



Congresso Nazionale
Rimini
Palacongressi
18-21 Ottobre
2012

Cons. pregressuali
18 Ottobre 2012



SALA DELLA MARINA 9.00 - 13.30

TRAUMA CRANICO

Moderatori: M. Chiesa, A. M. Ferrari

- 9.00 Il medico d'urgenza e il trauma cranico lieve
A. Fabbri
- 9.20 Trauma cranico severo: la gestione
G. Bini
- 9.40 Neuromonitoring
D. Payen
- 10.00 Indicazioni neurochirurgiche
C. Iaccarino
- 10.20 Discussione

Trauma cranico grave: la gestione

Dott. Giovanni Bini

Neurorianimazione - Ospedale Bufalini - Cesena

Centro Traumi Romagna

Cosa è un «Trauma cranico grave»?

GCS 3-8
«in coma»

JOURNAL OF NEUROTRAUMA
Volume 24, Supplement 1, 2007
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MANAGEMENT AND PROGNOSIS OF SEVERE TRAUMATIC BRAIN INJURY

**Part I: Guidelines for the Management of Severe
Traumatic Brain Injury**

**Part II: Early Indicators of Prognosis in Severe
Traumatic Brain Injury**

The Brain Trauma Foundation also gratefully acknowledges the following members of the Review Committee and the professional societies they represent:

Elenco facile

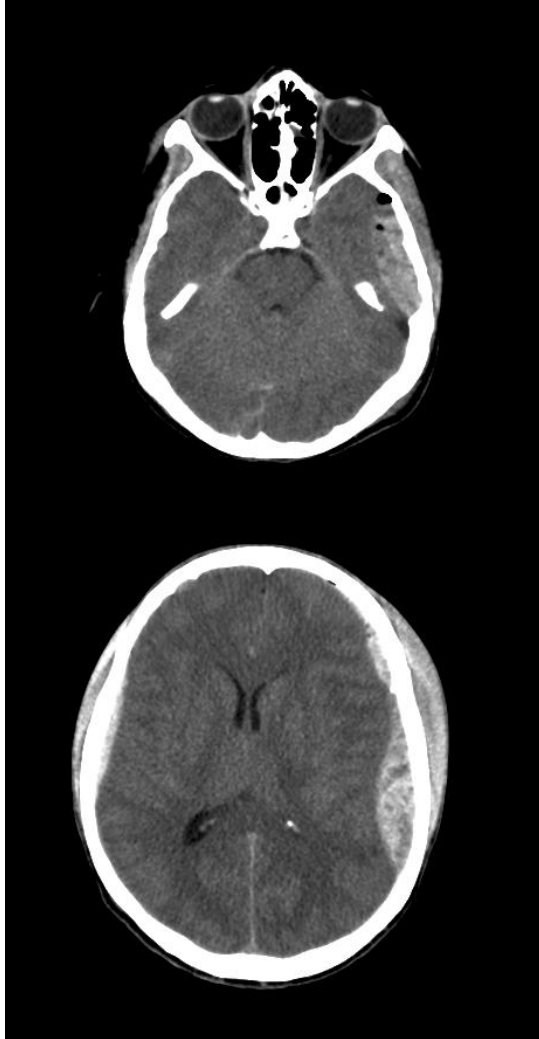
PART I: GUIDELINES FOR THE MANAGEMENT OF SEVERE TRAUMATIC BRAIN INJURY

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E' un trauma cranico grave?

preH GCS 14



15 gg di ICU

preH GCS 6



Estubato il I gg

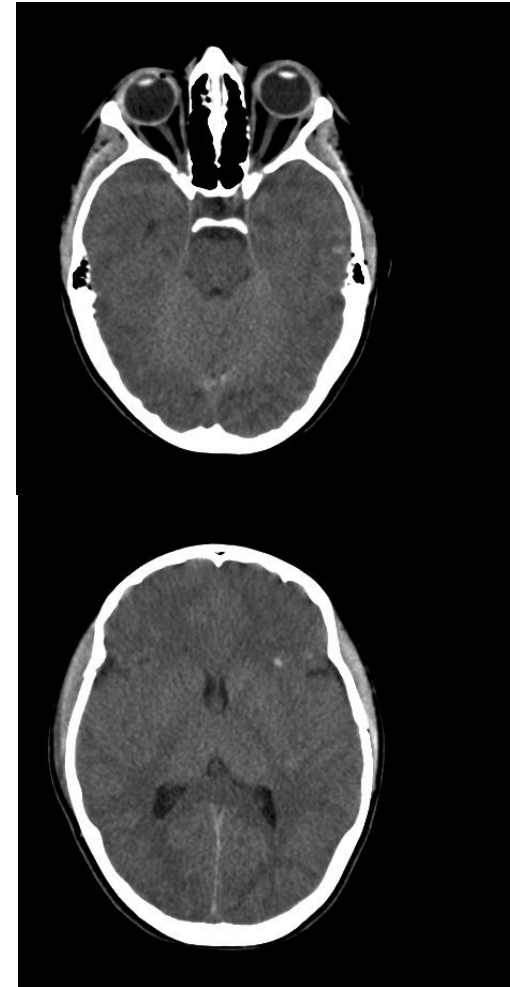
E' un trauma cranico grave?

preH GCS 12



6 gg di ICU

preH GCS 6



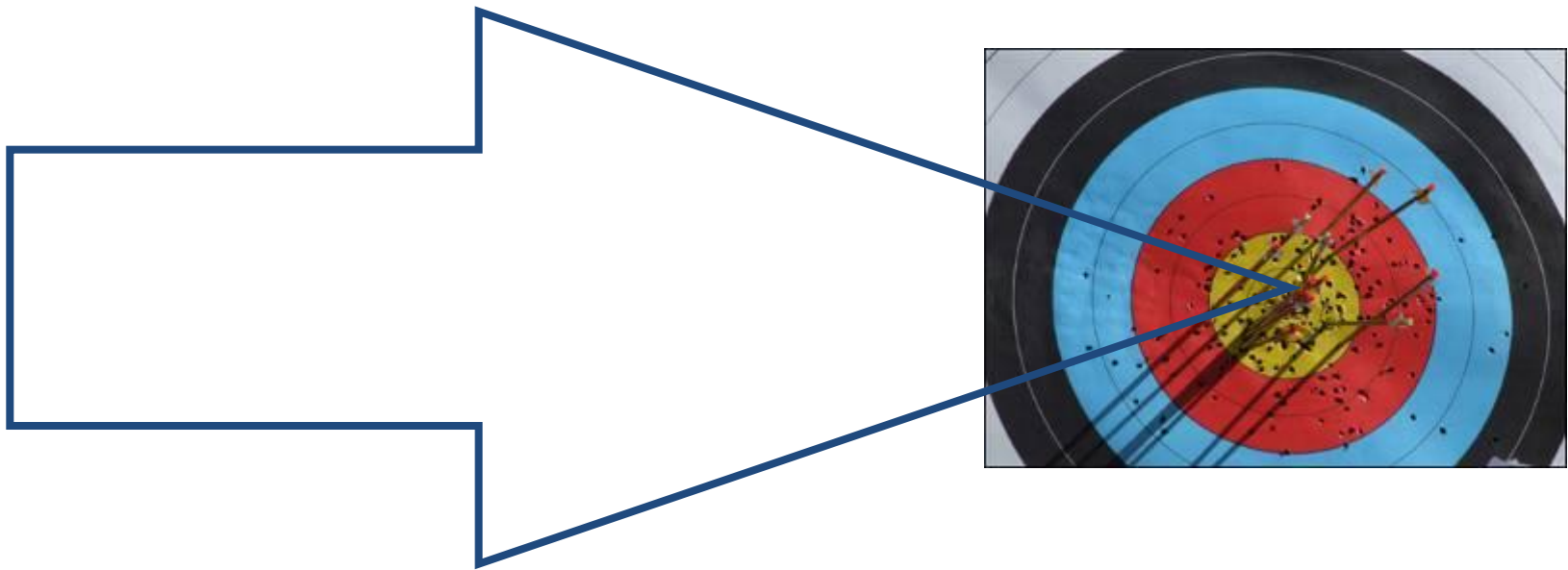
20 gg di ICU

Trauma cranico grave...

È quello che richiede una gestione
NEUROPROTETTIVA
cioè di prevenzione del danno secondario

È un paziente in cui c'è un danno cerebrale
ma ci sono **neuroni salvabili**

C'è una **evolutività** che merita attenzione e cura



Obiettivi per un medico d'urgenza nel trauma cranico grave (o potenzialmente evolutivo)

1. Omeostasi, neuroprotezione
2. Precoce riconoscimento di patologie tempo dipendenti
3. Smascherare i falsi gravi e i falsi moderati (pitfalls)

Omeostasi - Neuroprotezione

ABCDE della neuroprotezione: omeostasi

A normossia

B normocapnia

C normotensione

D sedazione

E normotermia

Danno ischemico secondario: cause sistemiche ed intracraniche

Ipossia

arresto respiratorio
ostruzione vie aeree
aspirazione
pnx - emotorace
contusione polmonare

Ipotensione

shock emorragico
IMA
tamponamento cardiaco
pnx iperteso
shock spinale

Anemia

Ipertermia

Ipo-iperglicemia

Ipercapnia

Iperensione intracranica

Ematoma intracranico

Edema cerebrale

Iperemia

Vasospasmo

Convulsioni

Ridotta
disponibilità
di ossigeno

O₂

Aumento
delle
richieste
energetiche

*Danno ischemico
secondario*

A

AIRWAY

Obiettivo:

vie aeree pervie

normossia

Criticità:

non protezione delle vie aeree

ipossia

O₂ ad alti flussi

IOT se GCS < 9 (migliora l'outcome)

IOT se GCS < 12 sulla scena (riduce le complicanze)

IOT preferita a IRT

per IOT preferire farmaci a breve durata d'azione

B

BREATHING

Obiettivo:

normocapnia

Criticità:

ipo - iper - capnia

i comatosi vanno ventilati

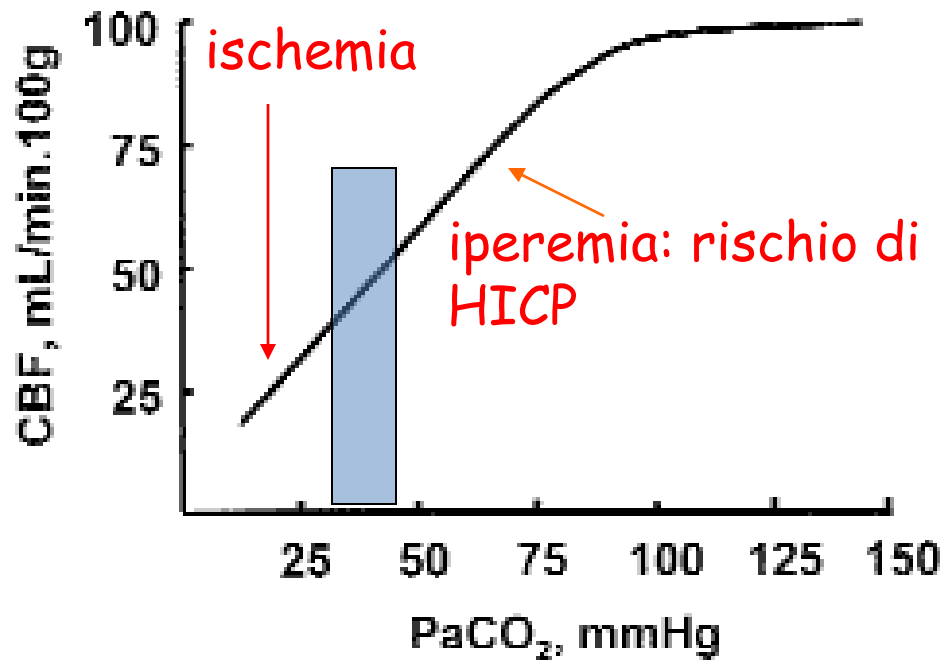
consigliato monitoraggio ETCO₂

VENTILAZIONE



Le false sicurezze del saturimetro

CBF - PaCO₂

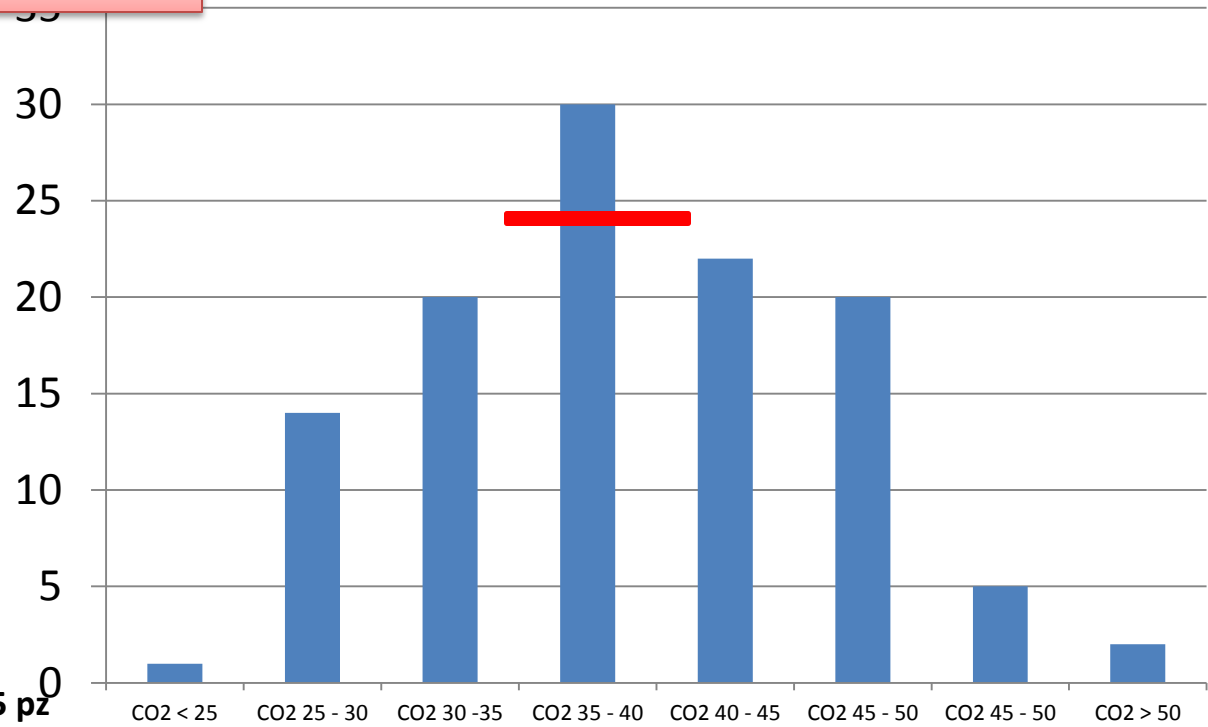


Range di normalità di flusso: PaCO₂ 35 - 45

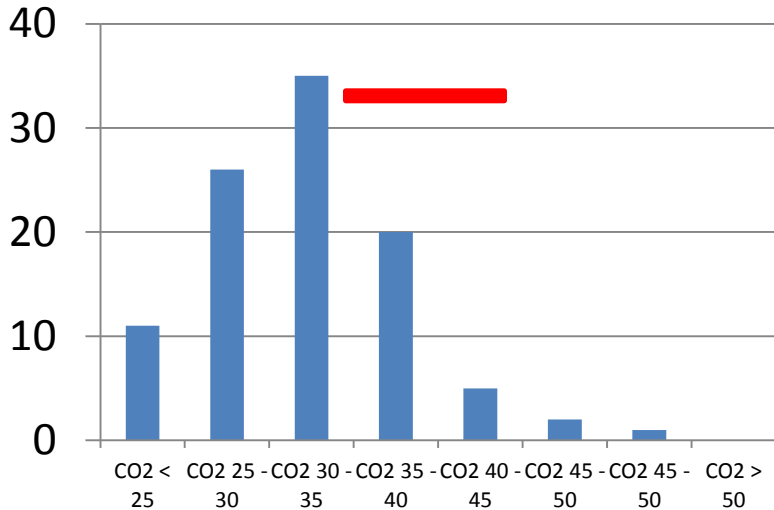
Omeostasi - Neuroprotezione

Pazienti intubati con trauma e emorragia cerebrale: EGA appena giunti al PS dall'ambulanza. 2063 paz

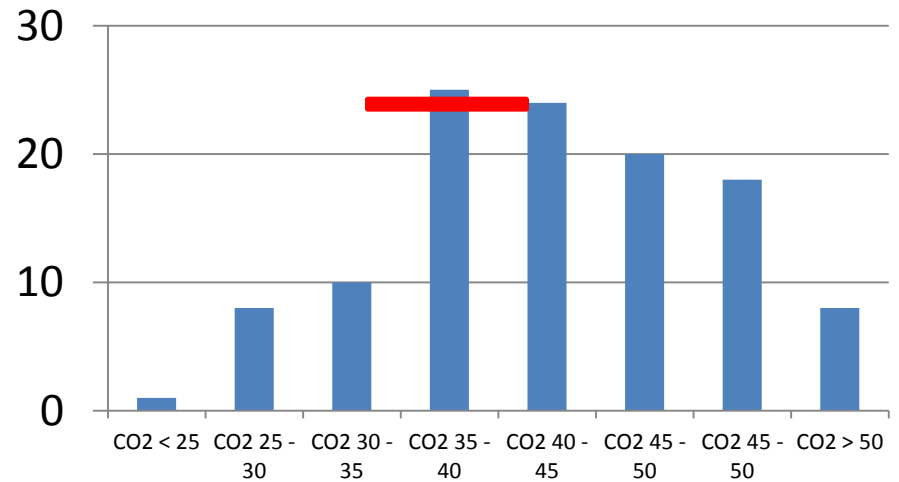
EGA PS tutti 2063 paz



EGA PS (va e vieni) 355 paz



EGA PS (respiro spontaneo) 201 paz



VENTILAZIONE

VENTILAZIONE MECCANICA

SEMPRE

MAI IN RESPIRO SPONTANEO
o con ventilazione spontanea assistita
con va e viene



IPERVENTILAZIONE

Solo in caso di coma con anomalie pupillari
In associazione con altre terapie

C

CIRCULATION

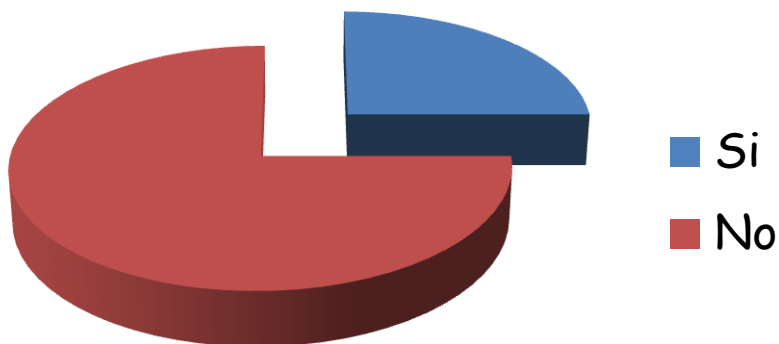
Obiettivo:
normotensione

Criticità:
ipotensione

Obiettivo della PAS nel trauma cranico

PAS > 110 - 120 mmHg (PAM > 90: CPP 70 con ICP 20)

Ipotensione pre H



931 traumi primari Cesena 2001-2012		
IPOTENSIONE preH		PS
Tot	25%	18%
HI	8%	7%
Poli HI	31%	22%

Traditional systolic blood pressure targets underestimate hypotension-induced secondary brain injury

Megan Brenner, MD, MS, Deborah M. Stein, MD, MPH, Peter F. Hu, MS, Bizhan Aarabi, MD, Kevin Sheth, MD, and Thomas M. Scalea, MD, *Baltimore, Maryland*

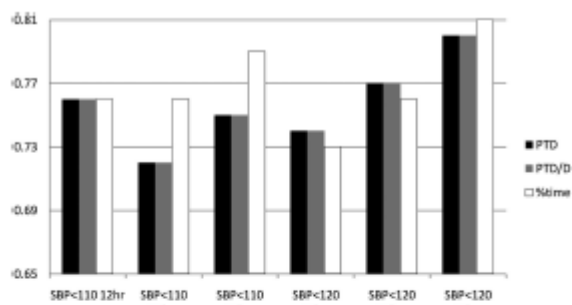


Figure 1. Mortality predicted by ROC for SBP <110 mm Hg and <120 mm Hg at 12 hours, 24 hours, and 48 hours.

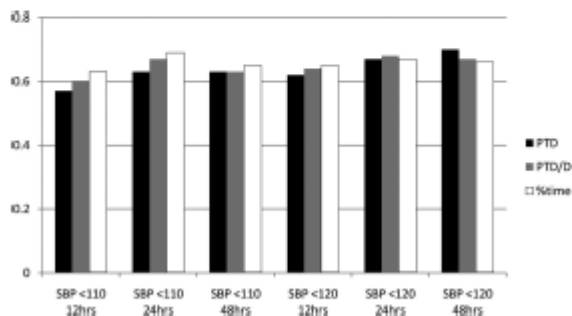


Figure 2. Twelve-month GOSE predicted by ROC for SBP <110 mm Hg and <120 mm Hg for 12 hours, 24 hours, and 48 hours of admission.

J Trauma
Volume 72, Number 5

CONCLUSION

Within the first 48 hours of ICU admission, hypotension was not predictive of outcomes at traditionally defined thresholds of SBP <90 mm Hg or <100 mm Hg. Systemic blood pressure targets >120 mm Hg may be more efficacious in minimizing secondary insults and particularly useful in settings without invasive intracranial monitoring capabilities.

C

CIRCULATION

Obiettivo:

normotensione

Criticità:

ipotensione

Obiettivo della PAS nel trauma cranico

PAS > 110- 120 mmHg (PAM > 90: CPP 70 con ICP 20)

Cristalloidi o colloidi isotonici

Evita ipotoniche (glucosata 5%)



normo osmolarità

E la glicemia?

Crit Care Med 2008 Vol. 36, No. 12

Neurologic Critical Care

Impact of tight glyceemic control on cerebral glucose metabolism after severe brain injury: A microdialysis study*

Mauro Oddo, MD; J. Michael Schmidt, PhD; Emmanuel Carrera, MD; Neeraj Badjatia, MD, MS; E. Sander Connolly, MD; Mary Presciutti, RN; Noeleen D. Ostapkovich, MS; Joshua M. Levine, MD; Peter Le Roux, MD; Stephan A. Mayer, MD, FCCM

CLINICAL INVESTIGATION

Intensive Insulin Therapy Increases the Risk of Hypoglycemia in Neurocritical Care Patients

Charlotte A.M. Tiemessen, BSc,† Cornelia W.E. Hoedemaekers, MD,†
Freya M. van Iersel, MD,*† Gerrit H.J. Rösken, MD,*† Johannes van der Hoeven, MD,†
Geert-Jan Biessels, MD,‡ and Arjen J.C. Slooter, MD**

Background: Intensive insulin therapy protocols are widely used in intensive care medicine. A disadvantage of these protocols may be the occurrence of hypoglycemic episodes. Neurocritical

Key Words: intensive insulin therapy, effects, protocol, neurocritical care patients, intensive care, hypoglycemia

(*J Neurosurg Anesthesiol* 2011;23:206–214)

Intensive Insulin Therapy in Brain Injury: A Meta-Analysis

Syed Nabeel Zafar,¹ Aftab Iqbal,² Mauricio F. Farez,³ Suyog Kamatkar,⁴ and Marc A. de Moya⁵

Target: 180 - 215

REVIEW ARTICLE

Glycemia Management in Neurocritical Care Patients A Review

*Federico Bilotta, MD, PhD, Federico Giovannini, MD, Remo Caramia, MD,
and Giovanni Rosa, MD*

Target: 80 - 155
100 - 200

Abstract: Intensive research investigating the relation between the management of glycemia and outcome in patients receiving neurocritical care has underlined the possible benefits and adverse events related to glucose control. Here, we review experimental and clinical studies investigating the effects of hypoglycemia and hyperglycemia on the brain that advance current knowledge on managing glycemia in patients receiving neurocritical care.

