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HAEMODYNAMIC AND NEUROCRITICAL CARE

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SARTD-CHGUV Sesión de Formación Continuada
Valencia 4 de Diciembre de 2012

The 1990's have been declared to be 'the decade of the brain' and, unsurprisingly, there has been what can only be described as an 'avalanche' of pertinent material for review. For example, in his review on neuroprotection Dr Menon (pp. 485-496) has cited 199 references. However, he tells me that in the 12 month period covered by this issue there were more than 2000 worthy of consideration. Clearly therefore selection has been necessary, not only as far as references are concerned but, in addition, in regard to subject material. Thus as Editor for the 1998 and 1999 *Neuroanaesthesia and Neurointensive Care* sections, for Current Opinion in Anaesthesiology, I have elected to choose a number of subject areas which I hope will be of interest and divide them between the 1998 and 1999 issues.

Current Opinion in
Anaesthesiology

Fitch W. Neuroanesthesia (editorial comment). 1998;11:457-8

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HAEMODYNAMIC AND NEUROCRITICAL CARE

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1. Symptomatic Cerebral Vasospasm Following Resection of a Medulloblastoma in a Child.
Rao VK, Haridas A, Nguyen TT, Lulla R, Wainwright MS, Goldstein JL.
Neurocrit Care. 2012 Aug 22. [Epub ahead of print]
PMID: 22911499 [PubMed - as supplied by publisher]
Related citations

2. The Effect of APRV Ventilation on ICP and Cerebral **Hemodynamics**.
Marik PE, Young A, Sibole S, Levitov A.
Neurocrit Care. 2012 Jul 25. [Epub ahead of print]
PMID: 22829002 [PubMed - as supplied by publisher]
Related citations

3. Intra-arterial Dantrolene for Refractory Cerebral Vasospasm After Aneurysmal Subarachnoid Hemorrhage.
Majidi S, Grigoryan M, Tekle WG, Qureshi AI.
Neurocrit Care. 2012 Jul 20. [Epub ahead of print]
PMID: 22815125 [PubMed - as supplied by publisher]
Related citations

4. Comparison of high- and low-dose corticosteroid regimens for organ donor management.
Dhar R, Cotton C, Coleman J, Brockmeier D, Kappel D, Marklin G, Wright R.
J Crit **Care**. 2012 Jul 2. [Epub ahead of print]
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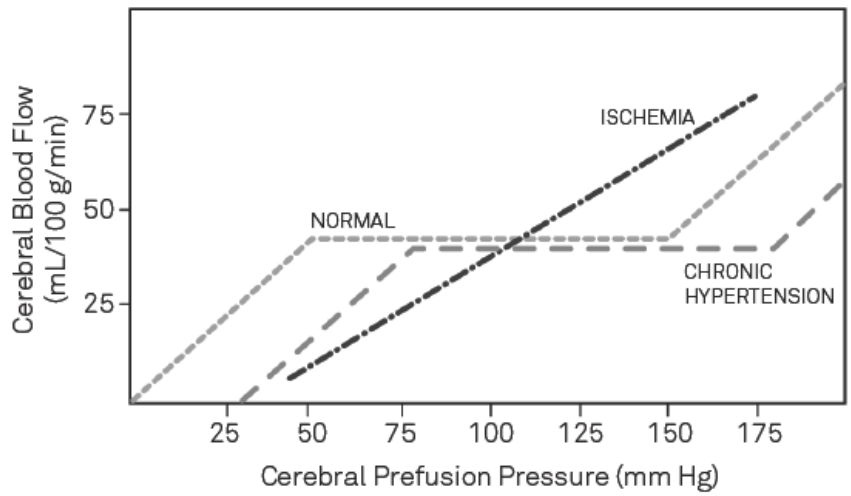
Cerebral hemodynamics: concepts of clinical importance



CBF: cerebral blood flow.

Fig 2. Cerebrovascular autoregulation in health, chronic hypertension and during autoregulation loss. In chronic hypertension, values for lower and upper limits of pressure autoregulation are higher than those under normal conditions, resulting in a degree of resetting as high as 40 mmHg. Therefore, acceptable ABP levels for healthy subjects may be associated with lower CBF. Ischemia can impair cerebral autoregulation allowing CBF to vary directly with blood pressure leading to brain lesion.

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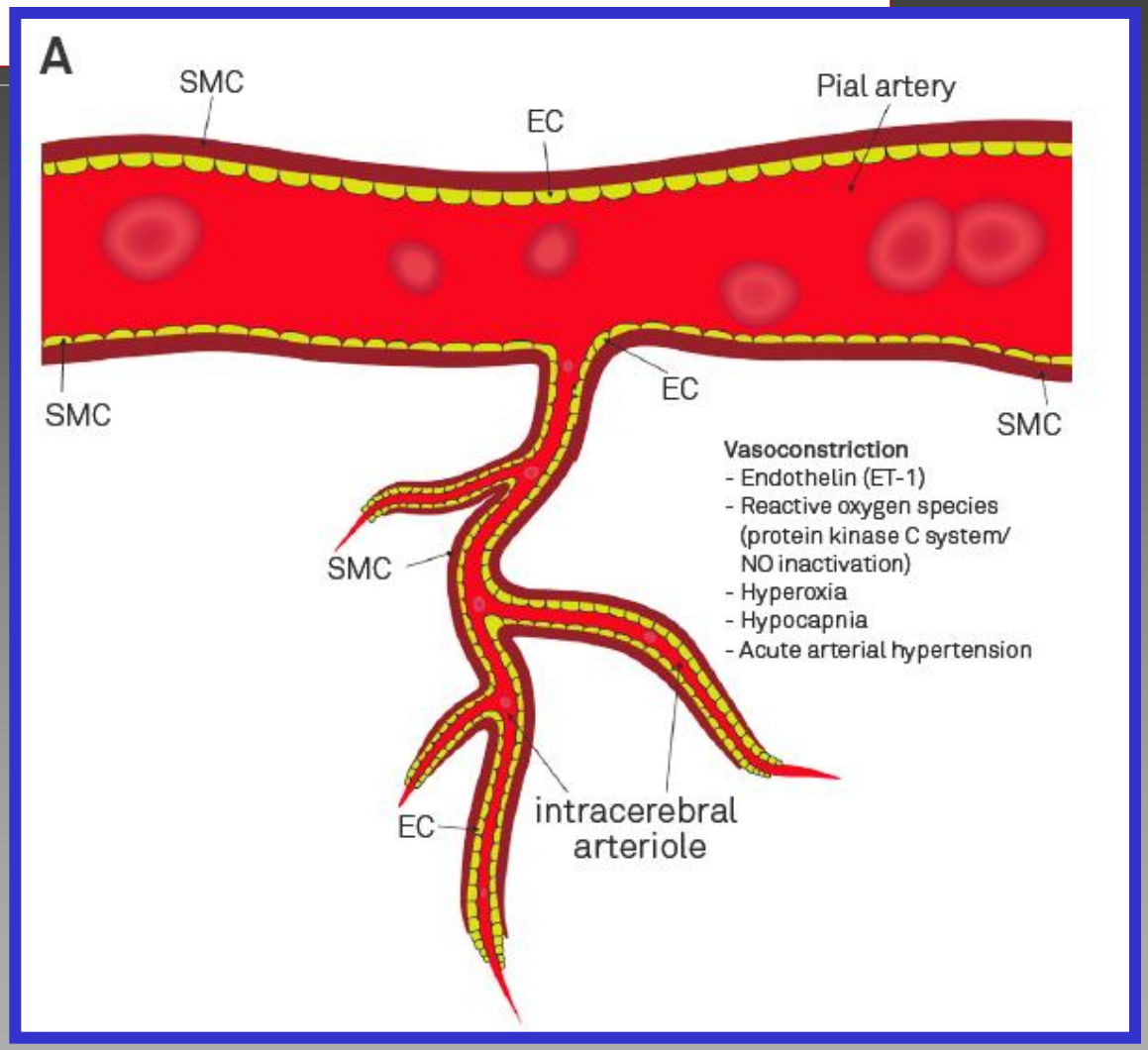
Cerebral hemodynamics: concepts of clinical importance



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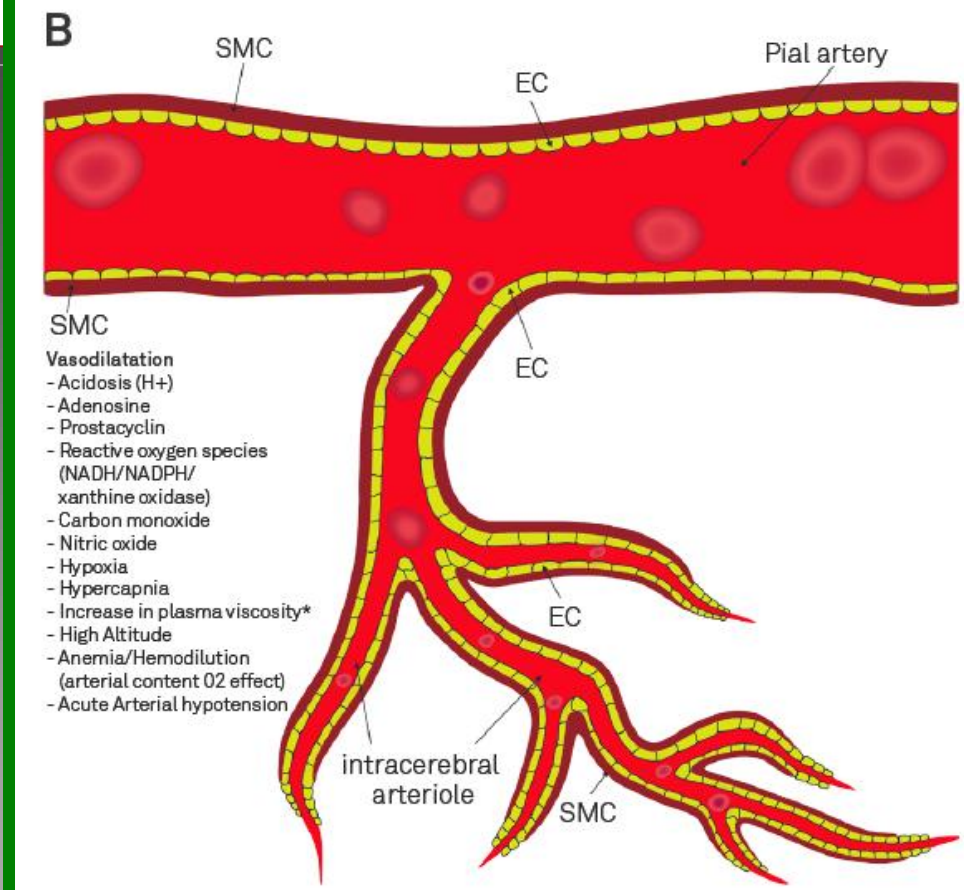
Cerebral hemodynamics: concepts of clinical importance



SMC: smooth muscle cell; EC: edothelium cells.

Fig 3. (A) factors that can induce vasoconstriction; (B) factors that can induce vasodilation.

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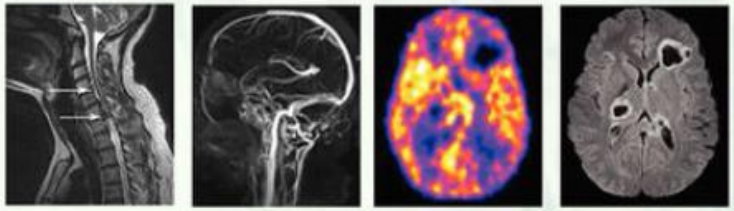
Core Topics in

Neuroanaesthesia and Neurointensive Care



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Section 4 Neurointensive care

Chapter 19

Systemic complications of neurological disease

Magnus Teig and Martin Smith

Non-neurological complications are common after brain injury and their importance as independent contributors to morbidity and mortality are well recognized.

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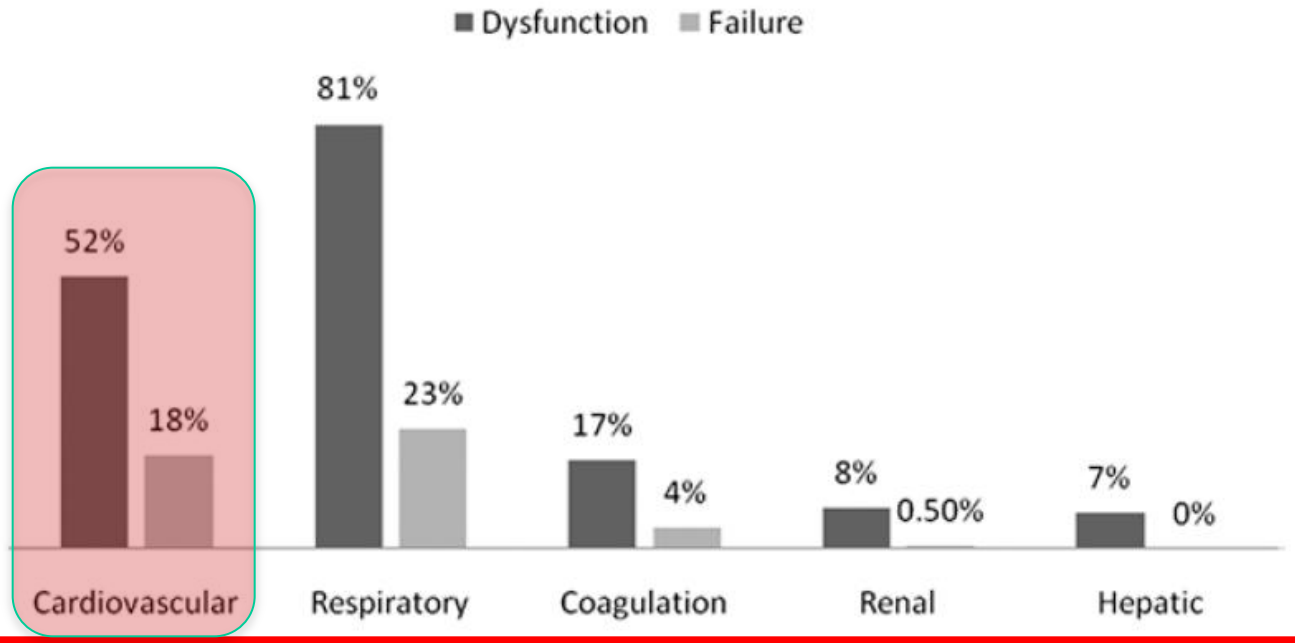

Fig. 19.1. The incidence of non-neurological organ dysfunction and failure after severe head injury.

More than 80% of patients suffer dysfunction of at least one non-neurological organ system after traumatic brain injury (TBI), with multiple organ dysfunction occurring in 60%. Organ failure develops in around 35%

Impact of Acute Lung Injury and Acute Respiratory Distress Syndrome After Traumatic Brain Injury in the United States

*Department of Neurology, Division of Critical Care, Thomas Jefferson University, Philadelphia, Pennsylvania; ‡Department of Neurosurgery, Division of Neurotrauma, Thomas Jefferson University, Philadelphia, Pennsylvania; §Department of Biostatistics, The Rothman Institute, Philadelphia, Pennsylvania; ¶Division of Cerebrovascular Diseases, Thomas Jefferson University, Philadelphia, Pennsylvania; ||Department of Neurosurgery, Stanford University School of Medicine, Stanford, California

Neurosurgery 71:795–803, 2012

During the 20-year study period, there were more than 750 million hospitalizations in the United States. The demographic characteristics and coexisting conditions in the population of admissions of patients with TBI with and without ARDS/ALI are shown in Table 1.

