ID NUMBER: ☐ ☐ ☐ ☐ ☐

University Hospital, Geelong

Emergency Medicine

Trial Fellowship Exam

Short Answer Questions (SAQ)

Week 12

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 (18 marks)

a. What is the role of the ROSIER scale in the Emergency Department? State three (3) points in your answer. (3 marks)

1. _____

2. _____

3. _____

A patient presents after a sudden onset of dense right hemiparesis.

b. List five (5) inclusion criteria that must be met for the patient to be considered for thrombolysis. (5 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

Question 1 (continued)

A non-contrast CT brain is taken- refer to the props booklet- page 1.

c. State the diagnosis, based on this CT scan and the clinical features provided.(1 mark)

_____

The patient undergoes thrombolysis and suffers a lethal intracerebral bleed. It is discovered that the patient had a recognised contraindication to thrombolysis.

d. List the five (5) elements involved in the process of open disclosure. (5 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

Question 1 (continued)

e. In general, list four (4) factors that would support the role for decompressive craniectomy. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 2 (12 marks)

A 75 year old woman presents following a fall from standing. She is complaining of bilateral hip pain only.
Her observations are:

BP	75/ 50	mmHg
HR	135	/ min
RR	20	/ min
Oxygen saturations	98%	room air
GCS	13	E3, V4, M6

A pelvis xray is taken- refer to the props booklet- page 2.

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

1. _____

2. _____

3. _____

4. _____

b. List four (4) further imaging studies that you would consider for this patient (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 2 (continued)

c. Assuming that you have IV access, list four (4) steps in your approach to managing her pain control. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 3 (12 marks)

A 25 year old presents following a stab wound to the neck.

a. List four (4) key historical features that are important in this case. (4 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

b. List four (4) examination features that are important in this case. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 3 (continued)

c. As they pertain to the neck, list the boundaries of the following. (3 marks)

Zone 1:_____

_____

Zone:2:_____

_____

Zone 3:_____

_____

d. What feature of the wound would allow definitive repair in the emergency department. (1 mark)

_____

Question 4 (12 marks)

A 3 month old infant presents with shortness of breath and difficulty breathing.
Her observations are:

BP	85/ 50	mmHg
HR	125	/ min
RR	80	/ min
Oxygen saturations	98%	room air
Temperature (rectal)	36.5	°C

a. List four (4) likely differential diagnoses for this patient (each to be from a different pathological category). For each, list the method of confirmation of diagnosis. (8 marks)

	Differential diagnosis (4 marks)	**Method of confirmation of diagnosis (4 marks)**
1		
2		
3		
4		

Question 4 (continued)

b. List four (4) historical factors that would suggest a serious illness for this patient. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 5 (12 marks)

An 87 year man presents from a nursing home with acute deterioration.

An ECG is taken- refer to the props booklet- page 3.

a. What is the most likely diagnosis? (1 mark)

_____

b. List three (3) features of this ECG that support this diagnosis. (3 marks)

1. _____

2. _____

3. _____

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

1. _____

2. _____

3. _____

4. _____

Question 5 (continued)

d. List four (4) treatments that you would consider for this patient. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 6 (12 marks)

A 45 year old man presented following a fall from a ladder. He has suffered an isolated injury to his right arm.

An elbow X-Ray is taken- refer to the props booklet- page 4.

a. List four (4) potential complications of this injury in the first 7 days. (4 marks)

1. _____

2. _____

3. _____

4. _____

He last ate 2 hours ago. He has received 20 mg morphine IV en route in the ambulance. His BP is 140 mmHg and HR 110 / min.

b. List your preferred analgesic/ sedative regime for the correction of this injury in the Emergency Department (include doses and routes). State three (3) points in your answer. (3 marks)

1. _____

2. _____

3. _____

Question 6 (continued)

c. Assuming adequate analgesic/ sedation, list five (5) steps in your measures to correct this abnormality. (5 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

Question 7 (10 marks)

A 47 year old man with a history of chronic liver disease and schizophrenia is brought to your emergency department with acute confusion.

His observations are:

BP	120/60	mmHg
HR	120	/min
RR	40	/min
GCS	12	E3, V4, M5

Selected biochemistry are taken- refer to the props booklet- page 5.

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

Derived value 1: _____

_____

_____

Derived value 2: _____

_____

_____

Question 7 (continued)

b. List four (4) likely explanations for these results. (4 marks)

1. _____

2. _____

3. _____

4. _____

You assess the patient as being moderately dehydrated.

c. List four (4) points in your approach to his fluid replacement regime. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 8 (12 marks)

A 26 year old man presents four hours following a recreational drug binge. A friend reports that he has been using large doses of "ICE".

a. List four examination findings that may be seen with ICE use. (4 marks)

1. _____

2. _____

3. _____

4. _____

The patient refuses to remain for assessment. You are required to chemically sedate the patient.

b. List your preferred drug regime in this situation, for the stated circumstances (include drug, dose and route): (3 marks)

i) Will accept oral treatment_____

_____

ii) Refuses oral medication, moderate degree of agitation_____

_____

iii) Refuses oral medication, going “nuts” _____

_____

Question 8 (continued)

The patient is sedated. Physical restraint is not required. Your complete assessment detects no organic pathology.

c. List five (5) key components to the ongoing care of this patient. (5 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

Question 9 (18 marks)

A 5 year old girl is referred by a GP with pallor and lethargy.
On examination she is extremely pale but appears alert and interactive.
Her observations:

BP	100/60	mmHg
HR	110	/min
RR	20	/min
O2 saturations	98%	on room air
Temperature	36.8	°C
GCS	15	

Selected blood tests are taken- refer to the props booklet- page 6.

a. List four (4) different pathological causes for these results. (4 marks)

1. _____

2. _____

3. _____

4. _____

Question 9 (continued)

b. List six (6) further investigations that you would perform in the emergency department for this girl. (6 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

6. _____

Question 9 (continued)

The mother refuses any blood products based on religious beliefs.

c. List five (5) situations under which you may override these wishes. (5 marks)

1. _____

2. _____

3. _____

4. _____

5. _____

None of these situations are met.

d. List three (3) alternative treatments that you could institute, other than the provision of blood products for this patient. (3 marks)

1. _____

2. _____

3. _____

ID NUMBER: ☐ ☐ ☐ ☐ ☐

University Hospital, Geelong

Emergency Medicine

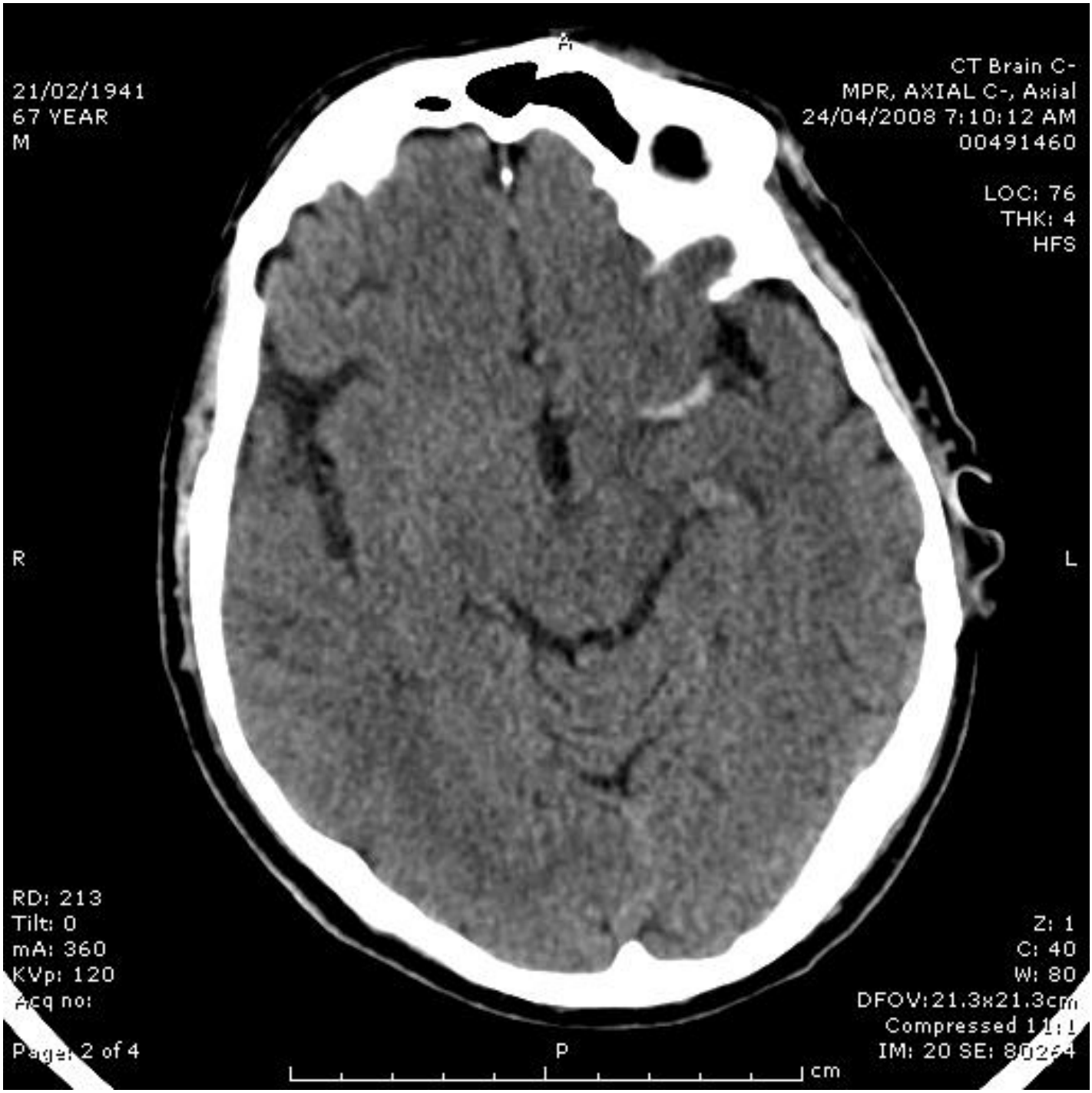
Trial Fellowship Exam

Short Answer Questions (SAQ)

