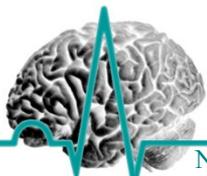


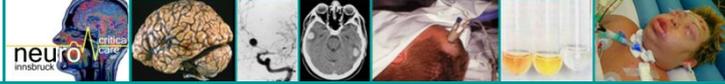
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DIAGNOSTICS and THERAPY of INCREASED INTRACRANIAL PRESSURE in ISCHEMIC STROKE

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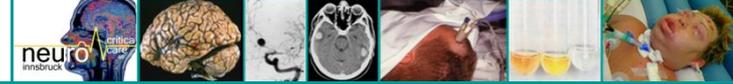


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Conflict of interest:

Speaker's honoraria from ZOLL Medical and
Honoraria for manuscripts from Pfizer





OUTLINE

Introduction



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Definition: ICP, CPP, PbtO₂, metabolic monitoring, lactate, pyruvate, brain temperature etc

Epidemiology of ischemic stroke and ICP elevation in ischemic stroke

ACM infarction and the role of collaterals and/or lack of collaterals for ICP etc

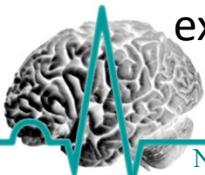
Hydrocephalus in posterior fossa (mainly cerebellar) infarction

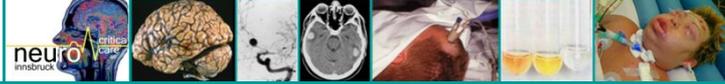
Diagnosis of ICP etc

Monitoring of ICP etc

Therapeutic management:

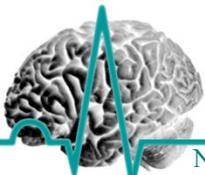
decompressive craniectomy – meta-analysis of DESTINY HAMLET und co
deepening of analgosedation, ventilation, hyperventilation, osmotherapy,
CPP-oriented th, MAP management,
hypothermia, prophylactic normothermia,
external ventricular drainage





Introduction

- Malignant cerebral edema following ischemic stroke is life threatening.
- The pathophysiology of brain edema involves failure of the sodium-potassium adenosine triphosphatase pump and disruption of the blood-brain barrier, leading to cytotoxic edema and cellular death.
- The Monro-Kellie doctrine clearly states that - since the brain is encased in a finite space - increased intracranial pressure (ICP) due to cerebral edema can result in herniation through the foramen magnum and openings formed by the falx and tentorium.
- Moreover, elevated ICP can cause secondary brain ischemia through decreased cerebral perfusion and blood flow, brain tissue hypoxia, and metabolic crisis.
- Direct cerebrovascular compression caused by brain tissue shifting can lead to secondary infarction, especially in the territories of the anterior and posterior cerebral artery.
- Tissue shifts can also stretch and tear cerebral vessels, causing intracranial hemorrhage such as Duret's hemorrhage of the brainstem.



