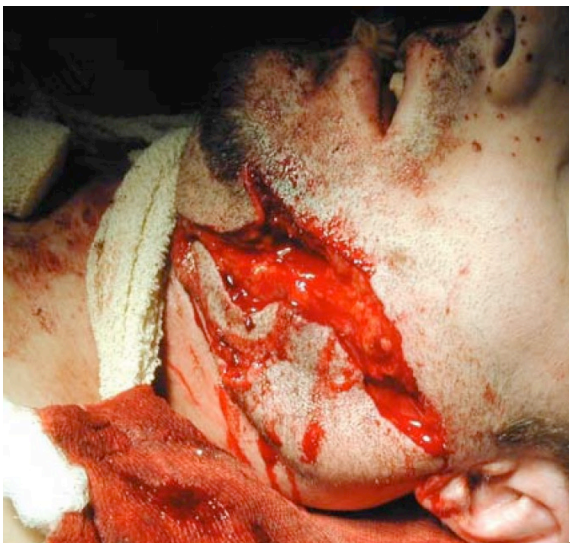
- Risk factors identified
- Electrolyte abnormalities
- Diagnosis in doubt
- Assessed as severe dehydration

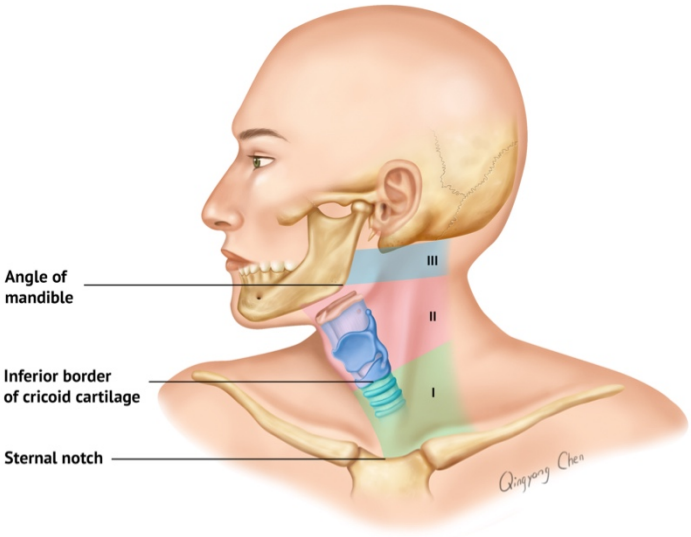
Consider transfer when:

- severe electrolyte abnormalities
- severe dehydration or shock

Question 4 (12 marks)

A 25 year old man has been brought to your emergency department after sustaining a knife wound to his neck in an assault. His vital signs and GCS are normal.





a. State four (4) important features of the injury shown in the photo. (4 marks)
   - **injury involves all 3 zones of the neck**
   - **deep injury likely to have breached platysma**
   - **extends from anterior midline to lateral ear lobe, therefore potentially involves all major neck structures (aerodigestive, vascular, nervous system)**
   - **large haematoma present**

b. List four (4) deep structures that may be injured in this patient. (4 marks)
   - **Airway: Trachea and larynx**
   - **Vascular e.g. carotid artery and jugular veins**
   - **Neurological e.g. phrenic nerve (lies on anterior scalene) or recurrent pharyngeal nerve (near thyroid)**
   - **Oesophagus**

c. State four (4) key features on history that you would obtain. (4 marks)
   - **details weapon/s: number, type, length, edge, site stabbings**
   - **airway issues e.g. noise breathing, hoarseness**
   - **breathing difficulties e.g. shortness of breath, pleuritic CP**
   - **circulation issues e.g. lightheadedness, blood lost, collapse**
   - **neurological symptoms e.g. focal weakness or parasthesia**

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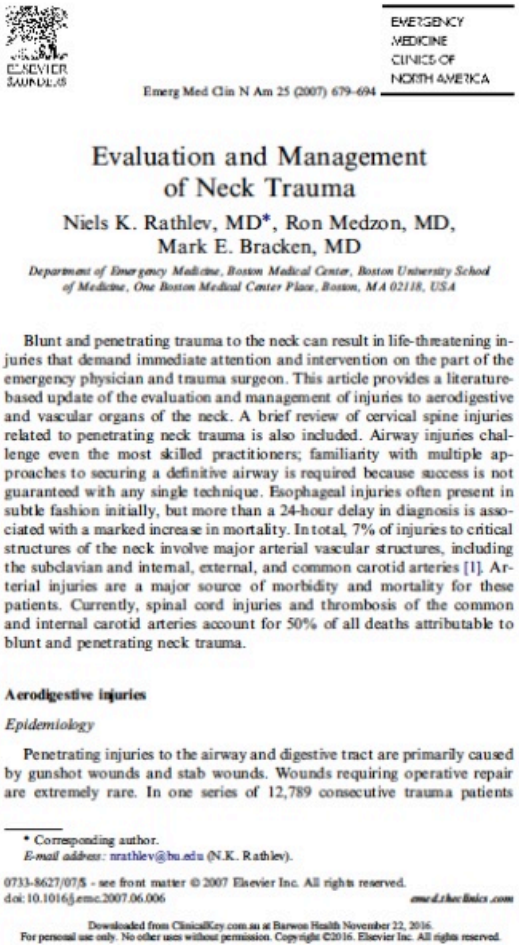
These 2 articles have a great summary of the important issues