Key Words: glycemia management, neurocritical care patients, hypoglycemia, hyperglycemia

(*J Neurosurg Anesthesiol* 2009;21:2–9)

The importance of blood glucose concentrations during acute cerebral injury and their effects on the brain were first described in 1977 by Myers and Yamaguchi⁵ who reported that the infusion of a glucose solution instead of saline in experimental animals, before cerebral ischemia, was related to a significantly worse postischemic outcome. Several papers have retrospectively described that, in patients with acute cerebral injury, hyperglycemia is related to a poor long-term neurologic outcome.^{3,6,7} Recent advances have shown that intensive insulin infusion and strict glycemia control is related to lower morbidity and mortality in postcardiac surgery patients.^{8,9} Preliminary results in limited groups of patients with brain injury suggest that strict glycemic control

D

DISABILITY

Obiettivo:
sedazione

Criticità:
segni di HICP

VALUTA: GCS e pupille

SEDAZIONE

per buon adattamento al respiratore

farmaci a breve durata d'azione e in infusione continua

*Ma perché sedare
uno...già in coma?*



Razionale della sedazione in pazienti con neurolesione

- Controllo ICP
- Riduzione del CMRO₂; metabolic suppression; neuroprotezione
- Prevenzione e trattamento delle crisi epilettiche precoci

E

EXPOSURE

Obiettivo:
normotermia

Criticità:
ipotermia

ipotermia

acidosi

coagulopatia

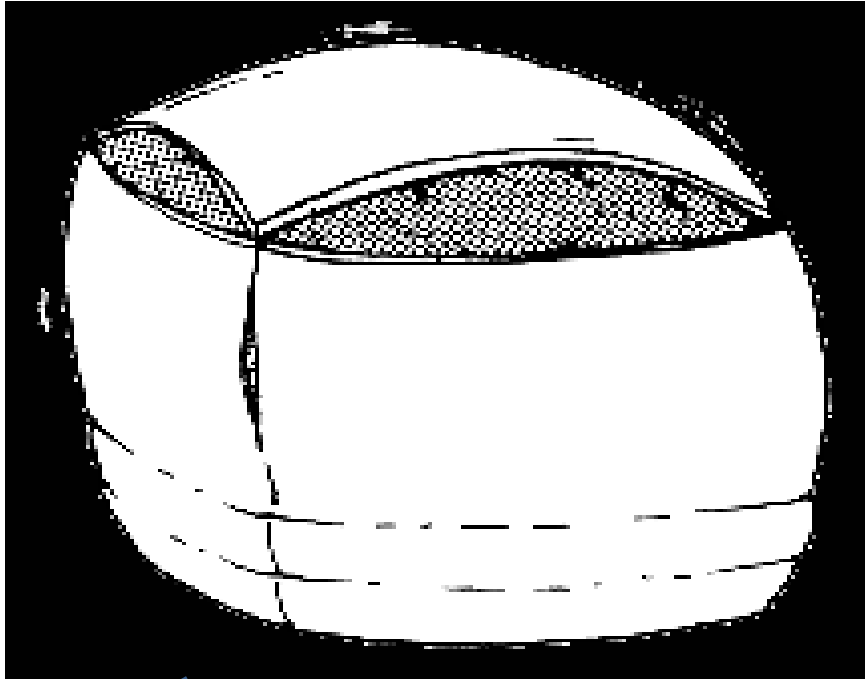
esami di
laboratorio
della
coagulazione

BE
lattati

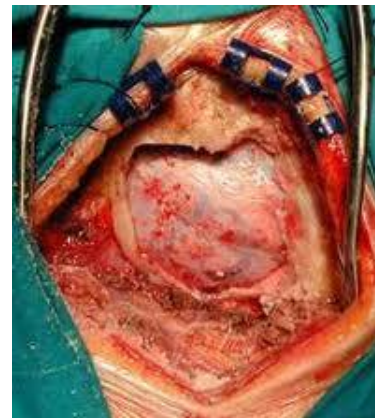
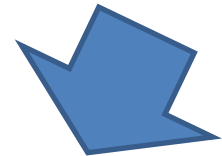
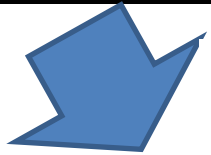
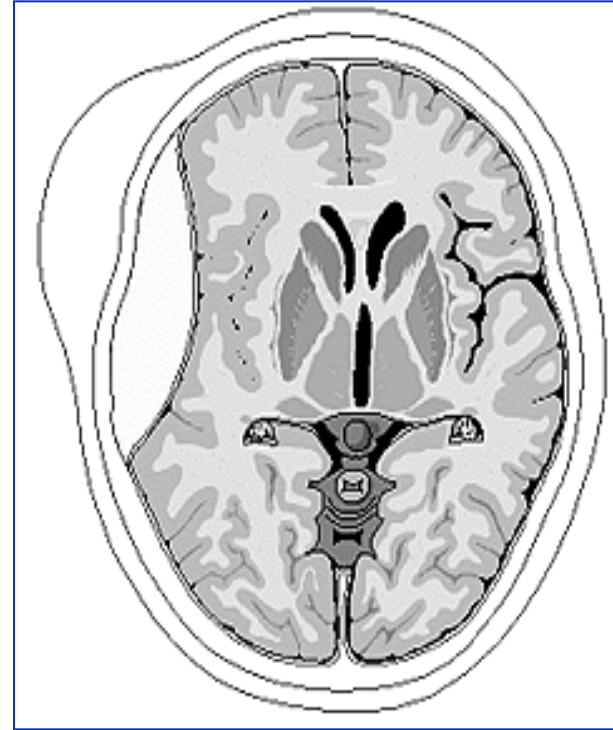
++ PREVENZIONE++

Precoce riconoscimento di patologie tempo-dipendenti

ICP increasing ?



Mass evolving ?



Ipertensione endocranica

GCS motorio	Monitoraggio ICP	HICP	Assenza di monitoraggio ICP	Sospetta HICP
1	54	28 (52%)	88	20 (23%)
2	27	19 (70%)	46	7 (15%)
3	36	18 (50%)	28	4 (14%)
4	55	28 (51%)	60	10 (17%)
5	75	41 (55%)	141	8 (6%)
6	30	19 (63%)	107	4 (4%)
Non valutabile	33	24 (73%)	27	9 (33%)
Non registrato	34	13 (38%)	68	7 (10%)
Totale	344	190 (55%)	565	69 (12%)

...non solo *GCS*...



Glasgow Coma Scale: limiti

In vari studi emerge una ampia variabilità inter operatore

Può essere utilizzata parzialmente, considerando il solo score motorio nei pazienti intubati, in cui non è valutabile la risposta verbale

Deve essere valutata ad avvenuta metabolizzazione dei farmaci utilizzati

Può essere inficiato da abuso di sostanze stupefacenti, da alcool, o da quadro di ipossia, ipercapnia, shock emorragico.

Va quindi calcolato a rianimazione effettuata, cioè al raggiungimento della stabilità emodinamica e ventilatoria

C'è un grosso rischio di inaccuratezza del primo GCS

Classification of a traumatic brain injury: the Glasgow Coma scale is not enough

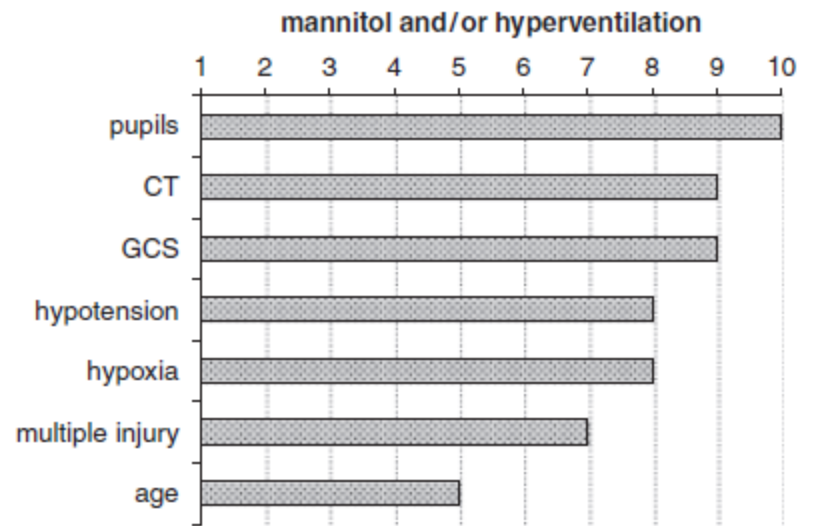
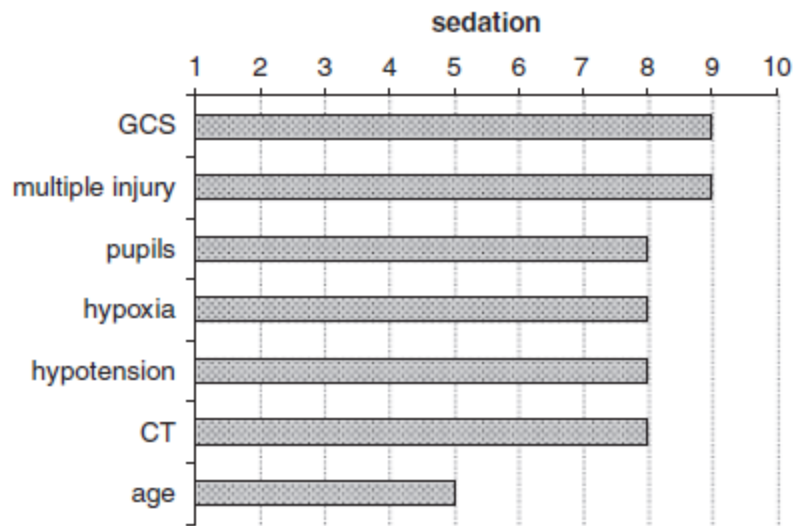
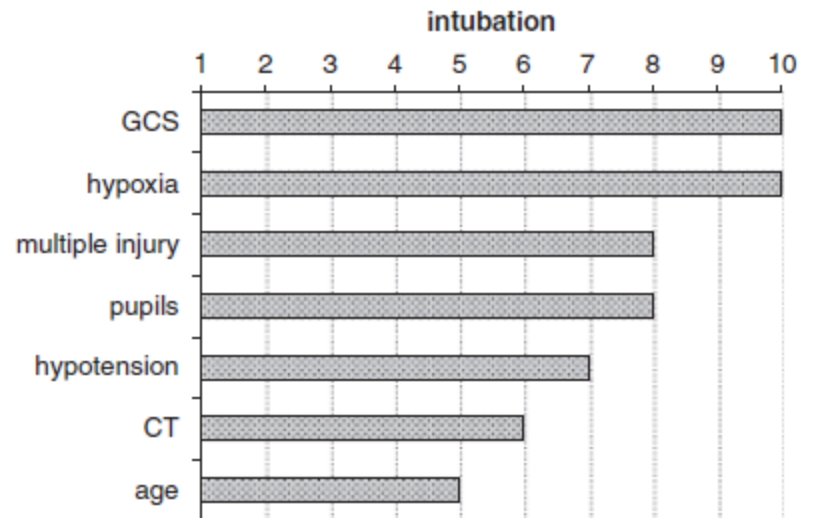
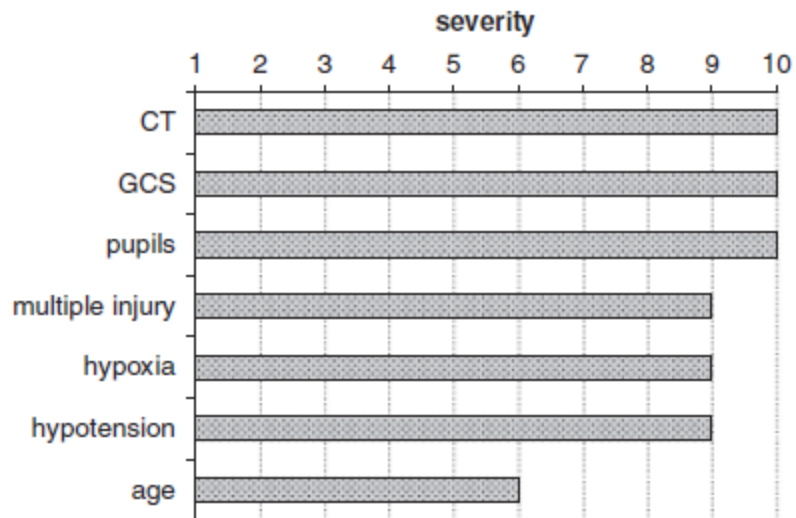
A. CHIEREGATO¹, C. MARTINO¹, V. PRANSANI¹, G. NORI¹, E. RUSSO¹, A. NOTO² and B. SIMINI³

