ID NUMBER: ☐ ☐ ☐ ☐ ☐

University Hospital, Geelong

Emergency Medicine

Trial Fellowship Exam

Short Answer Questions (SAQ)

Week 24

DIRECTIONS TO CANDIDATE

1. Answer each question in the space provided in this question paper.
2. Do not write your name on this question paper.
3. Enter your examination number in the space below.
4. Cross out any errors completely.
5. Do not begin the exam until instructed to do so.
6. Do not take examination paper or materials from this room.
7. The booklet binder may be removed during the exam.

QUESTION & ANSWER

BOOKLET

Question 1 (14 marks)

A 3 year old boy presents to your emergency department after taking 20 Lomotil tablets approximately 1 hour ago.

a. What is the role of decontamination for this patient? Include two (2) points in your answer. (2 marks)

1. _____

2. _____

b. List four (4) examination features that you would expect at this stage. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 1 (continued)

All of these features are present. You assess the patient to have severe toxicity.

c. State five (5) key steps in the management of this patient. (5 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

d. State the time frame that you would expect the patient to require hospitalisation, if he experiences no further complications of his ingestion. (1 mark)

_____

Question 2 (18 marks)

You are the consultant in charge of a regional base hospital ED. A 17 year-old girl is brought in by her parents with a 6-month history of weight loss.

a. State the four (4) key components to the diagnosis of Anorexia Nervosa. (4 marks)

1. _____

2. _____

3. _____

4. _____

b. List seven (7) examination findings that you would seek on examination for this patient. (7 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

6. _____

7. _____

Question 2 (continued)

A rapid assessment nurse has organised a venous blood gas which shows a serum K^+ level of 2.2.

c. State three (3) clinical factors that would lead you to choose IV replacement as the route of choice. (3 marks)

1. _____

2. _____

3. _____

d. State one (1) pro and one (1) con for oral and IV route for potassium replacement. (4 marks)

Route	Pros (2 marks)	Cons (2 marks)
Oral	1.	1.
Intravenous	1.	1.

Question 2 (continued)

Before you have completed your assessment, she asks to get dressed and discharge herself.

e. State five (5) key issues in this situation. (5 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

Question 3 (18 marks)

A 35 year old woman presents with suspected thyroid storm.

a. State four (4) diagnostic features of thyroid storm. (4 marks)

1. _____

2. _____

3. _____

4. _____

b. List three (3) likely precipitants for thyroid storm. (3 marks)

1. _____

2. _____

3. _____

Question 3 (continued)

c. List three (3) medications that may be used for this patient. For each medication, state one (1) reason why this medication is used. (6 marks)

	Medication **(3 marks)**	**Why is this medication used?** **(3 marks)**
1		
2		
3		

Question 3 (continued)

d. List one (1) medication that is specifically contraindicated in thyroid storm (1 mark)

_____

e. Other than intravenous fluids and oxygen, list four (4) non-medicinal treatments that may be utilised for this patient. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 4 (12 marks)

A 64 year old man is involved in a high speed, roll over motor car collision. He was unrestrained. On ambulance handover he has obvious bilateral femur #, severe pelvic pain, widespread chest and abdominal bruising and a suspected pelvic fracture. Time from injury on arrival is 90 minutes. He has received 4 L NS prehospital.
His observations on arrival are:

BP	60/30 mmHg
HR	145 bpm
RR	30 bpm
Sats 90%	15L via non rebreather mask

a. Based on the CRASH-2 study findings, state four (4) points relating to the use of Tranexamic acid for this patient. (4 marks)

1. _____

2. _____

3. _____

4. _____

b. State two (2) criticisms for the CRASH-2 study. (2 marks)

1. _____

2. _____

Question 4 (continued)

You are situated in an outer suburban hospital. After discussion with the regional retrieval service, it is decided to transfer the patient to a trauma centre 30 minutes by road.
It is requested that you arrange placement of a REBOA prior to transport.

c. What is REBOA? (1 marks)

_____

d. List three (3) features of this patient that may support the use of a REBOA. (3 marks)

1. _____

2. _____

3. _____

e. In general, list two (2) specific indications for a Zone 1 REBOA. (2 marks)

1. _____

2. _____

Question 5 (11 marks)

A 75 year old man presents with palpitations.

On examination:

BP	140/ 60	mmHg supine
RR	40	/ min
Oxygen saturation	88%	on 6L via Hudson mask
GCS	15	

An ECG is taken- refer to the prop booklet page 2.

a. What is the ECG diagnosis? (1 mark)

_____

b. State three (3) abnormalities on this ECG to support this diagnosis. (3 marks)

1. _____

2. _____

3. _____

Question 5 (continued)

c. List four (4) likely causes for this ECG diagnosis. (4 marks)

1. _____

2. _____

3. _____

4. _____

d. What is the clinical relevance of this ECG diagnosis? State three (3) points in your answer. (3 marks)

1. _____

2. _____

3. _____

Question 6 (12 marks)

A 32 year old man, John Smith (UR 123456), presents to your emergency department following a high pressure injury to his left middle finger, 1 hour ago.

Two photographs are taken- refer to the prop booklet page 3.

a. List three (3) pathophysiological mechanisms for harm from this mechanism of injury. (3 marks)

1. _____

2. _____

3. _____

b. List three (3) factors associated with a poor outcome from this injury in this patient. (3 marks)

1. _____

2. _____

3. _____

Question 6 (continued)

You decide to refer the patient after your care.

c. Using an ISBAR approach, list five (5) pieces of information that you would pass onto the receiving Doctor. (6 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

Question 7 (12 marks)

A 32 year old man is involved in a roll over motor car collision. He is known to be taking a NOAC.

a. Under what circumstances would you take measures to reverse the action of the NOAC? List two (2) circumstances. (2 marks)

1. _____

2. _____

b. What is the role of charcoal in the reversal of a NOAC? State two (2) points in your answer. (2 marks)

1. _____

2. _____

c. What is the role of dialysis in the reversal of a NOAC? State two (2) points in your answer. (2 marks)

1. _____

2. _____

Question 7 (continued)

d. Other than Packed cells/ whole blood, state three (3) agents which may be used for reversal of the effects of Dabigatran. (3 marks)

1. _____

2. _____

3. _____

e. What is the role of thromboelastography for this patient? State three (3) points in your answer. (3 marks)

1. _____

2. _____

3. _____

Question 8 (12 marks)

a. At what height does acute mountain sickness appear in a person who is not acclimatised to altitude? (1 mark)

_____

b. List three (3) examination features of a patient with high altitude cerebral oedema. (3 marks)

1. _____

2. _____

3. _____

c. List three (3) management steps for a patient with high altitude cerebral oedema. (3 marks)

1. _____

2. _____

3. _____

_

Question 8 (Continued)

d. List three (3) examination features of a patient with high altitude pulmonary oedema. (3 marks)

1. _____

2. _____

3. _____

e. List three (3) management steps for a patient with high altitude cerebral oedema. (3 marks)

1. _____

2. _____

3. _____

Question 9 (12 marks)

a. List three (3) cardinal features of a patient with Neuroleptic Malignant syndrome. (3 marks)

1. _____

2. _____

3. _____

b. List three (3) risk factors for the development of Neuroleptic Malignant syndrome. (3 marks)

1. _____

2. _____

3. _____

Question 9 (Continued)

c. List two (2) antidotes that may be beneficial for a patient with Neuroleptic Malignant Syndrome. (2marks)

1. _____

2. _____

d. Other than antidote use, list four (4) key components to the management of a patient with Neuroleptic Malignant syndrome. (4 marks)

1. _____

2. _____

3. _____

4. _____

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University Hospital, Geelong

Emergency Medicine

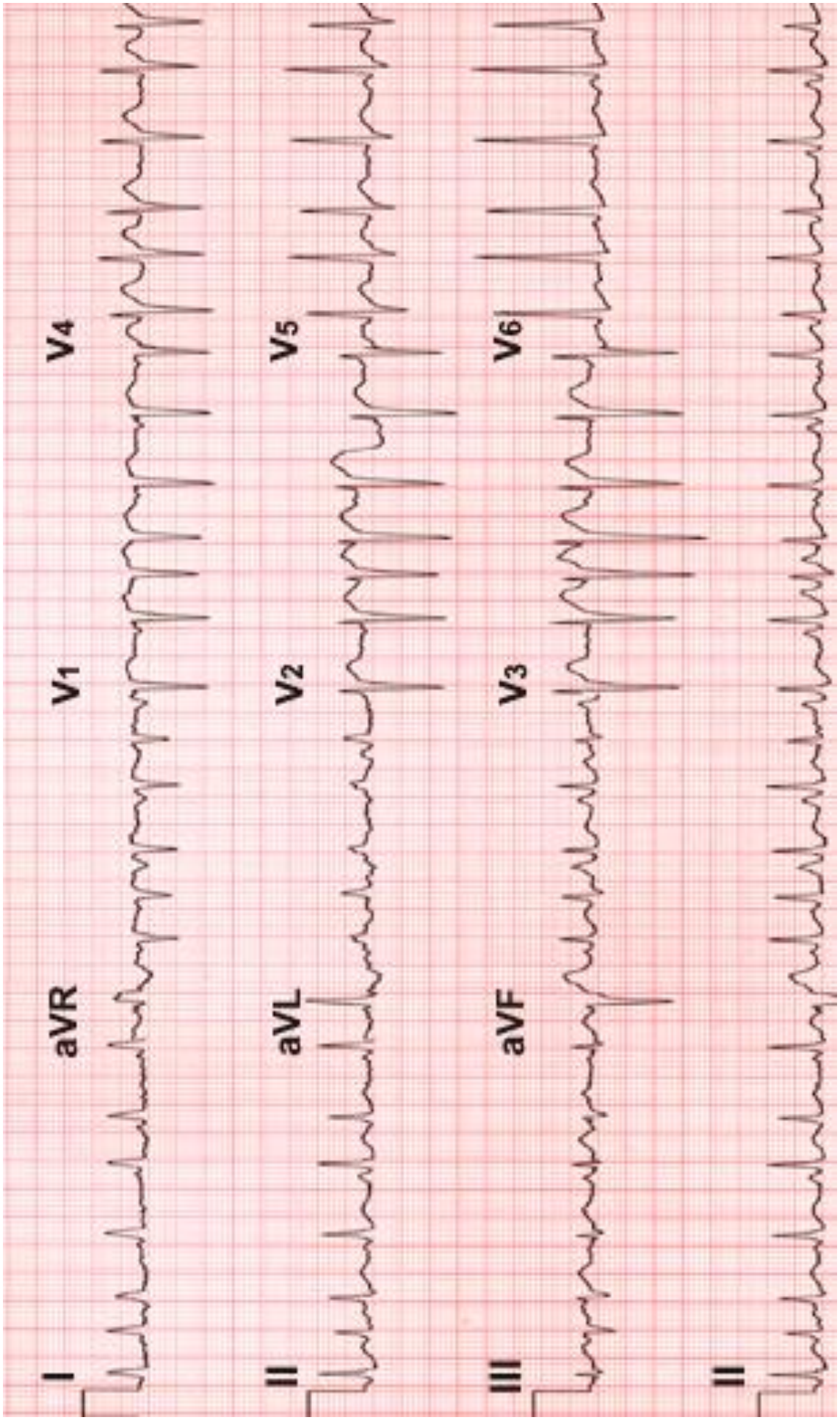
Trial Fellowship Exam

Short Answer Questions (SAQ)

