Critical care in the country

Dr Dan Ellis

FCEM, FACEM, FFICM, FRCS, MRCP, MRCA, EDIC, FIMC, DipRTM, DMCC

Consultant in Emergency, Critical Care and Pre-Hospital & Retrieval Medicine

- “the aggregation of marginal gains”
- “attention to detail”

- A - Airways, ABG, Access
- B - Ventilation, ventilators, NIV
- C - Inotropes, fluids

TRAUMA!

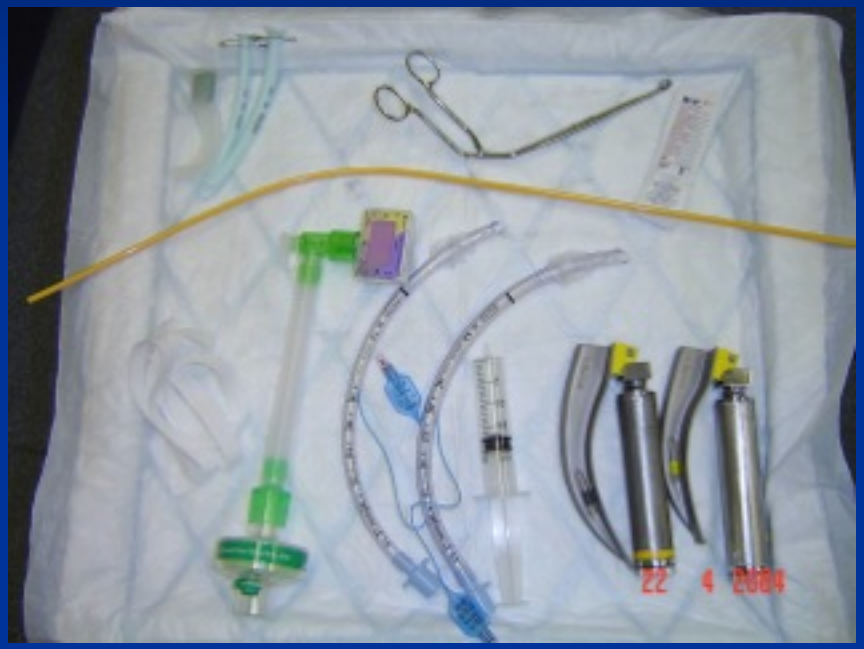
- D - Drugs (bleeding,) neuroprotection
- Others - Sepsis, monitoring, transfer, end of life

Airways

- TT
  - RSI - Weingart
  - Apnoeic oxygenation
- LMA
- BVM – just do it right….

Airway – 3 stage RSI

1. Patient selection
2. The procedure:
   - Location
   - Team – you, assistant, drugs, CP. (+/- C-spine)
   - Kit & kit dump – EtCO2, sux, roc
   - Failed airway drill – OPA, NPA, LMA, Mcoy, cric
   - Talk through then the procedure
3. Post intubation critical care management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Techniques and Procedures

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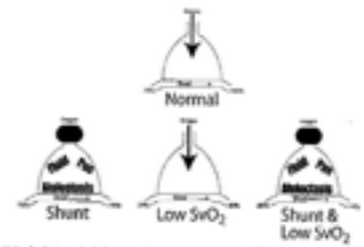
PREOXYGENATION, REOXYGENATION, AND DELAYED SEQUENCE INTUBATION IN THE EMERGENCY DEPARTMENT

INTRODUCTION

DISCUSSION



Cricoid Pressure in Emergency Department Rapid Sequence Tracheal Intubations: A Risk-Benefit Analysis

Daniel Y. Ellis, FCEM
Tim Harris, FACEM
David Zideman, FRCA

From the Department of Emergency Medicine, The Townsville Hospital, Douglas, Queensland, Australia (Ellis); the Department of Emergency Medicine and Pre-hospital Care (London Helicopter Emergency Medical Service), Royal London Hospital, London, UK (Harris); and the Department of Anaesthesia, Hammersmith Hospital, London, UK (Zideman).

Cricoid pressure is considered an integral part of patient safety in rapid sequence tracheal intubation and emergency airway management. Cricoid pressure is applied to prevent the regurgitation of gastric contents into the pharynx and subsequent aspiration into the pulmonary tree. This review analyzes the published evidence supporting cricoid pressure, along with potential problems, including increased difficulty with tracheal intubation and ventilation. According to the evidence available, the universal and continuous application of cricoid pressure during emergency airway management is questioned. An awareness of the benefits and potential problems with technique allows the practitioner to better judge when cricoid pressure should be used and instances in which it should be removed. [Ann Emerg Med. 2007;50:653-665.]

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INTRODUCTION

Cricoid pressure was described by Sellick[1] in 1961 as a method to reduce the risk of aspiration during the induction phase of anesthesia. Sellick's[1] technique was to apply backwards pressure to the cricoid cartilage, compressing the esophagus against the underlying vertebral body. Theoretically, this would occlude the esophageal lumen, preventing the passage of regurgitated gastric contents into the pharynx and subsequent aspiration into the pulmonary tree. The cricoid cartilage is a complete ring, with a larger posterior than anterior surface. Sellick[1] tested his theory on a cadaver and then on human subjects. Sellick's publications will be discussed in more detail below.

Death from aspiration during anesthesia was first described by Simpson[2] in 1848. In 1946, Mendleson[3] identified acid aspiration in 66 of 44,016 obstetric patients, all of whom underwent facemask anesthesia for labor and delivery. The Confidential Enquiry into Maternal Deaths in England and Wales in the 1950s and 1960s noted aspiration as a major cause of maternal morbidity and mortality.[4]

After Sellick's[1] article, cricoid pressure was incorporated into an overall approach to minimizing the risk of aspiration through "rapid sequence induction" of anesthesia.[5] Unlike traditional anesthesia practice, in this technique there is no interspersed ventilation (and delay) between the induction agent and the muscle relaxant. The goal is to minimize gastric insufflation and place a cuffed endotracheal tube as quickly as possible. After preoxygenation, the induction agent and muscle relaxant were given in rapid sequence, cricoid pressure was applied, and positive-pressure ventilation was withheld until the endotracheal tube was placed.[6] Indications for rapid sequence induction have since been expanded from the obstetric patients to include all anesthesia patients considered at high risk of aspiration, particularly patients believed to have a full stomach.[7,8]

"Rapid sequence induction" was adapted by emergency physicians to allow ventilation as required to prevent hypoxia and subsequently termed *rapid sequence tracheal intubation* (rapid sequence tracheal intubation will refer to this technique in this article).[9,10] Rapid sequence tracheal intubation is the now most widely used technique for tracheal intubation in the emergency department (ED),[11,12] and cricoid pressure is taught as a standard component of emergency airway management.[13,14]

In modern anesthesia practice, although cricoid pressure is widely used, its method of application, its timing, and its role in difficult airways are not standardized.[15-20] Cricoid pressure has been described as the "lynchpin of physical prevention [of aspiration]"[21] and a minimum standard of care, implying any trials to prove its worth could be unethical.[22] Conversely, more recent reviews and case reports have questioned the effectiveness and safety of the technique, even in obstetric anesthesia.[22-27] Questions have arisen about whether cricoid pressure should be abandoned altogether,[22-25] and some anesthetists have, in their own words, "more or less discontinued the application of cricoid pressure."[26] Doubt has also been cast on the efficacy of cricoid pressure from within emergency medicine practice.[28]

Induction agents

- Propofol & thio
  - Beware the BP…
- Ketamine
  - SNS stimulation v direct suppressive effects
  - Caution if SNS reserves depleted…
- Fentanyl?
- Midazolam?

