NEURO UPDATE

Traumatologia cranica

Aneurismi intracranici

Torino, 9-10 marzo 2017

Il monitoraggio sistemico

Marina Munari

U.O.S Neurorianimazione e Neuroanestesia

U.O. Anestesiologia e Terapia Intensiva Direttore Prof. C.Ori

Azienda Ospedaliera di Padova

Università degli Studi di Padova

Consensus summary statement of the International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care

Table 1 Reasons why we monitor patients with neurologic disorders who require critical care

Detect early neurological worsening before irreversible brain damage occurs
Individualize patient care decisions
Guide patient management
Monitor the physiologic response to treatment and to avoid any adverse effects
Allow clinicians to better understand the pathophysiology of complex disorders
Design and implement management protocols
Improve neurological outcome and quality of life in survivors of severe brain injuries
Through understanding disease pathophysiology begin to develop new mechanistically oriented therapies where treatments currently are lacking or are empiric in nature

Intensive Care Med 2014;40:1189

MONITORAGGIO SISTEMICO ?!?

Advanced Monitoring of Systemic Hemodynamics in Critically Ill Patients with Acute Brain Injury

Fabio Silvio Taccone · Giuseppe Citerio ·
And the Participants in the International Multi-disciplinary Consensus Conference on Multimodality Monitoring

The end-points of this review (in patients with ABI) were to answer the following questions:

118/10 ⇨ 12%

1. What is the proportion of patients who have altered systemic hemodynamics and how many will develop circulatory failure, inadequate perfusion or organ dysfunction?
2. Can monitoring of systemic hemodynamics help understand the r
inadequate perfusi
3. Does hemodynam
optimizing brain
specific therapy?
4. What is the impact of systemic hemodynamic monitoring and related therapies on morbidity, mortality, and neurological outcome?
5. How can fluid responsiveness be assessed in ABI patients?
6. What hemodynamic monitoring is indicated in ABI patients, in particular to diagnose and support the management of unstable or at-risk patients?

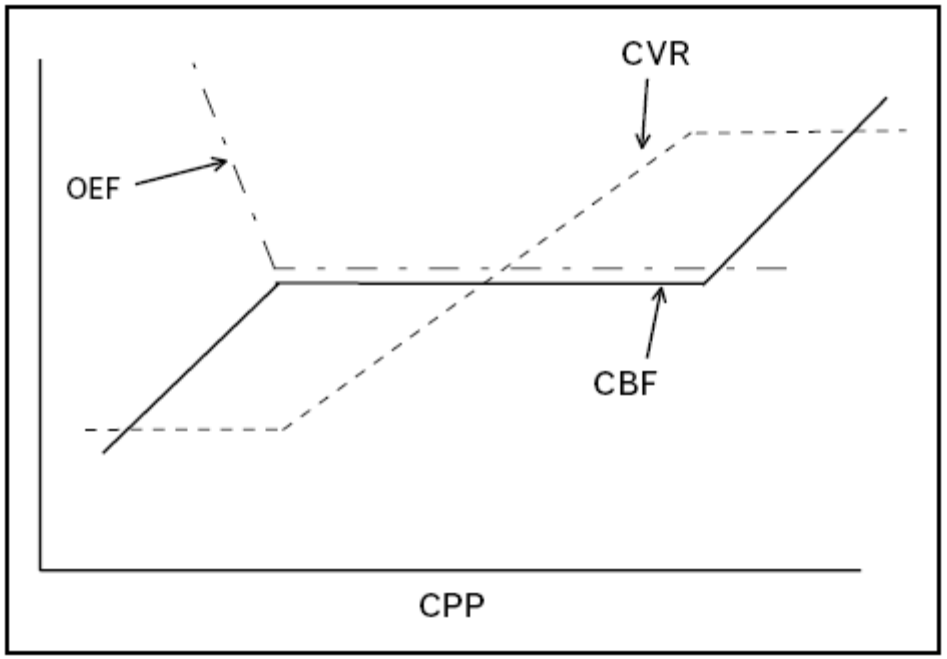
We recommend that hemodynamic monitoring be used to establish goals that take into account cerebral blood flow (CBF) and oxygenation. These goals vary

Neurocrit Care 2014;21:38

Obiettivi del monitoraggio

Manipolazione emodinamica

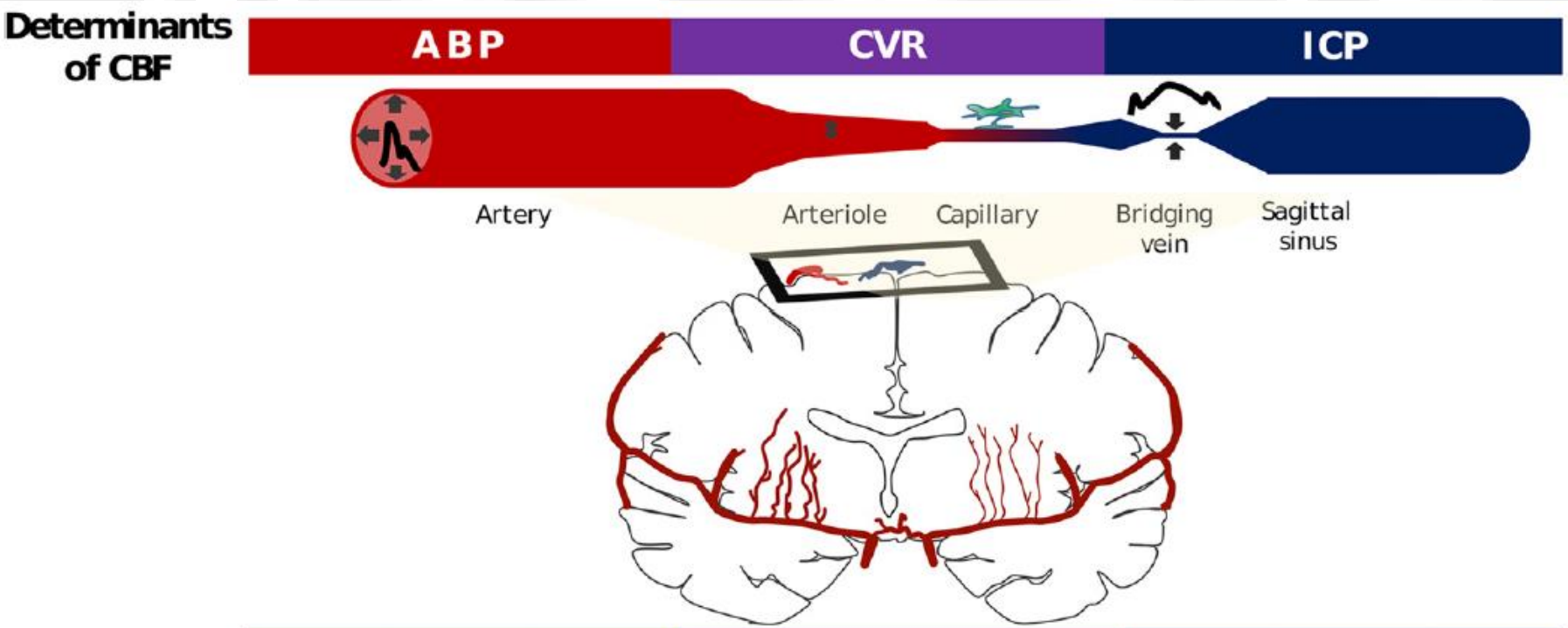
CPP, MAP, CBF, OEF, CVR



CBF, cerebral blood flow; CPP, cerebral perfusion pressure; CVR, cerebrovascular resistance; OEF, oxygen extraction fraction.

Regulation of the cerebral circulation: bedside assessment and clinical implications

Joseph Donnelly[1], Karol P. Budohoski[1], Peter Smielewski[1] and Marek Czosnyka[1,2*]



Influences	SV	HR	TPR	Obstruction	Vascular tone	CBV	CSF V	Tissue
Pathology	Cardiogenic	Arrhythmia	Inflammation	Stroke	Vasospasm. Metabolic, Neural, or Myogenic challenge	IIH, A and B waves	Hydrocephalus	Oedema
Therapies	Inotropes	Rate control	Vasopressors	Thrombolysis	Ventilation Vasoactive drugs	Ventilation head position	Shunt EVD	Mannitol Hypertonic saline

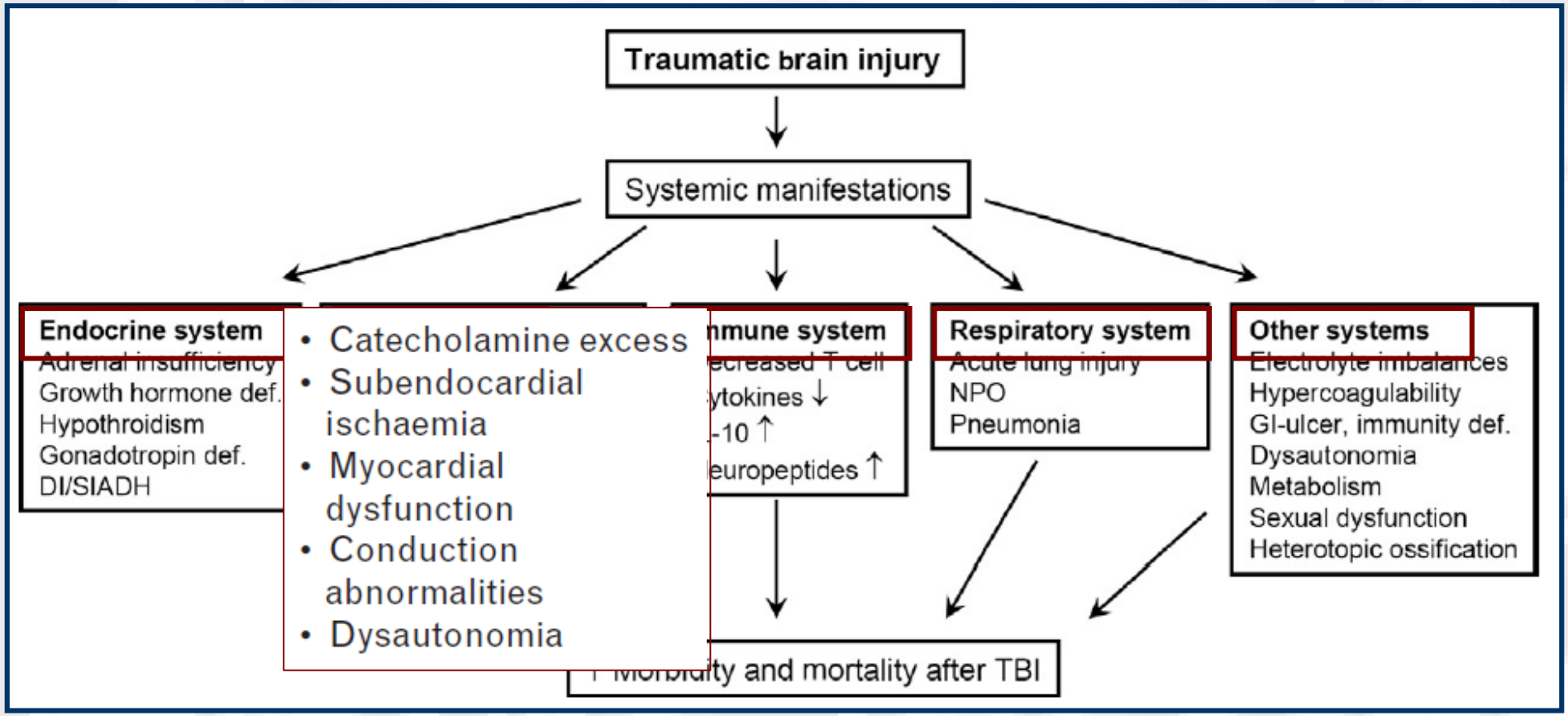
Critical Care 2016; 20:129

1. **Manifestazioni sistemiche primarie** legate al TBI, la cui natura e severità dipendono principalmente dalla gravità del danno cerebrale

