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

UNIVERSITY HOSPITAL, GEELONG
FELLOWSHIP WRITTEN EXAMINATION
WEEK 21– 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 (18 marks)

You are standing at the Triage desk of your Emergency Department. A distressed man presents to the Triage desk carrying his 5 year old son. He states that he witnessed his son being bitten on the right thigh by a snake, about 30 minutes earlier.
The child is conscious and alert. You note a small bruised area on the child's right thigh.
The child is placed in a resuscitation cubicle with full external monitoring applied.

a. State three (3) key, immediate steps in your management of this child. (3 marks)
Bold required 1 mark each, 4th mark any of other options
- **Keep child immobile- Mandatory**
- **Pressure bandage to right lower limb- Mandatory**
- **Splint right lower limb- Mandatory**
- **Obtain IV access (draw blood for pathology)**
- **Reassure father**

b. List two (2) symptoms and two (2) signs that would be consistent with envenomation in this child. (4 marks)
i) Symptoms: - **Headache**
- **Abdo pain**
- **Vomiting**
- **Myalgia**
- **Diarrhoea**
- **Sweating**

ii) Signs: - Neurotoxic- **cranial nerves/ peripheral focal signs/ respiratory fatigue**
- Coagulopathic- **bruising, bleeding**
- **Hypotension/ circulatory failure**
- **Lymphadenopathy**
- **Muscle tenderness**

c. State your preferred technique (ie bite site or urine or blood) to collect a sample for Venom detection. State two (2) justifications for choice. (3 marks)
- **Skin**

Justification:
- **Best accuracy (ie highest sensitivity and specificity)**
- **Both Urine and blood have unacceptable False +ve rate**
- **Urine false -ve in massive envenomation**
- **Blood false -ve rate unacceptable**

An FBE and U+E are taken.

d. List three (3) other key, blood tests that you would perform for this patient. (3 marks)
- **Coagulation screen- INR, APTT**
  - **Fibrinogen** *(part of a coagulation screen, but usually requires separate ordering)*
  - **DDimer** *(part of a coagulation screen, but usually requires separate ordering)*
- **Blood film**
- **CK**
- **LDH**
- **LFT**

e. Complete the table below. State one (1) justification for each choice. (6 marks)

Management step		Justification
Antivenom type	**Polyvalent** **Monovalent- area specific**	**If severe envenomation - until VDK confirms** **Monovalent following VDK/ area specific choice**
Antivenom dose	**1 amp polyvalent**	**Child=adult** **1 ampoule** **Repeat/ Total dose controversial- seek expert help**
Likelihood of serum sickness	**Increase risk vs adult**	**Child > risk than adult** **↑in line with amount of antivenom- esp if polyvalent given (> 10%)**

Clinical pathway: Snake bite envenomation in Victoria

This clinical pathway applies ONLY to community-acquired snake bites in patients who are not snake handlers. Specific advice regarding bites in snake handlers and from exotic snakes should be obtained from a clinical toxicologist (e.g. Poisons Centre 13 11 26).

Clinical patterns

Snake	Coagulopathy	Neurotoxicity	Myotoxicity	Systemic symptoms	Cardiovascular effects	TMA	Antivenom
Brown	VICC	Rare and mild	-	<50%	Collapse (33%) Cardiac arrest (5%)	10%	Brown
Tiger	VICC	Uncommon	Uncommon	Common	Rare	5%	Tiger
Red-bellied black	Anticoagulant	-	Uncommon	Common	-	-	Tiger

VICC = Venom-induced consumptive coagulopathy (abnormal INR, fibrinogen very low, d-dimer high)
Anticoagulant = aPPT 1.5–2.5 x normal ± minor elevation INR. D-dimer and fibrinogen usually normal
TMA = thrombotic microangiography. Fragmented red blood cells on blood film, thrombocytopenia and a rising creatinine

Indications for antivenom: seek advice from a clinical toxicologist (e.g. Poisons Centre 13 11 26)

- Neurotoxic paralysis (e.g. ptosis, ophthalmoplegia, limb weakness, respiratory effects)
- Significant coagulopathy (e.g. unclottable blood, INR>1.3, prolonged bleeding from wounds and venepunctures)
- History of unconsciousness, collapse, convulsions or cardiac arrest

There are a number of relative indications for antivenom that require expert interpretation. It is strongly recommended that significant systemic symptoms or any abnormality of INR, APTT, fibrinogen, d-dimer, full blood count (leucocytosis, evidence of TMA) or CK >1000 is discussed with a clinical toxicologist to determine if antivenom is required.

Choice of antivenom: seek advice from a clinical toxicologist (e.g. Poisons Centre 13 11 26)

If there is a delay in contacting a clinical toxicologist and there is clear indication for antivenom, administer 1 vial of tiger snake antivenom and 1 vial of brown snake antivenom.

It is strongly recommended that all cases of envenomation be discussed with a toxicologist to guide treatment and appropriate disposition.

Prepare to manage anaphylactoid reactions	**Tick if completed**
• Critical care area with monitoring • IV line in situ • Further IV fluids available • Adrenaline available	
Preparation and administration of antivenom	**Tick if completed**
• Dilute in 100–500mls of isotonic saline • Administer over 15-30 minutes	
• Release pressure bandage immobilisation **after** antivenom has been administered	

Monitor progress: seek advice from a clinical toxicologist (e.g. Poisons Centre 13 11 26)	**Tick if completed**
Monitor, investigate and treat for complications such as occult bleeding, electrolyte abnormality (e.g. hyperkalaemia, developing renal impairment)	
6 hours post anti-venom: INR, APPT, fibrinogen, d-dimer, EUC, CK and FBE If not improving/unsure, seek advice from a clinical toxicologist (e.g Poisons centre 13 11 26)	
12 hours post anti-venom: INR, APPT, fibrinogen, d-dimer, EUC, CK and FBE If not improving/ unsure, seek advice from a clinical toxicologist (e.g Poisons centre 13 11 26)	

NOTE: Coagulopathy may not begin to improve until about 12 hours. Persistent coagulopathy is not an indication for additional antivenom. Seek advice if concerned**.**	
Daily thereafter until resolved: INR, APPT, fibrinogen, d-dimer, EUC, CK and FBE	

Location	**List criteria**
ED observation unit	
Ward	
ICU/ HDU	
Transfer	

Criteria for discharge during daytime (do not discharge at night): seek advice from a clinical toxicologist (e.g. Poisons Centre 13 11 26)	
Uncomplicated myotoxicity and mild neurotoxicity	Once clinical features are resolving and blood tests, at least 12 hours post antivenom, are normalising
VICC	INR, APTT, creatinine and platelet count normalising

Discharge advice	**Tick if completed**
Explanation of the risk of serum sickness (~30%) characterised by flu-like symptoms, fever, myalgia, arthralgia and rash developing 4–14 days post antivenom	
Letter to GP including advice regarding recognition and treatment of serum sickness	

Notes for participating emergency departments:

1. Snake venom detection kit use: This is a decision for individual health services based on local resources and experience. The role of snake venom detection kits in bites occurring in the community within Victoria who are not snake handlers is controversial, because of the narrow range of snakes that might be involved and a significant misclassification rate of tiger snake venom as brown snake venom. Use of the kits requires training and results need to be interpreted in the light of all clinical and laboratory data.