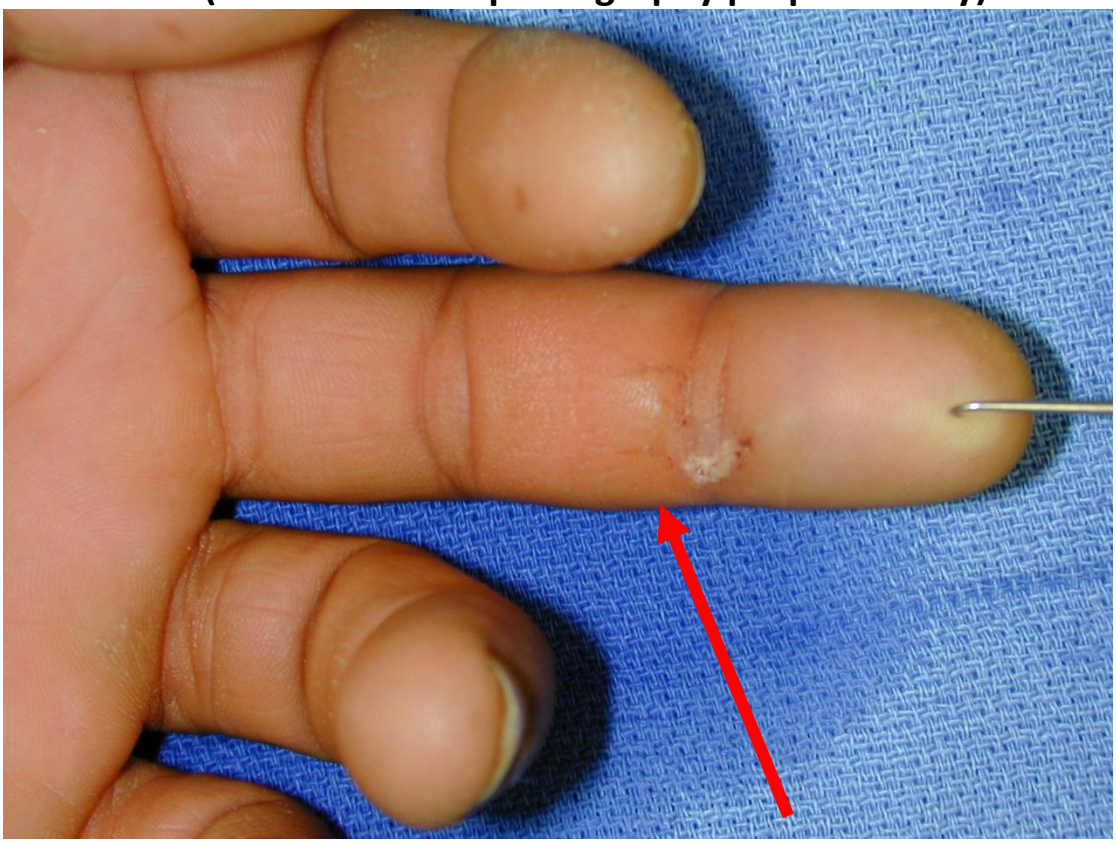
Week 24

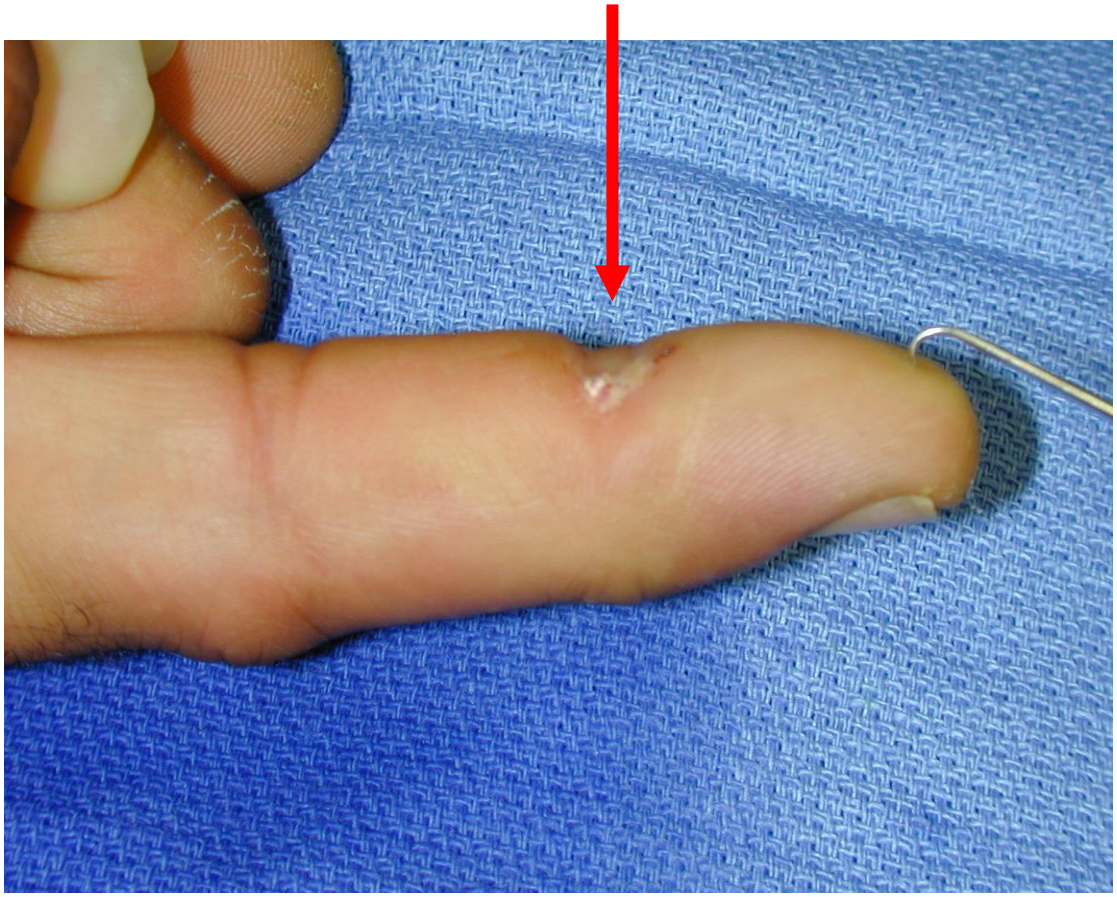
PROP BOOKLET

Question 5



Question 6 **Red arrow marks the point of entry**
(metal hook for photography purposes only)





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

UNIVERSITY HOSPITAL, GEELONG
FELLOWSHIP WRITTEN EXAMINATION
WEEK 24– 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 (12 marks)

A 3 year old boy presents to your emergency department after taking 10 Lomotil tablets approximately 1 hour ago.
NB: (Diphenoxylate-atropine- well known trade names may be used in the exam rather than generic)

a. What is the role of decontamination for this patient? Include two (2) points in your answer. (2 marks)
   - **Charcoal is indicated**
   - **If patient cooperative and alert**
   - **Not required for favourable outcome**
   - **May reduce naloxone requirement**
   - **May reduce LOS**

b. List four (4) examination features that you would expect at this stage. (4 marks)
   - **Opiod:**
     - **Decreased GCS**
     - **Respiratory depression**
     - **Miosis**
   - **Anticholinergic:**
     - **Delirium/ agitation**
     - **Tachycardia**
     - **Urinary retention**
     - **(dry skin)**

All of these features are present. You assess the patient to have severe toxicity.

c. State five (5) key steps in the management of this patient. (5 marks)
   - **Naloxone bolus**
   - **Naloxone infusion**
   - **Support A/B as required- not likely to require intubation**
   - **Admit to HDU facility- continuous non invasive monitoring**
   - **Feedback to family about safe storage of medications**

d. State the time frame that you would expect the patient to require hospitalisation, if the patient experiences no further complications of his ingestion. (1 mark)
   - **> 48 hrs**

Question 2 (18 marks)

You are the consultant in charge of a regional base hospital ED. A 17 year-old girl is brought in by her parents with a 6-month history of weight loss.

a. State the four (4) key components to the diagnosis of Anorexia Nervosa. (4 marks)

NB: DSM-5 is undergoing a review and this answer should be updated in near future. Below is the answer based on recommended texts:

- **Self induced wt loss/restriction of energy intake**
- **Body wt > 15% below expected or BMI < 17.5**
- **Pathological/ intense fear of wt gain/ becoming fat**
- **Body image distortion**
- (amenorrhoea ≥ 3 menstrual cycles- this obviously cannot be applied to males, pre/post menstrual females)
- (associated endocrine dysfunction)

b. List seven (7) examination findings that you would seek on examination for this patient. (7 marks)

- **BMI- ht/wt- REQUIRED**
- **Loss of subcutaneous fat**
- **Hypotension**
- **Bradycardia**
- **Hypothermia**
- **Signs of CCF**
- **Reduced capillary refill**
- **Hair loss**
- **Teeth- enamel loss from vomiting**
- **Parotid gland swelling**
- **Insensitivity to pain**
- **Skin sores**
- **Poor healing with malnutrition**
- **Evidence of self harm – often associated**
- **Hyporeflexic**
- **Gen weakness**
- **Examination for possible alternative causes for wt loss- cancers- skin, breast, abdominal**

University Hospital, Geelong Fellowship Exam Short Answer Questions
Week 24

A rapid assessment nurse has organised a venous blood gas which shows a serum K^+ level of 2.2.

c. State three (3) clinical factors that would lead you to choose IV replacement as the route of choice. (3 marks)

- **Extreme weakness**
- **Cardiac arrhythmias**
- **Dehydration requiring IV therapy**
- **Vomiting**

d. State one (1) pro and one (1) cons for oral and IV route for potassium replacement. (4 marks)

Route	Pros	Cons
Oral	• **Rapid absorption (chlorvescent)** • **More acceptable to pt** • **Avoids risks of IV**	• **Unpleasant taste** • **May refuse oral intake**
Intravenous	• **Avoids compliance issues** • **Titratable to repeat VBG measurements**	• **OD- incorrect rate-cardiac arrhythmias/death** • **Pain at site** • **Fast rates require CVC**

Potential additional Q:
Before you implement your management plan, she asks to get dressed and discharge herself.

