An update in contemporary trauma management

ACEM Winter Symposium Rotorua NZ 2019

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Trauma Facts

- 6 x10^6 trauma deaths each year
- 100 x 10^6 hospital admissions / yr
- Road trauma by 2030 – 5th cause of death worldwide
- Trauma in the elderly on the rise

- Haemorrhagic shock – most preventable deaths
- Traumatic brain injury (TBI) –most trauma-related deaths in HICs
- Musculoskeletal injury – most surgery and most disability

Clinical Series on Trauma Surgery. Lancet 2012

What has changed over the last 10-20 years ?

- Airway management
- Spinal injury- immobilization and imaging
- Shock management
- ? Head injury management
- Elderly trauma

Airway management

- Pre-intubation checklist
- Significant increase in successful first attempt at intubation
- ANZEDAR (Aust NZ Airway Registry)

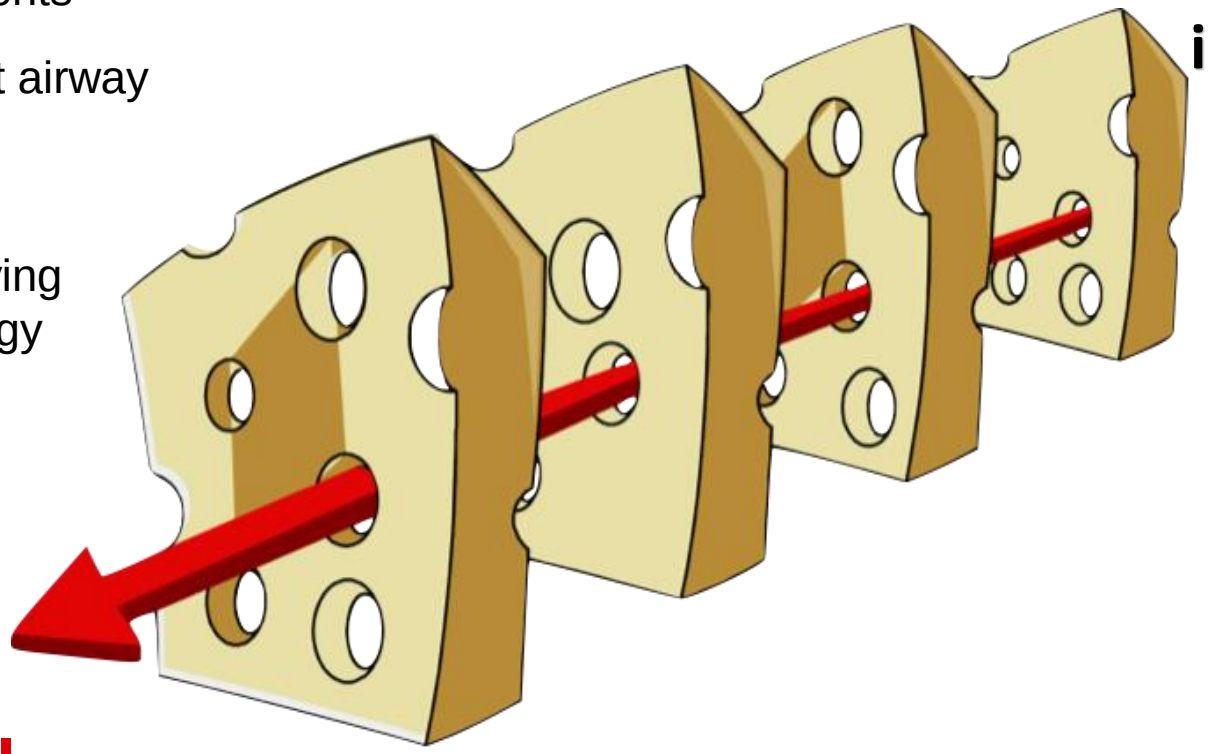
Poor identification of high risk patients

Equipment adequacy and redundancy

Decision to intubate

Difficult airway

Underlying pathology



Critical desaturation

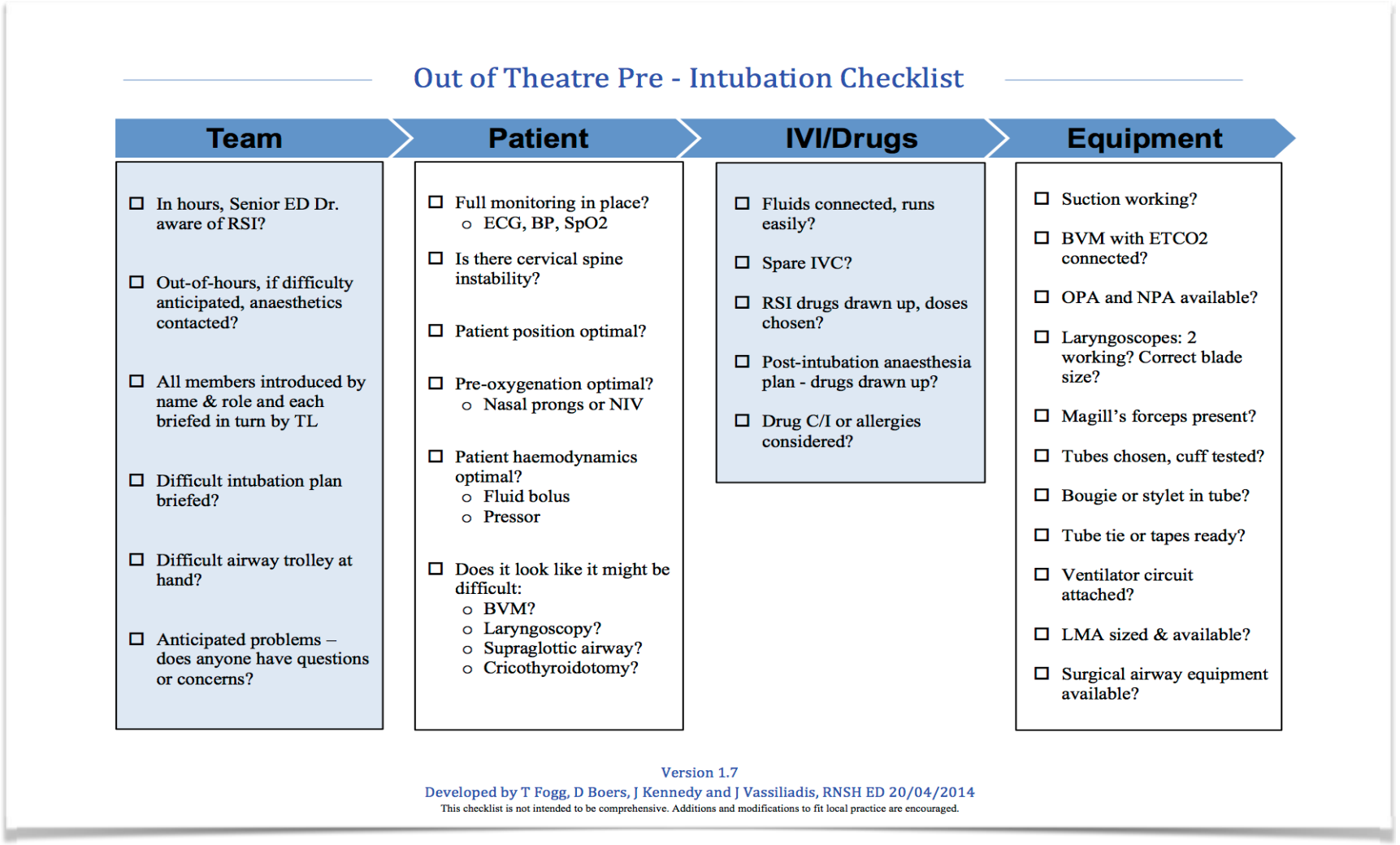
Failure to plan for failure

Poor patient optimisation

Communication errors

Steep (or absent) authority gradient

ED Pre-intubation checklist



Out of Theatre Pre - Intubation Checklist

Team

- ☐ In hours, Senior ED Dr. aware of RSI?
- ☐ Out-of-hours, if difficulty anticipated, anaesthetics contacted?
- ☐ All members introduced by name & role and each briefed in turn by TL
- ☐ Difficult intubation plan briefed?
- ☐ Difficult airway trolley at hand?
- ☐ Anticipated problems – does anyone have questions or concerns?

Patient

- ☐ Full monitoring in place?
  - ECG, BP, SpO2
- ☐ Is there cervical spine instability?
- ☐ Patient position optimal?
- ☐ Pre-oxygenation optimal?
  - Nasal prongs or NIV
- ☐ Patient haemodynamics optimal?
  - Fluid bolus
  - Pressor
- ☐ Does it look like it might be difficult:
  - BVM?
  - Laryngoscopy?
  - Supraglottic airway?
  - Cricothyroidotomy?

IVI/Drugs

- ☐ Fluids connected, runs easily?
- ☐ Spare IVC?
- ☐ RSI drugs drawn up, doses chosen?
- ☐ Post-intubation anaesthesia plan - drugs drawn up?
- ☐ Drug C/I or allergies considered?

Equipment

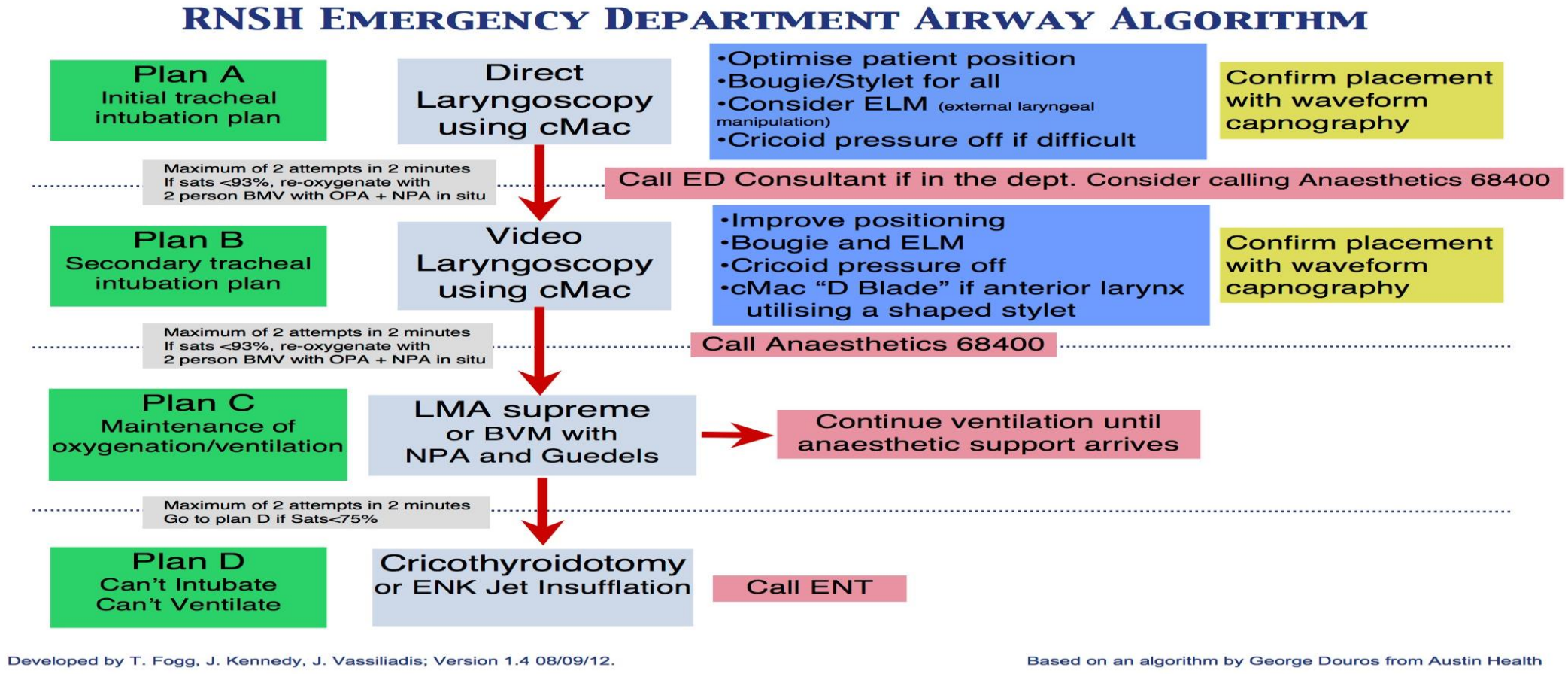
- ☐ Suction working?
- ☐ BVM with ETCO2 connected?
- ☐ OPA and NPA available?
- ☐ Laryngoscopes: 2 working? Correct blade size?
- ☐ Magill's forceps present?
- ☐ Tubes chosen, cuff tested?
- ☐ Bougie or stylet in tube?
- ☐ Tube tie or tapes ready?
- ☐ Ventilator circuit attached?
- ☐ LMA sized & available?
- ☐ Surgical airway equipment available?