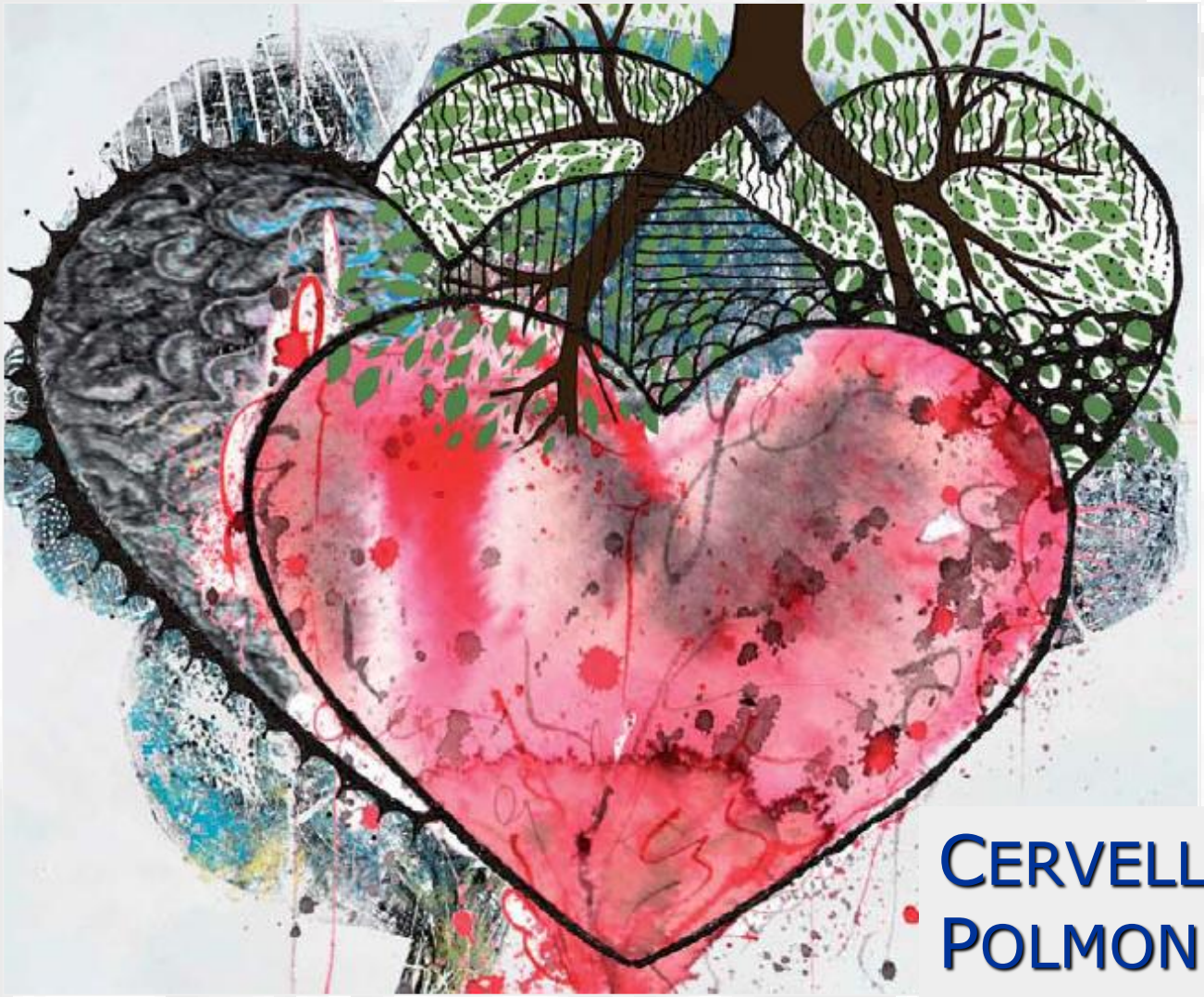
2. **Manifestazioni sistemiche secondarie** che includono disordini dovuti all'effetto di diverse strategie di trattamento utilizzate nel TBI

Systemic manifestations of traumatic brain injury

SAMSON SUJIT KUMAR GADDAM, THOMAS BUELL, AND CLAUDIA S. ROBERTSON*
Department of Neurosurgery, Baylor College of Medicine, Houston, TX, USA



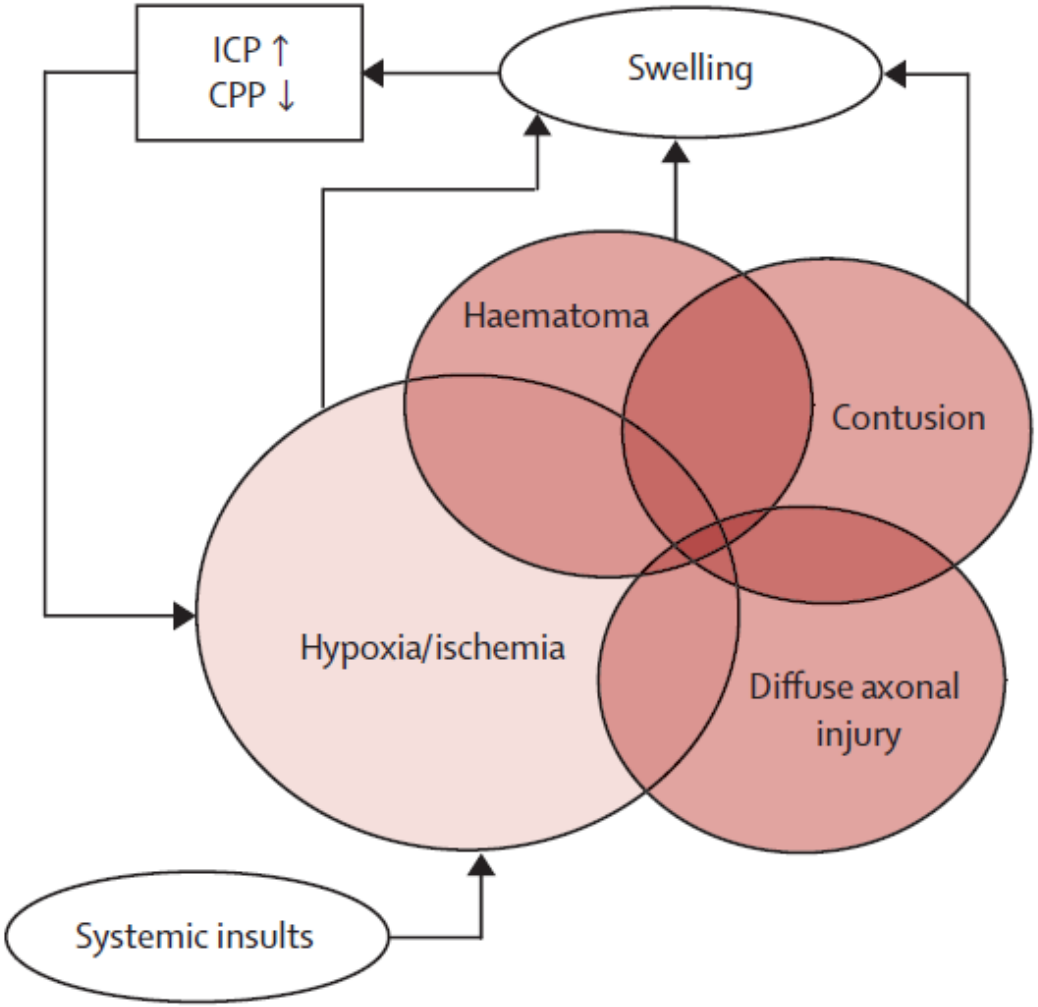
Handbook of Clinical Neurology Vol.127: 2015



CERVELLO, CUORE E POLMONE: UNA TRIADE INSIDIOSA

Moderate and severe traumatic brain injury in adults

Andrew I R Maas, Nino Stocchetti, Ross Bullock



QUALE INCIDENZA?

ALTERAZIONI ECG

- TACHICARDIA
- ANOMALIE RIPOLARIZZAZIONE
- ALTERAZIONI SEGMENTO ST
- ALTERAZIONI ONDA T
- ALLUNGAMENTO QT
- ONDE U

In TBI, up to 73% of patients will dem
ECG changes [4]. Changes can include anythi
sinus tachycardia through to ischaemic mir
repolarization abnormalities, such as ST segment changes, pathological T waves, QTc prolongation and U waves [4,5]. These ECG changes correlate with the severity of TBI and are associated with worse outcomes [5]. These electrographic changes can occur in the absence of known atherosclerotic coronary artery disease. The resulting subendocardial ischaemia, subendocardial haemorrhage, contraction band necrosis and cardiac dysfunction are associated with elevated cardiac biomarkers including troponin and creatine phosphokinase MB isoenzyme

Curr Opin Anest. 2015;525

ALTERAZIONI ECOCARDIOGRAFICHE

- **17.5**% alterazioni motilità parete
- **12**% alterazione funzione sistolica
- *Neurogenic stressed myocardium*

Takotsubo Cardiomyopathy in Traumatic Brain Injury

Chun Fai Cheah[1,2] · Mario Kofler[1] · Alois Josef Schiefecker[1] · Ronny Beer[1] · Gert Klug[3] · Bettina Pfausler[1] · Raimund Helbok[1]

References	Number of patients	Patients severe TBI (%)	Pathology	Abnormal ECG (%)	Increased CK or troponin level (%)[a]	Abnormal echocardiography (%)[b]	Patients with myocardial dysfunction (%)[c]
Bahloul et al. [20]	7	5/7 (85)	EDH, SDH, cerebral edema, contusion	7/7 (100)	2/7 (28.5)	3/7 (42.8)	7/7 (100)
Prathep et al. [21]	139	78/139 (56)	SDH, tSAH contusion	NA	98/139 (30.6)	31/139 (22.3)	31/139 (22.3)
Hasanin et al. [22]	50	50/50 (100)	SDH, tSAH, IVH, DAI, contusion	31/50 (62)	27/50 (54)	21/50 (42)	25/50 (50)

TBI traumatic brain injury, *tSAH* traumatic subarachnoid hemorrhage, *ECG* electrocardiography, *EDH* extradural hemorrhage, *SDH* subdural hematoma, *IVH* intraventricular hemorrhage, *DAI* diffuse axonal injury, *CK* creatinine kinase, *NA* not available

ALTERAZIONI PRESSIONE ARTERIOSA

Hypotension is the most common cardiovascular complication after severe TBI. In the diagnosis of this problem, it is important to exclude and treat hemorrhage as a result of other systemic injuries but hypotension can be caused by severe neurologic injury. Newer noninvasive hemodynamic monitors, including bedside ultrasound and continuous pulse contour analysis, can help with distinguishing the contributions of low intravascular volume and cardiac dysfunction. Treatment should be directed at the underlying hemodynamic state.

Handbook of Clinical Neurology Vol.127: 2015

Brain Trauma FOUNDATION

Guidelines for the Management of Severe Traumatic Brain Injury

4th Edition

The traditional definition of hypotension has been a SBP <90 mm Hg, and this was the target recommended in the first iterations of these guidelines. As will be noted, the literature now supports a higher level that may vary by age. The interrelationship between SBP, mean arterial pressure (MAP), and cerebral perfusion pressure (CPP) should be kept in mind as one considers threshold recommendations in these guidelines.

RECOMMENDATIONS

Level I and II

- There was insufficient evidence to support a Level I or II recommendation for this topic.

Level III

- Maintaining SBP at ≥100 mm Hg for patients 50 to 69 years old or at ≥110 mm Hg or above for patients 15 to 49 or over 70 years old may be considered to decrease mortality and improve outcomes.

Redefining hypotension in traumatic brain injury

Cherisse Berry, Eric J. Ley, Marko Bukur, Darren Malinoski, Daniel R. Margulies, James Mirocha, Ali Salim *

Patients and methods: A retrospective database review of all adults (≥15 years) with isolated moderate to severe TBI (head abbreviated injury score (AIS) ≥ 3, all other AIS ≤ 3), admitted from five Level I and eight Level II trauma centres (Los Angeles County), between 1998 and 2005. Several fit statistic analyses were performed for each admission SBP from 60 to 180 mm Hg to identify the model that most accurately defined hypotension for three age groups: 15–49 years, 50–69 years, and ≥70 years. The main outcome variable was mortality, and the optimal definition of hypotension for each group was determined from the best fit model. Adjusted odds ratios (AOR) were then calculated to determine increased odds in mortality for the defined optimal SBP within each age group.