State five (5) key issues in this situation. (5 marks)

- **Autonomy vs Duty of Care**
- **Determine level of Competence**
- **Assessment has not been completed**
- **Reasons for wanting to leave**
- **Attempt to secure pt's trust and confidence**
- **Address these reasons if possible**
- **Seek assistance: NOK, nursing**
- **Empower pt with options**
- **Involuntary intervention only if indicated and legally empowered**

Question 3 (18 marks)

A 35 year old woman presents with suspected thyroid storm.

a. State four (4) diagnostic features of thyroid storm. (4 marks)

- **Biochemical evidence of hyperthyroidism (↑ T4 +/- T3 and ↓TSH)**
- **Temp ≥ 37.8 °C**
- **Altered mental state**
- **Cardiovascular dysfunction - egTachycardia out of proportion to fever (usually 120-140)**

NB: no widely accepted absolute criteria

b. List three (3) likely precipitants for thyroid storm. (3 marks)

- **UnDx/ under Rx Graves**
- **Withdrawal of anti-thyroid drugs**
- **Infection**
- **AMI**
- **DKA**
- **Sx- thyroid or elsewhere**
- **Iodine administration**
- **Thyroxine toxicity**
- **Vigorous palpation of the thyroid gland**

c. List three (3) medications that may be used for this patient. For each medication, state one (1) reason why this medication is used. (6 marks)

Medication (3 marks)	Why is this medication used? (3 marks)
BBlocker- propranolol is the usual agent	control the symptoms and signs induced by increased adrenergic tone Blocks central and peripheral
thionamide	block new hormone synthesis
iodinated radiocontrast agent	inhibit the peripheral conversion of thyroxine (T4) to triiodothyronine (T3)
Glucocorticoids	reduce T4-to-T3 conversion, promote vasomotor stability, and possibly treat an associated relative adrenal insufficiency
Bile acid sequestrants	decrease enterohepatic recycling of thyroid hormones

d. List one (1) medication that is specifically contraindicated in thyroid storm (1 mark)

- **Aspirin** (displaces T4 from thyroglobulin)

e. Other than intravenous fluids and oxygen, list four (4) non-medicinal treatments that may be utilised for this patient. (4 marks)

- **External cooling**
- **DC cardioversion for arrhythmias**
- **Peritoneal dialysis**
- **Plasmapheresis**
- **Charcoal haemoperfusion**

Question 4 (12 marks)

A 64 year old male is involved in a high speed, roll over motor car collision. He was unrestrained. On ambulance handover he has obvious bilateral femur #, widespread chest and abdominal bruising and a suspected pelvic fracture. Time from injury on arrival is 90 minutes. He has received 4 L Ns prehospital.
His observations on arrival are: BP 60/30 mmHg HR145 bpm RR30 bpm Sats 90%15L via non rebreather mask

a. Based on the CRASH-2 study findings, state four (4) points relating to the use of Tranexamic acid for this patient. (4 marks)
    - **Indicated as he is in haemorrhagic shock (most likely)/ at risk of severe haemorrhage**
    - **Should be given as early as possible**
    - **1g over 10 min, then 1g over 8 hr**
    - **Most benefit in severe shock group- applicable to this pt**
    - **Not expected to affect blood requirements**
    - **Not expected to affect need for OT**

b. State two (2) criticisms for the CRASH-2 study. (2 marks)
    - **TXA2 group got more FVIIa**
    - **most benefit appeared to be in the severe shock group**
    - **many of the centers were in developing countries**

You are situated in an outer suburban hospital. After discussion with the regional retrieval service, it is decided to transfer the patient to a trauma centre 30 minutes by road.
It is requested that you arrange placement of a REBOA prior to transport.

c. What is REBOA? (1 marks)
    - **Resuscitative balloon occlusion of the aorta**
    - **Insertion of an intra-aortic balloon to reduce distal blood flow**

d. List three (3) features of this patient that may support the use of a REBOA. (3 marks)
    - **Haemorrhagic shock**
    - **Suspected severe pelvic injury**
    - **Time to definitive Rx < 60-90 minutes**

e. In general, list two (2) specific indications for a Zone 1 REBOA. (2 marks)
    - **High grade injury of liver (≥ Grade 3)**
    - **High grade injury of spleen (≥ Grade 3)**
    - **High grade injury of kidney (≥ Grade 3)**
    - **Mesenteric disruption**
    - **Named abdominal vessel injury**

CRASH-2 Trial Collabaorators (2010) "Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial" Lancet 276:23-32

- DB MCRCT (274 hospitals, 40 countries)
- n = 20,211 adults within 8 hours of injury (blunt and penetrating) at risk of severe haemorrhage or in haemorrhagic shock
- Intervention: tranexamic acid 1g over 10 min then 1g over 8h IV
- Control: placebo
- Primary outcome: all cause mortality within 4 weeks of injury (bleeding, vascular occlusion – MI, CVA, PE, MOF, HI, other)
- secondary outcomes: vascular occlusive events (MI, CVA, PE, DVT), surgical intervention (neurosurgery, thoracic, abdominal, pelvic), receipt of blood transfusion, units of blood products transfused, degree of dependency, FVIIa use and GI bleeding
- Results:
  -> all cause mortality reduced in the TXA2 group
  -> decreased mortality due to bleeding (RR 0.85) (which was 35% of deaths)
  -> trend toward more vascular occlusive events in placebo group
  -> no difference in transfusion and need for surgery
  -> trend towards early treatment being more effective
  -> NNT 65, ARR 1.5%, RR 0.91
- Commentary and criticisms:
  — TXA2 group got more FVIIa
  — most benefit appeared to be in the severe shock group
  — many of the centres were in developing countries

CRASH 2 a priori subgroup analysis 2011

- benefit for tranexamic acid was greater if given early
- NNT 125 (RR 0.68) for death from bleeding if given within 1 hour
- benefit up to 3 hours post-injury
- causes harm if given later than 3 hours

This article published in J Trauma (74(6), May 2013, p 1587–1598) gives an excellent summary of the current evidence as of 5/2013

Summary: What Do We Know?

- TXA is associated with a 1.5% reduction in 28-day all-cause mortality in adult trauma patients with signs of bleeding (SBP < 90 mm Hg, heart rate > 110 beats per minute, or both, within 8 hours of injury) in a large pragmatic prospective randomized placebo-controlled trial.
- What is critical is the modest effect on the overall population: All-cause mortality was "significantly" reduced from 16.0% to 14·5% (NNT, 67). The risk of death caused by bleeding overall was "significantly" reduced from 5.7% to 4·9% (NNT, 121).
- TXA signal for benefit was in the most severe shock group (admission SBP <= 75 mm Hg), 28-day all-cause mortality of 30.6% for the TXA group versus 35.1% for the placebo group (RR, 0.87; 99% CI 0.76–0.99).
- 1,063 deaths (35%) were caused by bleeding in the CRASH-2 Trial.
- TXA had greatest impact on reduction of death caused by bleeding in the severe shock group (SBP <= 75 mm Hg) (14.9% vs. 18.4%; RR, 0.81; 95% CI, 0.69–0.95).
- Early TXA (<=1 hour from injury) was associated with the greatest reduction (32% reduction) in deaths caused by bleeding (5.3% vs. 7.7%; RR, 0.68; 95% CI, 0.57–0.82; p < 0.0001).
- TXA given between 1 hour and 3 hours after injury also reduced the risk of death caused by bleeding (4.8% vs. 6.1%; RR, 0.79; 95% CI, 0.64–0.97; p = 0.03).
- TXA given after 3 hours after injury was associated with an increased risk of death caused by bleeding (4.4% vs. 3.1%; RR, 1.44; 95% CI, 1.12–1.84; p = 0.004).
- TXA had no impact on TBI outcomes, but the study was limited by small sample size.
- TXA treatment is not associated with an increased risk of vascular occlusive events.

What Is Still Unknown?

- Whether TXA has any impact on trauma outcomes when damage-control resuscitation or MT protocols are used;
- The mechanism by which TXA reduced mortality in trauma in the CRASH-2 Trial. Fibrinolysis assessment and coagulation testing were not part of the study design, and determination of time to cessation of hemorrhage was not required in the study;
- Whether fibrinolysis testing should be performed before consideration of TXA treatment;
- What is the optimal dose and timing of TXA in trauma;
- Whether other antifibrinolytic agents could be substituted for TXA use in trauma;

- Whether TXA is associated with higher seizure rates in trauma or TBI patients. Increased postoperative seizures have been reported in cardiac surgery with TXA doses that are 2-fold to 10-fold higher than those used in CRASH-2.75–80 These seizures have been associated with an increased incidence of neurologic complications (delirium and stroke), prolonged recovery, and higher mortality rates. A proposed mechanism for seizures is TXA-mediated inhibition of glycine receptors as a potential cause of neurotoxicity.81,82 A recent warning has been added to the FDA drug label: “Convulsions have been reported in association with tranexamic acid treatment.”83

A Rational Approach for TXA use in Trauma

- In adult trauma patients with severe hemorrhagic shock (SBP <= 75 mm Hg), with known predictors of fibrinolysis, or with known fibrinolysis by TEG (LY30 > 3%);
- Only administer TXA if less than 3 hours from time of injury;
- TXA administration: 1 g intravenously administered over 10 minutes, then 1 g intravenously administered over 8 hours.

MATTERS study

- retrospective observational study (i.e. low quality evidence)
- benefit found for tranexamic acid in the military setting (Camp Bastion, Afghanistan)
- included patients who required transfusion and were given tranexamic acid
- decreased amount of transfused PRBCs needed if tranexamic acid given

MATTERS 2 study

- retrospective observational study (i.e. low quality evidence)
- military setting (Camp Bastion, Afghanistan)
- synergistic decrease in mortality with tranexamic acid and cryoprecipitate
- mortality was 14.4% for TXA + cryo vs 28.8% if neither used
- despite higher ISS scores (severity of injury) in the intervention group

The MATTERs II study expanded the sample size of the MATTERs I study to further evaluate TXA and trauma outcomes. A review of 1,332 patients (identified from prospectively collected UK and US trauma registries) who required one or more RBC unit transfusion were analyzed to examine the impact of cryoprecipitate (CRYO) in addition to TXA on survival in combat injured patients.
Despite greater ISSs and RBC transfusion requirements, mortality was lowest in patients who received TXA (18.2%) or TXA/CRYO (11.6%) compared with CRYO alone (21.4%) or no-TXA/CRYO (23.6%). Logistic regression analysis confirmed that TXA and CRYO were independently associated with a similarly reduced mortality (OR, 0.61; 95% CI, 0.42–0.89; p = 0.01 and OR, 0.61; 95% CI, 0.40–0.94; p = 0.02, respectively). The combined TXA and CRYO effect versus neither in a synergy model had an OR of 0.34 (95% CI 0.20–0.58; p < 0.001), reflecting nonsignificant interaction (p = 0.21).

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

Original article

OPEN ACCESS

Resuscitative endovascular balloon occlusion of the aorta (REBOA): a population based gap analysis of trauma patients in England and Wales

Edward Benjamin Graham Barnard,[1,2] Jonathan James Morrison,[3,4] Ricardo Mondoni Madureira,[5] Robbie Lendrum,[6] Marisol Fragoso-Iñiguez,[7] Antoinette Edwards,[7] Fiona Lecky,[7,8] Omar Bouamra,[7] Thomas Lawrence,[7] Jan Olaf Jansen[9,10]

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ABSTRACT

Introduction Non-compressible torso haemorrhage (NCTH) carries a high mortality in trauma as many patients exsanguinate prior to definitive haemorrhage control. Resuscitative endovascular balloon occlusion of the aorta (REBOA) is an adjunct that has the potential to bridge patients to definitive haemostasis. However, the proportion of trauma patients in whom REBOA may be utilised is unknown.

