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**Goals of cranioplasty**

facilitate neurological recovery

improve cerebral blood flow

restore cerebrospinal fluid dynamics and normal cerebral compliance

prevent the consequences of hydrostatic pressure - restore the gradient between atmospheric- and intracranial pressure

provide better cosmetic results/appearance
Introduction

Definition:

**Cranioplasty**: surgical procedure to repair cranial defects for cosmetic and functional purposes.

Indications:

- traumatic injuries
- decompressive craniectomies
- congenital anomalies
- tumor removal

Contraindications:

- hydrocephalus
- infection
- brain swelling due to any reasons
Comparative Study of Outcomes between Shunting after Cranioplasty and in Cranioplasty after Shunting in Large Concave Flaccid Cranial Defect with Hydrocephalus
Major challenges in cranioplasty

What material to use?
When to perform?
Cranioplasty 1519
• Time/timing 332/44
  • Early 142
  • Late 60
Early - Late 142-60

The earlier the better!
Complications related to cranioplasty

Reabsorption
Cosmetic failure
Infection
EDH/SDH/ICH
Hydrocephalus
Complications related to cranioplasty

Reabsorption
Cosmetic failure
Infection
EDH/SDH/ICH
Hydrocephalus
Complications related to cranioplasty

Reabsorption
Cosmetic failure
Infection
EDH/SDH/ICH
Hydrocephalus
Calvarial reconstruction using high-density porous polyethylene cranial hemispheres

Nitin J Mokal, Mahinoor F Desai

IJPS; 2011; Vol44
Complications related to cranioplasty

Reabsorption
Cosmetic failure
Infection
EDH/SDH/ICH
Hydrocephalus
Complications are defined by:

age,  
gender,  
comorbidities,  
material,  
site of skull defect,  
time between decompression and cranioplasty
Key questions

What is a decompressive craniectomy?

• Of 24 manuscripts, only one defines the size
  • The average size of the bone defect was 69.5 (19.5-149.5)cm².

??????
Large defect - ample consequences
Small defect can also cause severe psychological problems (Gilmour, C., CMJ, 1919)
Key questions

What is early?
- Immediately after edema disappears
- Within 4 weeks
- Within 3 months

What is late?
- After 3 months
- After 6 months
- Over a year
Traditional thoughts about early cranioplasty

+  
  • Safe when edema decreased  
  • Prevents sinking skin flap  
  • Decreases the chance for altered CSF circulation  
  • Cosmetic solution --- psychological aid facilitating recovery/rehabilitation  
  • Prevents injury
Traditional thoughts about early cranioplasty

- Early second hit
- Pathobiology is not resolved, the brain is more vulnerable
- Higher tendency for edema/swelling
Complications of cranioplasty

CNS infection, hydrocephalus, intracranial hematoma and subdural fluid collection, All may prolong the hospitalization, unfavorable prognosis, death
Review

Early cranioplasty vs. late cranioplasty for the treatment of cranial defect: A systematic review

Hao Xu *, Chaoshi Niu, Xianming Fu, Wanhai Ding, Shiyong Ling, Xiaofeng Jiang, Ying Ji

Department of Neurosurgery, Anhui Provincial Hospital Affiliated to Anhui Medical University, Hefei, Anhui 230001, PR China
full text randomized and non-randomized controlled trials (1994-2014)

early CP (1–3 months after DC)
late CP (3–6 months after DC)

Traumatic brain injury, cerebral infarction, subarachnoid hemorrhage and ICH
significant difference in mean operating time / mean difference = -13.46 min/
No difference between the overall complications and infection rate
Hydrocephalus is significantly higher in the early cranioplasty group.
no difference between **intracranial hematoma rates** and **subdural fluid collection rates**
Chaturvedi et al, 2015 BrJNs

74 patients, mortality 1.35% overall complication rate 31%
operating time more than 90 min Odds ratio (OR) 4.77 (1.61-14.20);
timing of CP less than 3 months after craniectomy, OR 2.86 (1.48-8.11);
age more than 20 years, OR 2.59 (1.20-6.53);
female gender, OR 1.91 (1.13-4.17).
Early cranioplasty within 3 months and late cranioplasty after 3 months

Intergroup differences according to cranioplasty time after craniectomy were not observed (p=0.083).
independent risk factors for complications:

• Older age,
• poorer functional situation (worse Barthel index score)
• early surgery (≤85 days)

earlier surgery and larger bone defects increase clinical improvement
174 patients who underwent TC at two London units over seven years.