Version 1.7

Developed by T Fogg, D Boers, J Kennedy and J Vassiliadis, RNSH ED 20/04/2014

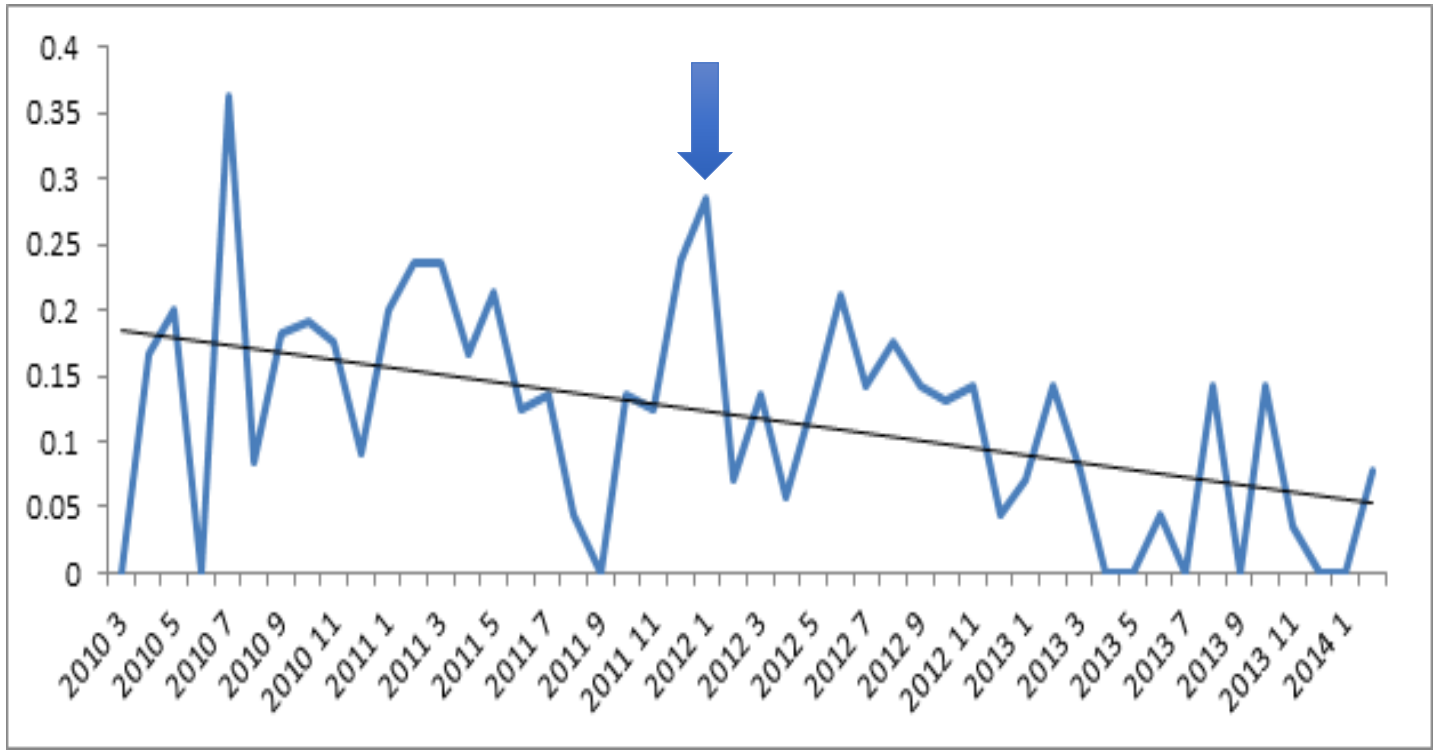
This checklist is not intended to be comprehensive. Additions and modifications to fit local practice are encouraged.

ED Airway Algorithm



First pass Intubation Failure Rate decrease over time (RNSH data)

- First pass success: 84% to 94% (P<0.001)



Spinal Injury Investigation and Management

- Soft collars use pre-hospital (convenience > evidence)
- Protect spine until can be cleared
- Use Nexus/ Canadian Cx spine rules
- Unable to clear clinically – CT Cx spine
- If spinal # detected - Rigid collar (Philadelphia or Miami J)
- MRI to detect ligament injury
- CT Angio for blunt cerebro-vascular injury (Denver criteria)

KEY PRINCIPLES

Agency for Clinical Innovation

Use of foam collars for cervical spine immobilisation

Initial management principles

Scientific evidence on the use of cervical collars

There is no scientific evidence that any type of cervical collar used in prehospital transport or initial trauma management is effective in stabilising an acutely injured cervical spine or preventing further neurological deterioration in those with spinal cord injury (SCI).[1] However, there is evidence that rigid collars can lead to significant complications and morbidity when used to immobilise the c-spine.[2-3]

- If cervical bony injury is identified, or if the patient cannot be cleared in ED due to competing priorities, apply a locally agreed cervical immobilisation collar such as a Philadelphia or Miami J collar, and refer to neurosurgery for advice.

Detection of Blunt Cerebrovascular Injury (BCVI)

CT angio Ix of choice

Denver screening criteria for BCVI

Risk factors for BCVI

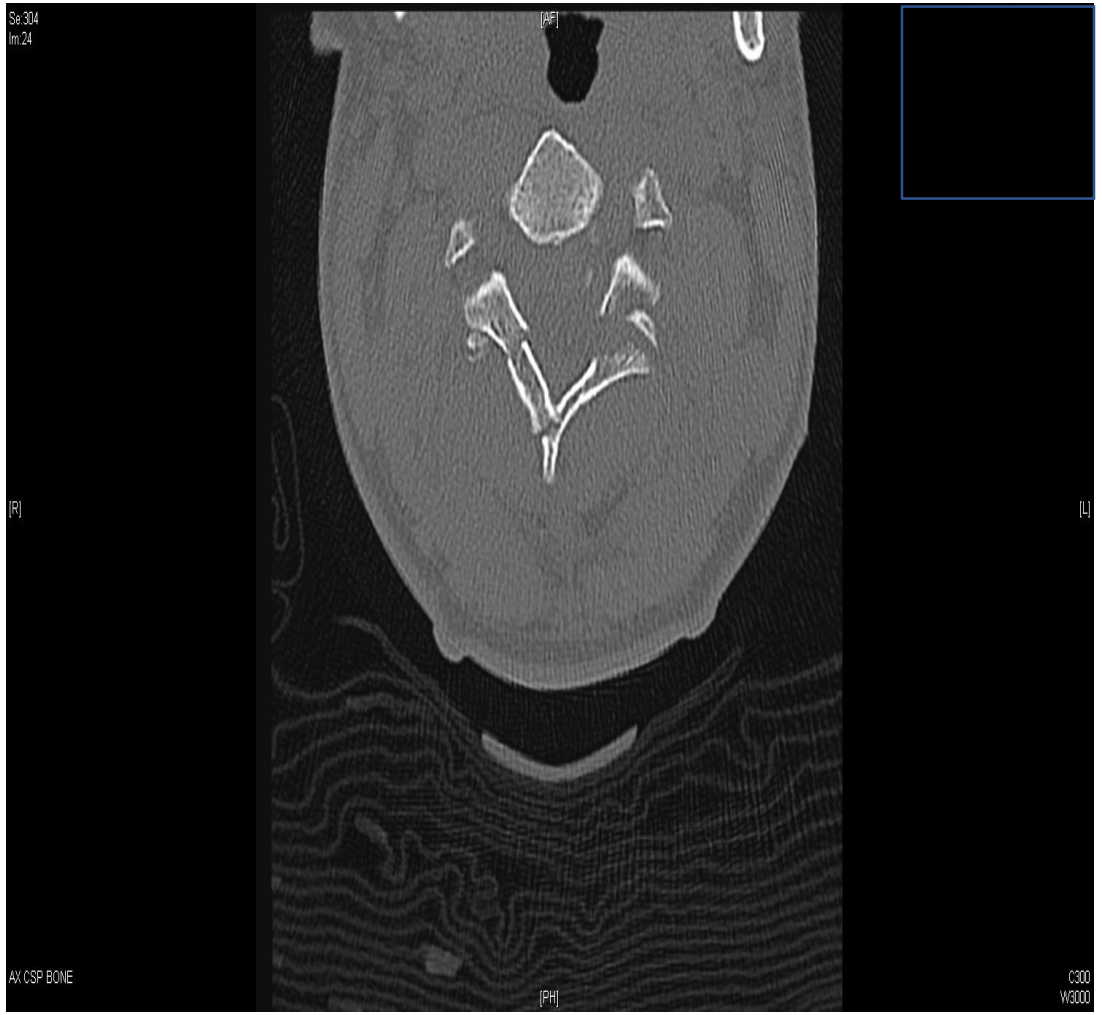
- High-energy transfer mechanism with
  - Lefort II or III fracture
  - Cervical spine fracture patterns: subluxation, fractures extending into the transverse foramen, fractures of C1-C3
  - Basilar skull fracture with carotid canal involvement
  - Diffuse axonal injury with GCS =6
  - Near hanging with anoxic brain injury

40 yr male BIBH post 15 m fall down cliff

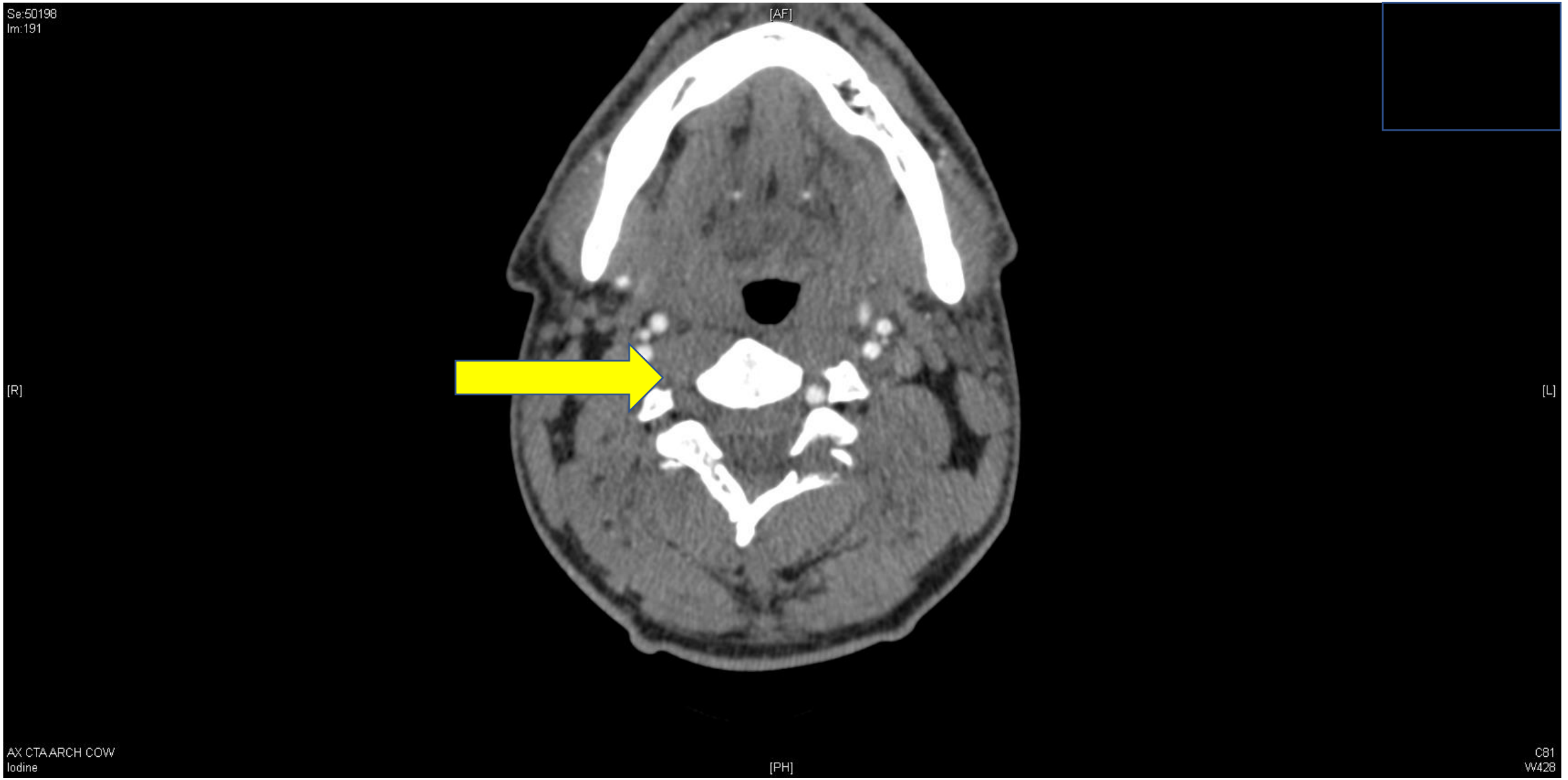
Neck pain

Paraesthesia arms and legs

40 yr male BIBH post 15 m fall down cliff
Neck pain / Paraesthesia arms and legs
Hangman's Fracture C2