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Impact of Acute Lung Injury and Acute Respiratory Distress Syndrome After Traumatic Brain Injury in the United States

Neurosurgery 71:795–803, 2012

TABLE 3. Multivariate Analysis of Predictors of In-hospital Mortality After Admission for TBI[a]

	OR	Low	High	P
Demographics				
Year of admission	0.941	0.940	0.942	<.001
Age, y	1.021	1.020	1.022	<.001
Female	0.84	0.81	0.88	<.001
Black (vs white)	0.93	0.87	0.99	.03
Asian (vs white)	0.92	0.86	0.98	.01
Hispanic (vs white)	1.08	0.94	1.21	NS
Native American (vs white)	0.99	0.79	1.26	NS
Other (vs white)	1.02	0.91	1.14	NS
Hospital characteristics				
Rural (vs urban-academic)	0.74	0.68	0.80	<.001
Urban private (vs urban-academic)	1.45	1.40	1.49	<.001
Midwest (vs Northeast)	1.08	0.94	1.07	NS
South (vs Northeast)	1.05	0.99	1.11	NS
West (vs Northeast)	1.05	0.99	1.11	NS
Medium (vs small)	1.69	1.58	1.81	<.001
Large (vs small)	1.87	1.75	2.00	<.001
Comorbidities				
CHF	1.44	1.34	1.55	<.001
DM	0.91	0.85	0.96	.002
HTN	2.15	1.86	2.49	<.001
COPD	0.96	0.88	1.04	NS
CRF	2.28	2.11	2.46	<.001
CLD	1.54	1.35	1.76	<.001
Cancer	4.86	3.42	6.91	<.001
Hospital complications				
ARDS/ALI	10.73	10.31	11.18	<.001
Sepsis	1.13	1.02	1.24	.022
Cardiovascular dysfunction	2.63	2.43	2.85	<.001
Renal dysfunction	2.16	1.99	2.34	<.001
Hepatic dysfunction	1.25	0.96	1.63	NS
Hematological dysfunction	2.45	2.28	2.63	<.001
Neurological dysfunction	1.94	1.81	2.11	<.001

Table 19.1 Non-neurological complications after subarachnoid haemorrhage

Complication	Total SAH population affected (%)	SAH patients with poor outcome (dead or severely disabled) affected (%)
Fever	54	75
Anaemia	36	48
Hyperglycaemia	30	48
Hypertension	27	35
Pneumonia	20	33
Hypotension	18	36
Hypernatraemia	18	34
Pulmonary oedema	15	25
Hyponatraemia	15	17

Non-neurological complications are also common after aneurysmal subarachnoid haemorrhage (SAH) (Table 19.1), with dysfunction and failure of at least one systemic organ system occurring in 80 and 26% of patients, respectively. Around 23% of deaths after SAH

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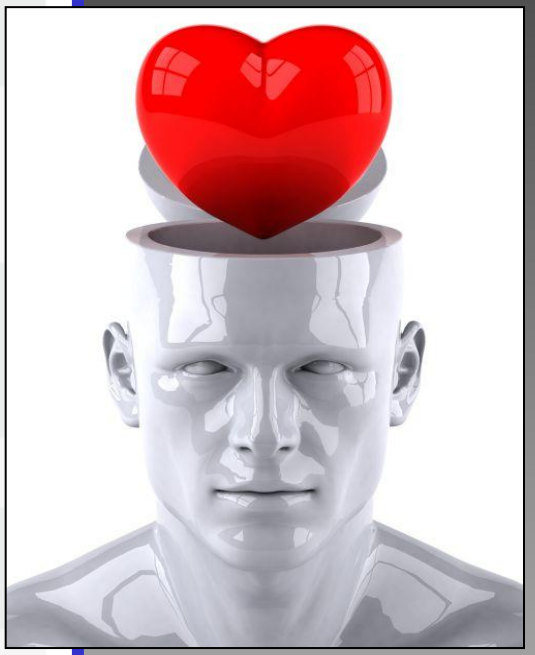
Table 19.2 Outcome effects of common systemic complications after subarachnoid haemorrhage

Complication	Odds ratio for poor outcome
Hypernatraemia (serum sodium >150 mmol l^{-1})	8.1
Hypotension (systolic blood pressure <100 mmHg treated with vasopressors)	7.1
Stunned myocardium syndrome (elevated cardiac troponin I and ECG changes)	6.2
Fever (>38.3°C)	4.4
Hyperglycaemia (blood glucose >11.0 mmol l^{-1})	4.2
Pulmonary oedema	4.2
Anaemia (haemoglobin concentration <9 mg dl^{-1})	2.5



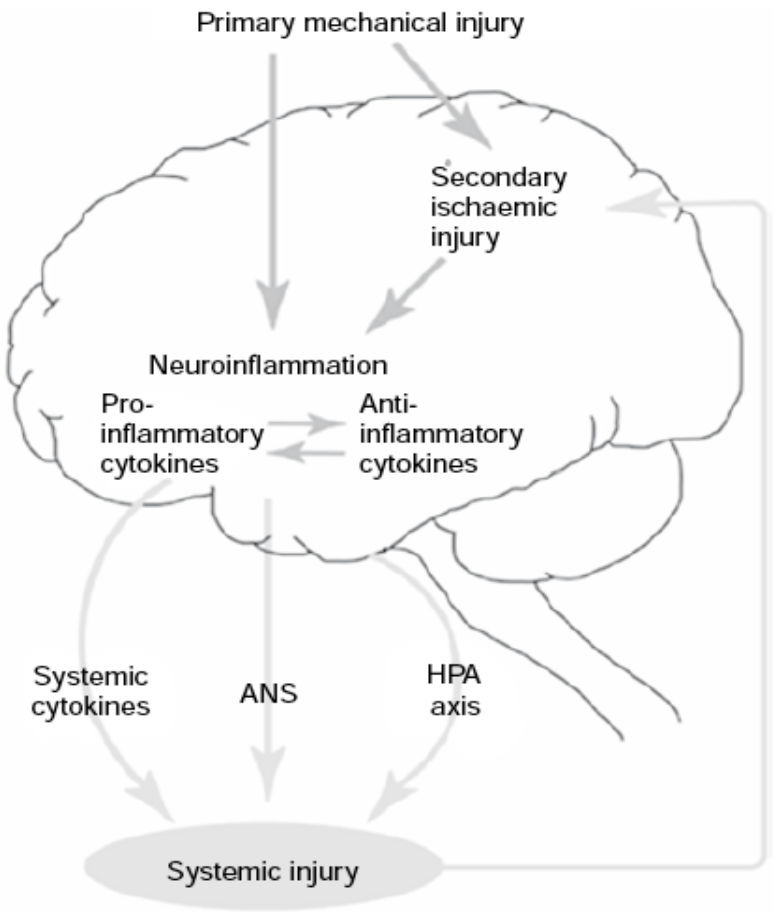


Fig. 19.4. The mechanism of systemic complications after traumatic brain injury. Primary mechanical injury and secondary ischaemic/reperfusion injury induces an acute neuroinflammatory reaction that can lead to systemic organ injury via three interrelated mechanisms: (i) overspill of intracranial cytokines into the systemic circulation; (ii) the autonomic nervous system (ANS); and (iii) the hypothalamic–pituitary–adrenal (HPA) axis. (Reproduced from Lim and Smith, *Anaesthesia*, **62**:474–482 with permission).

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Brain injury-related catecholamine release is the primary driver of neurogenic systemic complications. Plasma concentrations of norepinephrine, epinephrine and dopamine can rise to 1200, 145 and 35 times the normal levels, respectively, after SAH and may remain high for up to 10 days. The catecholamine surge is driven by the central neuroendocrine axis, which massively increases sympathetic outflow and activates the adrenal glands. It has been suggested that the catecholamine surge is a 'protective' mechanism designed to maintain cerebral perfusion in the presence of intracranial hypertension. However, it also has an adverse impact on many organ systems, with particularly important effects on the cardiovascular and respiratory systems (Fig. 19.2).

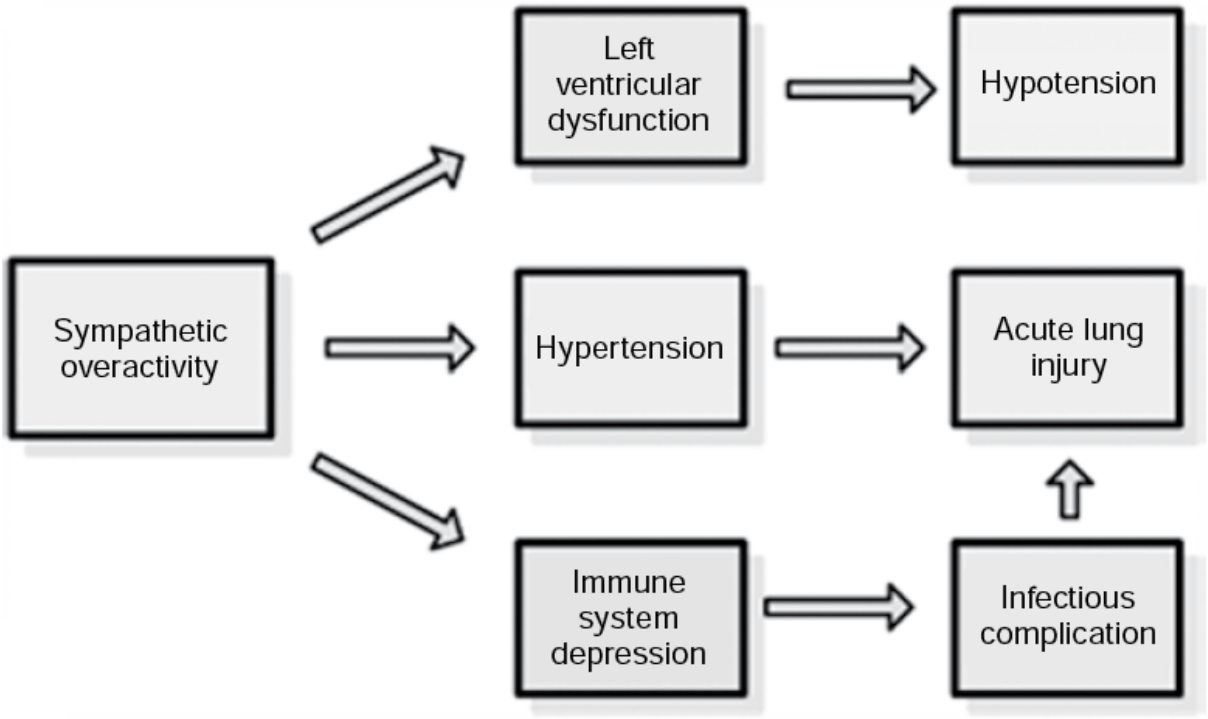


Fig. 19.2. Cardiorespiratory effects of the brain injury-related catecholamine surge (Reproduced from Lim and Smith, 2007, with permission).

Neurogenic stunned myocardium syndrome

Severe brain injury can be associated with a reversible cardiac myopathy that has been termed neurogenic stunned myocardium (NSM) syndrome. It is characterized by ECG changes, reversible left ventricular (LV) dysfunction and release of biomarkers of cardiac injury in the absence of a defect in coronary perfusion. Neurogenic

Cardiovascular complications

The clinical syndrome of NSM includes a transient metabolic acidosis, widespread ECG changes, reversible LV RWMA and, in severe cases, cardiogenic shock, pulmonary oedema and death. Neurogenic stunned myocardium is more common in women than in men and its severity is related to the severity of the brain injury.

Differentiation between neurogenic myocardium and acute coronary syndromes

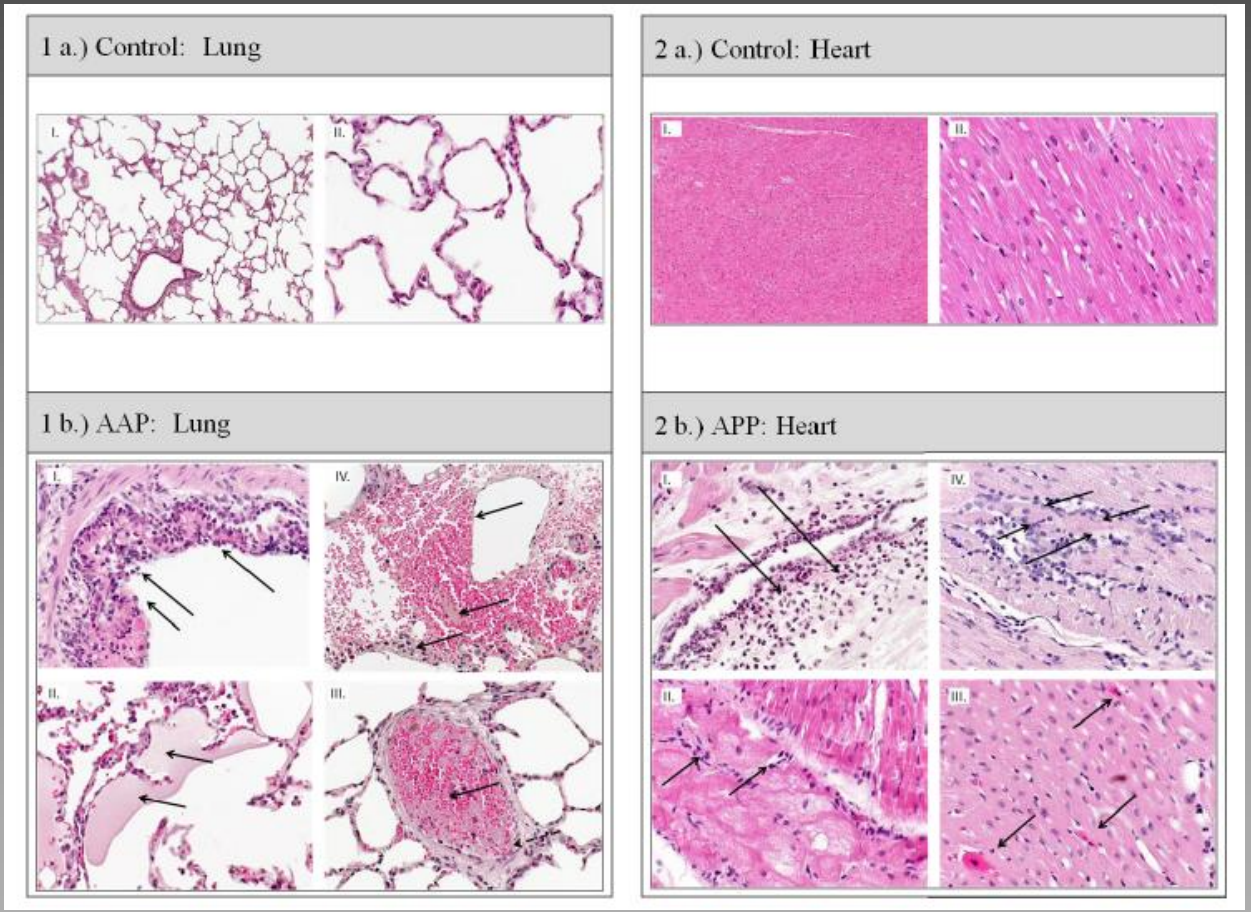
It is important for the clinician to be able to differentiate between stunned myocardium and acute coronary syndromes, but this is not always straightforward because of the common clinical and diagnostic features. Angiography is the definitive diagnostic tool

Effects of acute intracranial hypertension on extracerebral organs: a randomized experimental study in pigs.

Heuer JF, Selke M, Crozier TA, Pelosi P, Herrmann P, Perske C, Quintel M.

Department of Anaesthesiology, Emergency and Intensive Care Medicine, University Hospital Göttingen, Göttingen, Germany.

J Neurol Surg A Cent Eur Neurosurg. 2012 Sep;73(5):289-95.



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ECG changes

ECG abnormalities have been recognized after SAH for more than 50 years, but they also occur after TBI and other intracranial pathologies. In SAH, the over-

Table 19.4 ECG changes after subarachnoid haemorrhage

ECG abnormality	Reported incidence (%)
ST segment changes	15–51
Inverted or isoelectric T waves	12–92
QTc prolongation	11–66
Prominent U waves	4–47
Sinus bradycardia	16
Sinus tachycardia	8.5

ECG changes

ECG abnormalities have been recognized after SAH for more than 50 years, but they also occur after TBI and other intracranial pathologies. In SAH, the over-

malities. There is no evidence that treatment guided by morphological ECG changes improves outcome after brain injury, although anti-arrhythmic agents offer short-term benefit during periods of haemodynamic instability while intracranial hypertension is brought under control. The presence of minor ECG changes

ECG changes

ECG abnormalities have been recognized after SAH for more than 50 years, but they also occur after TBI and other intracranial pathologies. In SAH, the over-

Sudden cardiac death

Most neurogenic arrhythmias are innocuous but others carry a poor prognosis and may progress to sudden cardiac death. This is a rare event, although its true incidence is uncertain because it may be the cause of pre-hospital mortality in some patients. The exact mechanism is also unclear, but severe QTc prolongation, driven by abnormalities in the insula, may be responsible. Drugs that prolong the QTc interval

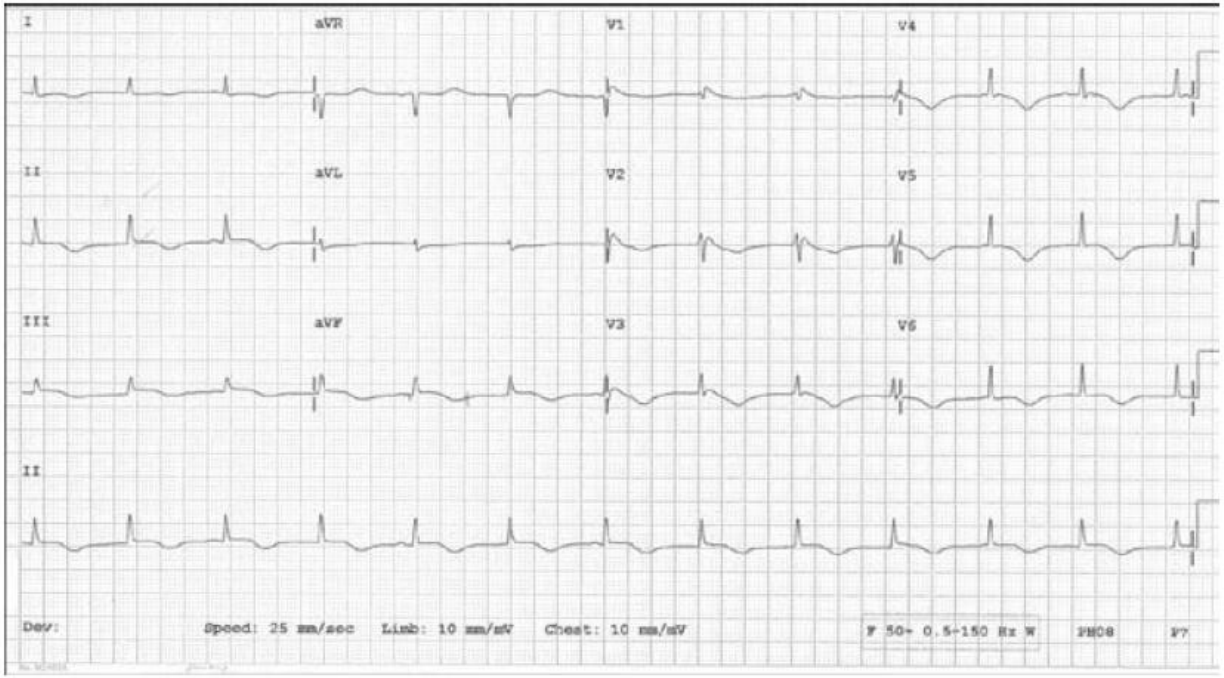


Fig. 19.5. Typical ECG changes after subarachnoid haemorrhage. Note the deep T-wave inversion and prolonged QTc interval.