EMERGENCY MEDICINE CLINICS OF NORTH AMERICA

Emerg Med Clin N Am 25 (2007) 679–694

Evaluation and Management of Neck Trauma

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Blunt and penetrating trauma to the neck can result in life-threatening injuries that demand immediate attention and intervention on the part of the emergency physician and trauma surgeon. This article provides a literature-based update of the evaluation and management of injuries to aerodigestive and vascular organs of the neck. A brief review of cervical spine injuries related to penetrating neck trauma is also included. Airway injuries challenge even the most skilled practitioners; familiarity with multiple approaches to securing a definitive airway is required because success is not guaranteed with any single technique. Esophageal injuries often present in subtle fashion initially, but more than a 24-hour delay in diagnosis is associated with a marked increase in mortality. In total, 7% of injuries to critical structures of the neck involve major arterial vascular structures, including the subclavian and internal, external, and common carotid arteries [1]. Arterial injuries are a major source of morbidity and mortality for these patients. Currently, spinal cord injuries and thrombosis of the common and internal carotid arteries account for 50% of all deaths attributable to blunt and penetrating neck trauma.

Aerodigestive injuries

Epidemiology

Penetrating injuries to the airway and digestive tract are primarily caused by gunshot wounds and stab wounds. Wounds requiring operative repair are extremely rare. In one series of 12,789 consecutive trauma patients

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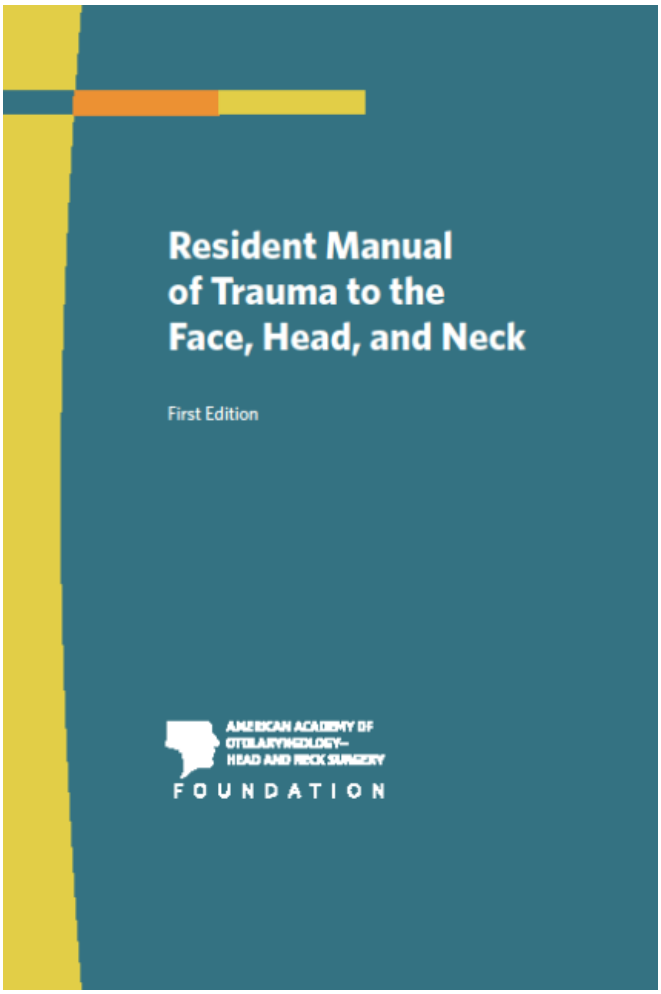
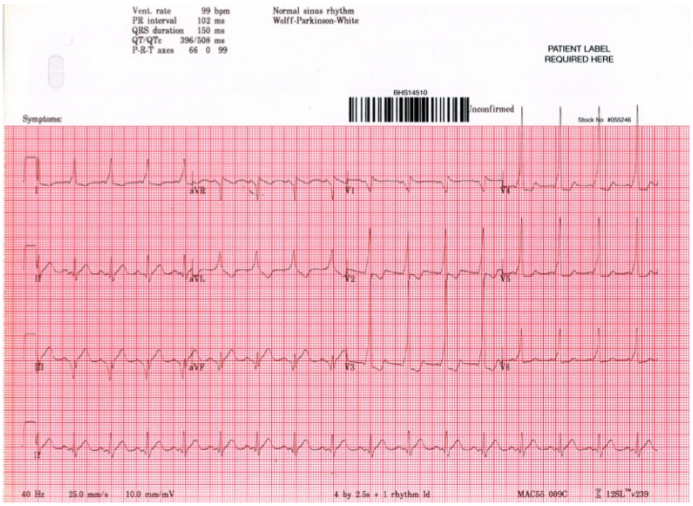