Non-significant trend: craniectomy-to-cranioplasty interval of 4-8 months with the lowest complication rate and shortest postoperative hospital stay.

Patients with a skull defect larger than 100 cm(2) had the highest complication rate (p < 0.001), highest plate removal rate (p = 0.039), and longest postoperative hospital stay (p = 0.019).

Bifrontal versus unilateral cranioplasty was associated with a significantly higher complication rate (40 vs 14%).
Special issues – Pediatric population
in 3 of 4 manuscripts the effect of time between craniectomy and cranioplasty on complication rate, the authors found no significant effect,
in 1 of 4 the incidence of bone resorption was significantly lower in children who had undergone early cranioplasty
Sixty-one patients were divided into early (< 6 weeks; 28 patients) and late (≥ 6 weeks; 33 patients) cranioplasty cohorts. Bone resorption after cranioplasty was significantly more common in the late (42%) than the early (14%) cranioplasty cohort (p < 0.05; OR 5.4). No other complication differed in incidence between the cohorts.
Special issues - DC-related Hydrocephalus
Cranioplasty and Ventriculoperitoneal Shunt Placement after Decompressive Craniectomy: Staged Surgery Is Associated with Fewer Postoperative Complications.

41 cranioplasty procedures with simultaneous or staged VPS placement

overall complication rate: 27%

47% vs. 12%; P = 0.03
Cranioplasty and ventriculostomy followed by a second stage placement of a ventriculoperitoneal shunt are associated with fewer complications in the treatment of hydrocephalus after DC.
Conclusions

Decompressive craniectomy may harbor significant consequences: not a harmless intervention!

We lack solid scientific evidence to define the optimal timing of cranioplasty

In case of hydrocephalus stage treatment with the priority of CP (or CP+ventriculostomy) seems advisable

In the pediatric population late cranioplasty may increase the rate of reabsorption
J Neurotrauma. 2015 Nov 5. [Epub ahead of print]

THE EFFECT OF CRANIOPLASTY ON CEREBRAL HEMODYNAMICS AS MEASURED BY PERFUSION CT AND DOPPLER ULTRASONOGRAPHY.

Paredes I1, Castaño-Leon AM2, Cepeda S3, Alen JF4, Salvador E5, Millán JM6

Author information

Abstract

Cranioplasties are performed to protect the brain and correct cosmetic defects, but there is growing evidence that this procedure may result in neurological improvement. We prospectively studied cranioplasties performed at our hospital over a 5

KEYWORDS:
CBF autoregulation; CEREBRAL VASCULAR DISEASE; Cranioplasty; Lindegaard ratio; PE

PMID:
26541365
[PubMed - as supplied by publisher]
Thank You!
Decompressive craniectomy is a method to open/expand the space that has defined closed by the Monro-Kellie doctrine in order to reduce ICP

- Primary DC:
  - Preventive/preemptive craniectomy upon evacuation of a space occupying lesion

- Secondary DC:
  - Decompressive craniectomy aimed at the reduction of ICP in lack of a space occupying lesion
A

PRESSURE-VOLUME CURVE: NORMAL ADULT
(ICP vs ΔV)

ICP (mmHg)

ΔV (ml)

Compliance = 0.14

Compliance = 0.62

b

a

B

PRESSURE-VOLUME INDEX (PVI) IN NORMAL ADULT
(log ICP vs ΔV)

Log ICP (mmHg)

ΔV (ml)

PV1 = 25 ml
(Calculated volume to raise ICP x 10)
Kocher, 1901...

...there is an ongoing debate about:

• Indications
• Timing
• Methods
  • Cranium
  • Dura
  • Parenchyma
• EBM
Indications

Primary indication is uncontrollable ICP...

...what does this mean?
CPP = MABP - ICP

60 = 80 - 20
ICP-threshold of 20mmHg

- 6m outcome in 428 sTBI cases
- Occurrence of ICP periods over 20 is associated with adverse outcome

Impact of ICP instability and hypotension on outcome in patients with severe head trauma

Anthony Marmarou, Ph.D., Randy L. Anderson, Ph.D., John D. Ward, M.D., Sung C. Choi, Ph.D., and Harold F. Young, M.D.
Division of Neurosurgery and Department of Biostatistics, Medical College of Virginia, Richmond, Virginia

Howard M. Eisenberg, M.D.
Division of Neurosurgery, University of Texas Medical Branch, Galveston, Texas

Mary A. Foukes, Ph.D.
Biometry and Field Studies Branch, National Institute of Neurological Disorders and Stroke, Bethesda, Maryland

Lawrence F. Marshall, M.D.
Division of Neurological Surgery, University of California, San Diego, California

John A. Jane, M.D.
Department of Neurosurgery, University of Virginia School of Medicine, Charlottesville, Virginia

In 207 sTBI cases:

ICP was over 20 if CT positive in 60%
ICP was over 20 if CT negative in 13%
ICP was over 20 if CT negative but two of the following occurred:
- age over 40
- BPsyst under 90
- decerebrate/decorticate posturing

Intracranial pressure: to monitor or not to monitor?

