Prehospital diagnostik

- systematik og improvisasjonsevne

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Oslo Universitets Sygehus

Gøteborg April 2015


Investec
G-EHMS
AMBULANCE
LONDON'S AIR AMBULANCE


U-481


LEGE


VH-FDZ


RESCUE


S-181

Plan....

- Diagnostik + Magiske spørgsmål
- Ansigtsskader
- Pneumothorax
- RSI – taktik
- Thorakotomi

...eller...."Hvad mener du om...????...."

Hvad og Hvorfor

ABCDE

Udvalgte emner....

Og med stor tak til de som jeg har lånt,
stjålet, købt, kopieret, byttet slides med.....

Why train and practise ?

YES

We can do it !

But we have to train, think, cerebrate and practise to meet these high demands

«...with tailored treatment...»

What is prehospital care

Bringing the emergency department to the patient.....

Advanced critical emergency care on the road side.....

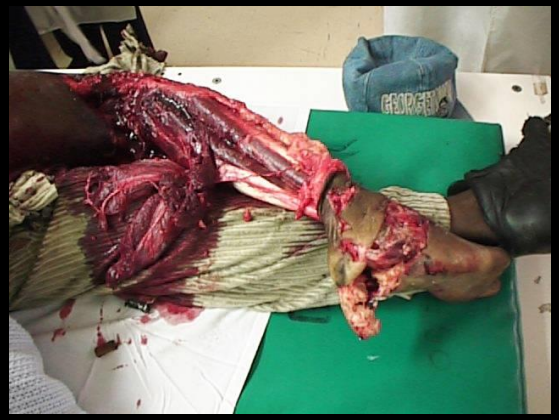
WHY

Advanced airway interventions

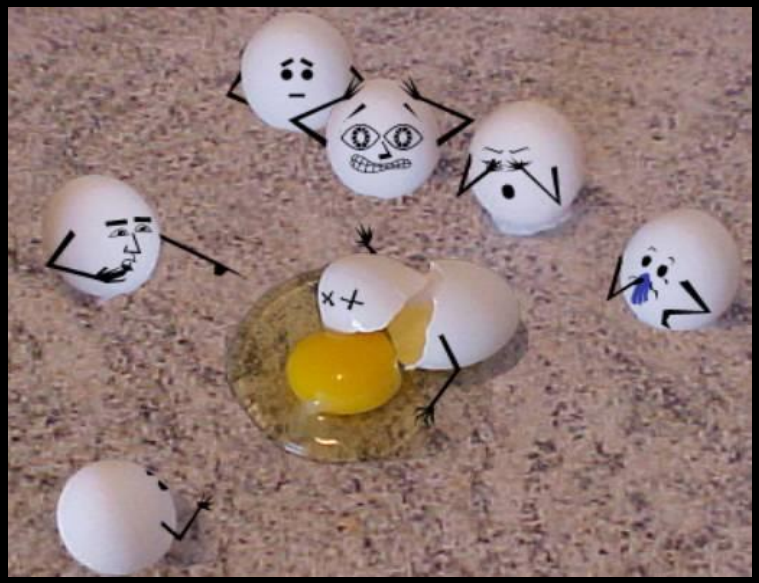
Pharmocologic intubation (RSI)

Triage desicions

Ultimate pain relief



”Why do we have doctors out there ?”



Because they make a difference.....

But it is hard to prove.....

Addition of physicians to paramedic helicopter services decreases blunt trauma mortality

Retrospectice study from NSW, Australia

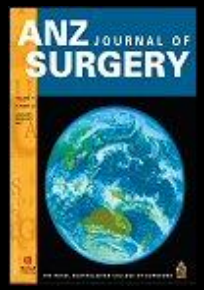
28 months

207 patients

The statistic suggesting that there would be between eight and 19 extra survivors per 100 patients treated in the physician group compared with the paramedic group.

arner A Rashford S Lee A Bartolacci R

Aust N Z J Surg. 2000 Sep;70(9):681.



The experience of Teesside helicopter emergency services: doctors do not prolong prehospital on-scene times

Compared scene time for teams with, and without doctors at the scene

This study did not show any significant prolongation of mean on-scene times for PPP missions either overall or for any of the subgroups A-C

Dissmann PD, **Le Clerc S**

Emerg Med J. 2007 Jul;24(7):521

Prehospital advanced life support provided by specially trained physicians: is there a benefit in terms of life years gained?

Results: The expert panels estimated a benefit of **504 LYG in 74 patients (7% of the total study population)**, with a median age of 54 years (range 0–88). The cause of the emergency was cardiac diseases (including cardiac arrest) in **61%** of the 74 patients, trauma in 19%, and cardio-respiratory failure as a result of other conditions in 20%. The LYG were equally divided between air and ground missions, and the majority (88%) were attributed solely to ALS by the **anesthesiologist.**

H. M. Lossius, E. Søreide, R. Hotvedt, S. A. Hapnes, O. V. Eielsen, O. H. Førde, P. A. Steen (2002)

Acta Anaesthesiologica Scandinavica 46 (7), 771–778.

Impact of pre-hospital care in patients with acute myocardial infarction compared with those first managed in-hospital

T.P. Mathew, I.B.A. Menown, D. McCarty, H. Gracey, L. Hill and A.A.J. Adgey*

+ Author Affiliations

*Correspondence: Professor A. A. J. Adgey, Regional Medical Cardiology Centre, Royal Victoria Hospital, Belfast BT12 6BA, Northern Ireland, U.K.

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This Article

Eur Heart J (2003) 24 (2): 161-171.
doi: 10.1016/S0195-668X(02)00521-3

Abstract
» Full Text (HTML)
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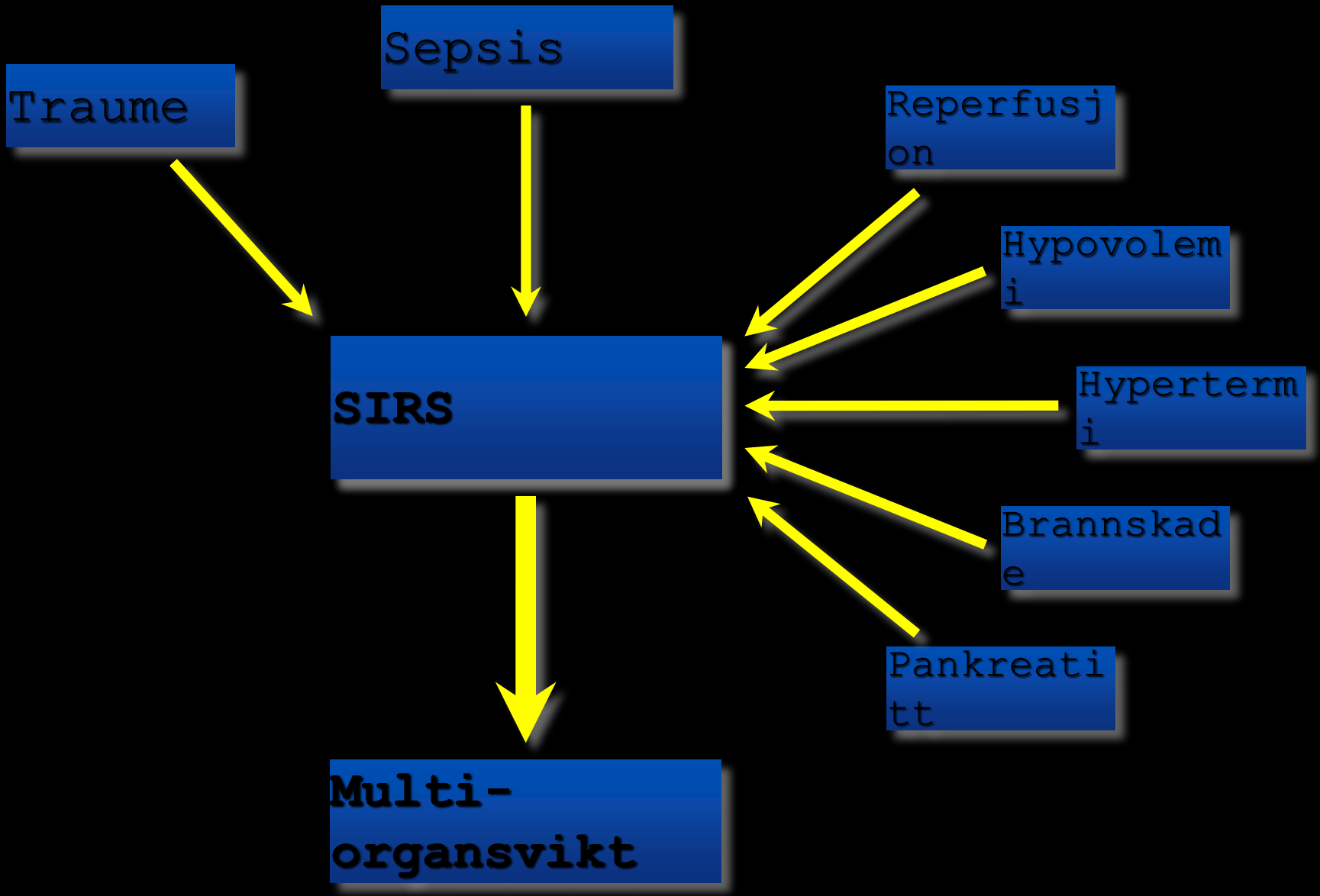
Abstract

Aims To compare prospectively the impact of pre-hospital care by a physician-staffed mobile coronary care unit with patients managed initially in-hospital, all with

WHY

Bucause it matters....