If health services decide to include the use of a snake venom detection kit in their pathway, it should be inserted under the 'Choice of anti-venom' section along with a strong recommendation/ requirement that the results are discussed with a clinical toxicologist.

2. Disposition criteria: Each health service should decide its own disposition criteria, taking into account resources, expertise and clinical risk. These should be clearly documented in the pathway.

Click on the image below to view the entire PDF (& print/save if necessary)

HEALTH CARE

Changes in serial laboratory test results in snakebite patients: when can we safely exclude envenoming?

Graham Ireland, Simon G A Brown, Nicholas A Buckley, Jeff Stormer, Bart J Currie, Julian White, David Spain and Geoffrey K Isbister for the Australian Snakebite Project Investigators

ABSTRACT

Objectives: To determine which laboratory tests are first associated with severe envenoming after a snakebite, when (ie, how long after the bite) the test results become abnormal, and whether this can determine a safe observation period after suspected snakebite.

Design, patients and setting: Prospective cohort study of 478 patients with suspected or confirmed snakebite recruited to the Australian Snakebite Project from January 2002 to April 2009, who had at least three sets of laboratory test results and at least 12 hours of observation in hospital after the bite. Severe envenoming was defined as venom-induced consumption coagulopathy (VICC), myotoxicity, neurotoxicity or thrombotic microangiopathy.

Main outcome measures: International normalised ratio (INR), activated partial thromboplastin time (aPTT), creatine kinase (CK) level, and neurological examination.

Results: There were 280 patients with severe envenoming, 75 with minor envenoming and 163 non-envenomed patients. Of 206 patients with VICC, 178 had an INR > 1.2 (abnormal) on admission, and the remaining 28 had an INR > 1.2 within 12 hours of the bite. Of 33 patients with myotoxicity, a combination of CK > 250 U/L and an abnormal aPTT identified all but two cases by 12 hours; one of these two was identified within 12 hours by leukocytosis. Nine cases of isolated neurotoxicity had a median time of onset after the bite of 4 hours (range, 35 min – 12 h). The combination of serial INR, aPTT and CK tests and repeated neurological examination identified 213 of 222 severe envenoming cases (96%) by 6 hours and 238 of 240 (99%) by 12 hours.

Conclusion: Laboratory parameters (INR, aPTT and CK) and neurological reassessments identified nearly all severe envenoming cases within 12 hours of the bite, even in this conservative analysis that assumed normal test results if the test was not done.

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The majority of patients presenting to Australian emergency departments with suspected snakebite do not develop envenoming.[1-3] The accepted policy in Australia is for patients to be observed and have serial blood samples tested for up to 24 hours after a bite, as well as removal of any first aid such as pressure bandages with immobilisation (PBI). This practice has some support from anecdotal case reports of delayed envenoming, but has never been formally tested in snakebite cases.[4-8] According to one study from southern Queensland, some hospitals discharge asymptomatic patients with normal blood test results 4 to 6 hours after the bite. However, the study included only 34 envenomed patients, and there are concerns about its applicability to other geographical regions.[9]

No Australian study has systematically examined the changes in early laboratory test results observed in envenomed or non-envenomed patients after snakebite. Some early blood test results may be indicative of severe envenoming and therefore useful to determine if envenoming has occurred. Our aim was to determine which laboratory tests are first associated with severe envenoming after a snakebite, when (ie, how long after the bite) the test results become abnormal, and whether this information might assist in determining a safe observation period after suspected snakebite.

METHODS

Setting and study design

This was a cohort study of patients with confirmed or suspected snakebite recruited to the Australian Snakebite Project (ASP). ASP recruitment and data collection processes have previously been described in detail.[10-12] In brief, ASP is a national, prospective, multicentre study that recruits adults and children (aged >2 years) with suspected or definite snakebite from over 100 hospitals. All patients have demographic and clinical information, laboratory test results and treatments recorded on a clinical research form, which is then entered into a purpose-built relational database. Ethics approval has been obtained from 19 human research ethics committees covering all institutions involved in the study.

Study participants

Patients recruited to ASP from January 2002 to April 2009 were included in this study if at least three sets of laboratory test results were available and the patient was observed in hospital for at least 12 hours after the bite or was admitted to hospital with envenoming. We compared envenomed and non-envenomed patients, using cases classified as severe envenoming, minor envenoming or non-envenomed.

Severe envenoming was defined as any of the following:

- Venom-induced consumption coagulopathy (VICC): evidence of a complete consumption coagulopathy, indicated by either an undetectable fibrinogen level or a raised D-dimer level (at least 10 times the assay cut-off or > 2.5 mg/L), with an international normalised ratio (INR) > 3.0.
- Myotoxicity: a creatine kinase (CK) level > 1000 U/L, with myalgia and/or muscle tenderness.
- Neurotoxicity: with either two nerve groups (eg, ocular and bulbar) involved, respiratory muscle paralysis, or requirement for intubation or mechanical ventilation.
- Thrombotic microangiopathy: defined as the presence of intravascular haemolysis on the blood film, thrombocytopenia and an abnormal creatinine level with or without acute renal failure.[13]

Patients with minor envenoming were those with evidence of envenoming but in whom specific treatment or antivenom were rarely required. This included patients with isolated systemic symptoms (at least three of nausea, vomiting, abdominal pain, diarrhoea, diaphoresis and headache), anticoagulant coagulopathy, mild or partial VICC

Research

Tiger snake (*Notechis* spp) envenoming: Australian Snakebite Project (ASP-13)

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Abstract

Objectives: To describe the clinical syndrome associated with definite tiger snake (*Notechis* spp) envenoming and to examine the ability of tiger snake antivenom (TSAV) to bind free venom in vivo.

Design, setting and participants: We conducted a prospective cohort study within the Australian Snakebite Project, reviewing all definite tiger snake envenoming cases between October 2004 and June 2011. Definite cases were identified by venom-specific enzyme immunoassay or expert snake identification.

Main outcome measures: Clinical effects of tiger snake envenoming; peak venom concentrations; number of vials of antivenom administered.

Results: Fifty-six definite tiger snake envenomings were identified. Clinical effects included venom-induced consumption coagulopathy (VICC) ($n = 53$), systemic symptoms ($n = 45$), myotoxicity ($n = 11$) and neurotoxicity ($n = 7$). Thrombotic microangiopathy occurred in three patients, all of whom developed acute renal failure. There were no deaths. A bite-site snake venom detection kit test was done in 44 patients, but was positive for tiger snake in only 33 cases. Fifty-three patients received TSAV and eight of these patients had immediate hypersensitivity reactions, severe enough in one case to satisfy diagnostic criteria for severe anaphylaxis. The median peak venom concentration in 50 patients with pre-treatment blood samples available was 3.2 ng/mL (interquartile range [IQR], 1–12 ng/mL; range 0.17–152 ng/mL). In 49 patients with post-treatment blood samples available, no venom was detected in serum after the first antivenom dose. Ten patients were given 1 vial of TSAV; the median dose was 2 vials (range, 1–4 vials). Pre-treatment serum venom concentrations did not vary significantly between patients given 1 vial of TSAV and those given 2 or more vials.

