
10
EMERGE
Celebrating ten
years of Emergency
Medicine research

Programme- 22nd November

EMERGE

Celebrating ten years of Emergency Medicine research

Trauma Care
Chairs: Dr Dave Caesar and Professor Alasdair Gray

11:00-11:20: Professor Alasdair Gray- *Welcome to EMERGE10 and History of EMERGE*

11:20-11:50: Professor Ian Roberts– *TXA in trauma and other bleeding emergencies*

11:50-12:15: Professor Heather Jarman– *Leading the way– establishing a trauma service*

12:15-12:40: Dr Youri Yordanov– *Lessons from the Paris Attacks*

12:40-13:00: Dr Martin McKechnie– *Trauma Care Scotland– an update*

13:00-14:00 **Lunch**

Novel Technologies in Emergency Care
Chairs: Dr Matt Reed and Professor Heather Jarman

14:00-14:30: Professor Jim Quinn– *Using smart phones to improve diagnosis-iFaint*

14:30-14:50: Dr Matt Reed– *How smart phones can change practice– the IPED results*

14:50-15:10: Professor Rustam Al-Shahi Salman– *Brain Imaging– friend or foe*

15:10-15:30: Polly Black and Rachel O'Brien– *Using staff surveys to improve research participation*

15:30-16:00 **Break**

Chest Pain
Chairs: Dr Anoop Shah and Dr Gareth Clegg

16:00-16:20: Dr Marc Dweck– *Picturing the heart in 2020*

16:20-17:20: Dr Edd Carlton– *Troponin– all that glitters is not gold*

Professor Rick Body– *Troponin– is only part of the story*

Professor Nick Mills– *Troponin is everything*

The future of chest pain assessment panel discussion

17:20-17:25: Julia Grahamslaw and Miranda Odam– *Closing address*

17:25-17:30: Professor Colin Robertson– *Poster Prize Winner Announcement*

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iRhythm

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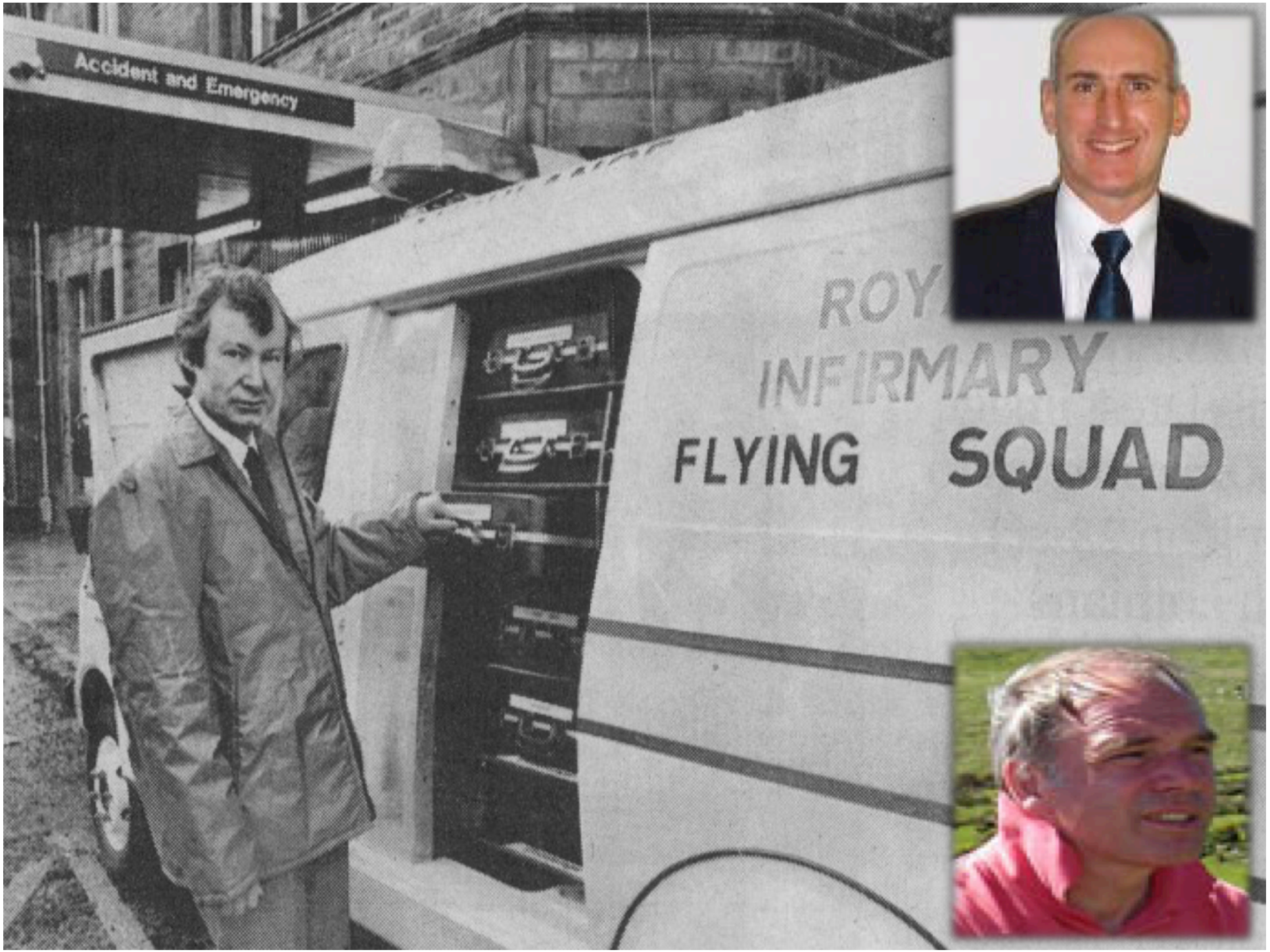
In the
Beginning


Accident and Emergency
ROY
INFIRMARY
FLYING SQUAD

USE OF ETHER IN LIFE-THREATENING ACUTE SEVERE ASTHMA

C. E. ROBERTSON D. STEEDMAN
C. J. SINCLAIR D. BROWN
N. MALCOLM-SMITH

Department of Accident and Emergency Medicine and Department of Anaesthetics, Royal Infirmary, Edinburgh

Summary Two patients with acute severe asthma who had failed to improve with conventional treatment, mechanical ventilation, and halothane showed prompt bronchodilator response following administration of diethyl ether by inhalation. Airway pressures fell, and there was a dramatic improvement in blood gas analysis and clinical condition.

mmol/l, pO_2 26 kPa. Halothane was discontinued and diethyl ether was administered by inhalation using a Mapleson type A circuit (approximate inspired concentration 15–20%). Before this the inflation pressures had continued to be greater than 80 cm water. Within 10 min of the administration of ether the inspiratory airway pressure had fallen to 20–30 cm water, with a striking improvement in bronchospasm on auscultation of the chest. Arterial blood gas analysis after a further 15 min showed H^+ 164 nmol/l, pCO_2 20·2 kPa, bicarbonate 23 mmol/l, pO_2 48 kPa. He required intubation and assisted ventilation for 3 h, and after extubation he was self-ventilating and breathing air. Blood gas analysis was normal. He was discharged from intensive care after 12 h with no evidence of hypoxic brain damage.