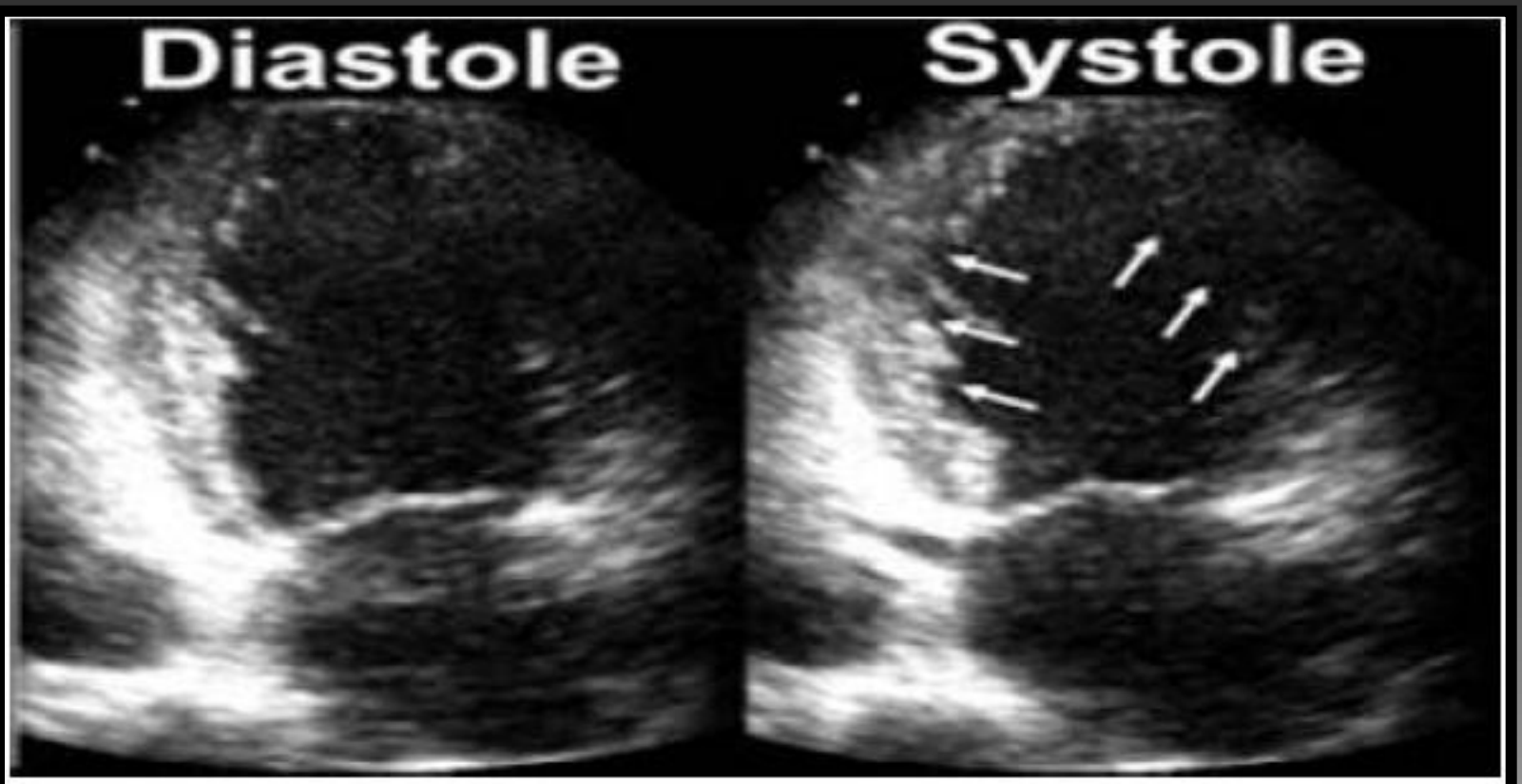
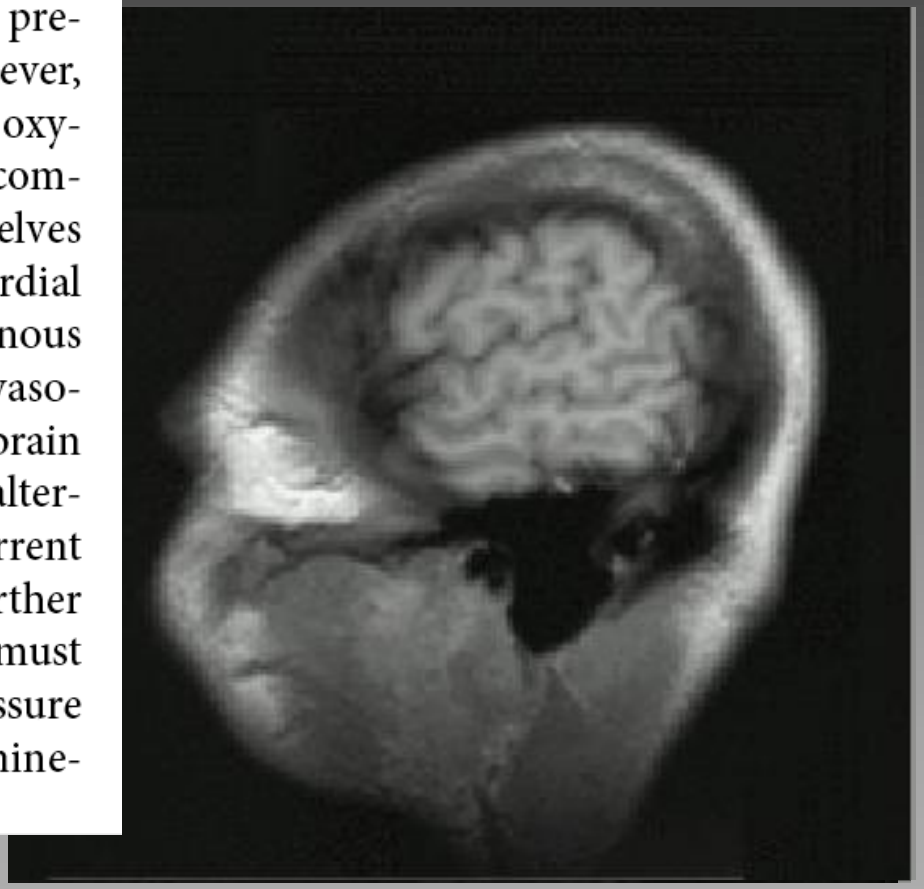


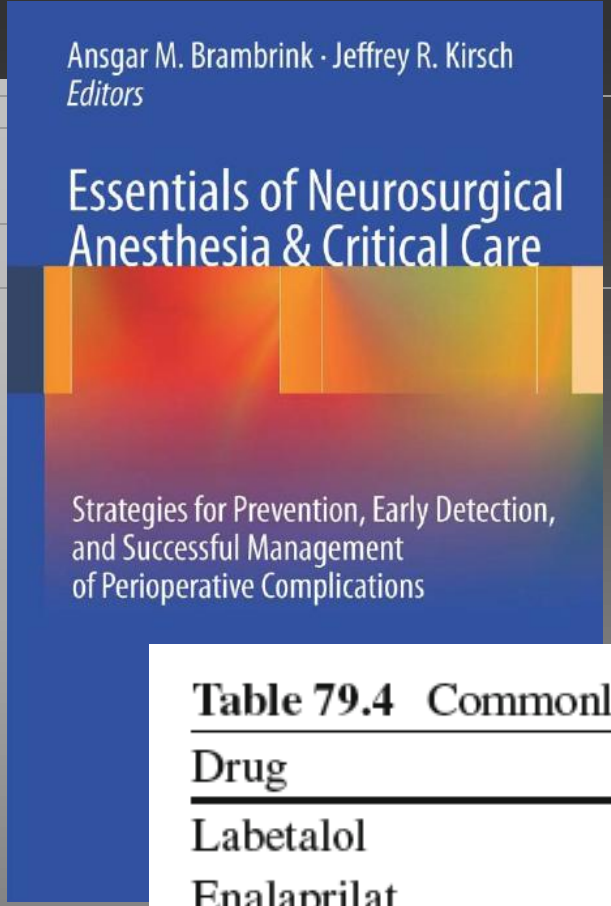
Fig. 19.6. Echocardiogram of a patient with Hunt and Hess grade 3 subarachnoid haemorrhage showing midventricular wall motion abnormalities (arrows) in the apical two-chamber view. Wall motion score was 1.9 and ejection fraction 30%. (Modified from Tanabe *et al.*, 2008, with permission.)

Blood pressure control

β-blockade might also accrue through decreased cerebral blood flow and metabolism, but this is speculative. Further studies must address the role of β-blockade after TBI and should identify the optimal type/dose and safety profile. Furthermore, it is important to establish whether post-injury treatment produces similar effects to pre-exposure. Other sympathomimetic therapies, such as magnesium sulfate and clonidine, have been investigated, but there is no clinical evidence to support their use after brain injury.

Regional wall motion abnormalities after SAH are associated with higher vasopressor requirement and this association is intriguing. Neurogenic stunned myocardium and associated events often result in hypotension and this is usually treated with vasopressors. In this context, high-dose vasopressor use is presumably a marker for more severe disease. However, sympathomimetic drugs increase myocardial oxygen demand and, if the myocardium is already compromised by NSM, vasopressors might themselves increase myocyte necrosis and worsen myocardial damage. Thus, if exogenous as well as endogenous catecholamines cause or worsen NSM, high-dose vasopressors would presumably be dangerous after brain injury and it would then be necessary to find alternative means to support cerebral perfusion. Current data cannot resolve this issue, which warrants further investigation. In the meantime, a careful balance must be struck between the need to support blood pressure and CPP and the risks of exogenous catecholamine-induced injury.





Chapter 79
Myocardial and Vascular Management in Neurosurgical Critical Care

E. Paige Gerbic and Valerie Sera

Table 79.4 Commonly used antihypertensive medications

Drug	Dose
Labetalol	5–10 mg IV q10′ as needed
Enalaprilat	
Hydralazine	0.625–1.250 mg IV q6h as needed
Esmolol	2.5–10 mg (up to 40 mg/dose) IV q4–6 h as needed
Nicardipine	0.25–0.5 mcg/kg load; 50–200 μg/kg/min

neurocritical care society Neurocrit Care
DOI 10.1007/s12028-011-9568-x

REVIEW

Advanced Hemodynamic Monitoring: Principles and Practice in Neurocritical Care

Christos Lazaridis

Goal-Directed Hemodynamic Management in the Neuro-ICU

Pressure-flow augmentation and volume status manipulation is commonly employed in the setting of prevention and treatment of delayed cerebral ischemia (DCI) after SAH and during CPP/ICP guided therapy in TBI. In SAH, this

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DOI 10.1007/s12028-011-9568-x

REVIEW

Advanced Hemodynamic Monitoring: Principles and Practice in Neurocritical Care

Christos Lazaridis

and during CPP/ICP guided therapy in TBI. In SAH, this particularly becomes relevant in patients who develop neurogenic stress cardiomyopathy or neurogenic pulmonary edema (NPE) independently or concurrently with hemodynamic augmentation for DCI [53–55]. Continuous measurement of MAP and CI becomes imperative and

Delayed cerebral ischemia is a major complication after SAH, traditionally treated with the so-called "triple-H" therapy (Hypertension, Hypervolemia, Hemodilution) [64, 65]. This regimen has never been prospectively tested in a randomized controlled fashion, and the individual components risk/benefit ratios are not well documented [66–68].

pressure [70, 71]. Use of advanced hemodynamic monitoring (AHM) could assist in optimizing these hemodynamic variables to specific clinical goals and/or direct measures of CBF and tissue parameters like brain tissue oxygenation ($PbtO_2$) and metabolic-microdialysis data. Importantly, AHM could be employed to limit the harmful effects of vasopressors or hypervolemia [69, 72].

Pulmonary edema

ELWI = 3.0–10.0 ml/kg

Lung US = A-line predominance

Hemodynamic augmentation

Normovolemic hypertension (Norepi/Neo/Dopa high-dose)

BP increments of 20%—titrate to exam, tolerance and indices of pressure autoregulation, brain oxygenation and/or microdialysis data

Preload + fluid responsiveness markers (general goals)

Primary CO augmentation (Dobutamine/Milrinone)

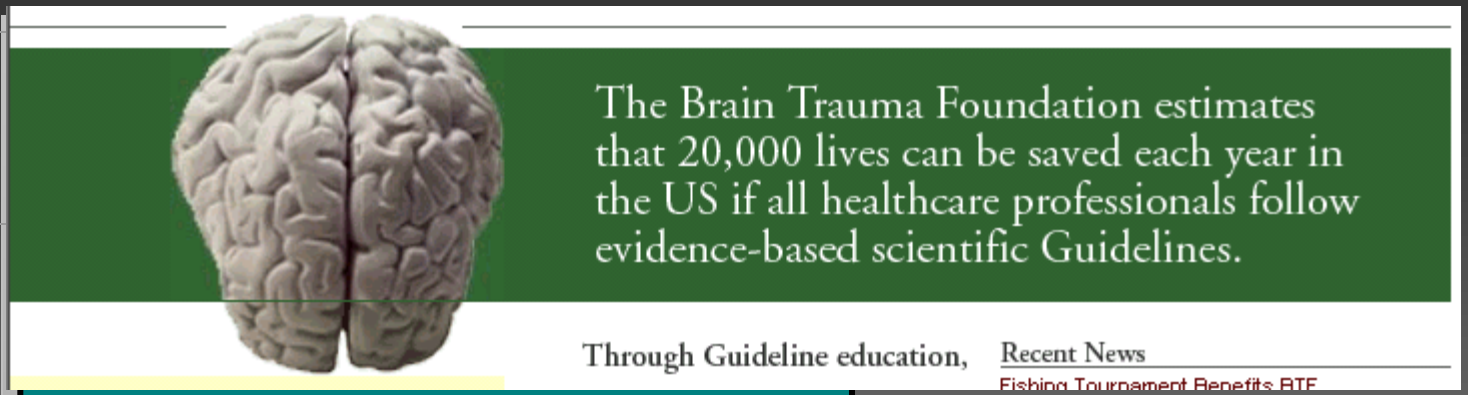
CI >3.5—titrate as above

Preload + fluid responsiveness markers (general goals)

Mild hypervolemia allowed under monitoring of ELWI and B-lines, fluid administration guided by dynamic markers

---

Management of severe TBI is centered in optimizing CPP and controlling ICP. Individualizing CPP targets taking into account the state of pressure autoregulation, brain oxygenation, and tissue metabolic parameters is encouraged [73]. On the contrary, indiscriminate, aggressive CPP augmentation is considered potentially harmful leading to ALI/ARDS [73, 74]. Fluid-volume goals, in these patients are complicated and often competing in the presence of multi-organ dysfunction; both hypovolemia and hypervolemia have been linked with unwanted effects to establish their safety and value. The addition of goal-directed regiments targeting tissue oxygenation and metabolism makes AHM an important tool in an effort to select potentially beneficial from harmful interventions and materialize better patient outcomes.



JOURNAL OF NEUROTRAUMA
Volume 24, Supplement 1, 2007

A Joint Project of the

Brain Trauma Foundation
Improving the Outcome of Brain Trauma Patients Worldwide

and

American Association of Neurological Surgeons (AANS)
Congress of Neurological Surgeons (CNS)
AANS/CNS Joint Section on Neurotrauma and Critical Care

Guidelines for the Management of Severe Traumatic Brain Injury

3rd Edition

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Valencia 4 de Diciembre de 2012

I. Blood Pressure and Oxygenation

I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

Blood pressure should be monitored and hypotension (systolic blood pressure < 90 mm Hg) avoided.

C. Level III

Oxygenation should be monitored and hypoxia (PaO_2 < 60 mm Hg or O_2 saturation < 90%) avoided.

IX. Cerebral Perfusion Thresholds

I. RECOMMENDATIONS

A. Level I

There are insufficient data to support a Level I recommendation for this topic.

B. Level II

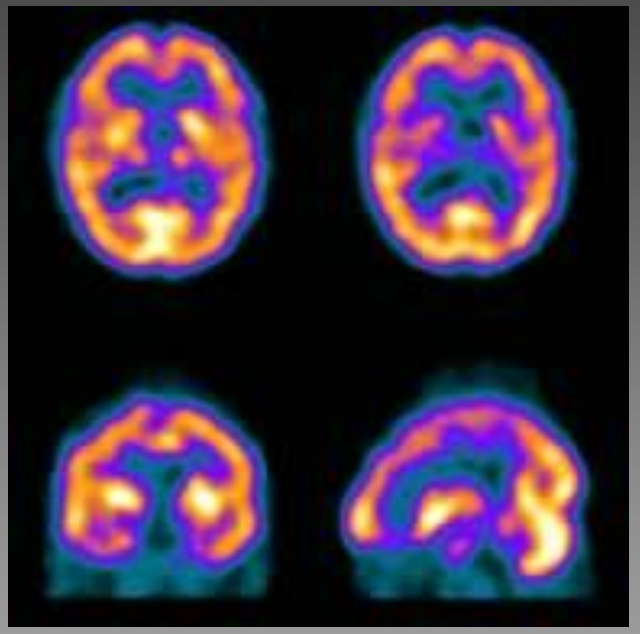
Aggressive attempts to maintain cerebral perfusion pressure (CPP) above 70 mm Hg with fluids and pressors should be avoided because of the risk of adult respiratory distress syndrome (ARDS).

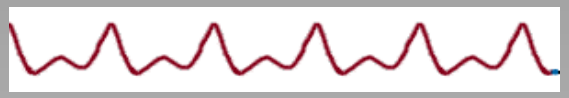
C. Level III

CPP of <50 mm Hg should be avoided.

The CPP value to target lies within the range of 50–70 mm Hg. Patients with intact pressure autoregulation tolerate higher CPP values.

Ancillary monitoring of cerebral parameters that include blood flow, oxygenation, or metabolism facilitates CPP management.





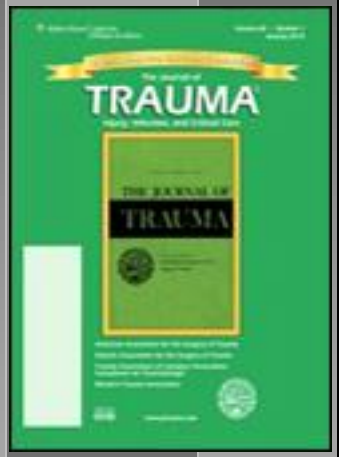
Process of Care

Impact on TBI Guidelines

Marked Improvement in Adherence to Traumatic Brain Injury Guidelines in United States Trauma Centers

Dale C. Hesdorffer, PhD, and Jamshid Ghajar, MD, PhD

J Trauma. 2007;63:841–848.