Results: A total of 15,733 patients were analysed. The optimal threshold of hypotension according to the best fit model was SBP of 110 mm Hg for patients 15–49 years (AOR 1.98, CI 1.65–2.39, $p < 0.0001$), 100 mm Hg for patients 50–69 years (AOR 2.20, CI 1.46–3.31, $p = 0.0002$), and 110 mm Hg for patients ≥70 years (AOR 1.92, CI 1.35–2.74, $p = 0.0003$).

Conclusions: Patients with isolated moderate to severe TBI should be considered hypotensive for SBP < 110 mm Hg. Further research should confirm this new definition of hypotension by correlation with indices of perfusion.

15.733 pts
27% GCS ≤ 8

Injury Int J Care Injured 2012;43:1833

Treatment Decision	American Heart Association/American Stroke Association[2,a]	Neurocritical Care Society[12,b]
Blood pressure control	Between the time of SAH symptom onset and aneurysm obliteration, blood pressure should be controlled with a titratable agent to balance the risk of stroke, hypertension-related rebleeding, and maintenance of cerebral perfusion pressure (Class I, Level B). The magnitude of blood pressure control to reduce the risk of rebleeding has not been established, but a decrease in systolic blood pressure to less than 160 mm Hg is reasonable (Class IIa, Level C).	Treat extreme hypertension in patients with an unsecured, recently ruptured aneurysm. Modest elevations in blood pressure (mean blood pressure of less than 110 mm Hg) do not require therapy. Premorbid baseline blood pressures should be used to refine targets and hypotension should be avoided (low quality of evidence, strong recommendation).
Intravascular volume status	Maintenance of euvolemia and normal circulating blood volume is recommended to prevent delayed cerebral ischemia (Class I, Level B).	Intravascular volume management should target euvolemia and avoid prophylactic hypervolemic therapy. In contrast, there is evidence for harm from aggressive administration of fluid aimed at achieving hypervolemia (moderate quality of evidence, strong recommendation).
Cardiopulmonary complications	No recommendations given.	Baseline cardiac assessment with serial enzymes, ECG, and echocardiography is recommended, especially in patients with evidence of myocardial dysfunction (low quality of evidence, strong recommendation). Monitoring of cardiac output may be useful in patients with evidence of hemodynamic instability or myocardial dysfunction (low quality of evidence, strong recommendation).

Monitoraggio Sistemico: come?

Consensus summary statement of the International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care

MONITORAGGIO EMODINAMICO

1. We recommend the use of electrocardiography and invasive monitoring of arterial blood pressure in all unstable or at-risk patients in the ICU. (Strong Recommendation, moderate quality of evidence.)
2. We recommend that hemodynamic monitoring be used to establish goals that take into account cerebral blood flow (CBF) and oxygenation. These goals vary depending on diagnosis and disease stage. (Strong recommendation, moderate quality of evidence.)
3. We recommend the use of additional hemodynamic monitoring (e.g., intravascular volume assessment, echocardiography, cardiac output monitors) in selected patients with hemodynamic instability. (Strong recommendation, moderate quality of evidence.)
4. We suggest that the choice of technique for assessing pre-load, after-load, cardiac output, and global systemic perfusion should be guided by specific evidence and local expertise. (Weak recommendation, moderate quality of evidence.)

1. ECG
   IBP

2. ECOcardiografia
   PiCCO

Intensive Care Med 2014;40:1189

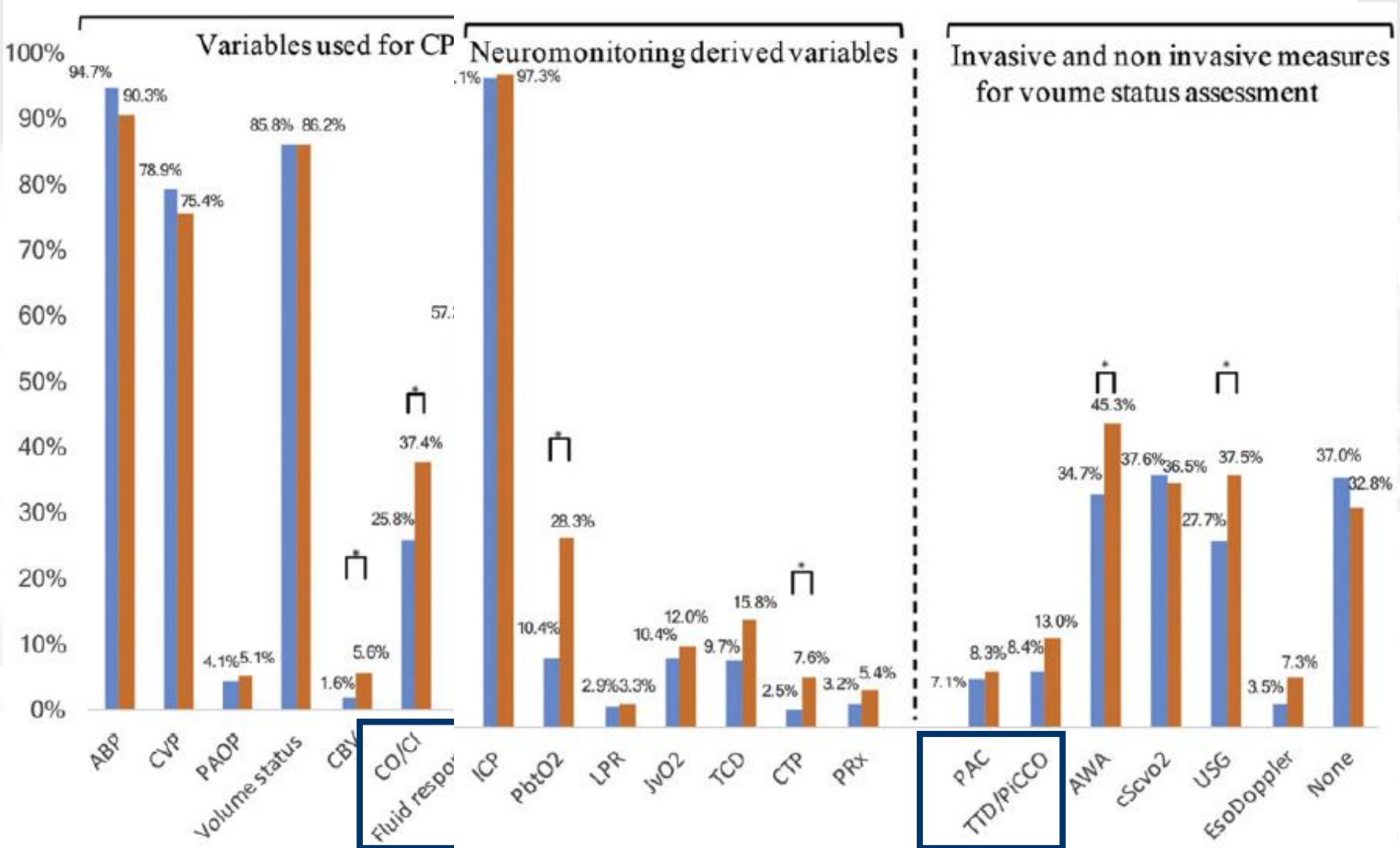
Rapporto nazionale TI neurochirurgiche - Anno 2015
Indicatori di processo - Pazienti adulti

Presidi e/o trattamenti (Missing=7)	Utilizzo		TBI Utilizzo		TI POLIVALENTI Utilizzo	
	N	%	N	%	N	%
Presidi (antibiotici esclusi)	3207	95.5	371	98.1	49231	94.0
Ventilazione invasiva	2332	69.4	320	84.7	35486	67.8
Ventilazione non invasiva	192	5.7	13	3.4	8393	16.0
Tracheostomia	535	15.9	135	35.7	5945	11.4
iNO (ossido nitrico inalatorio)	1	0.0	0	0.0	117	0.2
CVC (Catetere Venoso Centrale)	1363	40.6	259	68.5	34206	65.3
PICC	27	0.8	6	1.6	855	1.6
Catetere arterioso	3084	91.8	359	95.0	39385	75.2
Farmaci vasoattivi	653	19.4	129	34.1	17496	33.4
Farmaci antiaritmici	86	2.6	14	3.7	4135	7.9
Contropulsatore aortico	1	0.0	0	0.0	369	0.7
Monit. invasivo gittata	57	1.7	19	5.0	1842	3.5
Monitoraggio continuo ScVO2	0	0.0	0	0.0	146	0.3
Pacing temporaneo	1	0.0	0	0.0	211	0.4
Assistenza ventricolare	0	0.0	0	0.0	12	0.0
Defibrillazione	12	0.4	1	0.3	1106	2.1
Rianimazione cardio-polmonare (CPR)	14	0.4	0	0.0	1698	3.2
Trasfusione di sangue massiva	17	0.5	6	1.6	828	1.6
Monitoraggio PIC senza drenaggio liquor	132	3.9	81	21.4	400	0.8
Monitoraggio PIC con drenaggio liquor	300	8.9	26	6.9	397	0.8
Drenaggio ventricolare esterno senza PIC	57	1.7	1	0.3	176	0.3
Emofiltrazione	18	0.5	3	0.8	1882	3.6
Emodialisi	13	0.4	2	0.5	1202	2.3
ECMO	1	0.0	1	0.3	169	0.3

Hemodynamic and neuro-monitoring for neurocritically ill patients: An international survey of intensivists☆,☆☆,★

Sanjeev Sivakumar, MD [a], Fabio S. Taccone, MD, PhD [b], Mohammed Rehman, MD [c], Holly Hinson, MD [d], Neeraj Naval, MD [e]

422 (65%) were OI, and 226 (35%) were NI. More NI



3.4. Hemodynamic and neuromonitoring practices in TBI

For CPP optimization, most monitor invasive arterial blood pressure (ABP) (93%, n = 479) followed by clinical volume status assessment using input/output, daily weights (86%, n = 442) and central venous pressure (CVP) (77%, n = 398 Fig. 2A). Neurointensivists used significantly more often measurements of cardiac output/index (CO/CI) (37% vs 26%, $P = .006$), and global end-diastolic volume index (GEDVI) (9% vs 4%, $P = .018$); OI more frequently measured central venous oxygen saturation ($ScvO_2$) (54% vs 45%, $P = .045$ Fig. 2B). Among neuromonitoring-derived variables used to augment CPP, ICP monitoring is most used (97%, n = 452), followed by $PbtO_2$ (18%, $P = 82$). Significantly more NI used $PbtO_2$ (28% vs 10%, $P < .001$) and CTP (8% vs 2.5%, $P = .012$).

Among invasive and non-invasive measures of volume status assessment in CPP optimization, most respondents used arterial waveform analysis (AWA, 39%, n = 196), followed by continuous $ScvO_2$ (37%, n = 188) and bedside ultrasound (BUS, 31.5%, n = 159) (Fig. 2A). Use of AWA (45% vs 35%, $P = .019$) and BUS (37.5% vs 28%, $P = .023$) was significantly more among NI. A large proportion did not use any tool outside of clinical exam and standard ICU monitoring (35%, n = 178 Fig. 2B).