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Evidence-Based Medicine

ROCURONIUM VS. SUCCINYLCHOLINE IN THE EMERGENCY DEPARTMENT: A CRITICAL APPRAISAL

William K. Mallon, MD,* Samuel M. Keim, MD, MS,† Jan M. Shoenberger, MD,* and Ron M. Walls, MD‡

*Department of Emergency Medicine, Los Angeles County/University of Southern California Medical Center, Los Angeles, California, †Department of Emergency Medicine, The University of Arizona College of Medicine, Tucson, Arizona, and ‡Department of Emergency Medicine, Brigham and Women's Hospital, Boston, Massachusetts

Reprint Address: Samuel M. Keim, MD, MS, Department of Emergency Medicine, The University of Arizona College of Medicine, Arizona Health Sciences Center, PO Box 245057, Tucson, AZ 85724-5057

☐ **Abstract—Background: Two methods of paralysis are available for rapid sequence intubation (RSI) in the emergency department (ED): depolarizing agents such as succinylcholine, and non-depolarizing drugs such as rocuronium. Rocuronium is a useful alternative when succinylcholine is contraindicated. Contraindications to succinylcholine include allergy, history of malignant hyperthermia, denervation syndromes, and patients who are 24–48 h post burn or crush injury. Non-depolarizing drugs have the advantage of causing less pain due to post-paralysis myalgias. Clinical Question: Can rocuronium replace succinylcholine as the paralytic of choice for RSI in the ED? Evidence Review: Four relevant studies were selected from an evidence search and a structured review performed. Results: For the outcomes of clinically acceptable intubation conditions and time to onset, the two agents were not statistically significantly different. Succinylcholine seems to produce conditions that have higher satisfaction scores. Conclusion: Succinylcholine remains the drug of choice for ED RSI unless there is a contraindication to its usage. © 2009 Elsevier Inc.**

☐ **Keywords—evidence-based medicine; EBM; rapid sequence intubation; RSI; rocuronium; succinylcholine; intubation**

CASE

A 20-year-old man presents to the Emergency Department (ED) via paramedics after a rollover motor vehicle accident with associated blunt head trauma and an apparent left femur fracture. He is in a cervical collar on a backboard with oxygen by facemask. He has received 250 cc of normal saline en route. His Glasgow Coma Scale score (GCS) is 9 (Eye = 1, Verbal = 3, Motor = 4) and his left pupil is sluggish to light. The FAST (focused assessment with sonography in trauma), four views, is negative and his vital signs are: blood pressure 155/100 mm Hg, heart rate 122 beats/min, respiratory rate 16 breaths/min, and temperature 36.6°C (98.0°F). Airway evaluation suggests no apparent problems with intubation visualization except for low neck mobility secondary to the presence of a cervical collar. The decision is made to perform rapid sequence intubation (RSI) before immediate diagnostic imaging and neurosurgical consultation.

CLINICAL QUESTION

Can rocuronium replace succinylcholine as the paralytic agent of choice for ED RSI in patients similar to this patient?

CONTEXT

Although RSI has revolutionized ED airway management and is routinely used, clinicians continue to search

Received: 3 July 2008;
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Sux v roc

- Familiarity
- Decision:
  - Can I wake this patient up if it goes to custard?
- Real difference in intubating conditions…?

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CASE

A 29-year-old man presents to the Emergency Department (ED) via paramedics after a rollover motor vehicle accident with associated blunt head trauma and an apparent left femur fracture. He is in a cervical collar on a backboard with oxygen by facemask. He has received 250 cc of normal saline en route. His Glasgow Coma Scale score (GCS) is 9 (Eye = 1, Verbal = 3, Motor = 4) and his left pupil is sluggish to light. The FAST (focused assessment with sonography in trauma), four views, is negative and his vital signs are: blood pressure 155/100 mm Hg, heart rate 122 beats/min, respiratory rate 16 breaths/min, and temperature 36.6°C (98.0°F). Airway evaluation suggests no apparent problems with intubation visualization except for low neck mobility secondary to the presence of a cervical collar. The decision is made to perform rapid sequence intubation (RSI) before immediate diagnostic imaging and neurosurgical consultation.

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Received 3 July 2008;
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NM blocker

- Suxamethonium
  - Onset 45 secs, offset 3-6 mins, fasciculation
  - Give proper dose (2mg/kg), wait, no 2nd dose
  - K+, IOP, burns, sux apnoea, anaphylaxis
- Rocuronium
  - 0.6 mg/kg, onset 1 min, offset up to 20mins
  - Much less anaphylaxis, antagonist…?

Gases

- ABG or VBG
  - A good screen – better than most other tests….
  - Lactate

Ventilation I

- Low tidal volume
  - Reducing incidence of ARDS*
  - Pay attention when ventilating with bag
  - 'normoventilation'
  - No point in low Tv ventilation in ICU if damage already done in ED
- PEEP

*N Engl J Med 2000; 342: 1301–8

Ventilation II

- NIV (CPAP, BiPAP)
  - COPD – 1st line treatment!
  - APO
  - ARF – more caution; don't delay IPPV
  - Asthma – caution….
  - Lobar collapse
  - Nasal, mask, face, helmet
  - 'Trial' of NIV means re-evaluate after half an hour or so...

Access

- CVP lines
  - Who needs one and which vein? (eg femoral)
  - US v blind
  - Secured in place
  - Swann sheaths, angiocaths
  - EZIO
- Arterial lines
  - Gases and inotropes

Inotropes

- Usual suspects
  - adrenalin
  - noradrenalin

Vasopressors/inotropes

- Which vasopressor/inotrope?
- Answer – unknown….

Annane D. Norepinephrine plus dobutamine versus epinephrine alone for management of septic shock: a randomised trial. Lancet. 2007 Aug 25;370(9588):676-84.

Myburgh JA. An appraisal of selection and use of catecholamines in septic shock – old becomes new again. Crit Care Resusc. 2006 Dec;8(4):353-60. Review.

Beale R. Vasopressor and inotropic support in septic shock: an evidence-based review. Crit Care Med. 2004 Nov;32(11 Suppl):S455-65.

M Müllner Vasopressors for shock *Cochrane Database of Systematic Reviews* **2004**

Fluids

- Colloids v crystalloids
  - SAFE study – no difference but crystalloid cheaper
  - Possible benefit of albumin in sepsis
- FEAST
- VISEP
- CRISTAL
- Bottom line?
  - Balanced crystalloids
  - Not a lot

FAST

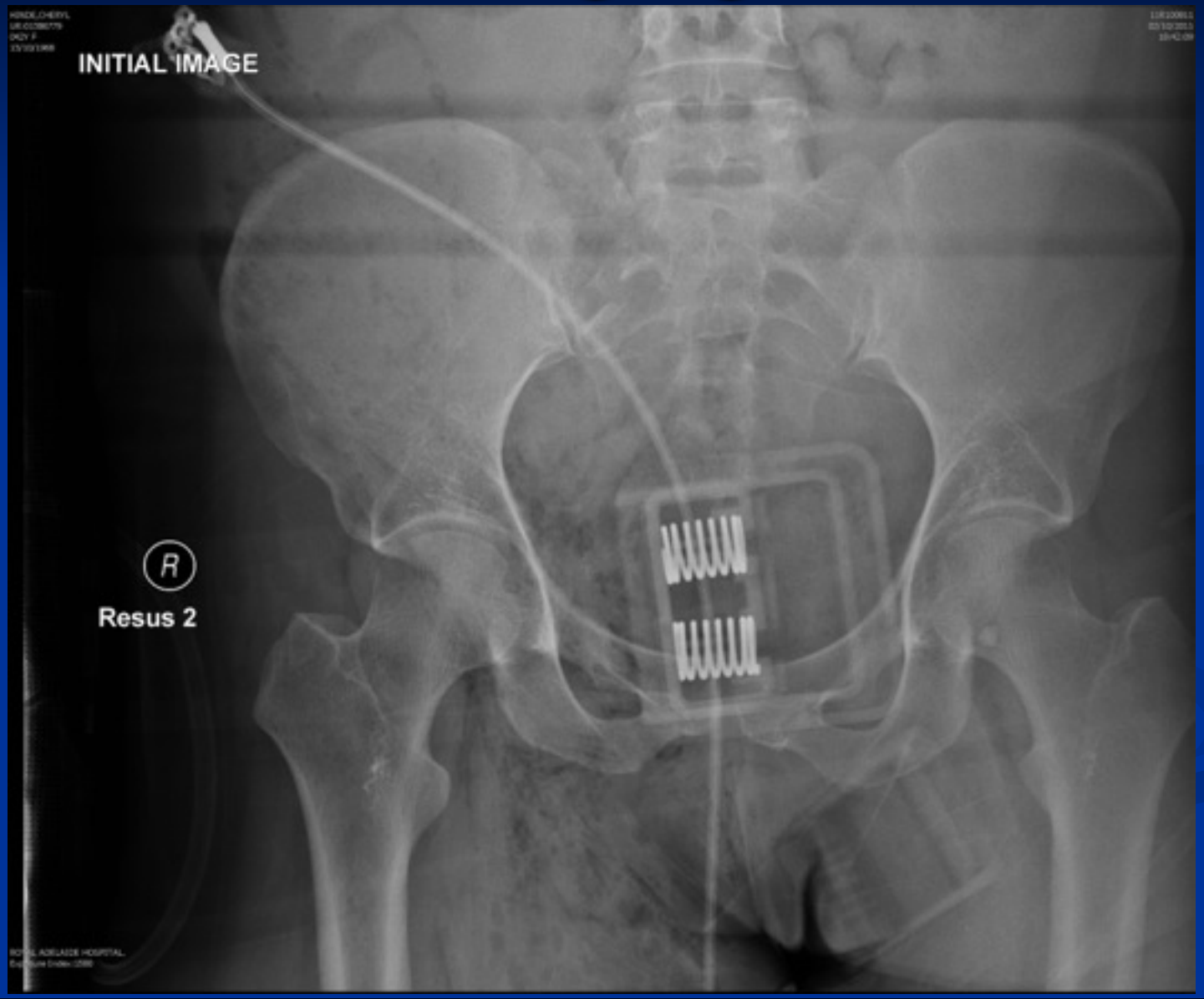
- Should be ready
  - Not every ED doctor is accredited or skilled…..
- Can look also at lungs
  - Effusion
  - Pneumothorax
- & the heart
  - Tamponade
  - function