Week 12

PROP BOOKLET

Question 1


A
21/02/1941
67 YEAR
M
CT Brain C-
MPR, AXIAL C-, Axial
24/04/2008 7:10:12 AM
00491460
LOC: 76
THK: 4
HFS
R
L
RD: 213
Tilt: 0
mA: 360
KVp: 120
Acq no:
Page: 2 of 4
P
cm
Z: 1
C: 40
W: 80
DFOV:21.3x21.3cm
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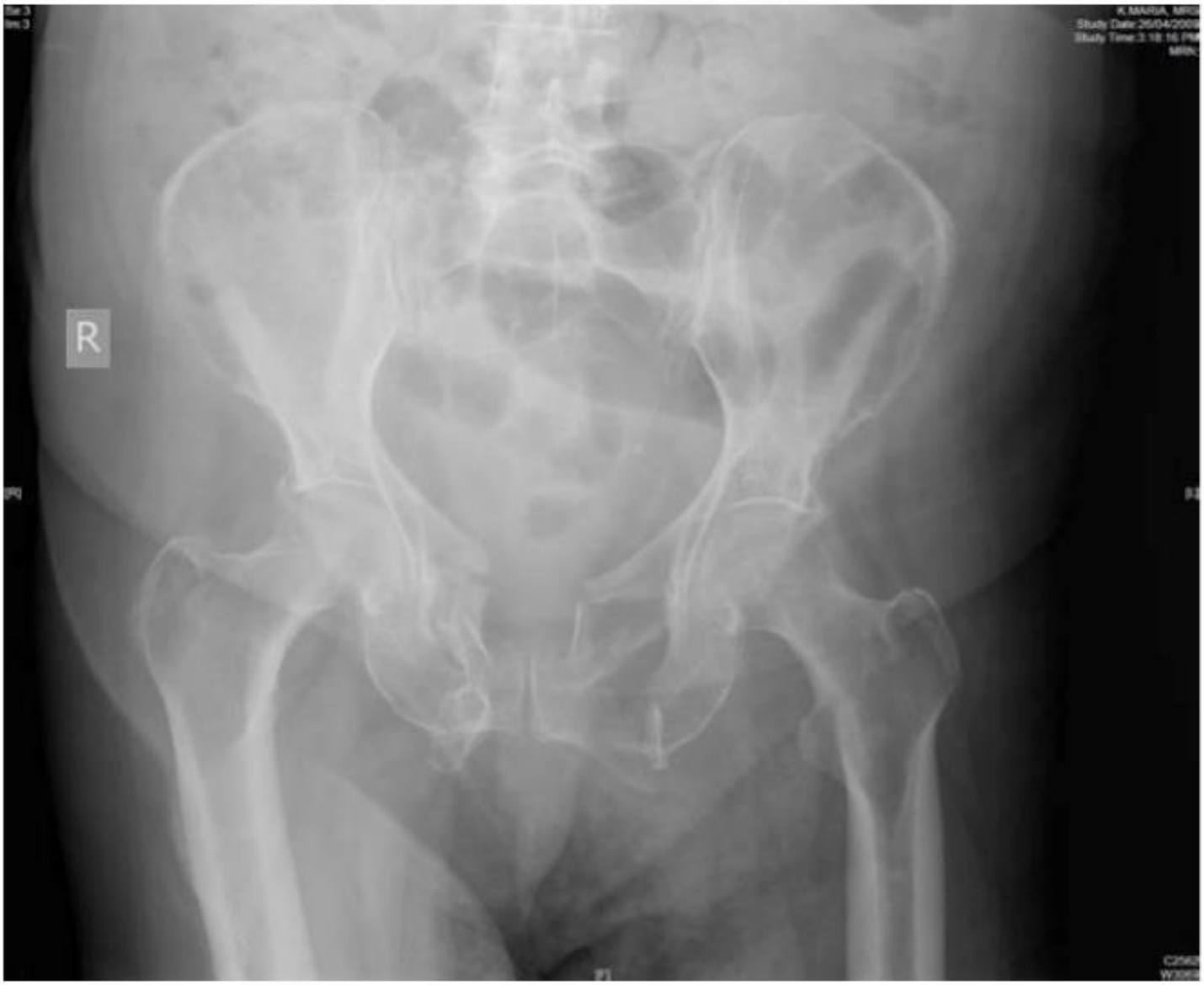
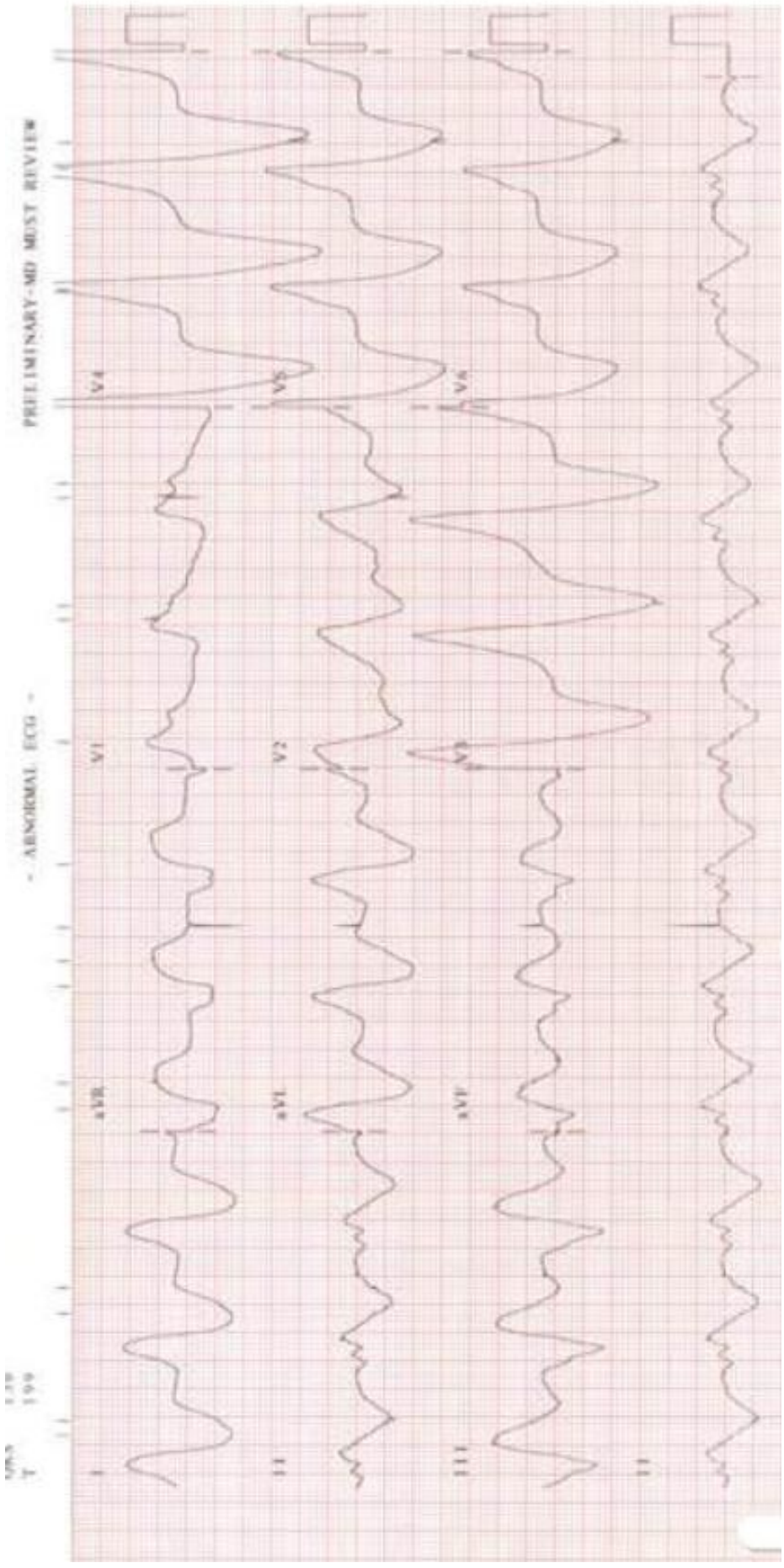
IM: 20 SE: 80264