-Survey of 413 US Trauma Centers

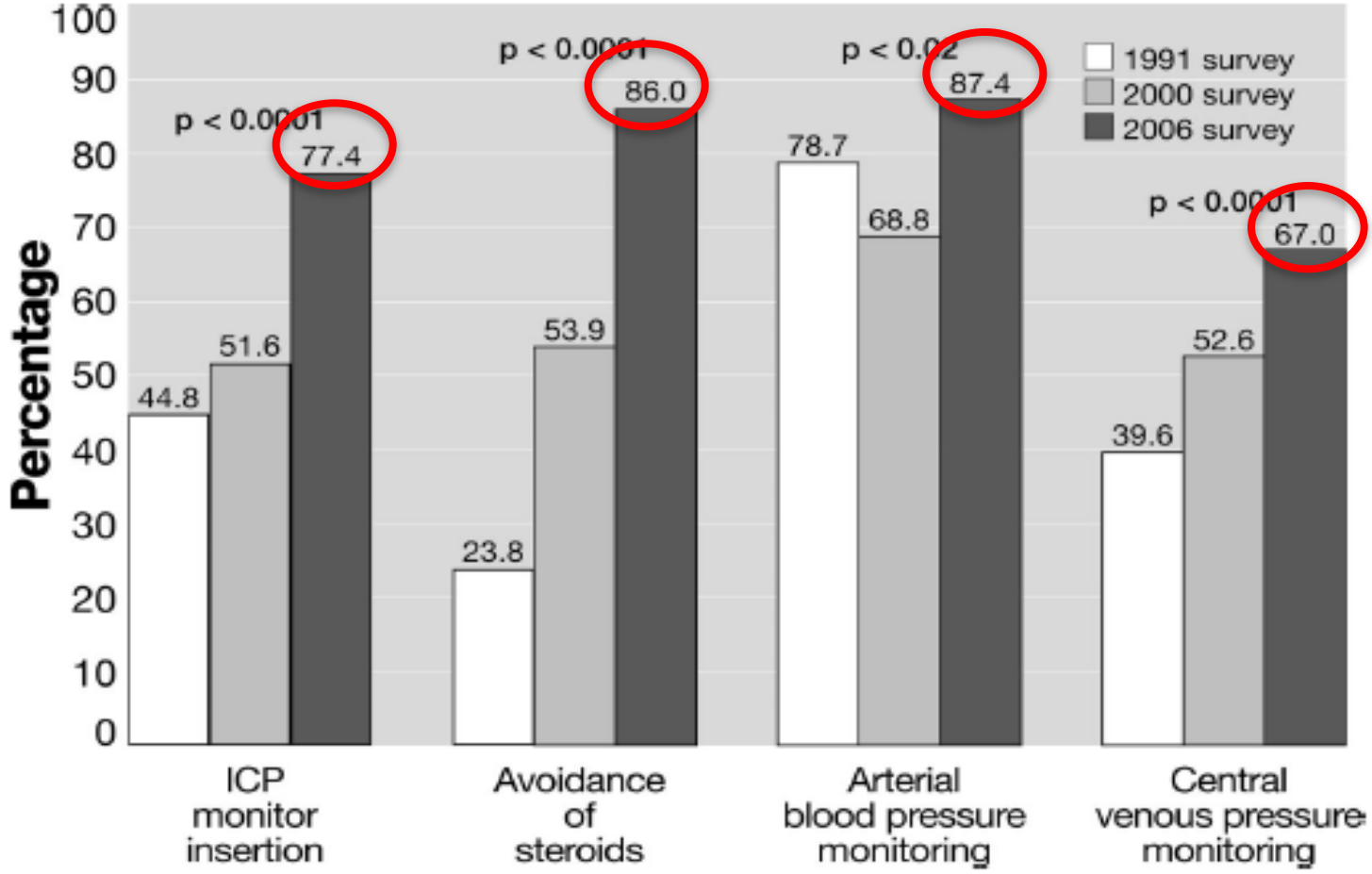
ICP monitoring rose from 32% in 1991 to 77% in 2006

Avoidance of steroids rose from 48% to 86%

Predictors of compliance: Level 1 Trauma Center designation, Presence of specific treatment protocols.

Marked Improvement in Adherence to Traumatic Brain Injury Guidelines in United States Trauma Centers

Dale C. Hesdorffer, PhD, and Jamshid Ghajar, MD, PhD



348.

Blood pressure control for acute ischemic and hemorrhagic stroke

Erin M. Grise and Opeolu Adeoye

Curr Opin Crit Care 2012, 18:132–138

Except in patients receiving thrombolytic therapy, there is insufficient evidence to recommend active BP management in ischemic stroke. In ICH, the Antihypertensive Treatment of Acute Cerebral Hemorrhage (ATACH) trial and Intensive Blood Pressure Reduction in Acute Cerebral Haemorrhage Trial (INTERACT) have demonstrated that systolic BP reduction to 140 mmHg is well tolerated and associated with attenuation of hematoma expansion. The impact of BP reduction on outcomes is being evaluated in the ongoing phase III ATACH II and INTERACT 2 trials. No evidence exists to recommend definitive BP management strategies in acute SAH, although hypertension should likely be avoided before an aneurysm is secured, and hypotension should be avoided altogether.

Blood pressure control for acute ischemic and hemorrhagic stroke

Erin M. Grise and Opec

Curr Opin Crit Care 2012, 18:132–138

KEY POINTS

- Extremes of SBP in acute ischemic stroke have a negative impact on patient outcome, although the thresholds for treatment are currently unknown and likely vary from patient to patient.
- Rapid, intensive BP reduction in ICH is well tolerated from a safety standpoint and attenuates hematoma expansion in the first 72 h, although it is unknown what effect this has on patient outcome.
- Lowering SBP below 160 mmHg may reduce the risk of aneurysmal rebleed in SAH.
- Therapeutic hypertension may be beneficial in the treatment of vasospasm after SAH, although a target SBP of 160–180 mmHg is somewhat arbitrary and should be tailored to each individual patient.

CONCLUSION

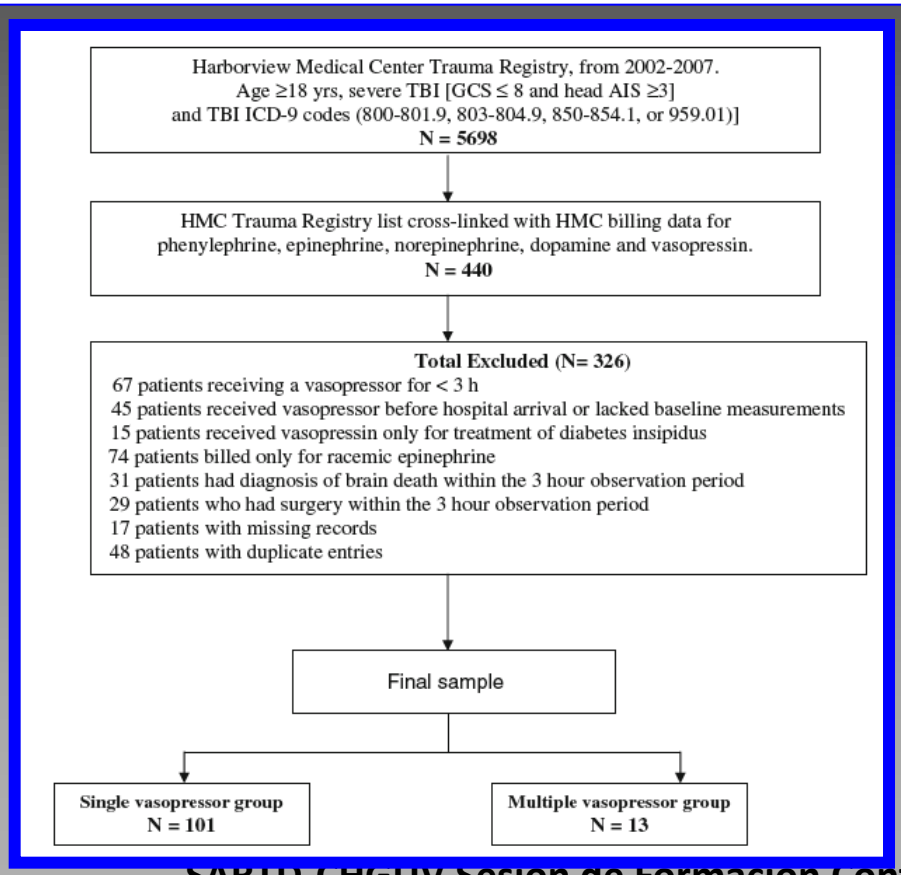
BP management in acute stroke represents a therapeutic area of growing knowledge with the potential to impact patient outcomes. For patients with acute ischemic stroke who are not candidates for thrombolytic therapy, it remains unclear whether BP reduction is beneficial. ENOS may provide further data to better answer this question. It is also unclear whether induction of hypertension is beneficial in 'relatively' hypotensive ischemic stroke patients. stroke patients are warranted. Recent early phase trials suggest intensive BP reduction in ICH may limit hematoma expansion and is well tolerated. ATACH II and INTERACT2 will hopefully provide definitive data on the effect BP reduction on ICH patient outcomes. In SAH, very little is known in regards to optimal BP management. Prospective, randomized trials on BP management in SAH are sorely needed.

neurocritical care society Neurocrit Care (2011) 15:46–54
DOI 10.1007/s12028-010-9448-9

ORIGINAL ARTICLE

Vasopressor Use and Effect on Blood Pressure After Severe Adult Traumatic Brain Injury

Pimwan Sookplung · Arunotai Siriussawakul · Amin Malakouti · Deepak Sharma · Jin Wang · Michael J. Souter · Randall M. Chesnut · Monica S. Vavilala



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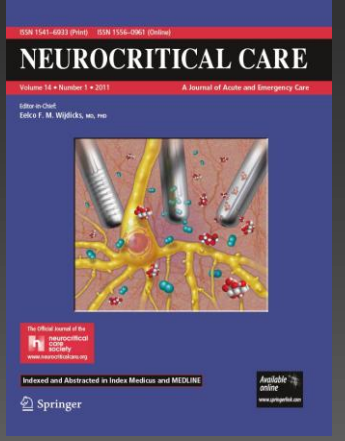

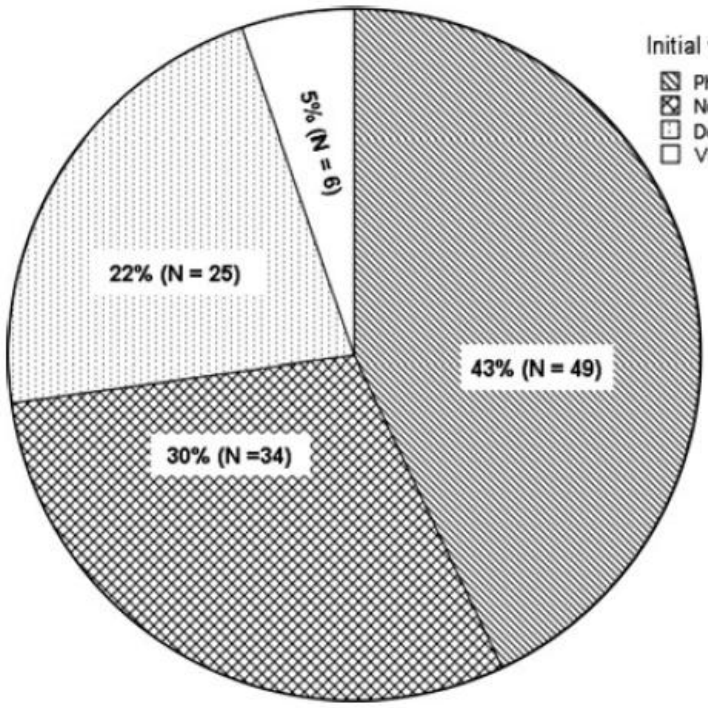

Fig. 2 Initial vasopressor used to augment blood pressure patients with severe traumatic brain injury ($n = 114$)

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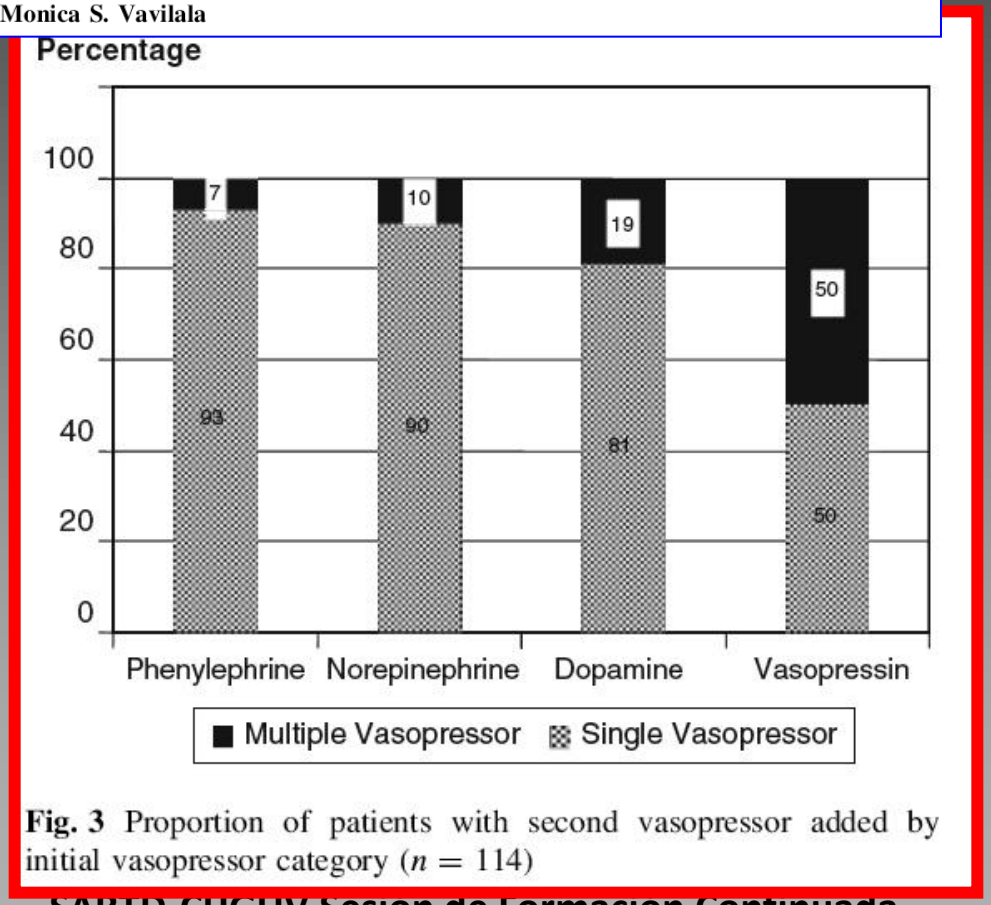

Fig. 3 Proportion of patients with second vasopressor added by initial vasopressor category ($n = 114$)

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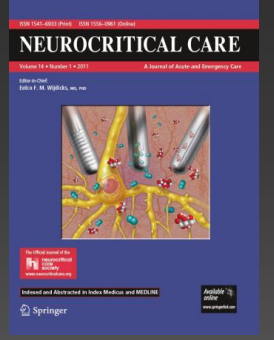

	Worst hemodynamics 1 h prior to vasopressor start	Hemodynamics during 3 h after vasopressor start	P^a	Hemodynamics at 3 h after vasopressor start	P^b
HR (bpm; $n = 99$)					
PHE ($n = 45$)	86 ± 3	80 ± 2.4	0.005**	79 ± 3	0.002***
NE ($n = 31$)	105 ± 3	101 ± 4	0.07	100 ± 4	0.09
DA ($n = 21$)	92 ± 7	100 ± 5	0.1	101 ± 5	0.2
AVP ($n = 2$)	96 ± 10	88 ± 1	–	118 ± 18	–
P	0.003*	<0.001*		<0.001*	
MAP (mmHg; $n = 101$)					
PHE ($n = 46$)	74 ± 3	86 ± 2	0.001**	83 ± 2	0.009***
NE ($n = 31$)	73 ± 2	78 ± 2	0.07	78 ± 2	0.06
DA ($n = 21$)	75 ± 4	81 ± 3	0.33	82 ± 4	0.34
AVP ($n = 3$)	87 ± 12	89 ± 10	–	95 ± 7	–
P	0.47	0.01*		0.07	
ICP (mmHg; $n = 58$)					
PHE ($n = 36$)	18 ± 2	16 ± 3	0.86	17 ± 3	0.63
NE ($n = 13$)	25 ± 4	28 ± 4	0.64	23 ± 5	0.94
DA ($n = 9$)	17 ± 6	17 ± 8	0.53	18 ± 8	0.53
P	0.08	0.35		0.26	
CPP (mmHg; $n = 56$)					
PHE ($n = 34$)	56 ± 4	67 ± 3	<0.001**	68 ± 3	<0.001***
NE ($n = 13$)	53 ± 5	53 ± 4	0.15	58 ± 5	0.44
DA ($n = 9$)	57 ± 7	64 ± 5	0.67	66 ± 5	0.41
P	0.58	0.03*		0.04*	
$PbtO_2$ (mmHg; $n = 5$)					
PHE ($n = 4$)	10.5 ± 5	22 ± 3	–	20 ± 4	–

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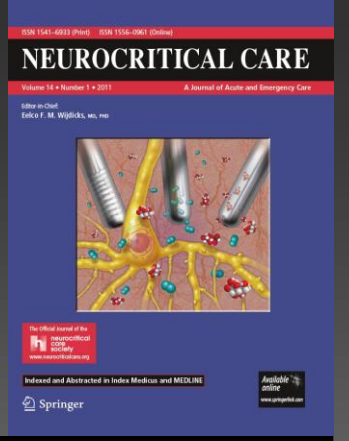

In summary, while we observed considerable variability in vasopressor use after severe adult TBI, phenylephrine was the preferred vasopressor and these patients had the greatest increase in MAP and CPP after start of infusion, compared to patients who received norepinephrine or dopamine. Prospective comparison of phenylephrine with other vasopressors is required to further understand the benefits of one vasopressor over another in severe TBI.