Methods We conducted a population based analysis of 2012–2013 Trauma Audit and Research Network (TARN) data. We identified the number of patients in whom REBOA may have been utilised, defined by an Abbreviated Injury Scale score ≥3 to abdominal solid organs, abdominal or pelvic vasculature, pelvic fracture with ring disruption or proximal traumatic lower limb amputation, together with a systolic blood pressure <90 mm Hg. Patients with non-compressible haemorrhage in the mediastinum, axilla, face or neck were excluded.

Results During 2012–2013, 72 677 adult trauma patients admitted to hospitals in England and Wales were identified. 397 patients had an indication(s) and no contraindications for REBOA with evidence of haemorrhagic shock: 69% men, median age 43 years and median Injury Severity Score 32. Overall mortality was 32%. Major trauma centres (MTCs) received the highest concentration of potential REBOA patients, and would be anticipated to receive a patient in whom REBOA may be utilised every 95 days, increasing to every 46 days in the 10 MTCs with the highest attendance of this injury type.

Conclusions This TARN database analysis has identified a small group of severely injured, resource intensive patients with a highly lethal injury that is theoretically amenable to REBOA. The highest density of these patients is seen at MTCs, and as such a planned evaluation of REBOA should be further considered in these hospitals.

Key messages

What is already known on this subject?
- Haemorrhage is the leading cause of potentially survivable death in trauma.
- Resuscitative endovascular occlusion of the aorta (REBOA) has been shown in large animal models to improve survival in non-compressible torso haemorrhage but early clinical data are equivocal.

What might this study adds?
- The number of patients in England and Wales in whom REBOA may be utilised is small, but they have a mortality of 32%.
- The highest density of potential REBOA patients is seen at major trauma centres.

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To cite: Barnard EBG, Morrison JJ, Madureira R M, *et al*. *Emerg Med J* 2015;32:926–932.

INTRODUCTION

Haemorrhage is the leading cause of potentially preventable death following traumatic injury.[1–5] Non-compressible torso haemorrhage (NCTH), defined as vascular disruption to the axial torso vessels, solid organs, pulmonary parenchyma or the bony pelvis, accompanied by shock,[6] has a mortality of approximately 45%.[9 10] A significant proportion of these deaths occur before the opportunity for definitive haemorrhage control in an operating theatre or interventional radiology suite.[2 10] There is therefore a need for a haemorrhage control adjunct, to achieve temporary cessation of bleeding, until definitive haemostasis can be achieved.

Resuscitative endovascular balloon occlusion of the aorta (REBOA) involves the temporary occlusion of the aorta, using a percutaneously deployed intravascular balloon, usually inserted via the femoral artery. REBOA has demonstrated potential application in large animal models and several human studies. In animal models of NCTH, REBOA has been shown to improve survival,[11 12] increase blood pressure (BP),[11 13 14] brain oxygenation and carotid arterial blood flow.[14 15] REBOA has also been demonstrated to be superior to haemostatic gauze in the control of pelvic haemorrhage.[12] In two human case series, of 13 and 6 patients, survival from presumed lethal NCTH was shown to be between 46% and 67%.[16 17] However, more recent publication of a large, retrospective, propensity score matched data study from the Japan Trauma Data bank suggested that the use of REBOA in blunt abdominal trauma is associated with increased mortality (OR of survival 0.3, 95% CI 0.23 to 0.40).[18] The decision to use REBOA in this study was based on the physicians' clinical decision making (ie, not according to a clinical practice

SYSTEMATIC REVIEW

A systematic review of the use of resuscitative endovascular balloon occlusion of the aorta in the management of hemorrhagic shock

Jonathan James Morrison, MD, PhD, Richard E. Galgon, MD, MS, Jan Olaf Jansen, FRCS, FFICM, Jeremy W. Cannon, MD, SM, Todd Erik Rasmussen, MD, *and* **Jonathan L. Eliason, MD,** *Glasgow, United Kingdom*

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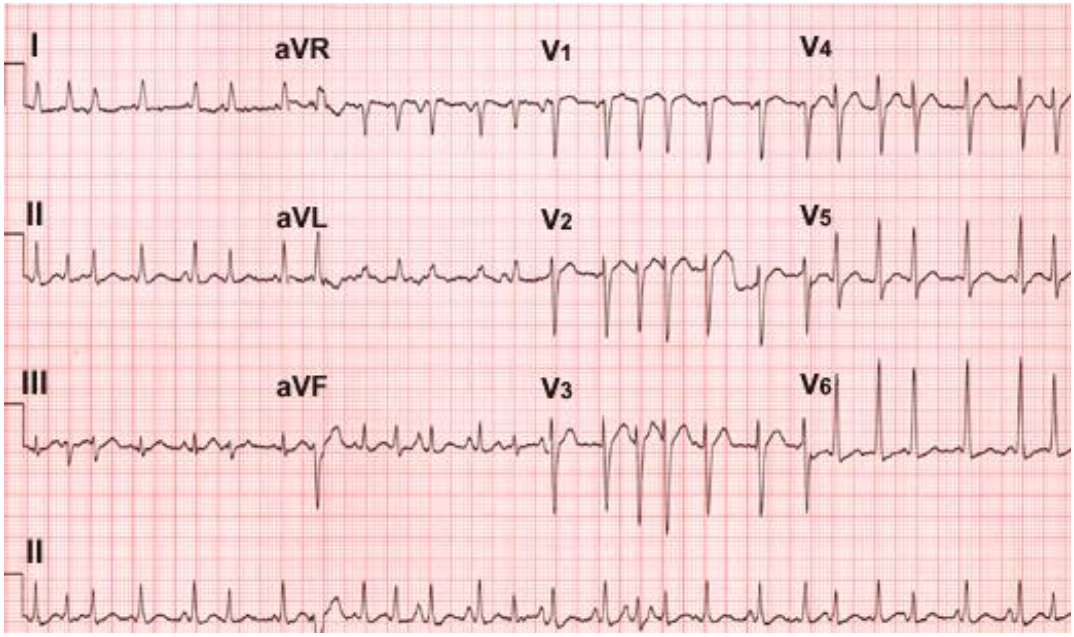
Question 5 (11 marks)

A 75 year old man presents with palpitations.

On examination: BP 140/ 60mmHg supine RR 40/ min Oxygen saturation 88% on 6L via Hudson mask
GCS 15



i) What is the ECG diagnosis? (1 mark)
- **Multifocal atrial tachycardia (MFAT)**

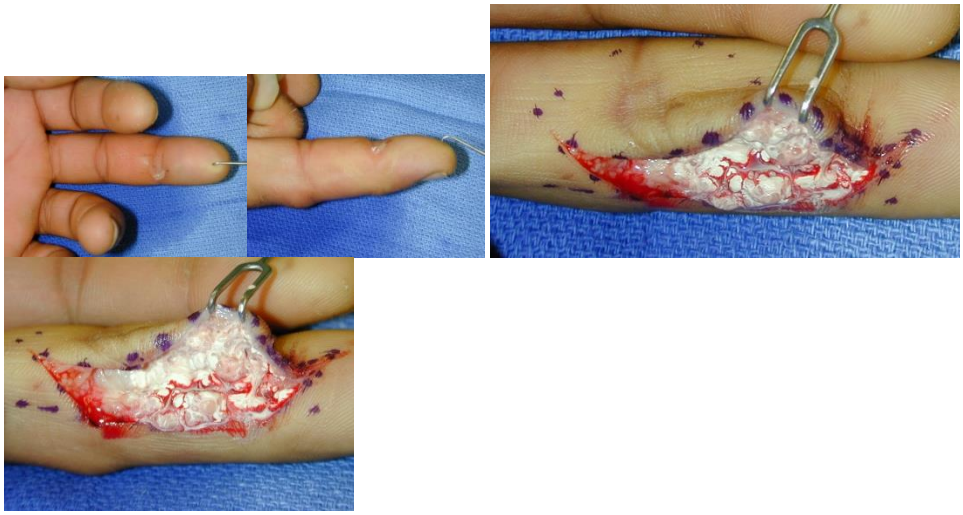
ii) State three (3) abnormalities on this ECG to support this diagnosis. (3 marks)
- **At least 5 atrial foci (≥3 for diagnosis)**
- **Ventricular rate > 100 (variable 130-170 here)**
- **Variable PP, PR, RR intervals**

iii) List four (4) likely causes for these ECG changes. (4 marks)
- **Severe airways disease**
- **Digitalis toxicity**
- **Theophylline toxicity**
- **Large PE**
- **Severe hypoxia**
- **Diabetes**

iv) What is the clinical relevance of this ECG diagnosis? State three (3) points in your answer. (3 marks)
- **Usually associated with serious illness/ respiratory failure**
- **Resolves with Rx of underlying disorder**
- **Poor prognostic sign (60% in hospital mortality, mean survival 1 yr- due to underlying disease, not arrhythmia itself)**

Question 6 (12 marks)

A 32 year old man, John Smith (UR 123456), presents to your emergency department following a high pressure injury to his left middle finger, 1 hour ago. **(Same pt at Sx shown below)**



a. List three (3) pathophysiological mechanisms for harm from this mechanism of injury. (3 marks)

- **Direct tissue injury/ inflammation from:**
  - **noxious material injected into pulp/flexor sheaths- progressive necrosis**
  - **chemical injury- local anaesthesia**
  - **heat- burn**
- **Ischaemia as a result of tissue under marked tension**
- **Infection**

b. List three (3) factors associated with a poor outcome from this injury. (3 marks)

- **Fuel/ paint injected** (70% amputation rate)
- **Distal fingertip injuries**
- **Low viscosity agents** (greater tissue spread/penetration)
- **Contamination/ waste water**
- **Delay to operative intervention**
- **Placement of ring block** (increases tension in tissue and worsens ischaemia)

You decide to refer the patient after your care.

c. Using an ISBAR approach, list five (5) pieces of information that you would pass onto the receiving Doctor. (5 marks)

- Identify- **My name, Emergency registrar, Pt John Smith 32M UR 123456** (Who you are and what is your role?) (Patient identifiers- at least 3) (2 marks)
- Situation- **High pressure injury, to L middle finger** (What is going on with the patient?)
- Background- **Details of injectant, 1 hour ago** (What is the clinical background/context?)
- Assessment- **Critically urgent (digit/ limb threatening) problem** (What do I think the problem is?)
- Recommendation- **Urgent review required with a view to urgent Sx.** (What would you recommend?)