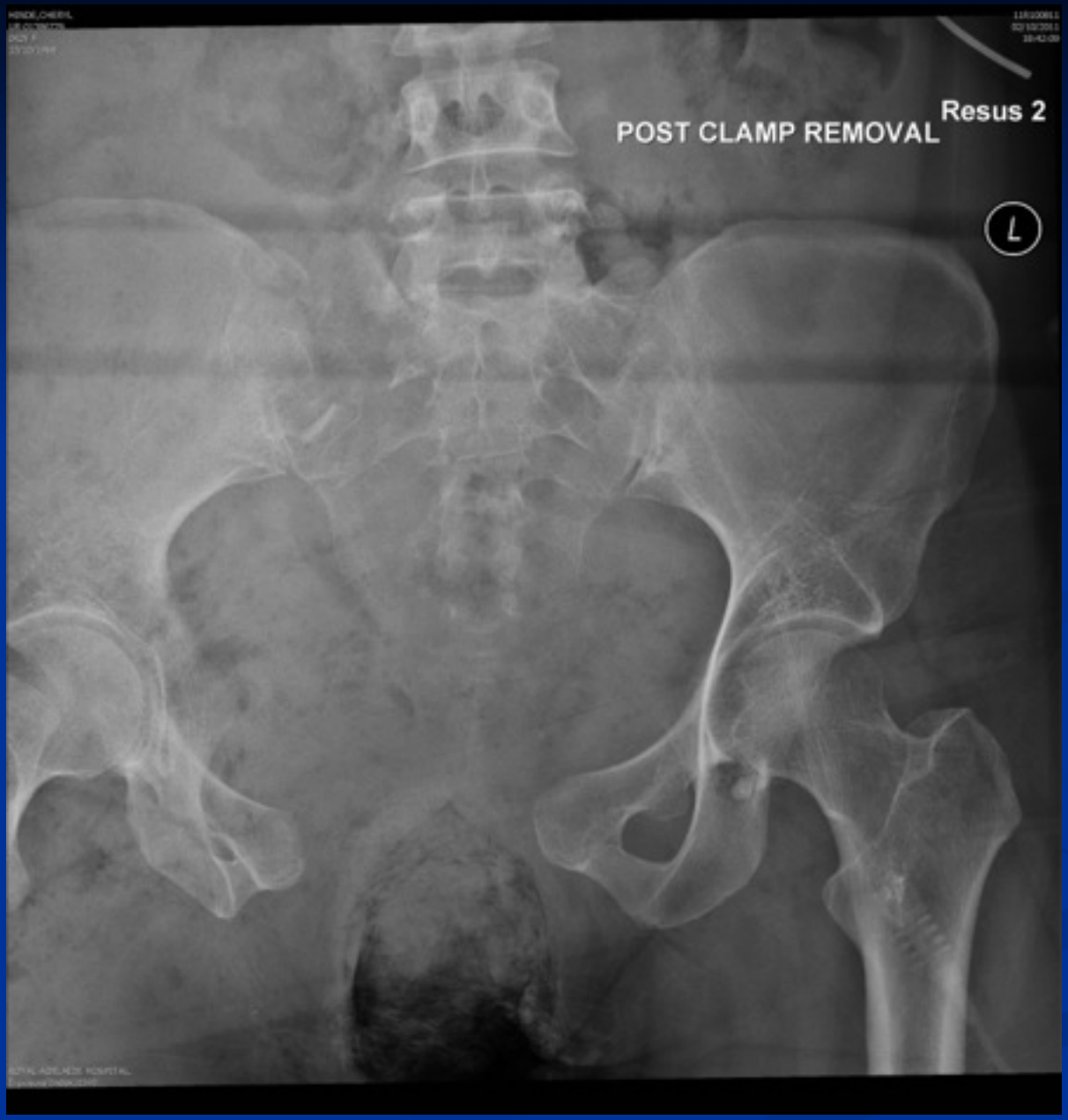
Exposure

- Log roll
- Keep warm
  - A team responsibility
- Indotherm on the bottom
- Bair hugger on the top
- CT is very cold…..

- TIME is of the essence….

Packaging

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POST CLAMP REMOVAL
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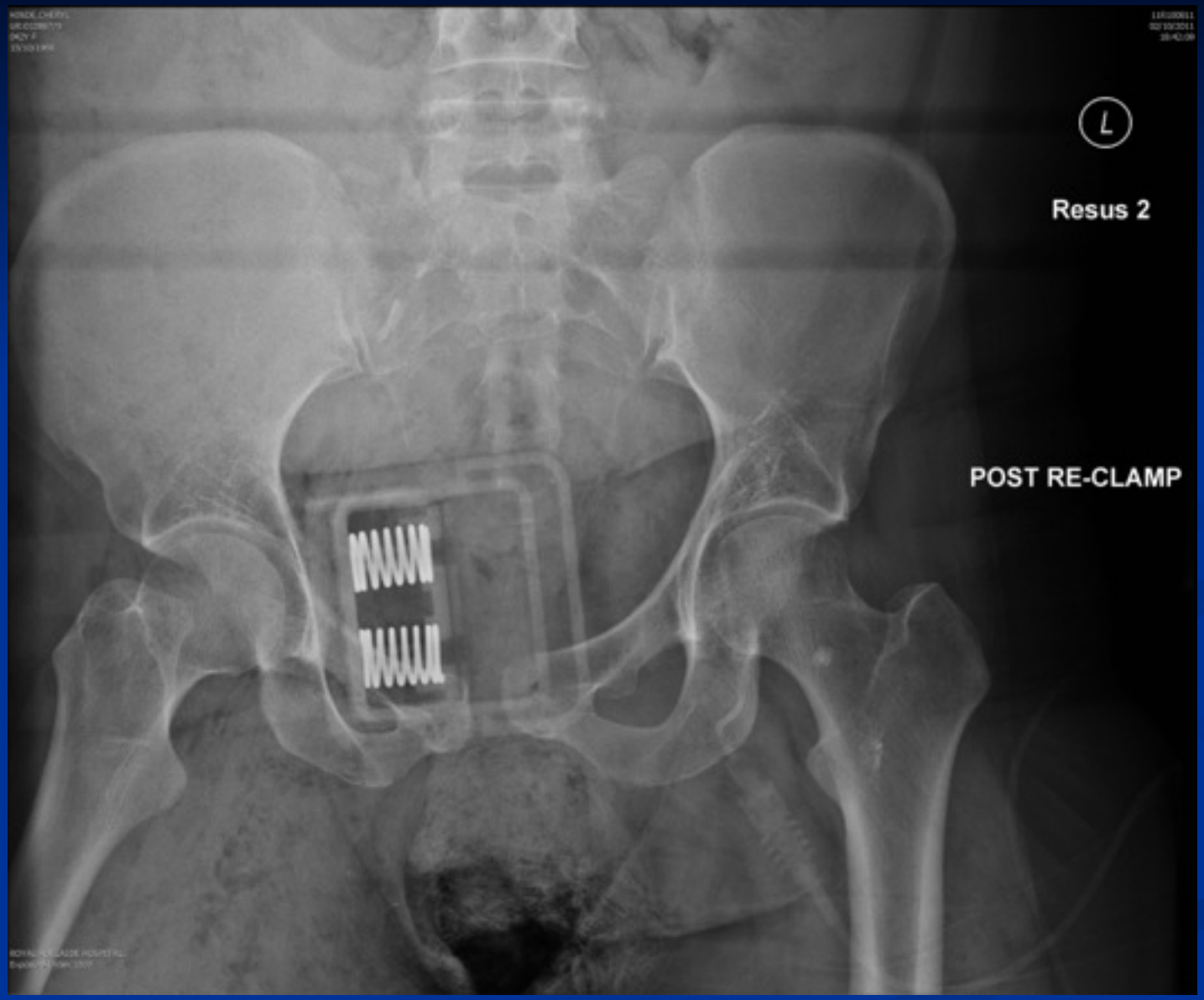
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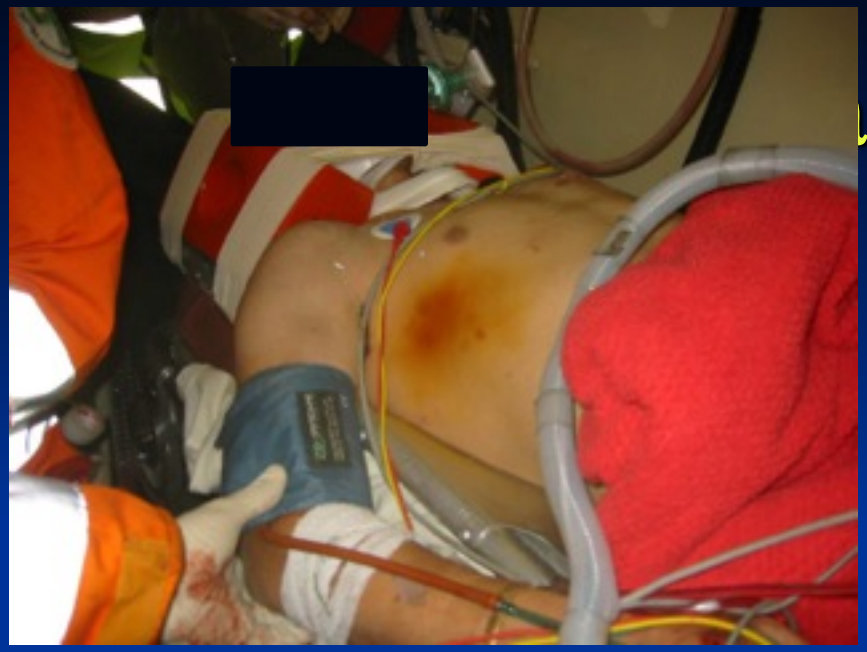
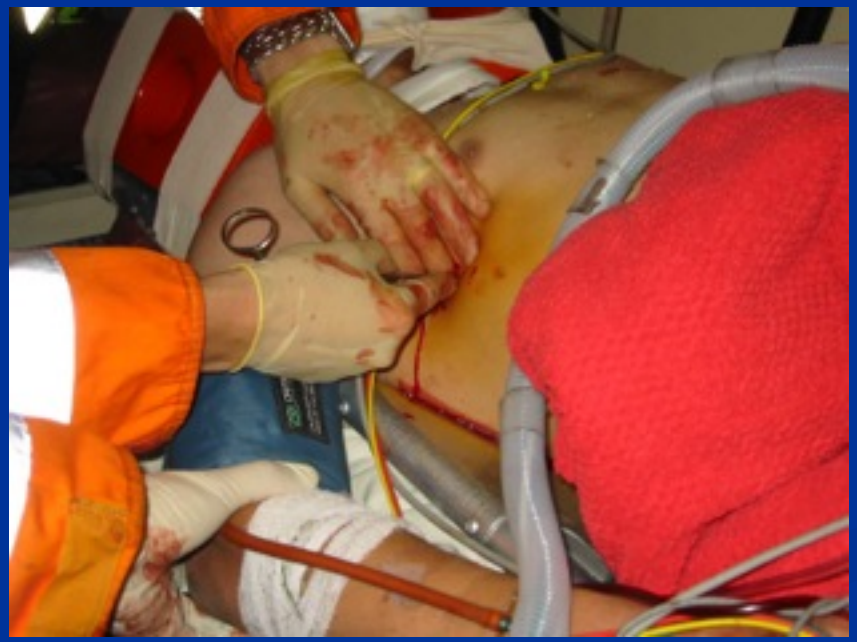
Resus 2
POST RE-CLAMP

