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ABC's or ICP? Goals for anesthetic management of patients with head injury

David P. Archer, M.D., MSc, FRCPC*

* Professor, Department of Anesthesia and Clinical Neurosciences University of Calgary Alberta, Canada.

GOALS OF THE PRESENTATION

- Review recommendations of the Brain Trauma Foundation 2001
- Review evidence for traditional strategies for "neuroprotection"
- Introduce some new concepts concerning potential therapeutic strategies

GUIDELINES 2001 (WWW.BRAINTRAUMA.ORG)

- Standards (Class I evidence)
- ICP normal - avoid prolonged hyperventilation
- No benefit from steroid administration
- No benefit from prophylactic anticonvulsants

GUIDELINES 2001

- Recommendations - class II evidence
- Regional trauma management system
- Avoid hypotension (BP systolic < 90 mmHg), hypoxemia ($SpO_2 < 90\%$, $PaO_2 < 60$ mmHg)
- Measure ICP if: GCS 3-8 with abnormal CT or 2 or more of the following: age > 40 yrs, motor signs, Bpsystolic < 90 mmHg

GUIDELINES 2001

- Treat ICP > 20-25 mmHg, mannitol 0.25-1 g/kg
- Avoid prophylactic hyperventilation ($PaCO_2 < 35$ mmHg) during the first 24 hours after traumatic brain injury

- Consider high dose barbiturates for treatment of uncontrollable intracranial hypertension

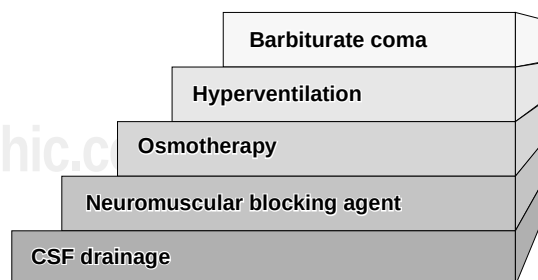
MANAGEMENT STRATEGIES YOU WILL ENCOUNTER

- Traditional
- Perfusion pressure based
- "Lund" strategy

TRADITIONAL MANAGEMENT

- Rapid evacuation of intracranial hematomas
- Initial management
 - airway control, tracheal intubation, oxygen therapy, ventilatory support
 - rapid correction of systemic hypotension
 - sufficient fluid administration to maintain intravascular volume

A. Traditional ICP-directed management strategy

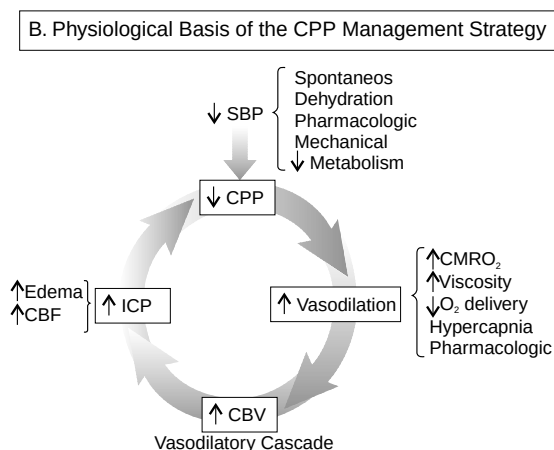


Robertson, C.S. Anesthesiology 2001

CPP HYPOTHESIS (J NEUROSURG 1995;83:949)

- The brain “sees” perfusion as the cerebral perfusion pressure (CPP) not the mean arterial pressure (MAP)
- ↑ ICP are caused by autoregulatory responses to reductions in CPP near the limits of autoregulation