COMMUNICATING WITHIN YOUR HEALTH CARE TEAM
CLINICAL DETERIORATION
INTRODUCTION
Introduce yourself, your role and location
Identify the patient
SITUATION
State the immediate clinical situation
BACKGROUND
Provide relevant clinical history and background
Presenting problems and clinical history
ASSESSMENT
Work through A-G physical assessment
What clinical observations are of particular concern?
What do you think the problem is?
Remember to have current observations and information ready!
RECOMMENDATION
What do you want the person you have called to do?
What have you done?
Be clear about what you are requesting and the timeframe
Repeat to confirm what you have heard
CLINICAL HANDOVER
INTRODUCTION
Introduce yourself, your role and location
Identify team leader
Clearly identify patient and family and carer if present
SITUATION
State the immediate clinical situation
State particular issues, concerns or risks
Identify risks - Deteriorating patient, Falls risk, Allergies, limitation to resuscitation
BACKGROUND
Provide relevant clinical history referring to medical record and/or eMR
ASSESSMENT
Work through A-G physical assessment
Refer to observations, medication and other patient charts
Summarise current risk management strategies
Have observations breached CERS criteria?
RECOMMENDATION
Recommendations for the shift
Refer to medical record or eMR
Provide expected date of discharge
What further assessments and actions are required by who and when
State expected frequency of observations
Request that receiver read back important actions required
NSW Health
Sydney Local Health District

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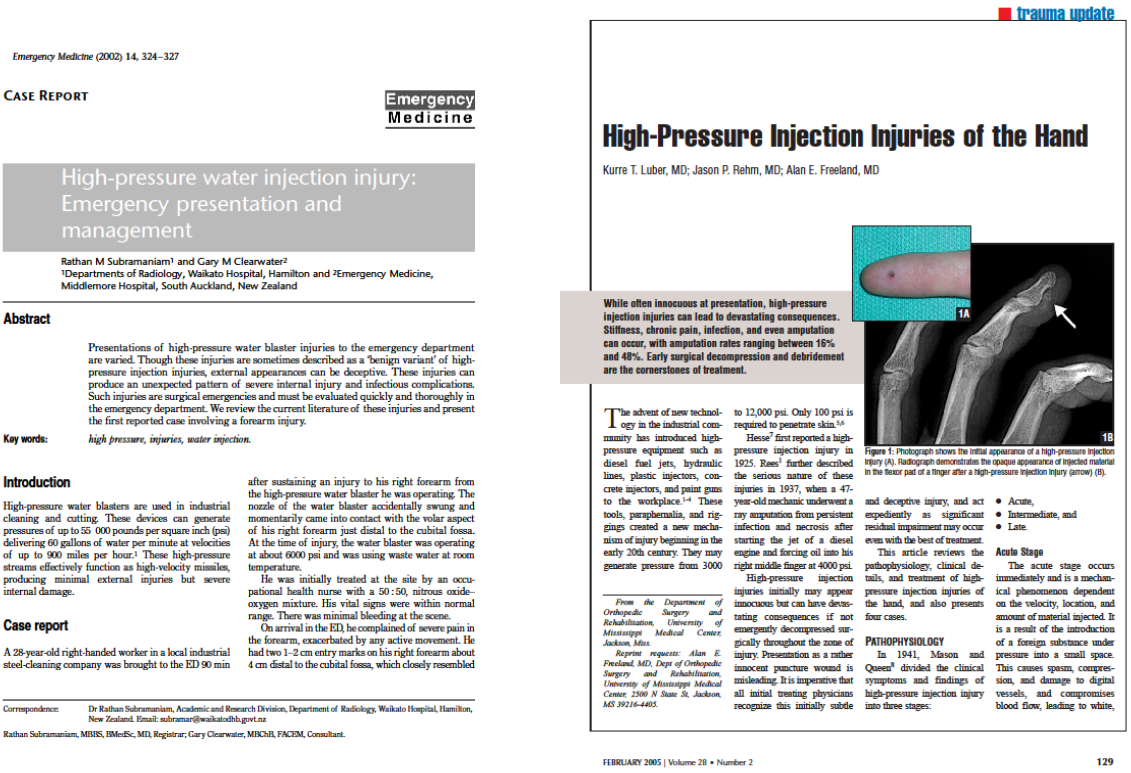

Emergency Medicine (2002) 14, 324–327

CASE REPORT

Emergency Medicine

High-pressure water injection injury: Emergency presentation and management

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[1]Departments of Radiology, Waikato Hospital, Hamilton and [2]Emergency Medicine, Middlemore Hospital, South Auckland, New Zealand

Abstract

Presentations of high-pressure water blaster injuries to the emergency department are varied. Though these injuries are sometimes described as a 'benign variant' of high-pressure injection injuries, external appearances can be deceptive. These injuries can produce an unexpected pattern of severe internal injury and infectious complications. Such injuries are surgical emergencies and must be evaluated quickly and thoroughly in the emergency department. We review the current literature of these injuries and present the first reported case involving a forearm injury.

Key words: *high pressure, injuries, water injection.*

Introduction

High-pressure water blasters are used in industrial cleaning and cutting. These devices can generate pressures of up to 55 000 pounds per square inch (psi) delivering 60 gallons of water per minute at velocities of up to 900 miles per hour.[1] These high-pressure streams effectively function as high-velocity missiles, producing minimal external injuries but severe internal damage.

Case report

A 28-year-old right-handed worker in a local industrial steel-cleaning company was brought to the ED 90 min after sustaining an injury to his right forearm from the high-pressure water blaster he was operating. The nozzle of the water blaster accidentally swung and momentarily came into contact with the volar aspect of his right forearm just distal to the cubital fossa. At the time of injury, the water blaster was operating at about 6000 psi and was using waste water at room temperature.

He was initially treated at the site by an occupational health nurse with a 50:50, nitrous oxide–oxygen mixture. His vital signs were within normal range. There was minimal bleeding at the scene.

On arrival in the ED, he complained of severe pain in the forearm, exacerbated by any active movement. He had two 1–2 cm entry marks on his right forearm about 4 cm distal to the cubital fossa, which closely resembled

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High-Pressure Injection Injuries of the Hand

Kurre T. Luber, MD; Jason P. Rehm, MD; Alan E. Freeland, MD

While often innocuous at presentation, high-pressure injection injuries can lead to devastating consequences. Stiffness, chronic pain, infection, and even amputation can occur, with amputation rates ranging between 16% and 48%. Early surgical decompression and debridement are the cornerstones of treatment.

Figure 1: Photograph shows the initial appearance of a high-pressure injection injury (A). Radiograph demonstrates the opaque appearance of injected material in the flexor pad of a finger after a high-pressure injection injury (arrow) (B).

The advent of new technology in the industrial community has introduced high-pressure equipment such as diesel fuel jets, hydraulic lines, plastic injectors, concrete injectors, and paint guns to the workplace.[1-4] These tools, paraphernalia, and rigging created a new mechanism of injury beginning in the early 20th century. They may generate pressure from 3000 to 12,000 psi. Only 100 psi is required to penetrate skin.[5,6]

Hesse[7] first reported a high-pressure injection injury in 1925. Rees[1] further described the serious nature of these injuries in 1937, when a 47-year-old mechanic underwent a ray amputation from persistent infection and necrosis after starting the jet of a diesel engine and forcing oil into his right middle finger at 4000 psi.

High-pressure injection injuries initially may appear innocuous but can have devastating consequences if not emergently decompressed surgically throughout the zone of injury. Presentation as a rather innocent puncture wound is misleading. It is imperative that all initial treating physicians recognize this initially subtle and deceptive injury, and act expediently as significant residual impairment may occur even with the best of treatment.

This article reviews the pathophysiology, clinical details, and treatment of high-pressure injection injuries of the hand, and also presents four cases.

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PATHOPHYSIOLOGY

In 1941, Mason and Queen[8] divided the clinical symptoms and findings of high-pressure injection injury into three stages:

- Acute,
- Intermediate, and
- Late.

Acute Stage

The acute stage occurs immediately and is a mechanical phenomenon dependent on the velocity, location, and amount of material injected. It is a result of the introduction of a foreign substance under pressure into a small space. This causes spasm, compression, and damage to digital vessels, and compromises blood flow, leading to white,

Historical Aspects

Rees[1], in 1937, was the first to describe a high pressure injection injury and note the potential severity of the injury. He documented the clinical course of a 47 year old mechanic who had a diesel fuel injection injury. The patient initially presented with an apparently innocuous injury. He developed pain after a few hours and then developed a systemic response to the injury with lymphadenitis, leucocytosis and fever. His finger progressed to gangrene within a week and required ray amputation.

In 1941, Mason and Queen[2] described three phases that define the natural history of high pressure injection injuries (early, intermediate and late) and their description is still in use today.

The prognosis for these injuries was traditionally so poor that Kaufman[3] in 1968 advocated amputation of the digit as the primary treatment.

History of Illness

Many types of high pressure injection device are now in frequent use within an industrial setting. The minimum pressure required to breach intact human skin is 100psi or 7 x10^5NM^2 (7 bar)[4] but pressures may exceed 2500 bar (35500 lbs/in^2). Most injuries are caused by grease guns, spray guns and diesel injectors but pneumatic hoses, plastic moulding or cement injectors, hydraulic lines, grease boxes, vaccination equipment and oil rig drilling devices can all produce these injuries. These devices are used, amongst other things, in painting, lubrication, cleaning, and mass farm immunization. A diverse spectrum of substances may be injected which vary in their local and systemic toxicity. These include paint, paint thinner, oil, diesel fuel, grease, hydraulic fluid, water, plastics, cement or biological vaccines.

Epidemiology

Schoo et al[5] estimated the incidence of high pressure injection injuries to be 1 in 600 hand injuries attending an emergency department. There are no other estimates of its incidence in the literature although it is certainly an uncommon injury, albeit a serious one, particularly if its significance is initially unrecognised.

High pressure injection injuries predominantly affect healthy young men, since they are largely occupational injuries. It is usually the non dominant hand that is affected, with the index finger being the commonest digit affected. However, any area of the body can be affected and there have been reports of injuries to all regions of the body including the scrotum[6].

Injuries to the digits tend to be serious as rapid infusion of a large volume of fluid into a small closed space leads to a rapid increase in interstitial pressure which may compromise the circulation to the digit.

Grease guns are the most common type of equipment involved in these injuries and this may be because its users are less likely to be skilled than those who use other high pressure devices[7].

Pathophysiology

Mason and Queen divided the response to high pressure injection injury into three phases: the early, intermediate and late phases.

The early response is of swelling, numbness and possible vascular insufficiency due to a combination of mechanical and chemical factors that may act synergistically. In injuries producing a greater inflammatory response, such as paint thinner

injuries, chemical inflammation is more likely to be causative of vascular compromise than the mechanical effect. In other injuries the predominant factor is uncertain. The volume of the injected substance itself acts together with the local inflammatory response to raise the interstitial pressure. This may result in vascular occlusion either as a direct effect of the fluid volatising or as a result of venous or arterial compression.
Some materials that produce local tissue destruction and necrosis may do so by lipid dissolution or by protein coagulation. Dickson[8] suggested that in paint thinner injuries, the severe chemical inflammation was secondary to the alkyl benzines in white spirit. Superadded infection might further compromise tissue viability and extend the zone of tissue necrosis and gangrene.
In the intermediate phase, there is the formation of foreign body granulomata or oleomata. This was first described by Hesse in 1925[9] who noted it in Russian recruits who injected themselves subcutaneously with grease to try to avoid national service. These are nodular tumours which are the result of a foreign body reaction to the injected material. Widespread vessel thrombosis occurs with an inflammatory reaction in the adventitia and thrombosis of the vasa vasorum and venae comitantes. This produces coagulative necrosis of the skin and subcutaneous tissue. Fat is lost from fat locules. Damage to the tendon sheath and perineural fibrosis results in late fibrosis and contracture. Oleomata may persist unchanged for years but the associated fibrosis may affect hand function.
The late phase is rarely seen in developed countries. Here the skin over the oleomata breaks down, producing persistent ulcers and sinuses which discharge grease and epithelial debris. They become secondarily infected and so increase inflammatory changes in the skin. There is a theoretical risk of malignant change in these longstanding sinuses.