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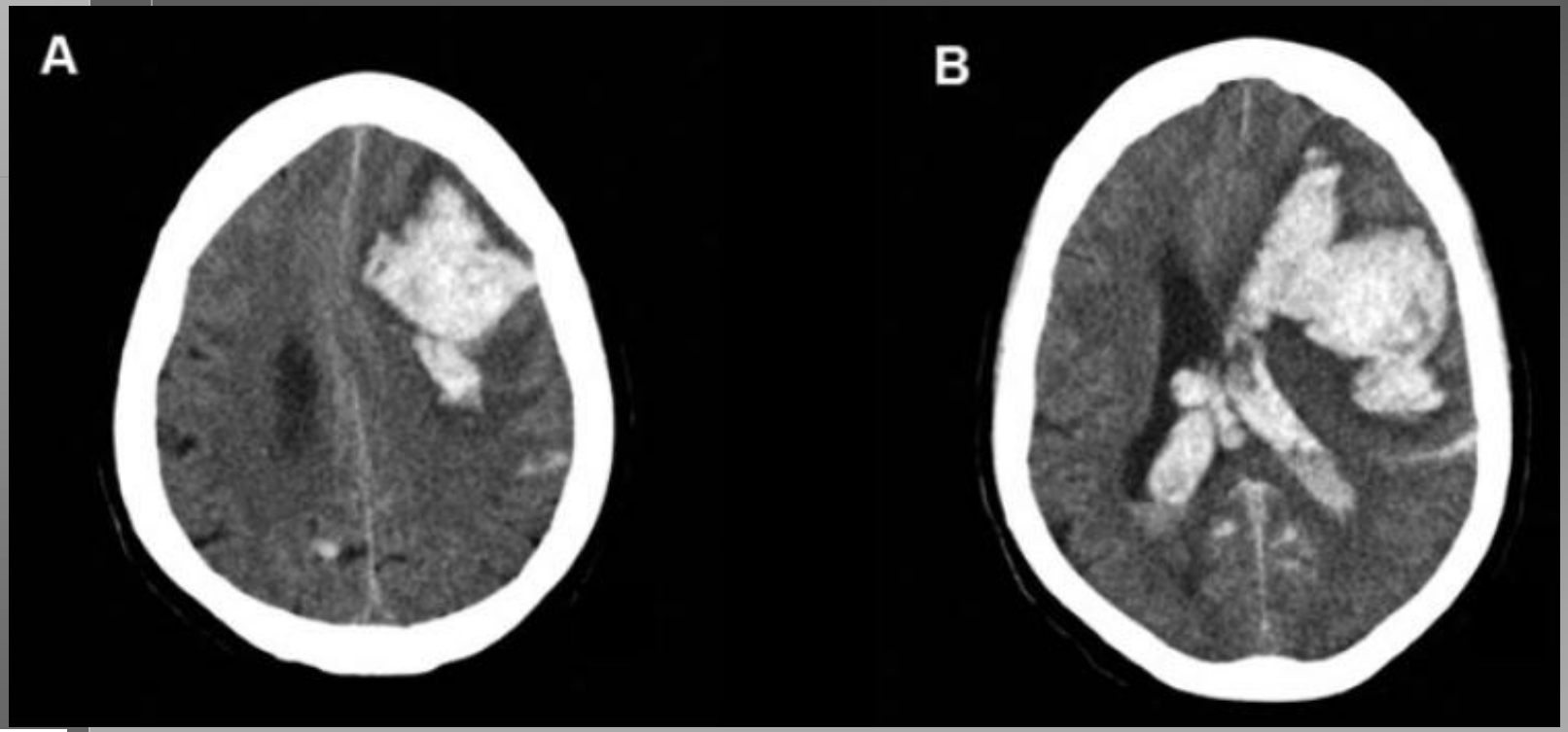
CME

The Acute Management of Intracerebral Hemorrhage: A Clinical Review

(Anesth Analg 2010;110:1419–27)

Justine Elliott, FRCA, and Martin Smith, FRCA



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CME

The Acute Management of Intracerebral Hemorrhage: A Clinical Review

(Anesth Analg 2010;110:1419–27)

Justine Elliott, FRCA, and Martin Smith, FRCA

BP CONTROL

BP monitoring and management is critical after ICH, but the targets for treatment remain controversial. Even in previously normotensive patients, hypertension is a very common finding[31] and associated with worse outcome, probably because excessive hypertension is a cause of hematoma expansion.[37,38] In a recent multicenter study, systolic BP (SBP) >140 to 150 mm Hg after ICH doubled the risk of subsequent death or dependency.[39]

The risks of a sudden therapeutic reduction in BP after ischemic stroke are well known,[40] but it is possible that

CME

The Acute Management of Intracerebral Hemorrhage: A Clinical Review

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geted management strategies. A greater understanding of the dynamic processes that occur after ICH is likely to result in the development of therapies aimed at the prevention of neurological deterioration and improve outcome by minimizing hematoma expansion, perihematoma edema, and secondary neuronal damage. An awareness of the

basis. Continuing randomized, controlled trials will clarify the correct approach to early BP management and the indications for surgical interventions after ICH. Promising

Intensive Care Med (2010) 36:1033–1037
DOI 10.1007/s00134-010-1793-9

BRIEF REPORT

Emmanuelle Mercier
Bruno Giraudeau
Guy Giniès
Dominique Perrotin
Pierre-François Dequin

Iatrogenic events contributing to ICU admission: a prospective study

Conclusion: Of admissions to the ICU, 19.5% resulted from IE, with high proportion of shock, leading to greater need for invasive treatments and longer stay in the ICU. Most cases of IE seemed preventable.

Physicians often do not recognize or appropriately treat IEs [21]. Therefore, we emphasize the role of ICU physicians, who are confronted with a high incidence of IEs, and whose units could reflect the efficiency of prevention programs.

Clinical practice guidelines for the sustained use of sedatives and analgesics in the critically ill adult

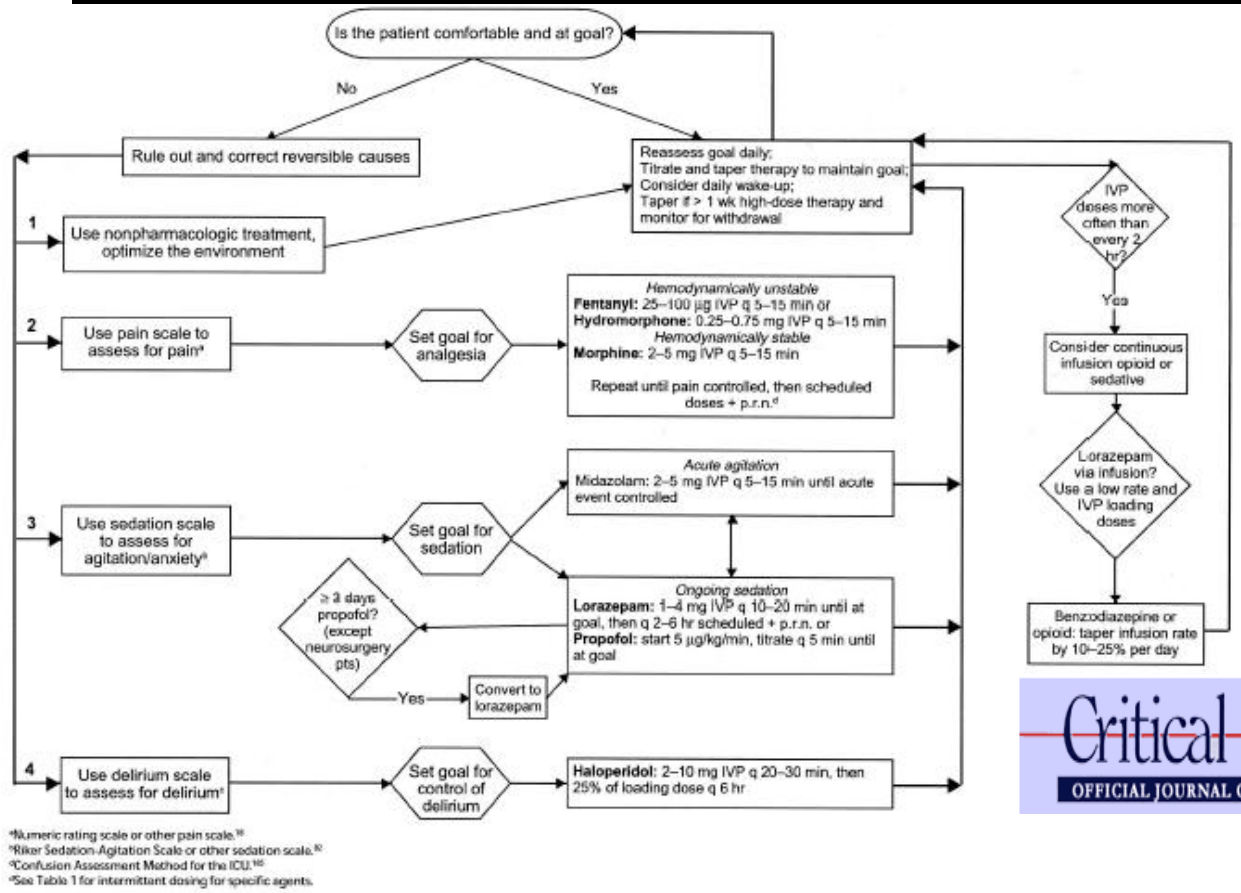

Figure 1. Algorithm for the sedation and analgesia of mechanically ventilated patients. This algorithm is a general guideline for the use of analgesics and sedatives. Refer to the text for clinical and pharmacologic issues that dictate optimal drug selection, recommended assessment scales, and precautions for patient monitoring. Doses are approximate for a 70-kg adult. IVP = intravenous push.

Critical Care Medicine
OFFICIAL JOURNAL OF THE SOCIETY OF CRITICAL CARE MEDICINE
2002;30:119-141

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Recommendations: Midazolam or diazepam should be used for rapid sedation of acutely agitated patients. (Grade of recommendation = C)

Propofol is the preferred sedative when rapid awakening (e.g., for neurologic assessment or extubation) is important. (Grade of recommendation = B)

Midazolam is recommended for short-term use only, as it produces unpredictable awakening and time to extubation when infusions continue longer than 48–72 hours. (Grade of recommendation = A)

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Clinical practice guidelines for the sustained use of sedatives and analgesics in the critically ill adult

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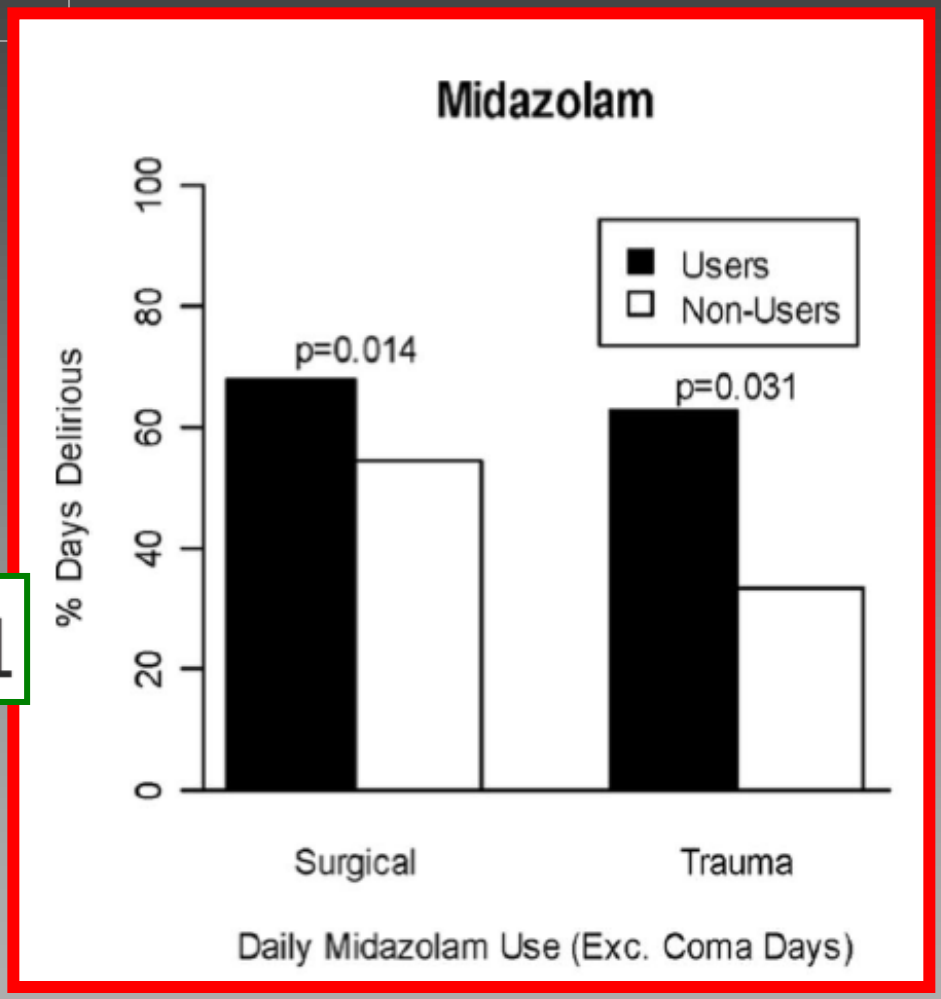
Prevalence and Risk Factors for Development of Delirium in Surgical and Trauma Intensive Care Unit Patients

J Trauma 2008;65:34-41



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Prevalence and Risk Factors for Development of Delirium in Surgical and Trauma Intensive Care Unit Patients

J Trauma 2008;65:34-41

Table 2 Multivariable Analysis of Sedative and Analgesic Medications as Risk Factors for Transitioning to Delirium

Medication	Odds Ratio (95% CI)*	$p^{\dagger}$
Surgical and trauma patients (n = 97)		
Anesthetics	0.52 (0.23–1.16)	0.108
H2 blockers	1.45 (0.80–2.62)	0.217
Lorazepam	0.45 (0.16–1.27)	0.131
Midazolam	2.75 (1.44–5.26)	0.002
Fentanyl	1.88 (0.99–3.55)	0.053
Morphine	0.36 (0.16–0.82)	0.015
Surgical patients (n = 45)		
Anesthetics	1.23 (0.37–4.04)	0.735
H2 blockers	1.71 (0.74–3.95)	0.212
Lorazepam	0.46 (0.10–2.05)	0.307
Midazolam	3.22 (1.27–8.20)	0.014
Fentanyl	3.99 (1.47–10.85)	0.007
Morphine	0.37 (0.13–1.08)	0.069
Trauma patients (n = 52)		
Anesthetics	0.18 (0.04–0.77)	0.020
H2 blockers	1.25 (0.52–3.04)	0.618
Lorazepam	0.51 (0.12–2.17)	0.360
Midazolam	2.45 (1.09–5.52)	0.031
Fentanyl	1.03 (0.47–2.25)	0.936
Morphine	0.22 (0.06–0.82)	0.024

* Odds ratios in this table can be interpreted as indicating the odds of transitioning to delirium for patients who received any dose of the given medication in the previous 24 hours, adjusted for the following baseline variables: age, body mass index, Charlson Comorbidity Index, APACHE II severity of illness score, and diagnosis of sepsis, septic shock, or ARDS.

$^{\dagger}p \leq 0.05$ considered statistically significant.