CTA C2- R Vertebral Art Dissection



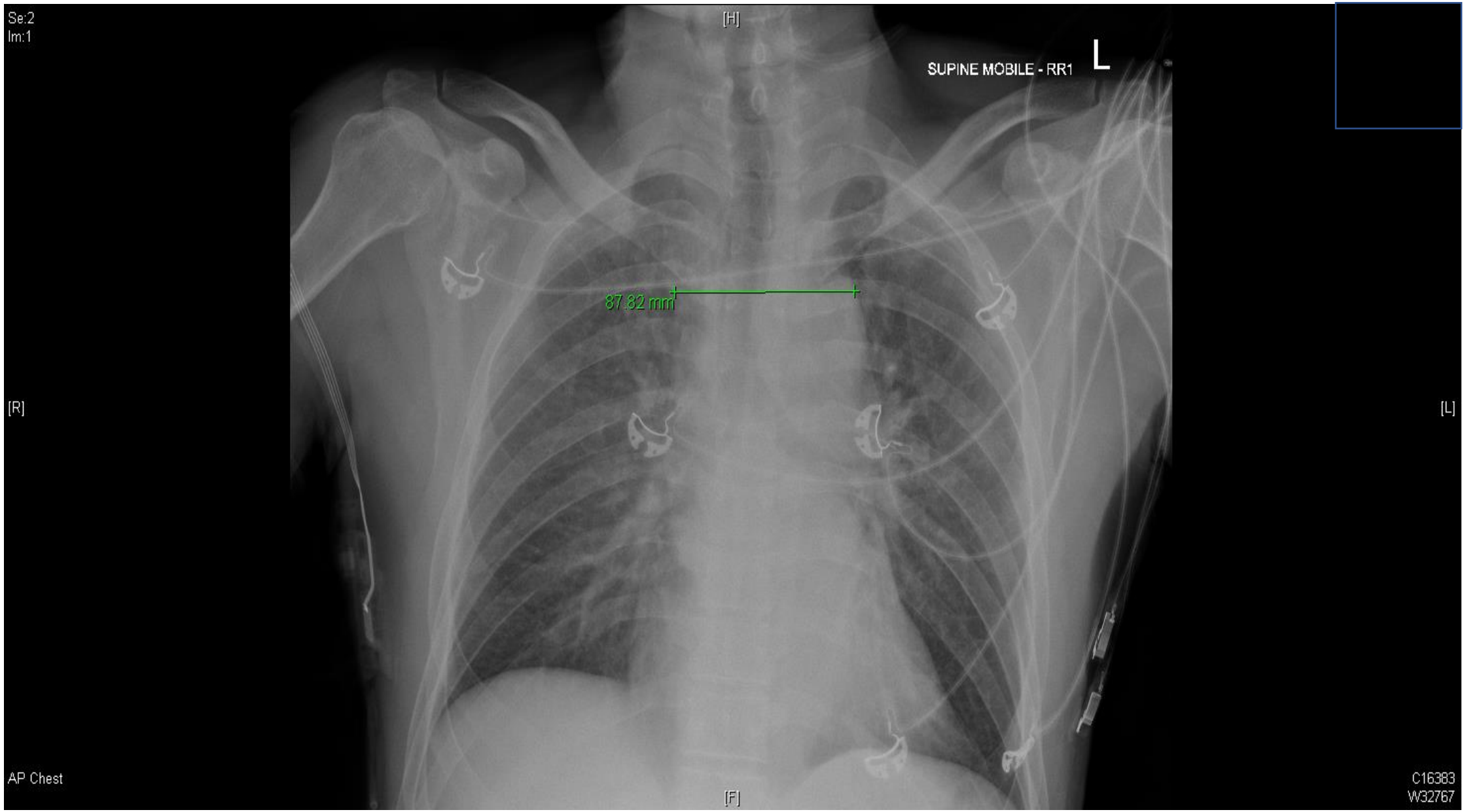

Se:405
Im:18
AR
10 cm
A
L
F
VR ARCH TO COW
CONTRAST
C128
W256

MRI in spinal injury

- Fracture dislocation at T11-12 with compression/ transection of the conus
- The anterior and posterior longitudinal ligaments, ligamentum flavum and the interspinous ligaments are disrupted.
- Anterior epidural haematoma maximal thickness of 10 mm upper border of T11 to the T11-12 interspace.

Chest trauma

52 yr male high speed MVA – chest pain



Traumatic Aortic Rupture

Aortic stent

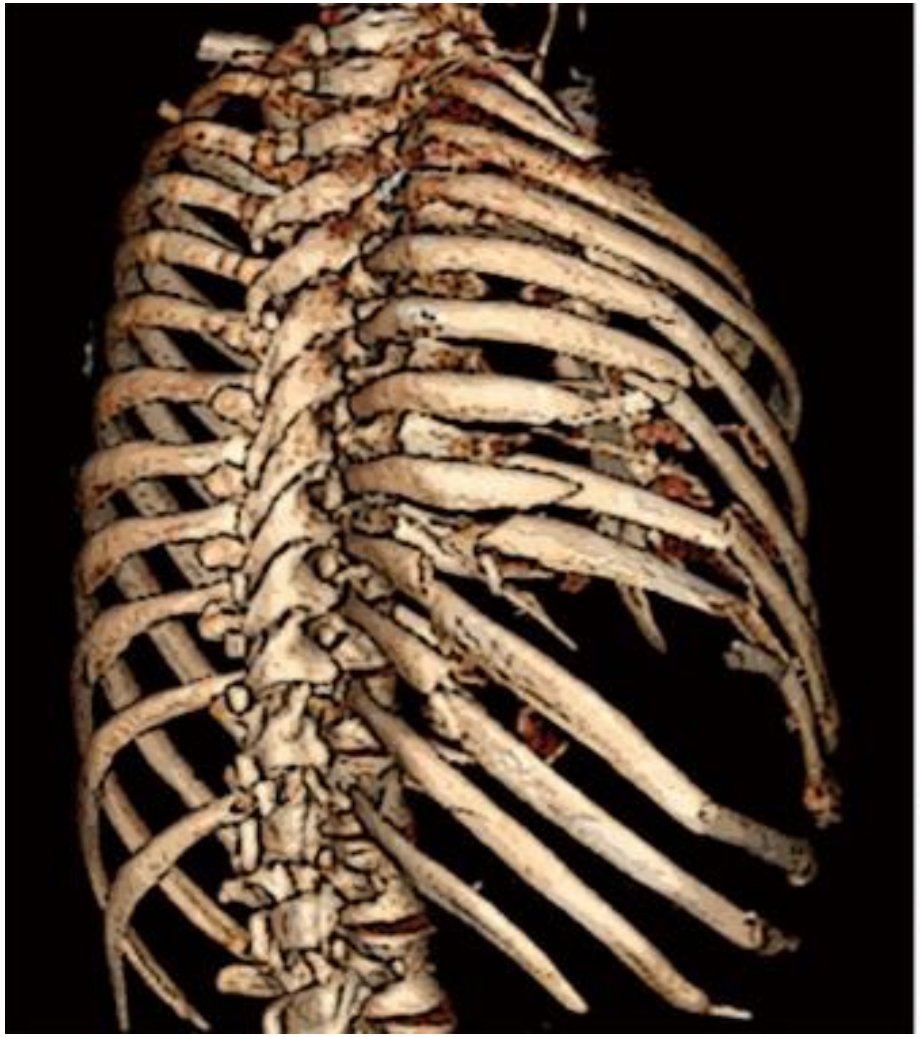
Rib Fracture Fixation

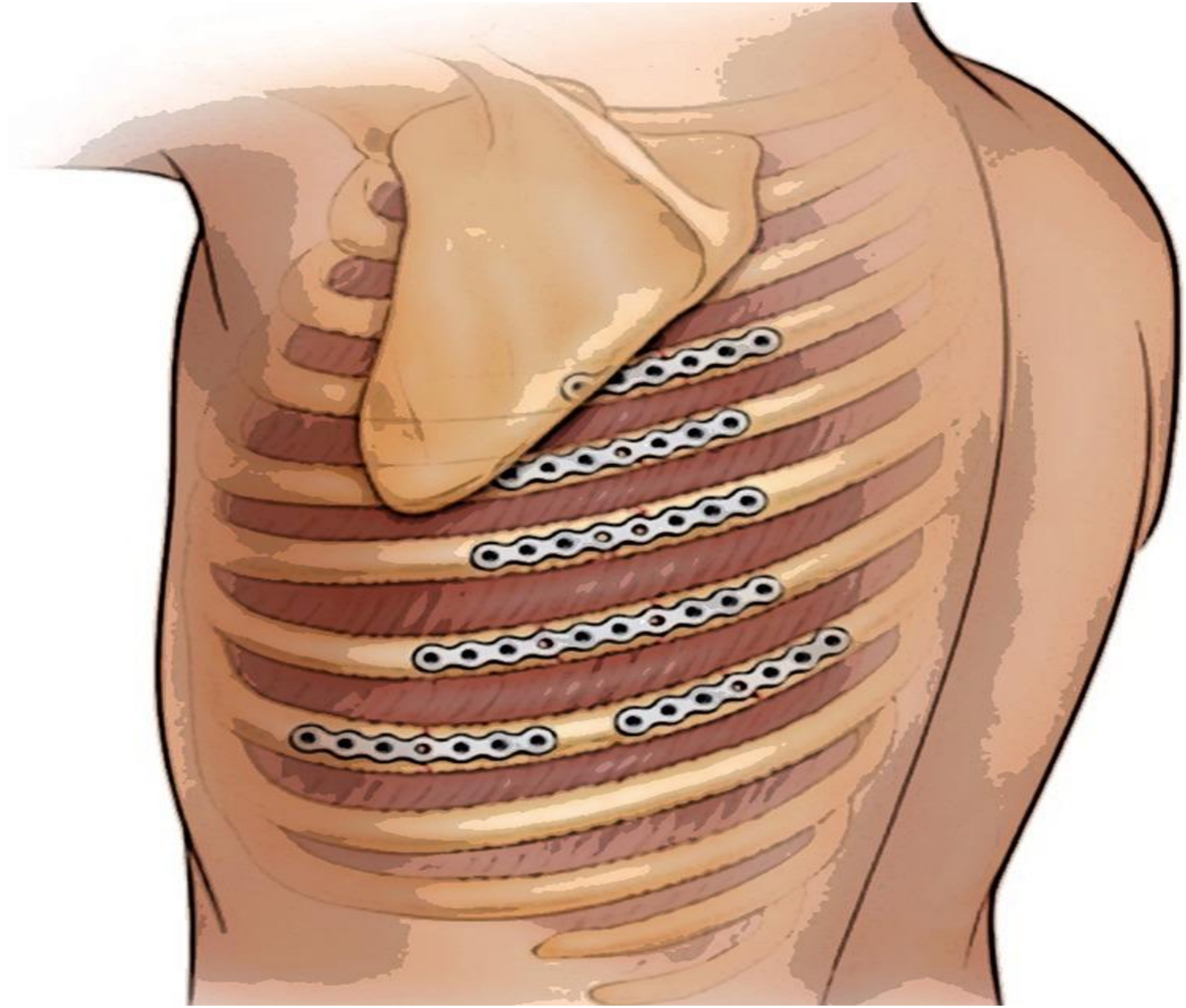
- Consider if 3 or more displaced rib #s
- Flail chest
- Fail non-operative Mx
- Require other chest procedures