Presentation

History

The history should alert the clinician to the severity of the injury. The patient may either be aware himself of the severity of the injury or may have been sent to the emergency room by his employer who should have operating instructions for the equipment being used and guidelines as to when to seek attention. Unfortunately, the clinician who is unaware of the potential consequences of these injuries may underestimate them and dismiss them as trivial.
Taking an adequate history of the pressure at which the equipment was operating, the time of the injury and the volume and nature of the material it contained will provide the diagnosis and suggest the likely prognosis.

Presenting complaints

The patient may present without any symptoms since pain is not always initially present. A few hours after the injury, there is increasing pain and the patient may complain of some numbness and discoloration.

Mechanism of Injury

Many studies suggest that inexperience in operating the equipment is a factor. Kaufman[3] found that most of the injuries were in workers who had operated this equipment for less than six months although they may have operated similar low pressure equipment where testing the nozzle on the end of the finger was safe. Typically injury occurs when the gun is being cleaned, the safety nozzle having been removed, or when tested after reassembly or after the nozzle jams.

Physical Examination

Inspection

Early signs are minimal, usually only a puncture wound at the site where the skin has been breached and oozing of the injected substance from the wound. There may be some local swelling. Occasionally the patient may present early with a digit which is pale, cool and numb showing obvious vascular compromise – these injuries do poorly even when appropriately treated. A digital Allen's test may demonstrate digital artery thrombosis but this is unnecessary, and it may be inadvisable to perform this test in this situation.
If the pain appears disproportionate to that expected of the injury, clinical evidence of raised compartment pressures should be sought. If a compartment syndrome is present, pain will be worsened by passively stretching of the muscles in that compartment. Test the anterior forearm compartment by passive wrist and finger extension, the wrist extensors and brachioradialis muscle by passively flexing wrist in ulnar deviation, and the dorsal forearm compartment by simultaneous wrist and digital flexion. Within the hand, test the adductor, thenar, hypothenar, and dorsal and volar interossei compartments and examine for an acute carpal tunnel syndrome.
Later presentation may show greater swelling and stiffness of the digits or a bluish discoloration if the venous circulation is compromised.
If the patient does not present for days or weeks, there may be gangrene present or a swollen, stiff digit with subcutaneous tumours, ulceration or discharging sinuses present. If left unattended, the sinuses become secondarily infected increasing inflammatory changes and fibrosis and producing more stiffness. There is a theoretical risk of malignant change, with squamous cell carcinoma developing within the chronic ulcers.

Palpation

The digit may be tender to touch along the path of the injected material. Sensation may decrease with swelling so there may be reduced two–point discrimination. Capillary refill will be brisk if there is venous compromise or slow or absent if there is arterial compromise. Where large amounts of air are injected, crepitus may be demonstrable.
Later the patient may show a low grade fever. Systemic symptoms are otherwise dependent on the substance injected, with acute renal failure being reported after injection of wax solvent and acute lead intoxication after injection of lead-based paint.

Quantification

Assessment of the severity of the injury is from a combination of history, physical and operative findings. The severity depends on the nature of the material concerned and its distribution. The nature of the material includes its toxicity, its viscosity and its volume. The distribution depends on the site of injection, depth of penetration, anatomical plane in which spread occurs and the ejection pressure. Some of these factors are interdependent.

Toxicity of Injected Material

The toxicity of the material is dependent on its chemical composition. Lipid soluble materials produce a greater inflammatory response and therefore, greater tissue destruction, than grease. They will cause lipid dissolution even when not under pressure.

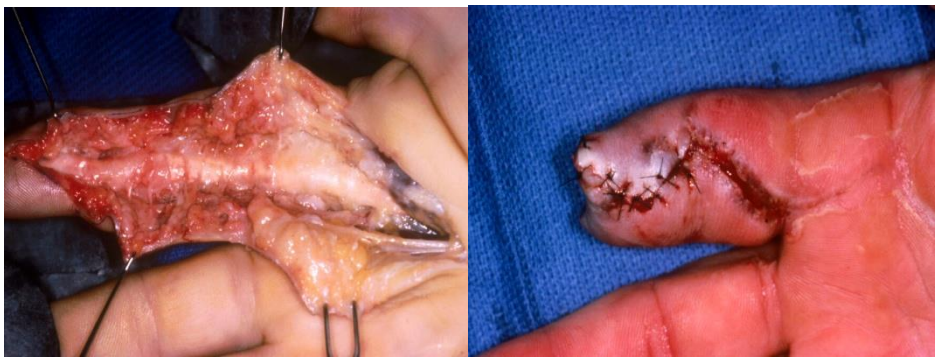

Fig 2(a). Extent of proximal solvent spread after high pressure injection to index finger
Fig 2(b) Outcome of injury

Paint solvents are more toxic than either paint or diesel fuel, resulting in amputation in 80% of cases in one series[5]. Paint is composed of solvents, vehicles and pigments and sometimes bacterial contaminants, all of which contribute to the inflammatory response and tissue destruction. Grease causes less destruction and has less severe inflammatory response so the risk of amputation in the same series was only 20%.

Water and air injuries are usually relatively benign. Even so, water injection injuries can mimic gunshot injuries in their tissue destruction and produce a compartment syndrome. Estimation of their severity should not be based purely on the appearance of the external wound. Bacterial, fungal or chemical inoculation (with sewage or oil lubricant) in water jet injuries may further complicate the clinical picture.
High energy gas injection from firing handgun blank rounds at close range can cause serious injury and gas embolism and death have been reported[10].

Viscosity

The more viscous the material, the less it will spread. Paint, therefore, does not disperse as far as paint solvents which, therefore, affect a greater volume of tissue[11].

Site

Once the material is injected, it travels until it meets resistance. Kaufman[7] using injections into cadaver hands defined clearly the expected course of the material according to the site of injection. The bones, tendons and flexor sheath act as points of resistance which deflect the material causing it to spread superficially through the soft tissues[3]. Deeper spread depends on the anatomical site of injection. If the site of penetration is at the interphalangeal joint crease where the flexor sheath is weak, the substance will travel within the sheath and may therefore spread more proximally directly into the palm or wrist. Spread within the sheath does not appear to affect the prognosis[12]. With pressures exceeding 5-10000 psi, the tendon sheath will always be at risk of penetration. The anatomical arrangement of radial and ulnar bursae makes proximal spread into the wrist more likely if the injection site is into the little finger or thumb.

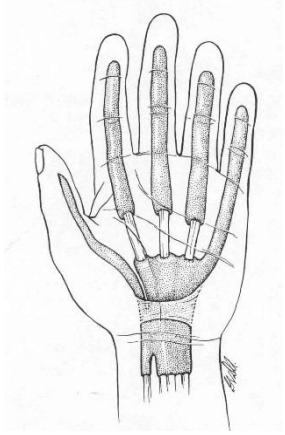

Diagram 1. Simplification of flexor sheath anatomy in the hand

If the puncture wound is eccentric, the dorsal surface of the digit is likely to be extensively involved. Material injected into the thenar or hypothenar spaces is likely to remain these compartments but may involve the intrinsic muscles. In the experimental situation, injection into the mid palmar space failed to show extension proximally into the wrist but extension to the dorsum did occur[7].
Injection distally in the digits carries a worse prognosis, possibly related to the smaller volume of the digits and their lack of distensibility producing a greater rise in interstitial pressure[13]. Kaufmann equated the amount of energy produced in a grease gun injury to a digit to a 1000kg weight falling from a height of 25cm. The velocity of the jet of material emitted may be up to 1550mph (2500km/hr) and the theoretical kinetic energy dissipated on impact may be calculated from the formula, $KE=1/2mv^2$. Therefore, the digits, having a smaller mass will have a greater amount of kinetic energy to absorb and will hence suffer a worse injury than more proximal parts.

Ejection pressure
Grease guns produce pressures of 350-700 bar. Spray guns, that are used in the application of paint, lacquer, semifluid cement, hydraulic fluids and solvents (paint thinner, turpentine or gasoline), operate in the range of 200-500 bar and diesel fuel injectors from 140-400 bar. Water guns operate between 400-550 bar[14] .

Volume
The volume tolerated at different sites of injection is variable. The digits can only tolerate 1cc whilst the palm may tolerate more than 5cc[3]. Chicken vaccine injury, despite being in an oil-based carrier, does not appear as dangerous as pig vaccine perhaps due to their different respective volumes (0.2cc versus 2cc) [15]. A greater volume at the same site is related to poorer functional results[16].

Investigations
Laboratory
After a few hours and particularly with the injection of oil based substances, a leucocytosis may develop.
Sometimes laboratory analysis of the fluid may help in gauging prognosis for recovery or bacteriology in assessing likely infecting organisms.

X-rays
Radiographs are not essential and often add little to the examination. Plain radiographs may give some idea of the degree of dispersion of the substance if it is radio-opaque or if they demonstrate subcutaneous emphysema. This may assist in planning the operative approach. Serial radiographs may be performed intraoperatively to ensure removal of all of the injected material.

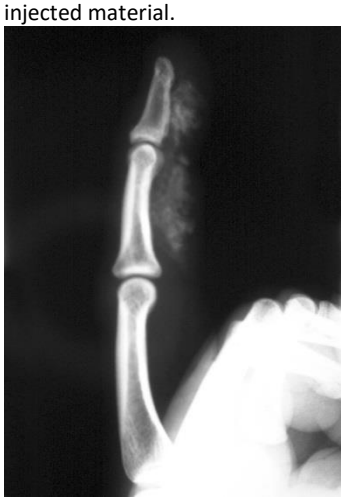

Fig 4. Lateral radiograph showing extent of proximal spread of radio-opaque paint in digit

Classification
The only classification used is that of early, intermediate and late stages of the disease as described by Mason and Queen[2]. Classifying these injuries in relation to the substance injected would be reasonable for the purposes of both treatment and prognosis. The most obvious grouping would be for oil based substances, solvents and paints to be grouped together, all requiring aggressive debridement and medical management, grease injuries to form an intermediate group, all requiring aggressive debridement but not necessarily requiring antibiotics, and water and air injection injuries to form a separate group which may be suitable for conservative management. .