¹UO Anestesia e Rianimazione, Rianimazione per la Traumatologia e le Neuroscienze, Ospedale Bufalini, Cesena, Italy, ²Unità Cardiovascolare e Toracica, Ospedale G. Martino, Università di Messina, via Consolare Valeria, Messina, Italy and ³UO Anestesia, Rianimazione e Terapia Antalgica, Ospedale Generale Provinciale, Lucca, Italy

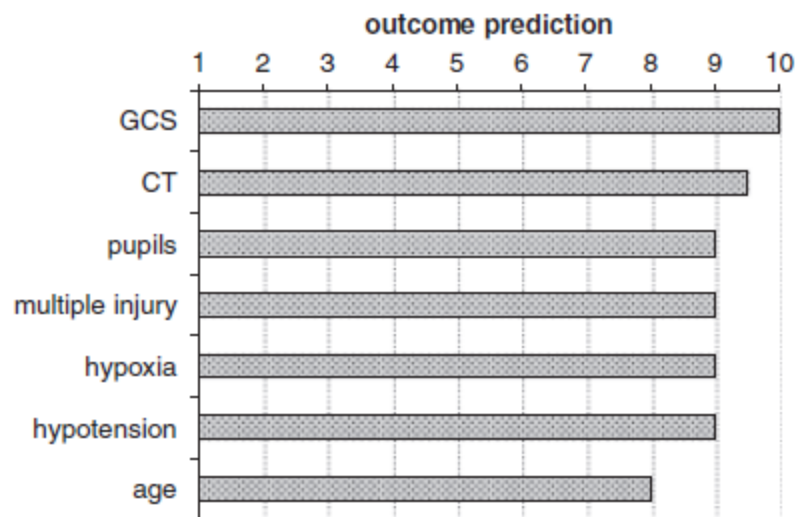
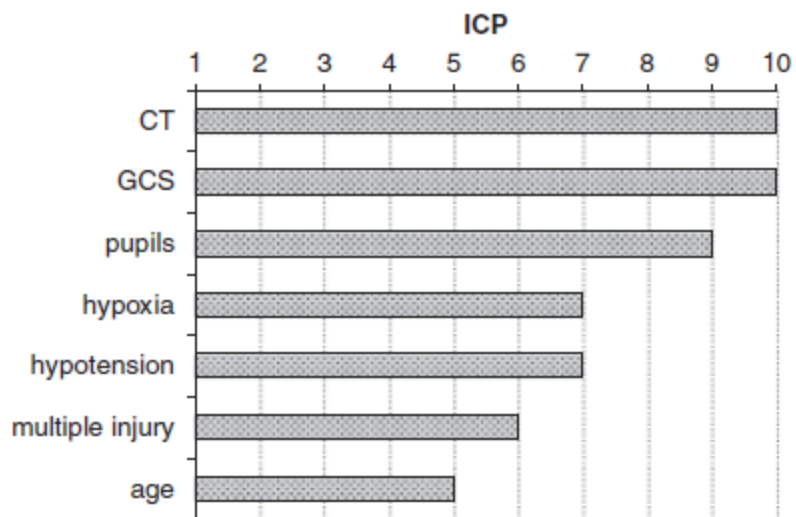
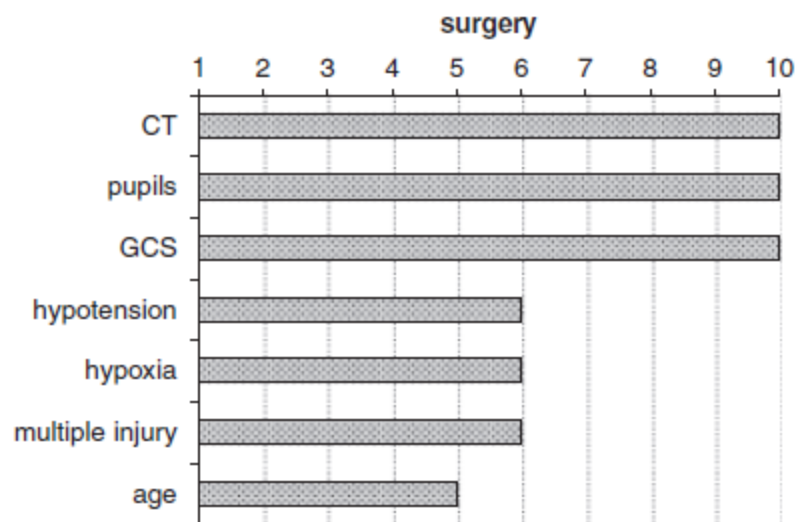
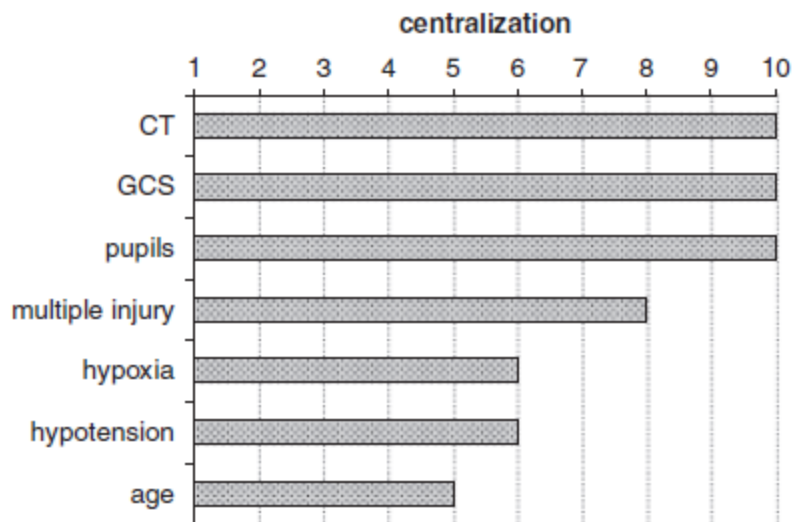
Questionnaires distributed to anesthetist during SIAARTI and SIARED courses in 2005

843 responded / 1334 questionnaires administrated

Precoce riconoscimento di patologie tempo-dipendenti



Precoce riconoscimento di patologie tempo-dipendenti



Moderato GCS 9 - 13

Grave GCS 8 - 3

GRAVITA' CLINICA

GCS + pupille + TAC

Precoce riconoscimento di patologie tempo-dipendenti

www.tbi-impact.org/?p=impact/calc

Come iniziare | Ultime notizie | Dettagli New York City... | Film in streaming | YouTube in MP3 conv... | storia dell'anestesia, hi...

Search | Safe | Weather | Facebook | Speedtest

Search: OK

IMPACT
International Mission for Prognosis and Analysis of Clinical Trials in TBI

Home | TBI | **IMPACT** | Publications | References | Links | Contact

You are here: TBI-IMPACT.org » **IMPACT** » Prognostic calculator

IMPACT

Prognostic calculator

Based on extensive prognostic analysis the IMPACT investigators have developed prognostic models for predicting 6 month outcome in adult patients with moderate to severe head injury (Glasgow Coma Scale ≤ 12) on admission. By entering the characteristics into the calculator, the models will provide an estimate of the expected outcome at 6 months. We present three models of increasing complexity (Core, Core + CT, Core + CT + Lab). These models were developed and validated in collaboration with the CRASH trial collaborators on large numbers of individual patient data (the [IMPACT database](#)). The models discriminate well, and are particularly suited for purposes of classification and characterization of large cohorts of patients. Extreme caution is required when applying the estimated prognosis to individual patients.



Prediction models for 6 month outcome after TBI

List of subpages

- Background
- Mission & Aims
- Collaboration
- Investigators
- Advisory Board
- IMPACT database
- Prognostic calculator
- IMPACT recommendations
- Common Data Elements (Draft)
- Data Sharing
- Acknowledgements

IMPACT

Prognostic models in TBI

- Clinical Practice**
 - informing relatives
 - support treatment decisions
 - allocating resources
- Research**



Prognostic calculator

Prediction models for 6 month outcome after TBI

Admission Characteristics Value

Core

Age (14-99 years)

Motor Score

Pupils

Core+CT

Hypoxia

Hypotension

CT Classification

tSAH on CT

Epidural mass on CT

Core+CT+Lab

Glucose (3-20 mmol/L)

Hb (6-17 g/dL)

This model predicts outcome in the following patients:

Adults with head injury, Glasgow Coma Scale 12 or less.