But this is complex.....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Reperfusj
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Hypovolem
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Hyperterm
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SIRS
Brannskad
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Pankreati
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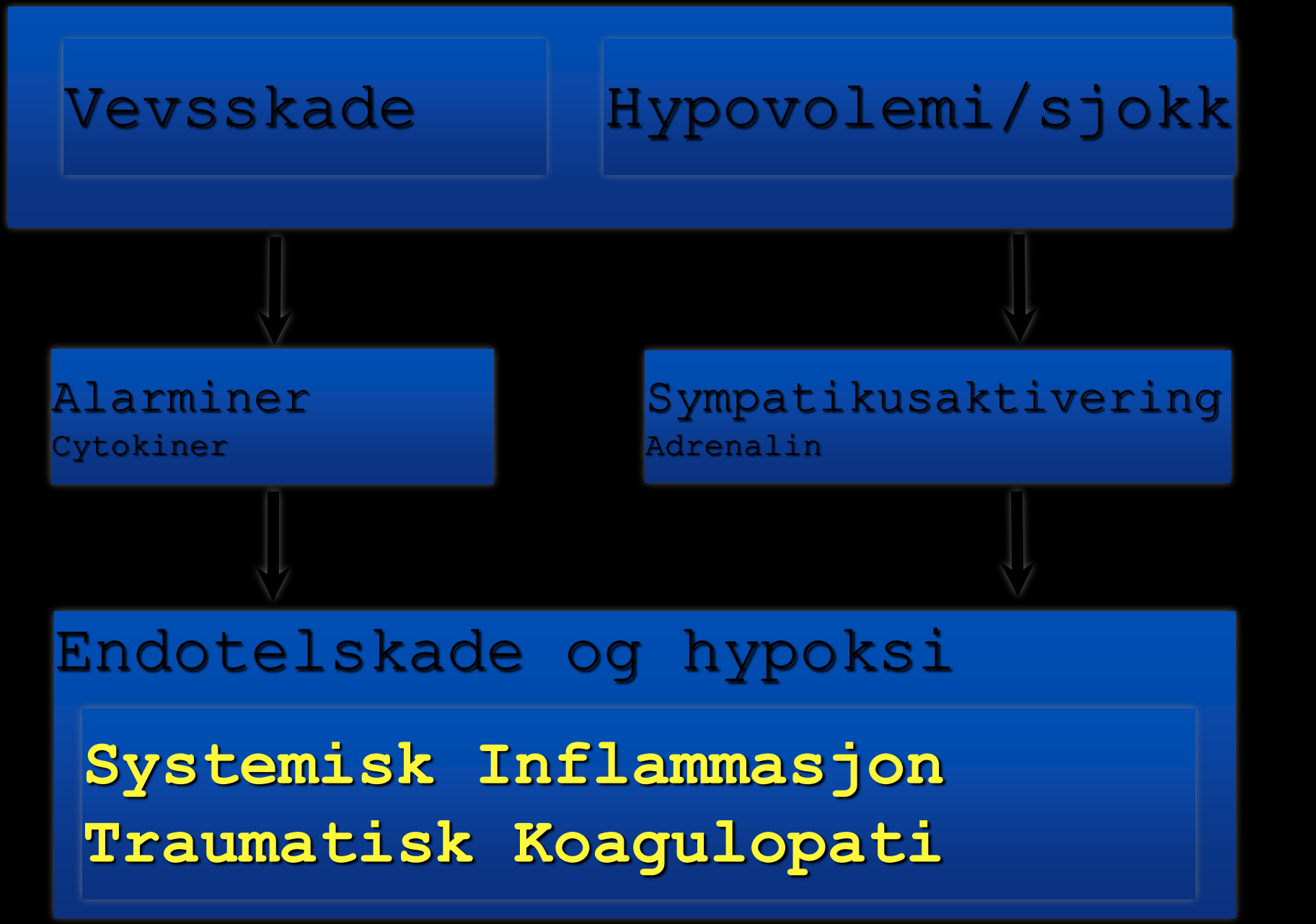
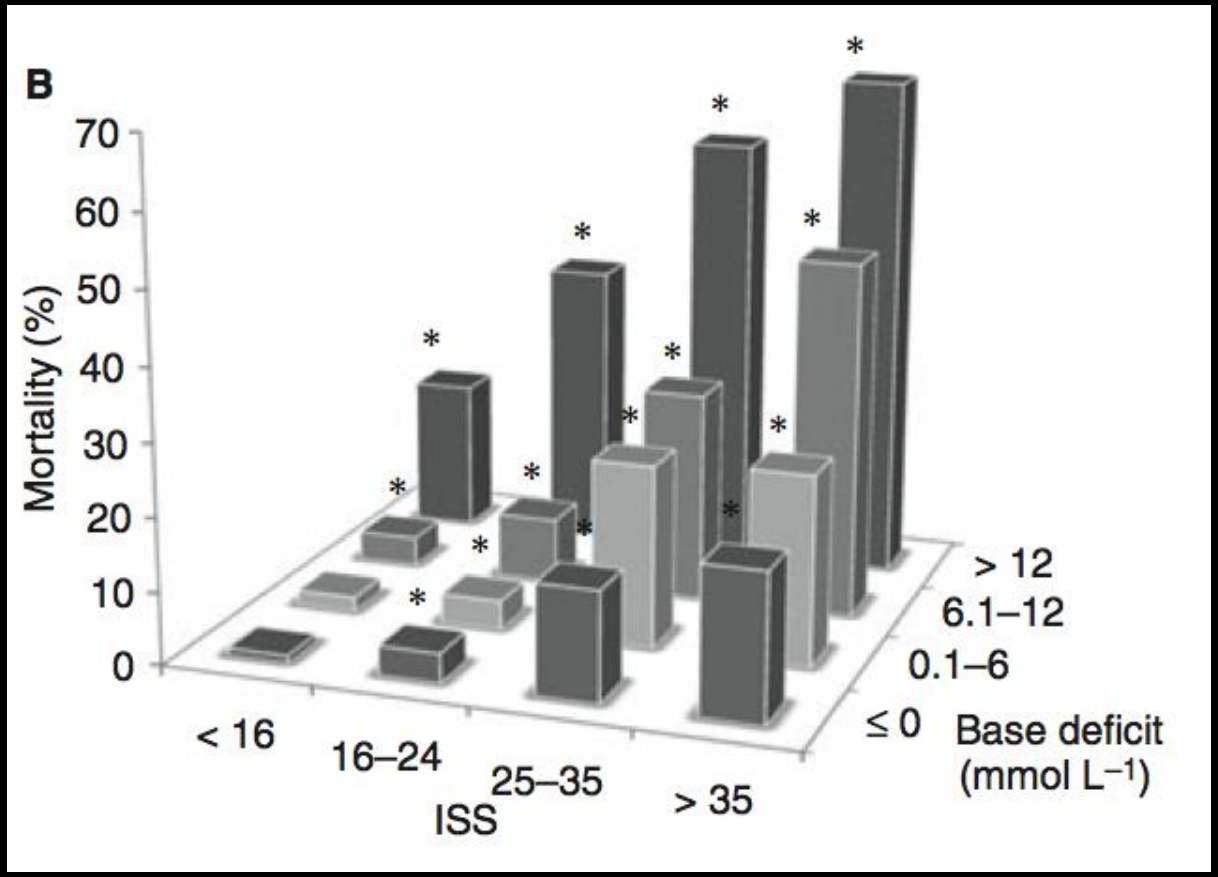

Vevsskade
Hypovolemi/sjokk
Alarminer
Cytokiner
Sympatikusaktivering
Adrenalin
Endotelskade og hypoksi
Systemisk Inflammasjon
Traumatisk Koagulopati

Traume / sjokk

Prehospitalt vindu

Økende grad av:

- inflammasjon
-koagulopati
-interaksjon
-redundance


B
Mortality (%)
70
60
50
40
30
20
10
0
< 16
16–24
25–35
> 35
ISS
> 12
6.1–12
0.1–6
≤ 0
Base deficit
(mmol L⁻¹)

High circulating adrenaline levels at admission predict increased mortality after trauma

Pär Ingemar Johansson, MD, DMSc, MPA, Jakob Stensballe, MD, PhD, Lars Simon Rasmussen, MD, PhD, DMSc, *and* **Sisse Rye Ostrowski, MD, PhD, DMSc,**
Copenhagen, Denmark

BACKGROUND: Trauma-induced acute coagulopathy predicts a poor outcome. Although its pathophysiology is unclear, severe injury and shock (hypoperfusion) are proposed drivers. This study investigated the association between sympathoadrenal activation (circulating catecholamines) and biomarkers of coagulopathy.

METHODS: Prospective study of 75 adult trauma patients admitted to a Level I trauma center directly from the scene of accident. Patients were selected blinded post hoc from three predefined Injury Severity Score groups (<16, 16–27, and >27) and had available blood samples on arrival. We measured activated partial thromboplastin time, international normalized ratio, hematology, biochemistry, circulating adrenaline and noradrenaline, 11 biomarkers of tissue and endothelial damage, glycocalyx degradation, natural anticoagulation and fibrinolysis (histone-complexed DNA fragments, high-mobility group box 1, syndecan-1, von Willebrand factor, soluble thrombomodulin, protein C, tissue factor pathway inhibitor, antithrombin, tissue-type plasminogen activator, plasminogen activator inhibitor-1, D-dimer) and registered 30-day mortality. Biomarkers were compared between survivors and nonsurvivors.