Case 2

A 4
admit

Emergency Care

ARE ACCIDENT FLYING SQUADS REALLY CLEARED FOR "TAKE-OFF"?

COLIN ROBERTSON DAVID J. STEEDMAN

Department of Accident and Emergency Medicine, Royal Infirmary, Lauriston Place, Edinburgh EH3 9YW

THE concept of "first aid to the wounded" is not new. Napoleon's surgeon-in-chief, Dominique Jean Larrey appreciated the requirement for rapid evacuation and early

The two major preventable causes of death following trauma are respiratory obstruction and/or inadequate ventilation, and hypovolaemia. Several attempts have been made to quantify these components. Lauppi indicated that 14% of patients who died within 2 days of their injury did so because of respiratory obstruction. Ruffnell-Smith reported the frequency of death from respiratory obstruction to be 5%.[5] In a major study in 1976 casualty officers found airway obstruction in 10·7% of patients who died as a consequence of trauma, but only in 0·7% of survivors. They also reported inhalation of blood and vomitus in 36% of all deaths related to road traffic accidents.[6] In 1982 the same workers reported that nearly 40% of patients who subsequently died had an obstructed airway at the scene of the accident.[7] In Yates'
f deaths over a 5 year period, of patients who died in
l as a consequence of trauma, those who had airway
tion had less severe injuries as measured by a standard
system than did those without such obstruction.[8]
iding strongly implied that airway obstruction in such
xt contributed to mortality.
situation with regard to blood loss is less well
ented. Hoffman showed that for patients who died
sociated blood loss, 59% died instantly and 85% died
6 h, suggesting that perhaps 25% of deaths from
ilaemia could have been prevented.[7] Sherriff has
ted that 33% of patients who died of trauma from road
accidents did so because of blood loss and that 7–10%

study, but this is unlikely. Men who undertake regular outdoor activity have been shown to have a lower prevalence of hip fracture than those who do not,[3] and active men have a higher bone density than sedentary men.[4] Our data suggest that any exercise is beneficial to bone and that, within the normal range, the more the better.

Slemenda *et al*[5] reported that the high leg and pelvic bone densities in young female figure skaters aged 10-25 were not evident until their mid-teens, suggesting that selection of skaters from a biased population was unlikely. Furthermore, the effect of exercise on bone appears to be related to site. Most of the activities we recorded were weight bearing and likely to stress the upper femur. The mean difference in femoral bone density across the range of activities recorded was 12%, equivalent to 18 years of bone loss.[1] The observation that bone density was related to physical activity only in those aged up to 50 suggests that exercise may have its major effect on peak bone density.

1 Wishart JM, Need AG, Horowitz M, Morris HA, Nordin BEC. Effects of age on bone density and bone turnover in men. *Clin Endocrinol* (in press).
2 Wickham CAC, Walsh K, Cooper C, Barker DPJ, Margetts BM, Morris J, Bruce SA. Dietary calcium, physical activity, and risk of hip fracture: a prospective study. *BMJ* 1989;299:889-92.
3 McArdle WD, Katch FI, Katch VL. *Exercise physiology: energy, nutrition and human p*
4 Snow-Har
density,
1992;7:
5 Slemenda
specific
125-32.

(Accepted 21

Archives of Emergency Medicine, 1992, **9**, 203–207

THE LANCET

Vol 341 Saturday 13 March 1993 No 8846

ARTICLES

Interleukin-8 and development of adult respiratory distress syndrome in at-risk patient groups

SEAMAS C. DONNELLY ROBERT M. STRIETER STEVEN L. KUNKEL
ALFRED WALZ COLIN R. ROBERTSON DAVID C. CARTER
IAN S. GRANT ANTONY J. POLLOK CHRISTOPHER HASLETT

Neutrophils have been implicated in the pathogenesis of the adult respiratory distress syndrome (ARDS). We have measured concentrations of the neutrophil attractant interleukin-8 in blood and bronchoalveolar lavage fluid (BAL) from patients at risk of ARDS.

We studied 29 patients from three groups at risk of developing ARDS: multiple trauma (n=16), perforated bowel (n=6), and pancreatitis (n=7). ARDS developed in 7 of these patients. Interleukin-8 in BAL and blood samples taken on initial hospital presentation was measured by a sandwich enzyme-linked immunosorbent assay. The mean BAL interleukin-8 concentration was significantly higher for the patients who subsequently progressed to ARDS than for the non-ARDS group (3·06 [SE 2·64] *vs* 0·053 [0·010] ng/mL, p=0·0006). There was no difference between the groups in plasma interleukin-8 (6·23 [2·60] *vs* 5·12 [2·22] ng/mL, p=0·31). Immunocytochemistry suggested that the alveolar macrophage is an important source of interleukin-8 at this early stage in ARDS development.

This study provides evidence of a relation between the presence of interleukin-8 in early BAL samples and the development of ARDS. The early appearance of interleukin-8 in BAL of patients at risk of ARDS may be an important prognostic indicator for the development of the disorder and reinforces the likely importance of neutrophils and the effects of their accumulation and activation in the pathogenesis of many cases of ARDS.

Lancet 1993; **341**: 643–47.

Introduction

Adult respiratory distress syndrome (ARDS) characteristically develops after a latent period of hours or days since the initiating or provoking insult. The progressive respiratory failure is associated with pathological features caused by the breakdown of alveolar capillary integrity within the lung and leakage of protein-rich fluid into the alveolar space. Despite improvements in intensive care support for patients with ARDS, mortality has remained at 50–90%.[1] Research has tended to concentrate on patients with established ARDS in an intensive care setting. However, the disease process is likely to have been under way for hours or days before clinical presentation. The risk of progression to ARDS varies from 1% to 35%, depending on the initiating or provoking insult.[2] At present it is not possible to identify clearly individuals or subgroups of patients who are at very high risk of this disorder.

It is generally believed that ARDS arises as a result of tissue injury secondary to inflammatory-cell sequestration, emigration, activation, and secretion of their histotoxic products. Neutrophils have received much attention as a key part of this process.[3] Although ARDS has been described in

ADDRESSES: **Respiratory Medicine Unit, University of Edinburgh, City Hospital, Edinburgh, UK** (S. C. Donnelly, MRCPI, Prof C. Haslett, FRCP); **Department of Pathology and Medicine, Division of Pulmonary and Critical Care Medicine, University of Michigan, Ann Arbor, USA** (R. M. Strieter, MD, S. L. Kunkel, PhD); **Theodor Kocher Institute, University of Berne, Switzerland** (A. Walz, PhD); **Departments of Accident and Emergency Medicine** (C. R. Robertson, FRCPE), **Surgery** (Prof D. C. Carter, FRCSE), **and Anaesthetics** (A. J. Pollok, FFA RCS), **Royal Infirmary, Edinburgh; and Intensive Therapy Unit, Western General Hospital, Edinburgh** (I. S. Grant, FFA RCS). Correspondence to Prof Christopher Haslett, Respiratory Medicine Unit, University of Edinburgh, City Hospital, Edinburgh EH10 5SB UK.