B. PHYSIOLOGICAL BASIS OF THE CPP MANAGEMENT STRATEGY

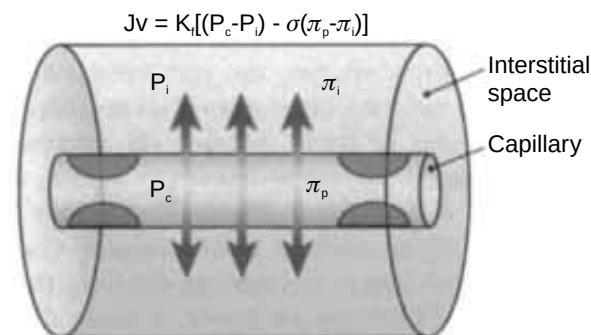


Robertson, C.S. Anesthesiology 2001

“LUND” STRATEGY (↓ CEREBRAL EDEMA FORMATION)

- Supine position
- “stress” reduction - thiopental (0.5 -3 μg/kg/h, fentanyl 2-5 μg/kg/h, metoprolol, clonidine
- ↓ of cerebral blood volume - thiopental, dihydroergotamine - 0.1-0.9 μg/kg/h

C. Physiological Basis of the Lund Management Strategy



Robertson, C.S. Anesthesiology 2001

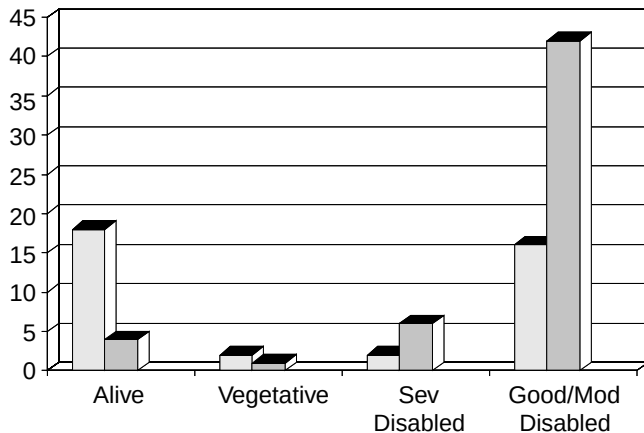
Differences in management approaches to the head-injured patient

Approaches to the critical care management of traumatic brain injury

	Traditional	CPP management ²	Lund therapy ^{4,15}	Individualized therapy ⁶
General management:				
Head position	15-30° elevation	Flat	Flat	whater position best CPP
Sedation	morphine + lorazepam	No	low dose thiopental	and ICP morphine + lorazepam
Treatment of systemic hypertension	Treat SBP > 160 mmHg, using labetalol	No	metoprolol + clonidine	Ischemia/hypoperfusion pattern, do not treat
Nutritional support	Yes, avoid hyperglycemia	No	Yes, avoid hyperglycemia	Adequate perfusion, may treat with labetalol yes, avoid hyperglycemia
Treatment of intracranial hypertension				
Neuromuscular blockade	Yes	Yes	No	Yes
Hyperventilation	Yes	No	No	Ischemia/hypoperfusion pattern, don't use
CSF drainage	Yes	Yes	No	Adequate perfusion, may use
Osmotherapy	Yes	Yes	No	Yes
Barbiturate coma	Yes	No	No	Hypoperfusion/edema pattern, yes
Management of cerebral perfusion pressure				
Goal for CPP	Not considered, keep ICP < 20 mmHg and normal BP	> 70-80 mmHg (above lower limit of autoregulation)	> 50-60 mmHg (whatever provides adequate perfusion)	Hypoperfusion/ischemia pattern-increase CPP to improve CBF, especially if autoregulation is impaired
				Adequate perfusion, maintain normal CPP

CPP = cerebral perfusion pressure; ICP = intracranial pressure; SBP = systolic blood pressure; CSF = cerebrospinal fluid; BP = blood pressure; CBF = cerebral blood flow

"LUND STRATEGY" MORTALITY (%) VS ICP (CRIT CARE MED 1998;26:1881)