RESULTS: The adrenaline level was increased in nonsurvivors ($p = 0.026$), it was independently associated with increased activated partial thromboplastin time ($p = 0.034$) and syndecan-1 ($p = 0.007$), a marker of glycocalyx degradation, and it correlated with biomarkers of tissue and endothelial damage (histone-complexed DNA, high-mobility group box 1, soluble thrombomodulin) and hyperfibrinolysis (tissue-type plasminogen activator, D-dimer). Furthermore, nonsurvivors had higher syndecan-1, tissue factor pathway inhibitor, and D-dimer levels (all $p < 0.05$). Circulating adrenaline was independently associated with 30-day mortality (OR, 5.92 [95% CI, 1.48–23.73]; $p = 0.012$) together with age ($p = 0.001$) and severe head injury (Abbreviated Injury Scale head >3; $p = 0.011$).

CONCLUSIONS: The trauma-induced catecholamine surge is closely associated with biomarkers of tissue and endothelial damage, glycocalyx degradation, coagulopathy including hyperfibrinolysis and independently predicts mortality. (*J Trauma.* 2012;72: 428–436. Copyright © 2012 by Lippincott Williams & Wilkins)

KEY WORDS: Acute coagulopathy of trauma; sympathoadrenal activation; catecholamines; adrenaline; noradrenaline; hyperfibrinolysis; endothelial glycocalyx; mortality.

The New England
Journal of Medicine

Volume 331 OCTOBER 27, 1994 Number 17

IMMEDIATE VERSUS DELAYED FLUID RESUSCITATION FOR HYPOTENSIVE PATIENTS WITH PENETRATING TORSO INJURIES

WILLIAM H. BICKELL, M.D., MATTHEW J. WALL, JR., M.D., PAUL E. PEPE, M.D., R. RUSSELL MARTIN, M.D., VICTORIA F. GINGER, M.S.N., MARY K. ALLEN, B.A., AND KENNETH L. MATTOX, M.D.

Abstract *Background.* Fluid resuscitation may be detrimental when given before bleeding is controlled in patients with trauma. The purpose of this study was to determine the effects of delaying fluid resuscitation until the time of operative intervention in hypotensive patients with penetrating injuries to the torso.

Methods. We conducted a prospective trial comparing immediate and delayed fluid resuscitation in 598 adults with penetrating torso injuries who presented with a prehospital systolic blood pressure ≤90 mm Hg. The study setting was a city with a single centralized system of prehospital emergency care and a single receiving facility for patients with major trauma. Patients assigned to the immediate-resuscitation group received standard fluid resuscitation before they reached the hospital and in the trauma center, and those assigned to the delayed-resuscitation group received intravenous cannulation but no fluid resuscitation until they reached the operating room.

Results. Among the 289 patients who received delayed fluid resuscitation, 203 (70 percent) survived and were discharged from the hospital, as compared with 193 of the 309 patients (62 percent) who received immediate fluid resuscitation ($P = 0.04$). The mean estimated intraoperative blood loss was similar in the two groups. Among the 238 patients in the delayed-resuscitation group who survived to the postoperative period, 55 (23 percent) had one or more complications (adult respiratory distress syndrome, sepsis syndrome, acute renal failure, coagulopathy, wound infection, and pneumonia), as compared with 69 of the 227 patients (30 percent) in the immediate-resuscitation group ($P = 0.08$). The duration of hospitalization was shorter in the delayed-resuscitation group.

Conclusions. For hypotensive patients with penetrating torso injuries, delay of aggressive fluid resuscitation until operative intervention improves the outcome. (N Engl J Med 1994;331:1105-9.)

Table 3. Systemic Arterial Blood Pressure, Heart Rate, and Laboratory Findings at the Time of Initial Operative Intervention in Patients with Penetrating Torso Injuries, According to Treatment Group.*

VARIABLE	IMMEDIATE RESUSCITATION (N = 268)	DELAYED RESUSCITATION (N = 260)	P VALUE
Systolic blood pressure (mm Hg)	112±33	113±30	0.98
Diastolic blood pressure (mm Hg)	57±22	60±21	0.10
Heart rate (beats/min)	102±25	104±23	0.25
Hemoglobin (g/dl)	10.7±5.8	11.5±2.6	<0.001
Platelet count ($\times 10^{-3}$/mm^3)	195±97	198±105	0.99
Systemic arterial pH	7.27±0.16	7.28±0.15	0.75
Serum bicarbonate concentration (mmol/liter)	21±5	20±4	0.39

*Plus–minus values are means ±SD. To convert values for hemoglobin to millimoles per liter, multiply by 0.62.

Table 3. Classes Of Shock By ATLS® Designation*

	Class 1	Class 2	Class 3	Class 4
Blood loss, %	< 15%	15%-30%	30%-40%	> 40%
Heart rate, beats per minute	< 100	> 100	> 120	> 140
Blood pressure, mm Hg	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate, breaths per minute	14-20	20-30	30-40	> 35
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

Resuscitation 81 (2010) 1142–1147

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Clinical paper

Testing the validity of the ATLS classification of hypovolaemic shock☆

H.R. Guly[a,*,1], O. Bouamra[b,1], R. Little[b,1,2], P. Dark[c,1], T. Coats[d,1], P. Driscoll[e,1], F.E. Lecky[b,1]

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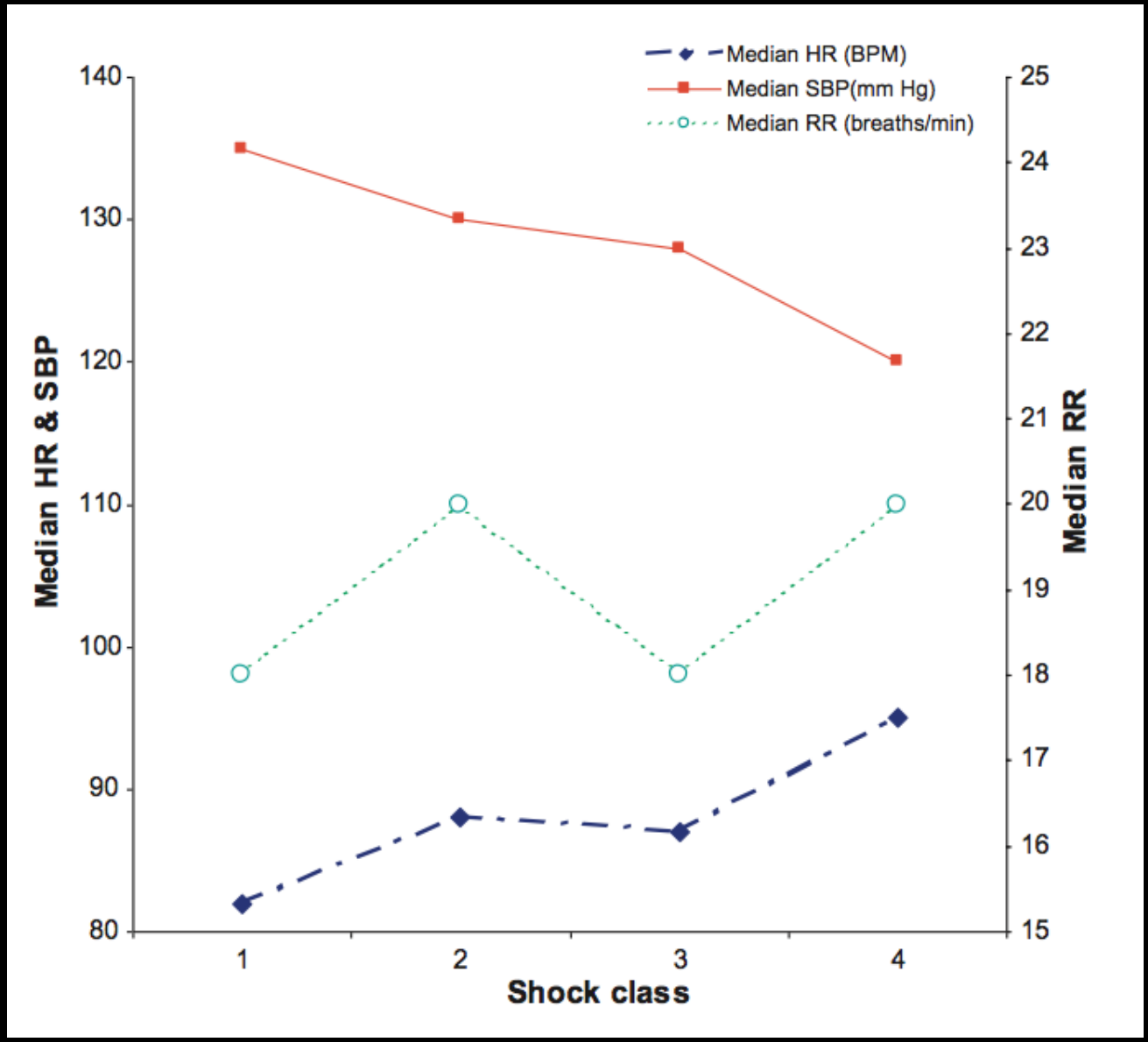
ATLS
Vital signs

ABSTRACT

Aim: The Advanced Trauma Life Support system classifies the severity of shock. The aim of this study is to test the validity of this classification.
Methods: Admission physiology, injury and outcome variables from adult injured patients presenting to hospitals in England and Wales between 1989 and 2007 and stored on the Trauma Audit and Research Network (TARN) database, were studied. Patients were divided into groups representing the four ATLS classes of shock, based on heart rate (HR) systolic blood pressure (SBP), respiratory rate (RR) and Glasgow Coma Score (GCS). The relationships between variables were examined by classifying the cohort by each recorded variable in turn and deriving the median and interquartile range (IQR) of the remaining three variables. Patients with penetrating trauma and major injuries were examined in sub-group analyses.
Results: In blunt trauma patients grouped by HR, the median SBP decreased from 128 mm Hg in patients with HR < 100 BPM to 114 mm Hg in those with HR > 140 BPM. The median RR increased from 18 to 22 bpm and the GCS reduced from 15 to 14. The median HR in hypotensive patients was 88 BPM compared to 83 BPM in normotensive patients and the RR was the same. When grouped by RR, the HR increased with increasing RR but there were no changes in SBP.
Conclusion: In trauma patients there is an inter-relationship between derangements of HR, SBP, RR and GCS but not to the same degree as that suggested by the ATLS classification of shock.