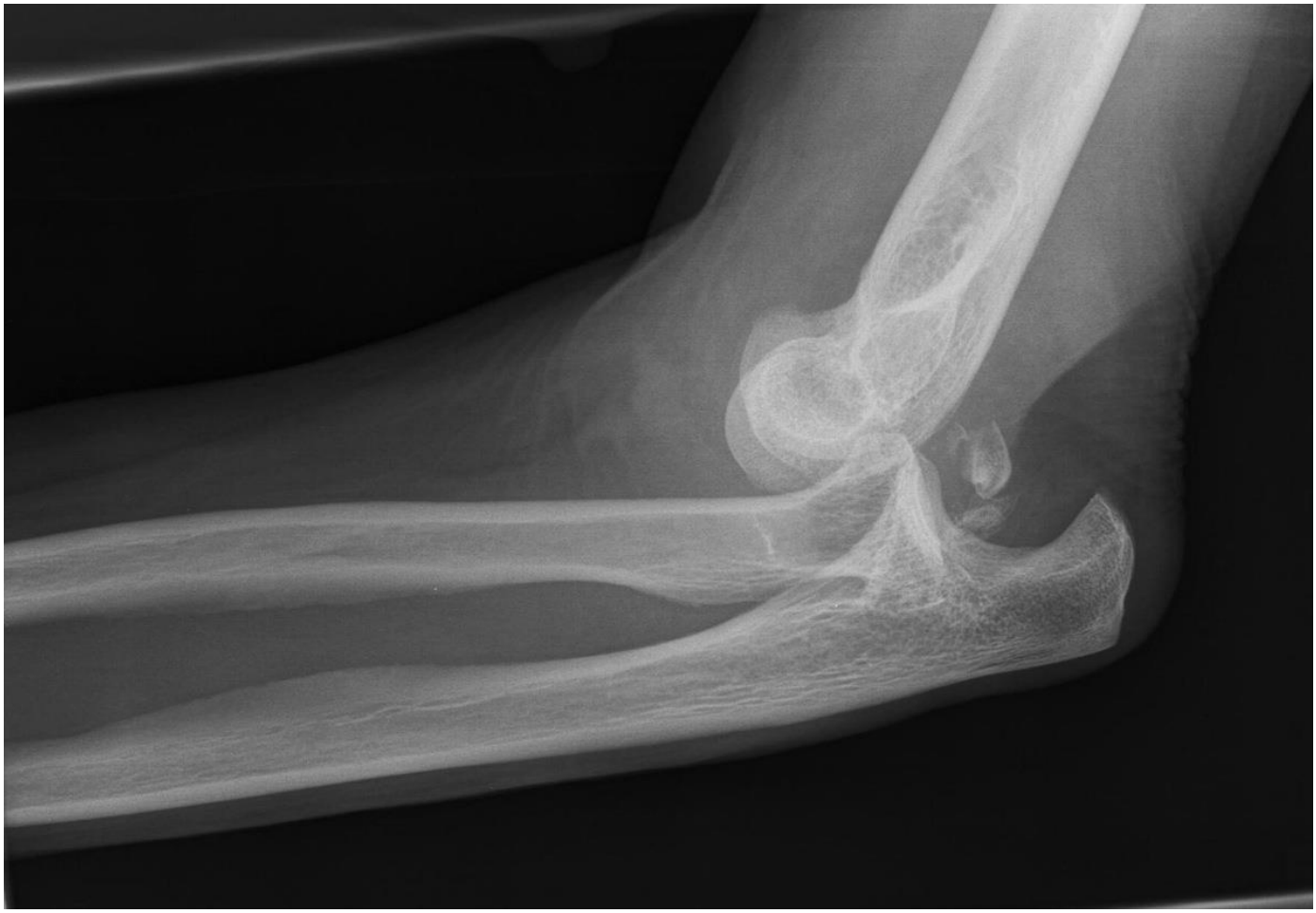
Question 2



Question 5


PRELIMINARY-MD MOST REVIEW
- ABNORMAL ECG -
V1
V2
V3
V4
V5
V6
aVR
aVL
aVF
I
II
III
II

Question 6



Question 7

			Reference Range
Arterial Blood Gas			
FiO_2	21	%	
pH	7.30		7.35-7.45
pO_2	91	mmHg	80-95
pCO_2	15	mmHg	35-45
$HCO3^-$	9	mmol/L	22-28
Lactate	14	mmol/L	< 2.0
Electrolytes			
Na^+	101	mmol/L	134-146
K^+	4.7	mmol/L	3.4-5
Cl^-	73	mmol/L	98-106
Glucose	10.5	mmol/L	3.5-5.5

Question 9

Her full blood count results are as follows

		Reference Range	Units
Hb	35	(101-131)	g/L
WCC	9.1	(6.0-11.0)	10^9/L
PLT	260	(150-450)	10^9/L
RBC	2.18	(3.9-5.3)	10^{12}/L
MCV	56.0	(75-85)	fL
MCH	16	(23-31)	pg
MCHC	286	(310-355)	g/L
Retic %	3.6	(0.2-2.0)	

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

UNIVERSITY HOSPITAL, GEELONG
FELLOWSHIP WRITTEN EXAMINATION
WEEK 12– 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)

a. What is the role of the ROSIER scale in the Emergency Department? State three (3) points in your answer. (3 marks)

NB: Recognition Of Stroke In Emergency Room – used in many AUS EDs

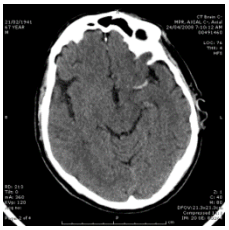
- **Clinical assessment tool for used (specifically for ED's) to risk stratify patients according to likelihood of stroke**
- **Only score validated for use in emergency departments following triage**
- **Comprises 5 pieces information pertaining to GCS, BP, BGL, unilateral weakness, speech or visual disturbance to rule out stroke**
- **Widely recommended for use in EDs (Eg. by NICE, NICS, NSF, SIGN)**

A patient presents after a sudden onset of right hemiparesis.

b. List five (5) inclusion criteria that must be met for the patient to be considered for thrombolysis. (5 marks)

- **Patient factors:**
  - **Age > 18 (upper age is currently undefined)**
  - **Time of onset < 4.5 hrs at time initiation treatment**
  - **Clinically definite stroke- with new persisting focal neurological deficit e.g. speech disturbance or neglect**
  - **Significant measurable deficit (NIHSS >4 detail not required)**
  - **CTB has excluded ICH**
- **Hospital factors:**
  - **Immediate access to imaging facilities and staff trained to interpret images**
  - **Authority has been given by a Neurologist/ Emergency Physician**
  - **Access to stroke management team who have expert knowledge in the delivery and monitoring of a patient who has received thrombolysis**
  - **Pathways/ protocols are available to guide post thrombolysis Mx, specifically BP control**

NB: - question asks for inclusion criteria, so "exclusion criteria" or the absence of exclusion criteria is not strictly acceptable
- consent is not required for TGA approval for administration



c. State the diagnosis, based on this CT scan and the clinical features provided.(1 mark)

- **Left middle cerebral artery infarction** (within MI division with hyperdense clot sign)

The patient undergoes thrombolysis and suffers a lethal intracerebral bleed. It is discovered that the patient had a recognised contraindication to thrombolysis.

d. List the 5 elements involved in the process of open disclosure. (5 marks)

- **an <u>apology</u> or expression of regret, which should include the words 'I am sorry' or 'we are sorry'**
- **a factual <u>explanation</u> of what happened**
- **an <u>opportunity</u> for the patient to relate their experience of the adverse event**
- **a <u>discussion</u> of the potential consequences of the adverse event**
- **an <u>explanation</u> of the steps being taken to manage the adverse event and prevent recurrence**

e. In general, list four (4) factors that would support the role for decompressive craniectomy. (4 marks)

NB: Decompressive craniectomy is used in pt with large strokes and cerebral oedema assoc with a deteriorating neurological state

- **Age < 60**
- **Decreased conscious state**
- **Signs of ↑ ICP**
- **Absence of thrombolysis/stroke unit facilities**
- **Malignant MCA infarct involving > 50% territory on imaging**
- **Surgery within 48 hrs of stroke onset**

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EMA Emergency Medicine Australasia

Emergency Medicine Australasia (2016) 28, 496–510 doi: 10.1111/1742-6723.12653

REVIEW ARTICLE

Review article: Why is there still a debate regarding the safety and efficacy of intravenous thrombolysis in the management of presumed acute ischaemic stroke? A systematic review and meta-analysis

Lachlan DONALDSON,[1] Emily FITZGERALD,[1,2] Oliver FLOWER[1,3] and Anthony DELANEY[1,3,4]

[1]Malcolm Fisher Department of Intensive Care Medicine, Royal North Shore Hospital, Sydney, New South Wales, Australia, [2]Faculty of Health, University of Technology Sydney, Sydney, New South Wales, Australia, [3]Northern Clinical School, Sydney Medical School, The University of Sydney, Sydney, New South Wales, Australia, and [4]ANZIC Research Centre, Department of Epidemiology and Preventative Medicine, Monash University, Melbourne, Victoria, Australia

Abstract

Objective: The objective of the present study is to independently and systematically assess the harms and benefits of intravenous thrombolysis for patients with presumed acute ischaemic stroke.

Methods: We performed a systematic review and meta-analysis of randomised clinical trials of intravenous thrombolysis compared with control in patients with presumed acute ischaemic stroke. The effectiveness of thrombolysis on functional outcome, symptomatic intracranial haemorrhage, early mortality and mortality at final follow up was assessed using a fixed-effect meta-analysis.

Results: A total of 26 studies that randomised 10 431 participants were included. The use of thrombolysis was associated with an increased odds of good functional outcome, estimated odds ratio (OR) 1.14 (95% confidence interval [CI] 1.04–1.25, $P = 0.004$), and also a significantly increased risk of symptomatic intracranial haemorrhage, estimated OR 4.28 (95% CI 3.34–5.48, $P < 0.0005$) and an increased risk of early mortality, estimated OR 1.51 (95% CI 1.27–1.78, $P < 0.0005$). There was no statistically significant evidence that the effect of recombinant tissue plasminogen activator (rt-PA) was different from that of other thrombolytic agents. There was also an increase in mortality at final follow up associated with treatment with thrombolysis, estimated OR 1.17 (95% CI 1.06–1.30, $P = 0.003$), although this result was not consistent when limited to studies of rt-PA, estimated OR 1.04 (95% CI 0.92–1.18, $P = 0.49$).