gcs

Ipertensione endocranica

Ipertensione endocranica documentata

Nel **60%** di 120 pazienti con **GCS < 9**

Chan

Nel **56%** di 121 pazienti con **GCS < 9**

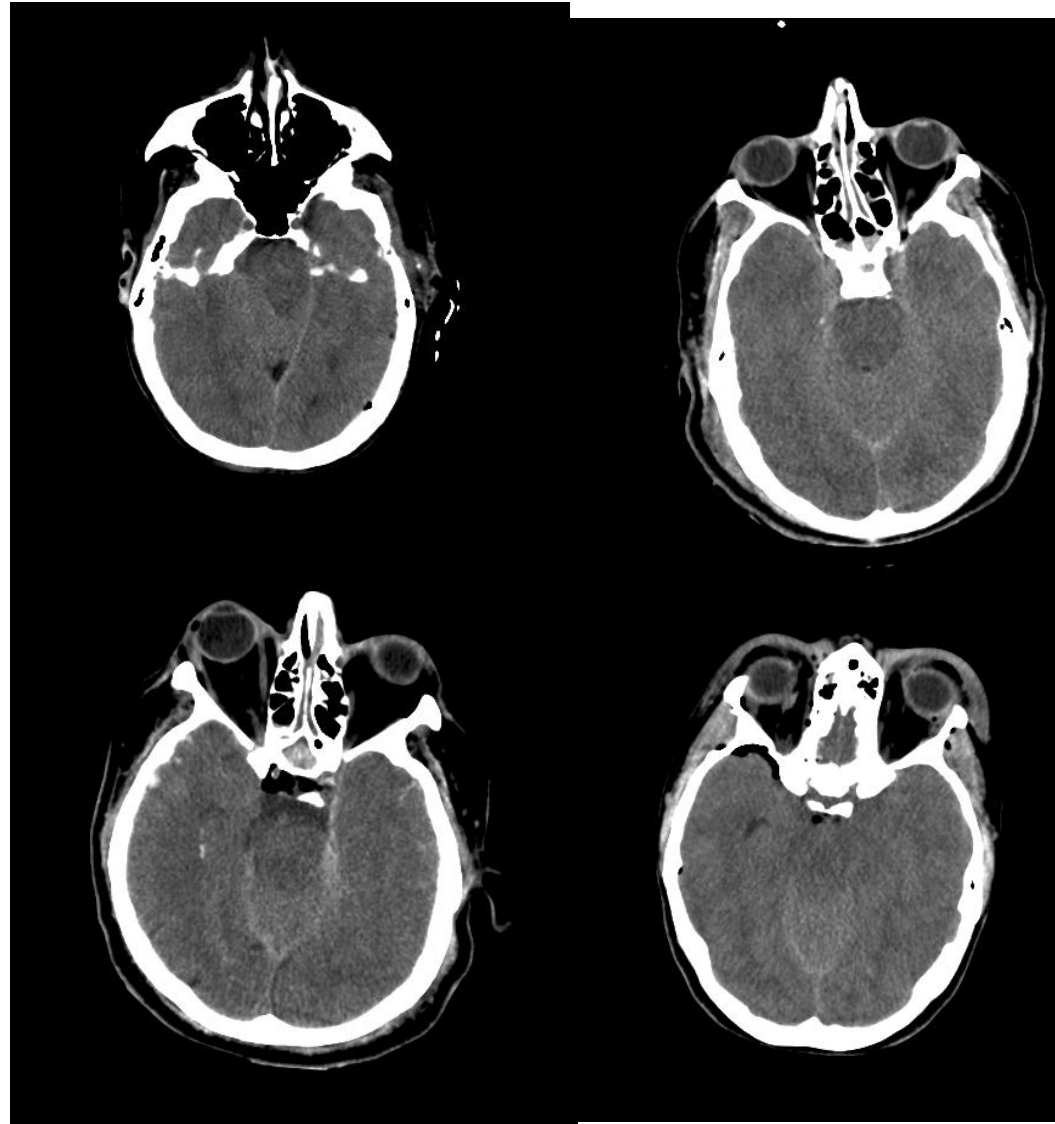
Neurolink

Nel **65%** di 138 pazienti con **GCS < 9**

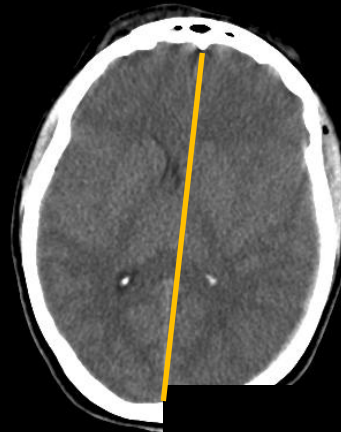
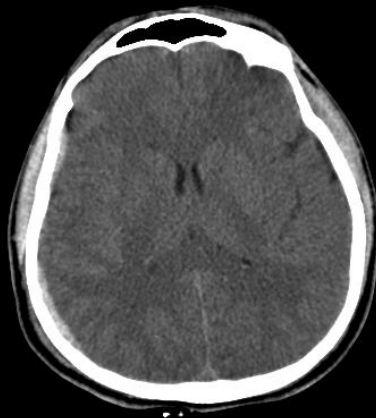
Stocchetti

tac

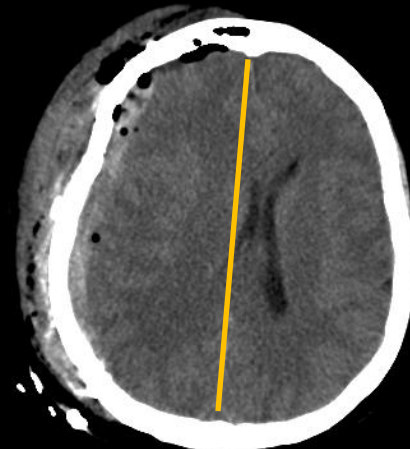
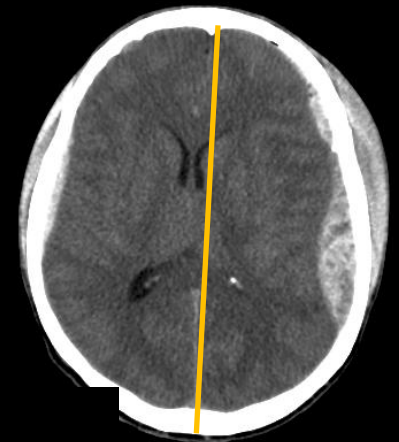
CISTERNE



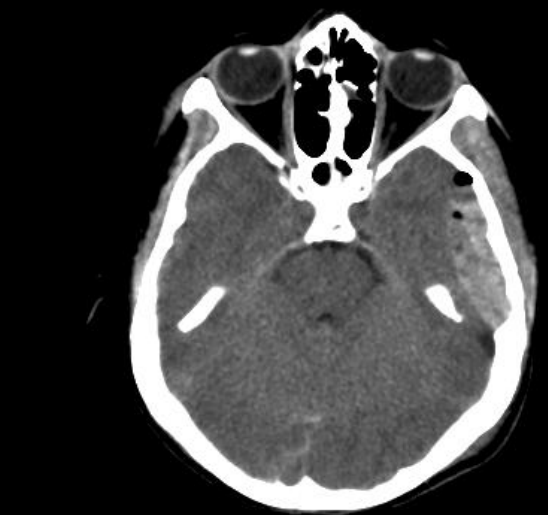
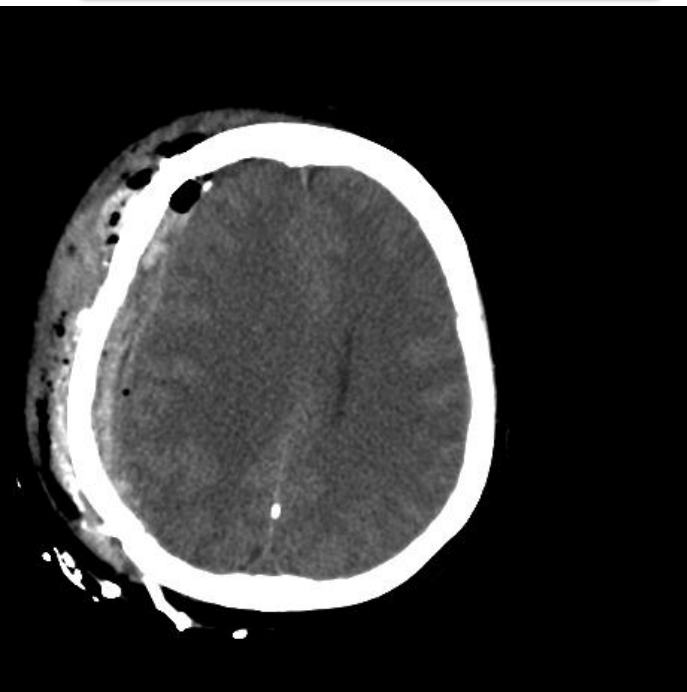
Precoce riconoscimento di patologie tempo-dipendenti



SHIFT



EMATOMI



CT PROGRESSION

Predictive factors of CT progression

- GCS at admission
- Presence of brain contusion at admission
- The amount of subarachnoid blood
- Marshall classification

- Male sex and older age
- Presence of brain contusion at admission
- Early first CT (< 2 hours)
- Increased aPTT



Chierigato Neurosurgery 2005



Oertel J Neurosurg 2002

pupille

Occhio alle pupille!
Se anomali pupillari:
terapia per erniazione



Herniation treatment



Paziente che diventa
anisocorico o midriatico
sotto i nostri occhi

Come ponte per arrivare
in sala operatoria



Mannitolo o Salina ipertonica

+

Iperventilazione

TPS o propofol ev in bolo

**Incremento pressorio - fluidi -
amine**

**UNICO
PACCHETTO**

Randomized, controlled trial on the effect of a 20% mannitol solution and a 7.5% saline/6% dextran solution on increased intracranial pressure after brain injury*

Claire Battison, BA Hons; Peter J. D. Andrews, MD; Catriona Graham, MSc; Thomas Petty

Objective: The aim of this pilot study was to compare the effects of equimolar doses of hypertonic saline and dextran solution (HSD, Rescueflow) with 20% mannitol solution for reduction of increased intracranial pressure.

Design: Prospective, randomized, controlled, crossover trial in the intensive care unit of a large teaching hospital.

Setting: Academic hospital and tertiary referral center for neurosciences.

Patients: Nine patients with an intracranial pressure of >20 mm Hg were recruited and received two treatments of each, HSD and 20% mannitol, in a randomized order.

Intervention: Equimolar, rapid intravenous infusions of either 200 mL of 20% mannitol or 100 mL of 7.5% saline and 6% dextran-70 solution (HSD) over 5 mins.

Measurements: Intracranial pressure, blood pressure, serum and urine sodium and osmolality, and urine output.

Main Results: Treatments reduced intracranial pressure with both mannitol (median decrease, 7.5 mm Hg, 95% confidence interval, 5.8–11.8) and HSD (median decrease, 13 mm Hg; 95% confidence interval, 11.5–17.3). HSD caused a significantly greater decrease in intracranial pressure than mannitol ($p = .044$). HSD had a longer duration of effect than mannitol ($p = .044$).

Conclusion: When given in an equimolar, rapid, intravenous infusion, HSD reduces intracranial pressure more effectively than mannitol. (Crit Care Med 2005; 33:196–202)

Key Words: Intracranial pressure; mannitol; hypertonic solutions; natriuretics

Isovolume hypertonic solutes (sodium chloride or mannitol) in the treatment of refractory posttraumatic intracranial hypertension: 2 mL/kg 7.5% saline is more effective than 2 mL/kg 20% mannitol

Renaud Vialet, MD; Jacques Albanèse, MD; Laurent Thomachot, MD; François Antonini, MD; Aurélie Bourgoin, MD; Bernard Alliez, MD; Claude Martin, MD, FCCM

Neurologic Critical Care

Long-term outcome after medical reversal of transtentorial herniation in patients with supratentorial mass lesions

Adnan I. Qureshi, MD; Romergryko G. Geocadin, MD; Jose I. Suarez, MD; John A. Ulatowski, MD, PhD

Mannitolo e
Sodio ipertonico

Ipertoniche

Stesso effetto
osmotico

No effetto diuretico

Bene nel trauma
cranico con lesioni
extracraniche
emorragiche
associate

Mannitolo o Salina ipertonica

+

Iperventilazione

TPS o propofol ev in bolo

**Incremento pressorio - fluidi -
amine**

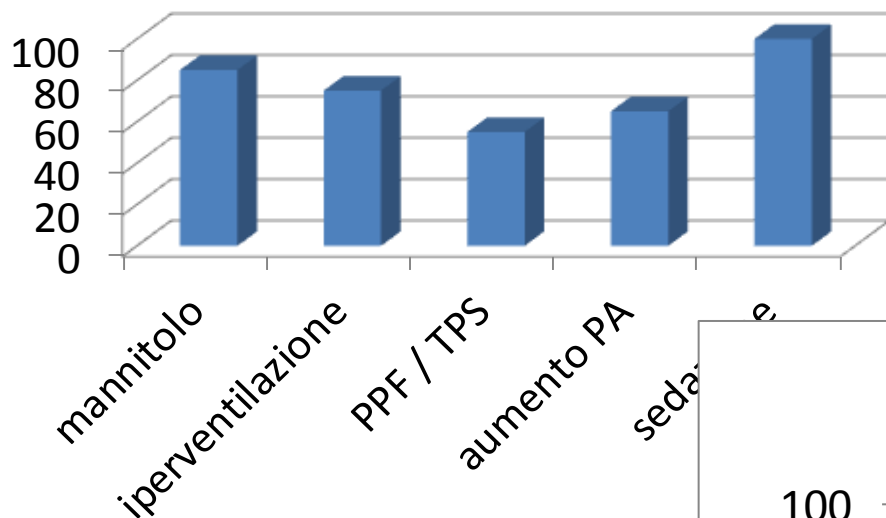
**Solo in caso di segni
di erniazione
cerebrale**

Rianimazione - Ospedale Cesena - Centro Traumi Romagna

2001- 2012

931 pazienti

Anomalie pupillari



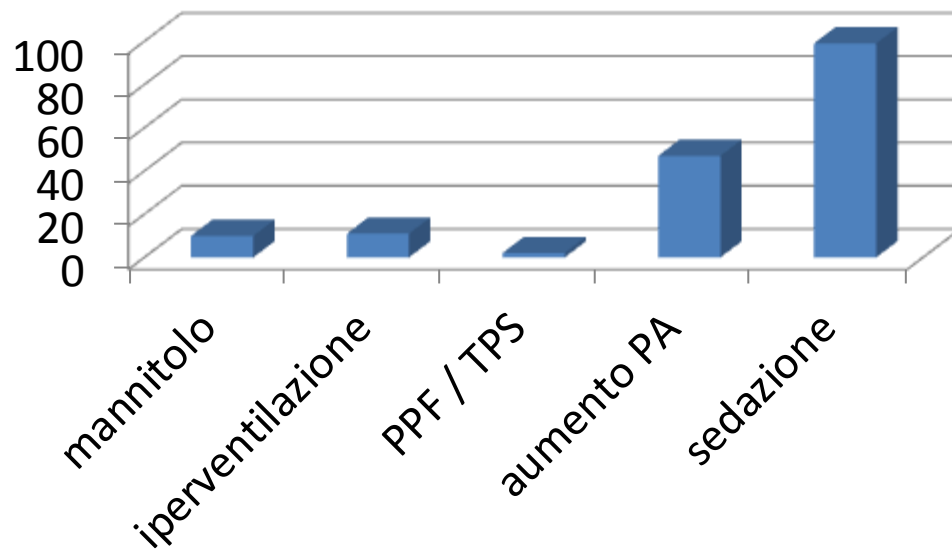
Terapia Trauma Cranico

PS Cesena

Anomali pupillari 142 paz (16%)