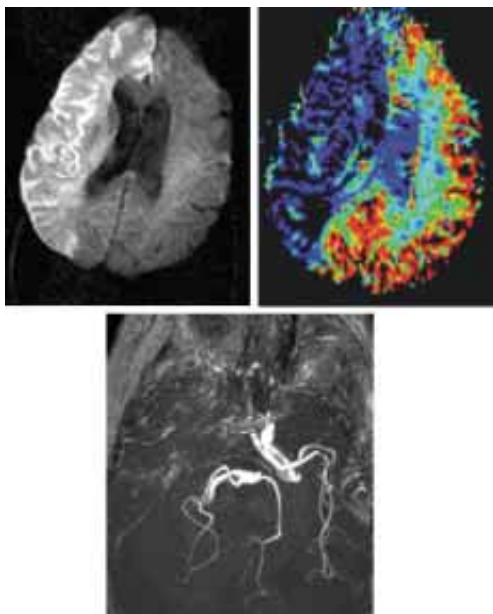


Robertson, C.S. Anesthesiology, 2001

WHAT BLOOD PRESSURE SHOULD I AIM FOR?

- Concept of "Traumatic Penumbra" - perilesional regions are the most affected by therapeutic interventions. (Menton D. Anesthesiology 98, 2003)
- The penumbral region is probably best defined by physiological imaging (PET, fMRI ect).
- Frontal and temporal contusions (motivation and memory) - these regions are frequently contused

MULTIMODAL MRI IMAGING WHAT BLOOD PRESSURE?



WHAT BLOOD PRESSURE?

- Traditional management (targeted at reduction of ICP)
CPP $\geq$ 60 mmHg, Psystolic $\geq$ 90 mmHg
- CPP strategy - CPP $\geq$ 70 mmHg (Rosner et al. J Neurosurg 1995)
- "Lund" strategy - CPP 50-70 according to the effect on ICP. The traumatic penumbra shows an increased extracellular lactate at CPP $<$ 50 mmHg

POTENTIAL INTERVENTIONS

- Improve perfusion
 - $\uparrow$ MAP
- Reduce metabolic demand
 - Hypothermia, anesthetic agents
- Intervene in the ischemic cascade
- Intervene in the white matter injury. (Baker, AJ et al. J Neurotrauma, 2002;19:587)

HYPOTHERMIA

- Archives of neurology. 59(7):1077-83, 2002
- This meta-analysis of randomized controlled trials suggests that hypothermia is not beneficial in the management of severe head injury

ANESTHETIC CHOICE

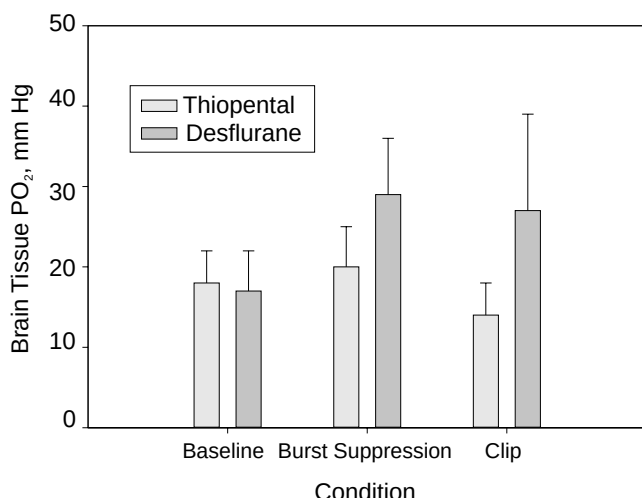
Does it matter which drugs that I use?

BRIEF HISTORY OF WHAT "NOT TO USE" FOR NEUROANESTHESIA IN MY CAREER

- Late 1970's - No halothane ($\uparrow$ ICP)
- mid - 1980's - No sux ($\uparrow$ - ICP), ? crystalloids
- mid - 1990's - No sufentanil ($\uparrow$ - ICP), no N₂O ($\uparrow$ - metabolism, ICP) crystalloids O.K.
- early 2000's ? crystalloids? propofol ($\downarrow$ Cerebral venous O₂ sat), protective effects of volatiles?