- Contraindications
  - Unstable spine #
  - Severe Traumatic brain injury

Chest wall stabilization in trauma patients: why, when, and how? J Thor Dis 2018

Rib Fracture fixation





Benefits of chest wall stabilisation

Shortened duration of mechanical ventilation

Decreased incidence of pneumonia

Decreased need for tracheostomy

Shortened ICU length of stay

Shortened hospital length of stay

Decreased mortality in patients with flail chest

Rapid return to work

Cost effectiveness

Decreased use of narcotics

Earlier mobilization

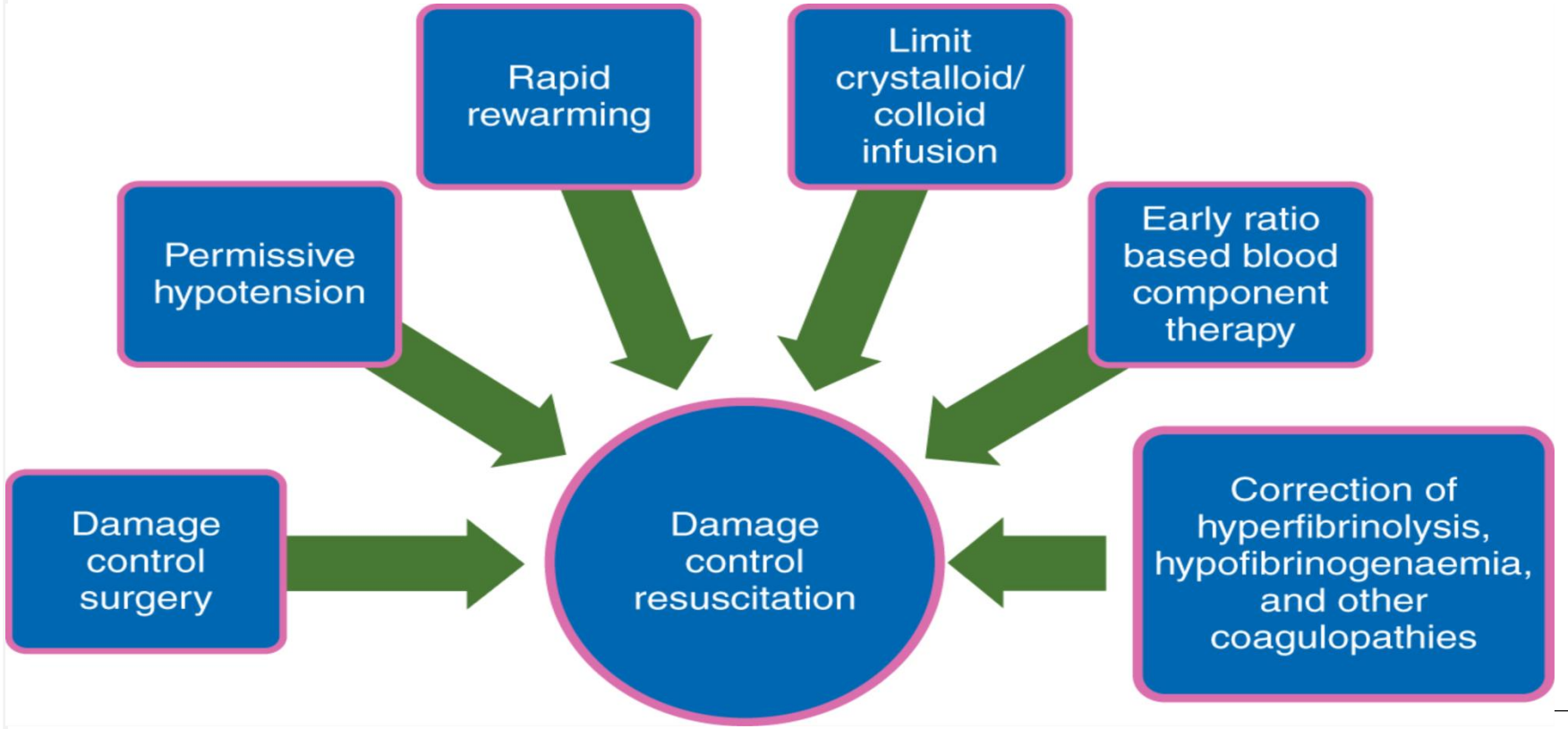
Haemorrhage and shock management

Some Science ?

Management of Acute Traumatic Haemorrhage

- Exsanguination is a common cause of death in injured patients
- Early surgical (or Radiological) control of haemorrhage
- Goals of early damage control resuscitation
- Early detection and management of coagulopathy

Damage control resuscitation



From: Acute traumatic coagulopathy: pathophysiology and resuscitation

Br J Anaesth. 2016;117(suppl_3):iii31-iii43. doi:10.1093/bja/aew328


14 RBC
3 FFP
0 platelets
0 cryo
25 liters
of crystalloid
2006

Acute Traumatic Coagulopathy
Karim Brohi J Trauma 2003

Acute Traumatic Coagulopathy

Karim Brohi, BSc, FRCS, FRCA, Jasmin Singh, MB, BS, BSc, Mischa Heron, MRCP, FFAEM, and Timothy Coats, MD, FRCS, FFAEM

Background: Traumatic coagulopathy is thought to be caused primarily by fluid administration and hypothermia.

Methods: A retrospective study was performed to determine whether coagulopathy resulting from the injury itself is a clinically important entity in severely injured patients.

Results: One thousand eight hundred sixty-seven consecutive trauma patients were reviewed, of whom 1,088 had full data sets. Median Injury Severity Score was 20, and 57.7% had an Injury Severity Score > 15; 24.4% of patients had a significant coagulopathy. Patients with an acute coagulopathy had significantly higher mortality (46.0% vs. 10.9%; χ^2, $p < 0.001$). The incidence of coagulopathy increased with severity of injury, but was not related to the volume of intravenous fluid administered ($r^2 = 0.25$, $p < 0.001$).

Conclusion: There is a common and clinically important acute traumatic coagulopathy that is not related to fluid administration. This is a marker of injury severity and is related to mortality. A coagulation screen is an important early test in severely injured patients.

Key Words: Traumatic coagulopathy, Hypothermia, Fluid administration.

J Trauma. 2003;54:1127–1130.

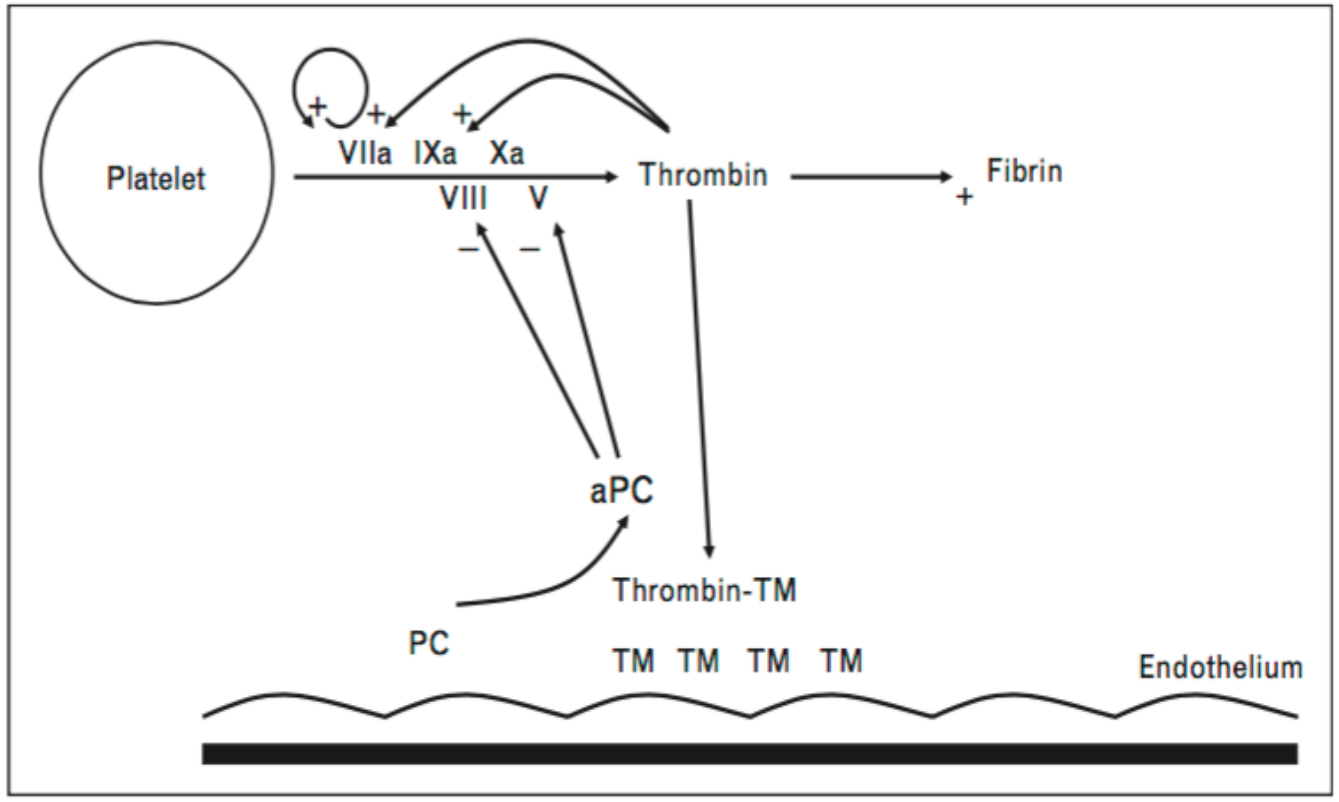
What is ATC?