No anomalie 789 paz

No anomalie pupillari



Caso clinico LZ

Ore 11:30 ciclista investito da furgone (sbalzato a terra)

Ore 11:50 118 automedica
GCS 10, M5 V2 E3
 anisocoria dx>sx, ferita scalpo a dx
PA 190
SpO2 92%

 midazolam, fentanyl IOT VAM

SPOKE A 5'

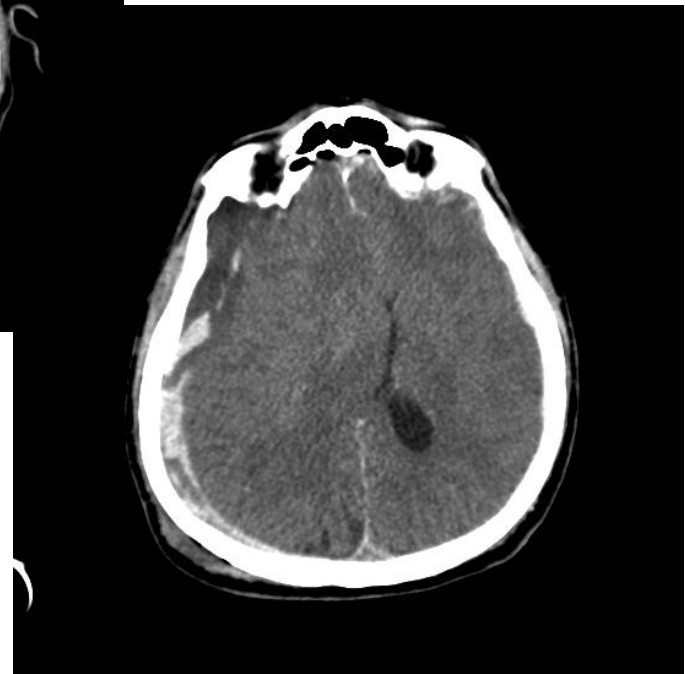
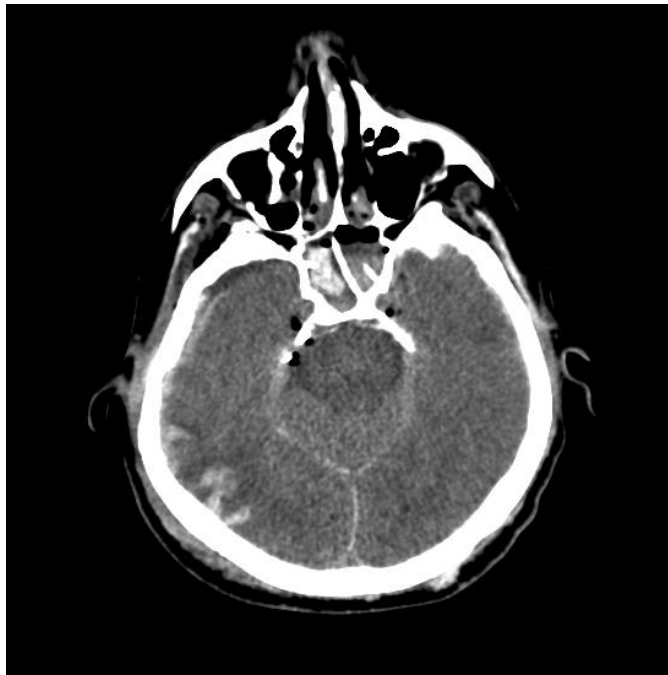
HUB a 25'

Che fare?

Ore 12:23 PS SPOKE
GCS 3, anisocoria dx>sx, ferita scalpo a dx
PA 130
SpO2 100%

Ore 13:24 TAC e cons NCH telematica

TAC H spoke



Consulenza nch telemedicina:
Indicazione alla centralizzazione

ore

12

13

14

15

16

Ore 14:20

trasporto per centralizzazione
GCS 3, media midriasi areagente
PA 130
SpO2 100%

midazolam, fentanyl

Ore 15:19

PS HUB

GCS 3, media midriasi areagente
PA 130 BE 0-7 Lattati 1.3
SpO2 100% PaO2 500 PaCO2 42
terapia deerniante (mannitolo, HV, PPF MDZ FNT)
no modifiche pupillari