Median HR (BPM)
Median SBP(mm Hg)
Median RR (breaths/min)
Median HR & SBP
Median RR
Shock class
140
130
120
110
100
90
80
25
24
23
22
21
20
19
18
17
16
15
1
2
3
4

The Journal of TRAUMA® Injury, Infection, and Critical Care

Hypotensive Resuscitation during Active Hemorrhage: Impact on In-Hospital Mortality

Richard P. Dutton, MD, MBA, Colin F. Mackenzie, MD, and Thomas M. Scalea, MD

Background: Traditional fluid resuscitation strategy in the actively hemorrhaging trauma patient emphasizes maintenance of a normal systolic blood pressure (SBP). One human trial has demonstrated improved survival when fluid resuscitation is restricted, whereas numerous laboratory studies have reported improved survival when resuscitation is directed to a lower than normal pressure. We hypothesized that fluid resuscitation titrated to a lower than normal SBP during the period of active hemorrhage would improve survival in trauma patients presenting to the hospital in hemorrhagic shock.

Methods: Patients presenting in hemorrhagic shock were randomized to one of two fluid resuscitation protocols: target SBP > 100 mm Hg (conventional) or target SBP of 70 mm Hg (low). Fluid therapy was titrated to this endpoint until definitive hemostasis was achieved. In-hospital mortality, injury severity, and probability of survival were determined for each patient.

Results: One hundred ten patients were enrolled over 20 months, 55 in each group. The study cohort had a mean age of 31 years, and consisted of 79% male patients and 51% penetrating trauma victims. There was a significant difference in SBP observed during the study period (114 mm Hg vs. 100 mm Hg, $p < 0.001$). Injury Severity Score (19.65 ± 11.8 vs. 23.64 ± 13.8, $p = 0.11$) and the duration of active hemorrhage (2.97 ± 1.75 hours vs. 2.57 ± 1.46 hours, $p = 0.20$) were not different between groups. Overall survival was 92.7%, with four deaths in each group.

Conclusion: Titration of initial fluid therapy to a lower than normal SBP during active hemorrhage did not affect mortality in this study. Reasons for the decreased overall mortality and the lack of differentiation between groups likely include improvements in diagnostic and therapeutic technology, the heterogeneous nature of human traumatic injuries, and the imprecision of SBP as a marker for tissue oxygen delivery.

Key Words: Resuscitation, Hemorrhage, Hypotension, Trauma, Shock.

J Trauma. 2002;52:1141–1146.

Acta Anaesthesiol Scand 2012; ••: ••–••
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ACTA ANAESTHESIOLOGICA SCANDINAVICA
doi: 10.1111/j.1399-6576.2012.02763.x

Review Article

Fluid therapy in uncontrolled hemorrhage – what experimental models have taught us

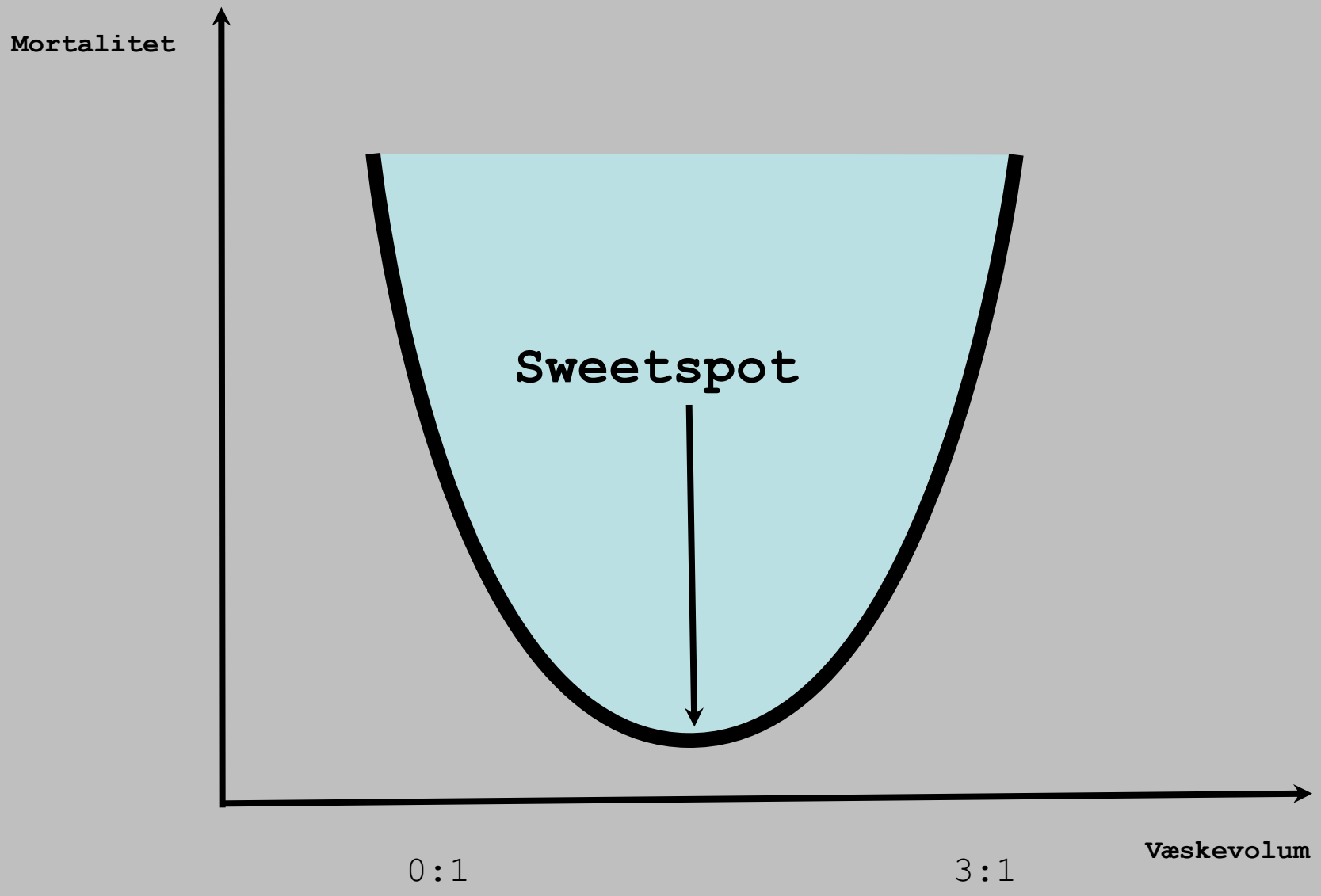
R. G. HAHN[1,2]
[1]*Anaesthesia and Intensive Care, Linköping University, Linköping, Sweden and* [2]*Research Unit, Södertälje Hospital, Södertälje, Sweden*

Intravenous fluid is life-saving in hypovolemic shock, but fluid sometimes aggravates the bleeding. During the past 25 years, animal models have helped our understanding of the mechanisms involved in this unexpected effect. A key issue is that vasoconstriction is insufficient to arrest the bleeding when damage is made to a major blood vessel. 'Uncontrolled hemorrhage' is rather stopped by a blood clot formed at the outside surface of the vessel, and the immature clot is sensitive to mechanical and chemical interactions. The mortality increases if rebleeding occurs. In the aortic tear model in swine, hemorrhage volume and the mortality increase from effective restoration of the arterial pressure. The mortality vs. amount of fluid curve is U-shaped with higher mortality at either end. Without any fluid at all, irreversible shock causes death provided the hemorrhage is sufficiently large. Crystalloid fluid administered in a 3 : 1 proportion to the amount of lost blood initiates serious rebleeding. Hypertonic saline 7.5% in 6% dextran 70 (HSD) also provokes rebleeding resulting in higher mortality in the recommended dosage of 4 ml/kg. Uncontrolled hemorrhage models in rats, except for the 'cut-tail' model, confirm the results from swine. To avoid rebleeding, fluid programs should not aim to fully restore the arterial pressure, blood flow rates, or blood volume. For a hemorrhage of 1000 ml, computer simulations show that deliberate hypovolemia (–300 ml) would be achieved by infusing 600–750 ml crystalloid fluid over 20–30 min or 100 ml of HSD over 10–20 min in an adult male.