Question 5 (12 marks)

A 50 year old man presents following an episode of palpitations and syncope. At the time of the ECG shown he is asymptomatic.



a. State three (3) abnormalities shown in this ECG. (3 marks)
   - **Short PR interval < 80ms**
   - **Broad QRS complex approx 120ms with delta wave consistent with Wolff Parkinson White** (type B)
   - **q wave II, III and aVF, aVR and V1**
   - **ST segment depression in lateral leads 1mm aVL, V5, V6 2mm V2-V4**
   - **T wave inversion high lateral leads I and aVL**

The patient experiences palpitations and is noted to be in atrial fibrillation on the monitor at a rate of 160. Other than palpitations, he is asymptomatic and appears well. His BP is 150/85. There are no other new changes to his ECG. The rhythm persists. He is placed in a resuscitation cubicle with full monitoring applied.

b. State four (4) steps in his ongoing management over the next 20 minutes. (4 marks)
   - **Attach defibrillation pads in anterior-posterior location and turn defibrillation machine on**
   - **As haemodynamically stable- acceptable 1st line options :**
     - **flecanide 150mg IV over 30 minutes** (appropriate if normal echo and no known coronary artery disease, whilst preparing for electrical cardioversion)

     OR:
     - **procedural sedation and electrical cardioversion**
   - **If becomes haemodynamically unstable- procedural sedation and electrical cardioversion**
   - **Synchronised biphasic DC shock 150J, increased to 200J if first shock unsuccessful**
   - **Optimise electrolytes in particular potassium and magnesium aiming K > 4.0 and Mg > 1.0 mmol using 10mmolKCL or MgSO4 over 30 minutes intravenous**
   - **Cardiology review- will require a period of observation in monitored bed as inpatient**
   - **Ensure no other reversible cases e.g. digoxin toxicity**

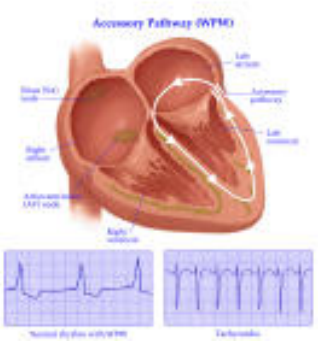
c. State three (3) points to justify your selected management approach. (3 marks)
   - **AF in WPW may progress to VF at any stage**
   - **Therefore rhythm control (vs rate control) strategy indicated**
   - **Current haemodynamic stability does not reassure**
   - **Flecainide is the DOC (in structurally normal heart without CAD)**
   - **If information re structural normality and presence of CAD is not known→ Rx of choice is DCR**

WPW

- Syndrome of supraventricular tachyarrhythmias → accessory pathway
- 1% of population

Pathophysiology:

- Accessory connections between the atrium and ventricle are the result of anomalous embryonic development of myocardial tissue bridging the fibrous tissues that separate the 2 chambers.
- Although dozens of locations for bypass tracts can exist in preexcitation, including atriofascicular, fasciculo ventricular, intranodal, or nodoventricular, **the most common bypass tract is an accessory atrioventricular (AV) pathway otherwise known as a Kent bundle. This is the anomaly seen in WPW syndrome.**
- Conduction through a Kent bundle can be anterograde, retrograde, or both.



ECG

- Delta waves → depolarisation of free ventricular wall

LOCATION		V1	V2	QRS axis
left posteroseptal (type A)	60%	+ve	+ve	left
right lateral (type B)	30%	-ve	-ve	left
left lateral (type C)		+ve	+ve	inferior (90 degrees)
right posteroseptal	8%	-ve	-ve	left
anteroseptal		-ve	-ve	normal

- Orthodromic → conduction in 95% of SVT in accessory PW (ie down usual PW, then reenters ∴ narrow QRS)
  ∴ normal pathway for ventricular depolarization, accessory tract is used for reentry
  ECG → delta wave absent, QRS complex is normal, and P waves inverted in inf & lat leads.
- Antidromic → 5%
  Less commonly, shorter refractory period in the accessory tract may cause block of an ectopic atrial impulse in the normal pathway, with anterograde conduction down the accessory tract and then retrograde reentry of the normal PW
  QRS is wide, which is an exaggeration of the delta wave during sinus rhythm (ie, wide-QRS tachycardia)
- Thus, the mechanism underlying the majority of the tachycardias in patients with WPW syndrome is macroreentry caused by anterograde conduction over the AV node His bundle pathway and retrograde conduction over an accessory pathway (orthodromic). Less common in patients with WPW syndrome is antidromic tachycardia. Even when the accessory pathway conducts only in retrograde fashion, it can still participate in the reentrant circuit and produce an orthodromic AV reciprocating tachycardia.