Conclusion: Tiger snake envenoming causes VICC, systemic symptoms, neurotoxicity and myotoxicity. One vial of TSAV, the dose originally recommended when the antivenom was first made available, appears to be sufficient to bind all circulating venom.

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Tiger snakes are one of the most frequent causes of envenoming in Australia, particularly in Victoria, and are the most important venomous snake in Tasmania. There are two species of tiger snake — *Notechis scutatus* (mainland tiger snake) and *Notechis ater* (black tiger snake) — although a recent taxonomic revision suggests the distinction may be somewhat arbitrary.[1] Mainland tiger snakes average 1.2 metres in length and have significant variability in appearance, from the classical banded dark grey/black and yellow skin to unbanded types with a range of colours from black to light grey.[2]

Most previous reports on tiger snake bites have been case reports of one or a few cases,[3-8] but there have been two case-series studies, one from South Australia[9] and another from Western Australia.[10] Both series were retrospective, with case confirmation based on clinical effects and on a positive result for tiger snake venom using the commercially available snake venom detection kit (sVDK).[10] The frequency of different envenoming syndromes and the severity of clinical effects in tiger snake envenoming has not been fully characterised.

Tiger snake antivenom (TSAV) was the first snake antivenom to be released by the then Commonwealth Serum Laboratories (which later became CSL Ltd) in 1930. The initial dose was 1 vial. However, over the past few decades, the recommended dose has increased to 4 vials[11] and, in some cases, many more are given.[10,12] Recent laboratory and clinical studies have shown that far less antivenom is required to neutralise the effects of Australian snake venoms[13,14] and to fully bind venom in vivo.[15-17]

The aims of our study were to report the clinical syndrome that occurs with definite tiger snake envenoming and to examine the ability of TSAV to bind free venom in vivo.

Abbreviations

ASP	Australian Snakebite Project
CK	Creatine kinase
EIA	Enzyme immunoassay
INR	International normalised ratio
sVDK	Snake venom detection kit
TSAV	Tiger snake antivenom
VICC	Venom-induced consumption coagulopathy

Methods

We conducted a prospective cohort study of tiger snake envenomings recruited to the Australian Snakebite Project (ASP). The ASP is a prospective multicentre study of suspected snakebites or snake envenomings from over 100 Australian hospitals. The study design, recruitment and data collection have been described previously,[10,18] and approval has been obtained from 19 human research and ethics committees covering all institutions involved.

Demographic details, clinical information, laboratory results, treatments and reactions are documented on clinical research forms by treating staff. Data are entered into a purpose-built relational database. Blood samples from each patient are centrifuged and aliquoted, and the serum is stored at –80°C for later analysis of venom concentrations.

Clinical syndromes

Clinical envenoming syndromes were defined for each patient based on the clinical and laboratory features, using a previously developed classification: venom-induced consumption coagulopathy (VICC) (partial or complete), myotoxicity, neurotoxicity, thrombotic microangiopathy and systemic symptoms.[19] In complete VICC, there is undetectable fibrinogen and/or a raised D-dimer concentration in citrate plasma (either at least 10 times the assay cut-off point or > 2.5 mg/L) and an international normalised ratio

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TOXICOLOGY

EMA

Effectiveness of pressure-immobilization first aid for snakebite requires further study

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Abstract

In the prospective Royal Darwin Hospital snakebite study, pressure-immobilization first aid (PI) was used more often than in previous studies. However, bandages were not uncommonly too loose or not applied to the whole limb and immobilization was often neglected. While PI should continue to be promoted as the standard for Australia for the present, prospective multicentre studies of snakebite with quantitative assays for blood venom concentration will hopefully better elucidate the real effectiveness of PI and define the limitations of timing of application and determine the optimum types of bandage materials to use and the pressure required to be maintained.

Key words: *Australia, first aid, snake bite.*

In 1979, the National Health and Medical Research Council formally endorsed pressure-immobilization first aid (PI) for snakebite management.[1] However, the clinical evidence base for use of PI remains limited to anecdotal case reports,[2,3] analogous to the evidence supporting the previous policy of tourniquets.[4] It is not widely appreciated in Australia that many overseas authorities remain unconvinced that PI should be used.[5,6] An editorial in a leading emergency medicine journal even suggested that 'eventually the pressure methodology will fall by the wayside as have many other physical modalities'.[7] The issue is whether the dramatic effectiveness of PI seen in restrained monkeys in the original experiments[8] has been translated into an effective first aid method for those bitten by snakes. Two aspects for consideration are how often PI is attempted, and once applied, how often PI retards venom in a bite from a venomous snake.

PI remains poorly adhered to in Australia, despite widespread publicity for over 25 years and universal acceptance as policy by all national first aid organizations. Around 18% of children were treated correctly in the field in Queensland,[9] while at least 31% of children with suspected snakebite in Western Australia had no first aid applied in a study from 1984 to 1993.[10] A more recent review of children presenting to the same Perth hospital between 1994 and 2004 found that the use of effective PI had actually dropped since the previous decade, with around only a third of the 151 children having appropriately applied PI.[11] In another

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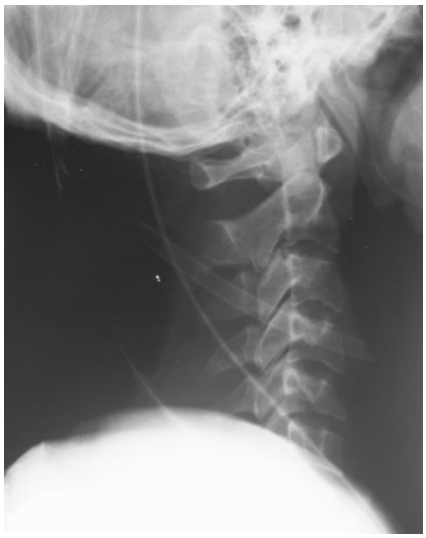
Question 2 (12 marks)

A 25 year old male presents via ambulance to your rural emergency department. He fell from a motorbike and complains of neck pain. After complete examination and investigation, he is found to have an isolated neck injury.
Observations: GCS 15



a. State three (3) abnormal findings shown in this Xray. (3 marks)
    - **Anterior teardrop # C5**
    - **Posterior teardrop # C5**
    - **Loss of continuity of posterior spinal line ay C5-6**
    - **Retrolisthesis of infero-posterior C5**

It is determined that the patient requires transfer by road ambulance to a trauma centre 150 km away.

b. State six (6) preparations for transfer that are **specific for this injury.** (6 marks)
    - **Complete spinal immobilisation**
    - **IDC**
    - **XR images- hardcopy/CD**
    - **Antiemetic**
    - **Analgesia**
    - **Airway:**
        - **secure only if pt non-compliant with spinal immobilisation/ drug affected/ other significant injuries**
    - **Steroids only after discussion with referral centre**
    - **Communication with destination to minimise patient bed transfers**
    - **Keep warm: at risk of hypothermia**