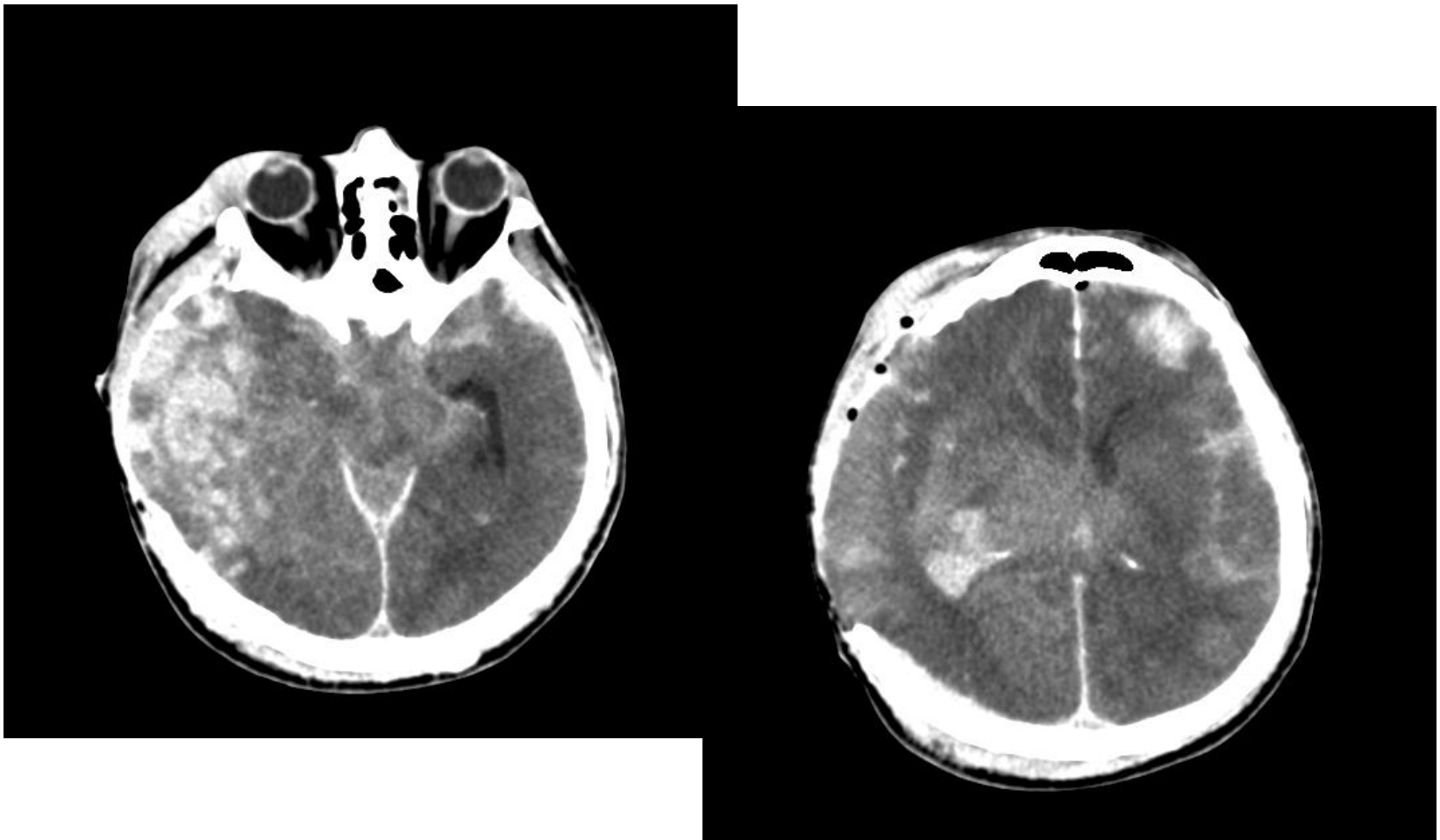
Ore 16:00

sala operatoria NCH

GCS 3, media midriasi areagente

evacuato SDH, rigonfiamento cerebrale

midriasi areagente a fine chirurgia

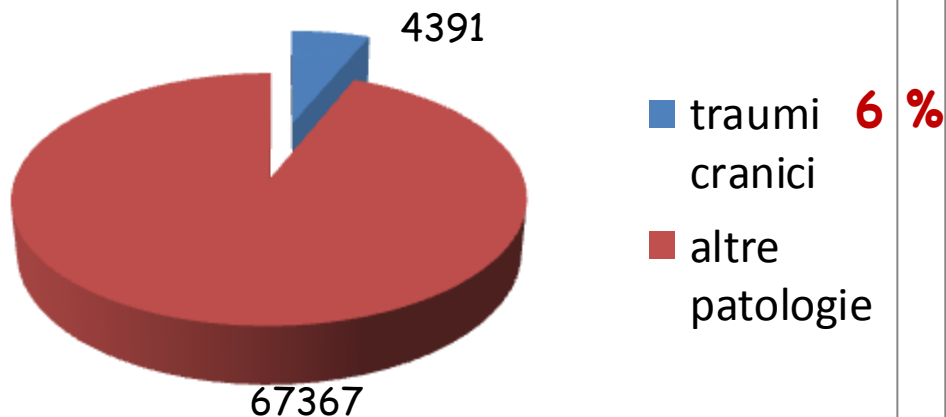


Il giorno successivo: morte cerebrale

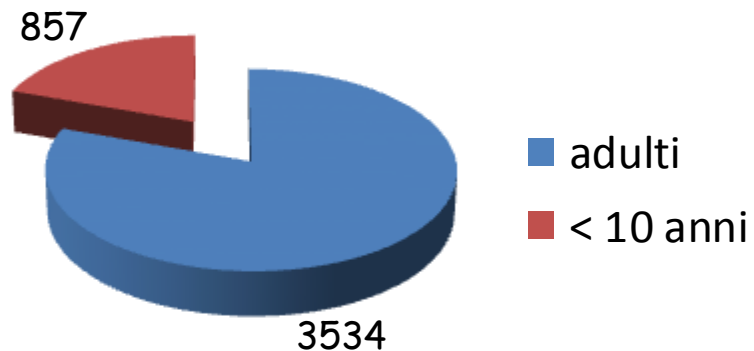
i falsi moderati

Pronto Soccorso Cesena anno 2011

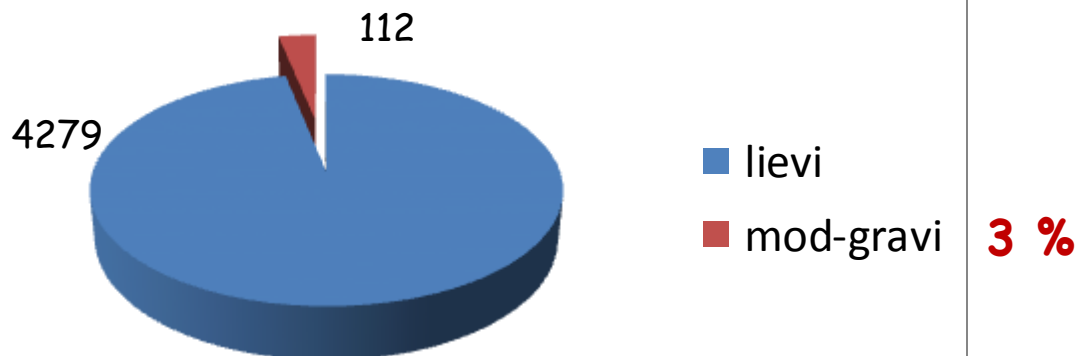
PS Cesena 2011

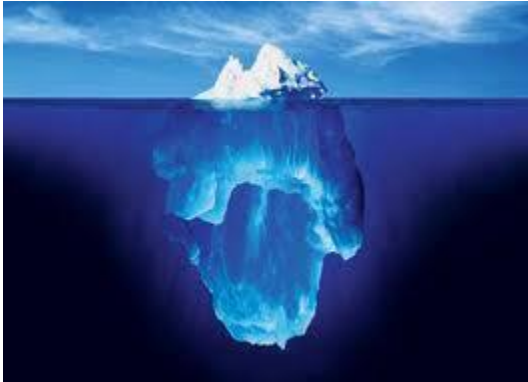


Traumi Cranici



Traumi Cranici adulti





}

Traumi cranici gravi

}

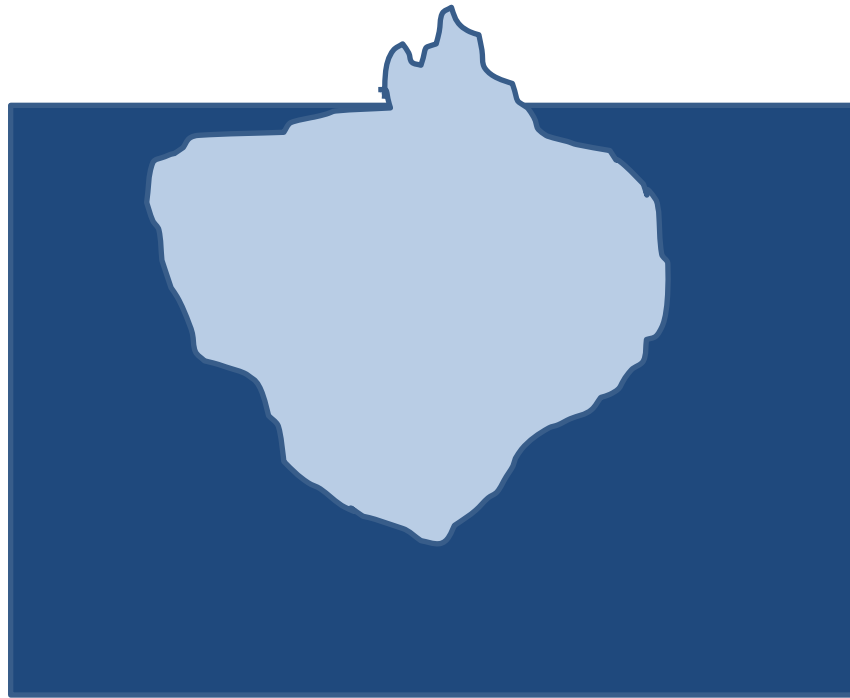
Traumi cranici lievi e moderati

ATLS

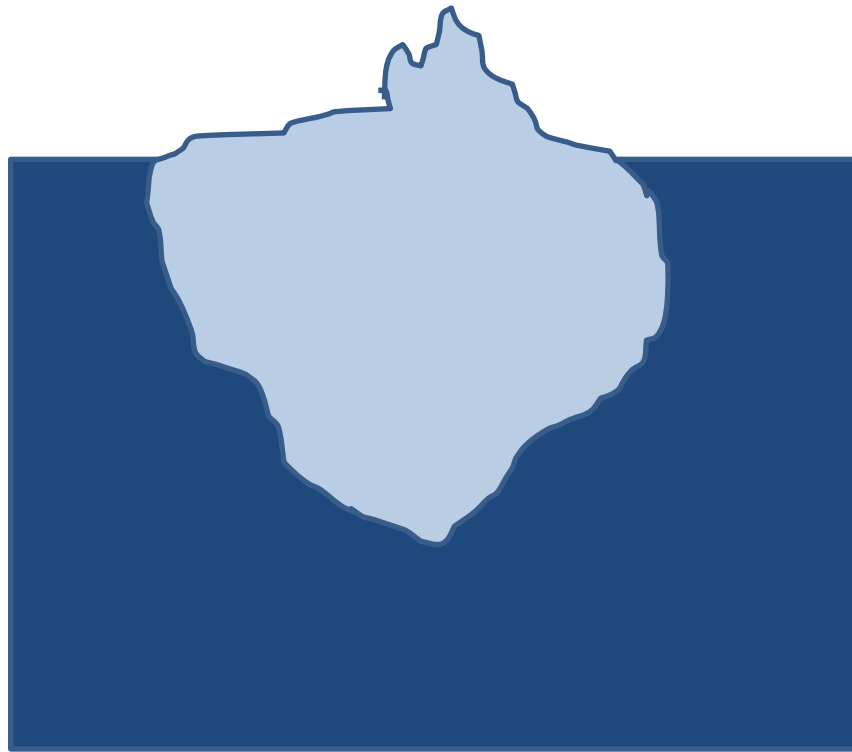
**Advanced Trauma
Life Support®**

Programma per Medici

**ATLS:
«Rivaluta in
continuazione...»**



Precoce riconoscimento di patologie tempo-dipendenti



Quanti sono i lievi-moderati che peggiorano?

pochi

Fra i pazienti che arrivano con trauma cranico grave in rianimazione quanti all'inizio erano lievi o moderati?

Circa la metà

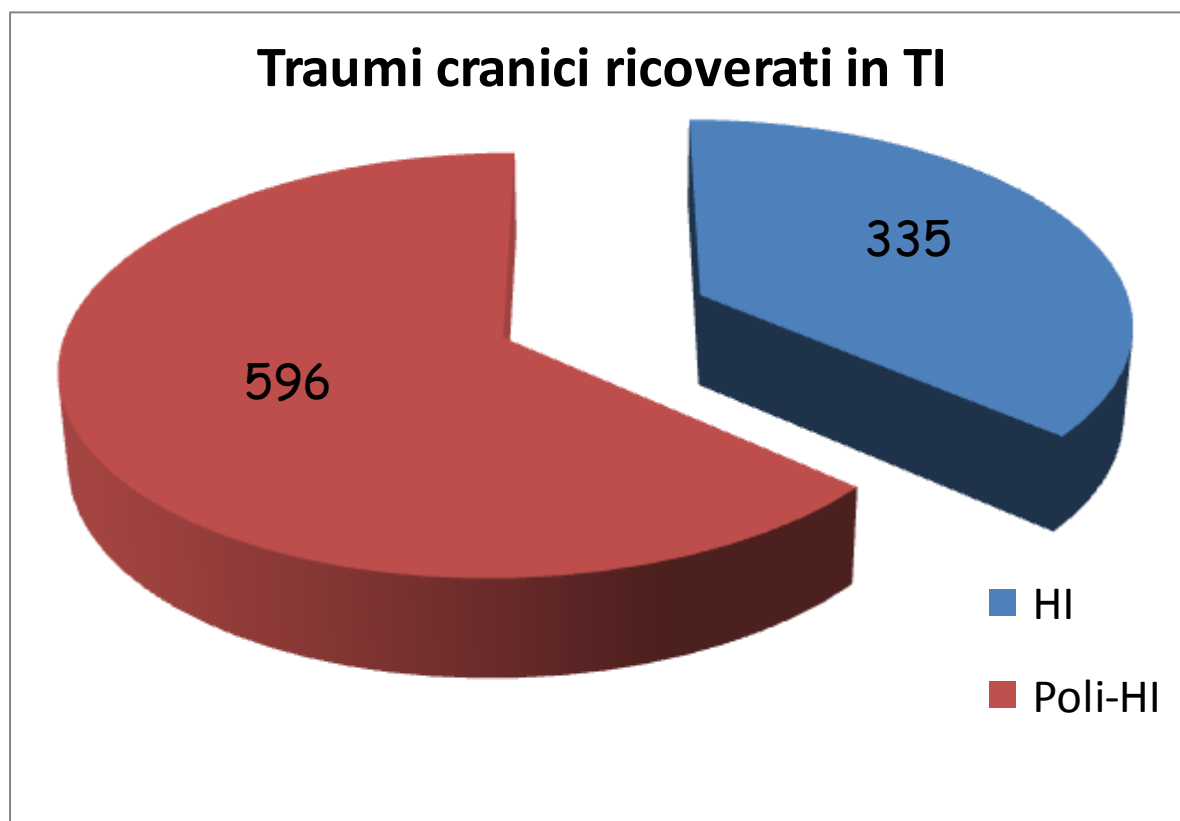


a. Respiratory pattern	<p>Regular sustained hyperventilation or Rarely, Cheyne-Stokes</p>
b. Pupillary size and reactions	<p>ipsilateral pupil widely dilated Does not constrict</p>
c. Oculocephalic and oculovestibular responses	<p>DOLL'S HEAD MANEUVER ICE WATER CALORICS ipsilateral eye doesn't move medially, but contralateral eye retains full lateral movement</p>
d. Motor responses at rest and to stimulation	<p>Decorticate or decerebrate responses</p>

Rianimazione - Ospedale Cesena - Centro Traumi Romagna

2001 - 2012

931 pazienti con trauma cranico, ricoveri primari

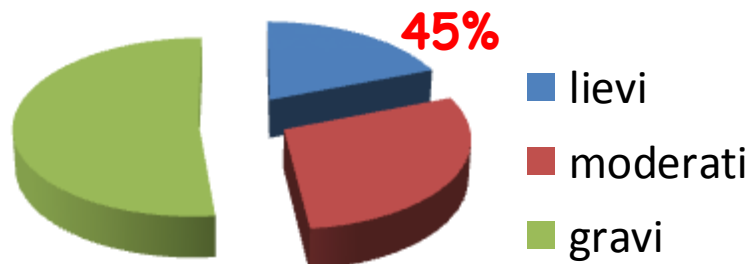


Rianimazione - Ospedale Cesena - Centro Traumi Romagna

2001- 2012

931 pazienti

GCS preH



TAC: massa evacuata



Il 45% dei pazienti ricoverati con trauma cranico in rianimazione nel preH aveva GCS > 8

Il 45% dei pazienti con ematoma evacuato e ricoverati in rianimazione nel preH aveva GCS > 8

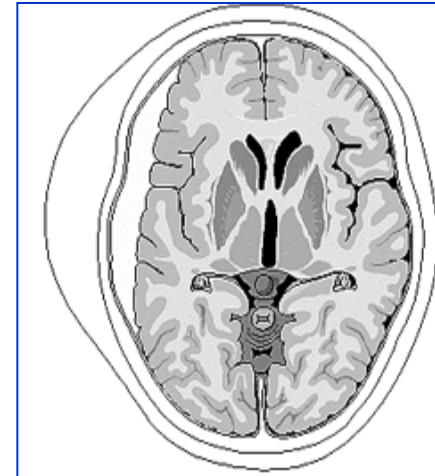
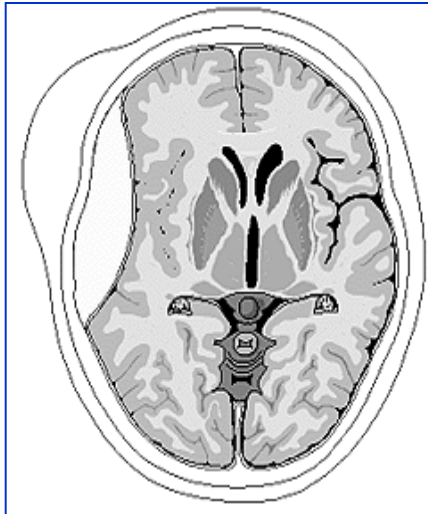
pitfalls