Accepted for publication 31 July 2012

Væskebehandling

- **Kontekst-sensitiv volumeffekt !**
- Ringer/NaCl er mer effektiv volumekspander enn tidligere antatt: 60-75% i blodbanen etter 30 min
- Bolus vs kontinuerlig
- Ta hensyn til skademekanisme, skadeomfang og anatomisk lokalisasjon


Mortalitet
Sweetspot
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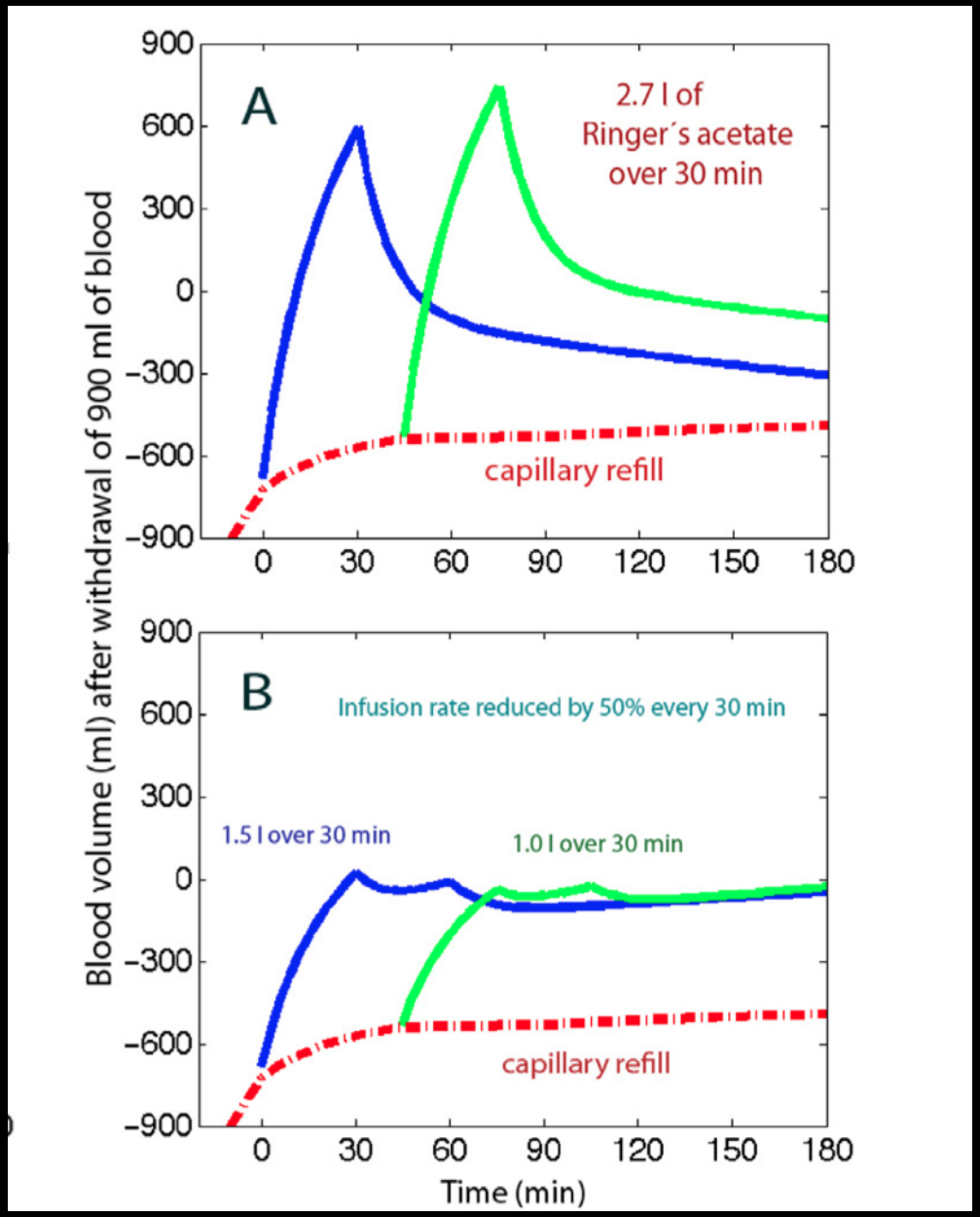
3:1
Væskevolum


A
2.7 l of
Ringer´s acetate
over 30 min
capillary refill
B
Infusion rate reduced by 50% every 30 min
1.5 l over 30 min
1.0 l over 30 min
capillary refill
Blood volume (ml) after withdrawal of 900 ml of blood
Time (min)
900
600
300
0
−300
−600
−900
0
30
60
90
120
150
180

Anestesi/smertelindring

- Redusert stressrespons, redusert adrenalinnivå
- Redusert oksygenforbruk
- Vasodilatasjon og bedret perfusjon ?

What do we need out there ?

Paramedics
Helicopters
Cars
Doctors
Motorbikes
Bicycles
Ambulances

A TEAM

And each member should be a team-player

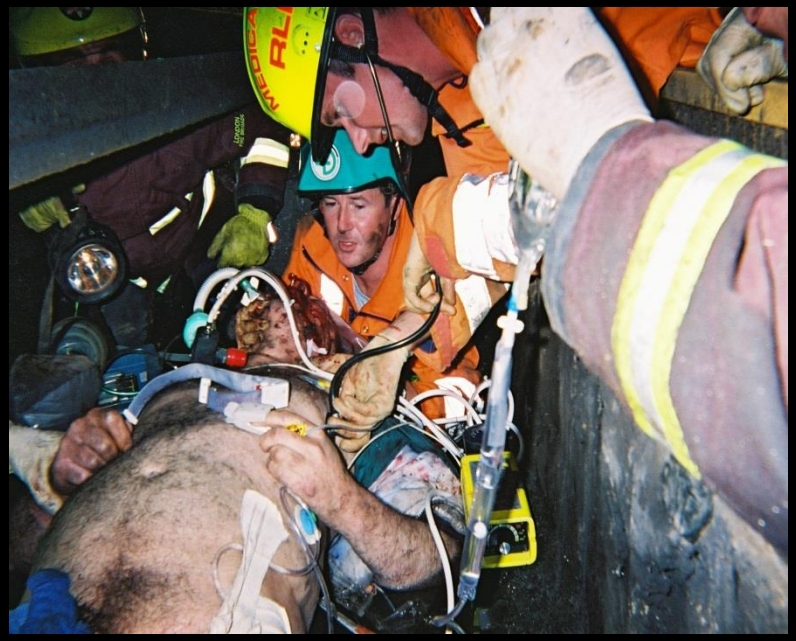
What is prehospital care

Bringing the emergency department to the patient....

TIDLIG DIAGNOSTIK + KORREKT TRIAGE

Advanced critical emergency care on the road side.....

Vanskeligt.... ?



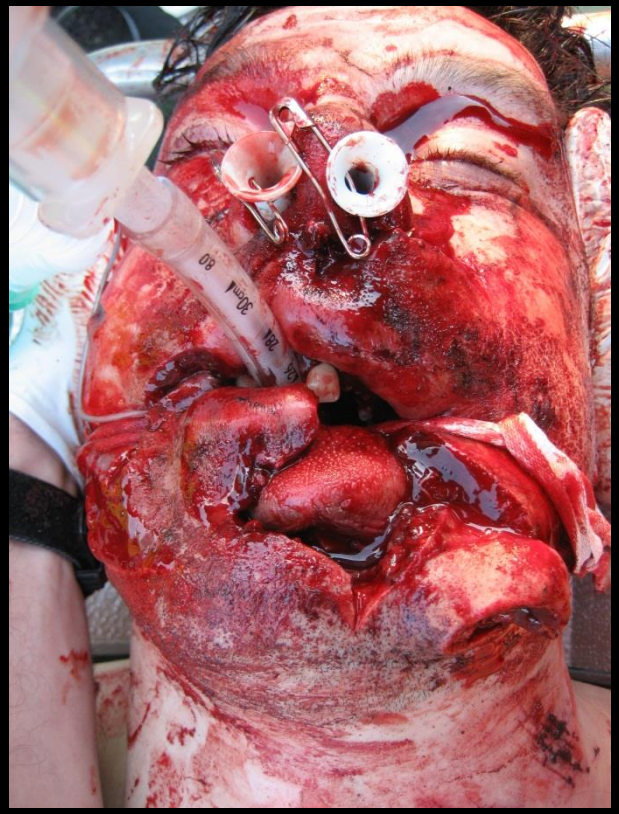
JA !

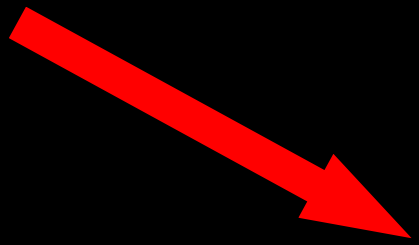
New concept ?