Conclusions: There is clear evidence of increased early mortality, increased rates of symptomatic intracranial haemorrhage and also of improved functional outcomes for patients with presumed acute ischaemic stroke treated with thrombolysis. The available data are unlikely to resolve the controversy regarding the use of intravenous thrombolysis in this population, and further randomised controlled trials are urgently required.

Key words: *stroke, thrombolytic therapy, tissue plasminogen activator.*

Key findings

- There is clear evidence of harm (increased incidence of symptomatic intracranial haemorrhage and early mortality) related to the use of thrombolysis for patients with presumed acute ischaemic stroke, but also some evidence that thrombolysis may be associated with improved functional outcomes.
- The debate regarding the utility of thrombolysis for the treatment of presumed acute ischaemic stroke cannot be resolved given the current evidence and further high-quality randomised trials are required.

Introduction

Thrombolysis for the treatment of presumed acute ischaemic stroke was first countenanced as a therapeutic option in the 1960s.[1] Early studies using a variety of agents did not produce promising results,[2–4] in contrast to the success of thrombolytic agents used to treat acute myocardial infarction.[5,6] However, the results of the NINDS study[7] led to the licensing of recombinant tissue plasminogen activator (rt-PA) for the treatment of acute ischaemic stroke, in spite of some controversy.[8–10] This controversy persists[11–13] and is considered to be an important factor[14] in the persistently low rates of utilisation of this therapy.[15]

Currently, rt-PA for the treatment of presumed acute ischaemic stroke is

Correspondence: Dr Lachlan Donaldson, Malcolm Fisher Department of Intensive Care Medicine, Royal North Shore Hospital, St Leonards, NSW 2065, Australia. Email: lachlandonaldson@gmail.com

Lachlan Donaldson, MBBS, Advanced Trainee; Emily Fitzgerald, BN, Clinical Nurse Specialist, Academic; Oliver Flower, BMedSci, MBBS, FCICM, Staff Specialist, Lecturer; Anthony Delaney, MBBS, MSc, PhD, FACEM, FJFICM, Senior Staff Specialist, Senior Lecturer, Adjunct Senior Research Fellow.

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EDITORIAL

EMA

How is more negative evidence being used to support claims of benefit: The curious case of the third international stroke trial (IST-3)

Jerome R Hoffman and Richelle J Cooper
UCLA School of Medicine, Los Angeles, California, USA

> Still a man hears what he wants to hear, and disregards the rest. 'The Boxer' – Paul Simon and Art Garfunkel, 1969.

Just before the release of the results of the third international stroke trial (IST-3),[1] the largest trial of thrombolysis in acute ischaemic stroke (AIS), the journal *Stroke* published a remarkable pre-emptive strike – a commentary in which the author identifies a legion of concerns regarding the study's methodology, only to reassure us about the study's value.[2] In one astonishing section he lucidly catalogues a host of important biases likely to skew the study's results in ways that increase the chance of finding a spurious benefit from the use of tissue plasminogen activator (tPA) – but then proceeds to trivialise the very concerns he has elucidated. He starts by noting that (among many other problems) the study determined ultimate patient outcome using a score that is highly unreliable even when calculated by a neurologist performing an in-person neurologic evaluation. He then acknowledges that this problem was enormously exacerbated in IST-3 because the scoring was done by a layperson; to make matters even worse, this was a layperson who, like the patient himself, was unblinded to treatment group. After all this, he nevertheless concludes with the soothing statement that 'reassuringly, all images in IST-3 have been read by a blinded central observer, so outcomes based on imaging will have meaning free from any recall bias.' This seems to be another way of saying 'sure, there are many ways in which our measurements are very distorted … but don't worry about that, because there is one other way in which we got it right.'

The author of this commentary – and by extension the editors of *Stroke* who approved it – ultimately concludes that despite its many flaws, there is much to learn from IST-3.[2] We agree … although given that the actual results of IST-3 uniformly failed to show benefit, even in the face of severe bias, we believe the lessons are precisely the opposite of those being trumpeted by the study's own authors.[1]

The third international stroke trial is the latest addition to the long-running controversy over the use of thrombolysis in AIS, but the claims being made about 'benefit' in this trial seem to go beyond the common situation, where well meaning people can look at the same information and come to wildly disparate conclusions.[1] In this commentary we focus on IST-3, while also briefly revisiting the other two randomised controlled trials (RCTs)[3,4] that are commonly cited as providing support for the use of tPA in AIS. We will not review the many RCTs that found either no benefit, or clear harm, even though we believe the limited attention given to these trials, simply because they are negative, greatly distorts the discussion of thrombolysis in AIS.[5–7] When the European Cooperative Acute Stroke Study III (ECASS III) was published in one of the world's most prominent journals, its claims of efficacy received enormous publicity;[4] meanwhile, the other contemporaneous RCT of thrombolytic treatment of AIS, which reported negative findings, appeared in a less prominent specialty journal and received essentially no notice after its publication – it was negative, so it is somehow considered irrelevant.[8] Nor will we address the many non-randomised 'effectiveness' studies other than to note that they too are mostly very negative, and that the few that purport to show utility suffer from the very same types of biases and misinterpretations that plague the 'positive' RCTs we will be addressing.

However, first we would like to touch on the role of chance when multiple studies evaluate a treatment that

Jerome R Hoffman, MA, MD, Professor Emeritus of Medicine/Emergency Medicine; Richelle J Cooper, MD, MSHS, Associate Professor of Medicine/Emergency Medicine.

RESEARCH

Thrombolysis for acute stroke in Australia: outcomes from the Safe Implementation of Thrombolysis in Stroke registry (2002–2008)

Marion A Simpson, Helen M Dewey, Leonid Churilov, Niaz Ahmed, Christopher F Bladin, David Schultz, Romesh Markus, Jonathan W Sturm, Christopher R Levi, David J Blacker, Jim Jannes, Richard I Lindley and Mark W Parsons

ABSTRACT

Objective: To report Australian outcomes from the Safe Implementation of Thrombolysis in Stroke International Stroke Thrombolysis Register (SITS-ISTR).

Design: Observational study using data collected prospectively from December 2002 to December 2008.

Setting: Centres administering thrombolysis for acute stroke in Australia and worldwide.

Patients: All patients treated with recombinant tissue plasminogen activator for acute stroke in participating centres, regardless of stroke severity, time of treatment and other clinical factors.

Intervention: Thrombolysis for acute stroke, administered according to local protocol.

Main outcome measures: Functional outcome as 3-month modified Rankin score (mRS), and frequency of symptomatic intracerebral haemorrhage (ICH).

Results: During the study period, a total of 32 countries participated, and confirmed baseline data were available for 581 Australian patients and 20 953 patients in the rest of the world. Australian patients were older (median age, 73 v 69 years; $P < 0.001$), were less independent before stroke (premorbid mRS of 0–1, 87.5% v 91.2%; $P < 0.005$), and had more comorbidities and more severe strokes. Comparing the Australian cohort with the rest of the world, the odds ratio of 3-month mRS of 0–2 was 0.98 (95% CI, 0.88–1.08; $P = 0.63$), the odds ratio of symptomatic ICH was 0.98 (95% CI, 0.83–1.16; $P = 0.85$ [by the definition used by the National Institute of Neurological Disorders]) and the odds ratio of death was 1.04 (95% CI, 0.91–1.19; $P = 0.54$). Good outcome in the Australian cohort was predicted by younger age, presence of hyperlipidaemia, lower premorbid mRS, absence of infarct on early brain imaging, less severe stroke, and lower baseline blood glucose level.

Conclusion: Clinical outcomes after thrombolysis in Australia were similar to those worldwide.

MJA 2010; 193: 439–443

For editorial comment, see page 436

Thrombolysis for acute stroke with recombinant tissue plasminogen activator (rt-PA) is increasingly being used in metropolitan and regional hospitals.[1-3] In randomised controlled trials, rt-PA given within 3 hours of onset of stroke symptoms has been shown to improve outcome with reduced disability at 3 months, albeit with a small risk of serious harm, usually related to intracranial haemorrhage.[4-6]

In response to concerns that rt-PA given in normal clinical practice might not have the same risk–benefit ratio as that given in trials, a consensus statement from the Karolinska Stroke Update meeting in 2000 recommended continuous audit of clinical rt-PA use.[7] The result was Safe Implementation of Thrombolysis in Stroke (SITS), a collaborative of over 700 centres in 35 countries committed to the audit of thrombolysis for stroke with the aim of standardising services, maintaining safety and improving outcomes. The SITS International Stroke Thrombolysis Register (ISTR)[8,9] has two main aims: to assess whether the favourable results from the original clinical trials are maintained in clinical practice; and to allow individual centres to monitor their own treatment time lines and outcomes, and compare these to national and international data.

For the first time, we report the Australian experience of thrombolysis for stroke from December 2002 to December 2008, as recorded in the SITS-ISTR, and compare these Australian data to worldwide data for the same period. We also used the Australian data to explore the demographic and clinical characteristics that may predict outcome after thrombolysis.

METHODS

In Australia, participation in the SITS-ISTR is not compulsory but is strongly encouraged for all centres administering rt-PA for stroke. Participating centres answer a series of questions on a password-protected, interactive website. Participants commit to registering all treated patients and consent to audit of the data. The inclusion criteria encompass all patients treated with rt-PA for ischaemic stroke, regardless of age, time of treatment or other clinical factors. De-identified data are collected on:

- patient demographics and medical history — age, sex, premorbid function as modified Rankin score (mRS) (Box 1), cardiovascular risk factors and premorbid drug therapy
- treating centre location and previous experience with rt-PA
- details of the acute stroke — time of stroke, arrival to hospital, brain imaging and treatment; classification of stroke according to the International Classification of Diseases (10th revision); and baseline National Institutes of Health Stroke Scale (NIHSS) score[11]
- results of brain imaging — presence of infarct or haemorrhage on any scan, and findings from additional cerebrovascular imaging
- primary outcomes — presence of symptomatic intracerebral haemorrhage (ICH) according to each of three recognised definitions (Box 2), mortality, and functional status at 3 months after stroke as mRS assessed by face-to-face or telephone interview (a "good functional outcome" was defined as an mRS of 0–2 at 3 months).