- ✓ PA
- ✓ BILANCIO
- ✓ PVC
- ✓ CO/CI
- ✓ GEDVI

J Crit Care 2017; 39:40

TECNICHE DISPONIBILI PER IL MONITORAGGIO EMODINAMICO IN PAZIENTI CON NEUROLESIONE ACUTA

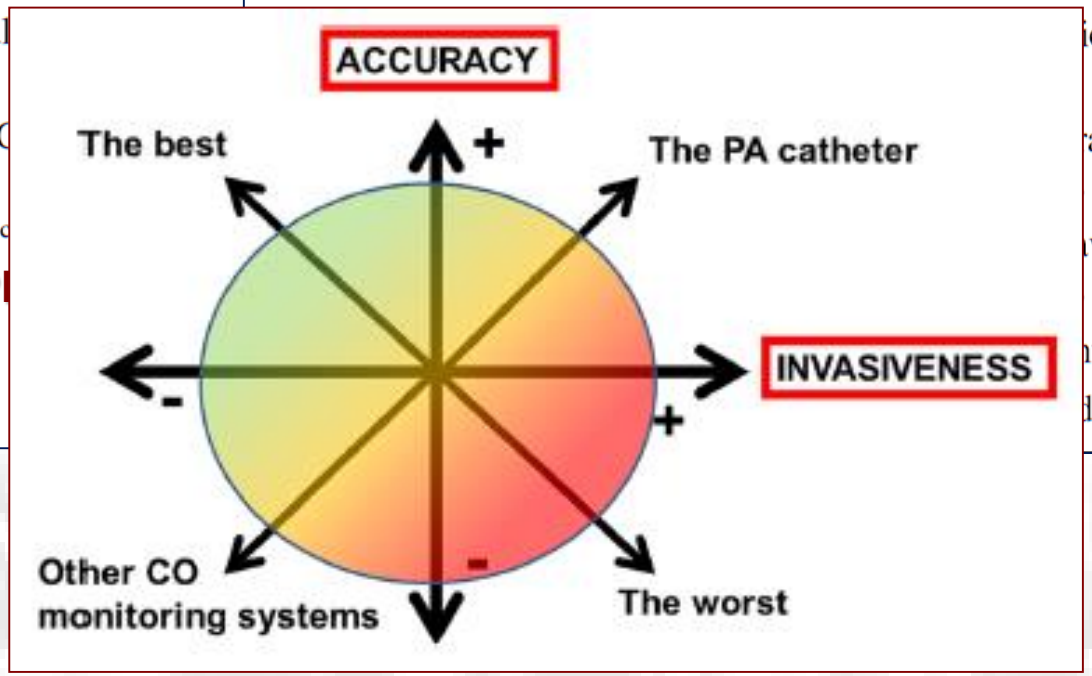
Techniques	Cardiac output	LV function	Preload	Fluid responsiveness	Afterload
PAC	+	−(LV) +(RV)	+	−	+
Trans-pulmonary thermodilution[a]	+	+	+	+	+
External+internal calibrated PCM[a]	+	+	−	+	+
PiCCO					
Internal-calibrated PCM[b]	+	−	−	+	+
FloTrac					
Non-calibrated PCM[c]	+	+	−	+	+
PRAM					
Echocardiography	+	+	+	+	−

PAC pulmonary artery catheter, *PCM* pulse contour method, *ScvO*$_2$ central venous oxygen saturation, *SvO2* pulmonary artery pressure, *PAOP* pulmonary artery occlusive pressure, *LV* left ventricle, *RV* right ventricl

Neurocrit Care 2014;21:S38

TECNICHE DISPONIBILI PER IL MONITORAGGIO EMODINAMICO IN PAZIENTI CON NEUROLESIONE ACUTA

Techniques	Advantages	Disadvantages
PAC	Measure of PAP, PAOP Measure of SvO_2	Invasiveness not beat-by-beat analysis
Trans-pulmonary thermodilution[a]	Less invasive No need for PAC positioning	Requires a specific femoral arterial cathete not beat-by-beat analysis
External+internal cal		ion every 4-6 h requires a specifi arterial catheter
Internal-calibrated PC		racy for CO sensitive to SVR specific catheter
Non-calibrated PCM[9]		available Less accuracy for CO (optimal arterial pressure tracing
Echocardiography		t use
		dequate training



Neurocrit Care 2014;21:S38

GiViTi 2015

Monit. invasivo gittata (N=1842)	N	%
Swan Ganz	368	20.0
PICCO	1128	61.2
LIDCO	6	0.3
Vigileo-PRAM	300	16.3
Altro	37	2.0
Missing	3	

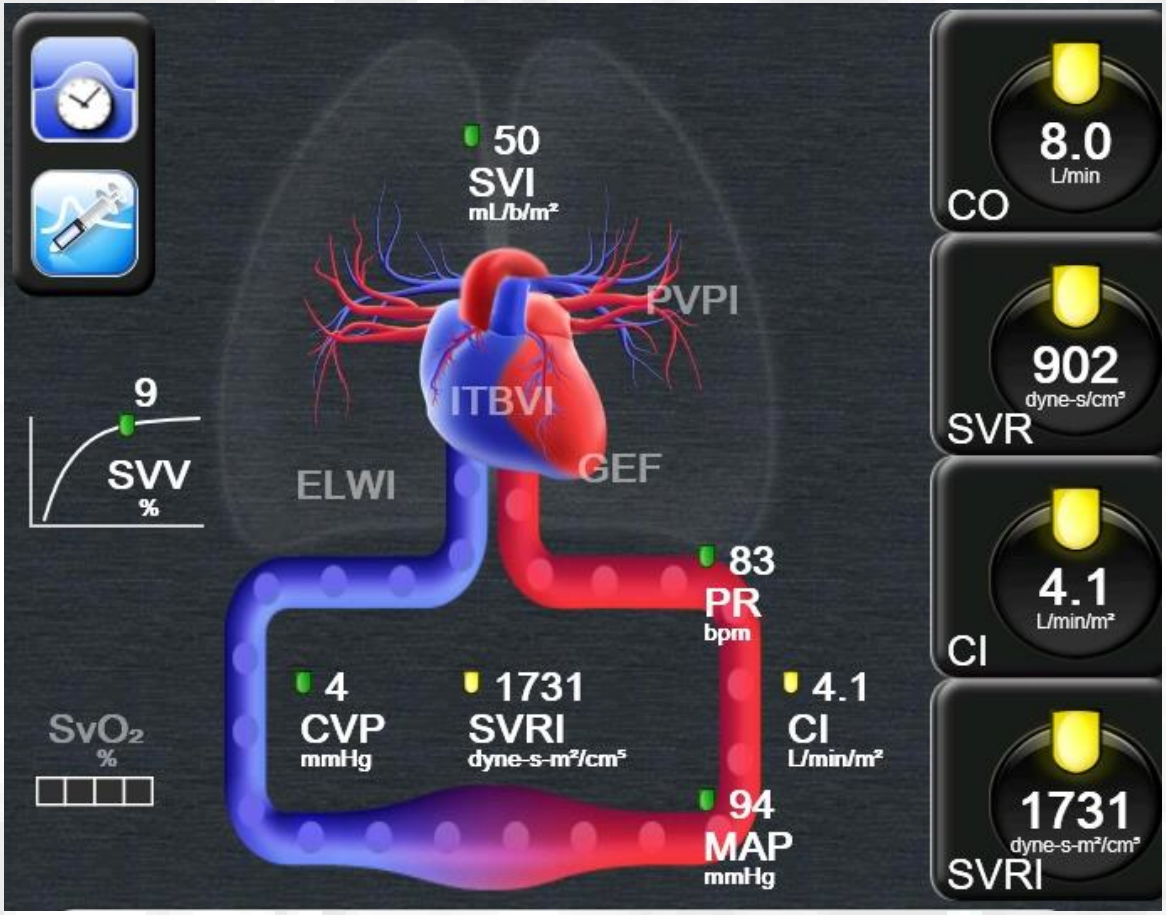
TI Polivalenti

Monit. invasivo gittata (N=1478)	N	%
Swan Ganz	266	18.0
PICCO	940	63.6
LIDCO	4	0.3
Vigileo-PRAM	234	15.8
Altro	34	2.3
Missing	0	

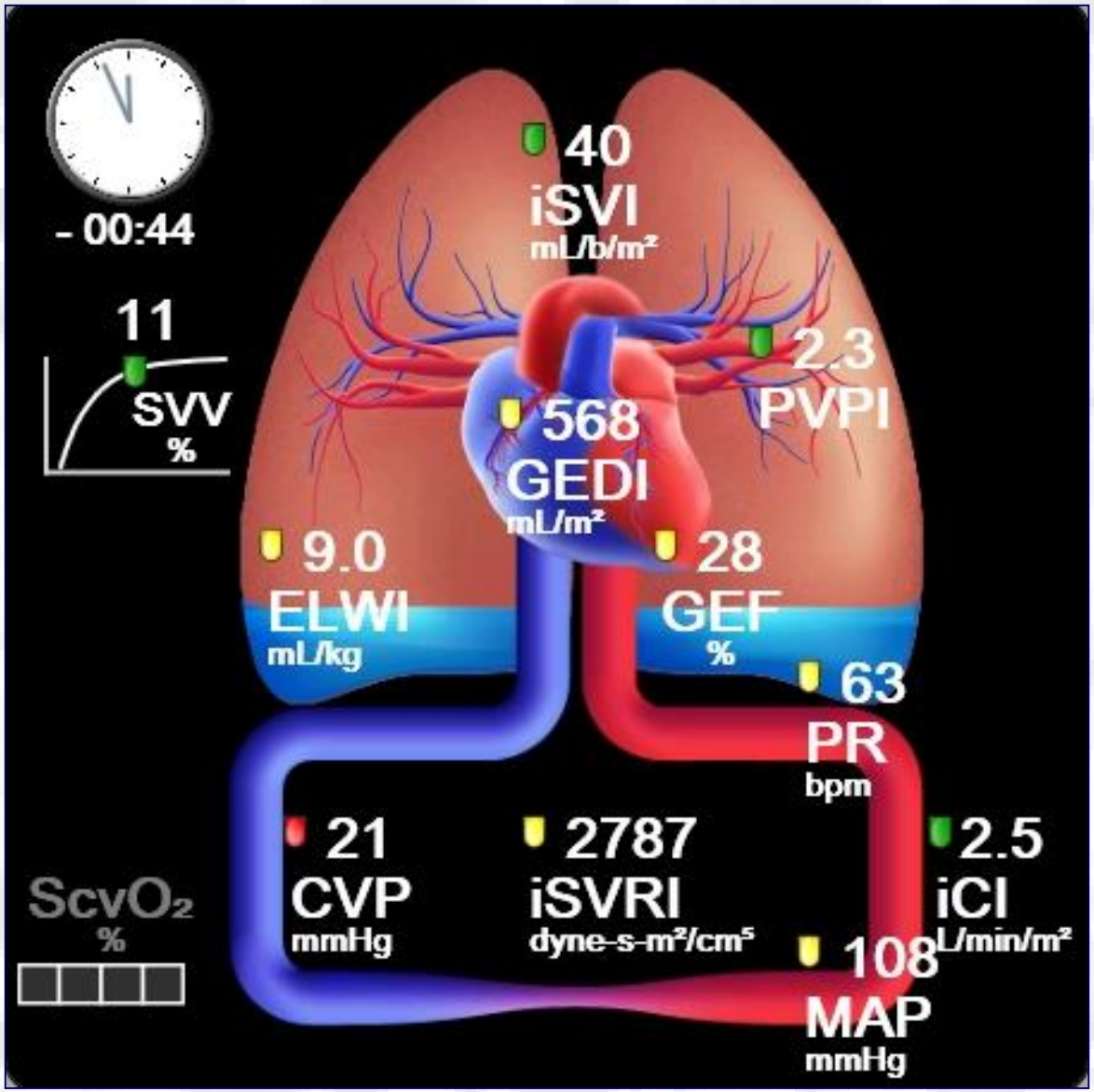
TI Polivalenti
Ricovero > 24h

Monit. invasivo gittata (N=57)	N	%
Swan Ganz	2	3.5
PICCO	53	93.0
LIDCO	0	0.0
Vigileo-PRAM	0	0.0
Altro	2	3.5
Missing	0	