You suspect a spinal injury.

c. List one (1) pro and two (2) cons for using steroids for this patient. (3 marks)
    Pros:
    - **Minor motor benefit- in small trials (needs to be given early)**
    - **For partial injuries only**

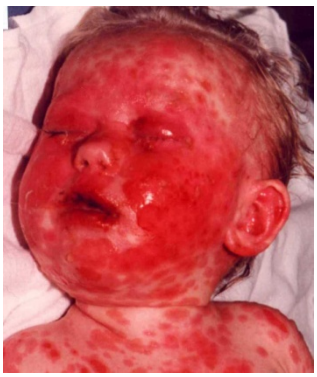
    Cons:
    - **Use not universally accepted- need advice from specialist unit**
    - **Insufficient evidence of benefit to recommend routine use**
    - **Complications:**
        - **Infection**
        - **GI bleed**

Question 3 (12 marks)

You are working in a mixed emergency department in an outer suburban hospital with an inpatient Paediatric service.
An 11 month old female developed a rash over a 48 hr period. The rash is present over the entire body, sparing the palms and soles.

a. What is the diagnosis ? (1 mark)
   - **Toxic epidermal necrolysis**

b. List three (3) likely underlying causes for this condition (**each cause to be a different aetiology type**). (3 marks)
   - **Drugs- sulphonamides, carbamazepine, phenobarbital, lamotrigine, aspirin/NSAIDS**
   - **Infections- underlying HIV, mycoplasma, CMV**
   - **Vaccination**
   - **Contrast medium**
   - **External chemical exposure**
   - **Herbal medicines**
   - **Food**
   - **UV therapy**
   - **Systemic diseases- eg SLE**
   - **Malignancies- leukaemia, lymphoma**



c. What is your preferred disposition for this patient? State two (2) points in your answer. (2 marks)
   - **Urgent transfer to a Tertiary paediatric centre (1) with ICU and Burns unit (1)**
   - **Early Opthalmological referral**
   - **Early gynaecologic referral**

d. Justify your preferred disposition for this patient. State two (2) points of justification for your choice. (2 marks)
   - **prognosis is better for patients transferred promptly to a burn care unit or intensive care unit**
   - **managed largely as a major burn**
   - **Early Opthalmological referral- eye inflammation can evolve quickly in the first few days of the illness**
   - **Early gyanecologic referral- should be performed in all female patients with SJS/TEN. The goal of treatment of vaginal involvement is decreasing the formation of adhesions and labial agglutination**

e. Other than disposition arrangements, list four (4) key steps management of this condition. (4 marks)
   - **Withdraw/treat inciting agent**
   - **Non adherent dressings**
   - **Analgesia**
   - **Fluid management as per burns**
   - **Infection prevention- Sterile handling, antiseptic solutions**
   - **Eye care- lubrication**

   NB: Prophylactic Abs are not recommended
   - *Topical steroids use is controversial*
   - *Ig use is controversial*

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe mucocutaneous reactions, most commonly triggered by medications, characterized by extensive necrosis and detachment of the epidermis. **According to a widely accepted classification, SJS and TEN are considered variants of a disease continuum and are distinguished chiefly by severity, based upon the percentage of body surface involved with blisters and erosions**

- SJS is the less severe condition, in which skin detachment is <10% of the body surface (picture A1-C). Mucous membranes are affected in over 90 % of patients, usually at two or more distinct sites (ocular, oral, and genital).
- **TEN involves detachment of >30 % of the body surface area Mucous membranes are involved in the majority of cases.**
- SJS/TEN overlap describes patients with skin detachment of 10 to 30 % of body surface area.
- "**Dunn" defines Erythema multiformae major as interchangeable with SJS.**

The incidence of SJS/TEN is approximately 100-fold higher among HIV-infected individuals than in the general population.
The overall mortality rate among patients with SJS/TEN is approximately 30%, ranging from approximately 10 % for SJS to more than 30 % for TEN. Mortality continues to increase up to one year after disease onset.
Clinical features:

- Fever, often exceeding 39°C, and influenza-like symptoms precede by one to three days the development of mucocutaneous lesions. Photophobia and conjunctival itching or burning, and pain on swallowing may be early symptoms of mucosal involvement. Malaise, myalgia, and arthralgia are present in most patients.

- In some patients, an exanthematous eruption can be the heralding sign of SJS/TEN. Signs and symptoms that should alert the clinician to the possibility of Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) include fever >38°C mucositis, skin tenderness, and blistering.

- **Cutaneous lesions** — The skin lesions typically begin with ill-defined, coalescing erythematous macules with purpuric centers, although many cases of SJS/TEN may present with diffuse erythema). The skin is often tender to the touch and skin pain can be prominent and out of proportion to the cutaneous findings. Lesions start on the face and thorax before spreading to other areas and are symmetrically distributed. The scalp is typically spared, and palms and soles are rarely involved. Atypical target lesions with darker centers may be present. As the disease progresses, vesicles and bullae form and within days the skin begins to slough.

- Nikolsky sign (ie, the ability to extend the area of superficial sloughing by applying gentle lateral pressure on the surface of the skin at an apparently uninvolved site) may be positive. The Asboe-Hansen sign or "bulla spread sign" (a lateral extension of bullae with pressure) may also be present. The ultimate appearance of the skin has been likened to that of extensive thermal injury.

- **Mucosal lesions** — Mucosal involvement occurs in approximately 90 % of cases of SJS/TEN and can precede or follow the skin eruption. Painful crusts and erosions may occur on any mucosal surface.

- **Oral** — The oral mucosa and the vermilion border are almost invariably involved, with painful haemorrhagic erosions covered with a greyish-white membrane Stomatitis and mucositis lead to impaired oral intake with consequent malnutrition and dehydration.

- **Ocular** — Ocular involvement is reported in approximately 80 % of patients. The most common change in the eyes is a severe conjunctivitis with a purulent discharge but bullae may develop. Corneal ulceration is frequent, and anterior uveitis or panophthalmitis may occur. Pain and photophobia are accompanying symptoms. The eye changes often regress completely, but scarring with the development of synechiae between the eyelids and conjunctiva may be late sequelae.

- **Urogenital** — Urethritis develops in up to two-thirds of patients, and may lead to urinary retention. Genital erosions are frequent. In women, vulvovaginal involvement may present with erosive and ulcerative vaginitis, vulvar bullae, and vaginal synechiae, and may lead to long-term anatomic sequelae. These include labial and vaginal adhesions and stenosis, obstructed urinary stream and urinary retention, recurrent cystitis, or hematocolpos. Vulvovaginal adenosis (presence of metaplastic cervical or endometrial glandular epithelium in the vulva or vagina) also has been reported in women with SJS/TEN.

- Pharyngeal mucosa is affected in nearly all patients; tracheal, bronchial, and oesophageal membranes are less frequently involved. Intestinal involvement is rare.