Details of any adverse events relating to thrombolysis are also provided. Centres receive a computer-generated report of their own data and those of the global dataset.

In addition, data on country of origin are entered into the SITS central database, which was searched for "Australia" or "other". For this study, data for Australia were excluded from the worldwide dataset.

Statistical analysis

Statistical analyses were performed using Stata version 10.0 IC (StataCorp, College Station, Tex, USA). For comparison with

Editorials

The spectacular recent trials of urgent neurointervention for acute stroke: fuel for a revolution

How should we redesign our stroke services in light of neurointerventional advances?

Stroke medicine has come a long way from the nihilism of two decades ago, with numerous interventions now supported by high-level evidence

In 2013, neutral results from three trials of neurointervention for treating ischaemic stroke were simultaneously published — a triad of gloom.[1-3] In just over 2 years since, five positive trials have been reported.[4-8] What explains this extraordinary turnaround, and what are the implications for stroke services in Australia and around the world? The answers to these questions are surprising and reflect a mixture of science, technology and policy.

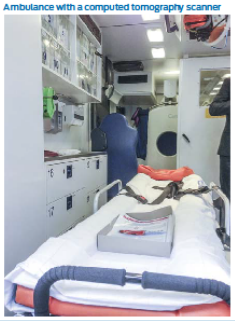
The roles of science, technology and policy

The science involved is the culmination of a decade of work on proving that brain imaging can identify the ischaemic penumbra — the area of the brain that has shut down and is on the path to infarction but, with successful reperfusion, is potentially salvageable. By recruiting patients with a favourable profile for reperfusion therapy (so-called target mismatch, where the ratio of perfusion lesion to established infarct is > 1.8, the perfusion lesion volume is > 15 mL, and the established infarct volume is < 70 mL),[9] we are now able to identify those who are likely to respond well. In addition, computed tomography (CT) angiography is now widely available and can demonstrate major cerebral vessel occlusion — a clear target for therapy.[9]

In contrast to the neutral trials, the recent trials all used either advanced imaging to identify patients with the "reperfusion responder" profile or angiography to prove major vessel occlusion, or both, then randomly assigned this population of likely responders to receive endovascular reperfusion (usually in addition to alteplase thrombolysis) or standard acute stroke care. The combination therapy resulted in potent reperfusion and a dramatic treatment effect (Appendix), such that three of the five neurointervention trials were stopped early.

The technology is all about the device. In today's fast-moving world, it is almost impossible to design, fund and complete a trial of a device without it becoming obsolete by the time the trial has finished — the fate of the previous studies.[1-3] Unlike in coronary intervention, the thrombus or embolus in ischaemic stroke must be physically extracted, and the new generation of retrievable stents are a major advance in this regard. One of the recent trials, MR CLEAN, demonstrated that carotid stenting (for extracranial occlusion) was also required for 13% of the patients receiving intra-arterial treatment.[4]

Ambulance with a computed tomography scanner



Finally, the unanticipated influence of policy can have profound effects. One of the neutral trials, IMS III, was conducted in the United States at a time when neurointervention was generously compensated.[3] This, together with the attractiveness of the technology (despite its lack of evidence), meant that most people were treated outside the trial. The difficulties in recruitment (only one or two patients per centre per year) and a possible selection bias of recruiting only "difficult" patients might have had an effect on the results of IMS III.

In contrast, the Dutch MR CLEAN trial provides an important lesson.[4] All the neurointervention centres in the Netherlands participated in this trial, and from 2013 there was no reimbursement for people treated outside the trial. This allowed some 500 patients to be recruited from 16 sites in just over 3 years, compared with IMS III, which needed 7 years to recruit 656 patients from 58 sites. If trial-only reimbursement for unproven devices were enforced, it is likely that reliable data on efficacy would have been available much earlier, potentially saving hundreds, if not thousands, of lives.

Richard I Lindley MD, FRCP(Edin), FRACP[1]
Christopher R Levi BMedSci, MRIH, FRACP[2]
1 Westmead Clinical School, University of Sydney, Sydney, NSW.
2 The George Institute for Global Health, Sydney, NSW.
3 John Hunter Hospital, Newcastle, NSW.
4 University of Newcastle, Newcastle, NSW.
richard.lindley@sydney.edu.au

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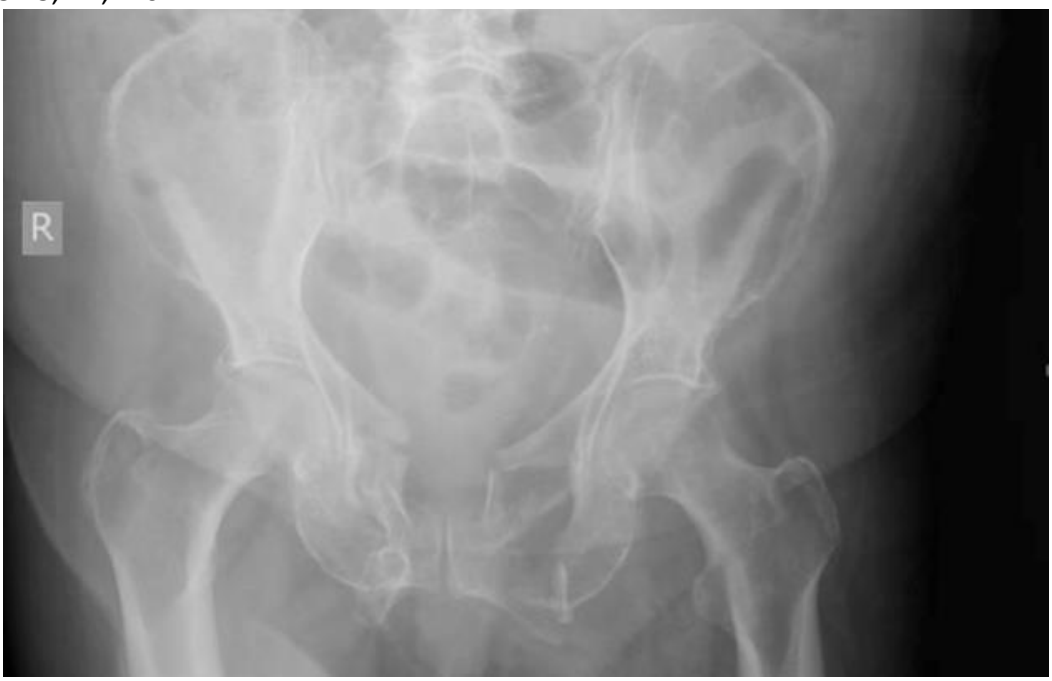
Question 2 (12 marks)

A 75 year old woman presents following a fall from standing. She is complaining of bilateral hip pain only. Her observations are: BP 75/ 50 mmHg HR 125/ min RR 20/ min Oxygen saturations 98% room air
GCS 13 E3, V4, M6



a. State four (4) abnormal findings shown in this xray. (4 marks)
   - **# R superior pubic ramus**
   - **# L Superior pubic ramus**
   - **# L inferior pubic ramus**
   - **R partially displaced sub capital femur #**
   - **# through ischium extending to R acetabulum**
   - **Bilateral hip OA- decreased jt space, peri articular sclerosis, peri articular osteophytes**

   NB: ok if either L or R OA only mentioned
   no # NOF is evident

b. List four (4) further imaging studies that you would consider for this patient. (4 marks)
   - **eFAST scan**
   - **CT angiography pelvis +/- embolisation**
   - **CT abdo/ pelvis (with contrast)**
   - **Cystogram/ retrograde cystogram/ retrograde urethrogram (all refer to the same study)**
   - **CT Brain +/- C spine**
   - **CXR (is a bit soft, but acceptable)**

c. Assuming that you have IV access, list four (4) steps in your approach to managing her pain control. (4 marks)
   - **Immobilisation of legs**
   - **Pelvic binder**
   - **Use non Haemodynamically significant drugs until BP improves:**

- **IV fentanyl 25 mcg boluses**
- **IV Ketamine 5mg bolus**

ie. Not just unqualified morphine IV

- **Consider PCA if patient able to use/ no CHI**
- **R femoral nerve block bupivocaine 0.5% 2mg/kg**

Question 3 (12 marks)

A 25 year old presents following a stab wound to the neck.

a. List four (4) key historical features that are important in this case. (4 marks)
1 mark for any of these:
- **Type of weapon- knife vs other**
- **Features of weapon- width, length, blade edge**
- **Estimated scene blood loss**
- **Symptoms of Airway compromise (eg stridor)**
- **Symptoms of Breathing compromise (eg SOB)**
- **Symptoms of Circulation/ neurological (eg focal neurological deficits)**
- **Other sites of blows- stab or otherwise**

½ mark/ no mark for:
- **PMHx**
- **medication eg anticoagulants (unlikely in a 25 yr old)**
- **Allergies**
- **Last ate**