DDx → Lown-Ganong-Levine (LGL) syndrome, patients have a short PR interval and SVT, but no delta wave

Management

- **Haemodynamically unstable →** DCR→ dose controversial (in gen. the fibrillations need ↑↑ doses) → M 100, B 150
  Electrical shock depolarizes all excitable myocardium, lengthens refractoriness, interrupts reentrant circuits, discharges foci, and establishes electrical homogeneity that terminates reentry
- **Critical in WPW is narrow vs wide & regular vs irregular**
  SVT ie narrow complex, regular - Rx as normal AVNRT (ie SVT) → vagal, IV adenosine, verapamil (NB AF may occur ∴ have DCR ready)
- **AF / wide complex tachycardia** → chaotic, rapid, QRS morphology may be all over the place
  AF in WPW is: 1) potentially serious 2) the deadliest arrhythmia
  →normal rate limiting effects of AV node bypassed.
  → anterograde conduction via accessory pathway
  ∴ ↑ventricular rates may lead to VF

Medical emergency- need to cardiovert AF → SR URGENTLY : IV Flecainide/ DC cardioversion are the Rx of choice
Digitalis shortens refractoriness in myocardium and bypass tract ∴may accelerate ventricular response in AF
→ lignocaine does not ↑ refractoriness of access. tract

OPD Mx

- Drug Rx
  - Class IA (eg, procainamide) and class IC (eg, flecainide, propafenone) block conduction in the accessory pathway.
  - Amiodarone and sotalol affect both AV node & bypass tract. Work in similar fashion but block only bypass tract.
  - Class IA and IC that prolong the refractory period in the bypass tract are indicated if drug therapy becomes necessary.
  - Class IC and IA drugs are best used in conjunction with an AV node blocker, such as metoprolol or verapamil.
  - Digoxin is CI in patients with WPW syndrome. Most deaths from WPW syndrome have been associated with digoxin
- Electrical ablation
- Surgical ablation

→ The best plan is to not use drugs at all; instead, refer all patients who have symptomatic WPW syndrome for ablation because this cures the tachycardia and eliminates the potential dangerous effects of drugs.

Question 6 (12 marks)

A 47 year old man arrives via ambulance with lethargy, extreme shortness of breath and wheeze despite IV adrenaline. He has a past history of poorly controlled asthma and morbid obesity. You decide to intubate him soon after arrival.

			Reference range
FiO_2	**0.50**		
pH	**7.12**		**(7.35- 7.45)**
pCO2	**80**	**mmHg**	**(35-45)**
PO2	**246**	**mmHg**	**(80- 95)**
Bicarbonate	**18**	**mmHg**	**(22-28)**
Base excess	**- 14**		**(-3 - +3)**
O2 saturation	**99**	**%**	**(> 95)**

a. Provide two (2) calculations to help you to interpret these results. (2 marks)

Derived value 1: **Expected CO_2**: PCO_2 = 1.5 x HCO3- + 8 = 33 but PCO_2 is 80

Derived value 2: **A-a gradient** PiO2= (atmospheric pressure - partial pressure of water) x FiO2

760 - 47 at sea level x ~ (0.21= RA)

PiO2 = 713x 0.5= 356

PAO2= (FIO2) (P_{atm} – 47 mm Hg) - (P_aCO2)/ 0.8

PAO2 = 356- 80/0.8= 356- 100 = 256

∴ A-a gradient = PAO2- Pa02 = 256- 246 = 10 which is normal

∴ suggests no shunt, diffusion limitation or V/Q mismatch

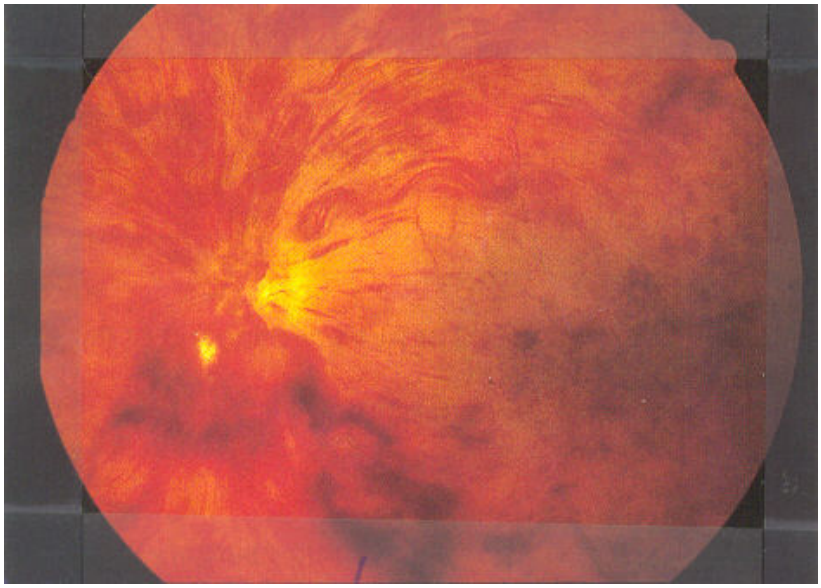
b. Using the scenario and the derived values, define the secondary acid/base abnormality/s. (2 marks)
- **Primary respiratory acidosis**
- **Primary metabolic acidosis**