Question 4 (12 marks)

Clinical handover in the emergency department can be performed using several techniques.

a. List one (1) pro and one (1) con for each of the techniques of handover listed below. (6 marks)

Handover technique	Pros	Cons
Paper/whiteboard	• **Free from unnecessary info** • **Confidentiality** • **(Acceptability)** • **Not -Ease of use**	• **Easy to lose info** • **Errors of info duplication** • **No trail of record changing**
Electronic	• **Rapid data** • **Flexible setup/ WR setting** • **Avoids info doubling up** • **Access Ix at same time** • **Soundproof/ private area**	• **Relies on IT infrastructure** • **Info scope may be limited by functionality** • **Needs electricity** • **System crashes info loss/ tracking**
Ward round/bedside	• **Direct pt viewing** • **Immediate info/ obs** • **Pt satisfaction** • **Direct pt questioning Pain etc** • **Allows provision of symptom care**	• **Time consuming** • **Confidentiality** • **Space for entire team to move** • **Potential threat to staff safety** • **Uncomfortable for patients**

b. Assuming appropriate staff participation, list six (6) other important components to a morning handover ward round. (6 marks).

- **Safe handover of patients seen**
- **Ongoing management of sick patients**
- **Identify salient issues with each pt**
- **Management plan should be clear**
- **Teaching and support where appropriate**
- **Delegate am staff member to each pt**
- **Handover of short stay admitted pts**
- **Debrief any problems overnight**
- **Ensure night staff documentation is complete**

Click on the image below to view the entire PDF (& print/save if necessary)

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ORIGINAL RESEARCH

Handover in the emergency department: Deficiencies and adverse effects

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Abstract

Objective: To determine problems resulting from ED handover, deficiencies in current procedures and whether patient care or ED processes are adversely affected.

Methods: A prospective observational study at three large metropolitan ED comprising three components: observation of handover sessions, 2 h post-handover surveys of the receiving doctors and a general survey of ED doctors.

Results: The handovers of 914 patients were observed during 60 handover sessions in a 3-month period. Medical information, including presenting complaints, was handed over better than communication and disposition information. Seven hundred and seven (77.4%) of 914 potential post-handover interviews were undertaken. Most (88.3%) doctors thought the handover was 'adequate/good'. However, information was perceived as lacking in 109 (15.4%) handovers, especially details of management (35, 5.0%), investigations (33, 4.7%) and disposition (33, 4.7%). There was a significant difference in the perceived quality of handovers (1–5 scale where 5 = excellent) when all required information was handed over and when it was not (median scores 4.0 *vs* 3.0, respectively, $P < 0.001$). As a result of perceived inadequate handovers, the doctor/ED and patient were affected adversely in 62 (8.8%) and 33 (4.7%) cases, respectively, for example, repetition of assessment, delays in disposition and care. Fifty doctors completed the general survey. Most believed communications made to inpatient units, inaccurate/incomplete information and disorganization were problematic.

Conclusion: Deficiencies in handover processes exist, especially in communication and disposition information. These affect doctors, the ED and patients adversely. Recommendations for improvement include guideline development to standardize handover processes, the greater use of information technology facilities, ongoing feedback to staff, and quality assurance and education activities.

Key words: *adverse event, communication, emergency department, handover.*

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ORIGINAL RESEARCH

Bedside review of patient care in an emergency department: The Cow Round

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Abstract

Objective: Clinical handover is a critical point in medical care in the ED, which can contribute to adverse effects for patient care and staff workloads. Over a 4 and a half months in a tertiary referral hospital ED, a centralized whiteboard handover was performed followed by a multidisciplinary review of each patient. This round was referred to as the 'Cow Round'.

Methodology: This observational study used a standardized feedback survey of clinicians leading each Cow Round. The survey asked participants in the round to report issues found, which were not handed over during the centralized whiteboard handover. Data were analysed for the number of issues identified, the type of issue identified, and to determine if there was a relationship between the number of issues reported and patients in the department.

Results: 204 surveys met inclusion criteria. Clinical issues not handed over at the standard whiteboard round were found in 64% of Cow Rounds. Of the 2411 patients reviewed on Cow Rounds, 14.1% had at least one clinical issue not handed over during the whiteboard round. A mean of 2.2 issues per round (95% CI 1.9–2.5) were found. Pearson correlation found a relationship between the number of issues identified and the total number of patients in the department ($r = 0.246$ $P = 0.005$).

Conclusion: Review of patients led by a senior member of medical staff, at the patient bedside enables the timely identification and management of issues not communicated during the whiteboard handover process. This review is important when more patients are receiving treatment in the department.

Key words: *clinical handover, emergency medicine, medical error.*

Introduction

Effective handover is the accurate and reliable transfer of information, responsibility and accountability to ensure coordination of independent people and technologies for a patient or group of patients.[1–3] This is particularly important in the ED, where work is characterized by evolving patient clinical condition, high staff turnover and shift changes. These challenges make the role of caring for multiple patients simultaneously, increasingly difficult.[4] Compounding these difficulties are variable skill mix and handover technique. Communication in the ED setting might significantly compromise patient safety and quality of care delivery.[2,3]

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ORIGINAL RESEARCH

Improving emergency department medical clinical handover: Barriers at the bedside

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Abstract

Objective: The present paper describes our experience of developing and piloting a best practice model of medical clinical handover. Secondary aims were to improve reliability of communication, identify negative effects on patient care and assess staff adherence and acceptance of the process.
Methods: We described existing handover practice. We designed and implemented a process incorporating bedside handover, the Identification, Situation, Background, Assessment, Requirements and Requests (ISBAR) tool and handover documentation. We audited the process and surveyed doctors before and after the intervention regarding their practice and preferences.
Results: Existing handover practice was remote from the patient, neither standardised nor documented. The new process resulted in a median 87% (95% CI 70.4–92.1) of handovers in the presence of the patient. ISBAR elements were consistently communicated, median 100% (95% CI 91.8–100). Risk events were directly identified in a median 8.3% (95% CI 0.0–13.8) of bedside handovers. Handover documentation did not improve. FACEM and registrar perception that bedside handover improves patient care fell from 71%, 80% to 56%, 58%, respectively. Preference for bedside handover fell from 79% and 80%, respectively, to being evenly divided between bedside and centralised models; 80.9% of respondents reported that ISBAR improved communication.
Conclusion: Bedside handover using ISBAR resulted in improved patient involvement, communication and a non-significant trend to improved patient safety. Despite a majority of doctors acknowledging these findings, preference remained for a centralised handover using ISBAR. Gaining staff acceptance of a process change is essential to its success. A barrier to acceptance could be that staff are time-poor. We suggest handover processes can be strengthened by adequate staffing and small, incremental improvements to existing models combined with auditing of outcomes.

Key words: *clinical handover, communication, ED, quality improvement.*

Key findings

- Bedside medical handover using ISBAR was implemented into a tertiary ED setting.
- We found improved patient involvement, staff communication and a trend to improved patient safety.
- Staff time pressures are a barrier to handover improvement.