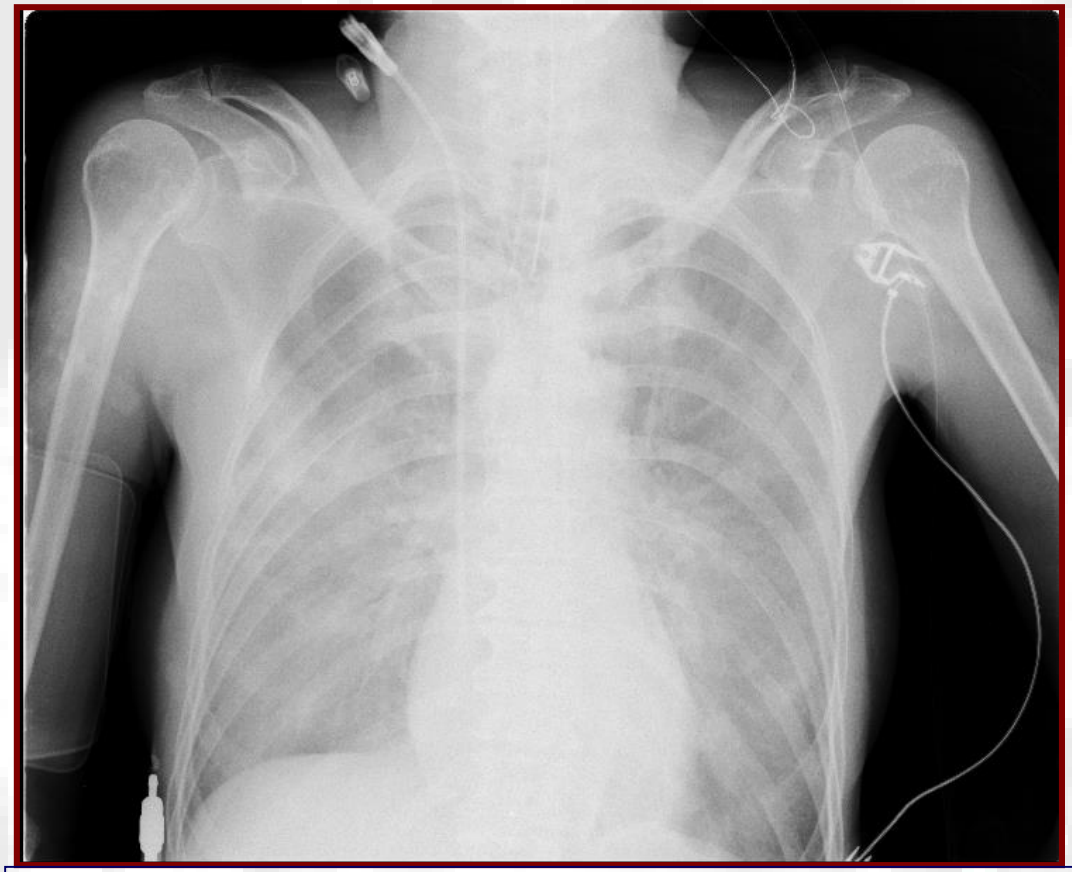
TI NCH



EV 1000/VolumeView system


- 00:44
11
SVV
%
40
iSVI
mL/b/m²
2.3
PVPI
568
GEDI
mL/m²
9.0
ELWI
mL/kg
28
GEF
%
63
PR
bpm
21
CVP
mmHg
2787
iSVRI
dyne-s-m²/cm⁵
2.5
iCI
L/min/m²
108
MAP
mmHg
ScvO₂
%

EDEMA POLMONARE NEUROGENO



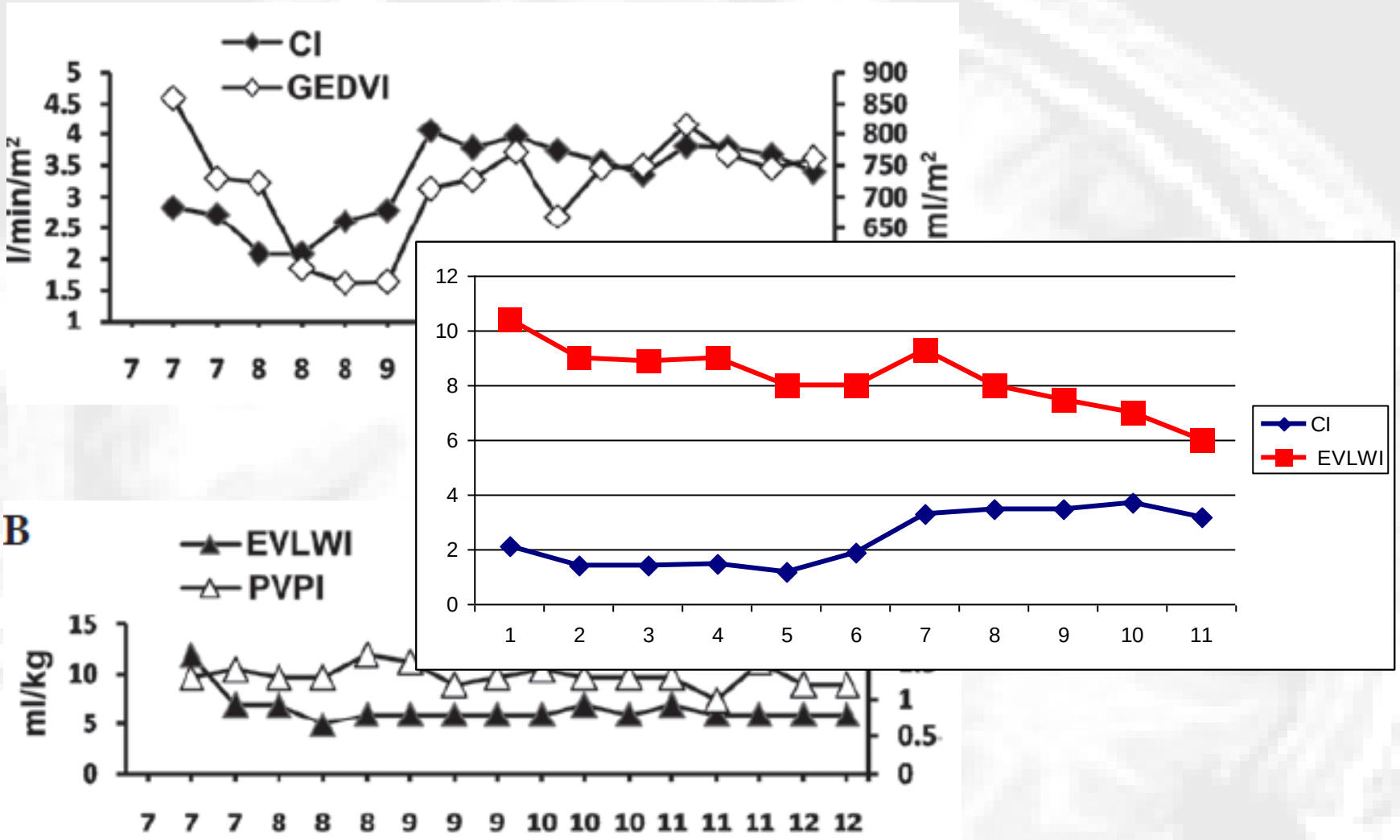
50 aa
Danno assonale diffuso

↑ GEDVI 840 ml/min

↑ EVLWI 12 ml/kg

↓ CI 2.5 l/min/m2

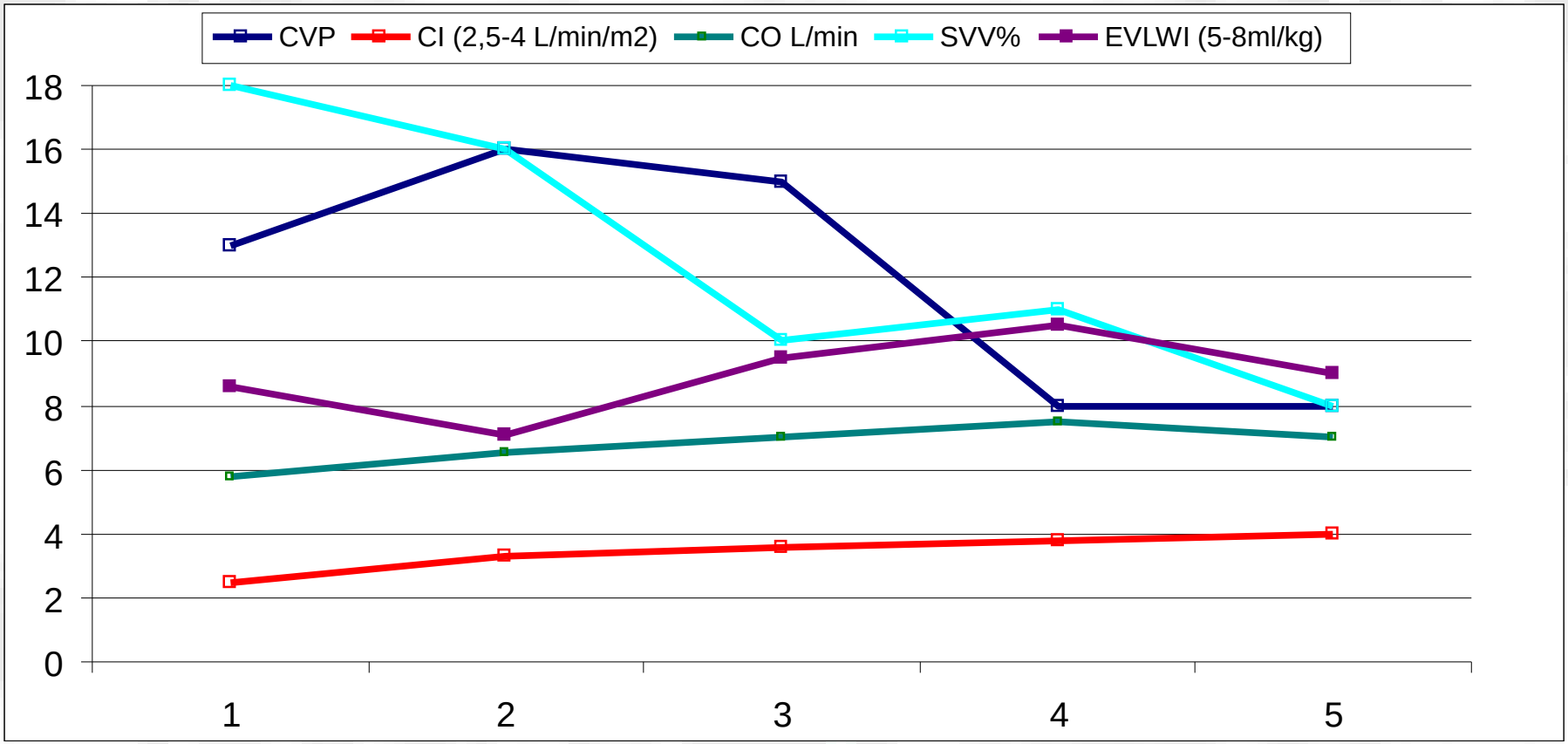
- ✓ Infiltrati bilaterali all'Rx Torace
- ✓ P/F < 200 mmHg
- ✓ Presenza di lesione cerebrale severa
- ✓ No ipertensione atriale sx
- ✓ Assenza di altre cause di distress repiratorio acuto o ARDS



Exp Therap med 2016; 12:2347

MONITORAGGIO EMODINAMICO

Monitoraggio AVANZATO



Manifestazioni sistemiche secondarie …

- SEDAZIONE ED ANALGESIA
- VENTILAZIONE MECCANICA
- IPOTERMIA
- COMA BARBITURICO

Rapporto nazionale TI neurochirurgiche - Anno 2015
Caratteristiche della popolazione in degenza - I

TI POLIVALENTI

Complicanze insorte	N	%	N	%
No	2365	70.8	20652	64.0
Sì	977	29.2	11639	36.0
Missing	24		2	

Insufficienze insorte	N	%	N	%
No	3095	91.9	27245	84.4
Sì	271	8.1	5048	15.6
A: Insufficienza respiratoria	122	3.6	1964	6.1
B: Insufficienza cardiovascolare	129	3.8	2507	7.8
C: Insufficienza neurologica	36	1.1	420	1.3
D: Insufficienza epatica	4	0.1	156	0.5
E: Insufficienza renale (AKIN)	34	1.0	1600	5.0
F: Insufficienza acuta dell'epidermide	0	0.0	6	0.0
G: Insufficienza metabolica	21	0.6	328	1.0
H: Insufficienza coagulatoria	4	0.1	226	0.7

Rapporto nazionale TI neurochirurgiche - Anno 2015
Caratteristiche della popolazione in degenza - Pazienti adulti

Complicanze insorte	N	%
Respiratorie	134	4.0
Atelettasia	51	1.5
Versamento pleurico	30	0.9
Pneumotorace/pneumomediastino	15	0.4
Patologia alte vie aeree	13	0.4
ARDS grave	12	0.4
Cardiovascolari	102	3.1
Arresto cardiaco	33	1.0
Aritmia grave acuta: tachicardie	32	1.0
Aritmia grave acuta: bradicardie	10	0.3
Crisi ipertensiva sistemica	10	0.3
Scompenso sinistro senza edema polm.	7	0.2
Infezioni	386	11.5
Polmonite	180	5.4
Inf. basse vie respiratorie NON polmonite	102	3.1
Infezione vie urinarie NON post-chir.	81	2.4
Batteriemia primaria sconosciuta	35	1.0
Batteriemia da catetere (CR-BSI)	19	0.6
Infezione delle alte vie respiratorie	13	0.4
Infezione vie urinarie post-chir.	10	0.3
Infezione del S.N.C. da device	9	0.3

7.1%

9.1%

Aritmia grave acuta: tachicardie	1208	3.7
Arresto cardiaco	695	2.2
Edema polmonare	275	0.9
Scompenso sinistro senza edema polm.	253	0.8
Aritmia grave acuta: bradicardie	223	0.7

11.9%

Manifestazioni sistemiche secondarie …

- Sedazione ed analgesia — IPOTENSIONE
- Ventilazione meccanica — POLMONITE 40%
- Ipotermia — COMPLICANZE CARDIO-POLMONARI
  INFEZIONI
- Coma barbiturico — IPOTENSIONE
  COMPLICANZE POLMONARI
  INFEZIONI
  INSUFF. RENALE EPATICA

IL MONITORAGGIO QUALE GUIDA TERAPEUTICA?