Treatment
Medications and doses
Anti tetanus toxoid should be administered if the patient is not covered but tetanus immunoglobulin is only rarely indicated.
A course of antibiotics, usually a combination of a cephalosporin and an aminoglycoside, is commonly given although the evidence for this is poor[17]. In an experimental model, all organic dyes and all solvents were bacteriocidal, as were some of

the vehicles used in paint although the inorganic dyes had no antibacterial action[18]. Those agents most likely to create a greater inflammatory response were also most likely to be bacteriocidal. This is weighed against the knowledge that the presence of a foreign material in a wound will impair the body's ability to resist infection and even sub-infective quantities of bacteria may result in frank infection, especially where there is any evidence of vascular compromise.
Some authors suggest the use of antiplatelet agents such as aspirin and low molecular weight Dextran to improve the microcirculation to the digit but this is not routine practice.
Non steroidal anti-inflammatory drugs may have some effect at reducing the inflammatory response but any effect is not dramatic[3].
Whether steroids are of any therapeutic benefit is disputed. There is evidence of benefit in animal models[18,13]. In vivo, some authors recommend their use routinely[19] , others use them for all except grease gun injuries where there is minimal tissue extension[12] and others consider them contraindicated due to their depression of the leucocyte response[20] Regional local anaesthetic blockade may be employed to improve the microcirculation by producing peripheral vasodilatation. Digital blocks should be avoided as they may compromise the microcirculation by increasing the interstitial pressure.

Splints
Splintage is used to reduce joint contracture and provide the best position from which to mobilize. The splint needs to be forearm based and maintain the hand in an intrinsic plus position. Night splintage may need to continue for some months following surgery.

Physical Therapy
Hand therapy is required in all cases whether treated surgically or conservatively. Even those who present late and require amputation are likely to require help with mobilization of their hand, as they are frequently left with residual stiffness in adjacent digits.

Conservative management
As a rule, these injuries require expeditious surgical intervention but there are instances where conservative management may be appropriate. The decision should be made on a case by case basis and only by an experienced hand surgeon.
Those cases that may be able to be managed without surgical intervention are those where the material, site and findings are favourable[21]. The few cases in the literature where chicken vaccine has been injected show that, although in an oil carrier, it is usually well tolerated[15]. Air and water injection injuries are also relatively benign[22,23] and may be sometimes treated conservatively with elevation, splintage with or without antibiotics and steroids. Water gun injuries only need decompression if there are signs of a compartment syndrome[14, 24].
Even if a decision is made to treat conservatively, these patients still require admission, careful observation and follow- up. Their digits tend to remain swollen for some weeks and their hands may become extremely stiff.

Surgical management
Surgical exploration should be the mainstay of management for this condition and should occur with the same urgency as for a compartment syndrome.

Surgical surprises
The unwary are especially likely to underestimate both the severity and the extent of this injury (see Fig 7. for the potential for spread in these injuries). The surgical approach should be planned so that proximal extension of the wound is simple.

Prognosis and outcomes of surgery

Multiple factors determine the outcome of these injuries. Death has been reported after abdominal high pressure water injuries that have caused caecal perforation[25] and after air embolism from high pressure air injection[10]. There is a morbidity whether or not the digit is salvaged.

Digital amputation rates in the literature vary from 16%[20] to 48%[5]. It is presumed that the prognosis is worsened if there is any delay to surgery. Several authors have suggested a lower morbidity if the time from injury to decompression is less than ten hours[26,27,17]. Others studies[13] have been unable to confirm this and in some those reaching surgery first appeared more likely to end in amputation[28,12]. In Schoo's series, 16 out of 21 that were amputated, were debrided within 24 hours of the injury and ten in less than six hours. This may be due to the greater severity of their injuries. The time factor may play less of a role in those injuries where chemically induced inflammation rather than pressure is the primary noxiant.

The risk of amputation varies with the material injected with a much worse prognosis for paint and paint thinners than grease probably due to a direct toxic effect on the tissues[12,29]. Schoo et al[5] demonstrated an 80% amputation rate with paint solvents compared to an overall amputation rate of 48% if all materials were included. Gelberman et al[13] had 83% amputation rate with paint injuries compared to 24% with other materials.

The higher the injection pressure of the appliance the more likely amputation will result. In the review by Schoo[5], all cases where the ejection pressure was >7000 psi (500 bar) culminated in amputation. This only consisted of three cases of the 127 reviewed so it is impossible to conclude that injuries at a specific pressure or greater should always be amputated.

Patients who show evidence of initial vascular compromise are likely to result in amputation[12]. Pinto et al[20] had a high digit salvage rate which he attributed to timely aggressive debridement, open wound packing and delayed primary closure rather than an attempt to close the wound primarily.

The volume of injected substance may contribute to the risk of amputation but this is difficult to ascertain as only animal vaccines come in a set volume[27].It is believed that the greater volume of material injected, the worse the prognosis but this is difficult to prove except in the case of animal vaccines where a set volume is given. Injuries to the digits where there is little room for dispersal do worse than more proximal injuries that can tolerate a greater volume of injected material.

Little work has been done documenting the quality of function of the hand following digit salvage. In one series, 92% returned to work with 62% who were considered to have functional hands[20]. Where the digit was salvaged, there was a correlation between the material injected and the time to return to work with grease gun injuries involving a longer rehabilitation period[5].

Christodoulou[28], in his study of fifteen patients an average of 73 months post injury, found that three of the six who had had amputations had changed occupation Only one of the nine with salvaged digits had altered his work. In comparison to the uninjured hand, grip strength was decreased by 15%, lateral key pinch by 23%, and chuck grip by 25%. Dynamic muscle power was reduced by 27%. Sensory evaluation, where it was possible, showed a decrease in sensibility with only one patient having normal sensation. Seven had diminished light touch, three had diminished protective sensation and one had loss of protective sensation.

Outcomes

Complications

Infection may occur despite antibiotic treatment and particularly when necrotic tissue is present. It may act synergistically with other factors to increase the likelihood of amputation or, if the digit is saved, to prolong swelling and stiffness and therefore, the period of rehabilitation. Most authors give antibiotics routinely but reported infection rates vary from 11.5%[13] to 60%[20]. This series had a low rate of digit amputation but in retaining digits there may have been more tissue with compromised vascularity which may have contributed to this high infection rate. Infections are commonly due to Staphylococcus epidermidis or aureus, Pseudomonas sp. or are polmicrobial.

Question 7 (12 marks)

A 32 year old male is involved in a roll over motor car collision. He is known to be taking a NOAC.

a. Under what circumstances would you take measures to reverse the action of the NOAC? List two (2) circumstances. (2 marks)
   - **Medication taken < 12/24 ago +**
   - **Clinically significant life threatening bleeding**

b. What is the role of charcoal in the reversal of a NOAC? State two (2) points in your answer. (2 marks)
   - **Indicated for all clinically significant bleeds**
   - **< 2/24 (up to 4/24 in some recommendations- Apixaban up to 6/24, Rivaroxaban to 8/24)**

c. What is the role of dialysis in the reversal of a NOAC? State two (2) points in your answer. (2 marks)
   - **Dabigatran only- no role in rivaroxiban/ apixaban (highly protein bound)**
   - **Life threatening bleeding**
   - **Renal function impairment or**
   - **aPTT > 80 sec**
   - **or Dabigatran level > 500 mg/ml**

d. Other than Packed cells/ whole blood, state three (3) agents which may be used for reversal of the effects of Dabigatran. (3 marks)
   - **Tranexamic acid**
   - **Prothrombin X**
   - **Idarucizumab** (Humanised monoclonal FAB fragment- biochemical reversal in 1/24, clinical reversal at 12/24 ie = to t ½ of drug)

e. What is the role of thromboelastography for this patient? State three (3) points in your answer. (3 marks)
   - **Re NOAC:**
     - **May have a role in detecting and monitoring NOAC activity** (role still evolving)
   - **Re Traumatic induced coagulopathy:**
     - **Predicts the need for blood transfusion**
     - **Guide transfusion strategy- FFP/Cryoprecipitate/ Platelet/ TxA_2use**

Use of Thromboelastography (TEG) for Detection of New Oral Anticoagulants

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Context.— The clinical introduction of new oral anticoagulants (NOACs) has stimulated the development of tests to quantify the effects of these drugs and manage complications associated with their use. Until recently, the only treatment choices for the prevention of venous thromboembolism in orthopedic surgical patients, as well as for stroke and systemic embolism in patients with atrial fibrillation, were vitamin K antagonists, antiplatelet drugs, and unfractionated and low-molecular-weight heparins. With the approval of NOACs, treatment options and consequent diagnostic challenges have expanded.

Objective.— To study the utility of thromboelastography (TEG) in monitoring and differentiating between 2 currently approved classes of NOACs, direct thrombin inhibitors (dabigatran) and factor Xa inhibitors (rivaroxaban and apixaban).

Design.— Blood samples from healthy volunteers were spiked with each NOAC in both the presence and absence of ecarin, and the effects on TEG were evaluated.

Results.— Both the kaolin test reaction time (R time) and the time to maximum rate of thrombus generation were prolonged versus control samples and demonstrated a dose response for apixaban (R time within the normal range) and dabigatran. The RapidTEG activated clotting time test allowed the creation of a dose-response curve for all 3 NOACs. In the presence of anti-Xa inhibitors, the ecarin test promoted significant shortening of kaolin R times to the hypercoagulable range, while in the presence of the direct thrombin inhibitor only small and dose-proportional R time shortening was observed.

Conclusions.— The RapidTEG activated clotting time test and the kaolin test appear to be capable of detecting and monitoring NOACs. The ecarin test may be used to differentiate between Xa inhibitors and direct thrombin inhibitors. Therefore, TEG may be a valuable tool to investigate hemostasis and the effectiveness of reversal strategies for patients

OVERVIEW OF COAGULOPATHY IN TRAUMA (From LITFL)

- new terms that are in vogue are trauma-induced coagulapathy (TIC) and acute traumatic coagulaopthy (ATC)
- not simply a ‘dilutional coagulopathy’ or ‘consumptive coagulopathy’!