c. Provide a unifying explanation for these gases in this clinical context. State three (3) points. (3 marks)
- **Type 2 respiratory failure (hypercapnic respiratory failure)**
- **Probably acute on chronic respiratory acidosis**
  - **acute likely exacerbation of airways disease +/- due to poor compliance/ precipitant/ infection**
  - **chronic secondary to ? COPD & obesity hypoventilation**
  - **potential that patient is tiring and hypoventilating if prolonged pre hospital period**
- **metabolic acidosis secondary to:**
  - **lactic acidosis 2˚ to adrenaline/ salbutamol or poor peripheral perfusion**
- **Severe acidosis- likely acute on chronic respiratory acidosis and metabolic acidosis**
  - **If simple metabolic acidosis expect: PCO_2 = 1.5 x HCO3- + 8 = 33, but PCO_2 is 80**
  - **If simple respiratory acidosis, expect $HCO3^-$ ↑**

d. State three (3) key aims in your support of this patients' ventilation. (3 marks)
- **<u>Ensure adequate oxygenation:</u> Given PaO2 on 50 %→ air mix reasonable,↑ to 100% oxygenation if deterioration**
- **<u>Limit peak pressures:</u> "Auto- PEEP" often present, avoid PEEP**
- **allow "permissive hypercarbia" (key phrase), if required, to avoid high PIPS (keep < 40)**
- **Inspiratory time titrated to keep PIPS < 55 mmHg**
- **<u>Avoid gas trapping</u>: Low I:E ratio (ideally < 1:3)**
- **RR titrated to lowest tolerable pH (eg ~ 10 bpm)**
- **Minimise Vt to avoid alveolar distension (eg. < 10 ml/kg- care with estimations as obese, use lean weight estimation or estimated Vt will be too high)**
- **Ensure adequate sedation and muscle relaxation (paralyse to allow intercostal relaxation eg. vecuronium)**
- **Volume cycled ventilator or hand ventilate- NOT time cycled ventilators (have long insp/ short exp phases)**
- **Continuous nebulised salbutamol may be required (high dose- ETT ↓ aerosol delivery)**
- **reverse trendelenberg as obese**
- **Consider inhalation anaesthesia for maintenance of anaesthesia (bronchodilator properties)**

Question 7 (10 marks)

A 75 year old female presents with gradual loss of vision in her left eye.



a. What is the diagnosis for this patient? (1 mark)
   - **Central retinal vein occlusion**

b. List five (5) likely underlying causes for this diagnosis in this patient. (5 marks)
   - **DM**
   - **HT**
   - **Hyperviscosity**
   - **Glaucoma**
   - **Atherosclerosis**

c. List four (4) key pieces of information that you would provide the patient. (4 marks)

- **<u>Further investigations</u> required: CT +/- angiography to assess circulation**
- **<u>Treatment:</u>**
  - **Nil acute**
  - **Specialist Rx (photocoagulation) aimed at preventing new vessels and 2˚ glaucoma**
  - **Diagnosis and Rx of the underlying condition**
- **<u>Prognosis:</u>**
  - **Poor despite best Rx- REQUIRED**
  - **Likely marked ↓ vision currently** (potentially light perception only)**, probably will not improve**
  - **Other eye at risk**

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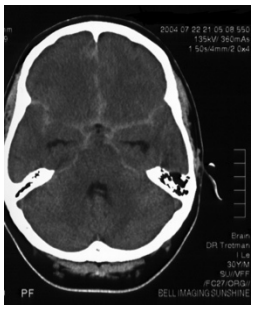
November 2017

Question 8 (12 marks)

You are in a regional emergency department. A registrar approaches you for assistance with one of his patients. The patient is a 30 year old man who presented with a headache.



a. State two (2) abnormalities shown in this CT scan. (2 marks)
   - **Subarachnoid haemorrhage with hyper attenuating material (blood) around the circle of Willis, inter-hemispheric fissure, and sylvian fissure.**
   - **Temporal horns visible raising possibility obstructive hydrocephalus**
   - **Loss grey-white matter differentiation suggestive raised intracranial pressure**

b. List four (4) features on examination that would indicate severe disease. (4 marks)
   - **Stupor, vegetative state, hemiparesis-** *Features suggestive grade 4 or 5 bleed according to Hunt and Hess grading system*
   - **GCS 3-6 with motor deficit-** *Features suggestive grade 4 or 5 according to World Federation of neurosurgeons*
   - **Decerebrate or decorticate posturing**
   - **Cushings response with bradycardia and hypotension**

Hunt & Hess Based on conscious state, severity of HA & neurological deficit	Grade I Asymptomatic, minimal HA, mild nuchal rigidity Grade II Mod→ severe HA, nuchal rigidity, No neurol (besides CN palsy) Grade III Drowsiness, confusion, mild focal deficit Grade IV Stupor, hemiparesis, vegetative Grade V Deep coma, decerebrate, moribund Grade I & II → independent 90-95% Grade IV-V → independent 10%, dead 80%
World Federation of Neurosurgeons based on most important factors to outcome are level of consciousness & hemiparesis	Grade I GCS 15, no motor deficit Grade II GCS 13-14, no motor deficit Grade III GCS 13-14, with motor deficit Grade IV GCS 7-12, +/- motor deficit Grade V GCS 3-6, +/-motor deficit