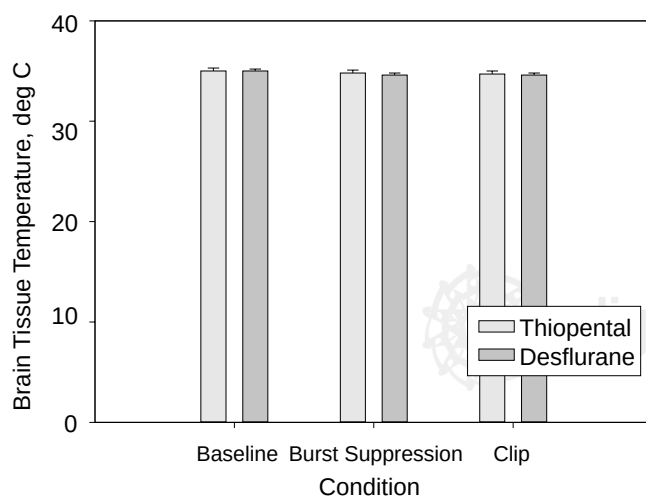
INTRAOPERATIVE BURST SUPPRESSION WITH THIOPENTAL VS DESFLURANE. HOFFMANN, WE ET AL J NEUROSURG 1998;43:1050-53

- RCT vascular neurosurg - randomized to receive STP (8) or Des (7)
- Outcome: brain PO₂, pH, PC=O₂, temperature



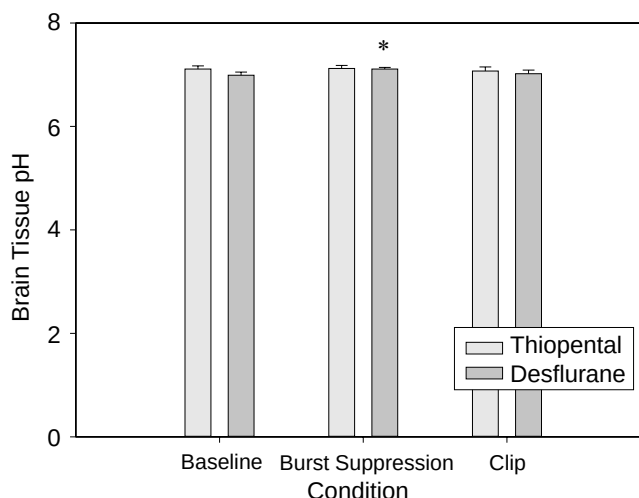
INTRAOPERATIVE BURST SUPPRESSION WITH THIOPENTAL VS DESFLURANE. BEFORE BRAIN OCCLUSION HOFFMANN, WE NEUROSURG 1998

- STP - 15. mg/kg + 1 mg/kg/min
- Desflurane - 9%
- Both adjusted to achieve 50% quiescence



INTRAOPERATIVE BURST SUPPRESSION WITH THIOPENTAL VS DESFLURANE. BACKGROUND ANESTHETIC HOFFMANN, WE 1998

- STP (3-5 mg/kg), Fentanyl 10-15 µg/kg, vecuronium
- Mild hypothermia (34 deg C)
- Desflurane - 3%
- PaCO₂ - 30-35 mmHg



IN SUMMARY

- There is currently no “evidence” to support the concept that the risk of cerebral ischemia in head injury can be reduced by manipulation of pharmacological variables

IN SUMMARY

- Current strategies are based upon reasonable extensions of preclinical (animal) or surrogate variable studies
- Since the benefit of these strategies is unknown, the risk must be very low to be acceptable

CEREBRAL HYPERPERFUSION SYNDROME - AN UNCOMMON CAUSE OF ↑ ICP

- *Clinical setting* - Post-AVM resection or after relief of a high-grade symptomatic stenotic lesion by CEA or stenting.
- *Pathophysiology* - impaired autoregulation leads to a rapid ↑ in CBF>>CMRO₂

- *Clinical syndrome* - headache, vomiting, arterial hypertension, confusion, seizures, neurological deficits, subarachnoid hemorrhage