The time of death after trauma

Jonathan Wyatt, Diana Beard, Alasdair Gray, Anthony Busuttil, Colin Robertson

The pre-eminence of trauma as a cause of death in young adults in the United Kingdom is well established, but little is known about the temporal distribution of these deaths.[1] The only complete data are from a frequently quoted paper, in which Trunkey described trauma deaths in San Francisco over two years.[2] These data are nearly two decades old and come from a country where the causes of trauma and the system for dealing with it differ from those in the United Kingdom.

Patients, methods, and results

All patients aged over 12 who died after trauma in the Lothian and Borders regions of Scotland between 1 February 1992 and 31 January 1994 were studied prospectively by the Scottish Trauma Audit Group and the university department of forensic medicine. The time and mechanism of injury and the time of death were recorded. Postmortem examinations were performed in every case, and injury severity scores calculated, using the abbreviated injury scale, 1990 revision. The definition of trauma used was that previously used by Trunkey, allowing direct comparison.

There were 331 deaths following trauma, including 26 murders and 98 suicides. Of the victims, 253 (76%) died within one hour of injury; 248 of these died instantaneously and had unsurvivable injuries (abbreviated injury scale 6, injury severity score 75) or were found dead. The remaining five patients died at the scene or in transit to hospital. Seventy eight patients survived more than one hour after injury; 59 surviving for more than four hours. The table compares the timing of deaths after trauma in this study with the United States data.

Comment

In his analysis Trunkey suggested that deaths after trauma follow a trimodal distribution.[2] The first and

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Flying squad response to out-of-hospital cardiac arrest — a decade of experience

S. CUSACK, D. J. STEEDMAN, C. E. ROBERTSON & K. LITTLE

Department of Accident & Emergency Medicine, Royal Infirmary Lauriston Place, Edinburgh

SUMMARY

The Flying Squad (MEDIC I) based at the Royal Infirmary, Edinburgh, commenced operation in 1980. The MEDIC I response to out of hospital non-traumatic cardiac arrest over the past decade is reported.

On-scene resuscitation was attempted in 384 patients. A total of 149 (39%) patients were successfully resuscitated and transferred to hospital. Thirty-six (9.4%) patients survived to discharge from hospital. Patients receiving basic life support prior to the arrival of MEDIC I and in ventricular fibrillation had a survival rate of 14.5% (25/174). During 1988–89, 21 patients were initially attended by ambulance crews equipped with semi-automatic external defibrillators and eight (38%) of these patients survived.

The response of a hospital-based flying squad to support trained ambulance crews, especially when equipped with a defibrillator may provide an economically and operationally feasible alternative to training all first responders in the full range of paramedic skills.

INTRODUCTION

Hospital-based accident flying squads have provided pre-hospital care in the United Kingdom since 1955 (Collins, 1966). The flying squad based at Edinburgh Royal Infirmary, MEDIC I, commenced operation in 1980 and since that time has responded to over 1,000 call-outs. The profile of MEDIC I response to medical and surgical emergencies over the past decade has been reported previously (Steedman,

Correspondence: D. J. Steedman MD, Consultant, Department of Accident & Emergency Medicine, Royal Infirmary, Lauriston Place, Edinburgh EH3 9YW.