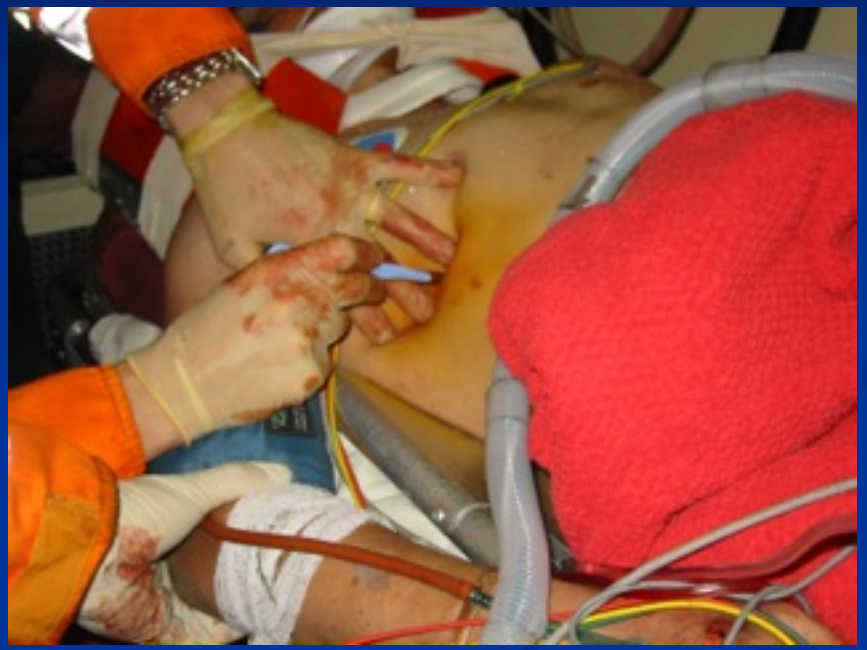
Thoracostomy

- Tube thoracostomy is time consuming
- Needle decompression is a 'one off' procedure which may not work in up to 50%
  - Stevens RL et al. Prehosp Emerg Care 2009 13(1);14-7
  - Zengerink I et al. J Trauma 2008 64(1);111-4
- Thoracostomy is a chest tube without the tube ie initial part of procedure is the same.
- Second stage (ie tube insertion) happens in ED

acostomy







- Thoracostomy allows pressure release before deleterious effects of tension become evident
- Keep thoracostomy patent by ‘re-fingering’
- Formal chest tube can occur in ED

Thoracotomy

- Ideally in theatre
- Penetrating v blunt
- Surgeon v non surgeon
- (needles don't work!)

ED/PH Thoracotomy

- Indication

  Penetrating chest / epigastric trauma associated with cardiac arrest

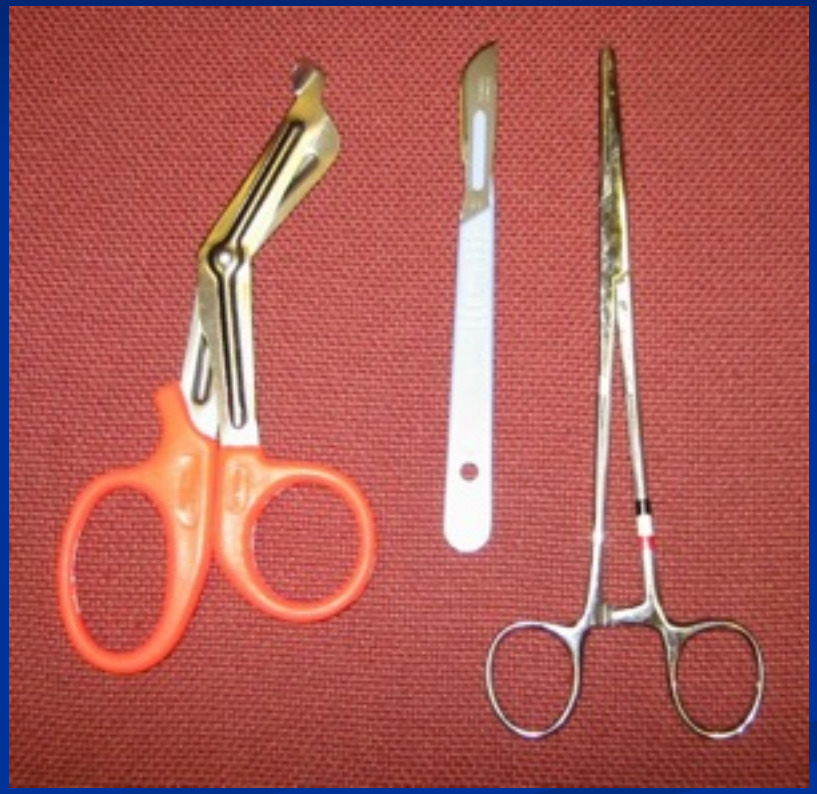
- Key treatable cause:

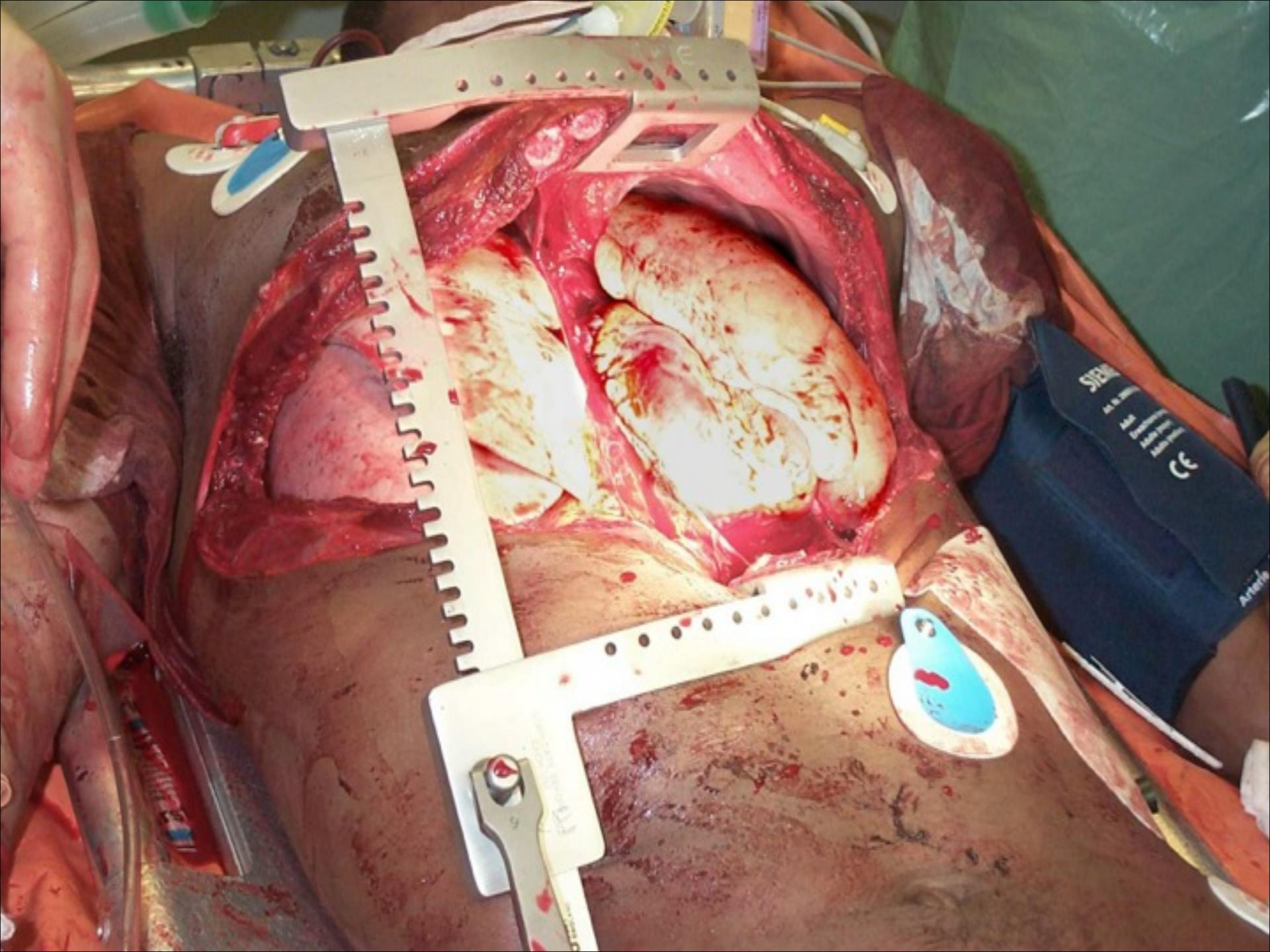
  Cardiac tamponade

- Decision <15seconds

Thoracotomy – the procedure

- Personnel
- Equipment
- Technique





DCR

- Damage control resuscitation
  - Permissive hypotension
  - Haemostatic resuscitation – stop the bleeding
    - Direct pressure
    - Tamponade
- Novel hybrid resuscitation

Hypotensive Resuscitation Strategy Reduces Transfusion Requirements and Severe Postoperative Coagulopathy in Trauma Patients With Hemorrhagic Shock: Preliminary Results of a Randomized Controlled Trial

C. Anne Morrison, MD, MPH, Matthew M. Carrick, MD, Michael A. Norman, MD, Bradford G. Scott, MD, Francis J. Welsh, MD, Peter Tsai, MD, Kathleen R. Liscum, MD, Matthew J. Wall, Jr., MD, and Kenneth L. Mattox, MD

Background: Trauma is a leading cause of death worldwide and is thus a major public health concern. Previous studies have shown that limiting the amount of fluids given by following a strategy of permissive hypotension during the initial resuscitation period may improve trauma outcomes. This study examines the clinical outcomes from the first 90 patients enrolled in a prospective, randomized controlled trial of hypotensive resuscitation, with the primary aim of assessing the effects of a limited transfusion and intravenous (IV) fluid strategy on 30-day morbidity and mortality.

Methods: Patients in hemorrhagic shock who required emergent surgery were randomized to one of the two arms of the study for intraoperative resuscitation. Those in the experimental (low mean arterial pressure [LMAP]) arm were managed with a hypotensive resuscitation strategy in which the target mean arterial pressure (MAP) was 50 mm Hg. Those in the control (high MAP [HMAP]) arm were managed with standard fluid resuscitation to a target MAP of 65 mm Hg. Patients were followed up for 30 days. Intraoperative fluid requirements, mortality, postoperative complications, and other clinical data were prospectively gathered and analyzed.

Results: Patients in the LMAP group received a significantly less blood products and total IV fluids during intraoperative resuscitation than those in the HMAP group. They had significantly lower mortality in the early postoperative period and a nonsignificant trend for lower mortality at 30 days. Patients in the LMAP group were significantly less likely to develop immediate postoperative coagulopathy and less likely to die from postoperatively bleeding associated with coagulopathy. Among those who developed coagulopathy in both groups, patients in the LMAP group had significantly lower international normalized ratio than those in the HMAP group, indicating a less severe coagulopathy.

Conclusions: Hypotensive resuscitation is a safe strategy for use in the trauma population and results in a significant reduction in blood product transfusions and overall IV fluid administration. Specifically, resuscitating patients with the intent of maintaining a target minimum MAP of 50 mm Hg, rather than 65 mm Hg, significantly decreases postoperative coagulopathy and lowers the risk of early postoperative death and coagulopathy. These preliminary results provide convincing evidence that support the continued investigation and use of hypotensive resuscitation in the trauma setting.

Key Words: Trauma, Hypotensive resuscitation, Coagulopathy.

(*J Trauma*. 2011;70: 652–663)

Trauma has long been a major cause of preventable deaths in the United States and worldwide. Approximately one-third of trauma deaths occur because the victims bleed to death within the first several hours after their injury.[1] For the past 50 years, high-volume fluid resuscitation strategies have typically been used by surgeons and emergency medical personnel in an attempt to reverse hemorrhagic shock by replacing lost blood with intravenous (IV) fluids or transfusions.[2] Although these strategies are currently considered to be the gold standard of care for trauma patients, they have considerable limitations[3–5] and, unlike many other treatment modalities, have not been extensively tested in prospective, randomized clinical trials.

Hypotensive resuscitation has recently been advocated as an alternative to the current standard of care. In contrast to standard fluid resuscitation, this strategy uses less fluids and blood products during the early stages of treatment for hemorrhagic shock. Although intraoperative hypotensive resuscitation has not been prospectively tested in a surgical trauma setting, it has been successfully used in animal models[6] and has also been safely used in patients undergoing elective operations.[7–11] To our knowledge, this study is the first randomized, prospective study of intraoperative hypotensive resuscitation for trauma in human subjects. The aim of this study is to assess patient outcomes after accrual of 90 patients to establish the safety of a hypotensive resuscitation strategy including its effects on intraoperative fluid administration, bleeding, postoperative complications, and mortality within the trauma population. Our hypothesis is that patients randomized to a hypotensive resuscitation strategy will have decreased transfusion and intraoperative fluid utilization, decreased transfusions, decreased operative blood loss, decreased postoperative coagulopathy, comparable postoperative morbidity, and decreased mortality compared with patients randomized to a standard fluid resuscitation strategy.

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Address for reprints: C. Anne Morrison, MD, MPH, Michael E. DeBakey, Department of Surgery, Baylor College of Medicine, Houston, TX; email: camorris@bcm.edu.

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- Changes to "significant" low BP

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Resuscitation

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Clinical paper

Systolic blood pressure below 110 mmHg is associated with increased mortality in blunt major trauma patients: Multicentre cohort study☆

Rebecca M. Hasler[a,*], Eveline Nuesch[c,d], Peter Jüni[c,d], Omar Bouamra[a], Aristomenis K. Exadaktylos[b], Fiona Lecky[a]

[a] Trauma Audit and Research Network (TARN), Health Sciences Research Group, School of Community Based Medicine, Manchester Academic Health Sciences Centre, University of Manchester, Salford Royal Hospital, Stott Lane, Salford M6 8HD, UK
[b] Department of Emergency Medicine, University Hospital Bern, Freiburgstr, 3010 Bern, Switzerland
[c] Institute of Social and Preventive Medicine (ISPM), University of Bern, Finkenhubelweg 11, 3012 Bern, Switzerland
[d] Clinical Trials Unit (CTU) Bern, University Hospital Bern, Finkenhubelweg 11, 3012 Bern, Switzerland

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Systolic blood pressure
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ABSTRACT

Introduction: Non-invasive systolic blood pressure (SBP) measurement is often used in triaging trauma patients. Traditionally, SBP< 90 mmHg has represented the threshold for hypotension, but recent studies have suggested redefining hypotension as SBP< 110 mmHg. This study aims to examine the association of SBP with mortality in blunt trauma patients.