Introduction

Shift work is necessary in the ED to provide continuous medical staff presence.[1] Clinical handovers occur when there is a changeover of staff. These are critical moments that influence patient care. High patient turnover and a wide range of presentations in the ED create a complex case load, which varies from hour to hour.[2] An effective clinical handover process, whereby information about a patient's care is passed from one healthcare professional to the next accurately and reliably, is essential to ensure continuity of care.[1]

Australian research examining medical clinical handover in EDs and in general, has identified that poor handover practices result in incomplete information transfer and consequently repetition of assessments,[3] delayed treatment,[3,4] medication errors, avoidable readmissions, increased patient morbidity and mortality.[5] Lack of training and poor knowledge of handover processes have been shown to contribute to these errors and inefficiencies.[5,6]

Reported 'best practice' consistently supports a structured handover led by a nominated senior practitioner, held at set times each day, preferably in a bedside environment allowing involvement of patients and their carers. Furthermore, handovers should incorporate recognised formats – for example ISBAR: Identification, Situation, Background, Assessment, Requirements and Requests[7] – and be documented following verbal discussion to prevent miscommunication.[7–9] The Australian Commission on Safety and Quality in Health Care (ACSQHC) and the ACEM have

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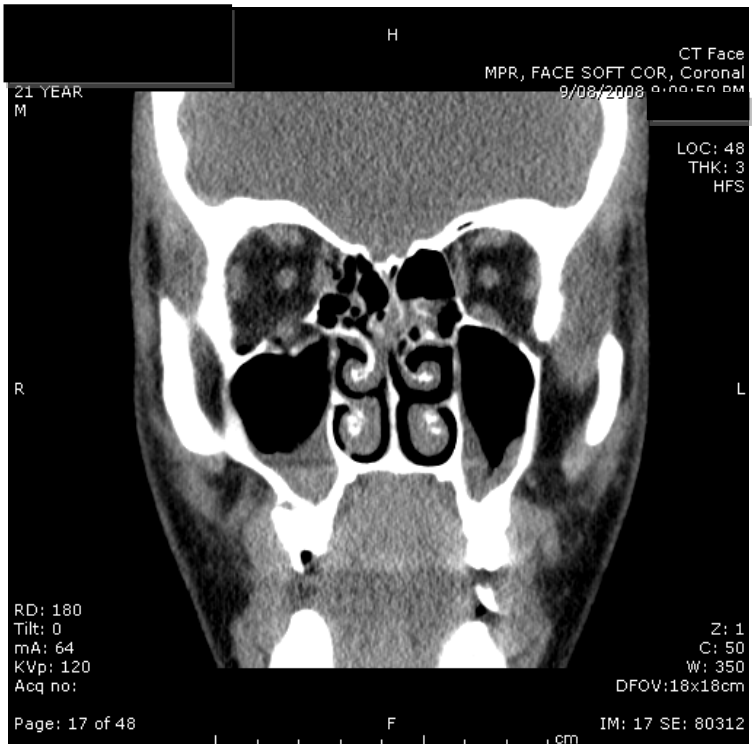
Question 5 (10 marks)

A 21 year old man presents following an assault with a painful right eye



a. State four (4) abnormal findings shown in this CT scan. (4 marks)

- **R Blow out #**
- **R trapped inf rectus**
- **R maxilliary sinus blood**
- **L maxilliary sinusitis**
- **Air in R orbit**

b. List (6) associated examination findings that you would expect to be associated with this injury. (6 mark)

- **Periocular bruising**
- **Infraorbital numbness**
- **Inability to look up/ upward gaze palsy**
- **Pain on eye movement**
- **Tenderness to orbital rim**
- **Facial subcutaneous emphysema**

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November 2017

Question 6 (12 marks)

An 82 year old woman presents to your emergency department with 10 hours of abdominal pain. Your clinical assessment leads to a clinical diagnosis of mesenteric ischaemia.

a. List three (3) options for definitive imaging in this patient. State one (1) relevant pro and one (1) con for each of these options. (9 marks)

NB: you only have 1 choice for each so make it a "goody". Make it clinically relevant.

Imaging option	Pro	Con
CT abdo + contrast	• **Most sensitive for mesenteric venous thrombosis** • **Can define embolic vs thrombotic- arterial and venous occlusion** • **Dx other Dx**	• **risk of contrast nephropathy** • **Sensitivity and specificity can be as low as 64% and 92%** • **contrast allergies**
Angiography	• **Specific and gold standard** • **Diagnostic and therapeutic** • **Good resolution of bowel wall oedema** • **Identify the type of occlusion, site of occlusion and state of collateral circulation**	• **Limited availability** • **Radiocontrast** • **Invasive**
MRI/MRA	• **Detailed information of the vasculature** • **Can define embolic vs thrombotic**	• **Limited resolution of bowel gas** • **Availability** • **Out of department** • **Time for procedure and interpretation**

NB: US –Limited value in acute setting. Useful in chronic state, assessing vascular flow- 87% and 98% sensitivity in identifying celiac and SMA stenosis respectively

A diagnosis of mesenteric ischaemia is supported by your chosen imaging.

b. State three (3) factors that may affect a decision regarding operative treatment for this patient. (3 marks)

- **Critical decision: 100% mortality without surgery; time critical if opt for it**
- **Patient's wishes**
  - **Advance directives**
  - **Patient potentially unfit to decide, even if *compos mentis* pre-morbidly**
- **Next of Kin:**
  - **Medical power of attorney if applicable**
  - **Choice must be informed and not coerced**
- **Co-morbidities:**
  - **Premorbid QOL**
  - **Complications of current illness – eg acidaemia, shock**
  - **Other illness – heightening risks of perioperative morbidity / mortality**
  - **Other risks: eg current warfarin or antiplatelet therapy**
- **Clinical progress- Response to initial resuscitation**
- **Current resources- Availability of urgent surgical services and ICU, and their opinion. If unavailable, pt unlikely to be suitable for transfer**

Question 7 (11 marks)

A 3 week old boy is brought to emergency with frequent vomiting over a 24 hour period.