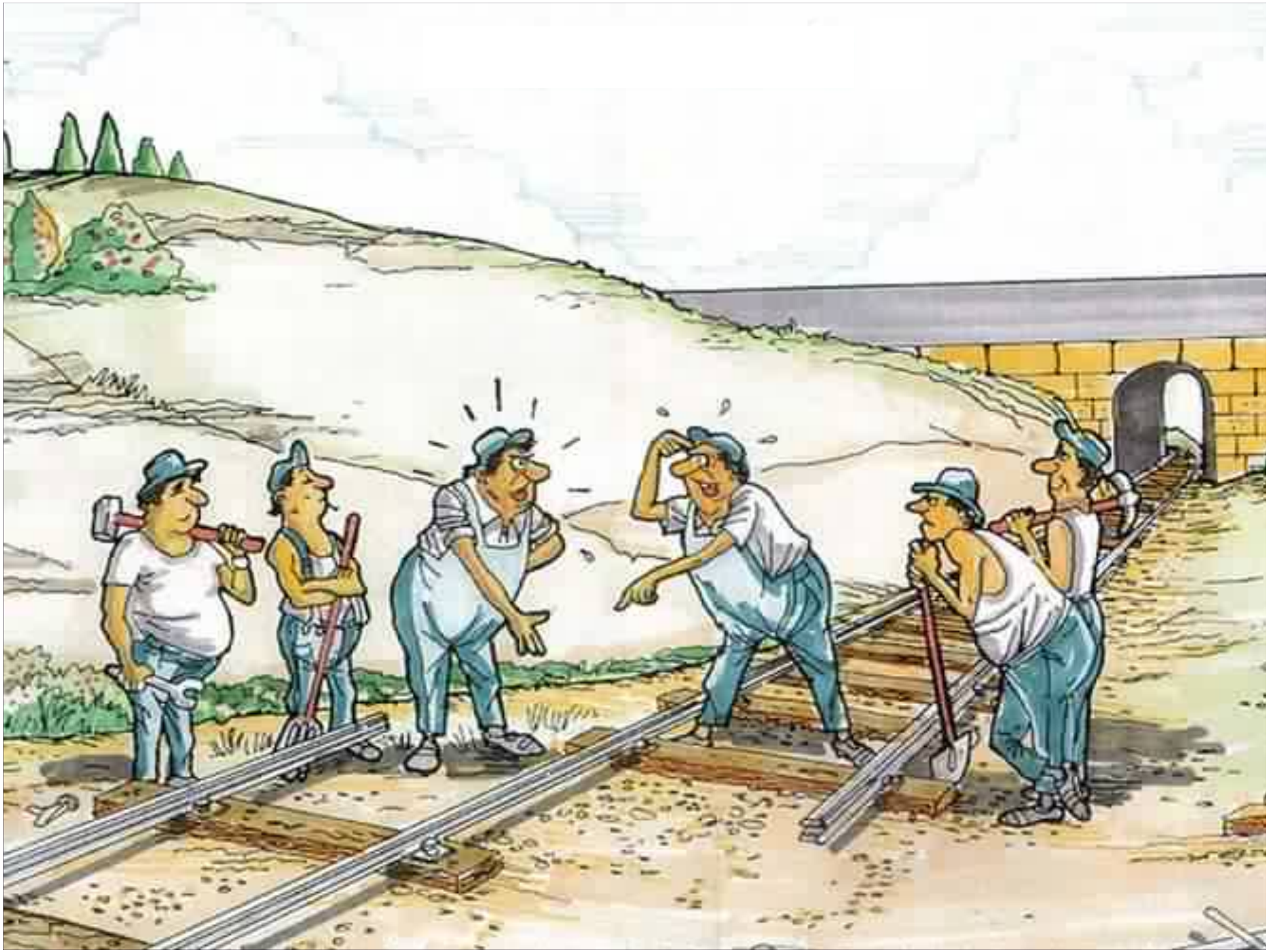

AMBULANCE

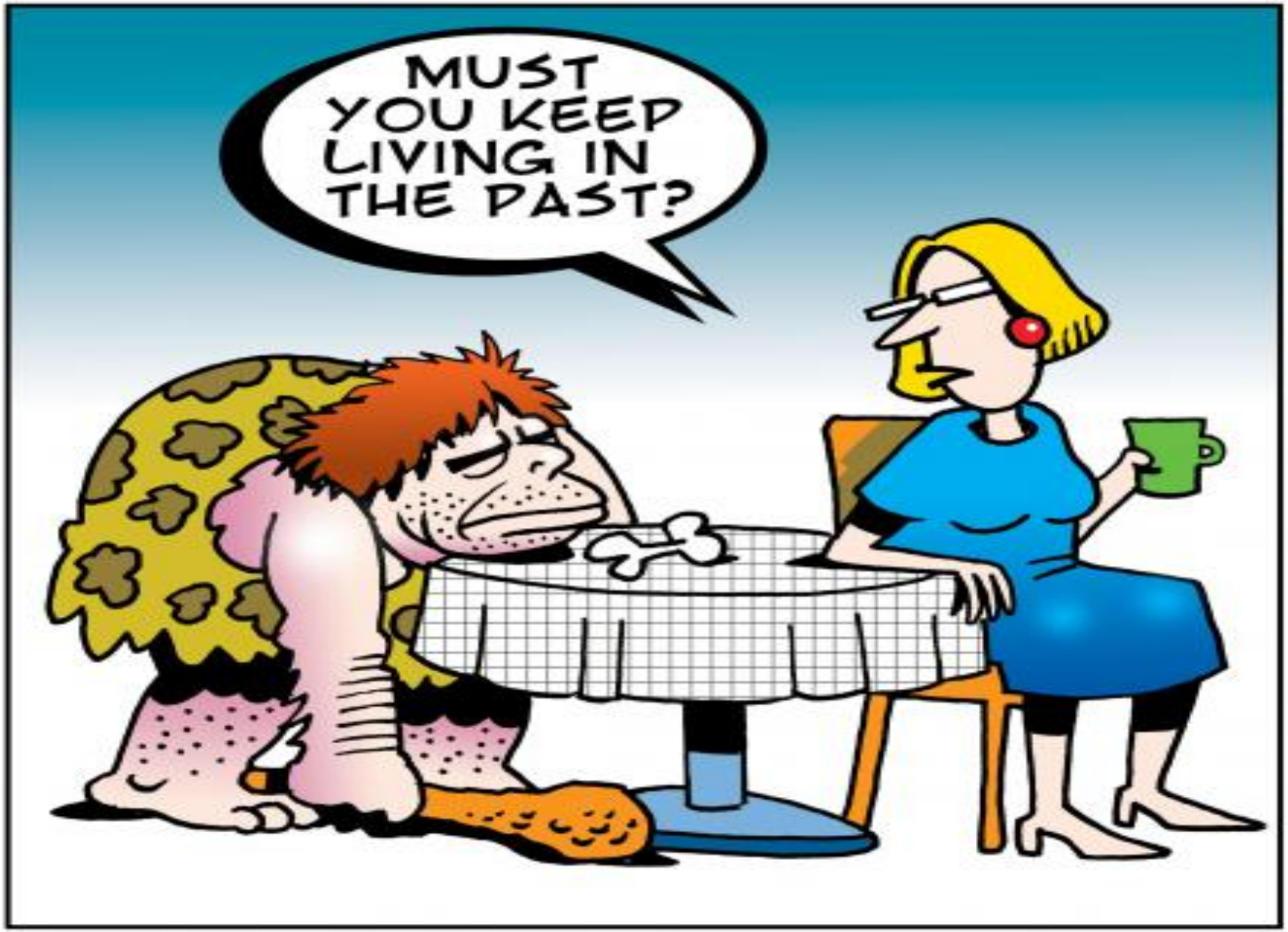

EMERGENCY DEPARTMENT

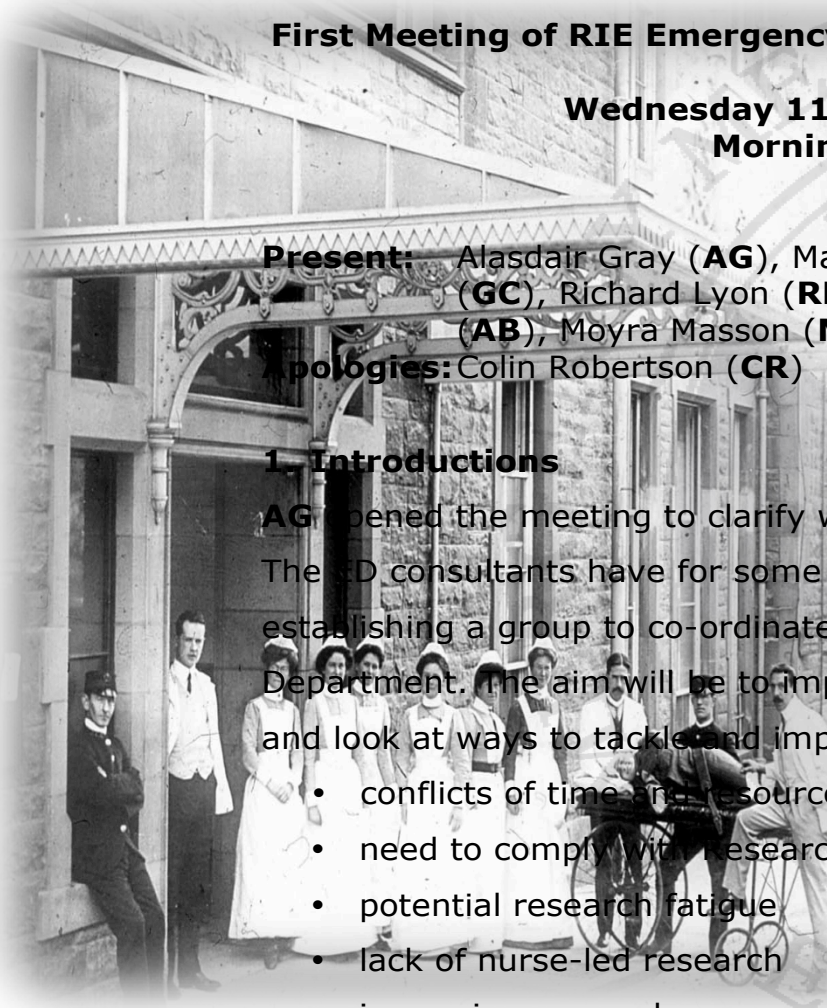
The 3Mg Study

The 3CPO Study


ONEONTA
GORGE
PCR




MUST YOU KEEP LIVING IN THE PAST?