- Multifocal global failure of coagulation
- Driven by tissue trauma / systemic hypoperfusion and shock
- Activated Protein C (aPC) and thrombomodulin central to process
- Hyperfibrinolysis
- Platelet dysfunction

Mechanism of ATC
Brohi et al 2007

Figure 1 Anticoagulation

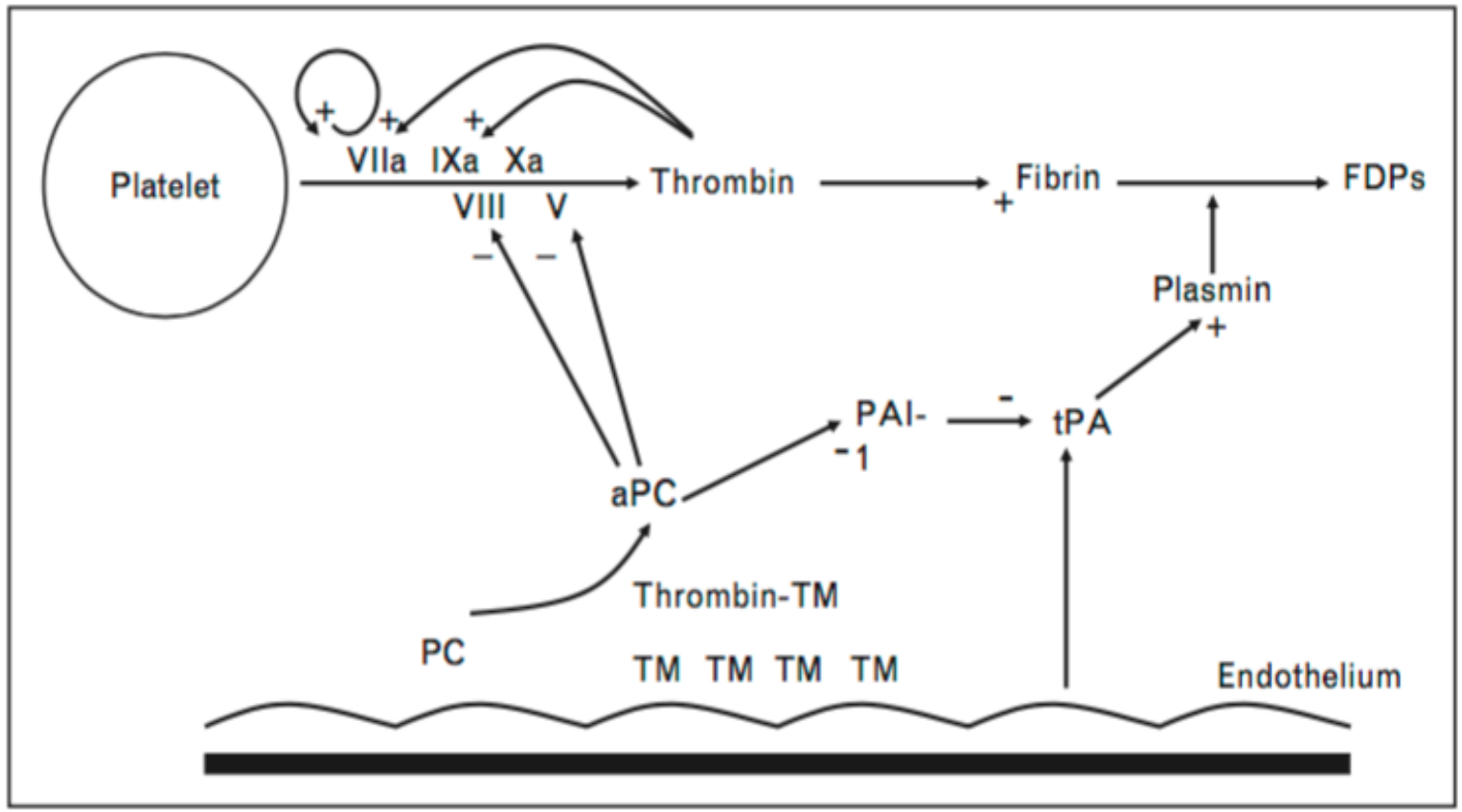
Thrombin is generated primarily via the 'extrinsic' pathway with multiple feed-forward loops. When thrombomodulin (TM) is presented by the endothelium, it complexes thrombin which is no longer available to cleave fibrinogen. This anticoagulant thrombin activates protein C which reduces further thrombin generation through inhibition of cofactors V and VIII.



Mechanism of hyperfibrinolysis
Brohi et al 2007

Figure 2 Hyperfibrinolysis

Tissue plasminogen activator (tPA) is released from the endothelium by injury and hypoperfusion and cleaves plasminogen to initiate fibrinolysis. Activated protein C (aPC) consumes plasminogen activator inhibitor-1 (PAI-1) when present in excess, and reduced PAI-1 leads to increased tPA activity and hyperfibrinolysis.



Clinical Significance of ATC

- Worse outcome
  - Increase risk of death by factor X 4-8
  - Increase ventilator time, LOS
- Increased transfusion requirements
- Increased thrombosis risk
  - Protein C depletion / TAFI (Thrombin -activatable fibrinolysis inhibitor)
- Increased risk of sepsis

Brohi et al Curr Opin Crit Care 2007

Problems with standard coagulation tests

- Lab PT/APTT ----20-60 mins
- Describe initial few mins of coagulation
  - Normal clotting time 15-30 mins
- No assessment of
  - Clot quality or strength
  - Fibrinolytic activity
  - Platelet function
- Role of TEG or RoTEM

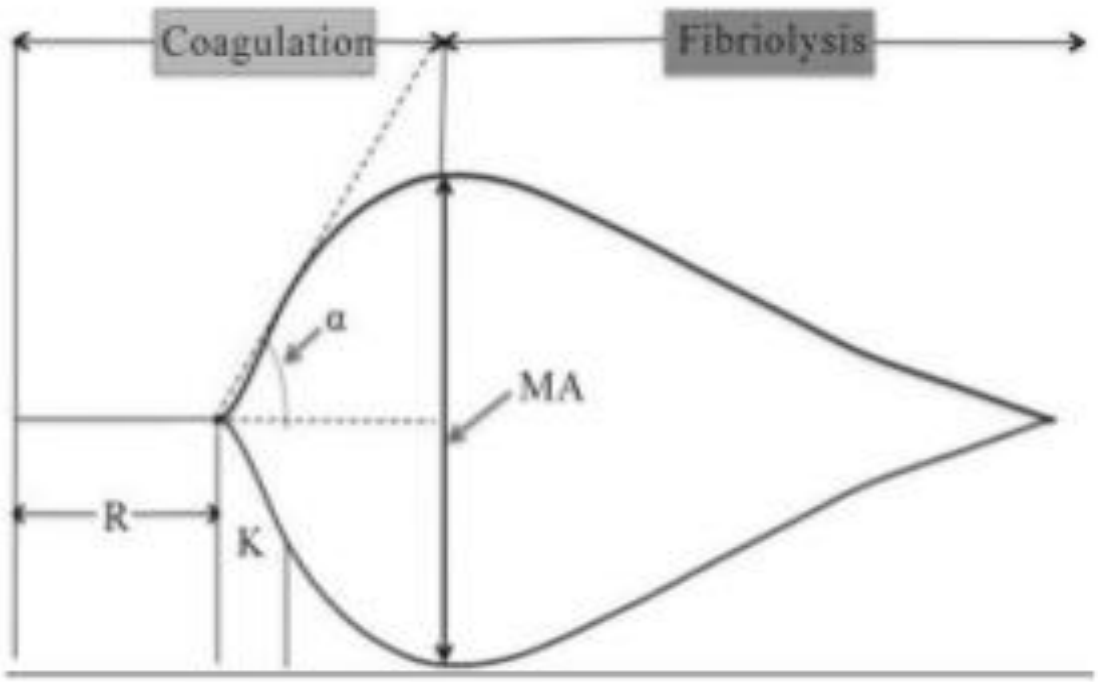
Role of TEG or ROTEM

Thromboelastography v Thromboelastometry

Thromboelastography

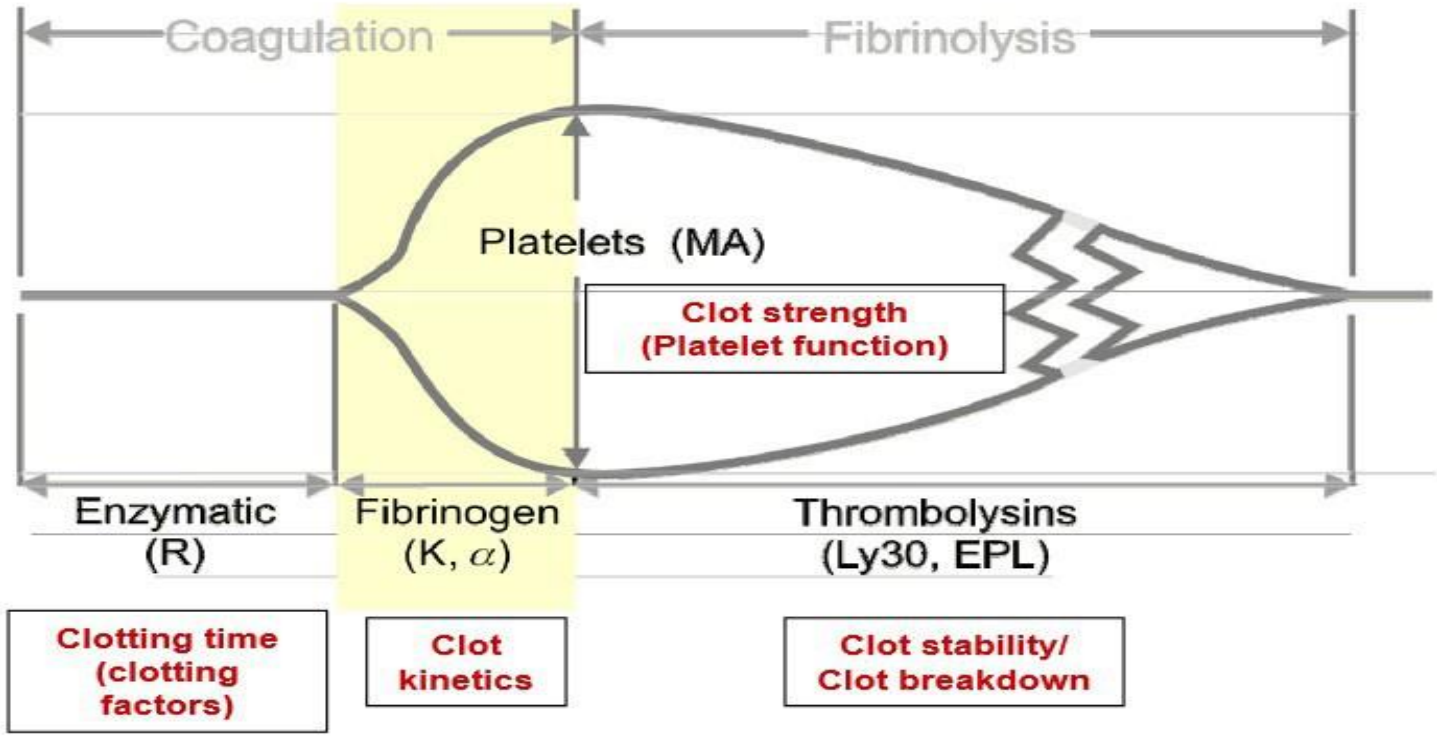
(Dr V Kumar Health and Medicine

Interpretation of TEG tracing



TEG parameter	Correlations with phase of coagulation
Reaction time (min)	Time between initiation of coagulation cascade to initial formation of fibrin
Kinetic time (min)	Time between initial formation of fibrin to specific clot firmness (20 mm)
α-angle (degrees)	Rate of fibrin formation and crosslinking
Maximum amplitude (mm)	Maximum clot strength
Lysis-30 (%)	Fibrinolysis 30 minutes after maximum amplitude

What Does TEG® Report?



TEG- Guided Transfusion Strategy (emDocs.net)

TEG Value	Transfuse
TEG-ACT > 140	FFP
R time > 10	FFP
K time > 3	cryoprecipitate
α angle < 53	cryoprecipitate +/- platelets
MA < 50	platelets
LY30 > 3%	tranexamic acid

Figure 5. Recommended Transfusion Strategies[3]

Massive Transfusion Guidelines

Massive transfusion – a transfusion of half of one blood volume (5 units) in 4 hours, or more than one blood volume (10 units) in 24 hours in adults (blood volume is aproximately 70 mL/kg)

Anticipate in patients with:

- severe thoracic, abdominal, pelvic or multiple long bone fracture
- penetrating torso injury with shock
- haemodynamic instability

Component Therapy v Whole blood

Component Therapy:

1U PRBC + 1U PLT + 1U FFP + 1 U cryo

680 COLD mL

- ***Hct 29%***
- ***Plt 80K***
- ***Coag factors 65%*** ***of initial concentration***
- ***1000 mg Fibrinogen***

WWB:

500 mL Warm

Hct: 38-50%

Plt: 150-400K

Coag: 100%

1000 mg Fibrinogen

Code Crimson

- Acute life-threatening haemorrhage requiring **life saving surgery**
- Shocked trauma patient- blunt or penetrating trauma with obvious thoracic or abdominal haemorrhage
- Activation: ED Team leader
  - De-activation Surgical consultant
- Notify
  - Blood bank – Massive Transfusion protocol
  - OR – Surgeon in OR / Anaesthetist in charge
  - OR Nursing staff aware

R Gruen et al

MJA 2013

Trauma and tranexamic acid

Research is needed to determine how patient selection and intercurrent treatment affect safety and efficacy

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

CRASH-2 trial collaborators*

Summary

Background Tranexamic acid can reduce bleeding in patients undergoing elective surgery. We assessed the effects of early administration of a short course of tranexamic acid on death, vascular occlusive events, and the receipt of blood transfusion in trauma patients.