You review the patient. He reports 8/10 on pain score for headache. He has been given 10mg oxycodone only prior to your review. He has 2 large bore IV access and full non invasive monitoring in situ and is in a resuscitation cubicle.
His observations are: BP 220/100 mmHg HR 90bpm Temperature 36.8°C Oxygen saturation99% on room air GCS15

c. List three (3) key steps in your management of this patient over the next 30 minutes. State one (1) point of detail for each management choice. (6 marks)

Management step (3 marks)	**Details (3 marks)**
Analgesia	Morphine 5mg bolus then 2.5mg aliquots intravenous titrated to sedation level and reduction pain score
Blood pressure control	Labetalol 10mg repeated 10 minutely IV then infusion at 1mg/min, endpoint sBP 140-160mmHg, in consultation with neurosurgeon
Minimise further increased in ICP	Nurse head up 30 degrees Prevent secondary injury (hypoxia, hyperthermia, abnormal glucose) Consider hyperosmolar therapy e.g. mannitol 0.5mg/kg IV Consider prophylactic anticonvulsant e.g. levetiracetam 500mg IV over 15 minutes Ondansetron 8mg intravenous TDS
Disposition planning	Urgent neurosurgical referral then retrieval service for immediate transfer to tertiary centre

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The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

MEDICAL PROGRESS

Cerebral Aneurysms

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N Engl J Med 2006;355:928-39.

SACCULAR INTRACRANIAL ANEURYSMS, ABNORMAL FOCAL OUTPOUCHings of cerebral arteries, cause substantial rates of morbidity and mortality. Recently, major changes have occurred in the way we think about and treat this disease. Previous concepts about the natural history, particularly the risk of rupture of certain aneurysms, have been challenged.[1,2]

When this topic was the subject of a Medical Progress article in the *Journal* in 1997, minimally invasive percutaneous endovascular treatment of intracranial aneurysms (a technique known as coiling) had been introduced as an experimental procedure for patients who were not good candidates for surgery.[3] After almost a decade of increased use and evaluation, endovascular coiling has proved to be a safe and durable alternative to the traditional neurosurgical treatment of craniotomy and clip ligation ("clipping"). Coiling has now surpassed clipping as the primary method of treatment for intracranial aneurysms in some centers. Since the Guglielmi detachable coil for the treatment of intracranial aneurysms was approved in 1995 by the Food and Drug Administration (FDA), an estimated 150,000 patients have been treated with this device.[4,5] In this article, the technological advances and supporting research contributing to this important change in practice patterns are reviewed.

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Intracranial aneurysms are common lesions; autopsy studies indicate a prevalence in the adult population between 1 and 5 percent,[6] which translates to 10 million to 12 million persons in the United States.[3] Fortunately, most aneurysms are small, and an estimated 50 to 80 percent of all aneurysms do not rupture during the course of a person's lifetime.[6] Intracranial aneurysms are considered to be sporadically acquired lesions, although a rare familial form has been described.[3] Associated conditions include autosomal dominant polycystic kidney disease, fibromuscular dysplasia, Marfan's syndrome, Ehlers–Danlos syndrome type IV, and arteriovenous malformations of the brain. An estimated 5 to 40 percent of patients with autosomal dominant polycystic kidney disease have intracranial aneurysms,[6,7] and 10 to 30 percent of patients have multiple aneurysms.[3] Screening with intracranial magnetic resonance angiography is indicated for people who have two immediate relatives with intracranial aneurysms and for all patients with autosomal dominant polycystic kidney disease.[3,8,9] Rescreening of patients with autosomal dominant polycystic kidney disease is recommended, although the frequency of the procedure depends on whether other affected family members are known to have intracranial aneurysms.[8]

The estimated incidence of subarachnoid hemorrhage from a ruptured intracranial aneurysm in the United States is 1 case per 10,000 persons, yielding approximately 27,000 new cases of subarachnoid hemorrhage each year.[3,5] Subarachnoid hemorrhage is more common in women than in men (2:1)[10]; the peak incidence

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ORIGINAL RESEARCH

Sensitivity of proposed clinical decision rules for subarachnoid haemorrhage: An external validation study

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Abstract

Objectives: Subarachnoid haemorrhage (SAH) is an uncommon but important cause of sudden-onset headache. Three clinical decision rules (CDRs) for investigation in sudden headache have been proposed, but concerns were raised about the generalisability of some variables. Our aim was to determine what proportion of patients with confirmed SAH has the identified high-risk factors and the sensitivity of the proposed CDR in an Australasian cohort.

Methods: This is a retrospective cohort study of alert and neurologically intact adult patients with confirmed SAH attending two community teaching hospitals between 2000 and 2011. The outcomes of interest were the proportion of patients with each high-risk criterion (descriptive statistics) and sensitivity of each proposed CDR (%, interquartile range [IQR]).