First Meeting of RIE Emergency Department Research Group

Wednesday 11/06/08 16:30
Morningside

Present: Alasdair Gray (**AG**), Matt Reed (**MR**), Gareth Clegg (**GC**), Richard Lyon (**RL**), Judy Coyle (**JC**), Anna Buckby (**AB**), Moyra Masson (**MM**).
Apologies: Colin Robertson (**CR**)

1. Introductions

AG opened the meeting to clarify why the group was convening. The ED consultants have for some time recognised the need for establishing a group to co-ordinate the research activities within the Department. The aim will be to implement a co-ordinated approach and look at ways to tackle and improve on the following:

- conflicts of time and resources
- need to comply with Research Governance
- potential research fatigue
- lack of nurse-led research
- improving research awareness amongst nurses

The 3Mg Study

The 3CPO Study

Emergency Medicine Research Group Edinburgh (EMeRGE)

Five-Year Strategic Research Plan; 2012-16

Alasdair Gray
Consultant and College Professor of Emergency Medicine, Royal Infirmary of Edinburgh;
Honorary Reader, University of Edinburgh;
Clinical Director, Emergency Medicine Research Group, Edinburgh

Matt Reed
Consultant and NRS Fellow in Emergency Medicine, Royal Infirmary of Edinburgh;
Honorary Senior Lecturer, University of Edinburgh.

Gareth Clegg
Senior Clinical Lecturer in Emergency Medicine, University of Edinburgh;
Honorary Consultant in Emergency Medicine, Royal Infirmary of Edinburgh

Moyra Masson
Research Coordinator, EMeRGE,
Department of Emergency Medicine, Royal Infirmary of Edinburgh

On behalf of EMeRGE

2. Aims and objectives

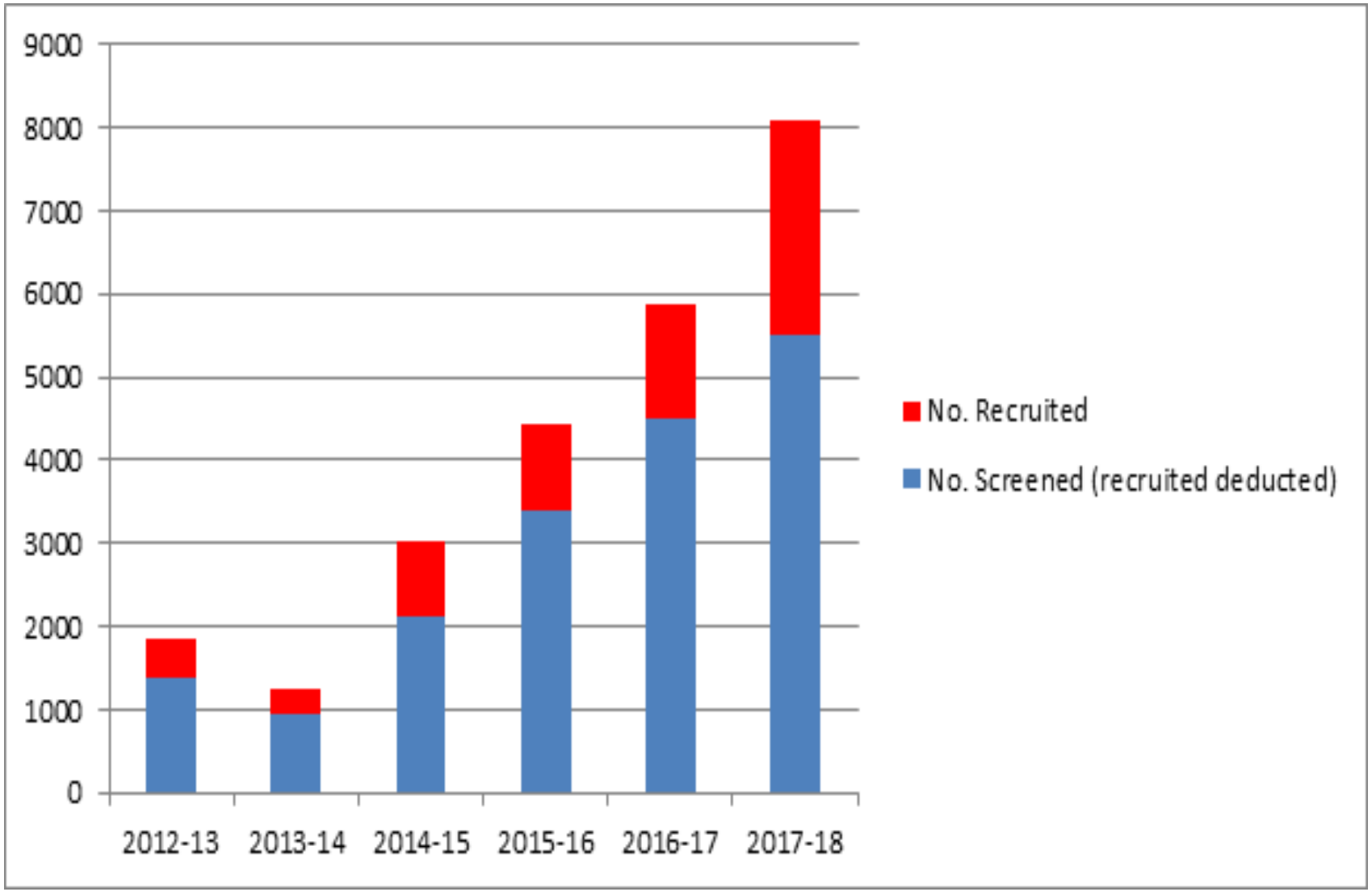
3.1. Aim:

The primary aim of the Emergency Medicine Research Group in Edinburgh is to develop and grow clinical research in emergency medicine in Edinburgh. We aspire to be the leading research centre for Emergency Medicine in the UK with robust national and international research collaborations. It is fundamental to our research philosophy that academic activity is fully integrated with other core clinical and educational activity in the Emergency Department.

3.2. Detailed objectives:

- Increase emergency medicine research activity and output.
- Increase research training opportunity and expertise.
- Further develop research collaboration; we have an explicit desire to foster increasing collaboration with the University of Edinburgh.
- Foster and develop patient involvement.
- Maintain rigorous research governance.
- Ensure research activity delivers patient centred clinical benefits and cost effective care in NHS Scotland.