Methods This randomised controlled trial was undertaken in 274 hospitals in 40 countries. 20 211 adult trauma patients with, or at risk of, significant bleeding were randomly assigned within 8 h of injury to either tranexamic acid (loading dose 1 g over 10 min then infusion of 1 g over 8 h) or matching placebo. Randomisation was balanced by centre, with an allocation sequence based on a block size of eight, generated with a computer random number generator. Both participants and study staff (site investigators and trial coordinating centre staff) were masked to treatment allocation. The primary outcome was death in hospital within 4 weeks of injury, and was described with the following categories: bleeding, vascular occlusion (myocardial infarction, stroke and pulmonary embolism), multiorgan failure, head injury, and other. All analyses were by intention to treat. This study is registered as ISRCTN86750102, Clinicaltrials.gov NCT00375258, and South African Clinical Trial Register DOH-27-0607-1919.

Findings 10 096 patients were allocated to tranexamic acid and 10 115 to placebo, of whom 10 060 and 10 067, respectively, were analysed. All-cause mortality was significantly reduced with tranexamic acid (1463 [14·5%] tranexamic acid group *vs* 1613 [16·0%] placebo group; relative risk 0·91, 95% CI 0·85–0·97; p=0·0035). The risk of death due to bleeding was significantly reduced (489 [4·9%] *vs* 574 [5·7%]; relative risk 0·85, 95% CI 0·76–0·96; p=0·0077).

Interpretation Tranexamic acid safely reduced the risk of death in bleeding trauma patients in this study. On the basis of these results, tranexamic acid should be considered for use in bleeding trauma patients.

Published Online
June 15, 2010
DOI:10.1016/S0140-6736(10)60835-5

See Online/Comment
DOI:10.1016/S0140-6736(10)60939-7

*Members listed at end of paper

Correspondence to:
Clinical Trials Unit, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK
crash@lshtm.ac.uk

Current widespread use of TxA in bleeding trauma patients is not supported by the current literature and may be causing harm

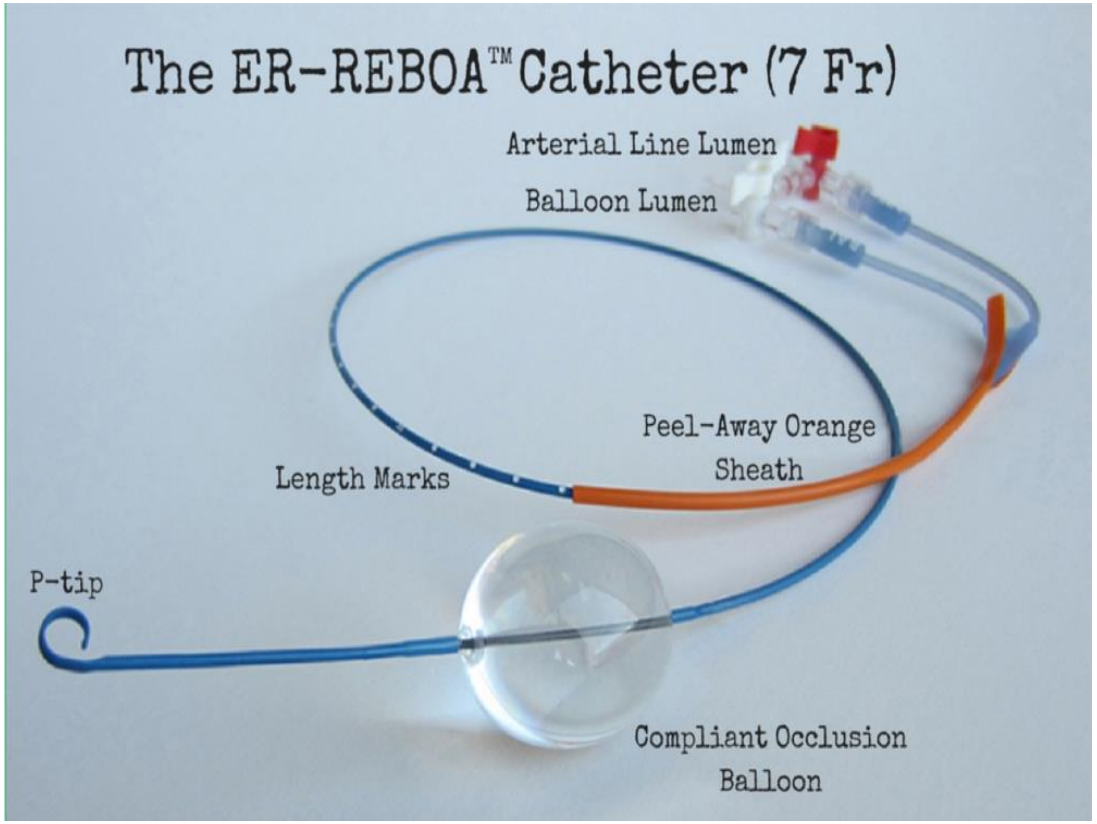
Gruen et al

- Effects of TxA on Acute Trauma Coagulopathy unknown as no coagulation studies were done in CRASH 2
- What is needed?
  - PRCT in controlled environment with laboratory monitoring of coagulation and standardised blood transfusion protocols essential before TxA is STANDARD care in trauma patients

- PATCH (Pre-hospital Anti-fibrinolytics for Trauma Coagulopathy and Haemorrhage) Study
  - early pre-hospital effect on mortality and 6/12 recovery as well as coagulation and clots

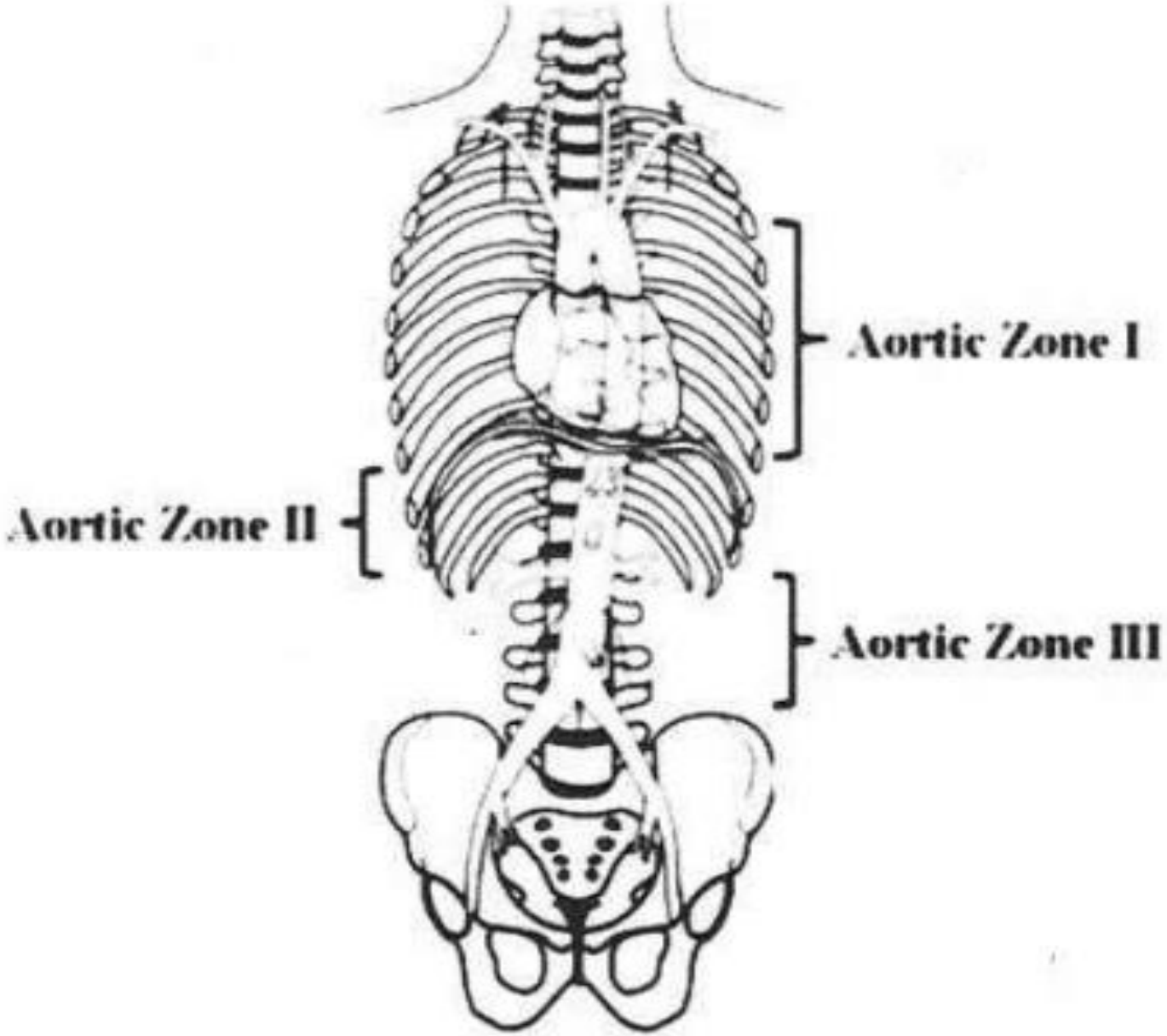
REBOA

- Resuscitative Endovascular Balloon Occlusion of the Aorta



REBOA

- Minimally invasive technique that uses a catheter to insert a balloon into the aorta to stop distal bleeding when inflated and allows time for patient to get to OR or IVR for definitive control of haemorrhage
- Indicated in life –threatening haemorrhage in chest, abdomen or pelvis
- Alternative to cross-clamping the aorta to control distal haemorrhage



Zone 1 = Origin of left subclavian artery to the celiac artery

Zone III = Lowest renal artery to aortic bifurcation

Utility of REBOA

- REBOA appears to be useful in sub-diaphragmatic haemorrhage
- Improves cardiac and cerebral perfusion
- Adjunct to standard surgical resuscitative tools
- Appears to improve survival but high mortality ?
- Patient selection may be an issue
- No extensive training necessary
- Should be widely available in MTS

Resuscitative Endovascular Balloon Occlusion of the Aorta in trauma: a systematic review of the literature