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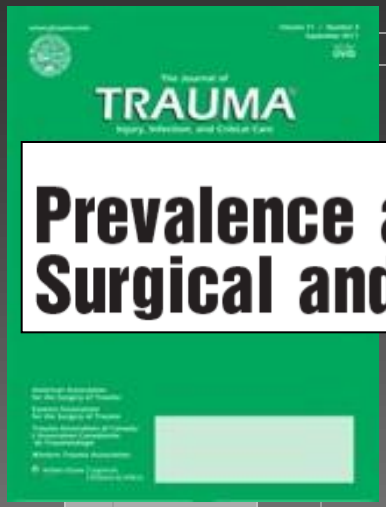

J Trauma 2008;65:34-41

Prevalence and Risk Factors for Development of Delirium in Surgical and Trauma Intensive Care Unit Patients

Delirium as a Predictor of Mortality in Mechanically Ventilated Patients in the Intensive Care Unit

JAMA

2004;291:1753-1762

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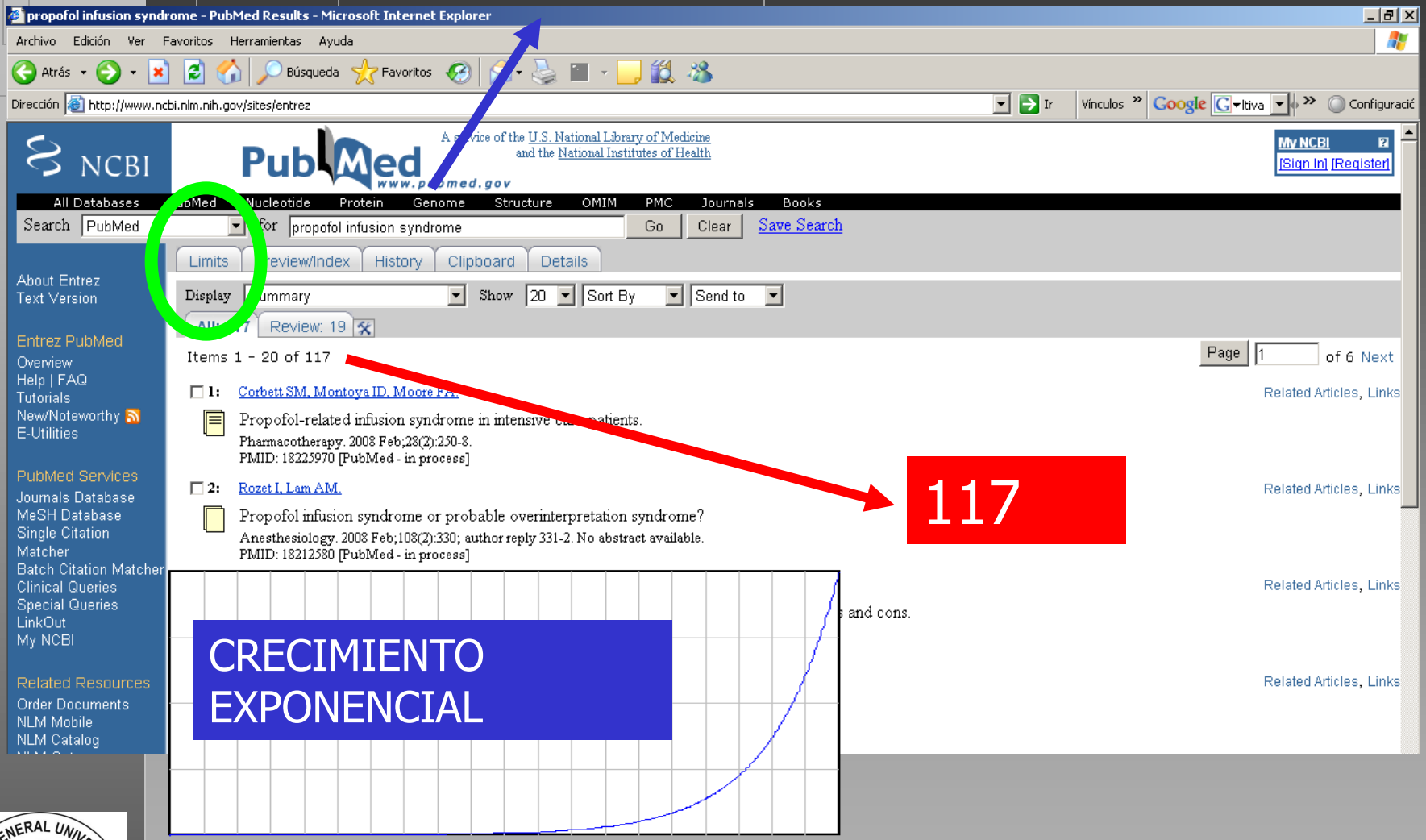
Propofol infusion syndrome



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The Journal of TRAUMA
Injury, Infection and Critical Care

Brief Review

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

Too Much of a Good Thing? Tracing the History of the Propofol Infusion Syndrome

Daniel J. Rosen, MD, Alina Nicoara, MD, Ninan Koshy, MD, and Raymond V. Wedderburn, MD

Case reports began to appear in the pediatric literature linking unexplained deaths with the prolonged use of high-dose propofol infusions. This led to an early warning issued by the Danish Side Effect Committee in 1990.[3] In 1992, as more case reports continued to come to light, the Committee on Safety of Medicines in the United Kingdom issued a serious adverse effect warning against the long-term use of propofol in pediatric patients.[4] This led to the immediate abandonment of propofol as a sedative agent in pediatric ICUs within Britain.[5] The warning also led to a corresponding change in the package insert provided by AstraZeneca.[6]

The Journal of TRAUMA Injury, Infection and Critical Care

Brief Review — *The Journal of* TRAUMA® *Injury, Infection, and Critical Care*

Too Much of a Good Thing? Tracing the History of the Propofol Infusion Syndrome

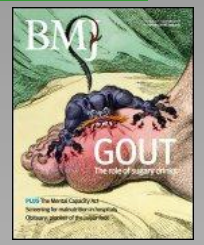
Daniel J. Rosen, MD, Alina Nicoara, MD, Ninan Koshy, MD, and Raymond V. Wedderburn, MD

5 niños fallecen en 1992

Metabolic acidosis and fatal myocardial failure after propofol infusion in children: five case reports. British Medical Journal 1992;305:613–616.

5 adultos en 2001

Long-term propofol infusion and cardiac failure in adult head-injured patients. The Lancet 2001; 357: 117–118



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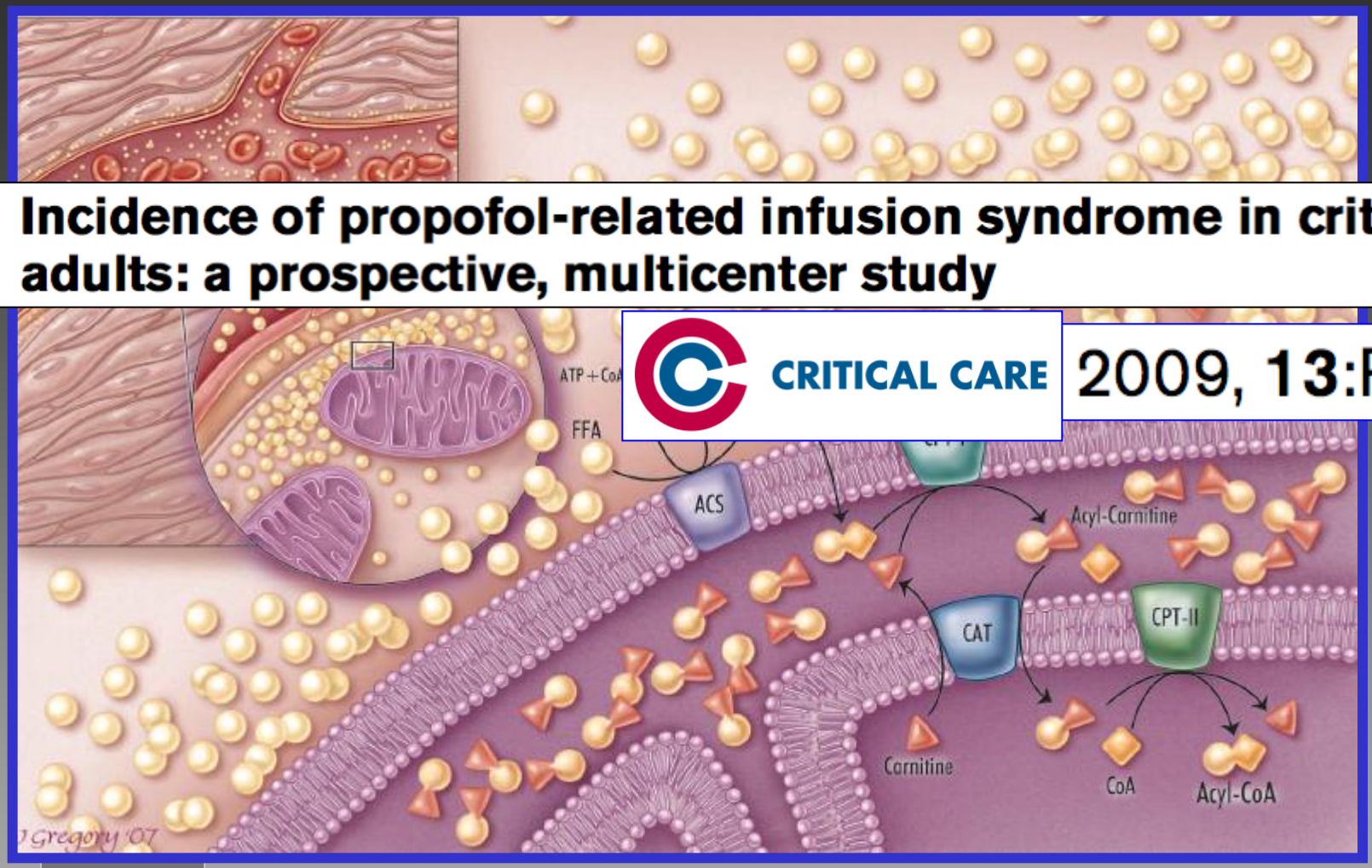
Incidence of propofol-related infusion syndrome in critically ill adults: a prospective, multicenter study

CRITICAL CARE 2009, 13:R169



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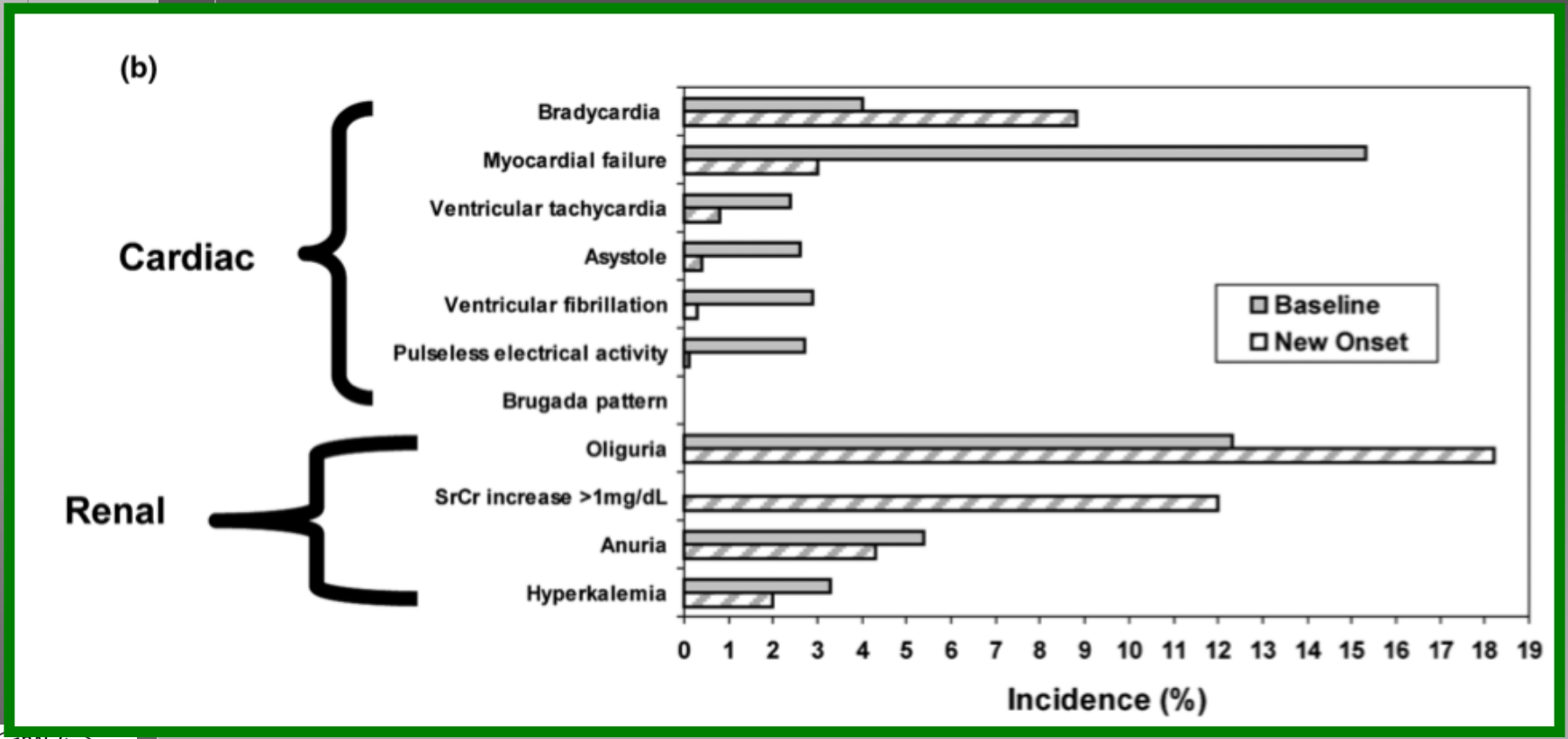

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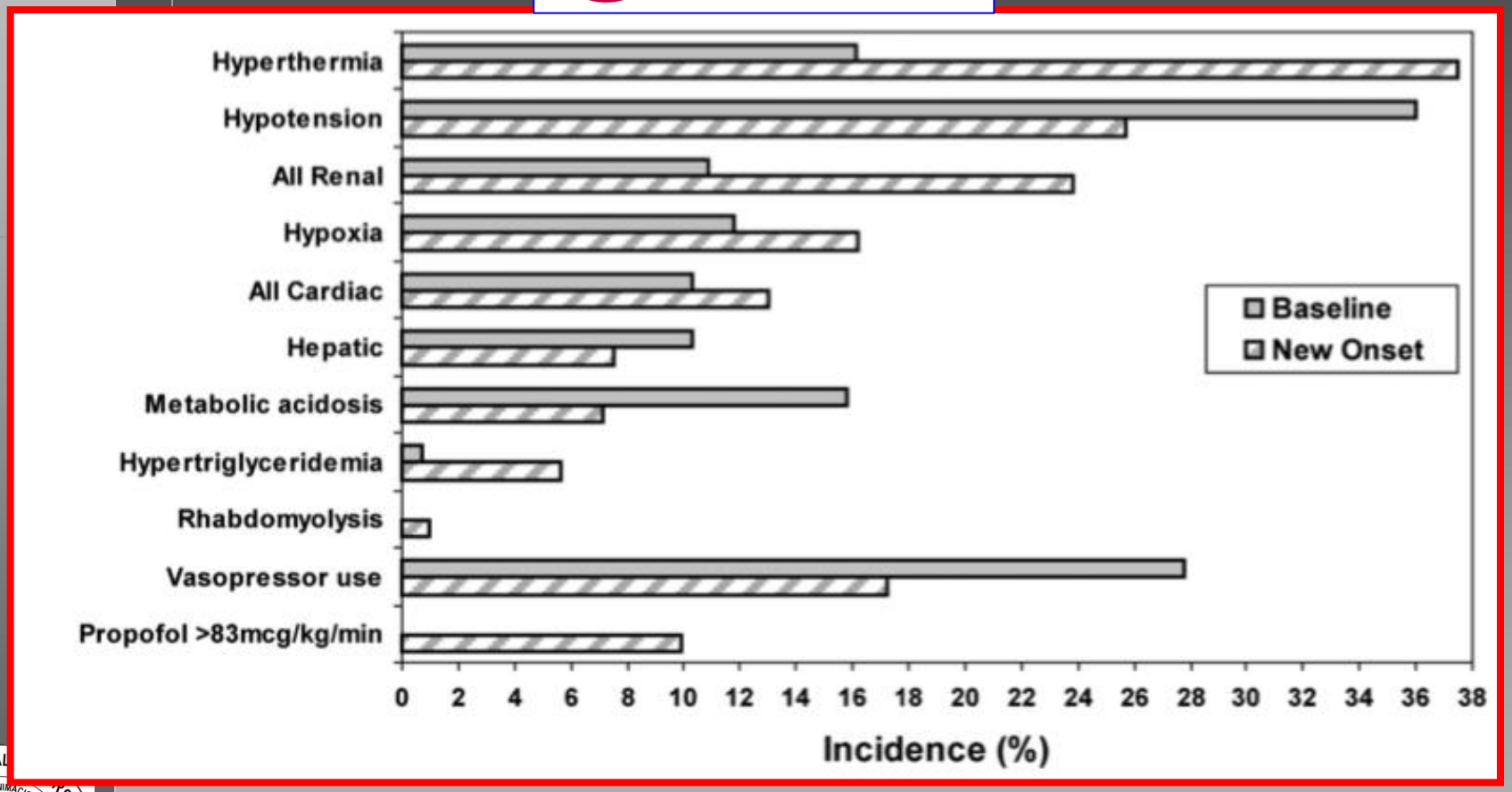

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Incidence of propofol-related infusion syndrome in critically ill adults: a prospective, multicenter study

CRITICAL CARE 2009, 13:R169

This study was the first to prospectively evaluate a large population of critically ill adults receiving longer-term propofol and to use an evidence-based and conservative definition for PRIS, and identified PRIS in 1.1% of patients.

Table 2. Demographic and Clinical Characteristics of 36 Adults with Propofol-Related Infusion Syndrome[17, 32–55]

Characteristic	Value
	Range
Age (yrs)	16–64
Mean propofol dose (mg/kg/hr)[a]	1.4–12
Duration of propofol administration (hrs)	4.5–192
	No. (%)
Sex	
Male	16 (44)
Female	12 (33)
Not reported	8 (22)
Admitting diagnosis	
Abdominal abscess	1 (3)
Accident (no head trauma)	2 (6)
Angina	1 (3)
Arteriovenous malformation	1 (3)
Asthma	3 (8)
Atrial fibrillation (radiofrequency ablation)	1 (3)
Brainstem cavernous angioma	1 (3)
Cerebral sinus thrombosis	1 (3)
Head trauma	16 (44)
Neonatal progeroid syndrome	1 (3)
Prostatectomy	1 (3)
Seizure disorder	6 (17)
Spinal cord injury	1 (3)
Formulation of propofol administered	
2% lipid emulsion	8 (22)
1% lipid emulsion	10 (28)
Not reported	18 (50)
Clinical features	
Arrhythmias	
Asystole	8 (22)
Bradycardia	8 (22)
Pulseless electrical activity	4 (11)
Tachyarrhythmias	19 (53)
Nonspecified type	2 (6)

Cardiac failure	
Cardiac arrest	11 (31)
Left bundle branch block	2 (6)
Left ventricular dysfunction	8 (22)
Right bundle branch block	1 (3)
Metabolic findings	
Fever	5 (14)
Hyperkalemia	17 (47)
Hypocalcemia	2 (6)
Hypotension	16 (44)
Lactic acidosis	11 (31)
Lipemia	6 (17)
Metabolic acidosis	30 (83)
Muscular involvement	
Elevated creatine kinase level	23 (64)
Myoglobinemia and/or myoglobinuria	8 (22)
Rhabdomyolysis	17 (47)
Elevated troponin I level	6 (17)
Renal involvement	
Acute renal failure	16 (44)
Urine discoloration	9 (25)
Respiratory involvement (hypoxia)	1 (3)
Concomitant drugs	
Catecholamines	26 (72)
Corticosteroids	6 (17)

Inhalation versus endovenous sedation in subarachnoid hemorrhage patients: Effects on regional cerebral blood flow

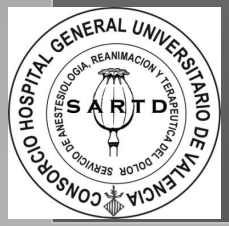
Federico Villa, MD; Cosimo Iacca, RN; Andrea Forastieri Molinari, MD; Carlo Giussani, MD, PhD; Giacomo Aletti, PhD; Antonio Pesenti, MD; Giuseppe Citerio, MD

Crit Care Med 2012 Vol. 40, No. 10

Table 1. Study population demographics, type of aneurysm, age, and treatment performed

Patient	Age	Glasgow Coma Score	Fisher Scale	Hunt and Hess Scale	World Federation of Neuro Surgeons Scale	Aneurysm	Treatment
1	59	6	4	4	5	Left IC	Clipping
2	58	9	2	3	3	Left MCA	Clipping
3	62	8	3	4	5	Right MCA	Clipping
4	56	12	3	3	3	Posterior communicating artery	Coiling
5	64	8	3	4	4	AcoA	Coiling
6	50	6	4	4	5	AcoA	Clipping
7	74	12	2	2	4	AcoA	Clipping
8	52	7	4	4	4	Right MCA	Clipping
9	58	13	4	2	3	AcoA	Clipping
10	51	9	4	4	4	Right IC	Clipping
11	53	8	4	4	4	Right MCA	Clipping
12	65	4	4	4	4	Right IC	Coiling
13	28	8	4	4	4	Right IC	Coiling

IC, internal carotid; MCA, middle cerebral artery; ACoA, anterior communicating artery.