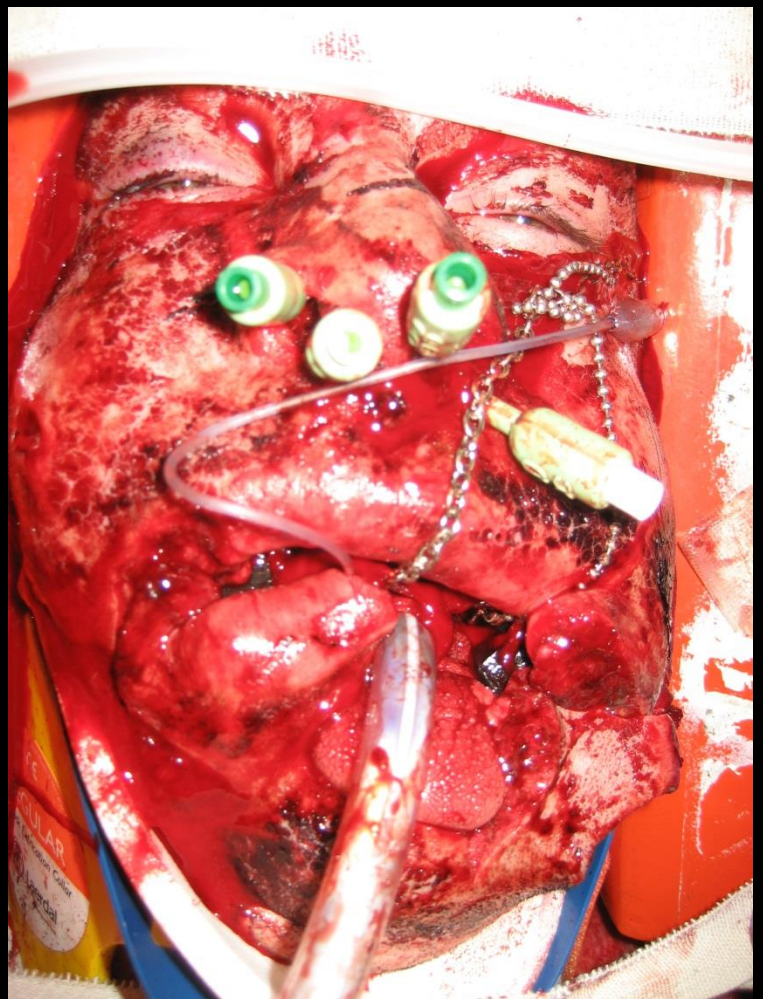
Cause

Condition

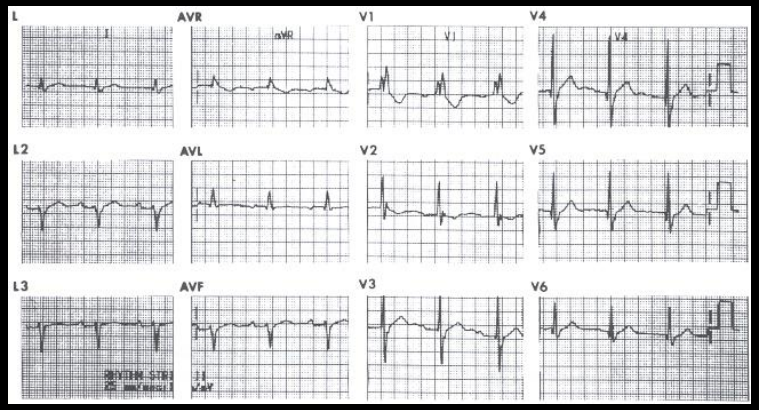
Complications







Mrs Smith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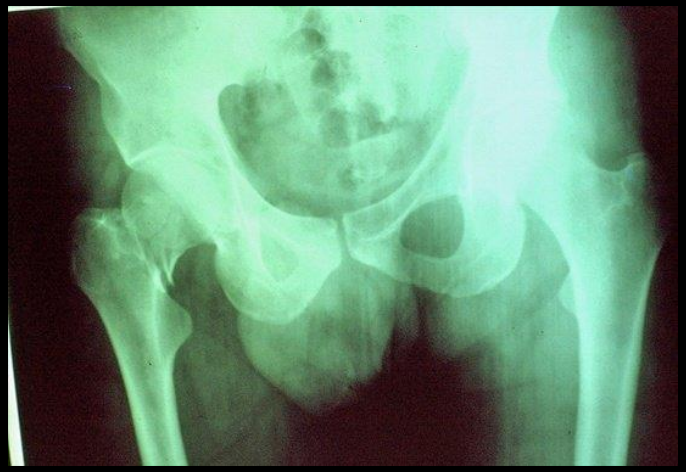
V5
L3
AVF
V3
V6




MESSIAS
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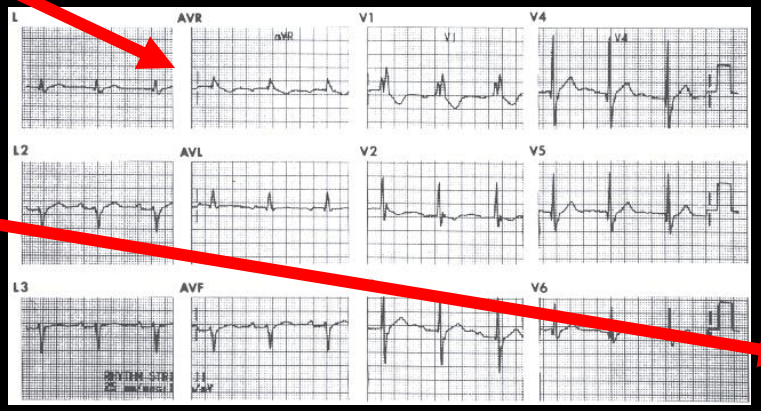
DOS VINHOS MESSIAS S.A.
V.N. DE GAIA - PORTUGAL
PRODUCT OF PORTUGAL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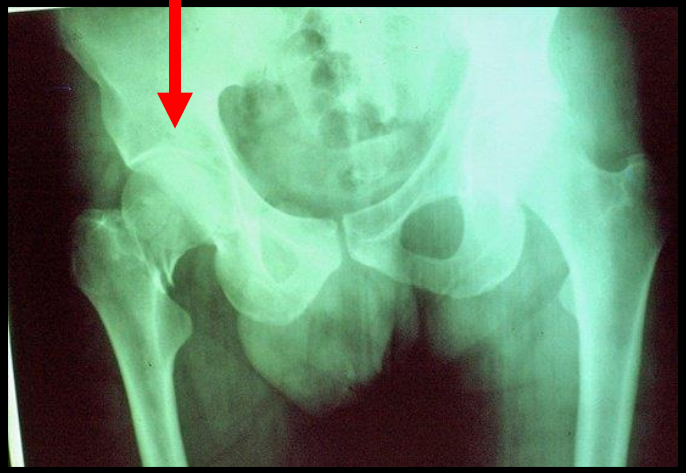
AVF
V6




MESSIAS
PORT
RUBY


(Gen)Kender du ham ?
Agitated
Reduced GCS
Combative
Drunk
Smells
Unpleasent friends
Urinates

Undifferentiated!

Agitated

Combative

Reduced GCS

- Trauma - SDH, EDH
- Toxicologic - alcohol, other
- Metabolic - hypoglycaemia, hyponatraemia
- Psychiatric
- Septic
- Oncologic

- Diagnostic skills
- Sedation
- Airway management, anaesthesia
- Mental health law

Smells

.....essentiel rolle i beslutningerne omkring denne patient

Structured approach

COMMON LANGUAGE

ABCDE

Need for a simple and common system

Prevents ”fuck ups”

Provides us with a common language

- In-hospital and pre-hospital
- Doctors, paramedics. nurses

Systematic

Primary Trauma Care Foundation

Definitive traume care course

Advanced trauma Life support - ATLS

European Trauma Course

Emergency Management of Severe Trauma EMST

Prehospital Trauma Life support - PHTLS

A B C D E

Airway

Breathing

Circulation

Disability

Exposure (”the rest”)