NB: discourage "AMPLE" as a standard answer- encourage "important in this case"

b. List four (4) examination features that are important in this case. (4 marks)
- **Vital signs- normality vs haemodynamic instability**
- **Airway threat- stridor/ expanding haematoma**
- **Breathing threat- PTX/ tension PTX, marked respiratory distress, subcutaneous emphysema, air bubbling through wound**
- **Circulation- active bleeding/ haematoma**
- **Site of wound- zone of the neck**

c. As they pertain to the neck, list the boundaries of the following: (3 marks)
- **ZONE 1: clavicles -> inferior border carotid cartilage**
- **ZONE 2: inferior border cricoid cartilage -> angle mandible**
- **ZONE 3: angle mandible -> base of skull**

d. What feature of the wound would allow definitive repair in the emergency department. (1 mark)
- **Platysma mm not breeched**

Dr Tom Reade (Staff Specialist, University Hospital, Geelong Emergency Department)
Email: **tomre@barwonhealth.org.au**

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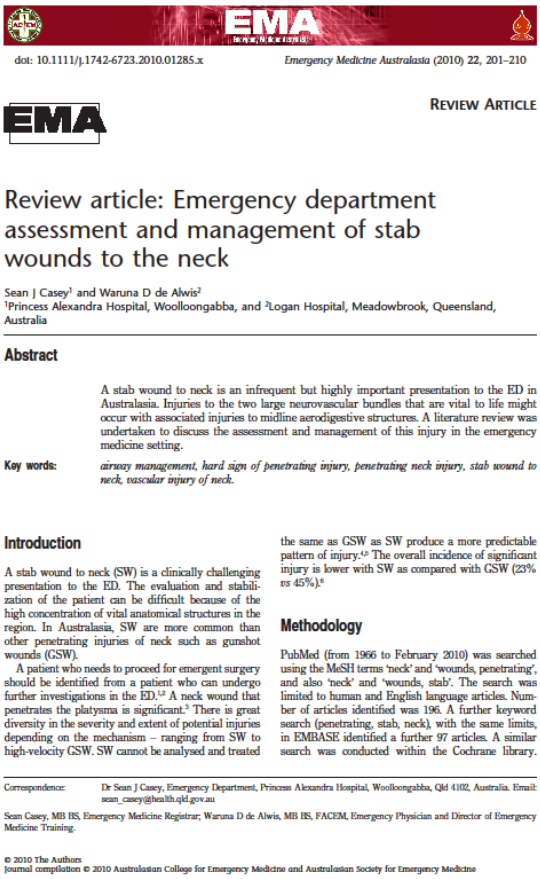

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REVIEW ARTICLE

Review article: Emergency department assessment and management of stab wounds to the neck

Sean J Casey[1] and Waruna D de Alwis[2]
[1]Princess Alexandra Hospital, Woolloongabba, and [2]Logan Hospital, Meadowbrook, Queensland, Australia

Abstract

A stab wound to neck is an infrequent but highly important presentation to the ED in Australasia. Injuries to the two large neurovascular bundles that are vital to life might occur with associated injuries to midline aerodigestive structures. A literature review was undertaken to discuss the assessment and management of this injury in the emergency medicine setting.

Key words: *airway management, hard sign of penetrating injury, penetrating neck injury, stab wound to neck, vascular injury of neck.*

Introduction

A stab wound to neck (SW) is a clinically challenging presentation to the ED. The evaluation and stabilization of the patient can be difficult because of the high concentration of vital anatomical structures in the region. In Australasia, SW are more common than other penetrating injuries of neck such as gunshot wounds (GSW).

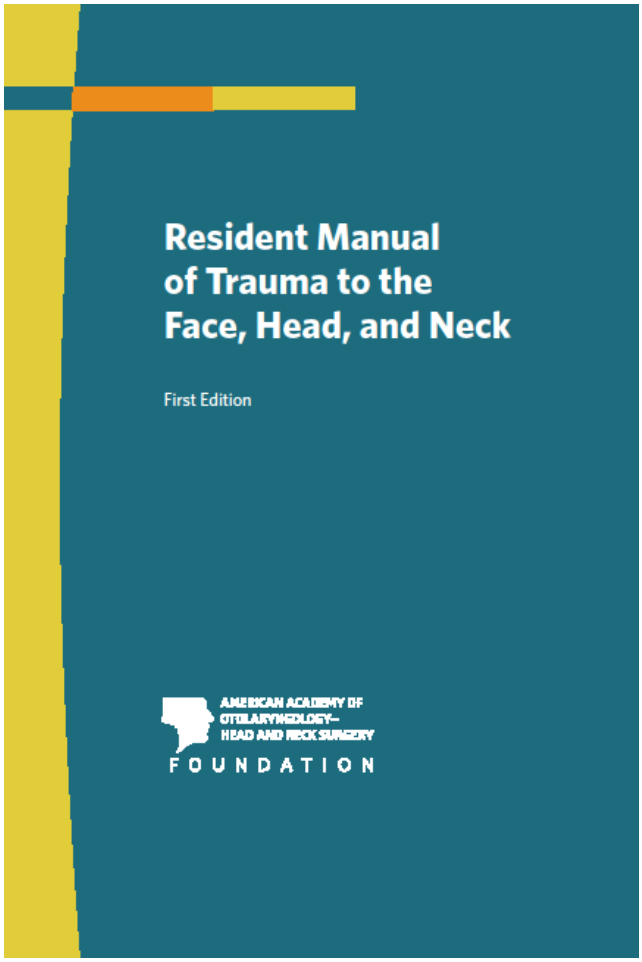
A patient who needs to proceed for emergent surgery should be identified from a patient who can undergo further investigations in the ED.[1,2] A neck wound that penetrates the platysma is significant.[3] There is great diversity in the severity and extent of potential injuries depending on the mechanism – ranging from SW to high-velocity GSW. SW cannot be analysed and treated the same as GSW as SW produce a more predictable pattern of injury.[4,5] The overall incidence of significant injury is lower with SW as compared with GSW (23% *vs* 45%).[6]

Methodology

PubMed (from 1966 to February 2010) was searched using the MeSH terms 'neck' and 'wounds, penetrating', and also 'neck' and 'wounds, stab'. The search was limited to human and English language articles. Number of articles identified was 196. A further keyword search (penetrating, stab, neck), with the same limits, in EMBASE identified a further 97 articles. A similar search was conducted within the Cochrane library.

Correspondence: Dr Sean J Casey, Emergency Department, Princess Alexandra Hospital, Woolloongabba, Qld 4102, Australia. Email: sean_casey@health.qld.gov.au

Sean Casey, MB BS, Emergency Medicine Registrar; Waruna D de Alwis, MB BS, FACEM, Emergency Physician and Director of Emergency Medicine Training.



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Emerg Med Clin N Am 25 (2007) 679–694

Evaluation and Management of Neck Trauma

Niels K. Rathlev, MD*, Ron Medzon, MD,
Mark E. Bracken, MD

Department of Emergency Medicine, Boston Medical Center, Boston University School of Medicine, One Boston Medical Center Place, Boston, MA 02118, USA

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

Aerodigestive injuries

Epidemiology

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

* Corresponding author.
E-mail address: nrathlev@bu.edu (N.K. Rathlev).

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Question 4 (12 marks)

A 3 month old infant presents with shortness of breath and difficulty breathing.
Her observations are: BP 85/ 50 mmHg HR 12/ min RR 80 / min Oxygen saturations 98% room air Temperature (rectal) 36.5 °C

a. List four (4) differential diagnoses for this patient (each to be from a different pathological category). For each, list the method of confirmation of diagnosis. (8 marks)

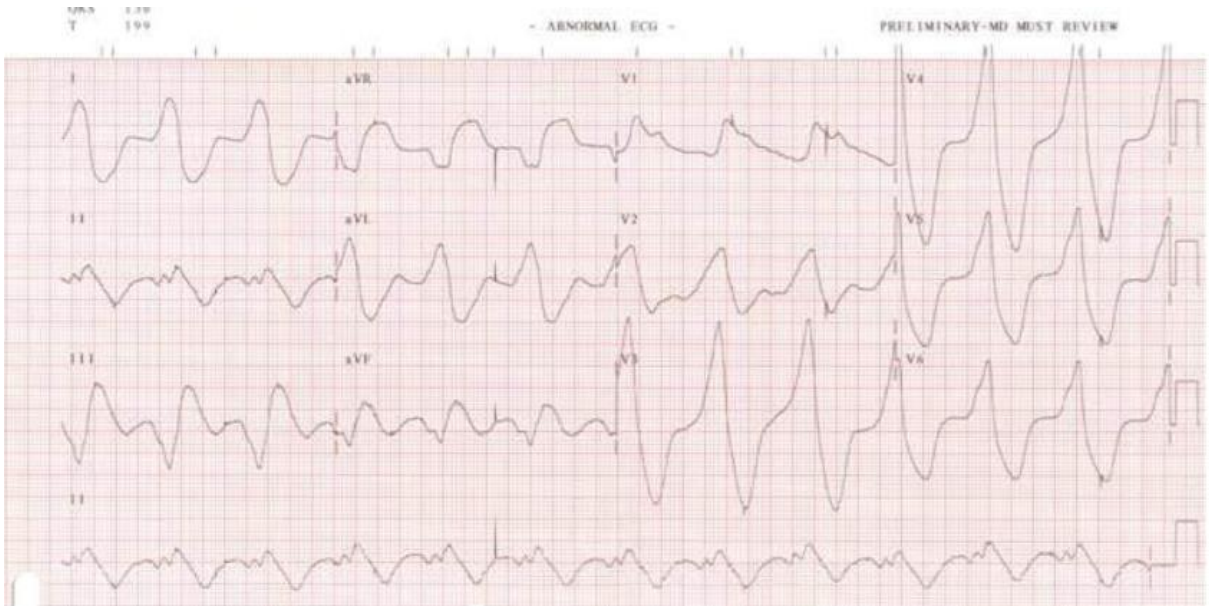
	Differential diagnosis (4 marks)	Method of confirmation of diagnosis (4 marks)
1	**Infection** **eg. Viral (croup, bronch)** **bacterial (pneumonia)**	**Septic work up: CXR, BC, NPA for Resp viruses**
2	**Congenital cardiac disease**	**Colour change during feed** **Hyperopia test** **ECG** **ECHO**
3	**Trauma eg lung contusion, # ribs, PTX (consider NAI)**	**Primary & secondary survey in eFAST** **Fundoscopy, skeletal survey, CXR**
4	**Inborn errors of metabolism eg DKA**	**BGL** **VBG** **LFT** **Urine for reducing substances**
	Congenital anatomical anomaly eg. Tracheomalacia, subglottic stenosis, diaphragmatic hernia	**Endoscopy** **(Hx of noisy breathing/ Increased WOB with feeding since birth)**
	Inhaled FB	**CXR** **Neck Soft tissue** **Indirect/ direct laryngoscopy**

b. List four (4) historical factors that would suggest a serious illness. (4 marks)