Methods: This is an analysis of prospectively recorded data from adult (≥16 years) blunt trauma patients. Included patients presented to hospitals belonging to the Trauma Audit and Research Network (TARN) between 2000 and 2009. The primary outcome was the association of SBP and mortality rates at 30 days. Multivariate logistic regression models were used to adjust for the influence of age, gender, Injury Severity Score (ISS) and Glasgow Coma Score (GCS) on mortality.

Results: 47,927 eligible patients presented to TARN hospitals during the study period. Sample demographics were: median age: 51.1 years (IQR = 32.8–67.4); male 68% (n = 28,094); median ISS 9 (IQR = 8–10); median GCS 15 (IQR = 15–15); and median SBP 135 mmHg (IQR = 120–152). We identified SBP< 110 mmHg as a cut off for hypotension, where a significant increase in mortality was observed. Mortality rates doubled at <100 mmHg, tripled at <90 mmHg and were 5- to 6-fold at <70 mmHg, irrespective of age.

Conclusion: We recommend triaging adult blunt trauma patients with a SBP< 110 mmHg to resuscitation areas within dedicated trauma units for close monitoring and appropriate management.

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1. Introduction

Trauma accounts for 10% of death and 14% of years of life lost worldwide and is the leading cause of death of people aged between 5 and 44 years in developed countries.[1,2]

Uncontrolled haemorrhage following traumatic injury accounts for 30–40% of trauma deaths,[3] and about 25% of patients with multiple trauma and haemorrhage suffer from coagulopathy, acidosis, and hypovolaemic shock.[4,5] In addition, excessive red blood cell transfusion is associated with a dose–response development of multiple organ failure,[6] a threefold increased intensive care unit admission rate and mortality.[7] Non-invasive systolic blood pressure (SBP) measurement is quick and easy to obtain and often used as a triage tool in the pre-hospital and hospital sector to identify patients with major bleeding.[8] Hypotensive trauma patients are usually referred to dedicated trauma centres to undergo specific care with maximum support and continued attention provided in dedicated resuscitation areas, whereas normotensive trauma patients might be considered less injured and therefore less observation and care might be provided. SBP levels are part of different guidelines in routine trauma care worldwide.[9,10] The American College of Surgeons Committee on Trauma recommends triage of patients with a SBP < 90 mmHg to dedicated trauma centres.[9] The multidisciplinary task force for advanced bleeding care in trauma also defines hypotension at a SBP of <90 mmHg[10] and an International Consensus Conference in 2007 agreed that SBP < 90 mmHg represents critical hypotension, but recommends that hypotension should not be used to define the state of shock.[11] Some authors argue that a SBP < 90 mmHg is a late sign of haemorrhage and rather an indicator for cardiovascular decompensation

☆ A Spanish translated version of the abstract of this article appears as Appendix in the final online version at doi:10.1016/j.resuscitation.2011.04.021.

* Corresponding author.
E-mail addresses: rebecca.hasler@gmail.com (R.M. Hasler), enuesch@ispm.unibe.ch (E. Nuesch), juni@ispm.unibe.ch (P. Jüni), omar.bouamra@manchester.ac.uk (O. Bouamra), aristomenis.exadaktylos@insel.ch (A.K. Exadaktylos), fiona.lecky@manchester.ac.uk (F. Lecky).

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Editorial

Blood pressure management in trauma: from feast to famine?

In 2007, the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) published a review of trauma services in England, Wales and Northern Ireland [1]. This demonstrated that almost 60% of major trauma cases received care that was judged to be less than good practice. This led to a review of major trauma care within England by the National Audit Office [2], which subsequently led to the creation of major trauma centres (MTCs). As a result of the advent of MTCs the management of trauma within the UK has changed radically over the past few years, from both organisational and clinical perspectives [3]. This has led to an upsurge in interest amongst medical professionals as to how best to manage trauma cases. The basis of much of UK trauma management over the past 30 years has been derived from teaching from the Advanced Trauma Life Support (ATLS) courses [4]. These guidelines have faced increasing criticism over the past decade, especially regarding their insensitivity to change (with updates typically occurring on a 3–4 year cycle) and applicability to UK practice [5, 6]. Despite these weaknesses, some ATLS concepts have become enshrined into trauma management and teaching, though without much supporting evidence. Examples of this include the trimodal distribution of death following trauma and the ATLS classification of shock, both of which have been shown to be theoretical concepts rather than useful clinical entities [7–10].

Further challenges to ATLS management recommendations came from the implementation of military trauma strategies. The conflicts in Iraq and Afghanistan led to the development of novel management strategies in both trauma resuscitation and surgery (discussed in detail in a recent supplement in this journal [11]), some of which have been translated back to civilian practice. One area of focus is the concept of permissive hypotension as part of a damage control resuscitation (DCR) strategy in the management of the bleeding trauma patient.

Permissive hypotension (or hypotensive resuscitation) was conceived because of the theoretical risk of excess fluid administration's interfering with the endogenous coagulation process, by inducing a dilutional coagulopathy, by clot disruption from an increase in arterial pressure, or through the abolition of reflex physiological vasoconstriction. This led to the recommendation that fluid administration should be delayed until haemorrhage has been controlled, even though this often would result in a period of suboptimal end-organ perfusion. Typical systolic blood pressure (SBP) targets in permissive hypotension are 70–90 mmHg although a recent article [12] has suggested that DCR targets should be a SBP and mean arterial pressure (*MAP*) of 80 and 50 mmHg, respectively. Over the past three years, guidelines from professional bodies (including the ambulance service) and review articles have been published, recommending the use of permissive hypotension in trauma management [13–16], but it is unclear whether there is robust evidence to support this practice, especially for the UK trauma population.

The concept of permissive hypotension was first described by Cannon et al. [17], a group of Captains in the Army Medical Corps, from their experience in the management of injuries received during the First World War. They noted that "*Injection of a fluid that will increase blood pressure has dangers in itself. If the pressure is raised before the surgeon is ready to check the bleeding that may take place, blood that is sorely needed may be lost.*" It is of note, however, that in the same series of articles, one of Cannon's co-authors, Cowell [18], recognised the problems associated with periods of prolonged hypoperfusion, stating "*...the treatment of*

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- Common sense approach
  - Eg in ED, PH etc
- Risk benefit analysis
  - Is the brain injured or not….?
- For theatre its less of an issue as the ‘tap’ should be turning off……

Drugs in haemorrhage

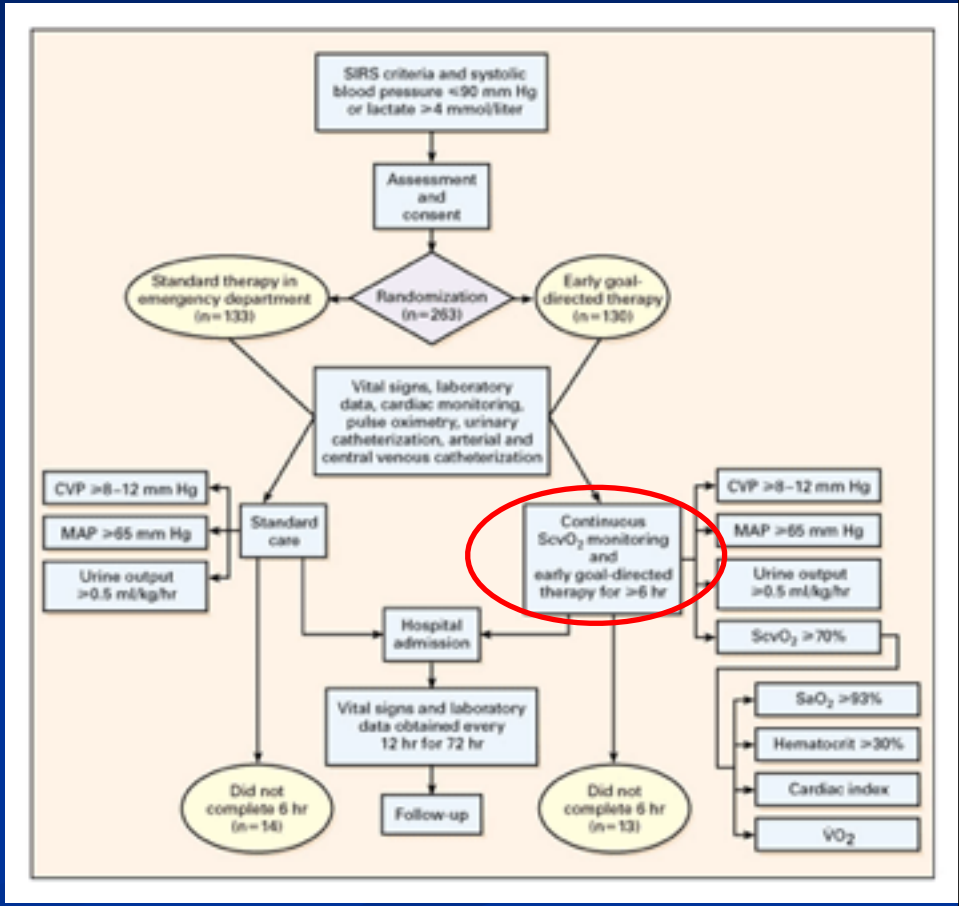
- CRASH 2
- PATCH

Neuroprotection

- Head up 30 degrees
- Tape in TT
- Avoid ↓ BP
- Avoid ↓ Sats
- Aim for $EtCO_2$ 35-40
- Keep adequatelyfilled