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Inhalation versus endovenous sedation in subarachnoid hemorrhage patients: Effects on regional cerebral blood flow

Federico Villa, MD; Cosimo Iacca, RN; Andrea Forastieri Molinari, MD; Carlo Giussani, MD, PhD; Giacomo Aletti, PhD; Antonio Pesenti, MD; Giuseppe Citerio, MD

Crit Care Med 2012 Vol. 40, No. 10

- Step 1 (Basal, T0): Standard sedation with propofol, 3–4 mg/kg/hr. Because the AnaConda system (AnaConDa) adds a significant dead space to the ventilator circuit that may cause an important raise in $PaCO_2$, the system was inserted in the ventilator circuit before the first step (without starting isoflurane) and minute ventilation was adjusted consequently, to maintain constant $PaCO_2$. The AnaConda system remained on the ventilator circuit during all the protocol steps. The only difference is the on/off of the isoflurane infusion pump during step 2.
- Step 2 (duration 1 hr): Sedation was switched from propofol, 3–4 mg/kg/hr, to isoflurane 0.8 % administered through an AnaConda. Data were recorded after 1 hr.
- Step 3: After 1 hr from the interruption of isoflurane and from the resumption of the infusion of propofol at the same previous infusion rate.

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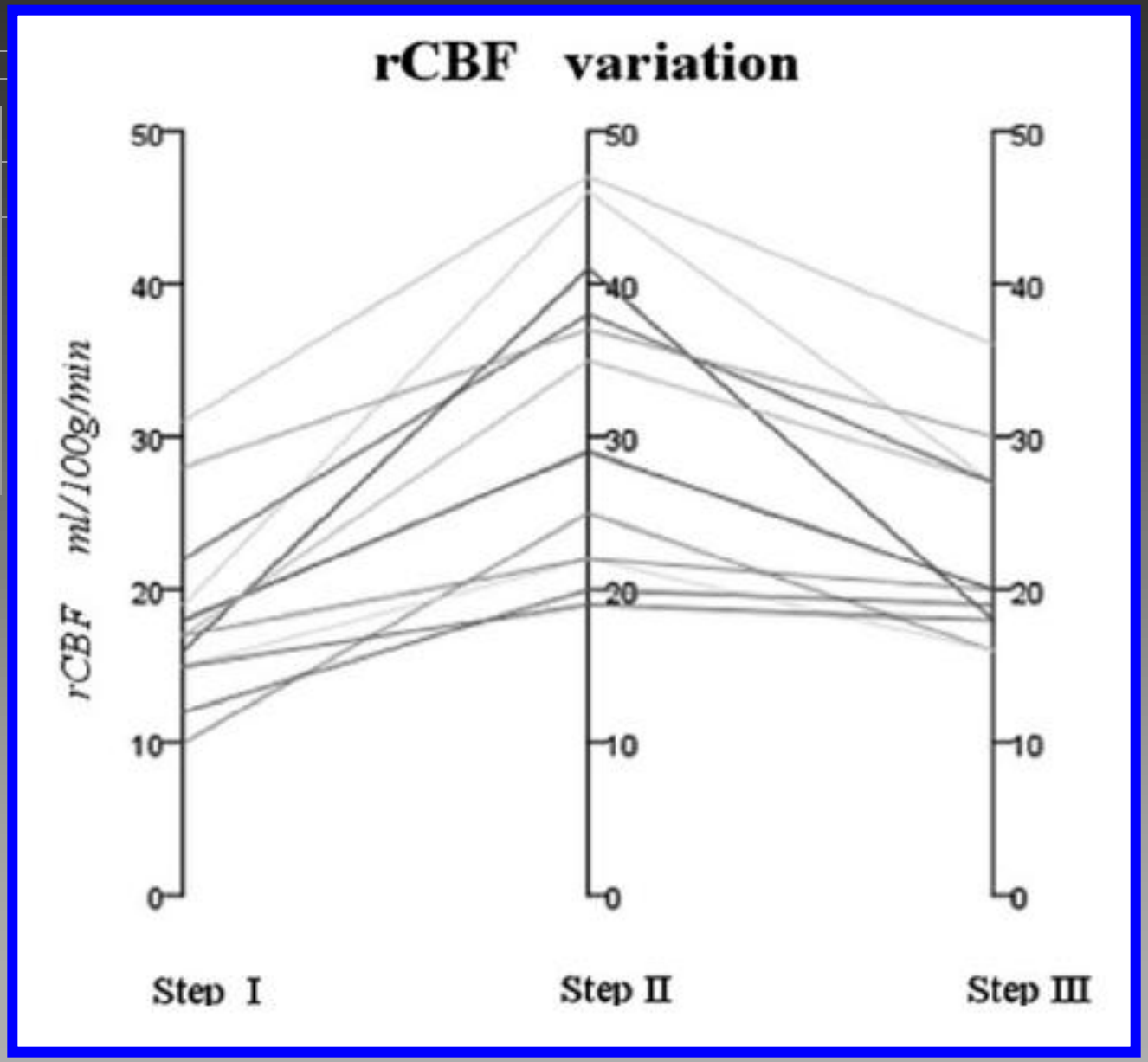
- A diagnosis of SAH, within 72 hrs from the initial bleeding.
- Aneurysm secured, either by clipping or coiling;
- Initial computed tomography Fisher scale 2–4 (16),
- World Federation of Neurosurgeons classification 3–5 (17),
- ICP and rCBF monitoring already in use due to clinical indication, with the correct position of both ICP and thermal diffusion probe controlled by a CT scan.
- Sedation and mechanical ventilation already in use due to clinical indication,
- Continuous electrocardiogram, central venous pressure, invasive arterial blood pressure, cerebral perfusion pressure (CPP), end-tidal CO_2 ($EtCO_2$), and core temperature (monitored by a bladder sensor).
- A normal ICP (<18 mm Hg) without medical therapies prior to the study.



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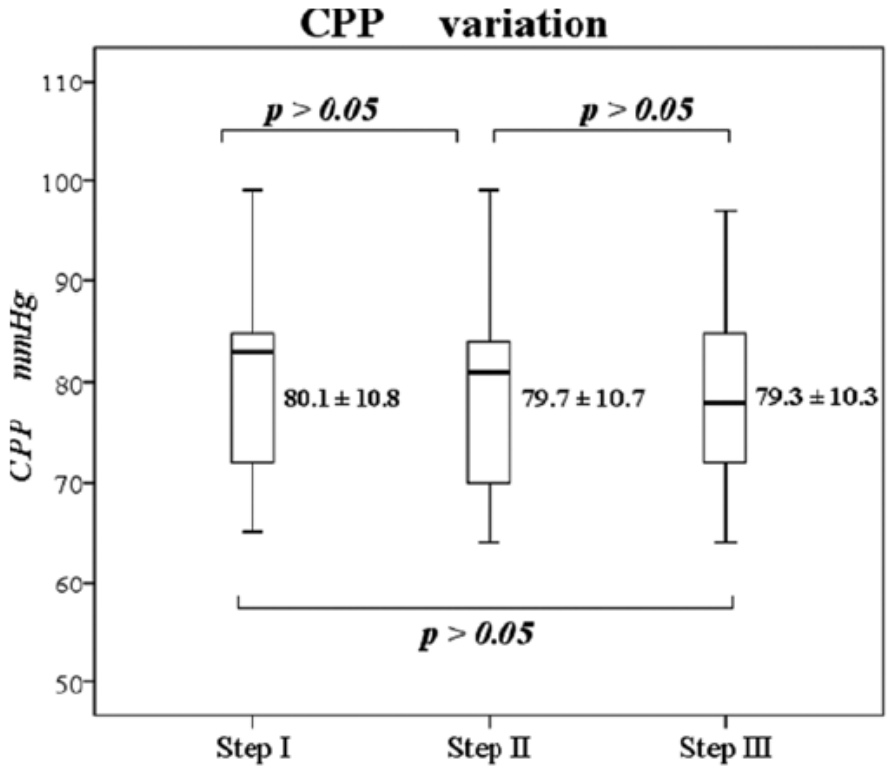

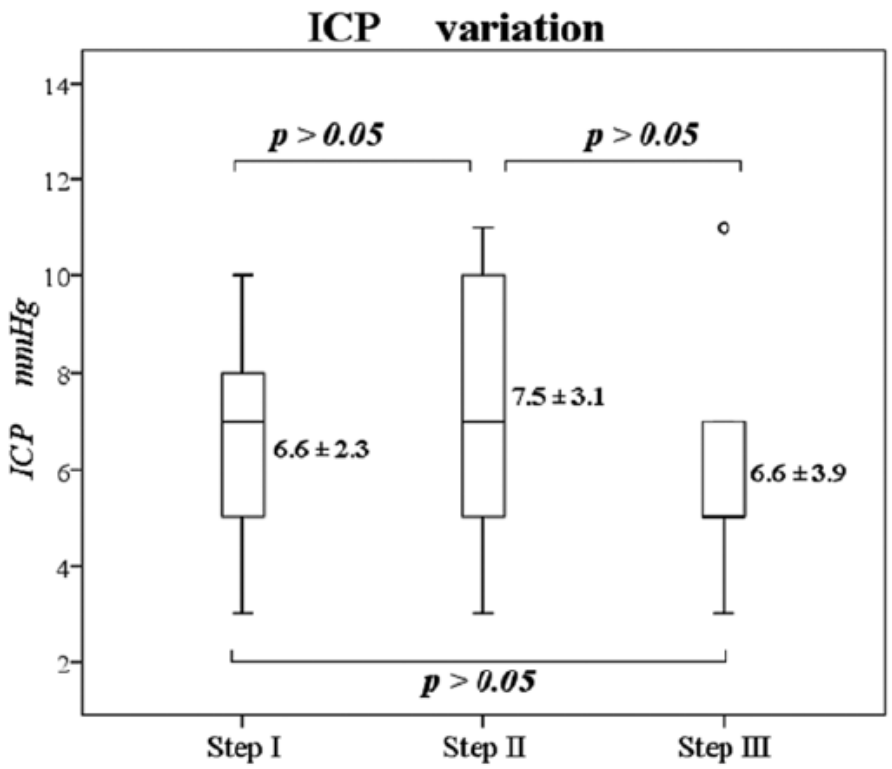

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CONCLUSIONS

Our data suggest that isoflurane is safe and could be used as sedative agent in SAH patients not affected by elevated ICP level, increasing CBF. More studies are needed to confirm the safety of its use and to investigate the real neuroprotective effects of this volatile anesthetic.

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EDITORIAL

Federico Villa
Giuseppe Citerio

Surpassing boundaries: volatile sedation in the NeuroICU

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Brief Review *The Journal of* TRAUMA® *Injury, Infection, and Critical Care*

Too Much of a Good Thing? Tracing the History of the Propofol Infusion Syndrome

Daniel J. Rosen, MD, Alina Nicoara, MD, Ninan Koshy, MD, and Raymond V. Wedderburn, MD

ment of this potentially lethal complication. Below, two patients are presented who were treated for severe traumatic brain injuries (TBIs) in our intensive care unit (ICU) during a period of 2 months. Both received high-dose, long-term propofol infusions to lower intracranial pressures (ICPs). Both developed findings consistent with PIS.

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CASE REPORTS

Patient 1

An 18-year-old White man without significant medical history was the unrestrained front seat passenger in a side-impact motor vehicle collision. After prolonged extrication from the vehicle, he was noted to have a Glasgow coma scale score of 3 and was intubated. Work up revealed severe TBI, pelvic and long-bone fractures, a right hemothorax, and an intraperitoneal bladder rupture. The patient was taken to the operating room for ICP monitor placement, tube thoracostomy, exploratory laparotomy with repair of a bladder laceration, and external fixation of his orthopedic fractures. The patient was then transferred to the ICU for continued monitoring.

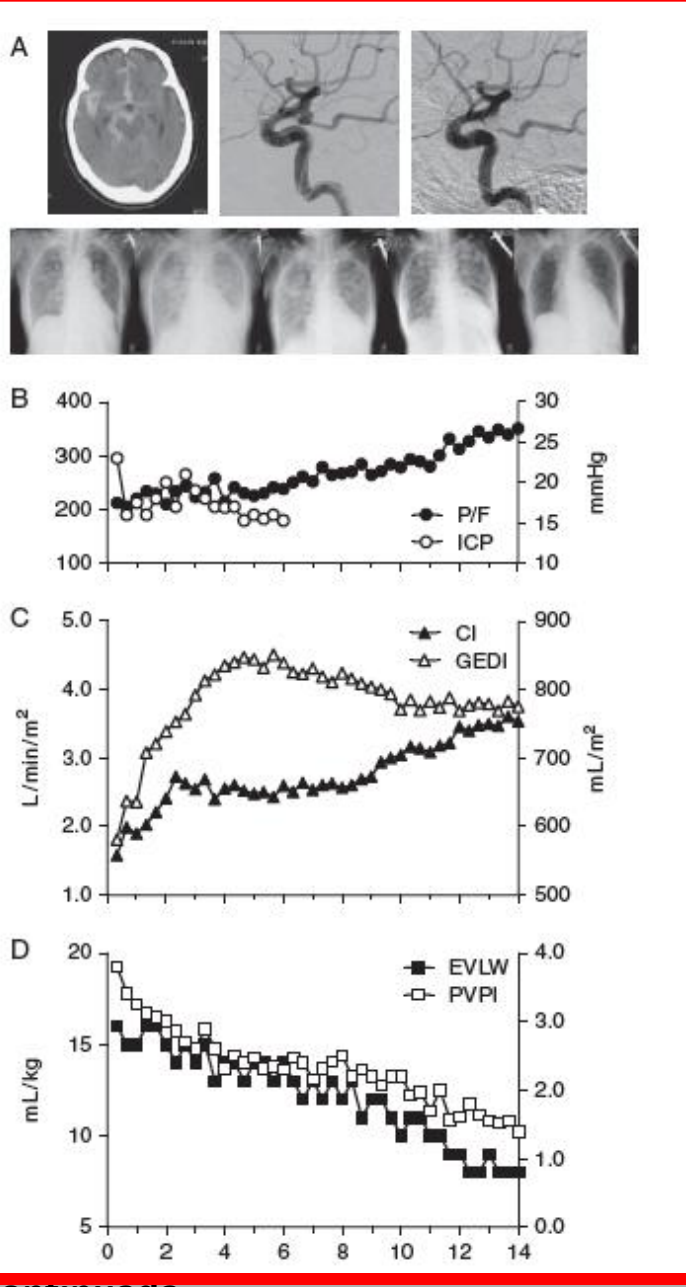
In the ICU, the patient was placed on a propofol infusion for both its sedative and neuroprotective effects. Analgesia was provided by a continuous morphine infusion. The propofol infusion was titrated to an ICP <20 mm Hg and a cerebral perfusion pressure (CPP) >60 mm Hg. During the ensuing week the patient's elevated ICP required increasing doses of propofol, reaching a maximum of 7.5 $mg/kg^{-1}/h^{-1}$. This high dose was continued for more than 72 hours during hospital days (HDs) 5 to 8. Low-dose vasopressors were started to elevate his mean arterial pressure in an attempt to maximize CPP.

On HD 8, the patient was noted to have dark urine. Creatine phosphokinase (CPK) was noted to be elevated at 15,312 U/L, up from 1,552 U/L from 2 days previous. During the course of HD 8, the patient developed progressive oliguria and worsening hemodynamics. CPK levels continued to rise rapidly, peaking at 95,440 U/L with no clear source of muscle breakdown.

On HD 9, the patient became anuric with serum creatinine elevated to 3.6 g/dL, from a baseline of 1.0 g/dL. The patient developed increasing metabolic acidosis with a pH of 7.10 and cardiac arrhythmias were noted. As plans for emergent dialysis were discussed, the patient clinically deteriorated and developed asystole. Advanced Cardiac Life Support (ACLS) protocol was initiated but resuscitation was unsuccessful.

Serial Measurement of Extravascular Lung Water and Blood Volume During the Course of Neurogenic Pulmonary Edema after Subarachnoid Hemorrhage: Initial Experience With 3 Cases

J Neurosurg Anesthesiol 2012;24:203–208



Serial Measurement of Extravascular Lung Water and Blood Volume During the Course of Neurogenic Pulmonary Edema after Subarachnoid Hemorrhage: Initial Experience With 3 Cases

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In summary, our clinical experience suggests that the present monitoring system is capable of distinguishing different etiologies for pulmonary edema complicating SAH that may assist with fluid management decisions; 3 cases of post-SAH NPE were presented to demonstrate the concepts.