The International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care: A List of Recommendations and Additional Conclusions

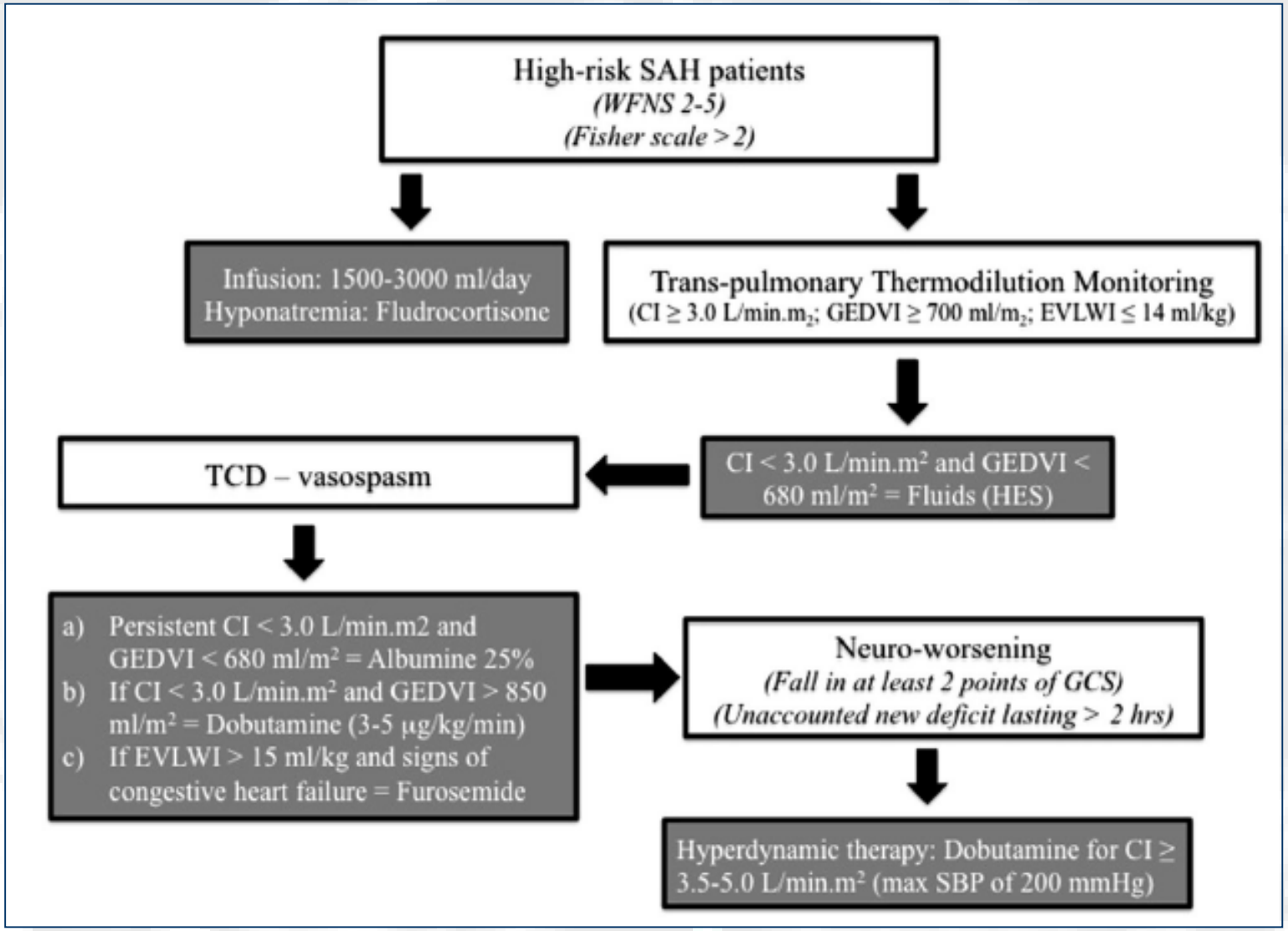
A Statement for Healthcare Professionals From the Neurocritical Care Society and the European Society of Intensive Care Medicine

Does Hemodynamic Monitoring Have a Specific Role in Optimizing Brain Perfusion and Oxygenation or Brain-Specific Therapy?

- Early optimization of cardiac index (CI) and preload during SAH-induced vasospasm can help improve regional cerebral oxygenation and CBF (very low quality of evidence).

How can Fluid Responsiveness be Assessed in Acute Brain Injury (ABI) Patients?

- Use of SVV or dICV monitoring can help predict fluid responsiveness in patients with acute brain injury (moderate quality of evidence).



Anesth Analg 2013;116:855
Neurocrit Care 2009;11:177

Cardiopulmonary haemodynamic changes after severe head injury

Haemodynamic studies were performed by pulmonary artery catheter in **15 patients** with severe head injury. …. We divided the patients into hypotensive and normotensive groups.

All patients showed a **high pulmonary vascular resistance** and a **high pulmonary capillary wedge pressure,** probably due to pulmonary vasoconstriction.

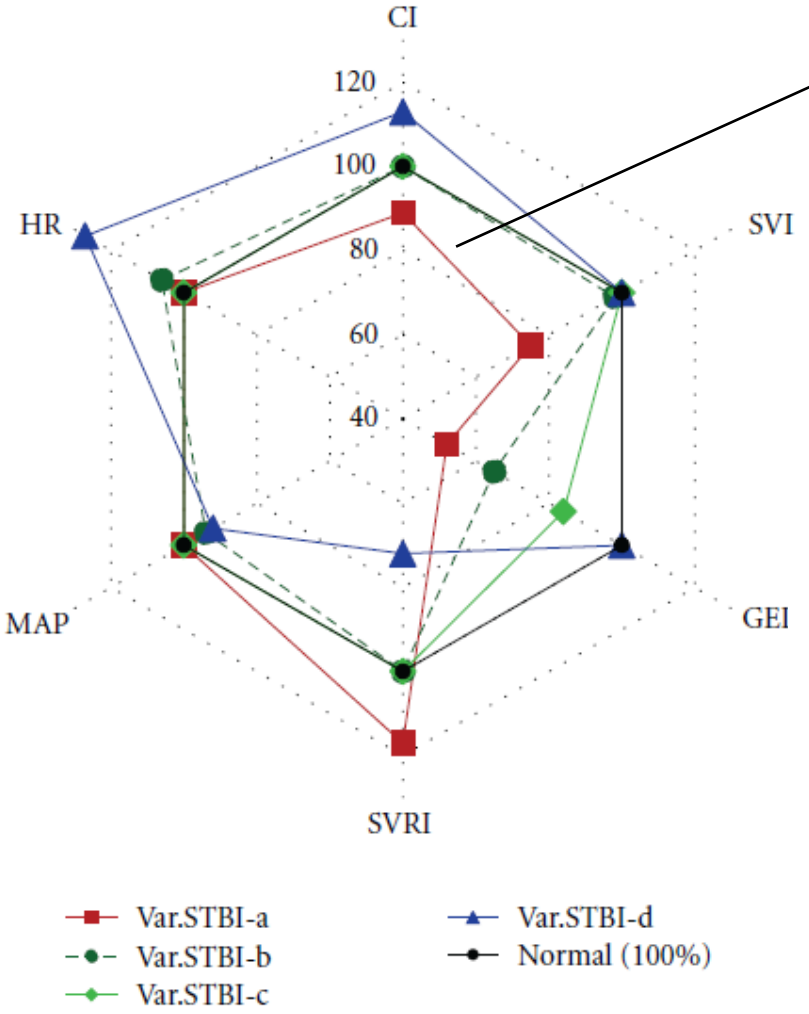
In the **hypotensive group**, the two major changes were a **marked decrease of the cardiac index** and **a slight increase of systemic vascular resistance**. The **low cardiac index** was the result of heart failure secondary to myocardial dysfunction.

In contrast, the **normotensive group** was characterized by a **high systemic vascular resistance** that was induced by generalized vasoconstriction. Increased intracranial pressure is initially associated with an increase of the cardiac index and systemic vascular resistance, so patients with severe head injury also suffer from profound circulatory disturbance.

Br J Neurosurg 2004 Apr;18(2):158

Research Article

Characteristics of Hemodynamic Disorders in Patients with Severe Traumatic Brain Injury