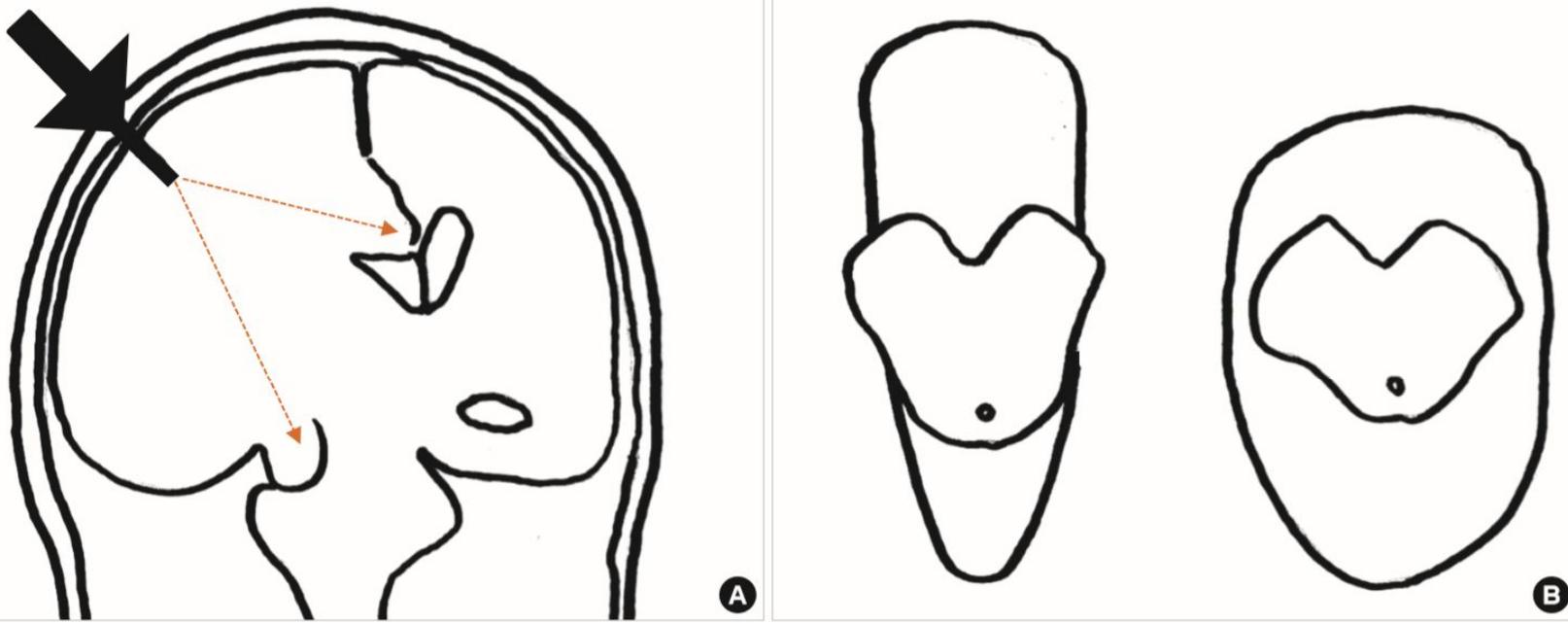


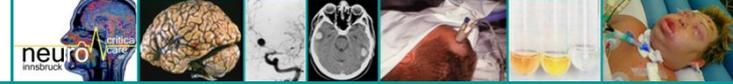
Figure 1. Measurement of ICP and herniation. (A) The distance (orange lines) from the ICP probe (black arrow) to the herniation site may contribute to the discrepancy between measured ICP and clinical deterioration. (B) Variations in the morphometric relationship between the tentorial aperture and the brainstem. Note the differences between the brainstem and the tentorial notches. The patient on the left is more susceptible to herniation syndromes and Kernohan's notch than the one on the right.

Critical Care for Patients with Massive Ischemic Stroke

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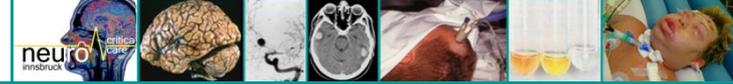




Escalation management of increased intracranial pressure I

- A. Consideration of surgical decompression and/or drainage of cerebrospinal fluid (EVD)
- B. General management
 - a. Mechanical ventilation after rapid sequence intubation
 - b. Head elevation to 30°
 - c. Neutral neck position and avoidance of constricting tube ties
 - d. Avoidance of hypoxia, hyper- and hypocapnia, electrolyte disturbances as hypo- or hypernatremia
 - e. Control of fever and hyper- and/or hypoglycemia
 - f. Treat seizures, do not use prophylactic anticonvulsants
 - g. close cardiopulmonary-, BP-, clin.-neurological (and invasive neuro-)monitoring



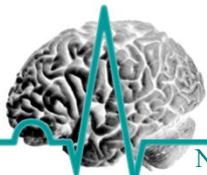


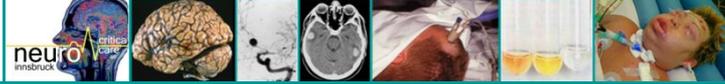
Escalation management of increased intracranial pressure II



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- C. Sedation and analgesia
- D. Moderate (short-term) Hyperventilation: PaCO₂ 30-35 mmHg (or 25-30 mmHg)
- E. Optimization of cerebral perfusion pressure:
intracranial pressure ≤ 22 mmHg
cerebral perfusion pressure ≥ 60 mmHg (individualized)
if present: treat low MAP (fluids, catecholamines)
- F. Osmotherapy: mannitol or hypertonic saline
- G. Therapeutic hypothermia
- H. Barbiturate coma therapy





ORIGINAL RESEARCH

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 **frontiers**
in Aging Neuroscience

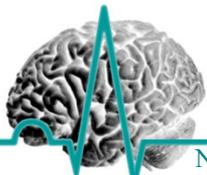


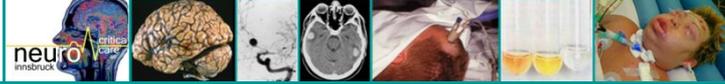
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Intracranial Pressure Elevation 24 h after Ischemic Stroke in Aged Rats Is Prevented by Early, Short Hypothermia Treatment