Today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2017-18
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Care
Medicine
Health
Orthopaedics
Global
Respiratory
Acute
Toxicology
Transfusion
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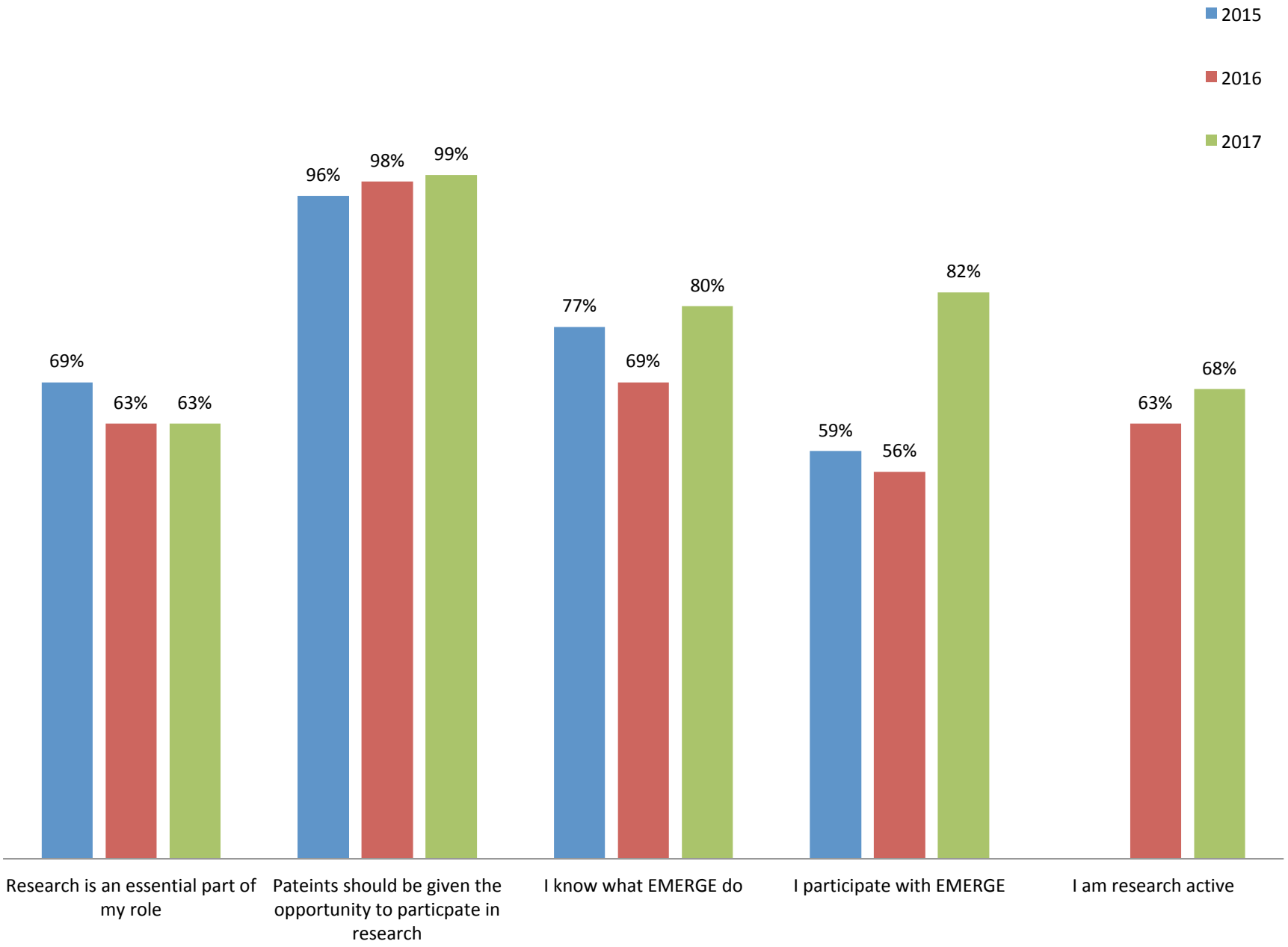
Public
Gastroenterology
Prehospital
Hepatology
Critical
Cardiology
Trauma
Radiology


2015
2016
2017
69%
63%
63%
96%
98%
99%
77%
69%
80%
59%
56%
82%
63%
68%
Research is an essential part of my role
Pateints should be given the opportunity to particpate in research
I know what EMERGE do
I participate with EMERGE
I am research active

Rapid rule out of myocardial infarction

NHS Lothian

Chest pain and suspected acute coronary syndrome (ACS)

Review of pre-hospital and departmental **ECG** by senior staff **within 10 mins**

Immediate IV access and bloods including **high-sensitivity cardiac troponin (hs-cTnI)**

Cardiac monitoring

Non-diagnostic

- Consider Aspirin 300 mg
- Sub-lingual nitrate or analgesia
- CXR as indicated

ST-depression/T-wave inversion

New horizontal or downsloping ST-depression ≥2mm ***or*** deep symmetrical T-wave inversion in 2 adjacent leads

Senior review *or* cardiology referral

- Aspirin 300 mg
- Sub-lingual nitrate or analgesia
- Consider Clopidogrel 300 mg
- Consider Fondaparinux 2.5 mg SC (if not anti-coagulated)
- CXR as indicated

ST-segment elevation

≥2mm in 2 adjacent chest leads
or >1mm in 2 adjacent limb leads
or new LBBB
or >2mm ST depression V1-V3 (posterior)

Bleep 5834 for emergency PCI

- Aspirin 300 mg
- Clopidogrel 600 mg
- Heparin 5,000 units IV (if not anti-coagulated)
- Sub-lingual nitrate or analgesia
- Consider Tirofiban IV bolus

Review baseline hs-cTnI

Admit and hs-cTnI at 6 hours

Myocardial infarction ruled out

A) Clear alternative diagnosis
Treat as appropriate

B) Atypical chest pain or recent negative investigations
Reassure patient low cardiac risk, advice and GP follow up

C) Typical cardiac pain on exertion with no previous investigations
Reassure, aspirin 75mg od and GTN spray, consider referral to the RACPC via Chest Pain Nurse

<5 ng/L*

≥5 ng/L **AND** ≤16 ng/L (women) ≤34 ng/L (men)

>16 ng/L (women) >34 ng/L (men)

Myocardial injury or Infarction

Arrange for admission, senior medical review and repeat hs-cTnI testing at 6 hours

A) Consider other causes of **myocardial injury** (e.g. heart failure, arrhythmia, sepsis, pulmonary embolism)

B) If diagnosis of **type 1 myocardial infarction** confirmed:

- Clopidogrel 300 mg
- Fondaparinux 2.5mg SC
- Sub-lingual nitrate

hs-cTnI 3 hours from presentation

CHANGE <3 ng/L **AND** ≤16 ng/L (women) ≤34 ng/L (men)

CHANGE ≥3 ng/L **AND** ≤16 ng/L (women) ≤34 ng/L (men)

>16 ng/L (women) >34 ng/L (men)

EMERGENCY DEPARTMENT AND PAA

HOSPITAL ADMISSION

Admit and hs-cTnI 6 hours from presentation

Myocardial infarction ruled out

Senior medical review or referral to cardiology

≤16 ng/L (women) ≤34 ng/L (men)

>16 ng/L (women) >34 ng/L (men)