A review of our experience with severe head injury

Raj K. Narayan, M.D., Pulla R. S. Kishore, M.D., Donald P. Becker, M.D.,
John D. Ward, M.D., Gregory G. Enas, B.S., Richard P. Greenberg, M.D., Ph.D.,
A. Domingues Da Silva, M.D., Maurice H. Lipper, M.D., Sung C. Choi, Ph.D.,
C. Glen Mayhall, M.D., Harry A. Lutz III, Ph.D., and Harold F. Young, M.D.

Divisions of Neurological Surgery, Neuroradiology, and Infectious Disease, and the Department of
Biostatistics, Medical College of Virginia, Virginia Commonwealth University, Richmond, Virginia

J. Neurosurg 56: 650-659, 1982
Pécs Severe TBI Database - 308 cases (06.2002-12.2008)
Prolonged, refractory ICP is a bad prognosticator...
Critical approach

- ICP and CPP are global measures
- Several studies, including the BEST TRIP trial highlight that „treating” ICP does not necessarily mean a treatment for brain injury
Deterioration is not exclusively caused by enlargement of the ICH ...

- cerebral infarction, measured by brain tissue oxygen monitoring, can occur despite normal ICP readings
- increased ICP detection is only responsible for half the episodes of cerebral ischemia.


low PbO2 was associated with normal CPP, indicating that CPP could be an inadequate estimate of regional CBF in focal ischemic areas

Multimodality monitoring in severe TBI

- MABP
- ICP
- CPP/PRx/CPPopt
- SATO$_2$/Astrup
- Brain temperature
- Brain oxygenation/Lycox
- Jugular bulb oxymetry
- ECG
- ECoG
- hemodynamics
- core/tympanic membrane temperature
- microdialysis/biomarkers
Causes of raised ICP following TBI

<table>
<thead>
<tr>
<th>Table 1. Causes of Post-TBI Increased ICP^{10,11}</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Cerebral edema</td>
</tr>
<tr>
<td>• Hyperemia</td>
</tr>
<tr>
<td>• Mass lesion: epidural hematoma; subdural hematoma; hemorrhagic contusions; depressed skull fracture; foreign body</td>
</tr>
<tr>
<td>• Cerebral vasodilation</td>
</tr>
<tr>
<td>• Systemic hypertension</td>
</tr>
<tr>
<td>• Hydrocephalus</td>
</tr>
<tr>
<td>• Venous sinus thrombosis or any other obstruction</td>
</tr>
<tr>
<td>• Posttraumatic seizure activity (status epilepticus, subclinical seizures)</td>
</tr>
<tr>
<td>• Increased intrathoracic or intraabdominal pressure, caused by mechanical ventilation, agitation, or abnormal motor posturing</td>
</tr>
<tr>
<td>• Hyperthermia or febrile states</td>
</tr>
<tr>
<td>• Lightening from coma with inadequate sedation</td>
</tr>
</tbody>
</table>

TBI = traumatic brain injury; ICP = intracranial pressure.
Timing of DC

• There is a lack of evidence to define when to perform DC
• Multimodality monitoring including trend- and waveform- analysis of ICP as well as PRx should provide a solid basis for this
• First exclude technical and extra-CNS causes
• Next, define what measures had been done and what other second tier therapies can be applied

• The decision is based on local guidelines and individual decision, case-by case
Forms

Frontal (bifrontal)
- With bony bridge over the SSS
- Without bony ridge over the SSS
  - With cut over the frontal insertion of the falx (crista galli)
  - Without

Lateral (bilateral) fronto-temporo-parietal

Dural opening
- Slit
- Curved-linear
- Wide radiate/stellate

Dural closure
- None
- Approximating
- Watertight expansion (duroplasty)
Size

The bigger the better...