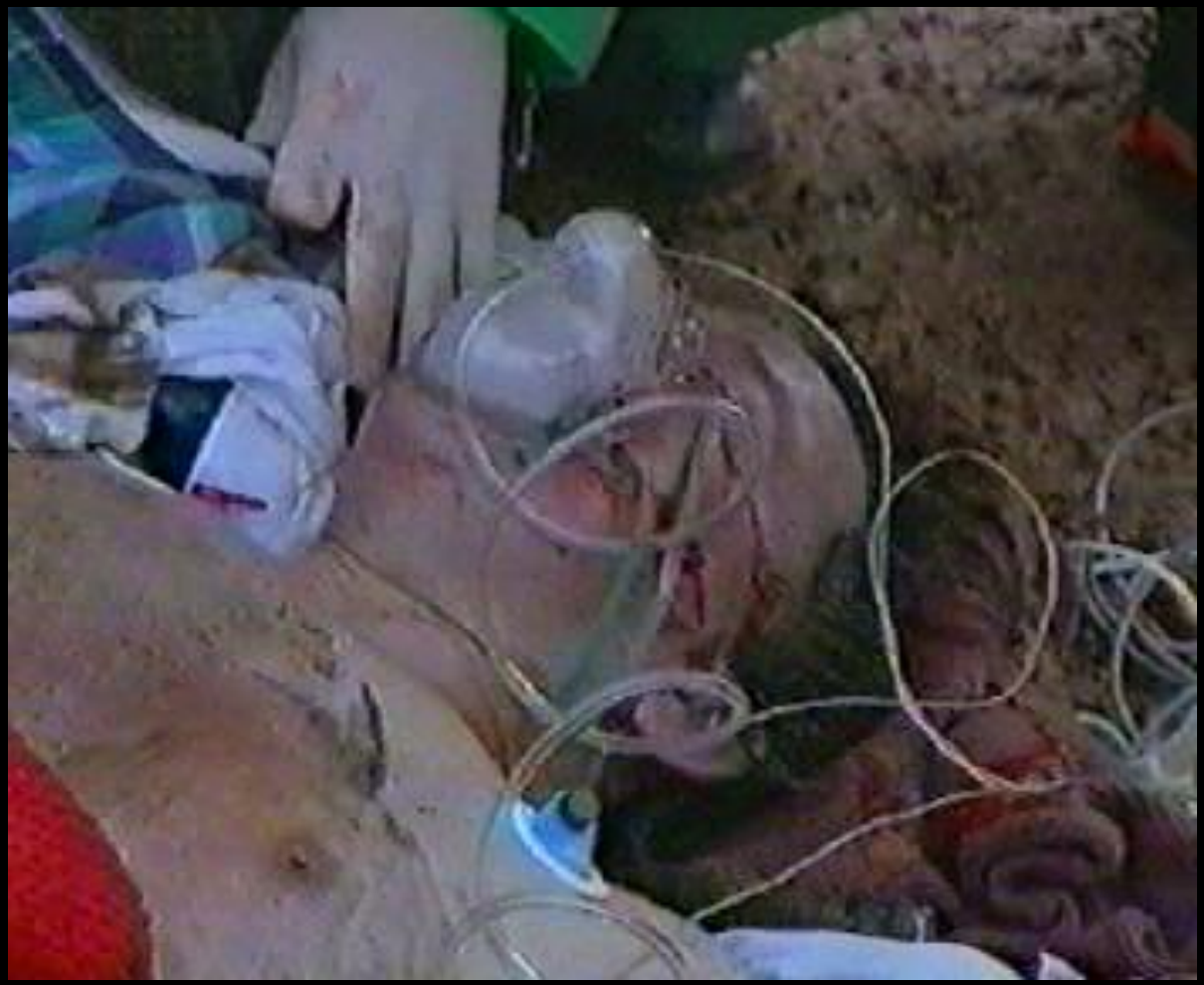
Airway

Se på patienten
Tal med patienten
Sekret
Slim
Blod

SpO2 (pulsoximetry)
EndTidal CO2

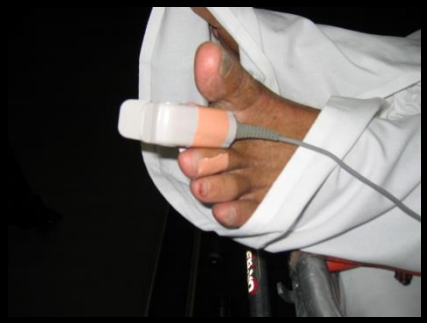
Breathing

Breathing



Breathing

SpO_2 (pulsoximetry)
EndTidal CO_2

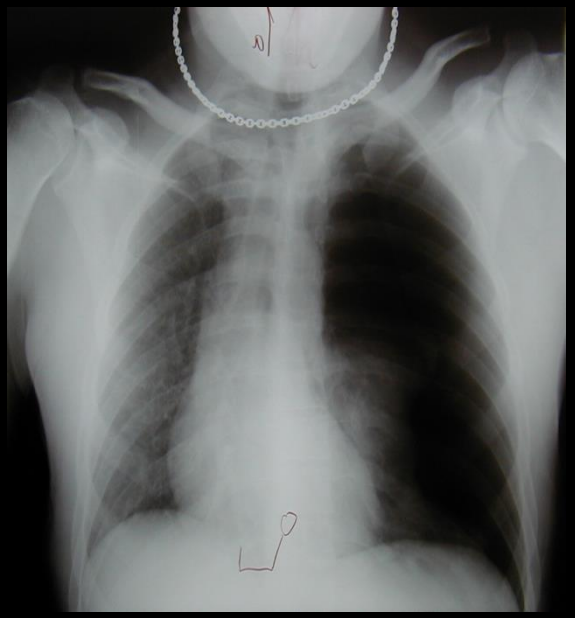


Skin color
Respiratory rate !!!
Symmetrical or not
Stressed or not
Accessory muscles
Pneumothorax

Ultrasound ????
Arterial blood gas ???

Thorax

- Respirasjons bevegelse
  - normal?
  - Symetrisk?
- Respirasjonslyder
  - normale?
  - sjekk før transport
- Subcutant emfysem
- Costafracturer
- Flail chest
- Kontusjonsmerker
- Luftveistrykk (intuberte)
- $ETCO_2$



Circulation

CNS

BP
Heart rate
CRT – value
Skin - moisture
ECG – 3 or 12 lead
EndTidal CO_2

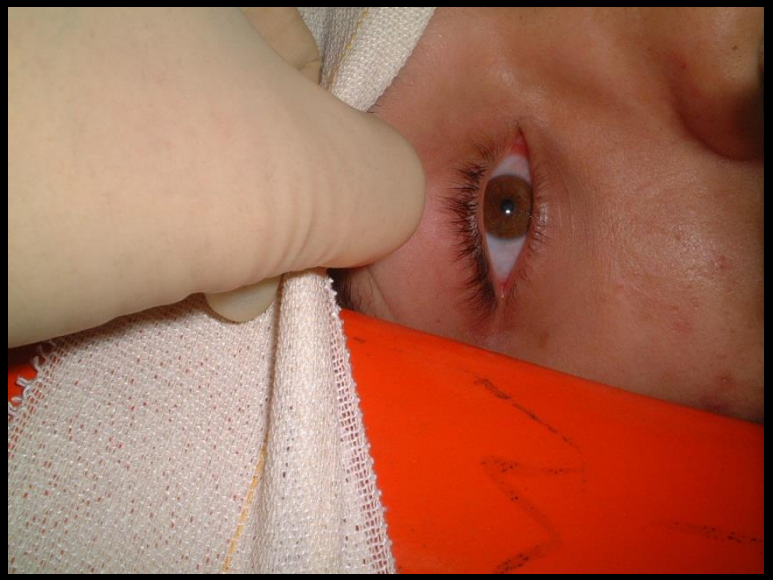
Ultrasound ????

Arterial blood gas ??

Disability

GCS (Glasgow Coma Scale Score)

Pupils



Blood glucose

DEFG = Don't Ever Forget Glucose

Motor score of GCS

Nevrologisk status

- Er pas. våken og klar?
- Urolig?
  - Årsak?

HYPOXI ?????

- Bevegelse i ekstremitetene?
  - spontant?
  - god kraft?

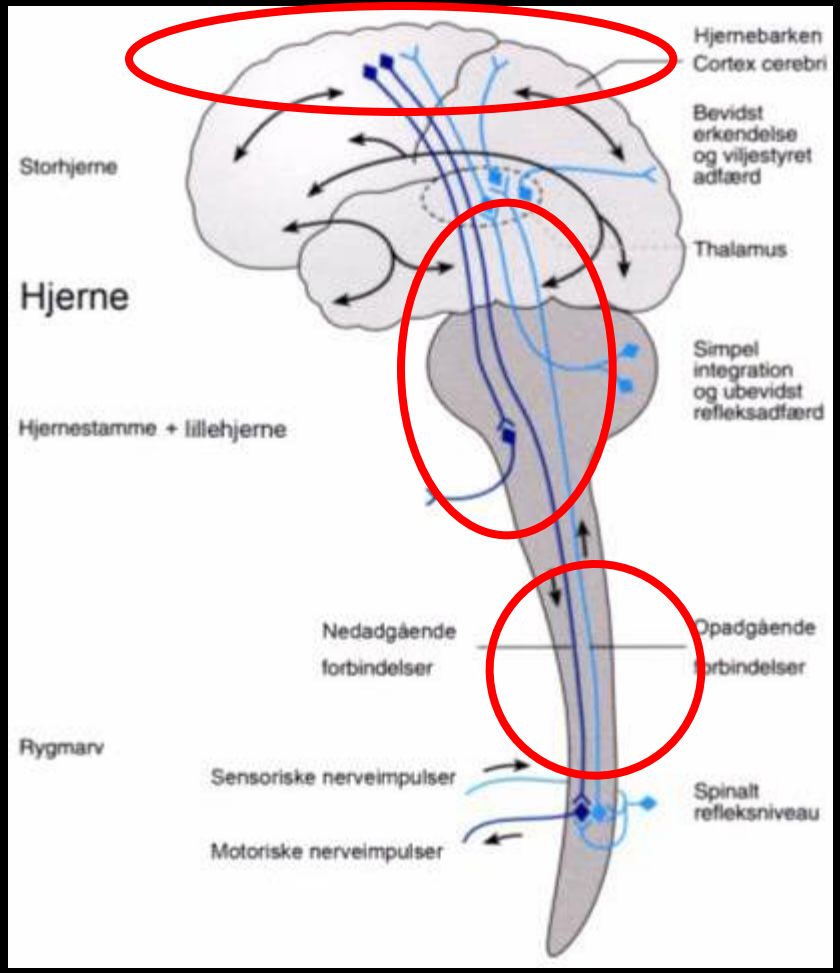
- Tilstand før vår ankomst?
- Smerter?
- GROV neurologi

- Husk pasientdata!

GCS

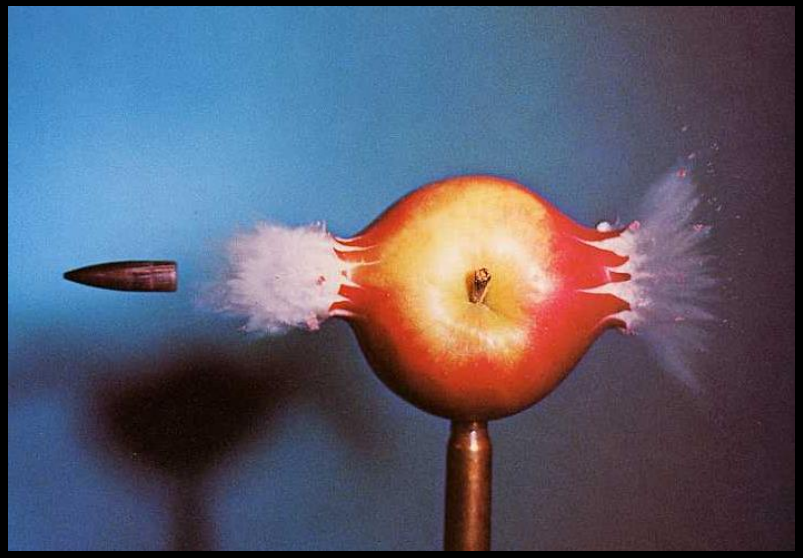
Hvad er det egentlig som testes ?