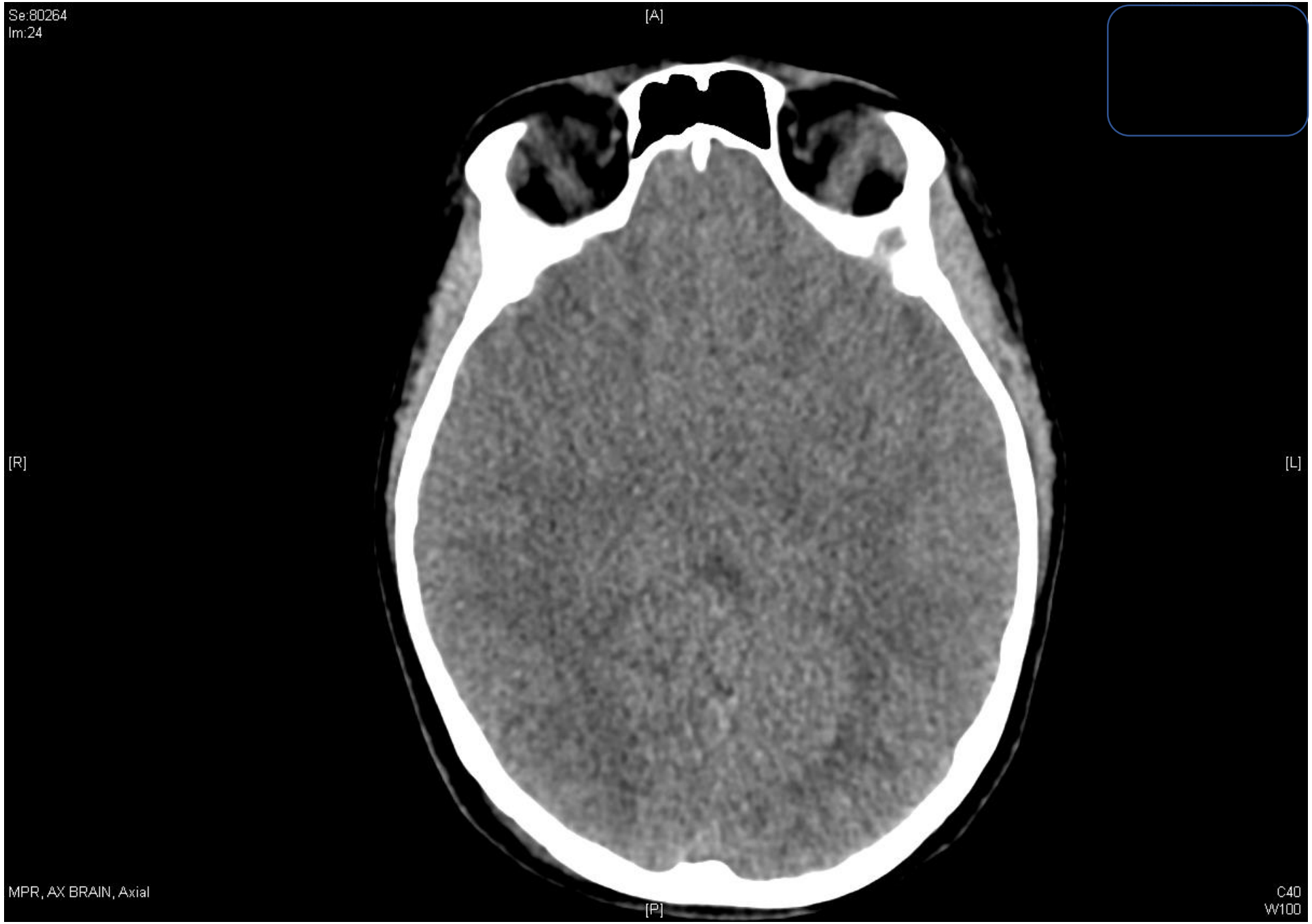
Gamberini at al World J Trauma Surgery 2017

Severe Traumatic Brain Injury

- Major health- care problem worldwide
- Progress in understanding pathophysiology
- Not translated to improved outcomes

Rosenfeld et al Early Management of Severe Traumatic Brain injury. Lancet Clinical Series in Trauma Surgery Sept 2012

35 yr female severe TBI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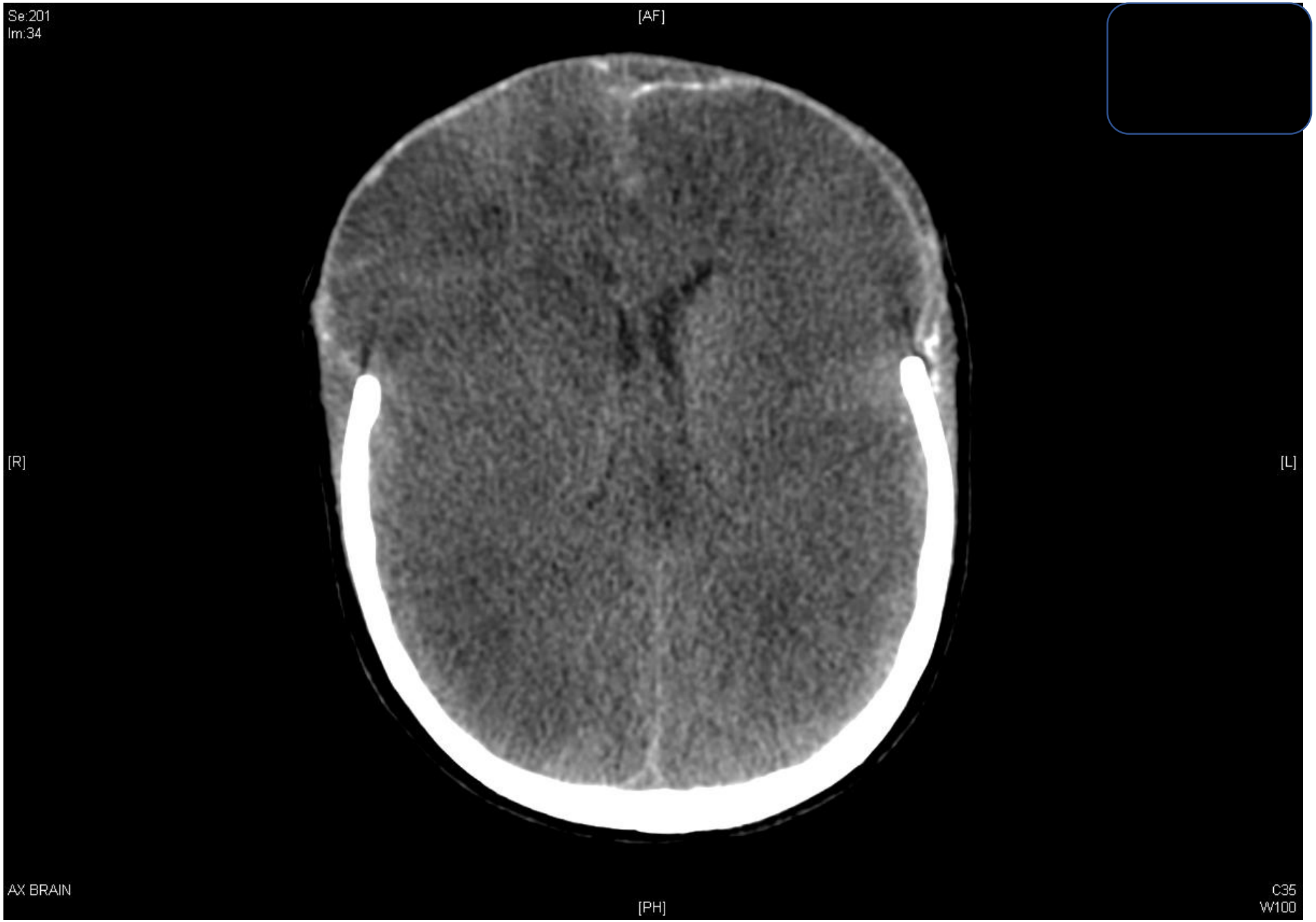
Post bi-frontotemporoparietal decompressive craniectomy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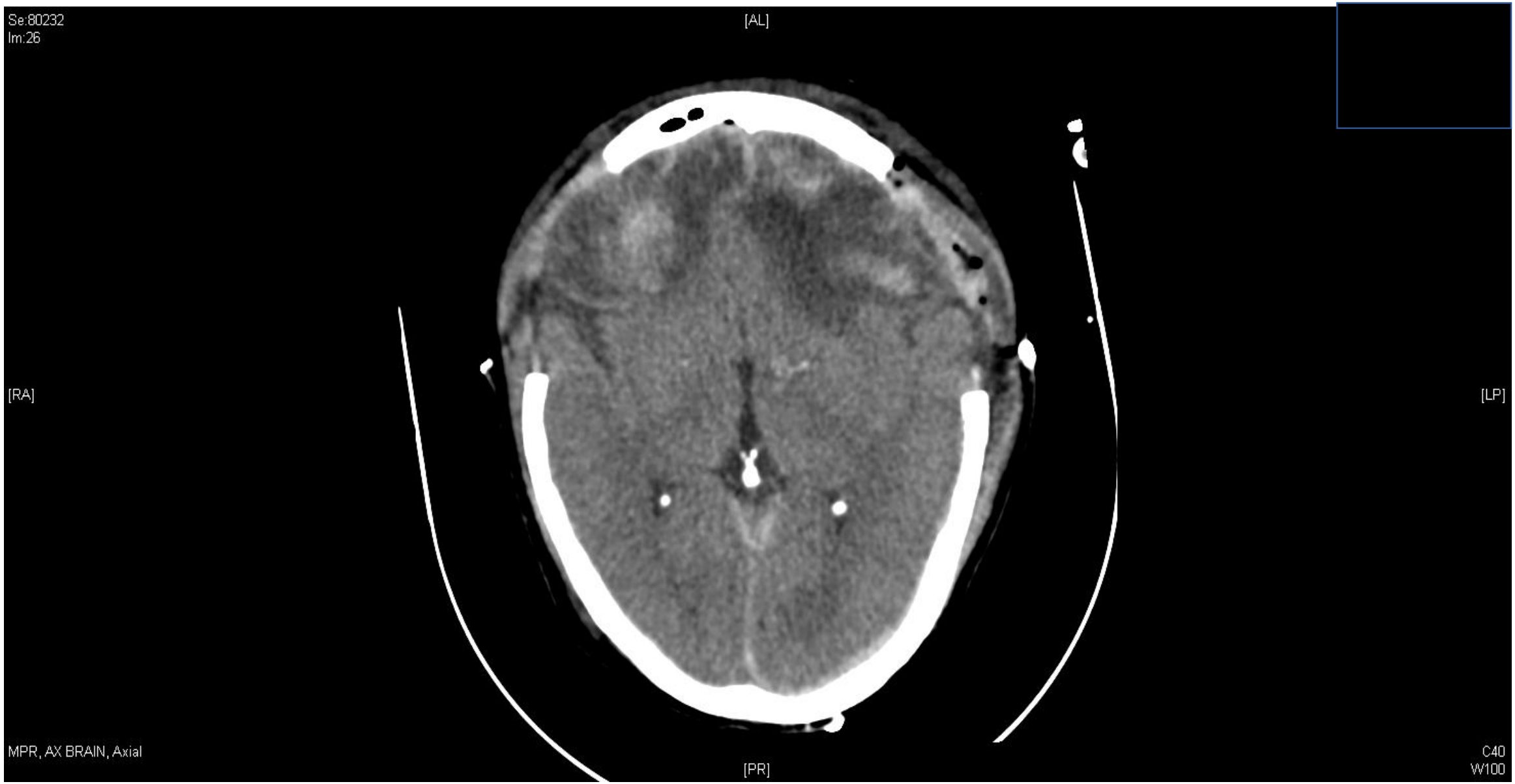
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The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 APRIL 21, 2011 VOL. 364 NO. 16