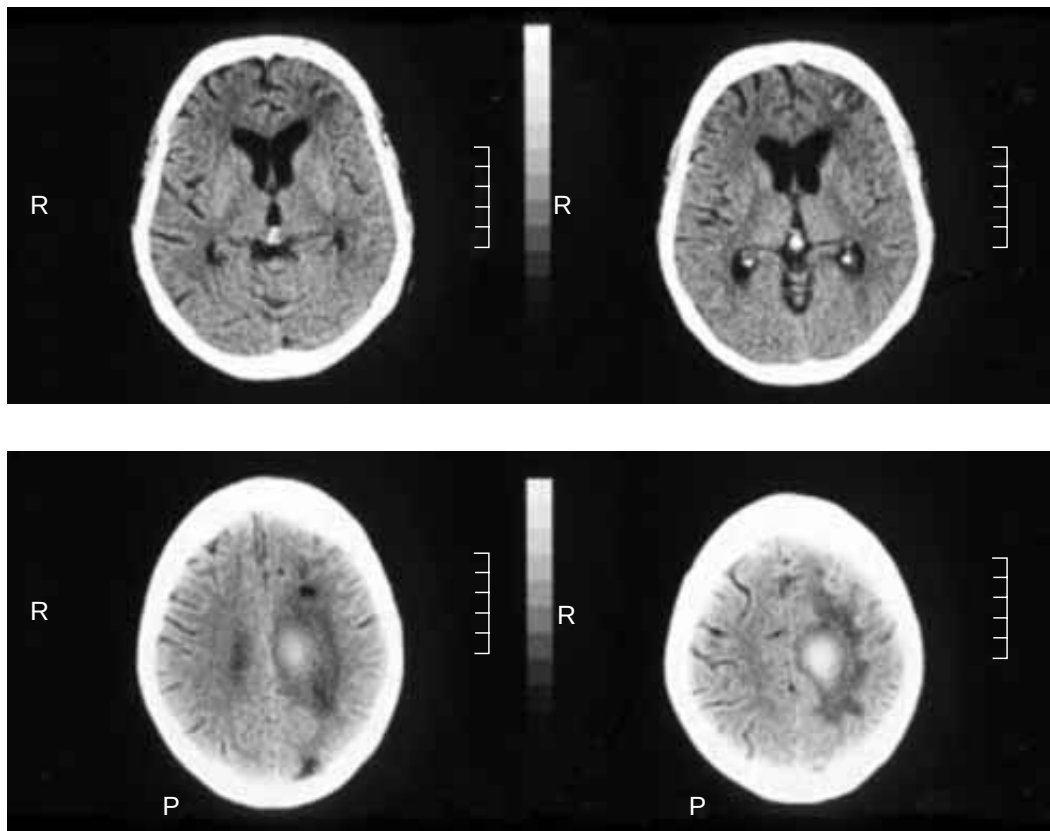
CEREBRAL HYPERPERFUSION SYNDROME

- Incidence - 0.3-1% after CEA. ? After angioplasty and stenting
- Risk factors
 - symptomatic high grade stenosis (> 80%)
 - poor distal perfusion and collateral flow
 - contralateral carotid occlusion