- Any technical modification will be unnecessary when the size is adequate
- Minimum of 10cmx10cm
- Optimal is 12cmx12cm or over

Large bone defect harbors more complications, particularly that of hydrocephalus
Complications related to DC (and CP)
<table>
<thead>
<tr>
<th>Complications arising from decompressive craniectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subdural hygroma (16%–50%)</td>
</tr>
<tr>
<td>Progression of hemorrhage/contusion (5%–58%)</td>
</tr>
<tr>
<td>Intracranial infection (2%–6%)</td>
</tr>
<tr>
<td>Contralateral SDH/EDH (6%–28%)</td>
</tr>
<tr>
<td>Hydrocephalus (2%–29%)</td>
</tr>
<tr>
<td>Herniation through skull defect (26% in 1 case study defining: herniation as brain tissue in the center of the defect &gt;1.5 cm above plane of normal outer table of skull)</td>
</tr>
<tr>
<td>Syndrome of the trephine: a late complication consisting of headaches, confusion, dizziness, memory difficulties, mood disturbances, and sometimes motor disturbances, consisting of progressive contralateral upper limb weakness not previously affected by injury (10 of 38 patients in 1 case series). In many cases, this syndrome can be reversed by cranioplasty; all patients developing motor symptoms in the case series experienced full and rapid motor recovery within days of their cranioplasty</td>
</tr>
<tr>
<td>Paradoxical herniation: has been reported as occurring as a result of lumbar puncture after large decompressive craniectomy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Complications of subsequent cranioplasty</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone flap resorption/sinking after cranioplasty (1.6%–12%)</td>
</tr>
<tr>
<td>Infection (11.3%)</td>
</tr>
<tr>
<td>Status epilepticus (1.6%): note that seizures after neurosurgical procedures are a well-recognized occurrence</td>
</tr>
</tbody>
</table>

SDH = subdural hematoma; EDH = extradural hematoma. Percentages (where available) are quoted from those reported from several individual studies.
Calvarial reconstruction using high-density porous polyethylene cranial hemispheres

Nitin J Mokal, Mahinoor F Desai

IJPS; 2011; Vol44
Pseudohypoxic Brain Swelling: A Newly Defined Complication after Uneventful Brain Surgery, Probably Related to Suction Drainage

Van Roost, Dirk M.D.; Thees, Christof M.D.; Brenke, Christopher M.D.; Oppel, Falk M.D.; Winkler, Peter A. M.D.; Schramm, Johannes M.D.

Abstract

OBJECTIVE: This is the first description of a severe and sometimes fatal complication after uneventful intracranial surgery. The clinical presentation and imaging features mimic those of global cerebral hypoxia. Extensive investigations were performed to discover the pathogenesis.

METHODS: Seventeen cases of pseudohypoxic brain swelling (PHBS) were collected from our institution and from various other neurosurgical departments and were studied for common features. PHBS can occur in a mild, moderate, or severe degree. It is characterized by a very early postoperative onset of clinical deterioration (clouded or lost consciousness and pupillary abnormalities), in association with typical bilateral computed tomographic or magnetic resonance imaging changes (hypodensities or altered intensities in the basal ganglia and/or thalamus). The following variables were considered: age, primary pathological lesion and intracranial location, previous cranial surgery, anesthetic risk, type of anesthesia, approach and duration of surgery, intraoperative observations, technical monitoring results, and blood gas analyses. The results of postoperative computed tomography and various other imaging studies, intracranial pressure measurements, transcranial Doppler sonography,
65 ys ♀

Sept. 06. 2015

Sept. 07. 2015

Sept. 14. 2015
65 ys ♀

Sept. 19. 2015

Sept. 20. 2015

Sept. 23. 2015
EBM...
Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury

- There is no evidence to support the routine use of secondary DC to reduce unfavorable outcome in adults with severe TBI and refractory high ICP.
- In the pediatric population DC reduces the risk of death and unfavorable outcome.
- …this treatment maybe justified in patients below the age of 18 when maximal medical treatment has failed to control ICP.
- To date, there are no results from randomized trials to confirm or refute the effectiveness of DC in adults.
- …results of non-randomized trials and controlled trials with historical controls involving adults, suggest that DC may be a useful option when maximal medical treatment has failed to control ICP.
Decompressive Craniectomy in Diffuse Traumatic Brain Injury