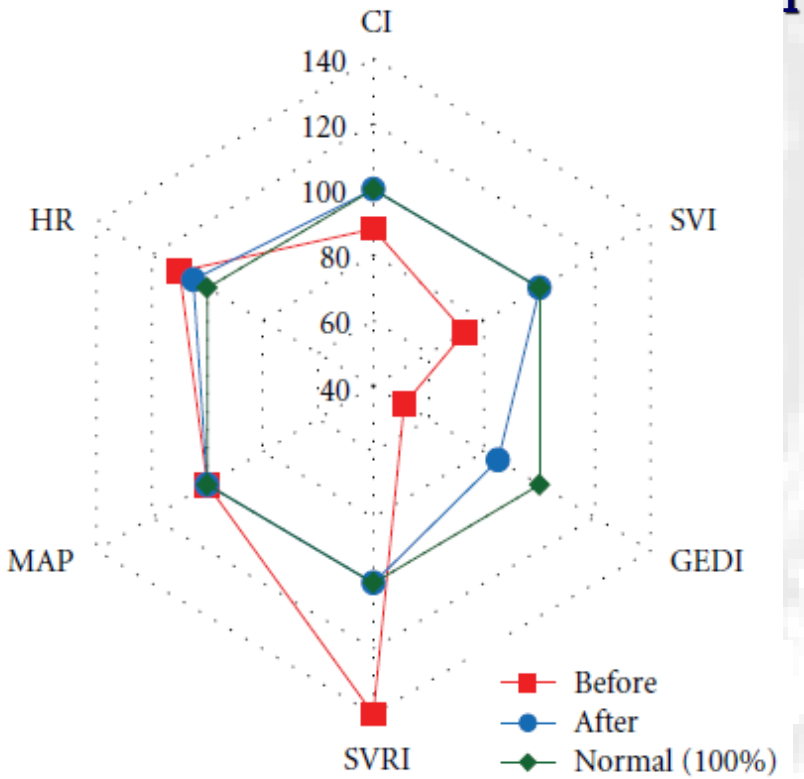
31% status ipodinamico

IPOVOLEMIA ASSOLUTA

↓ CI

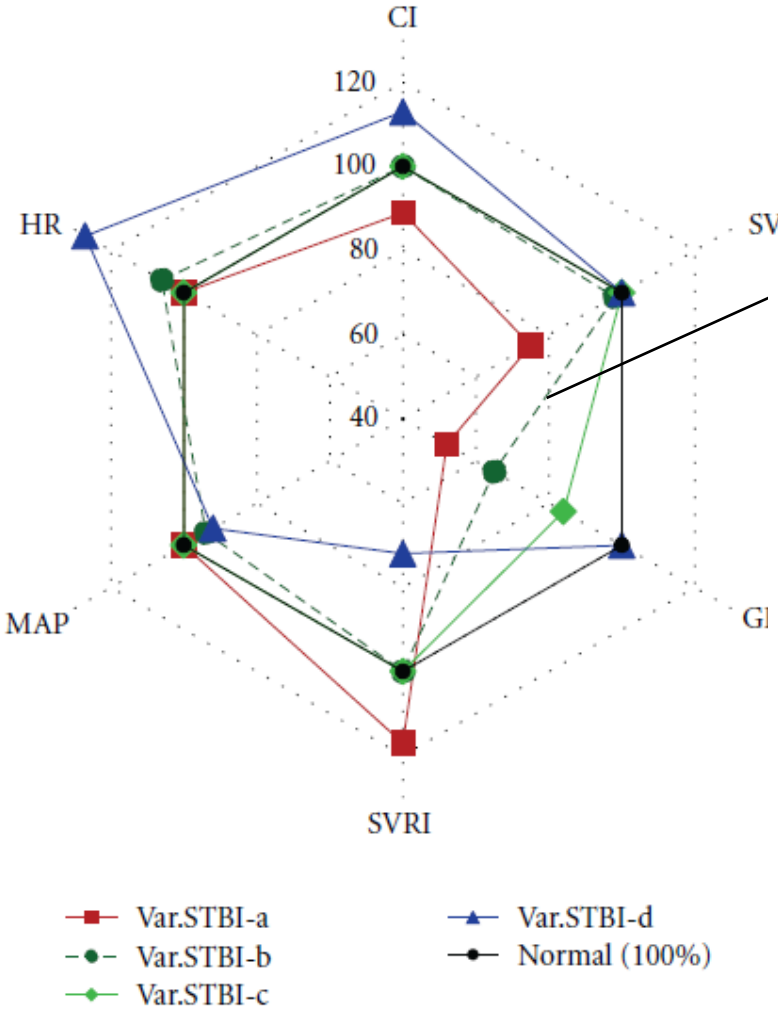
↑ SVRI

↓ GEDI



Research Article

Characteristics of Hemodynamic Disorders in Patients with Severe Traumatic Brain Injury



23 % status normodinamico

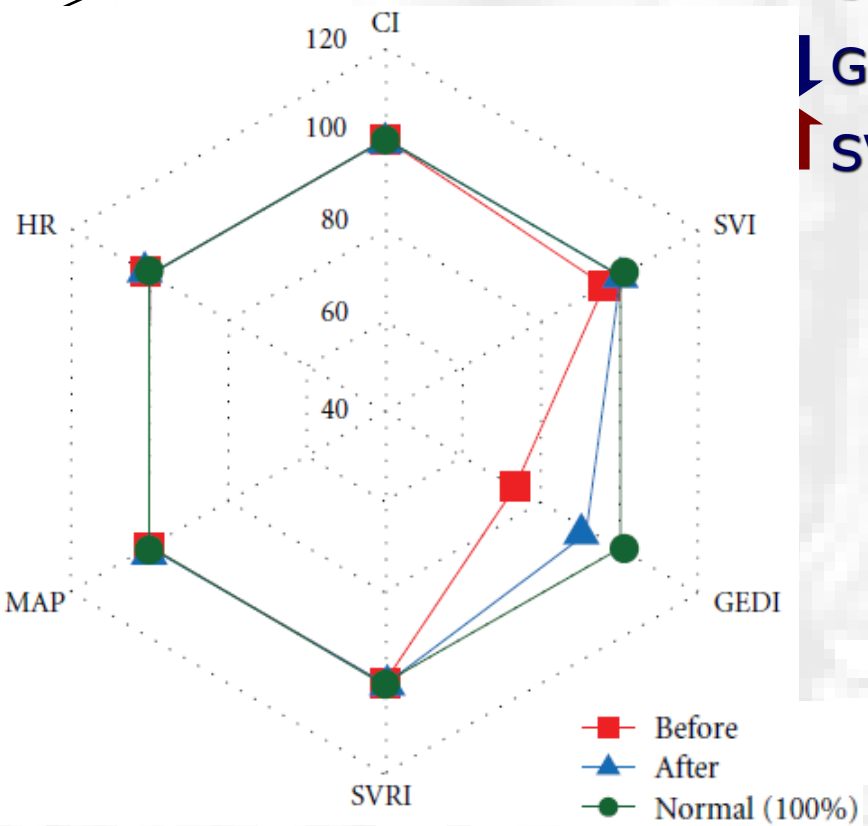
IPOVOLEMIA

= CI

= SVRI

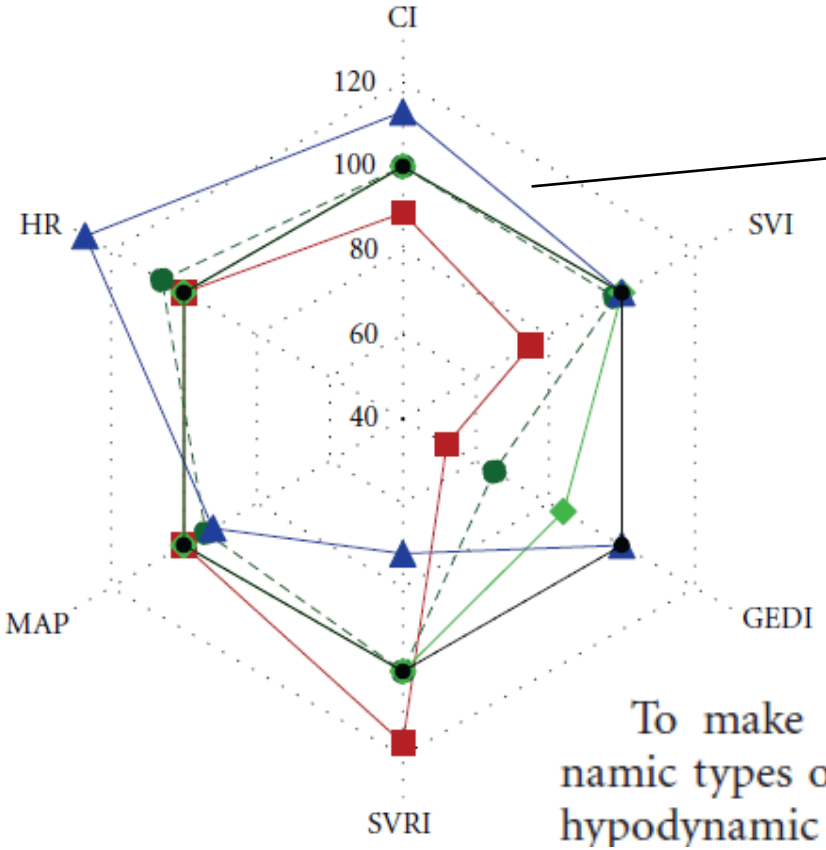
GEDI

SVV



Research Article

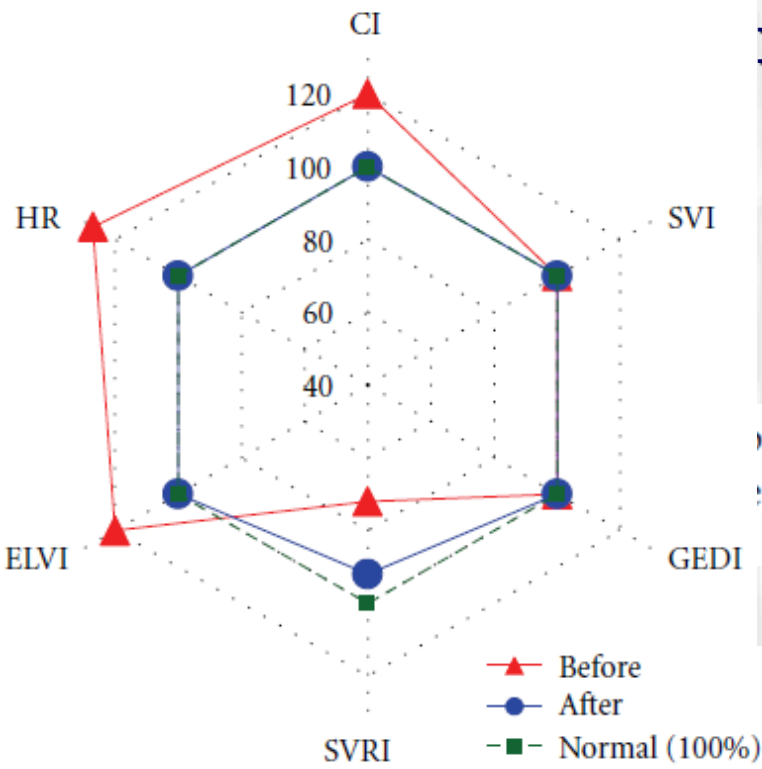
Characteristics of Hemodynamic Disorders in Patients with Severe Traumatic Brain Injury



15 % status iperdinamico

CI

SVRI



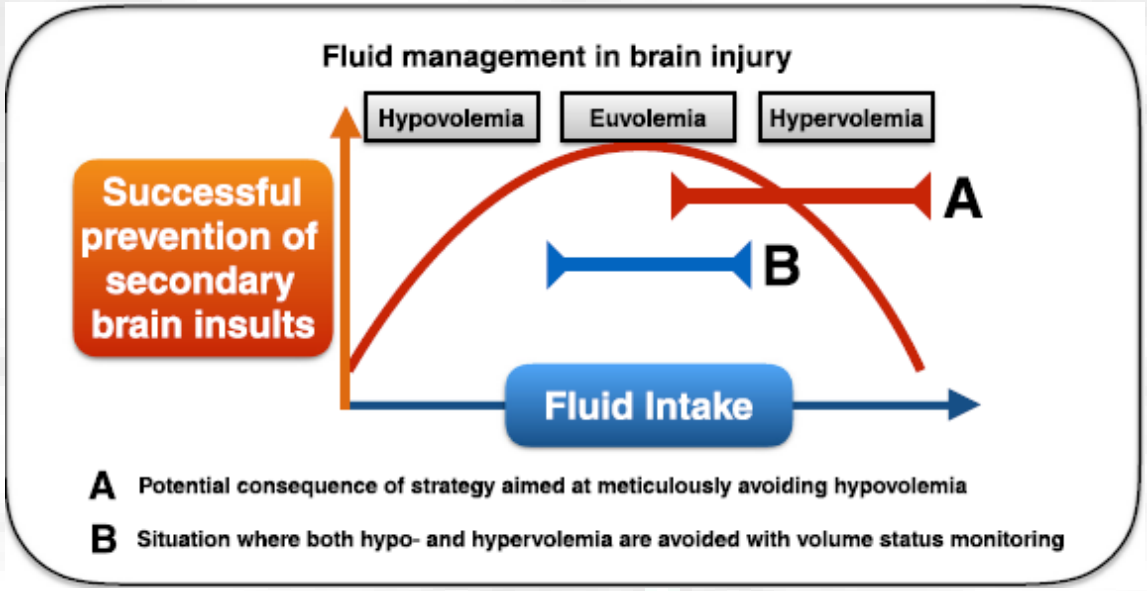
To make
namic types o
hypodynamic

erdy-
than

Var.STBI-a
Var.STBI-b
Var.STBI-c
Var.STBI-d
Normal (100%)

Fluid management of the neurological patient: a concise review

Mathieu van der Jagt



Critical Care (2016) 20:126

Fig. 3 Con
patients. Bo
clinical signi

ill brain-injured
tablish its

- Determinare la causa
- Effettuare una tempestiva correzione
- Ridurre il rischio di complicanze

QUALE IMPATTO SU MORBIDITA', MORTALITA' E OUTCOME NEUROLOGICO?

What is the Impact of Systemic Hemodynamic Monitoring and Related Therapies on Morbidity, Mortality, and Neurological Outcome?

- Use of systolic and diastolic dysfunctions to predict poor outcome after SAH is not always reliable (low quality of evidence).
- Hemodynamic monitoring-guided therapy can help reduce complications and improve outcome in SAH patients at risk for delayed cerebral ischemia (DCI) (moderate quality of evidence).
- Lactate levels on admission and lactate clearance may be used to evaluate prognosis of patients after CA (very low quality of evidence).
- $ScvO_2$-guided therapy may help improve hemodynamic stability and reduce mortality in patients after CA (low quality of evidence).