Results: There were 59 confirmed SAH that met the inclusion criteria. Sensitivity of proposed CDR 1 was 96.6% (95% confidence interval [CI] 88.5–99.1%), sensitivity of proposed CDR 2 was 100% (95% CI 93.8–100%) and sensitivity of proposed CDR 3 was 89.8% (95% CI 79.5–95.3%). The addition of vomiting to the criteria in CDRs 1 and 3 increased the sensitivity of both these CDRs to 100%.

Conclusion: CDR 2, or the refinement of CDRs 1 and 3 with the inclusion of at least one episode of vomiting as a criterion, has very high sensitivity. Although unlikely to reduce CT scan rates for patients in whom there is a clinical suspicion of SAH, they might be useful in guiding which patients require further testing (e.g. lumbar puncture) after a negative CT scan.

Key findings

- Proposed clinical decision rule 2 had 100% sensitivity for subarachnoid haemorrhage.
- With the addition of vomiting to the criteria, proposed clinical decision rules 1 and 3 achieved 100% sensitivity for subarachnoid haemorrhage.
- In this study, all cases of subarachnoid haemorrhage were diagnosed on CT scan.

Key words: *clinical decision rule, headache, subarachnoid.*

Introduction

Subarachnoid haemorrhage (SAH) is an uncommon but potentially life-threatening cause of headache presenting to EDs.[1–4] For patients with altered conscious state or neurological deficit, the decision to investigate is easy. Alert, neurologically intact patients pose the challenge. Investigation is time consuming and not without risk; however, a missed diagnosis of SAH can have catastrophic consequences.[4] Investigation for suspected SAH includes non-contrast head CT and, if that is negative, a lumbar puncture (LP) is recommended. The vast majority of CT scans (>95%) are normal,[2] and it can be hard to distinguish a traumatic tap from true SAH on LP.[5] Ideally, we would only investigate higher-risk patients where the risks and inconvenience of investigation were outweighed by the risks of the potential illness.

Perry *et al.*,[6] in a large prospective trial, have identified factors that are associated with high risk of SAH and have proposed that these might form the basis of an accurate clinical decision rule (CDR) regarding the need for investigation for SAH in patients with acute headache. The factors identified were age >40 years, complaint of neck pain or stiffness, witnessed loss of consciousness, onset with exertion, arrival by ambulance, vomiting at least once, diastolic BP >100 mmHg and systolic BP >160 mmHg. Three draft CDRs were developed for further testing. These are shown in Table 1. For each rule, a patient would be investigated if one or more of the criteria are present. In derivation, each

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FOR DEBATE

Spectrophotometry, not visual inspection for the detection of xanthochromia in suspected subarachnoid haemorrhage: A debate

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The case for

Kevin H Chu

The measurement of bilirubin in the cerebral spinal fluid (CSF) for the diagnostic work-up of suspected subarachnoid haemorrhage (SAH) is informed by basic science. Following a spontaneous SAH, red blood cells (RBCs) are lysed in the CSF. Free haemoglobin is converted to oxyhaemoglobin, which is then metabolised by macrophages to bilirubin. The former reaction occurs *in vitro* and *in vivo*. The latter reaction occurs only *in vivo*, with bilirubin appearing some 6 to 12 h post-haemorrhage.[1]

The presence of bilirubin resulting in yellow discolouration of the CSF is the contemporary definition for xanthochromia.[2] The finding of xanthochromia implies a SAH, a condition that is infrequent but has catastrophic morbidity and mortality if missed.[3] A cerebral angiogram in search of a treatable aneurysmal cause is indicated on finding xanthochromia or more specifically on detecting bilirubin in the CSF.

Spectrophotometry is a technique for measuring bilirubin in the CSF.[4] The usual laboratory analytical techniques cannot be used because bilirubin appears in too low a concentration. A spectrophotometer measures the light absorption of a material, thus quantifying its presence, analogous to the clinically ubiquitous pulse oximeter. Spectrophotometry is able to detect low concentrations of bilirubin in the CSF. Moreover, it can distinguish between bilirubin and oxyhaemoglobin. It is an objective test that is sensitive for SAH.[1]

Visual inspection is a subjective test that is insensitive for xanthochromia.[1] Unlike spectrophotometry, visual inspection is incapable of detecting low concentrations of bilirubin and differentiating between bilirubin and oxyhaemoglobin.[4] Moreover, its intraobserver and interobserver agreement is poor.[2] In sufficient concentrations, oxyhaemoglobin appears orange-red whereas bilirubin is yellow. When present, both contribute to the discolouration of the CSF. Visual inspection cannot reliably detect and distinguish between the two.

The distinction between oxyhaemoglobin and bilirubin is central to the analysis and interpretation of the CSF. Oxyhaemoglobin appears in both a traumatic lumbar puncture (LP) and following a SAH.

A traumatic LP confounds the interpretation of the CSF findings. The incidence of a traumatic LP is around 20% when arbitrarily defined as the presence of greater than 400×10^6 RBCs per litre of CSF.[4] In conventional teaching, a decreasing RBC count from the first to third or fourth collection tube is indicative of a traumatic LP, whereas a steady RBC count is said to be diagnostic of a SAH. However, a falling RBC count cannot reliably be equated to trauma unless it falls close to zero, because a traumatic LP can occur in the presence of a SAH.[3]

Bilirubin will only appear following a SAH. Following centrifuge after a traumatic LP, the CSF might look discoloured because of the presence of oxyhaemoglobin and not bilirubin. The presence of bilirubin is the key finding in SAH.