- **Rapid onset and progression**
- **Apnoeic episodes**
- **Cyanotic episodes**
- **Extreme lethargy**
- **Very poor intake**
- **Significantly decreased wet nappies**
- **Frequent vomiting- esp bilious**
- **Failure to thrive/ poor weight gain**
- **Prematurity- need for NICU**
- **Known congenital abnormality**
- **Hx of significant perinatal maternal disease**
- **Hx of significant neonatal disease**
- **Lack of immunisation**

Question 5 (12 marks)

An 87 year man presents from a nursing home with acute deterioration.



a. What is the most likely diagnosis? (1 mark)
   - **Life threatening hyperkalaemia**
   - **(Failure to pace/ capture -PPM spikes present in aVR/ aVL/ aVF/ V1/V2)- not required to get 1 mark**

b. List three (3) features of this ECG that support this diagnosis. (3 marks)
   - **Broad QRS > 400 msec**
   - **ST segment blurring**
   - **Absent p waves**
   - **"sine wave appearance"**
   - **Failure to pace/ failure to capture**

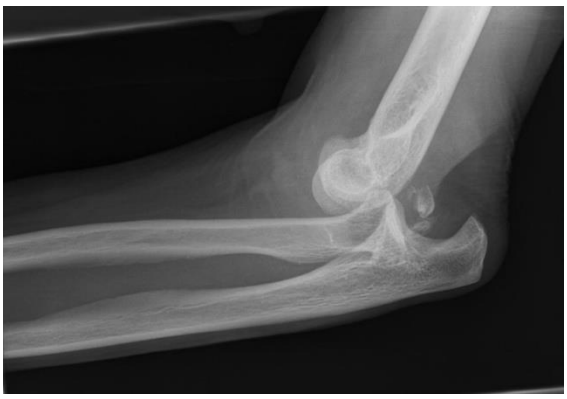
c. List four (4) likely causes for this diagnosis.(4 marks)
   - **Acute renal failure**
   - **Digoxin toxicity**
   - **K+ sparing diuretic +/- ACEI**
   - **K+ accidental or deliberate OD**
   - **Rhabdomyolysis**
   - **Much less likely: Transfusion/ Haemolysis/ Hyperthermia/ Addison's disease**

d. List four (4) treatments that you would consider for this patient. (4 marks)
   - **Calcium gluconate- 10ml 10% over 10 min - REQUIRED (Unless dig toxic- not required)**
   - **NaHCO3- 1 meq/kg over ½ hr**
   - **Dextrose 50% 50 ml + 5-10 U actrapid subcut**
   - **Salbutamol 5mg neb**
   - **Resonium**
   - **Dialysis if CRF**

- **Digibind if digoxin toxic**

Question 6 (12 marks)

A 45 year old man presented following a fall from a ladder. He has suffered an isolated injury to his right arm.



a. List four (4) potential complications of this injury in the first 7 days. (4 marks).
   - **Brachial a dissection/ occlusion**
   - **Median n neuropraxia**
   - **Compartment syndrome**
   - **Septic joint if open**
   - **Unstable elbow**
   - **Adverse drug reaction to Rx- eg morphine allergy**
   - **(Pain) NB: Pain is a complication, but the others are better answers**

b. He last ate 2 hours ago. He has received 20 mg morphine IV en route in the ambulance. His BP is 140 mmHg and HR 110 / min. List your preferred analgesic/ sedative regime for correction of this injury in the Emergency Department. (include doses and routes). State three (3) points in your answer. (3 marks)

   NB: Multiple options acceptable:
   - NB doses and routes requested
   - Propofol/ other GA is fatal error as unfasted. Addition of small doses of propofol may convert to GA given morphine on board
   - addition of fentanyl does not make sense given morphine on board
   - **Needs urgent reduction to reduce likelihood of complications/ pain**
   - **Ketamine 0.5- 1mg/ kg IV**
   - **Midazolam IV titrated to sedation eg 2 mg aliquots**
   - **Interscalene block La block- lignocaine/ bupivacaine**
   - **LAMP- Biers technique**

c. Assuming adequate analgesic/ sedation, list five (5) steps in your reduction technique. (5 marks)
   NB: Numerous techniques described. Not sufficient to just list the name of a technique.
   - **Counter traction of arm by assistant**
   - **Disengage with downwards pressure on proximal forearm**
   - **Longitudinal traction**
   - **+/- firm pressure over the olecranon**
   - **Splint at 90° flexion and elevate**

- **If unsuccessful due to intra- articular fragments → OT**

Question 7 (10 marks)

A 47 year old man with a history of chronic liver disease and schizophrenia is brought to your emergency department with acute confusion.
His observations are: GCS 12E3,V4,M5 HR 120/min BP 120/60mmHg RR 40/min

List two (2) derived values from these investigations. (2 marks)

Arterial Blood Gas			Reference Range
FiO_2	21	%	
pH	7.30		7.35-7.45
pO_2	91	mmHg	80-95
pCO_2	15	mmHg	35-45
$HCO3^-$	9	mmol/L	22-28
Lactate	14	mmol/L	< 2.0
Electrolytes			
Na^+	101	mmol/L	134-146
K^+	4.7	mmol/L	3.4-5
Cl^-	73	mmol/L	98-106
Glucose	10.5	mmol/L	3.5-5.5

a. Derived value 1
- **Anion gap 101+ 4.7 - 73 -9= 24 or K omitted 101-73-9 = 19**
- **Thus high/ raised**

b. Derived value 2

Delta ratio = ∆ Anion gap/∆ [HCO3-] or **↑anion gap/ ↓ [HCO3-**

= Measured anion gap – Normal anion gap
Normal [HCO3-] – Measured [HCO3-]

= (AG – 12)
(24 - [HCO3-])

- **Delta ratio (Print summary at end of this document for those who need)**
- **4 -12/24- 9 = 0.8 or 19-12/24-9= ~ 0.5**
- **Consider coexistent HAGMA and NAGMA**

Or

PAO2 - PaO2
PAO2= PiO2 – (PaCO2/R)
PiO2= (atmospheric pressure - partial pressure of water) x FiO2
760 - 47 at sea level x ~ (0.21= RA)
PAO2= (FIO2) (P_{atm} – 47 mm Hg) - (P_aCO2)/ 0.8
Normal Aa Gradient = 2.5 + (0.21) (age in years) OR = age+4/4

- **A- gradient**
- **PAO2= 760-47 x 0.21 - 15/0.8 = ~ 141**
- **PAO2 - PaO2= 141 - 91= 50**
- **Thus raised A-a gradient**

∴ HAGMA/ NAGMA/ 1° Resp alk

c. List four (4) explanations for these test results. (4 marks)
- **Acute decompensation of CLD- eg. H+M**
- **Post seizure**
- **Sepsis- esp resp source**
- **Toxic ingestion- eg ethanol/ salicylate/ chlorpromazine**
- **Head injury**
- **SIADH**

You assess the patient as being moderately dehydrated.

d. List four (4) points in your approach to his fluid replacement regime. (4 marks)
Change in Serum Sodium = (Fluid Sodium - Serum Sodium) / (Total Body Water + 1)
Total Body Water = (Wt in kg * % Water)
- **Na- urgent 3% 1- 2ml/kg/ hr via central line for 2-3/24**
  - **Aim to ↑ $SeNa^+$ by 1- 2 mmol/l per hr for 3-4 hrs**
  - **Aim ↑ < 12 mmol/ 24 hrs**
- **Volume- NSaline 150 ml/hr - slow**
- **Aims:**
  - **establish UO- > 0.5 ml/hr**
  - **BP > 90**

- **Conc albumin may be required to maintain BP**

The Delta Ratio (Δ/Δ)

The delta ratio is sometimes used in the assessment of elevated anion gap metabolic acidosis to determine if a mixed acid base disorder is present.

Delta ratio = Δ Anion gap/Δ [HCO3-] or ↑**anion gap/ ↓ [HCO3-]**

$$= \frac{\text{Measured anion gap} - \text{Normal anion gap}}{\text{Normal [HCO3-]} - \text{Measured [HCO3-]}}$$

$$= \frac{(AG - 12)}{(24 - [HCO3-])}$$

In order to understand this, let us re-examine the concept of the anion gap.

If one molecule of metabolic acid (HA) is added to the ECF and dissociates, the one H+ released will react with one molecule of HCO3- to produce CO2 and H2O. This is the process of buffering. **The net effect will be an increase in unmeasured anions by the one acid anion A- (ie anion gap increases by one) and a decrease in the bicarbonate by one meq.**

Now, if all the acid dissociated in the ECF and all the buffering was by bicarbonate, then the increase in the AG should be equal to the decrease in bicarbonate so the ratio between these two changes (which we call the delta ratio) should be equal to one.