HJERNEFUNKTION



Ett tidspunkt - én verdi

Single GCS observation = øjebliks billede



Hvordan scores en ordentlig GCS

GCS

Motorisk respons

Verbal respons

Åpning av øyne

HUSKELISTE er lov

Glasgow coma scale

		Score
Eye opening	spontaneously	4
	to speech	3
	to pain	2
	none	1
Verbal response	orientated	5
	confused	4
	inappropriate	3
	incomprehensible	2
	none	1
Motor response	obeys commands	6
	localises to pain	5
	withdraws from pain	4
	flexion to pain	3
	extension to pain	2
	none	1
Maximum score		15

A V P U

A Alert

V Verbal

P Pain

U Unresponsive

Barne GCS

Barne GCS

ØJNE og MOTOR score er det samme……

VERBAL:

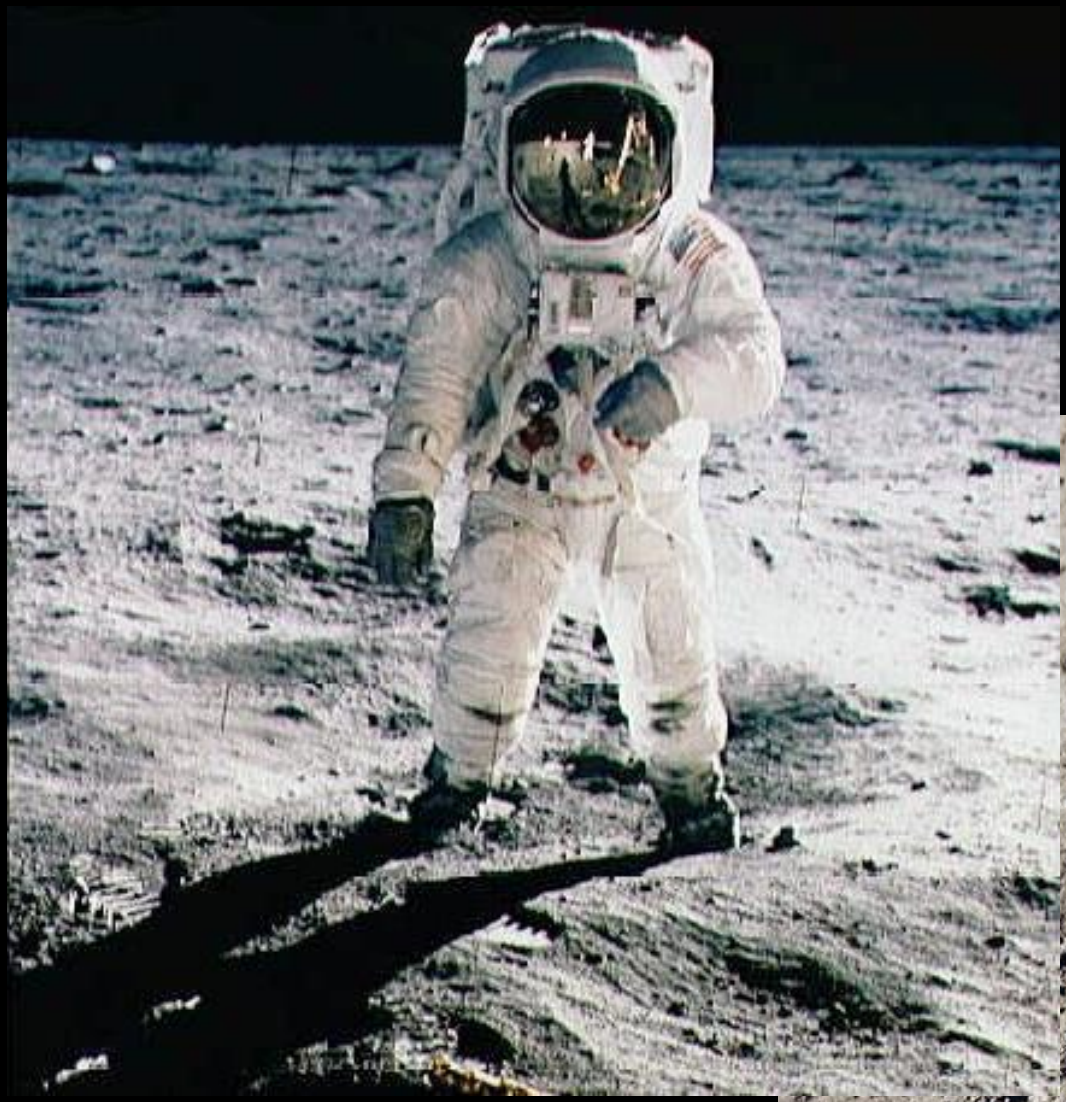
1 = No vocal response
2 = Inconsolable, agitated
3 = Inconsistently consolable, moaning
4 = Cries but is consolable, inappropriate interactions
5 = Oriented to sounds, follows objects, interacts

Beskriv hvad du ser

2 ”giant steps”

3
4
5
6
7
8
9
10
11
12
13
14
15

Glasgow Coma Scale



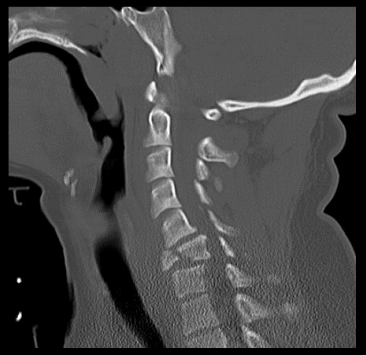
3

4
5
6
7
8
9
10
11
12
13
14

15

!

Spinal cord injuries



Neurogenic shock

Cord injury above T5 (sympathectomy below level of injury)

Bradycardia: HR 50-60

Hypotension: SBP 90-100 mmHg

unopposed vagal stimulation +

loss of sympathetic vascular tone

Loss of thermoregulation:

Loss of ability to redirect blood from the periphery to the core

Priapism

Motor and Sensory function

Exposure / Environment

Expose patient

But....Protect from environment

Fractures

Temperature

Check axilla + groin for wounds (+ anal)

Mand stukket i røven

Blev overfaldet på Christiania, men tilkaldte først hjælp, da han var kommet ind til Rådhuspladsen

13:27 - 18. apr. 2010 | Kristina Isabella Bergmann

Det var en noget omtumlet mand, Københavns Politi blev mødt af, da de blev kaldt ind til Rådhuspladsen tidligt søndag eftermiddag. Manden havde været udsat for vold i en noget ydmygende form, nemlig ved at få stukket en kniv i røven. Overgrebet skulle angiveligt have fundet sted på Christiania, hvor manden havde befundet sig i tiden op til overfaldet.

Mand stukket med kniv i røven på Christiania. (Foto: Ole Steen)

Gennem København med blødende røv

Da manden var blevet stukket i sin ene balde, tænkte han ikke på at ringe til hverken politi eller ambulancer. I stedet tog han turen ind til

3 Golden ?'s

1. Where does it hurt ?

2. Does it hurt anywhere else ?

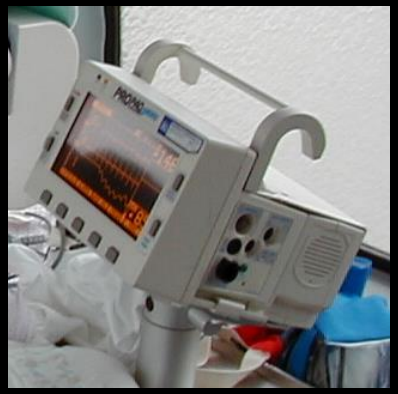
   (now concentrate)

3. Does your breathing feel normal ?

Documentation

Medical record: Legal document

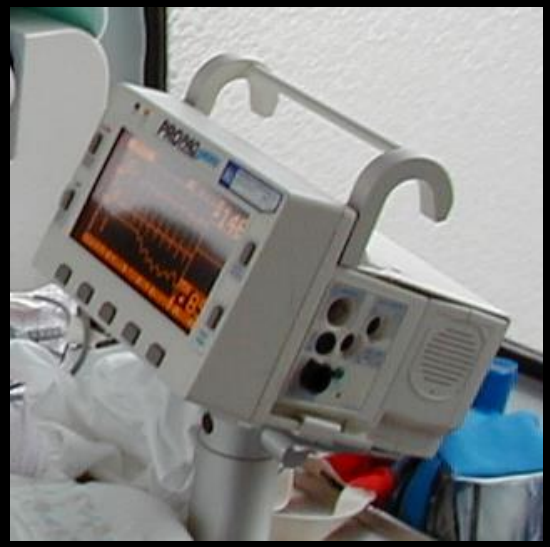
Personal details
Objective findings
Physiological values
Treatment
Response to treatment
Times



Monitorering

Nær sammenhæng med diagnostik

Gjentatt klinisk vurdering (se på, ta på)
BT (NIBP, evt invasivt)
EKG, pulsfrekvens, rytme
SpO2
Respirasjonsfrekvens, luftveistrykk
EtCO2 på intuberte
Pupiller, gjentatt GCS, bevegelse
Evt temp



Handover

Interface between PHC and hospital

One and only chance

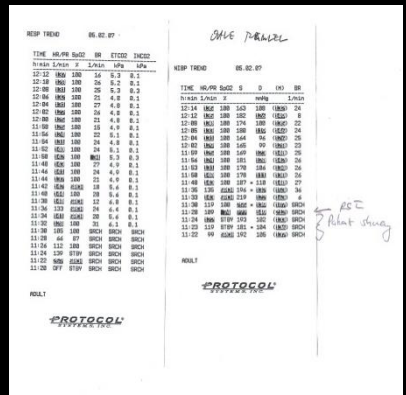
CLEAR and STRUCTURED

NO talking when moving patient....

Don’t be a Muppet….

”Control the scene”

Monitor print-out



Remember

"Pay attention to the detail"

Gareth Davies, Medical Director of HEMS-London

Summary

Structured approach

ABCDE

Monitor the patient

Documentation

Handover


The escape was meticulously planned and perfectly executed