Sepsis

- Rivers et al
- GDT not new - **early** is the key area
- Mortality 30% v 46% (N=263)
- Entry criteria
  - Lactate > 4
  - (or Base deficit >6) Or
  - Systolic BP <90 And
  - 2 of 4 Sirs criteria



SIRS	Two or more of: •Temperature > 38°C or <36°C •Tachycardia > 90 beats/minute •Respiratory rate > 20 breaths/minute or PaCO2 <4.3 kPa •White blood count > 12 x 10^9/l or < 4 x 10^9/l or > 10% immature (band) forms
Sepsis	SIRS due to infection
Severe sepsis	Sepsis with evidence of organ hypoperfusion
Septic shock	Severe sepsis with hypotension (systolic BP < 90mmHg) despite adequate fluid resuscitation or the requirement for vasopressors/inotropes to maintain blood pressure

Therapeutic hypothermia

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MILD THERAPEUTIC HYPOTHERMIA TO IMPROVE THE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

THE HYPOTHERMIA AFTER CARDIAC ARREST STUDY GROUP*

ABSTRACT

Background Cardiac arrest with widespread cerebral ischemia frequently leads to severe neurologic impairment. We studied whether mild systemic hypothermia increases the rate of neurologic recovery after resuscitation from cardiac arrest due to ventricular fibrillation.

Methods In this multicenter trial with blinded assessment of the outcome, patients who had been resuscitated after cardiac arrest due to ventricular fibrillation were randomly assigned to undergo therapeutic hypothermia (target temperature, 32°C to 34°C, measured in the bladder) over a period of 24 hours or to receive standard treatment with normothermia. The primary end point was a favorable neurologic outcome within six months after cardiac arrest; secondary end points were mortality within six months and the rate of complications within seven days.

Results Seventy-five of the 136 patients in the hypothermia group for whom data were available (55 percent) had a favorable neurologic outcome (cerebral-performance category, 1 [good recovery] or 2 [moderate disability]), as compared with 54 of 137 (39 percent) in the normothermia group (risk ratio, 1.40; 95 percent confidence interval, 1.08 to 1.81). Mortality at six months was 41 percent in the hypothermia group (56 of 137 patients died), as compared with 55 percent in the normothermia group (76 of 138 patients; risk ratio, 0.74; 95 percent confidence interval, 0.58 to 0.95). The complication rate did not differ significantly between the two groups.

Conclusions In patients who have been successfully resuscitated after cardiac arrest due to ventricular fibrillation, therapeutic mild hypothermia increased the rate of a favorable neurologic outcome and reduced mortality. (N Engl J Med 2002;346:549-56.)

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AN estimated 375,000 people in Europe undergo sudden cardiac arrest yearly.[1] Recovery without residual neurologic damage after cardiac arrest with global cerebral ischemia is rare. After cardiac arrest with no blood flow for more than five minutes, the generation of free radicals, together with other mediators, during reperfusion creates chemical cascades that result in cerebral injury.[2] Until recently, there was no therapy with documented efficacy in preventing brain damage after cardiac arrest.

Several studies have shown that moderate systemic hypothermia (30°C)[3] or mild hypothermia (34°C)[4-8] markedly mitigates brain damage after cardiac arrest in dogs. The exact mechanism for this cerebral resuscitative effect is not clear. A reduction in cerebral oxygen consumption[9,10] and other multifactorial chemical and physical mechanisms during and after ischemia have been postulated.[11-16] These include retardation of destructive enzymatic reactions, suppression of free-radical reactions, protection of the fluidity of lipoprotein membranes, reduction of the oxygen demand in low-flow regions, reduction of intracellular acidosis, and inhibition of the biosynthesis, release, and uptake of excitatory neurotransmitters.

Preliminary clinical studies have shown that patients treated with mild hypothermia after cardiac arrest have an improved neurologic outcome, without important side effects, as compared with the outcome in historical controls.[17-20]

We compared mild hypothermia with standard normothermia in patients who had had cardiac arrest due to ventricular fibrillation. The primary end point

Michael Holzer, M.D., Universitätsklinik für Notfallmedizin, Vienna, Austria, assumes overall responsibility for the integrity of the report. Address reprint requests to Dr. Fritz Sterz, Universitätsklinik für Notfallmedizin, Allgemeines Krankenhaus der Stadt Wien, Währinger Gürtel 18-20/6D, 1090 Vienna, Austria, or at fritz.sterz@akh-wien.ac.at.

*The investigators who participated in the Hypothermia after Cardiac Arrest Study Group are listed in the Appendix.

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TREATMENT OF COMATOSE SURVIVORS OF OUT-OF-HOSPITAL CARDIAC ARREST WITH INDUCED HYPOTHERMIA

STEPHEN A. BERNARD, M.B., B.S., TIMOTHY W. GRAY, M.B., B.S., MICHAEL D. BUIST, M.B., B.S., BRUCE M. JONES, M.B., B.S., WILLIAM SILVESTER, M.B., B.S., GEOFF GUTTERIDGE, M.B., B.S., AND KAREN SMITH, B.Sc.

ABSTRACT

Background Cardiac arrest outside the hospital is common and has a poor outcome. Studies in laboratory animals suggest that hypothermia induced shortly after the restoration of spontaneous circulation may improve neurologic outcome, but there have been no conclusive studies in humans. In a randomized, controlled trial, we compared the effects of moderate hypothermia and normothermia in patients who remained unconscious after resuscitation from out-of-hospital cardiac arrest.

Methods The study subjects were 77 patients who were randomly assigned to treatment with hypothermia (with the core body temperature reduced to 33°C within 2 hours after the return of spontaneous circulation and maintained at that temperature for 12 hours) or normothermia. The primary outcome measure was survival to hospital discharge with sufficiently good neurologic function to be discharged to home or to a rehabilitation facility.

Results The demographic characteristics of the patients were similar in the hypothermia and normothermia groups. Twenty-one of the 43 patients treated with hypothermia (49 percent) survived and had a good outcome — that is, they were discharged home or to a rehabilitation facility — as compared with 9 of the 34 treated with normothermia (26 percent, P=0.046). After adjustment for base-line differences in age and time from collapse to the return of spontaneous circulation, the odds ratio for a good outcome with hypothermia as compared with normothermia was 5.25 (95 percent confidence interval, 1.47 to 18.76; P=0.011). Hypothermia was associated with a lower cardiac index, higher systemic vascular resistance, and hyperglycemia. There was no difference in the frequency of adverse events.

Conclusions Our preliminary observations suggest that treatment with moderate hypothermia appears to improve outcomes in patients with coma after resuscitation from out-of-hospital cardiac arrest. (N Engl J Med 2002;346:557-63.)

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CARDIAC arrest outside the hospital is a major cause of unexpected death in developed countries, with survival rates ranging from less than 5 percent to 35 percent.[1-3] In patients who are initially resuscitated, anoxic neurologic injury is an important cause of morbidity and mortality.[4]

Currently, the treatment of patients with coma after resuscitation from out-of-hospital cardiac arrest is largely supportive. Because cerebral ischemia may persist for some hours after resuscitation,[5] the use of induced hypothermia to decrease cerebral oxygen demand has been proposed as a treatment option.[6] Although this suggestion has been supported by studies in animal models,[7-10] the studies in humans that have been reported to date have been uncontrolled or retrospective.[11-15]

After a pilot study that suggested the feasibility, safety, and possible efficacy of this treatment,[16] we conducted a prospective, controlled trial comparing moderate induced hypothermia with normothermia in comatose survivors of out-of-hospital cardiac arrest.

METHODS

Study Design

The study was performed in Melbourne, Australia, between September 1996 and June 1999. The ambulance service has treatment protocols that follow the recommendations of the Australian Resuscitation Council.[17] Patients were enrolled in the study when the following criteria were fulfilled: an initial cardiac rhythm of ventricular fibrillation at the time of arrival of the ambulance, successful return of spontaneous circulation, persistent coma after the return of spontaneous circulation, and transfer to one of four participating emergency departments. The exclusion criteria were an age of less than 18 years for men, an age of less than 50 years for women (because of the possibility of pregnancy), cardiogenic shock (a systolic blood pressure of less than 90 mm Hg despite epinephrine infusion), or possible causes of coma other than cardiac arrest (drug overdose, head trauma, or cerebrovascular accident). Patients were also excluded if an intensive care bed was not available at a participating institution.