Arterial blood gas, serum and urine biochemistry

			Reference range
FiO_2	0.21		
pH	7.54		7.35- 7.45
pCO2	50	mmHg	35-45
PO2	62	mmHg	80- 95
Bicarbonate	41	mmHg	22-28
Base excess	+ 10		-3 - +3
O2 saturation	99	% > 95	

Na^+	131	mmol/l	134-146
K^+	2.1	mmol/l	3.4- 5.0
Cl^-	66	mmol/l	98- 106
Bicarbonate	45	mmol/l	22- 28
Urea	10.5	mmol/l	2.5- 6.4
Creatinine	0.05	mmol/l	0.05- 0.1
Glucose	3.4	mmol/l	3.5- 5.5

Urine spot		
Na	22	mmol/l
K	28	mmol/l
Cl	<10	mmol/l

a. Provide one (1) calculation to help you to interpret these results. (1 mark)
   - Derived value 1: **Expected pCO2= PCO2 = 0.9 x HCO3- + 9 = 49.7**

b. What is the significance of this calculation finding? (1 mark)
   - **Appropriate respiratory component- metabolic alkalosis only, no resp component**

c. What is the most likely diagnosis? (1 mark)
   - **Pyloric stenosis**

d. List four (4) investigation findings from these blood tests to support this diagnosis. (4 marks)
   - **Severe metabolic alkalosis- pH 7.52 and Bicarbonate 45**
   - **Severe hypochloraemia**
   - **Severe hypokalaemia**
   - **Elevated Ur:Cr- suggesting dehydration**
   - **Others less good:**
     - **Mild hyponatraemia**
     - **Urinary sodium low**
     - **Increased urinary K loss**
     - **Decreased urinary Cl**
     - **Near normal glucose**

e. List two (2) urgent, key investigations that you would order for this patient. State one (1) justification for each choice. (4 marks)

Investigation (2 marks)	**Justification (2 marks)**
US	**Accuracy close to 100% (ie Sensitivity and specificity near 100%) "doughnut" or "null's eye" on X-section of pyloric channel**
Urine/ Septic screen	**Exclude infection as cause of vomiting**

Diagnosis/Evaluation

In the majority of patients with metabolic alkalosis the cause is readily established from the clinical picture. In those cases with obscure aetiology special consideration should be given to the possibility of surreptitious vomiting and diuretic administration, especially if severe to moderate hypokalaemia is present. Further information may be obtained from the urinary chloride concentration (*see Fig.* 5.6). The saline-responsive group (*see Table* 5.5) has a low concentration (<20 mmol/l) and the unresponsive group has a level greater than 20 mmol/l.

A high anion gap associated with a metabolic alkalosis suggests a concurrent metabolic acidosis. Although alkalaemia is associated with increased lactate production this does not raise the plasma anion gap more than 2–3 mEq/l.

If blood gas results are available the P_{CO_2} value should be checked for the possibility of an associated respiratory disorder. A low level (<35 mmHg) suggests a concurrent respiratory alkalosis, whilst a level greater than 60 mmHg indicates a possible underlying respiratory acidosis.

Principles of Management

The management of metabolic alkalosis depends on the cause and severity. In all cases the general principle is to reduce the alkalaemia by lowering the plasma bicarbonate level. This involves attention to the causes of generation and maintenance of the increased plasma bicarbonate.

In the volume-contracted or saline-responsive group the generating mechanism (vomiting, diuretics, etc.) should be returned to normal, the hypovolaemia resolved (intravenous saline if necessary), and any potassium deficit corrected. In mineralocorticoid excess the treatment depends on the aetiology but if it is of the endogenous type spironolactone administration will alleviate the problem until definitive treatment can be carried out.

Drastic measures aimed at lowering the bicarbonate level such as acid (HCl) administration, haemodialysis and carbonic anhydrase therapy are rarely necessary.

Case Examples

Vomiting

A 6-month-old infant was admitted to hospital with a 5-day history of projectile vomiting (pyloric stenosis). His admission acid–base parameters, and those 10 h later after intravenous normal saline infusion, with potassium supplements, are shown below.

		Date	13/02	14/02	
		Time (h)	2200	0800	
Plasma	Na		131	134 mmol/l	(132–144)
	K		2·1	3·6 mmol/l	(3·2–4·8)
	Cl		66	94 mmol/l	(98–108)
	HCO_3		>40	34 mmol/l	(23–33)
	Creat		0·05	0·04 mmol/l	(0·06–0·12)
Urine	Na		22	— mmol/l	
	K		28	— mmol/l	
	Cl		<10	— mmol/l	
Blood	pH		7·54	7·48	(7·35–7·45)
	H^+		29	33 nmol/l	(35–45)
	P_{CO_2}		50	43 mmHg	(35–45)
	P_{O_2}		51	75 mmHg	(80–110)
	$AHCO_3$		41	32 mmol/l	(24–32)

Comment

The admission blood gas and electrolyte values are typical of a patient who is vomiting from above the pylorus (gastric vomiting), e.g.

A. Loss of HCl and water (gastric juice):
HCl loss ⟶
1. Metabolic alkalosis (generation of HCO_3^-)
2. Hypochloraemia

Water loss ⟶ hypovolaemia ⟶
1. ↑ Aldosterone ⟶ ↑ renal K^+ loss
2. ↑ Renal NaCl reabsorption ⟶ ↓ urine $[Cl^-]$
3. ↑ Renal HCO_3^- reabsorption (maintenance of alkalosis)

B. The high plasma $[HCO_3^-]$ floods the renal reabsorption mechanism resulting in:
1. $NaHCO_3$ excretion ⟶ ↑ urine $[Na^+]$ (>20 mmol/l)
2. ↑ Distal nephron flow rate ⟶ ↑ renal K^+ excretion

C. The alkalaemia suppresses respiration producing:
1. ↑ P_{CO_2} (compensation)
2. ↓ P_{O_2}

In metabolic alkalosis complete compensation (pH to 7·40) is rarely achieved because the decreased respiratory response to alkalaemia not only results in hypercapnia, but also in hypoxia. Both of these are potent respiratory stimulants and they eventually over-ride the alkalaemic suppression of respiration.

The metabolic alkalosis of vomiting is an example of the saline-responsive type (hypovolaemia, urine $[Cl^-]$ <20 mmol/l). This is illustrated in the above case where after appropriate saline infusion the $[HCO_3^-]$ has dropped from 41 to 32 mmol/l within a few hours.

Diuretic Therapy

The electrolyte and blood gas values shown below are those of a 76-year-old female, with congestive cardiac failure, who had been on diuretic (thiazide) therapy for 4 months.

Plasma	Na	124 mmol/l	(132–144)
	K	2·4 mmol/l	(3·2–4·8)
	Cl	76 mmol/l	(98–108)
	HCO_3	38 mmol/l	(23–33)
	Creat	0·07 mmol/l	(0·06–0·12)

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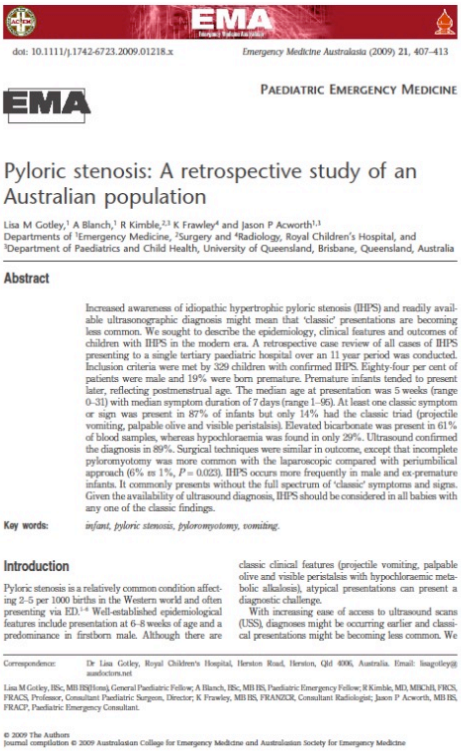

EMA
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EMA
PAEDIATRIC EMERGENCY MEDICINE