Hemodynamically Compromising Cardiac Arrhythmia During Surgery for Acute Traumatic Brain Injury: Management and Outcome

J Neurosurg Anesthesiol • Volume 24, Number 4, October 2012

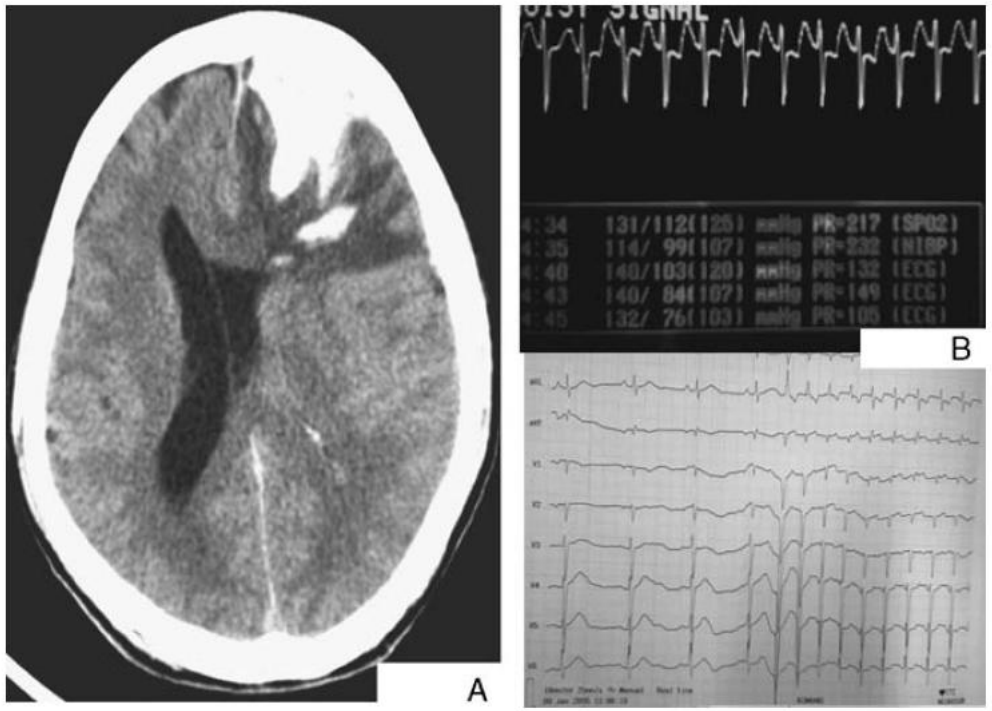

FIGURE 1. A, Axial computed tomography scan brain immediately on arrival in the emergency department. B, Electrocardiograms (ECGs) showing supraventricular tachyarrhythmia on the monitor and 12-lead ECG strip.

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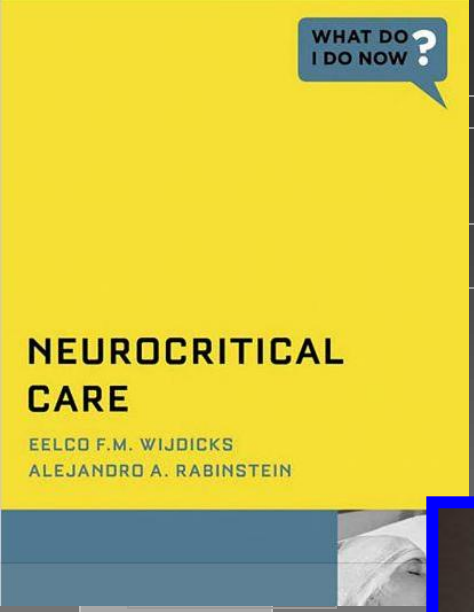
J Neurosurg Anesthesiol • Volume 24, Number 4, October 2012

Hemodynamically Compromising Cardiac Arrhythmia During Surgery for Acute Traumatic Brain Injury: Management and Outcome

At present, there is no sufficient evidence to support the prioritization of emergency surgery over management of arrhythmia and hemodynamics in TBI. This report, along with those published earlier, suggests that the time taken to optimize patients with cardiovascular compromise may not result in an adverse neurological outcome. The risk-benefit ratio for emergent surgery in this group of patients needs further evaluation.



Define resuscitation goal

A MAP goal higher than the usual 65 mmHg may be necessary in neurocritical patients with compromised cerebral perfusion

Start vasopressor if MAP below target after fluid challenge

Norepinephrine, low dose vasopressin, epinephrine

Phenylephrine is not adequate

Obtain echocardiogram and assess systolic function

Start dobutamine if decreased left ventricular ejection fraction

Conservative fluid strategy after resuscitation goal is achieved

Can use diuretics if MAP stable and evidence of cerebral edema or raised ICP

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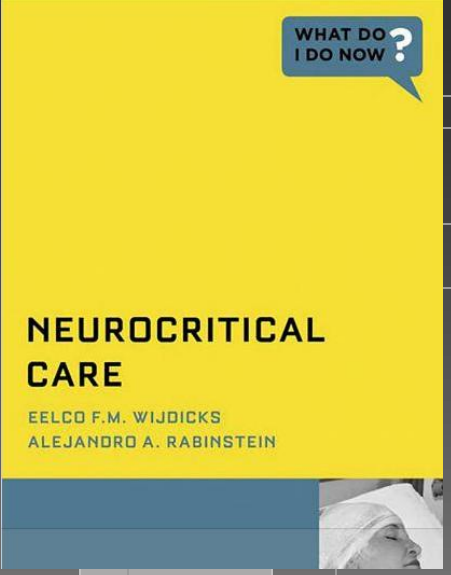

lines recommend reducing the systolic blood pressure below 185 mmHg and the diastolic blood pressure below 110 mmHg before infusing the

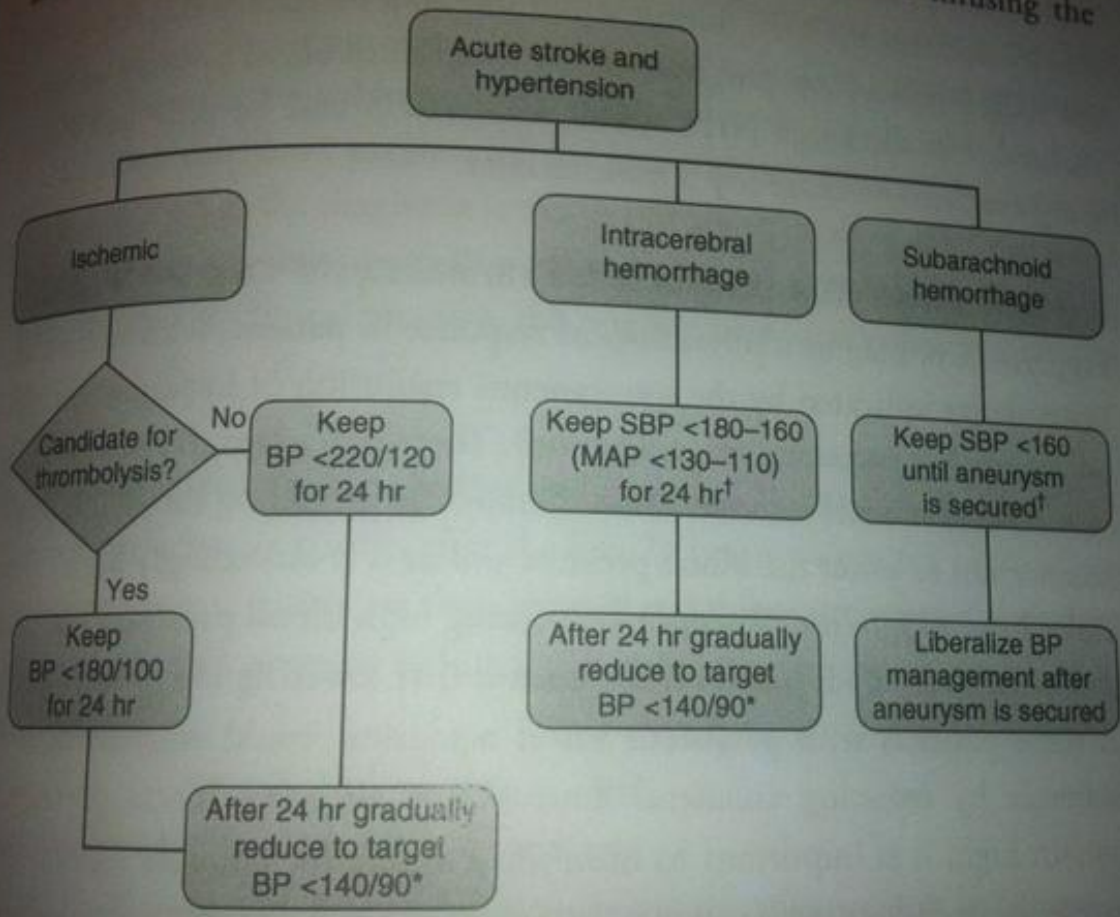

FIGURE 21.1 Recommended algorithm for control of hypertension in patients with acute stroke. *Target should be 130/80 mmHg in patients with diabetes mellitus. Ideal blood pressure <120/80 mm Hg. † If suspected increased intracranial pressure, then monitor intracranial pressure and maintain cerebral perfusion pressure > 60 mmHg. BP; blood pressure.

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neurocritical care society Neurocrit Care (2012) 16:63–71
DOI 10.1007/s12028-011-9620-x

ORIGINAL ARTICLE

Impact of a Neurointensivist on Outcomes in Critically Ill Stroke Patients

Lisa Knopf · Ilene Staff · Joao Gomes · Louise McCullough

It is clear from this study that factors that influence outcomes in critically ill stroke patients are complex and multifactorial, and are often more related to patient factors than the specifics of their ICU care. This work suggests that the NCCU improves outcomes for AIS patients, and the presence of a NI improves outcomes specifically in SAH patients. As evidence that the availability of a trained NI improves outcome in critically ill stroke patients need for this specialized physician will continue to rise.

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Changing trends in monitoring brain ischemia: from intracranial pressure to cerebral oximetry

Ganne S. Umamaheswara Rao[a] and Padmaja Durga[b]

Current Opinion in Anesthesiology 2011, 24:487–494

Table 1 Factors affecting the cerebral oxygenation

	Factors favoring cerebral oxygenation	Factors interfering with cerebral oxygenation
Systemic factors	High/normal MAP/CPP	Low MAP/CPP
	Normal hemoglobin	Anemia
	Normal/high cardiac output	Low cardiac output
	Normoxia/hyperoxia	Systemic hypoxemia
	Normocapnia	Hypocapnia
Intracranial factors	Metabolic suppression (sedatives/hypothermia)	Hyperthermia
	Normal ICP/CPP	Seizures
	Cerebral vasodilatation	High ICP/low CPP
		Cerebral vasospasm

CPP, cerebral perfusion pressure; ICP, intracranial pressure, MAP, mean arterial blood pressure.

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In conclusion, technology and concepts of monitoring cerebral ischemia have come a long way from indirect measures like ICP and CPP to direct $PbtO_2$ and microdialysis. With some of the current devices, it is possible to monitor changes in cellular physiology. The newer challenges are to comprehend the physiological basis and clinical implications of the monitored parameters and to identify an ideal combination of global and regional monitors that meet the requirements of given clinical situations. Multimodal monitoring with ICP, TCD, $PbtO_2$, microdialysis appears to be the future of monitoring and managing cerebral ischemia.

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Table 1 Methods for direct cerebral blood flow measurement

	Advantages	Disadvantages	Clinical applications
PET	CBF, CBV, $CMRO_2$, CMRglu Quantitative	Requires transfer Noncontinuous Radioactive compounds (^{15}O)	SAH TBI Stroke
Perfusion weighted MRI (PWI)	CBF, CBV, MTT, TTP measurement No radiation	Requires gadolinium IV contrast agent Longer scan time Transport and restrictions of MRI environment Semi-quantitative	SAH TBI Stroke
Xenon CT	CBF measurement Quantitative	Requires transfer Noncontinuous Inaccurate in lung disease	SAH TBI Stroke
SPECT	CBF	Requires transfer Noncontinuous Radioactive compounds Semi-quantitative	SAH TBI Stroke
CT perfusion	CBF, CBV, MTT, TTP assessment Economical Fast Quantitative	Requires iodinated IV contrast agent Requires transfer	SAH TBI Stroke

CBF, cerebral blood flow; CBV, cerebral blood volume; CT, computed tomography; MTT, mean transit time; PET, positron emission tomography; SAH, subarachnoid hemorrhage; SPECT, single-photon emission computed tomography; TTP, time to peak.

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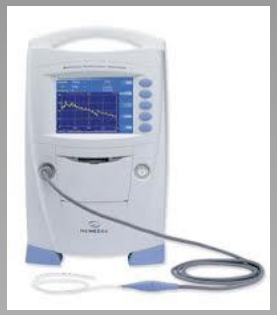
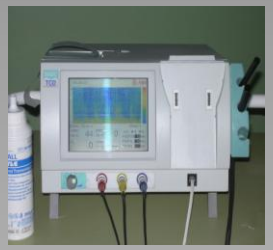
Table 2 Methods for indirect or bedside cerebral blood flow measurement

	Advantages	Disadvantages	Clinical applications
TDF	Direct and bedside monitoring Absolute CBF	Invasive Regional flow only	TBI SAH
LDF	Regional flow	Uses red cell flux as a surrogate Local CBF and relative flow only	Cerebral autoregulation testing CO_2 reactivity Assess responses to interventions
TCD	Noninvasive Real time with excellent temporal resolution Regional flow	Relative flow only Operator dependent 5–10% Failure rate	Cerebral autoregulation testing CO_2 reactivity TBI SAH Brain death confirmation
$SjvO_2$	Continuous monitoring Balance between flow and metabolism	Global and insensitive, invasive Thrombosis	TBI SAH Detecting A-V fistulas ICP management
$pbrO_2$	Bedside measurement Flow/metabolism balance	Invasive Measures local O_2 tension only, subject to drift	TBI SAH
NIRS	Noninvasive Real time Bedside measurement	Extracranial blood contamination, ambient light Probe limited to frontal location, dependent on arbitrary derived algorithms	TBI ICH CEA
Microdialysis	Measures local biochemistry and early detection of secondary injury	Invasive Hemorrhage Infection	TBI SAH Epilepsy Ischemic stroke Tumors

CBF, cerebral blood flow; CEA, carotid endarterectomy; ICH, intracranial hemorrhage; LDF, laser Doppler flowmetry; NIRS, near-infrared spectroscopy; $pbrO_2$, brain tissue oxygen monitoring; SAH, subarachnoid hemorrhage; $SjvO_2$, jugular venous oximetry; TBI, traumatic brain injury; TCD, transcranial Doppler; TDF, thermal diffusion flowmetry.





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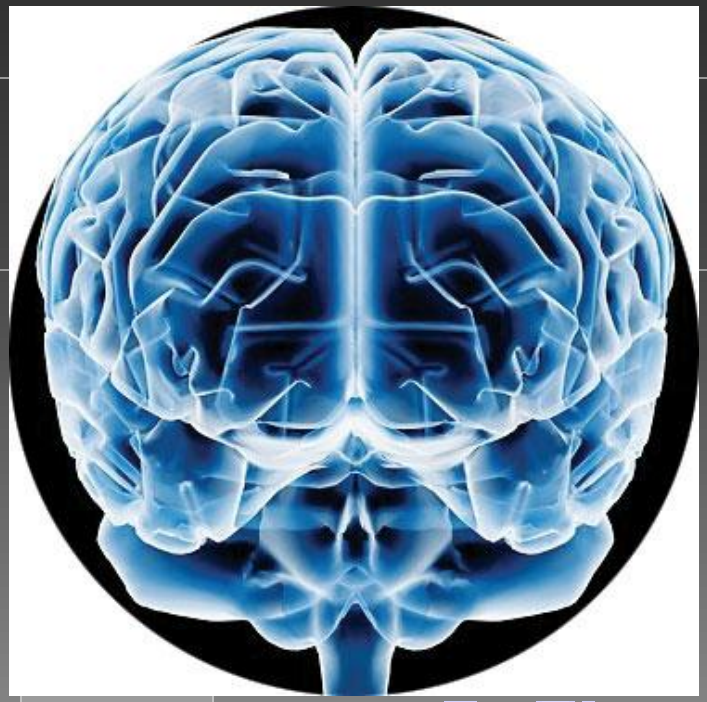

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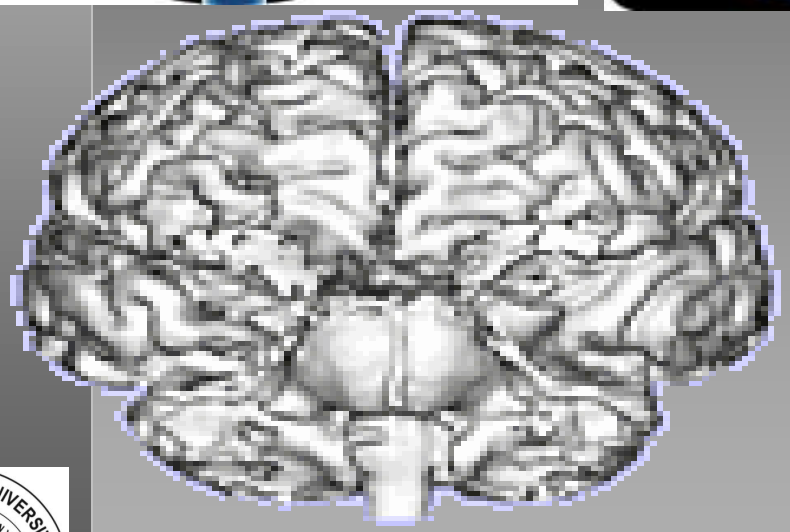

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