As described previously, more than 50% of excess acid is buffered intracellularly and by bone, not by HCO3- . In contrast, most of the excess anions remain in the ECF, because anions cannot easily cross the lipid bilayer of the cell membrane. **As a result, the elevation in the anion gap usually exceeds the fall in the plasma [HCO3-]. In lactic acidosis, for example, the Δ/Δ ratio averages 1.6:1**.

On the other hand, although the same principle applies to ketoacidosis, the ratio is usually close to 1:1 in this disorder because the loss of ketoacids anions (ketones) lowers the anion gap and tends to balance the effect of intracellular buffering. Anion loss in the urine is much less prominent in lactic acidosis because the associated state of marked tissue hypoperfusion usually results in little or no urine output.

A delta-delta value below 1:1 indicates a greater fall in [HCO3-] than one would expect given the increase in the anion gap. This can be explained by a mixed metabolic acidosis, i.e a combined elevated anion gap acidosis and a normal anion gap acidosis, as might occur when lactic acidosis is superimposed on severe diarrhoea. In this situation, the additional fall in HCO3- is due to further buffering of an acid that does not contribute to the anion gap. (i.e addition of HCl to the body as a result of diarrhoea)

A value above 2:1 indicates a lesser fall in [HCO3-] than one would expect given the change in the anion gap. This can be explained by another process that increases the [HCO3-],i.e. a concurrent metabolic alkalosis. Another situation to consider is a pre-existing high HCO3- level as would be seen in chronic respiratory acidosis.

Example

If the AG was say 26 mmols/l (an increase of 14 from the average value of 12), it might be expected that the HCO_3^- would fall by the same amount from its usual value (ie 24 minus 14 = 10mmols/l). If the actual HCO_3^- value was different from this it would be indirect evidence of the presence of certain other acid-base disorders (see Guidelines below).

Problem

A problem though: the above assumptions about all buffering occurring in the ECF and being totally by bicarbonate are not correct. Fifty to sixty percent of the buffering for a metabolic acidosis occurs intracellularly. This amount of H^+ from the metabolic acid (HA) does not react with extracellular HCO_3^- so the extracellular [HCO_3^-] will not fall as far as originally predicted. The acid anion (ie A^-) however is charged and tends to stay extracellularly so the increase in the anion gap in the plasma will tend to be as much as predicted.

Overall, this significant intracellular buffering with extracellular retention of the unmeasured acid anion will cause the value of the delta ratio to be greater than one in a high AG metabolic acidosis.

Caution

Inaccuracies can occur for several reasons, for example:

- Calculation requires measurement of 4 electrolytes, each with a measurement error
- Changes are assessed against 'standard' normal values for both anion gap and bicarbonate concentration.

Sometimes these errors combine to produce quite an incorrect value for the ratio. As an example, patients with hypoalbuminaemia have a lower 'normal' value for anion gap so using the standard value of 12 to compare against must lead to an error. Do not overinterpret your result and look for supportive evidence especially if the diagnosis is unexpected.

3.3.3 Guidelines for Use of the Delta Ratio

Some general guidelines for use of the delta ratio when assessing metabolic acid-base disorders in provided in the table below.

Overall Advice: **Be very wary of over-interpretation** - Always check for other evidence to support the diagnosis as an unexpected value without any other evidence should always be treated with great caution.

Delta Ratio	Assessment Guideline
< 0.4	Hyperchloraemic normal anion gap acidosis
0.4 - 0.8	Consider combined high AG & normal AG acidosis BUT note that the ratio is often <1 in acidosis associated with renal failure
1 to 2	Usual for uncomplicated high-AG acidosis Lactic acidosis: average value 1.6 DKA more likely to have a ratio closer to 1 due to urine ketone loss (esp if patient not dehydrated)
> 2	Suggests a pre-existing elevated HCO_3 level so consider: • a concurrent metabolic alkalosis, or • a pre-existing compensated respiratory acidosis

Warning

Be very wary of over-interpretation - Always check for other evidence to support the diagnosis as an unexpected value without any other evidence should always be treated with great caution.

A high ratio

A high delta ratio can occur in the situation where the patient had quite an elevated bicarbonate value at the onset of the metabolic acidosis. Such an elevated level could be due to a pre-existing metabolic alkalosis, or to compensation for a pre-existing respiratory acidosis (ie compensated chronic respiratory acidosis). With onset of a metabolic acidosis, using the 'standard' value of 24 mmol/l as the reference value for comparison when determining the 'decrease in bicarbonate' will result in an odd result.

A low ratio

A low ratio occurs with **hyperchloraemic (or normal anion gap) acidosis**. The reason here is that the acid involved is effectively hydrochloric acid (HCl) and the rise in plasma [chloride] is accounted for in the calculation of anion gap (ie chloride is a 'measured anion'). The result is that the 'rise in anion gap' (the numerator in the delta ration calculation) does not occur but the 'decrease in bicarbonate' (the denominator) does rise in numerical value. The net of of both these changes then is to cause a marked drop in delta ratio, commonly to < 0.4

Lactic acidosis

In **lactic acidosis**, the average value of the delta ratio in patients has been found to be is 1.6 due to intracellular buffering with extracellular retention of the anion. As a general rule, in uncomplicated lactic acidosis, the rise in the AG should always exceed the fall in bicarbonate level.

Diabetic ketoacidosis

The situation with a pure **diabetic ketoacidosis** is a special case as the urinary loss of ketones decreases the anion gap and this returns the delta ratio downwards towards one. A further complication is that these patients are often fluid resuscitated with 'normal saline' solution which results in a increase in plasma chloride and a decrease in anion gap and development of a 'hyperchloraemic normal anion gap acidosis' superimposed on the ketoacidosis. The result is a further drop in the delta ratio.

Question 8 (12 marks)

A 26 year old man presents four hours following a recreational drug binge. A friend reports that he has been using large doses of "ICE".

a. List four examination findings that may be seen with ICE use. (4 marks)
   - **Acute psychosis**
   - **Agitation**
   - **Sweating**
   - **Hypertension**
   - **Hyperthermia**
   - **Tremor/ tremulous**
   - **Poor dentition**
   - **Collingwood FC tattoos**

The patient refuses to remain for assessment. You are required to chemically sedate the patient.

b. List your preferred drug regime in this situation, for the stated circumstances (include drug, dose and route): (3 marks)

i) Will accept oral treatment:
   - **Lorazepam 2mg**
   - **Diazepam 20mg**

ii) Refuses oral medication, moderate degree of agitation
   - **IM midazolam 5-10 mg**
   - **IV Diazepam 2.5-5mg**
   - **IM ziprasidone 20mg + Lorazepam 2mg**
   - **IM Olanzepine 10mg**

iii) Refuses oral medication, going nuts:
   - **IM/IV Droperidol 5-10mg**
   - **IM ketamine 4mg/kg**

The patient is sedated. Physical restraint is not required. Your complete assessment detects no organic pathology.

c. List five (5) key components to the ongoing management of this patient. (5 marks)
   - **Resus cubicle**
   - **Close observation/ regular neuro obs**
   - **Airway protection/ avoid aspiration- L lateral**
   - **Continuous non invasive monitoring**
   - **IV fluids if dry**
   - **Avoid overstimulation**
   - **Serial assessments of state**
   - **Liaise with Psych- RV ASAP for disposition decision and follow up**
   - **Contact NOK**

- **Document progress and decisions made**

Question 9 (18 marks)

A 5 year old girl is referred by a GP with pallor and lethargy. On examination she is extremely pale but appears alert and interactive. Her observations: BP100/60 mmHg HR 110/min RR 20/min GCS 15 O2 saturations 98%on room air Temperature 36.8°C

		Reference Range	Units
Hb	35	(101-131)	g/L
WCC	9.1	(6.0-11.0)	10^9/L
PLT	260	(150-450)	10^9/L
RBC	2.18	(3.9-5.3)	10^{12}/L
MCV	56.0	(75-85)	fL
MCH	16	(23-31)	pg
MCHC	286	(310-355)	g/L
Retic %	3.6	(0.2-2.0)	

a. List four (4) different pathological causes for these results. (4 marks)

- **Fe deficiency *(Must be mentioned 1st as the most likely in a seemingly well child- per examiners- though Reticulocyte count should be < 2% for FDA)***
  - **poor dietary intake- reliance on cows milk**
  - **chronic blood loss**
  - **poor intestinal absorption**
    - **Coeliac disease**
    - **Crohn's disease**
- **Thalassaemia major**
- **Thalassaemia minor + a secondary medical condition eg unstable DM/ folate deficiency**
- **Haemolytic anaemia**

b. List six (6) further investigations that you would perform in the emergency department for this girl. (6 marks)

- **Iron studies**
- **Hb electrophoresis**
- **Peripheral blood film**
- **B12 level**
- **Folate level**
- **Faecal occult blood**
- **LDH**
- **Haptoglobin**
- **Unconjugated bilirubin**
- **Coombs test**

} **Haemolytic anaemia**

The mother refuses any blood products based on religious beliefs.

c. List five (5) situations under which you may override these wishes. (5 marks)

- **Likelihood death or serious permanent damage**
- **Approval by hospital superintendent**
- **Disagreement between parents, other parent consents**
- **Court order**
- **Mother not legal guardian**
- **Suspicion for NAI**
- **Mother does not have capacity e.g. mental health disorder**

None of these situations are met.

d. List three (3) alternative treatments that you could institute, other than the provision of blood products for this patient. (3 marks)

- **Iron infusion**

- **Synthetic haemoglobin**
- **EPO infection**