Decompressive Craniectomy in Diffuse Traumatic Brain Injury

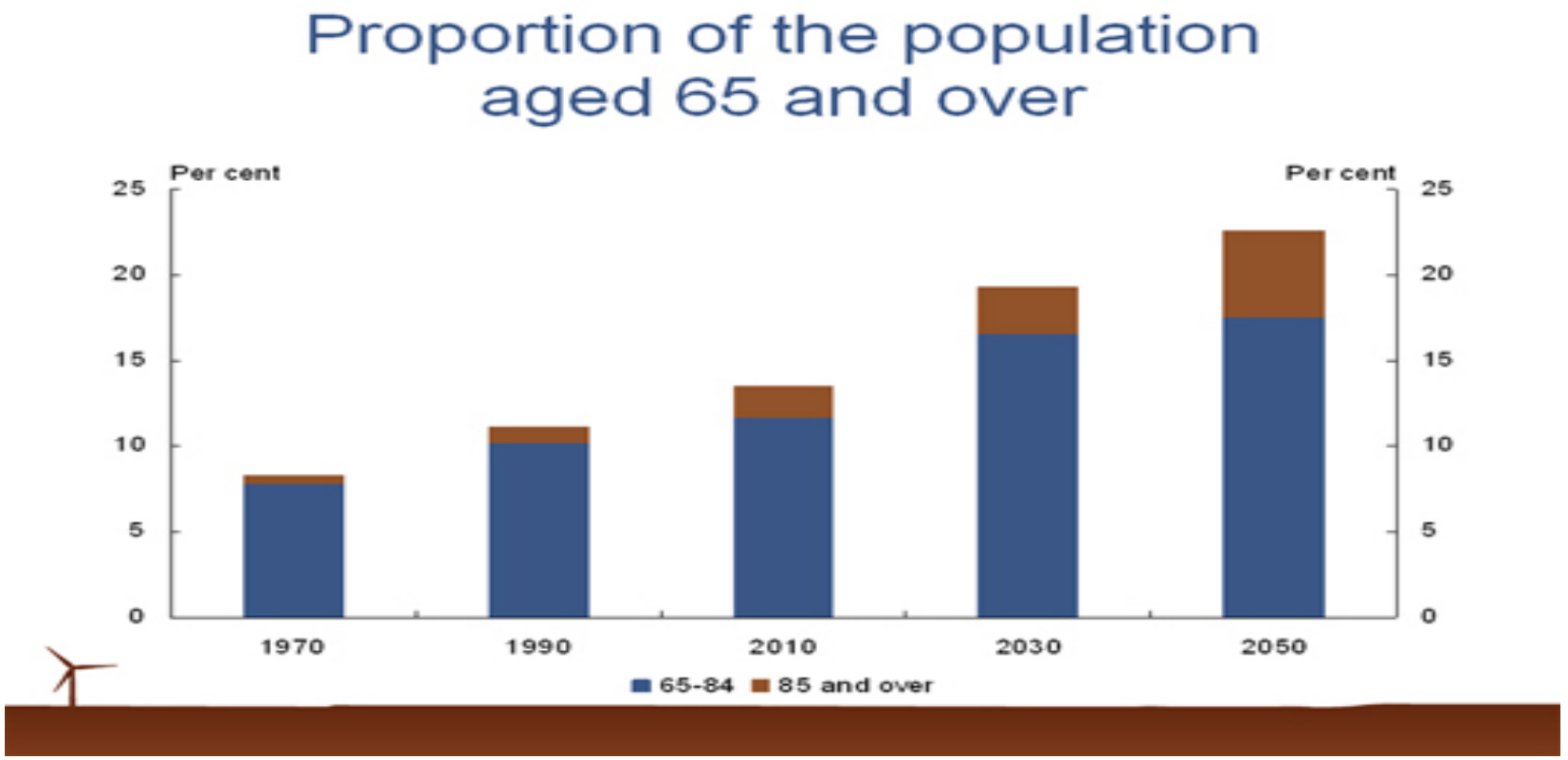
D. James Cooper, M.D., Jeffrey V. Rosenfeld, M.D., Lynnette Murray, B.App.Sci., Yaseen M. Arabi, M.D., Andrew R. Davies, M.B., B.S., Paul D'Urso, Ph.D., Thomas Kossmann, M.D., Jennie Ponsford, Ph.D., Ian Seppelt, M.B., B.S., Peter Reilly, M.D., and Rory Wolfe, Ph.D., for the DECRA Trial Investigators and the Australian and New Zealand Intensive Care Society Clinical Trials Group*

DECRA Conclusions

- In adults with severe diffuse traumatic brain injury and refractory intracranial hypertension, early bi-frontotemporoparietal decompressive craniectomy decreased intracranial pressure and the length of stay in the ICU but was associated with **more unfavourable outcomes**.
- Worse scores on Extended Glasgow Outcome scale than standard care (OR 1.84) and greater risk of unfavourable outcome (OR 2.21)
- Death rates similar at 6 months for surgery v standard care

Silver Tsunami of Elderly Trauma

Proportion of the Australian population aged 65 and over from 1970 – future prediction



Falls

Impaired driving ability

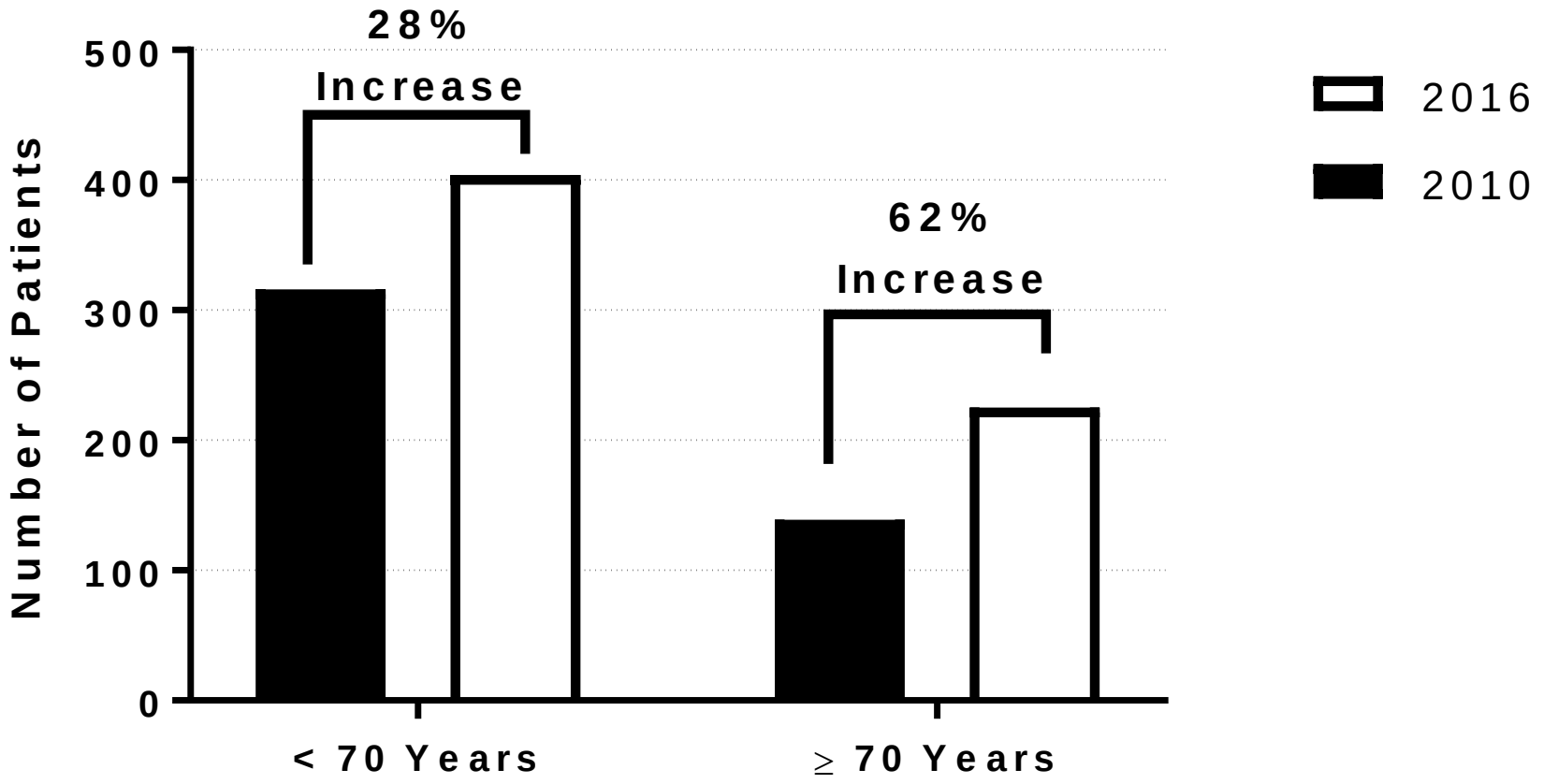
- **Runaway driver, 93, who caused Melbourne West Gate Bridge crash to be charged**

- **Debate continues to rage over age-limits on elderly drivers, following a shocking crash caused by a 93-year-old on a NSW highway.**

Alarming epidemic of major injuries… fuelled by Britain’s sprightly pensioners

Risk taking behaviour

Royal North Shore Hospital Trauma Data



NSW Institute of Trauma and Injury Management. Major Trauma in NSW 2015. Sydney: NSW Agency for Clinical Innovation, 2016

ROYAL NORTH SHORE HOSPITAL TRAUMA SERVICE

NSW GOVERNMENT Health Northern Sydney Local Health District

Australian Trauma Registry
Annual report 2016-17

- Total injuries ISS > 12 or died: 8423
- Total deaths: 897 (10%)

- Deaths due to injury in elderly
  - > 75 yrs : 40 %

Aging and Frailty

- **The estimated average prevalence of frailty among older people in the community is 10%**
  - **Range of 4–59% due to variability in definition used and studied population**

Collard *et al.*, 2012.

“Frailty” Definition

- **A state of vulnerability to minor homeostatic stressors due to an age-related decline in physiological reserve**
- **Frail people are at greater risk of adverse outcomes**
  - **Falls, increasing disability, hospitalisation, transfer to higher level of care and increased mortality**
- **Considerable heterogeneity**

Freid et al J Gerentology 2001
Joyce at al Current Opin Anaesth 2015

Frail patients

- Increased LOS
- Increased adverse events
- Increased costs

- Frailty Assessment allows :
  - Better prediction of potential outcome
  - Informed communication with family
  - Better allocation of resources

Frailty Assessment : Reported Edmonton Frail Scale

Hilmer Aust J Aging 2009

Cognition
General Health Status
Functional independence
Social support
Medication use
Nutrition
Mood
Continence
Self reported performance

- **Non frail: 0-7**
- **Frail: 8-18**

If we can make an early assessment of Frailty

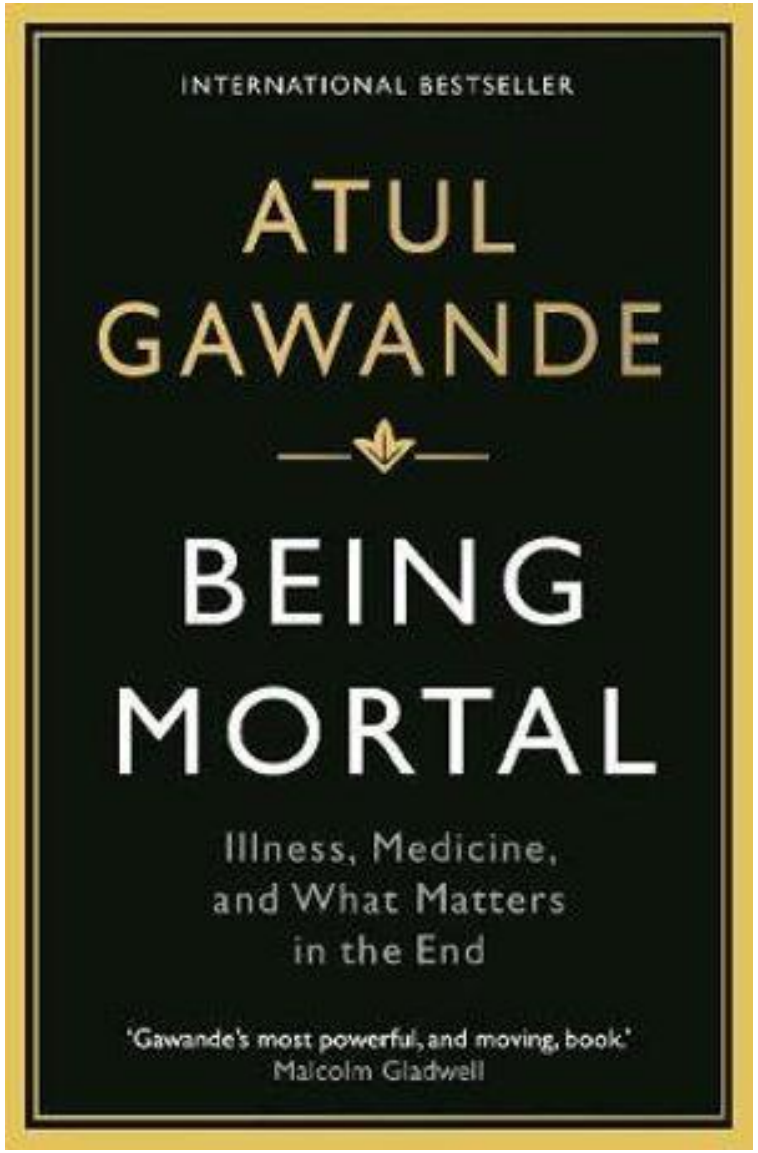
We can make a reasonable prediction of outcome after serious injury

Should be a part of the early assessment of elderly injured patients

“ The necessity of nature’s final victory was expected and accepted in generations before our own.

Doctors were far more willing to recognise the signs of defeat and far less arrogant about denying them.”

Sherwin Nuland “ How we die”



Thank you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