PATHOPHYSIOLOGY

- TIC was conventionally construed simply as depletion, dysfunction or dilution of procoagulant factors
- actually an imbalance of the dynamic equilibrium between procoagulant factors, anticoagulant factors, platelets, endothelium and fibrinolysis
- characterized by isolated factor V inhibition, dysfibrinogenaemia, systemic anticoagulation, impaired platelet function and hyperfibrinolysis
- exacerbated by hypothermia, acidosis (together with coagulopathy they form ‘the lethal triad’) and resuscitation with hypocoagulable fluids

MANAGEMENT

- early detection (ROTEM /TEG holds promise for this)
- early activation of massive transfusion protocols
- aggressive proactive blood product administration (PRBCs, FFP, platelets, cryopreciptitate)
- prevent and treat hypothermia and acidosis
- early use of tranexamic acid
- give calcium if hypocalcaemic
- consider Factor VII if non-surgical bleeding and all the other correctables have been corrected

OVERVIEW of Thromboelastography (TEG) (from LITFL)

- Thromboelastography is a viscoelastic hemostatic assay that measures the global visco-elastic properties of whole blood clot formation under low shear stress
- it shows the interaction of platelets with the coagulation cascade (aggregation, clot strengthening, fibrin cross linking and fibrinolysis)
- does not necessarily correlate with blood tests such as INR, APTT and platelet count (which are often poorer predictors of bleeding and thrombosis)
- This page describes TEG® predominantly, ROTEM® is the alternative viscoelastic hemostatic assay that is widely available

METHOD

- TEG® measures the physical properties of the clot in whole blood via a pin suspended in a cup (heated to 37C) from a torsion wire connected with a mechanical–electrical transducer
- The elasticity and strength of the developing clot changes the rotation of the pin, which is converted into electrical signals that a computer uses to create graphical and numerical output
- point of care test (quick, takes around 30min)
- can be repeated easily and compared and contrasted
- requires calibration 2-3 times daily
- should be performed by trained personnel
- susceptible to technical variations
- kaolin and more recently kaolin + tissue factor (TF) (RapidTEG®) are used as activators, NATEM (TEG® using native whole blood is slower)
- other tests are available including functional fibrinogen, a measure of fibrin-based clot function, and Multiplate which evaluates platelet function

USE

Indications

- prediction of need for transfusion (MA is a useful predictor in trauma)
- guide transfusion strategy

Studies show cost-effectiveness and reduction in blood products in:

- liver transplantation
- cardiac surgery

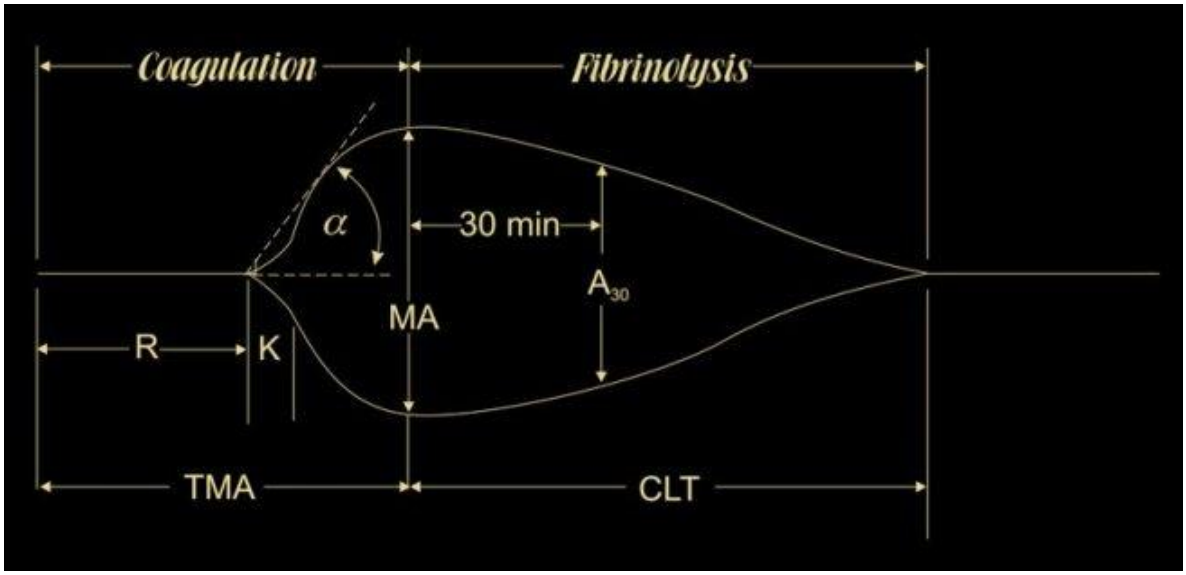
May be useful in:

- trauma (reduction in blood product use and mortality in cohort studies)
- obstetrics (some data to show that it may decrease transfusion rates; this is controversial)
- early detection of dilutional coagulopathy

Hard to interpret in certain situations:

- LMWH
- aspirin
- post cardiac bypass
- fibrinolysis
- hypercoagulability

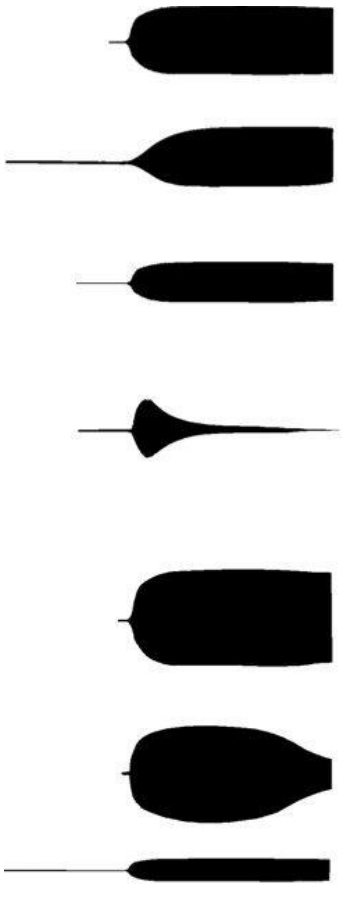
NORMAL TEG



Specific parameters represent the 3 phases of the cell-based model of haemostasis: initiation, amplification, and propagation

- R value = reaction time (s); time of latency from start of test to initial fibrin formation (amplitude of 2mm); i.e. initiation
- K = kinetics (s); time taken to achieve a certain level of clot strength (amplitude of 20mm); i.e. amplification
- alpha = angle (slope between R and K); measures the speed at which fibrin build up and cross linking takes place, hence assesses the rate of clot formation; i.e. thrombin burst
- TMA = time to maximum amplitude(s)
- MA = maximum amplitude (mm); represents the ultimate strength of the fibrin clot; i.e. overall stability of the clot
- A30 or LY30 = amplitude at 30 minutes; percentage decrease in amplitude at 30 minutes post-MA and gives measure of degree of fibrinolysis
- CLT = clot lysis time (s)

IMPORTANT PATTERNS



Normal
R;K;MA;Angle = Normal

Anticoagulants/hemophilia
Factor Deficiency
R;K = Prolonged;
MA;Angle = Decreased

Platelet Blockers
Thrombocytopenia/
Thrombocytopathy
R ~ Normal; K = Prolonged;
MA = Decreased

Fibrinolysis (UK, SK, or t-PA)
Presence of t-PA
R ~ Normal;
MA = Continuous decrease
LY30 > 7.5%; WBCLI30 < 97.5%;
Ly60 > 15.0%; WBCLI60 < 85%

Hypercoagulation
R;K = Decreased;
MA;Angle = Increased

D.I.C
Stage 1
Hypercoagulable state with
secondary fibrinolysis

Stage 2
Hypocoagulable state

TEG AS A GUIDE TO TREATMENT

- Increased R time => FFP
- Decreased angle => cryopreciptate
- Decreased MA => platelets (consider DDAVP)
- Fibrinolysis => tranexamic acid (or aprotinin or aminocaproic acid)

TEG® VERSUS ROTEM®

Comparison

- Two commercial types of viscoelastic tests are available: thromboelastography =TEG® (developed in 1948, now produced in the USA) and rotational thromboelastogram = ROTEM® (from Germany)
- differences in diagnostic nomenclature for identical parameters between the two
- TEG® operates by moving a cup in a limited arc (±4°45' every 5s) filled with sample that engages a pin/wire transduction system as clot formation occur
- ROTEM® has an immobile cup wherein the pin/wire transduction system slowly oscillates (±4°45'every 6s)
- results are not directly comparable as different coagulation activators are used
- ROTEM® is more resistant to mechanical shock, which may be an advantage in the clinical setting

Equivalent variables for ROTEM®

- Clotting time (CT) = *R* value (reaction time)
- *α* angle and clot formation time (CFT) = *K* value and *α* angle
- Maximum clot firmness (MCF) = Maximum amplitude (MA)
- Clot lysis (CL) = LY30

COMPARISON WITH PLASMA CLOTTING TESTS

Pros of viscoelastic hemostatic assays

- assessment of global haemostatic potential provides more information than time to fibrin formation
- can readily differentiate a coagulopathy due to low fibrinogen from one due to thrombocytopenia
- point-of-care (POC) device with rapid turnaround times so that many results available within 5–10 min of starting the test

Cons of viscoelastic hemostatic assays

- variable availability
- marked inter-operator variability and poor precision (UK NEQAS data suggests coefficients of variance ranging from 7.1% to 39.9% for TEG® and 7.0% to 83.6% for ROTEM®)
- may require specialist staff to perform

Question 8 (12 marks)

At what height does acute mountain sickness appear in a person who is not acclimatised to altitude? (1 mark)

- **> 2500 m**

a. List three (3) examination features of a patient with high altitude cerebral oedema. (3 marks)
   - **Lethargy**
   - **Altered consciousness**
   - **Coma**
   - **Truncal ataxia**

b. List three (3) management steps for a patient with high altitude cerebral oedema. (3 marks)
   - **Immediate descent**
   - **Oxygen**
   - **Dexamethasone**
   - **$HB0_2$**
   - **Coma care**

c. List three (3) examination features of a patient with high altitude pulmonary oedema. (3 marks)
   - **Tachycardia**
   - **Tachypnoea**
   - **Cyanosis**
   - **Crepitations**

d. List three (3) management steps for a patient with high altitude cerebral oedema. (3 marks)
   - **Immediate descent**
   - **Oxygen**
   - **Nifedipine**
   - **Maintain normothermia**

Question 9 (12 marks)

a. List three (3) cardinal features of a patient with neuroleptic malignant syndrome. (3 marks)
   - **Neuromuscular rigidity**
   - **Altered mental status**
   - **Autonomic instability**

b. List three (3) risk factors for the development of Neuroleptic Malignant syndrome. (3 marks)
   - **High doses of neuroleptic agent**
   - **Increased dose of neuroleptic agent within the last 5/7**
   - **Large magnitude dose increase**
   - **Parenteral administration**
   - **Simultaneous use of ≥2 neuroleptic agents**
   - **Haloperidol**
   - **Depot Fluphenazine**
   - **Young age**
   - **Male sex**
   - **Psychiatric comorbidity**
   - **Genetic factors**
   - **Pre-existing organic brain disorders**
   - **Dehydration**
   - **High CK levels during episodes of psychosis (not assoc with NMS)**
   - **Other pre-existing medical disorders (trauma, infection, malnutrition, premenstrual, thyrotoxicosis)**

c. List two (2) antidotes that may be beneficial for a patient with Neuroleptic Malignant Syndrome. (2 marks)
   - **Bromocriptine**
   - **Dantrolene**
   - **ECT**

d. Other than antidote use, list four (4) key components to the management of a patient with Neuroleptic Malignant syndrome. (4 marks)
   - **RSI if severe rigidity compromising ventilation/ or temp > 38.5°C**
   - **Correct hypoglycaemia**
   - **Correct hyperthermia- NM paralysis**
   - **Avoid any agent with dopamine antagonist effects**
   - **Rx HT an Tachycardia- Vasodilator (GTN/ nitroprusside)**
   - **+/- Bz (may play a role in the aetiology of NMS- therefore specific agents preferred**