Myocardial injury or Infarction

Referral to cardiology for in-patient assessment

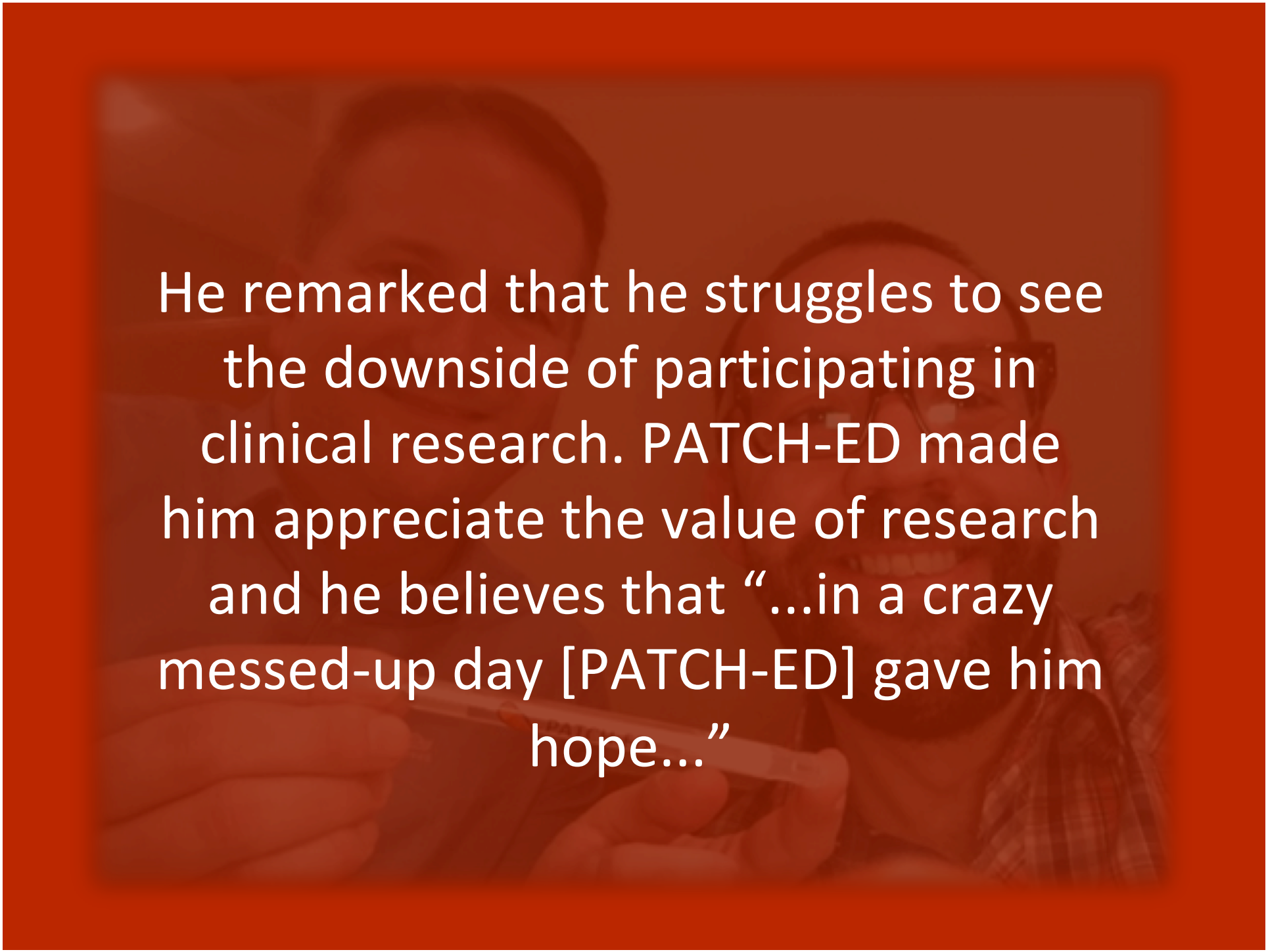
*** In all patients with chest pain for <2 hours repeat hs-cTnI at 3 hours**

Lothian protocol version 5.0; 21st January 2016

Comparison of outcomes after OHCA in Edinburgh 2005 vs 2013/14

	2005	2013/14
Number of resuscitations	258	357
Survival	6%	17%
Patients Home	15	61


‘Save 1000 lives by 2020…’
—Maureen Watt, MSP at the Scottish Cardiac Arrest Symposium 2015
OUT-OF-HOSPITAL CARDIAC ARREST
A STRATEGY FOR SCOTLAND
CARDIAC ARREST
CA 2179


He remarked that he struggles to see the downside of participating in clinical research. PATCH-ED made him appreciate the value of research and he believes that “...in a crazy messed-up day [PATCH-ED] gave him hope...”

Culture

Collaboration

Communication

Celebration


WHAT'S NEXT?

100% Research Active

Make research “normal”