CSF bilirubin spectrophotometry is supported by National Guidelines in the UK.[10] Proposed in 2003 and revised in 2008, the United Kingdom National External Quality Assurance Service (UKNEQAS) guidelines set out procedures for specimen requirements, transport, handling, analysis and interpretation. The guidelines also articulate the requirements for clinical governance and participation in an appropriate external quality assurance scheme.[11] There is no equivalent framework for visual inspection.

In Australia, CSF bilirubin spectrophotometry is available, although there is variability in its perceived need.[12] In Queensland, spectrophotometry is provided by Pathology Queensland, which services public hospitals in the state.[13] A spectrophotometer is a piece of desktop printer-sized equipment sitting on the

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RESEARCH

Sensitivity of computed tomography performed within six hours of onset of headache for diagnosis of subarachnoid haemorrhage: prospective cohort study

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Abstract

Objective To measure the sensitivity of modern third generation computed tomography in emergency patients being evaluated for possible subarachnoid haemorrhage, especially when carried out within six hours of headache onset.

Design Prospective cohort study.

Setting 11 tertiary care emergency departments across Canada, 2000-9.

Participants Neurologically intact adults with a new acute headache peaking in intensity within one hour of onset in whom a computed tomography was ordered by the treating physician to rule out subarachnoid haemorrhage.

Main outcome measures Subarachnoid haemorrhage was defined by any of subarachnoid blood on computed tomography, xanthochromia in cerebrospinal fluid, or any red blood cells in final tube of cerebrospinal fluid collected with positive results on cerebral angiography.

Results Of the 3132 patients enrolled (mean age 45.1, 2571 (82.1%) with worst headache ever), 240 had subarachnoid haemorrhage (7.7%). The sensitivity of computed tomography overall for subarachnoid haemorrhage was 92.9% (95% confidence interval 89.0% to 95.5%), the specificity was 100% (99.9% to 100%), the negative predictive value was 99.4% (99.1% to 99.6%), and the positive predictive value was 100% (98.3% to 100%). For the 953 patients scanned within six hours of headache onset, all 121 patients with subarachnoid haemorrhage were identified by computed tomography, yielding a sensitivity of 100% (97.0% to 100.0%), specificity of 100% (99.5% to 100%), negative predictive value of 100% (99.5% to 100%), and positive predictive value of 100% (96.9% to 100%).

Conclusion Modern third generation computed tomography is extremely sensitive in identifying subarachnoid haemorrhage when it is carried out within six hours of headache onset and interpreted by a qualified radiologist.

Introduction

Headache is a common symptom and accounts for about 2% of presenting complaints in the emergency department.[1] A foremost consideration in these patients is the exclusion of spontaneous subarachnoid haemorrhage, a neurosurgical emergency identified

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Question 9 (18 marks)

A 2 year old girl presents with agitation. She has been noted to have taken some of her grandmother's theophylline tablets.

a. List three (3) other early clinical features of theophylline toxicity. (3 marks)
   - **Vomiting**
   - **Tremor**
   - **Tachycardia**

b. List three (3) key investigations that are required for this patient. (3 marks)
   - **Theophlline levels (serial is best answer)**
   - **ECG**
   - **VBG/ ABG**

   Also acceptable:
   - **BSL**
   - **Potassium or magnesium**

c. What is the role of decontamination in this overdose? State three (3) points in your answer. (3 marks)
   - **Oral charcoal indicated** (1g/kg (max 50g) PO or NGT recommended)
   - **Even if delayed presentation**
   - **Need to consider protection of airway i.e. aggressive antiemetics or intubation if co-existent vomiting**

d. What is the role of enhanced elimination in this overdose? State three (3) points in your answer. (3 marks)
   - **HD indicated as the definitive life saving intervention**
   - **Highly effective if commenced early**
   - **Commence as soon as possibly life threatening toxicity is anticipated**
     - **based on serum levels** (> 500 acute and > 330 chronic)
     - **clinical manifestation of severe toxicity: arrhythmias, hypotension, seizures**
   - **multi dose charcoal is indicated in severe toxicity but should not delay more effective HD**

e. List three (3) specific features of severe theophylline toxicity and list the specific treatment of each of these features. (6 marks)

	Specific feature	Specific treatment
1	**Hypotension**	**Aggressive fluids with fluid bolus 1000ml titrated to SBP > 90mmHG** **Vasopressors e.g. noradrenaline - 1-1mcg/kg/hr intravenous**
2	**Seizures**	**Diazepam 5-10mg IV repeated once** **Phenobarbitone 100-300mg IV as second line agent** **+/- intubation**
3	**SVT**	**IV BBlocker i.e. metoprolol 5mg IV slow push titrated SVT (beware bronchospasm in asthma if pt's own meds)**
Also acceptable	**Severe hypokalaemia**	**K replacement**