After the return of spontaneous circulation had been accomplished outside the hospital, eligible patients were randomly assigned to hypothermia or normothermia according to the day of the month, with patients assigned to hypothermia on odd-numbered days. For these patients, the paramedics began measures in the field to initiate hypothermia by removing the patient's clothing and applying cold packs (CoolCare, Cheltenham, Victoria, Australia) to the patient's head and torso. The treatment of patients assigned to normothermia followed usual prehospital treatment protocols.

On arrival at a participating emergency department, the patients underwent routine initial assessment and treatment, includ-

From the Intensive Care Unit, Dandenong Hospital, Dandenong (S.A.B., M.D.B.); the Intensive Care Unit, Knox Hospital, Wantirna South, Melbourne (S.A.B., M.D.B., B.M.J.); the Metropolitan Ambulance Service, Victoria (S.A.B.); the Department of Emergency Medicine, Monash Medical Centre, Clayton (T.W.G.); the Department of Intensive Care, Austin and Repatriation Medical Centre, Heidelberg (W.S., G.G.); and the Monash University Department of Epidemiology and Preventive Medicine, St. Kilda (K.S.) — all in Australia. Address reprint requests to Dr. Bernard at Dandenong Hospital, David St., Dandenong, VIC 3175, Australia, or at s.bernard@cnedandenong.org.au.

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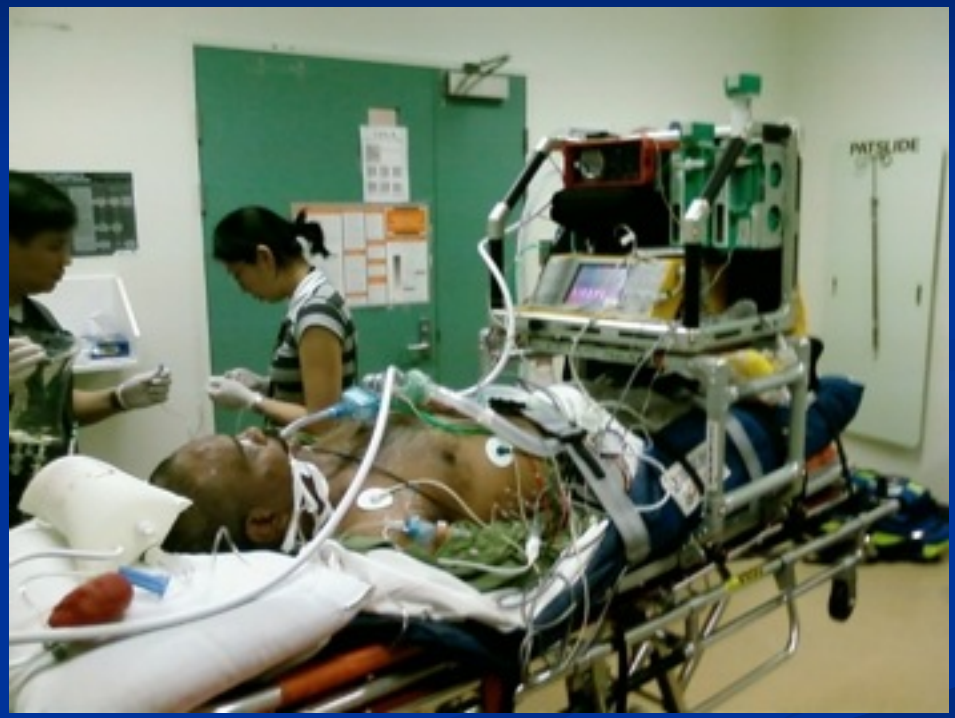
Observing the critical patient

- 1 to 1 nursing. Doctor immediately available.
- Ventilated child – 2 nurses (ED/ITU/Paeds)
- Recording:
  - P, BP, Sats, IBP,
  - Urine hourly
  - EtCO2 – the 'desert island' monitor….
- Sedation, suction, paralysis

Transfers

- Preparation….
  - Get things sorted before early
  - Oxygen, BVM, lines
  - Monitoring – EtCO2….
  - Drugs
  - Plans
    - what if it all goes wrong..?



Preparation for transport

- CABC's addressed
- Haemorrhage controlled
- Splinting / immobilisation
  - "SKIN TO SPLINT"
- IV access x2 – secured!
- Packaged
  - Vacuum mattress
- "Pink, warm, dry"

EMERGENCY MEDICAL RETRIEVAL

Activating MedSTAR - 13STAR (137 827) Option 1

For all critical care transfers consider early involvement of the retrieval service

WHO? – Patients who are critically ill or injured or are deteriorating and are likely to require critical/intensive care management.

WHY? – Early notification of MedSTAR will provide access to critical care advice to help manage patients locally and will allow timely activation of a retrieval response if required.

Critical illness

- Intubated patients (requiring respiratory support)
- Circulatory failure (requiring inotropes)
- Severe sepsis
- Complex multi-system disorders with clinical instability
- Specialised critical care needed (eg balloon pump)
- Premature infants

Referral may precede availability of results of tests or investigations

Major trauma

- Penetrating injuries (excluding isolated limb)
- Major head injury, blunt injuries or fractures
- Limb threatening injuries
- Uncontrolled significant haemorrhage
- Spinal injury with neurological signs
- Burns > 20%, special areas, paediatric burns
- Patients with high risk mechanism of injury whose vital signs deteriorate

Refer also to the South Australian Statewide Trauma Team Activation Criteria

Early activation and timely critical care transfer may improve clinical outcomes

VITAL SIGNS (persistent abnormalities despite appropriate therapy – seek critical care advice)

	Adult	Child (1-4 years)	Neonate (0-3 months)
Consciousness AVPU or GCS (best response)	GCS <13	V,P,U	Hypotonia, poor feeding, excessive sleepiness
Respiratory rate	<15 >30	<20 >40	<25 >60
Respiratory distress	present	present	present
Heart rate	<40 >120	< 90 >155	<100 >180
Systolic BP	<90	<70 >110	Cap refill >3 secs
SpO_2 (with O_2)	<90%	<95%	<90%

13STAR (137 827) Option 1

Statewide 24/7 (including telemedicine facilities)

For inter-hospital transfers (not requiring a Retrieval Team) Fax Request: 1300 730 800

PREPARATION FOR RETRIEVAL

Contact: 13STAR (137827) Option 1

Careful preparation for retrieval transport improves patient care and reduces risk.

AIRWAY

Ensure patient airway safety

1. Assess airway stability for all patients
2. Secure tracheal tube
3. Record tracheal tube (TT) size and length to lips
4. Oro/Naso-gastric tube (NGT/OGT) placed
5. CXR to confirm position of TT/NGT

BREATHING

Ensure ventilation optimised

1. Measure respiratory rate and record respiratory effort
2. Monitor SpO_2 and $ETCO_2$
3. Administer oxygen using appropriate device
4. Check blood gases if indicated and equipment available
5. Secure chest tubes if present

CIRCULATION

Control bleeding

1. Appropriate pressure/splintage
2. Consider reversing anti-coagulant treatment

Ensure patient IV access

1. Insert two peripheral IV lines – appropriate size
2. Secure all lines – ensure injection ports are accessible
3. Prepare drug infusions in 50ml Luer lock syringes
4. Record all IV fluids and drugs
5. Consider arterial and central venous access

DOCUMENTS

Ensure patient documentation completed

1. Provide copies in envelope
   - all patient charts
   - investigation results – pathology and ECG
   - imaging – x-rays / CT scans / ultrasound scans / MRI
2. Document and advise any 'limitation of treatment' orders

OTHER

1. Maintain appropriate temperature
2. Consider indwelling urinary catheter
3. Maintain fluid balance chart
4. Empty drainage bags prior to transport
5. Administer antiemetic if indicated
6. Maintain spinal precautions if required

ALERT

It is important you notify the MedSTAR coordinator of:

1. Significant deterioration in:
   - conscious state
   - blood pressure and/or heart rate
   - respiratory status and/or oxygenation
2. Major clinical developments such as significantly abnormal diagnostic tests, new clinical signs etc
3. The need for major interventions prior to the retrieval team arriving (e.g. intubation, surgery etc)

Ensure patient and/or family are aware of plans and that there is no guarantee that relatives can accompany the patient.

Withdrawal of Care

- Autonomy v Paternalism
- 'Futility'
- 'Best interests'
- Withholding v Withdrawal
- Surrogate decision makers
- Call MedSTAR....

- “the aggregation of marginal gains”
- “attention to detail”