Research active systems have better clinical outcomes


Emergency
Evidence Free Zone

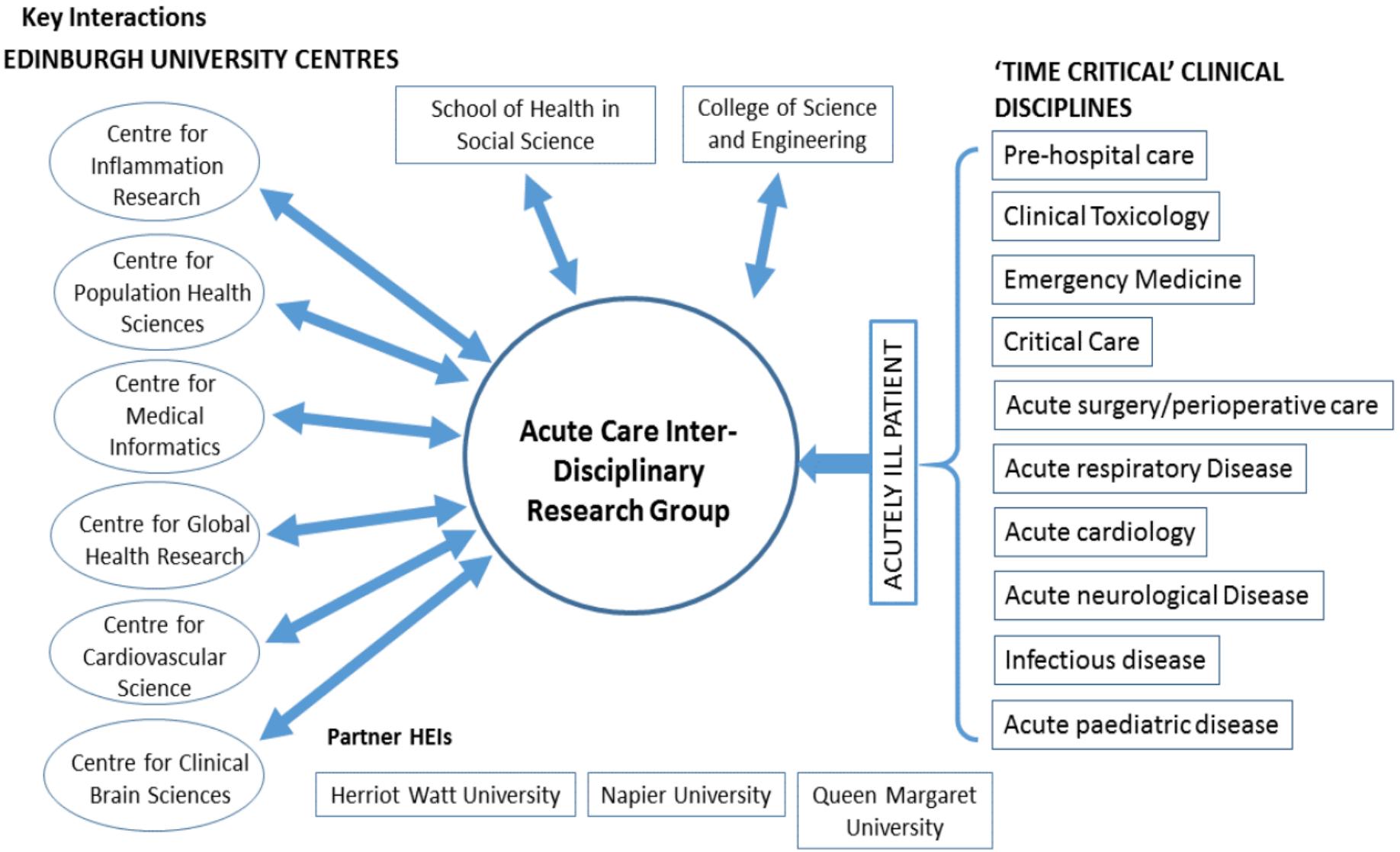

KEEP CLEAR

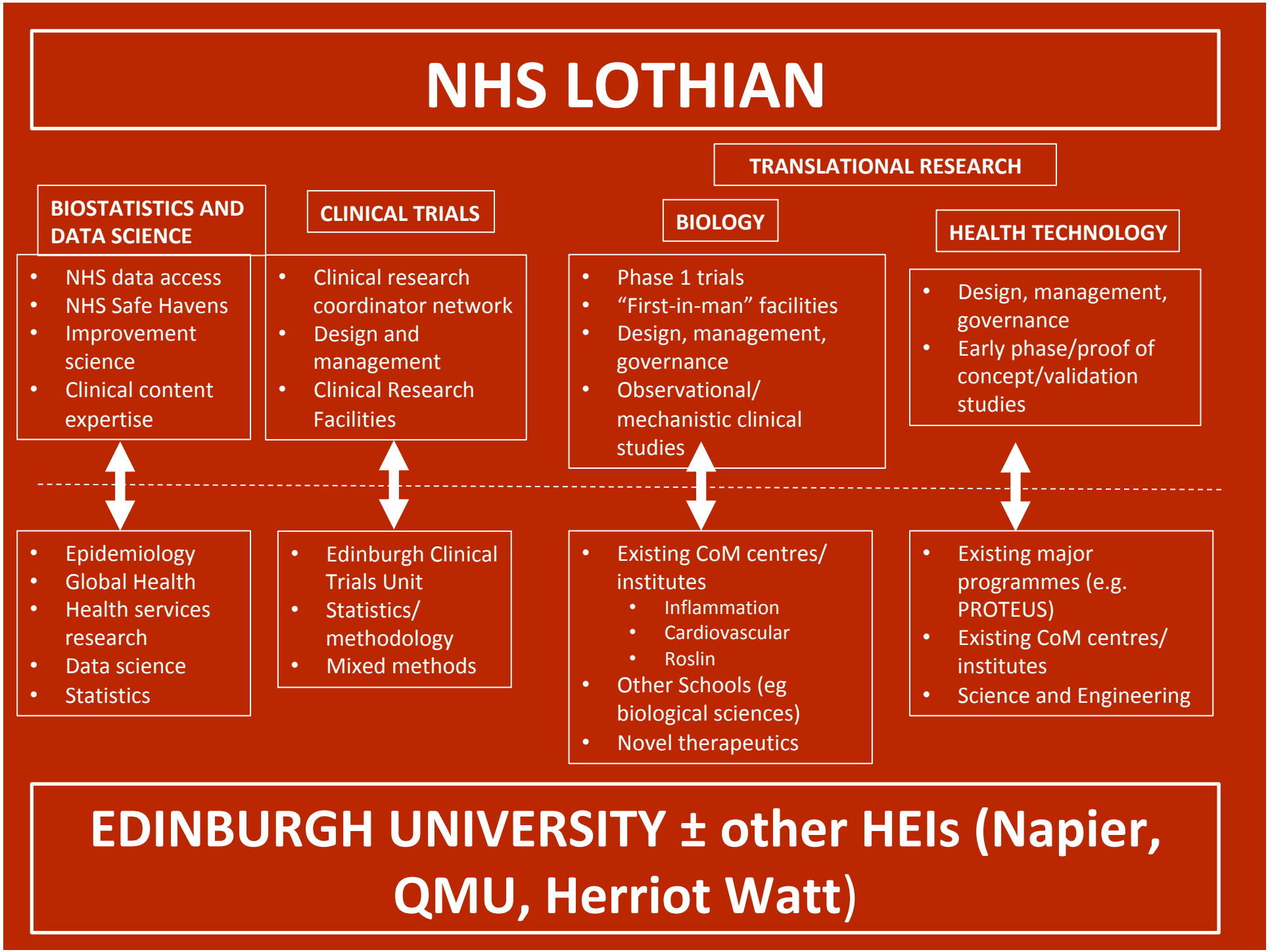
RESUSCITATION RESEARCH GROUP

EdinClinTox

Edinburgh Clinical Toxicology

Acute Care Research Facility




NHS LOTHIAN
TRANSLATIONAL RESEARCH
BIOSTATISTICS AND DATA SCIENCE
CLINICAL TRIALS
BIOLOGY
HEALTH TECHNOLOGY
• NHS data access
• NHS Safe Havens
• Improvement science
• Clinical content expertise
• Clinical research coordinator network
• Design and management
• Clinical Research Facilities
• Phase 1 trials
• "First-in-man" facilities
• Design, management, governance
• Observational/ mechanistic clinical studies
• Design, management, governance
• Early phase/proof of concept/validation studies
• Epidemiology
• Global Health
• Health services research
• Data science
• Statistics
• Edinburgh Clinical Trials Unit
• Statistics/ methodology
• Mixed methods
• Existing CoM centres/ institutes
• Inflammation
• Cardiovascular
• Roslin
• Other Schools (eg biological sciences)
• Novel therapeutics
• Existing major programmes (e.g. PROTEUS)
• Existing CoM centres/ institutes
• Science and Engineering
EDINBURGH UNIVERSITY ± other HEIs (Napier, QMU, Herriot Watt)


10
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Celebrating ten
years of Emergency
Medicine research

Programme- 22nd November

EMERGE

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11:50-12:15: Professor Heather Jarman– *Leading the way– establishing a trauma service*

12:15-12:40: Dr Youri Yordanov– *Lessons from the Paris Attacks*

12:40-13:00: Dr Martin McKechnie– *Trauma Care Scotland– an update*

13:00-14:00 **Lunch**

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Chairs: Dr Matt Reed and Professor Heather Jarman

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14:30-14:50: Dr Matt Reed– *How smart phones can change practice– the IPED results*

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15:10-15:30: Polly Black and Rachel O'Brien– *Using staff surveys to improve research participation*

15:30-16:00 **Break**

Chest Pain
Chairs: Dr Anoop Shah and Dr Gareth Clegg

16:00-16:20: Dr Marc Dweck– *Picturing the heart in 2020*

16:20-17:20: Dr Edd Carlton– *Troponin– all that glitters is not gold*

Professor Rick Body– *Troponin– is only part of the story*

Professor Nick Mills– *Troponin is everything*

The future of chest pain assessment panel discussion

17:20-17:25: Julia Grahamslaw and Miranda Odam– *Closing address*

17:25-17:30: Professor Colin Robertson– *Poster Prize Winner Announcement*

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