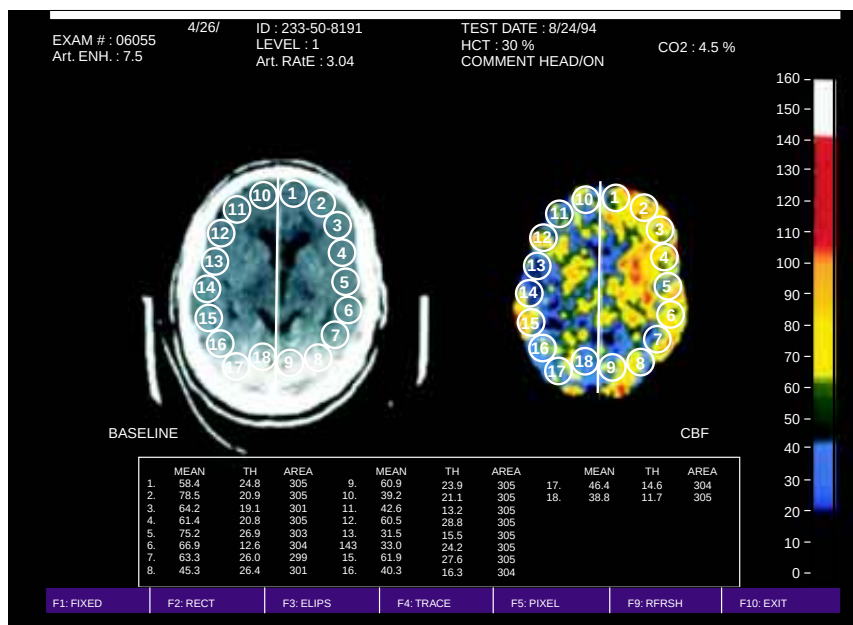
- perioperative hypertension
- Use of anticoagulants and antiplatelet agents

CEREBRAL HYPERPERFUSION SYNDROME

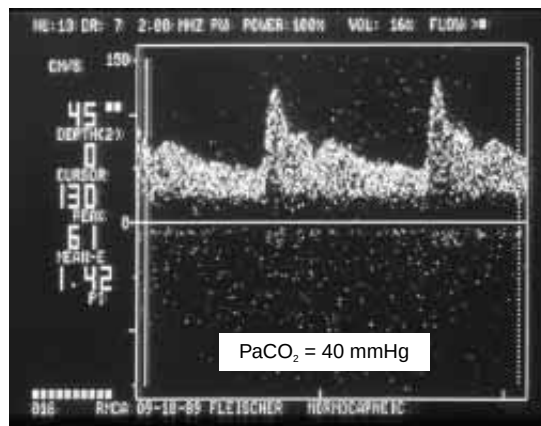
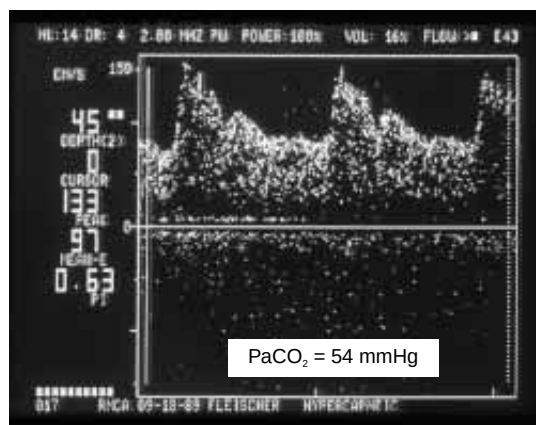
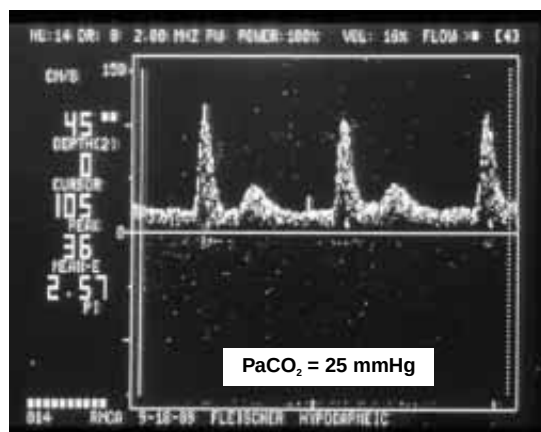
- Prevention - careful blood pressure control, early treatment of seizures
- DDX - edema, hemorrhage, infarct
- Transcranial Doppler, SPECT blood flow
- Management - aggressive blood pressure control, consider diltiazem (reduces BP with least cerebral vasodilation compared to hydralazine, NTG, nicardipine)



SPECT SCAN IN HYPERPERFUSION



DOPPLER IMAGES OF MCA FLOW



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