D. James Cooper, M.D., Jeffrey V. Rosenfeld, M.D., Lynnette Murray, B.App.Sci., Yaseen M. Arabi, M.D., Andrew R. Davies, M.B., B.S., Paul D’Urso, Ph.D., Thomas Kossmann, M.D., Jennie Ponsford, Ph.D., Ian Seppelt, M.B., B.S., Peter Reilly, M.D., and Rory Wolfe, Ph.D., for the DECRA Trial Investigators and the Australian and New Zealand Intensive Care Society Clinical Trials Group*
Characteristics of the trial

In 8y sTBI patients (19-59y) in 15 tertiary care hospitals in Australia, New Zealand, and Saudi Arabia.

treatment for ICP over 20 mm Hg

early refractory elevation in intracranial pressure:

• a spontaneous (not stimulated) increase in intracranial pressure for more than 15 minutes (continuously or intermittently) within a 1-hour period, despite optimized first-tier interventions.

interventions included

• optimized sedation, the normalization of arterial carbon dioxide pressure, and the use of mannitol, hypertonic saline, neuromuscular blockade, and external ventricular drainage
Randomization in 72 hours after injury to decompressive craniectomy plus standard care or to receive standard care alone

Standard care:

- Brain Trauma Foundation - Guidelines.

Second-tier options for refractory elevation of intracranial pressure:

- mild hypothermia (to 35°C),
- optimized use of barbiturates,
- both.

Patients randomized to continued standard care:

- protocol permitted the use of lifesaving decompressive craniectomy after a period of 72 hours had elapsed since admission.
Table 1. Baseline Characteristics of the Patients.*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Decompressive Cranectomy (N=73)</th>
<th>Standard Care (N=82)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age — yr</td>
<td></td>
<td></td>
<td>0.89</td>
</tr>
<tr>
<td>Medial</td>
<td>23.7</td>
<td>24.6</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>19.4–29.6</td>
<td>18.5–34.9</td>
<td></td>
</tr>
<tr>
<td>Male sex — no. (%)</td>
<td>59 (81)</td>
<td>61 (74)</td>
<td>0.44</td>
</tr>
<tr>
<td>Systolic blood pressure — mm Hg</td>
<td>135.4±32.0</td>
<td>135.7±27.6</td>
<td>0.95</td>
</tr>
<tr>
<td>Glasgow Coma Scale</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall score†</td>
<td></td>
<td></td>
<td>0.31</td>
</tr>
<tr>
<td>Medial</td>
<td>5</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>3–7</td>
<td>4–7</td>
<td></td>
</tr>
<tr>
<td>Motor score§</td>
<td></td>
<td></td>
<td>0.49</td>
</tr>
<tr>
<td>Medial</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>1–4</td>
<td>1–5</td>
<td></td>
</tr>
<tr>
<td>Maximum score for head injury on Abbreviated Injury Scale — no. (%)</td>
<td></td>
<td></td>
<td>0.52</td>
</tr>
<tr>
<td>3 or 4</td>
<td>35 (48)</td>
<td>44 (54)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>38 (52)</td>
<td>38 (46)</td>
<td></td>
</tr>
<tr>
<td>Injury Severity Score§</td>
<td></td>
<td></td>
<td>0.88</td>
</tr>
<tr>
<td>Medial</td>
<td>33</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>25–38</td>
<td>24–41</td>
<td></td>
</tr>
<tr>
<td>Trauma Score–Injury Severity Score**</td>
<td></td>
<td></td>
<td>0.46</td>
</tr>
<tr>
<td>Medial</td>
<td>0.74</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>0.42–0.88</td>
<td>0.51–0.90</td>
<td></td>
</tr>
<tr>
<td>Reactivity of pupils — no./total no. (%)</td>
<td></td>
<td></td>
<td>0.04</td>
</tr>
<tr>
<td>Neither pupil</td>
<td>19/71 (27)</td>
<td>10/80 (12)</td>
<td></td>
</tr>
<tr>
<td>One or both pupils</td>
<td>52/71 (73)</td>
<td>70/80 (88)</td>
<td></td>
</tr>
<tr>
<td>Hypotension — no. (%)</td>
<td>24 (33)</td>
<td>25 (30)</td>
<td>0.93</td>
</tr>
<tr>
<td>Hypoxemia — no. (%)</td>
<td>18 (25)</td>
<td>24 (29)</td>
<td>0.55</td>
</tr>
<tr>
<td>Traumatic subarachnoid hemorrhage — no. (%)</td>
<td>42 (58)</td>
<td>48 (59)</td>
<td>0.90</td>
</tr>
<tr>
<td>Cause of injury — no./total no. (%)</td>
<td></td>
<td></td>
<td>0.72</td>
</tr>
<tr>
<td>Motor-vehicle or motorcycle accident</td>
<td>45/70 (64)</td>
<td>55/81 (68)</td>
<td></td>
</tr>
<tr>
<td>Bicycle accident</td>
<td>4/70 (6)</td>
<td>4/81 (2)</td>
<td></td>
</tr>
<tr>
<td>Pedestrian accident</td>
<td>5/70 (7)</td>
<td>4/81 (5)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>16/70 (23)</td>
<td>20/81 (25)</td>
<td></td>
</tr>
</tbody>
</table>
Figure 1. Intracranial Pressure before and after Randomization.
Shown are the mean measurements of intracranial pressure in the two study groups during the 12 hours before and the 36 hours after randomization. The I bars indicate standard errors.
Table 2. Primary and Secondary Outcomes.*