Rischio delle TAC troppo precoci

Evolutività degli ematomi

Ematoma extradurale

Si sviluppa nelle prime
8 - 12 ore dal trauma

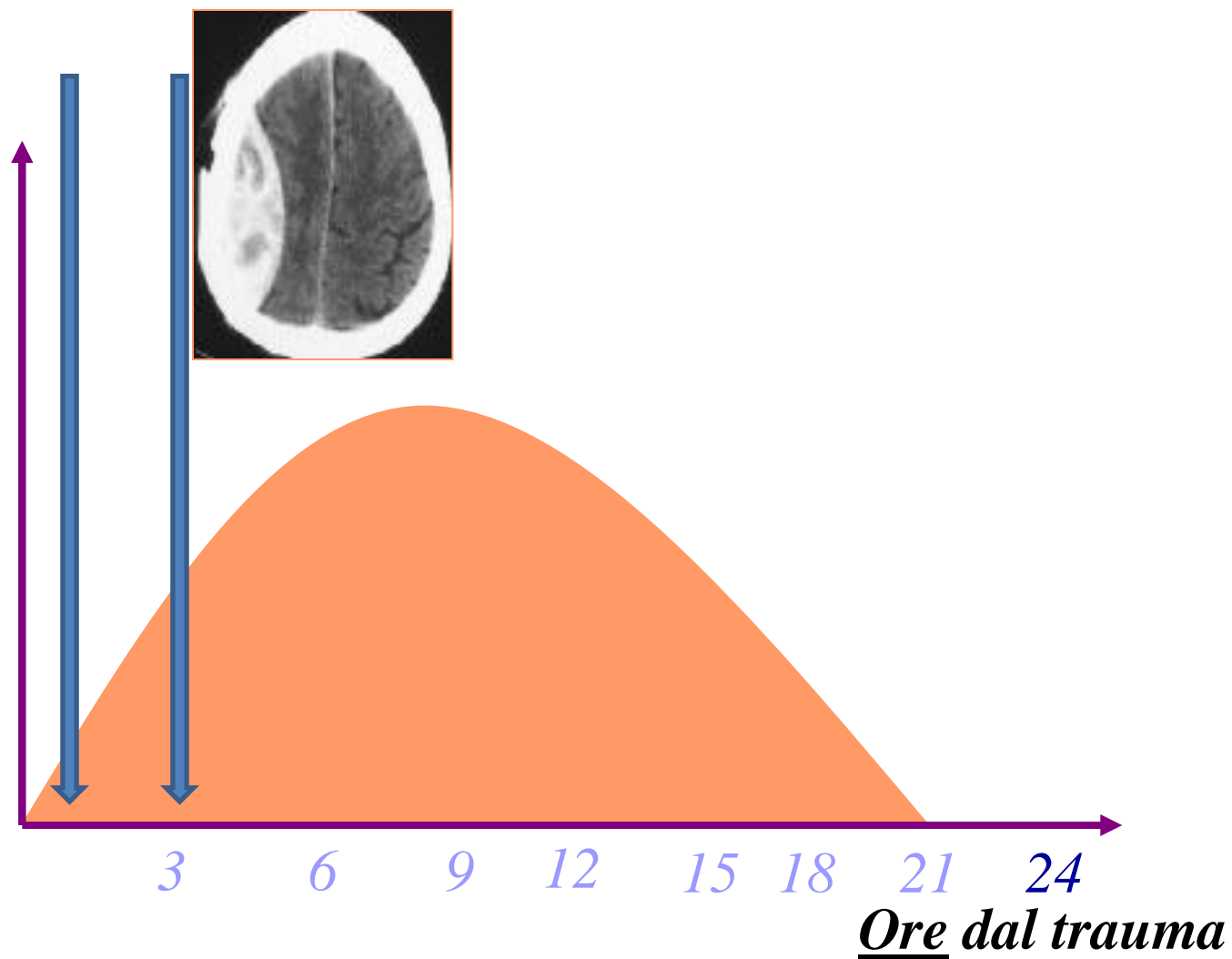


Ematoma sottodurale

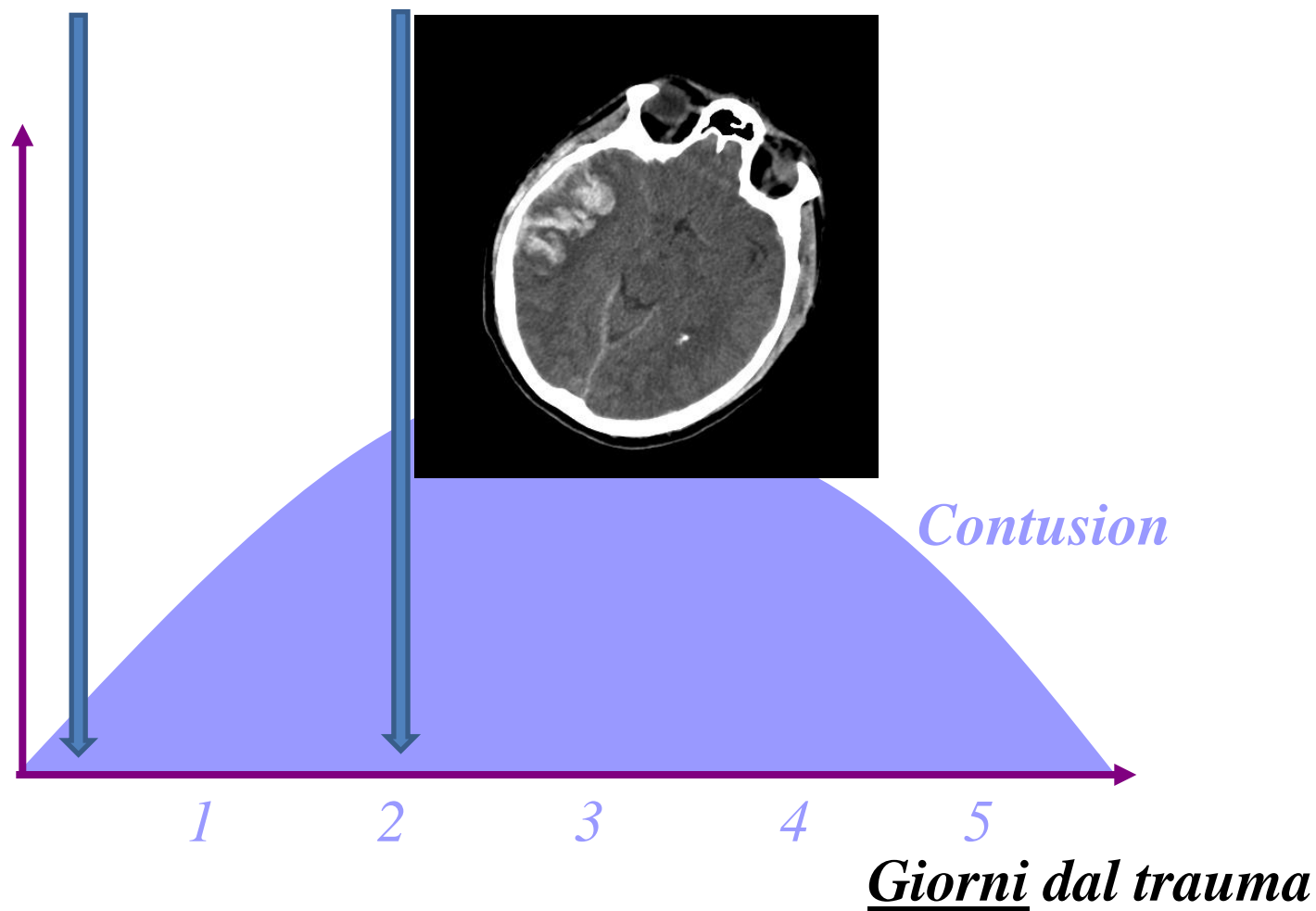
Si sviluppa nelle prime
24 ore dal trauma

Evolutività delle lesioni

Ematomi



Evolutività delle lesioni



pitfalls

pitfalls

i falsi gravi



Inaccurate Early Assessment of Neurological Severity in Head Injury

NINO STOCCHETTI,¹ FRANCESCA PAGAN,¹ EMILIANA CALAPPI,¹
KATIA CANAVESI,¹ LUIGI BERETTA,² GIUSEPPE CITERIO,³
MANUELA CORMIO,³ and ANGELO COLOMBO¹

In una certa proporzione pazienti con trauma cranico, non chirurgici, sedati e intubati, presentano una valutazione neurologica difficile e inficiata e possono dunque essere SOVRASTIMATI

Therefore MS patients were identified according to the following criteria:

- No surgical intracranial masses
- Could not follow commands at least at one point of neurological assessment
- Transferred from the ICU in ≤ 3 days to a regular ward
- Obeyed commands at discharge from the ICU

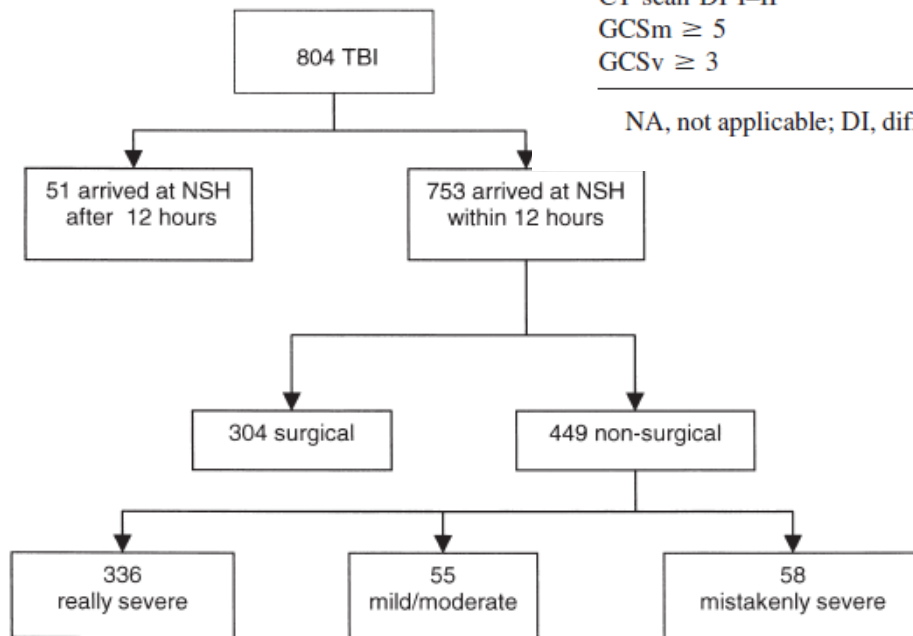
Inaccurate Early Assessment of Neurological Severity in Head Injury

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TABLE 4. ESTIMATED LOGISTIC REGRESSION COEFFICIENTS (β), ESTIMATED STANDARD ERRORS (SE), ESTIMATED ODDS RATIOS, AND 95% CONFIDENCE INTERVALS FOR THE ODDS RATIOS

<i>Variable</i>	β (SE)	<i>Estimated odds ratio (95% confidence interval)</i>
Constant	-4.8955 (0.655)	NA
Age < 40 years	0.9119 (0.382)	2.5 (1.2–5.3)
CT scan DI I–II	1.7312 (0.513)	5.6 (2.1–15.4)
GCSm \geq 5	1.4314 (0.469)	4.2 (1.7–10.5)
GCSv \geq 3	1.8584 (0.540)	6.4 (2.2–18.5)

NA, not applicable; DI, diffuse injury.



**Falsi gravi:
13%**

FIG. 1. Division of cases extracted from the data base.

The main features distinguishing MS from truly severe cases were

- younger age (< 40 years),**
- higher Glasgow Coma Scale (GCS) score at all time points,**
- Marshall classification of CT scan mostly Diffuse Injury I and II**
- fewer pupillary abnormalities,**
- a lower frequency of hypoxia, hypotension,**
- extra-cranial injuries.**

In these patients severity can be over-estimated

RACCOMANDAZIONE

- Dopo la stabilizzazione sistemica
- Dopo la TAC encefalo ed eventuale altra diagnosi
- Smaltiti farmaci e eventuali tossici

RIVALUTA IL GCS



Per modulare
l'inevitabile
OVER-TRIAGE
dei sistemi
traumi

Conclusioni

Take home message

Trauma cranico grave: GCS pupille TAC

Punta all'omeostasi, previeni il danno secondario

Rivaluta continuamente

Lavori di
squadra
**TRAUMA
TEAM**



Catena della sopravvivenza





Grazie!