Neurocrit Care 2014 Suppl

Incidence and outcome of cardiac injury in patients with severe head trauma

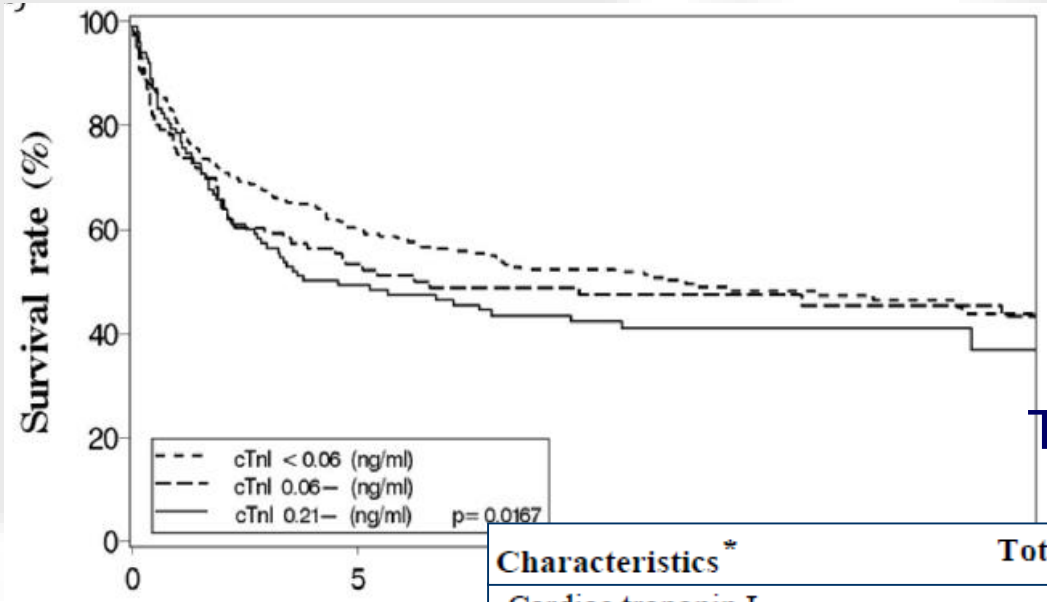
Ahmed Hasanin[1*], Amr Kamal[1], Shereen Amin[1], Dina Zakaria[1], Riham El Sayed[2], Kareem Mahmoud[3] and Ahmed Mukhtar[1]

Table 3 Risk factors for in-hospital mortality: univariate analysis. Data are presented as mean ± SD, median (IQR), and frequency (%)

	Non-survivors ($n = 18$)	Survivors ($n = 32$)	P value
Age	33.4 ± 12.7	26 ± 9.6	0.05
Male gender	29 (90.6 %)	16 (88.9 %)	1
APACHE II score	23.6 ± 4.4	16.8 ± 4.2	<0.001
GCS score	4.5 (4, 6)	7 (6, 7)	<0.001
Abnormal Echocardiography	13 (40.6 %)	2 (11.1 %)	0.052
Abnormal ECG	24 (75 %)	11 (61.1 %)	0.073
Hypotension	15 (46.9 %)	1 (5.6 %)	0.004
Troponin Day 1	1.1 ± 2.3	0.09 ± 0.18	<0.001
Elevated Troponin by Day 1	22 (68.8 %)	5 (27.8 %)	0.008
CIS	2 (1, 2)	0 (0,1)	<0.001
CIS > 1	18 (56.3 %)	1 (5.6 %)	0.001

GCS: Glasgow coma scale, *CIS*: cardiac injury score

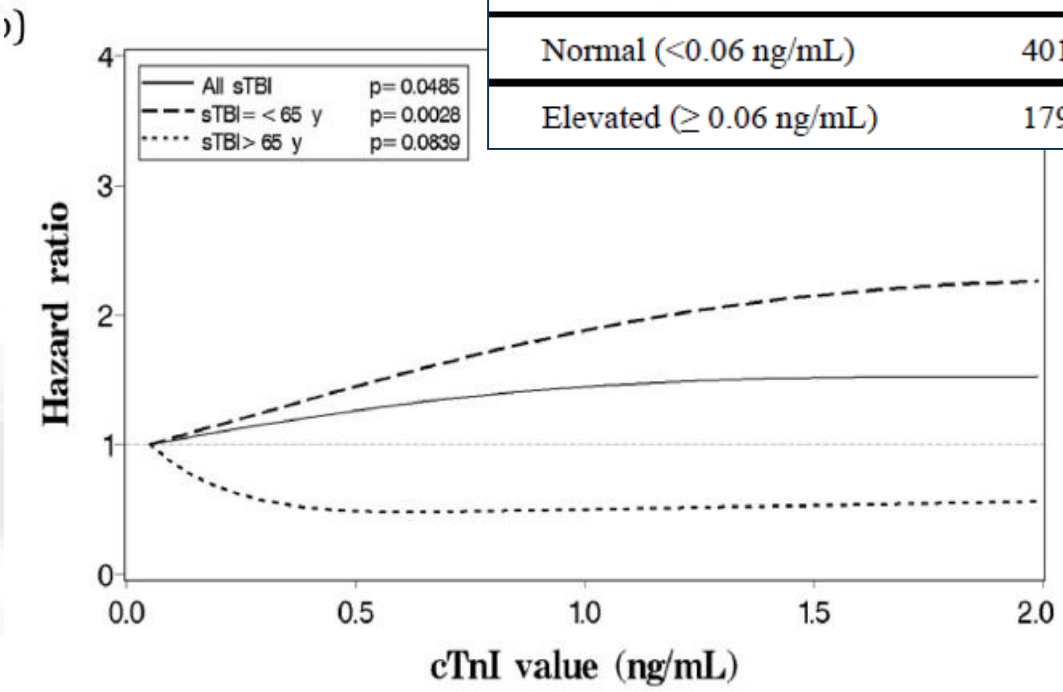
Scandinavia J Trauma 2016; 24:58



580 pts
31% troponina a 24h

TnI ≥ 0.21 ng/mL ⬆ mortalità

Characteristics*	Total (580)	Survived (293)	Expired (287)	P-value
Cardiac troponin I				
Normal (<0.06 ng/mL)	401 (69.1)	234 (79.9)	167 (58.2)	
Elevated (≥ 0.06 ng/mL)	179 (30.9)	59 (20.1)	120 (41.8)	< 0.001



Pts ≤65 aa

J Trauma Acute Care Surg 2016; 80:477

Survival, Hemodynamics, and Tissue Oxygenation after Head Trauma

Tim P Nicholls, MD, William C Shoemaker, MD, Charles CJ Wo, BS, J Peter Gruen, MD, Arun Amar, MD, BS, Alexis BC Dang, BS

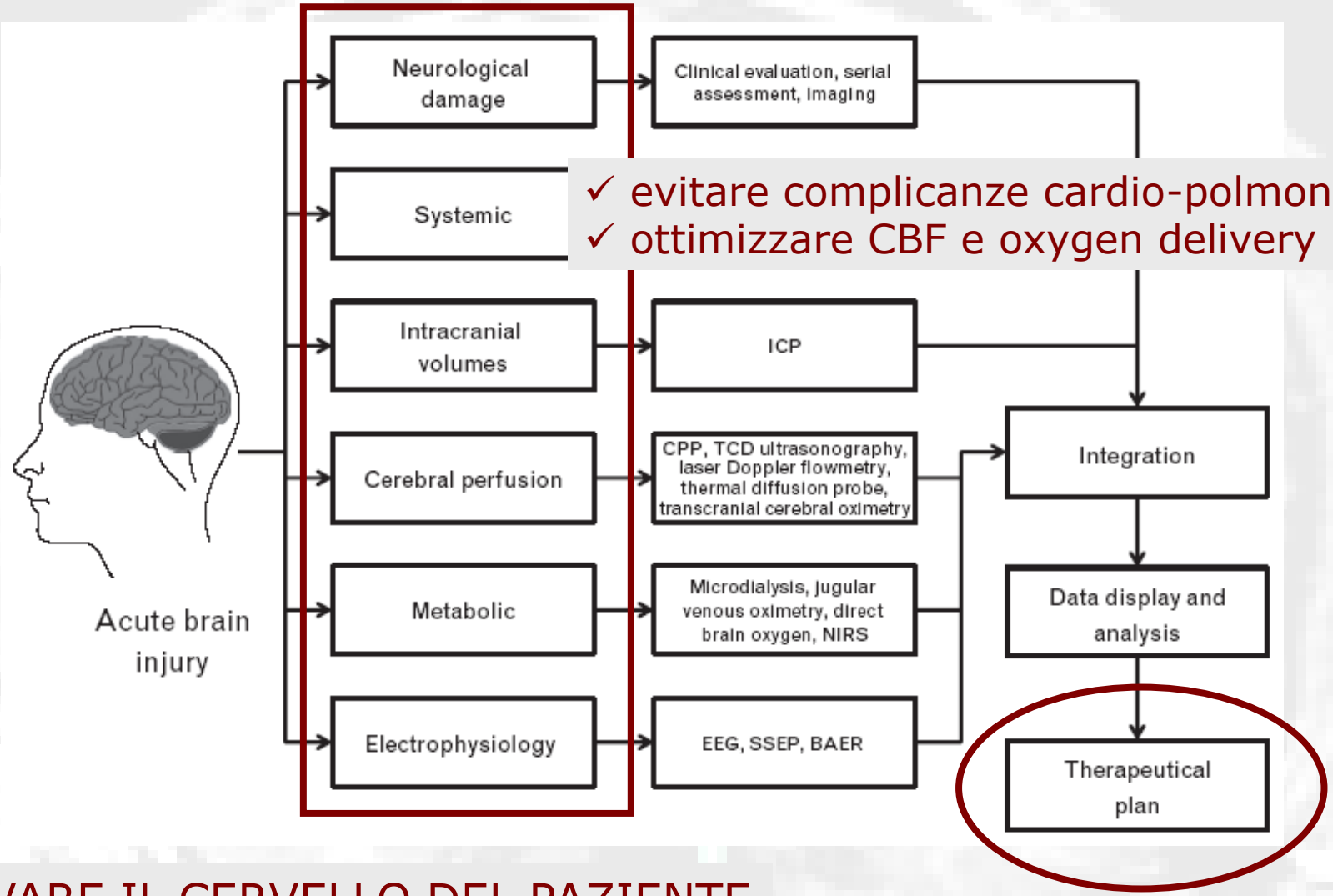
STUDY DESIGN: Sixty patients with head trauma were noninvasively monitored on arrival in the emergency department to assess the temporal hemodynamic patterns associated with head injury; patients who were brain dead were excluded because they have very different hemodynamic patterns. Cardiac index, mean arterial pressure, and heart rate were monitored to assess cardiac function, pulse oximetry to reflect changes in pulmonary function, and transcutaneous oxygen and carbon dioxide to reflect tissue perfusion function. Patients were stratified by inhospital survival outcomes, the Glasgow Coma Scale, and the presence or absence of associated somatic injuries.

RESULTS: Wh

Patient group	MAP, mmHg	HR, beats/min	CI, L/min/m²
Normal value (n = 809)	85–90	65–80	2.8–3.6
Survivors (n = 39)	92 ± 0.2	99 ± 0.3	4.02 ± 0.01
Nonsurvivors (n = 21)	74 ± .5*	99 ± 0.5	3.41 ± 0.03*
GCS ≥ 9 (n = 29)	92 ± 0.3	96 ± 0.3	3.81 ± 0.01
GCS ≤ 8 (n = 31)	82 ± 0.3*	103 ± 0.3*	4.00 ± 0.02*
Isolated head injury (n = 29)	85 ± 1	112 ± 1	4.12 ± 0.06
Multiple trauma† (n = 31)	88 ± 0.2	98 ± 0.2*	3.87 ± 0.01*

s were high
uced tissue
greater than
ormal) and
mmHg, p
atients with

CONCLUSIONS: The rial oxygen injury had normal blood flow with reduced tissue oxygenation that might have contributed to unfavorable outcomes. (J Am Coll Surg 2006;202:120–130. © 2006 by the American College of Surgeons)



- ✓ evitare complicanze cardio-polmonari
- ✓ ottimizzare CBF e oxygen delivery

SALVARE IL CERVELLO DEL PAZIENTE

USANDO IL NOSTRO CERVELLO

rent Opinion in Critical Care, 2015

...

GRAZIE

per la vostra attenzione