Pyloric stenosis: A retrospective study of an Australian population

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Abstract

Increased awareness of idiopathic hypertrophic pyloric stenosis (IHPS) and readily available ultrasonographic diagnosis might mean that 'classic' presentations are becoming less common. We sought to describe the epidemiology, clinical features and outcomes of children with IHPS in the modern era. A retrospective case review of all cases of IHPS presenting to a single tertiary paediatric hospital over an 11 year period was conducted. Inclusion criteria were met by 329 children with confirmed IHPS. Eighty-four per cent of patients were male and 19% were born premature. Premature infants tended to present later, reflecting postmenstrual age. The median age at presentation was 5 weeks (range 0–31) with median symptom duration of 7 days (range 1–96). At least one classic symptom or sign was present in 87% of infants but only 14% had the classic triad (projectile vomiting, palpable olive and visible peristalsis). Elevated bicarbonate was present in 61% of blood samples, whereas hypochloraemia was found in only 29%. Ultrasound confirmed the diagnosis in 89%. Surgical techniques were similar in outcome, except that incomplete pyloromyotomy was more common with the laparoscopic compared with periumbilical approach (6% *vs* 1%, $P = 0.023$). IHPS occurs more frequently in male and ex-premature infants. It commonly presents without the full spectrum of 'classic' symptoms and signs. Given the availability of ultrasound diagnosis, IHPS should be considered in all babies with any one of the classic findings.

Key words: *infant, pyloric stenosis, pyloromyotomy, vomiting.*

Introduction

Pyloric stenosis is a relatively common condition affecting 2–5 per 1000 births in the Western world and often presenting via ED.[1–4] Well-established epidemiological features include presentation at 6–8 weeks of age and a predominance in firstborn male. Although there are classic clinical features (projectile vomiting, palpable olive and visible peristalsis with hypochloraemic metabolic alkalosis), atypical presentations can present a diagnostic challenge.

With increasing ease of access to ultrasound scans (USS), diagnoses might be occurring earlier and classical presentations might be becoming less common. We

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Question 8 (18 marks)

A 25 year old woman presents following a deliberate aspirin overdose.

a. Complete the table below to demonstrate your dose related risk assessment.(10 marks)

Dose range mg/kg	Clinical effects	Acid/base disturbance
< 150 mg/kg	**Minimal symptoms** **(no further answer required)**	**Nil** **(no further answer required)**
150-300 mg/kg	**Hyperpnoea** **Tinnitus, decreased hearing** **CNS agitation** **Nausea vomiting**	**Resp alkalosis**
> 300 mg/kg	**Altered mental state** **Seizure**	**HAGMetabolic acidosis**
> 500 mg/kg	**Potentially lethal**	**Acidaemia**

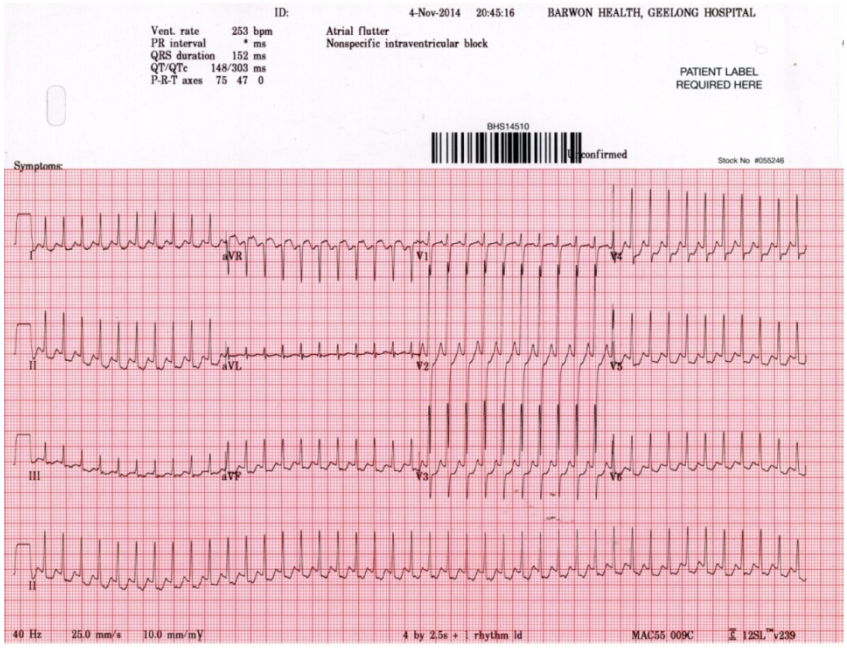
b. What is the role of serum salicylate levels? State three (3) points in your answer. (3 marks)
   - **Poor correlation between levels and severity of toxicity**
   - **Serial levels every 2-4 hrs useful to identify ongoing/ delayed absorption (tablet bezoar/ SR tablets)**
   - **Very high levels may be used as an indication for dialysis**
   - **Lower level is a concern in chronic poisoning or elderly**
   - **↑ levels post charcoal is an indication for repeated dosing of charcoal**

c. What is the role of decontamination in this poisoning? State two (2) points in your answer. (2 marks)
   - **Effective**
   - **> 150mg/kg : Oral charcoal up to 8/24**
   - **> 300 mg/kg NGT after airway secured**
   - **Repeated dose if serum levels rising**

d. What is the role of enhanced elimination in this poisoning? State three (3) points in your answer. (3 marks)
   - **Urinary alkalinisation for symptomatic**
   - **Haemodialysis rarely required if decontamination and urinary alkalinisation implemented early**
   - **Indicated if:**
     - **Urinary alkalinisation not feasible**
     - **↑ serum levels despite decontamination & urinary alkalinisation**
     - **Severe toxicity** *(altered mental state, acidaemia, ARF)*
     - **Very high salicylate levels**

Question 9 (11 marks)

An 18 month old girl presents with respiratory distress and pallor.



a. What is the diagnosis based on this ECG? (1 mark)
   - **SVT**

b. State four (4) features shown in this ECG that support this diagnosis. (4 marks)
   - **Regular**
   - **Narrow complex**
   - **Tachycardia: Rate 230-270** *(acceptable range)*
   - **Absent p waves**

c. State six (6) immediate steps in your management, demonstrating your escalation until this condition is adequately treated. (6 marks)

   NB: shock is suggested by presentation- SOB & pallor
   - **Consent/ explanation to parents**
   - **Vagal manoeuvre - Ice to face/ invert upside down/ head in bucket of water (!)**
   - **If rapid IV access available - IV adenosine 0.1 mg/kg**
   - **Repeat IV adenosine 0.2 mg/kg then 0.3 mg/kg (+/- 0.4 mg/kg)**
   - **If IV access delayed/ failure of IV adenosine- IM sedation (eg Ketamine 4 mg/kg) & DCR- dose 0.5-1J/Kg**
   - **Repeat DCR 2mg/kg**

   NB: NOT verapamil- (CI < 1 yr)

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Advanced Paediatric Life Support

Supraventricular Tachycardia (SVT) Management