*Lucy A. Murtha, Daniel J. Beard, Julia T. Bourke, Debbie Pepperall, Damian D. McLeod and Neil J. Spratt**

Translational Stroke Research Laboratory, Faculty of Health and Medicine, School of Biomedical Sciences and Pharmacy, Hunter Medical Research Institute and The University of Newcastle, Callaghan, NSW, Australia

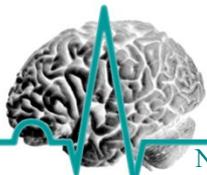


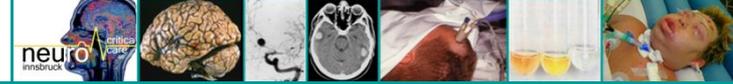


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Twenty-four hours post-stroke:

- ICP was significantly higher in normothermic animals compared to hypothermia-treated animals (27.4 ± 18.2 mmHg vs. 8.0 ± 5.0 mmHg, $p = 0.03$).
- Infarct and edema volumes were not significantly different between groups.
- Temperature management essential to contain ICP.
- Temperature management not so essential to reduce infarct-volume.





CPP and Collaterals

Therapeutic alterations in CPP (may) alter perfusion of the ischemic penumbra via collaterals

Collateral Failure Is Associated With Delayed Infarct Progression

Leptomeningeal collateral supply is typically good at the time of acute imaging, but deteriorates by the time of follow-up imaging (collateral failure)

ICP Elevation After Stroke: Importance for Collateral Blood Flow

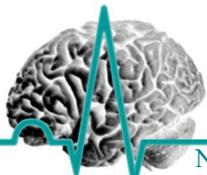
Elevated ICP is a significant problem in several forms of neurological injury including stroke; after exhaustion of all compensatory intracranial mechanisms ICP compromises collaterals thereby leading and/or adding to secondary neurological injury and (sometimes) death

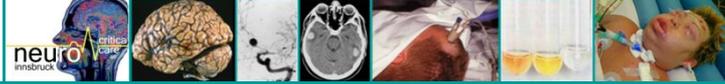
ICP Elevation After Stroke

ICP elevation is known to occur between 1 and 3 days after large hemispheric ischemic stroke and dramatic ICP rise is frequently a preterminal event

ICP and Collateral Blood Flow

ICP elevation reduces collateral flow post stroke





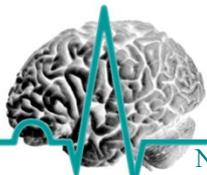
The morbidity associated with ischemic stroke extends beyond the initial insult because of the risk of

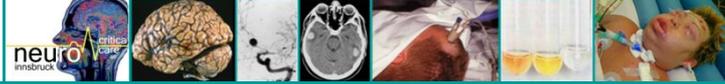
- generating clinically worrisome cerebral edema capable of
- raising ICP and
- causing compression of adjacent non-infarcted tissue.

Careful monitoring for evidence of

- hydrocephalus,
- herniation, and
- worsening midline shift in conjunction with evaluation for
- worsening neurological deficits is critical in the early postinfarct period. Decline in consciousness is the most common sign of worsening cerebral edema.

modified acc to McDowell et al 2017





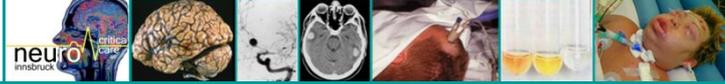
Medical management to reduce the volume of cerebral edema is the recommended initial intervention, but patients refractory to medical treatment should be considered for surgical decompression.

Routine placement of intracranial monitoring devices or EVDs before the onset of severe cerebral edema is not recommended.

Early treatment with thrombolytics or endovascular thrombectomy may reduce the risk of severe cerebral edema formation by reducing infarct volume.

modified acc to McDowell et al 2017





Stroke and ICP - important publications

Hacke W, Schwab S, Horn M, Spranger M, De Georgia M, von Kummer R. 'Malignant' middle cerebral artery territory infarction: clinical course and prognostic signs. *Arch Neurol* 1996;53:309-315

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Kasner SE, Demchuk AM, Berrouschot J, Schmutzhard E, Harms L, Verro P, et al. Predictors of fatal brain edema in massive hemispheric ischemic stroke. *Stroke* 2001;32:2117-2123.

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