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Decompressive Craniectomy (N=73)</th>
<th>Standard Care (N=82)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracranial pressure and cerebral perfusion pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intracranial pressure after randomization — mm Hg</td>
<td>14.4±6.8</td>
<td>19.1±8.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No. of hr of intracranial pressure &gt;20 mm Hg — median (IQR)</td>
<td>9.2 (4.4–27.0)</td>
<td>30.0 (14.9–60.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Intracranial hypertension index — median (IQR)‡</td>
<td>11.5 (5.9–20.3)</td>
<td>19.9 (12.5–37.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cerebral hypoperfusion index — median (IQR)¶</td>
<td>5.7 (2.5–10.2)</td>
<td>8.6 (4.0–13.8)</td>
<td>0.03</td>
</tr>
<tr>
<td>Duration of hospital intervention</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days of mechanical ventilation — median (IQR)</td>
<td>11 (8–15)</td>
<td>15 (12–20)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Days of ICU stay — median (IQR)</td>
<td>13 (10–18)</td>
<td>18 (13–24)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Days of hospitalization — median (IQR)</td>
<td>28 (21–62)</td>
<td>37 (24–44)</td>
<td>0.82</td>
</tr>
<tr>
<td>Extended Glasgow Outcome Scale</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score — no. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (dead)</td>
<td>14 (19)</td>
<td>15 (18)</td>
<td></td>
</tr>
<tr>
<td>2 (vegetative state)</td>
<td>9 (12)</td>
<td>2 (2)</td>
<td></td>
</tr>
<tr>
<td>3 (lower severe disability)</td>
<td>18 (25)</td>
<td>17 (21)</td>
<td></td>
</tr>
<tr>
<td>4 (upper severe disability)</td>
<td>10 (14)</td>
<td>8 (10)</td>
<td></td>
</tr>
<tr>
<td>5 (lower moderate disability)</td>
<td>13 (18)</td>
<td>20 (24)</td>
<td></td>
</tr>
<tr>
<td>6 (upper moderate disability)</td>
<td>6 (8)</td>
<td>13 (16)</td>
<td></td>
</tr>
<tr>
<td>7 (lower good recovery)</td>
<td>2 (3)</td>
<td>4 (5)</td>
<td></td>
</tr>
<tr>
<td>8 (upper good recovery)</td>
<td>1 (1)</td>
<td>3 (4)</td>
<td></td>
</tr>
<tr>
<td>Median score (IQR)</td>
<td>3 (2–5)</td>
<td>4 (3–5)</td>
<td>0.03</td>
</tr>
<tr>
<td>Unfavorable score of 1 to 4 — no. (%)</td>
<td>51 (70)</td>
<td>42 (51)</td>
<td>0.02</td>
</tr>
</tbody>
</table>
Conclusions of the study

The method „works”

The outcome is unaffected

?????
• Decompressive craniectomy does not improve outcome when it is done on patients who don’t need it!

(Chesnut, R., 2014, INTS, Budapest)
Why do patients not require DC?

- ICP is not elevated!

- Is ICP relevant to define the treatment options?

- Sometimes yes, sometimes not!
Let’s forget the „one size fits all” – approach!
Individual pathobiology matters!
Mortality according to the main intracranial pathology & ICP monitoring

<table>
<thead>
<tr>
<th>Condition</th>
<th>Total</th>
<th>ICP Monitoring+</th>
<th>ICP Monitoring-</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICH</td>
<td>47.83%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICH+SDH</td>
<td>52.33%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDH</td>
<td>42.22%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diffuse</td>
<td>42.86%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Penetrating</td>
<td>64.71%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Individual assessment of the pathobiology!

**CASE STUDY**
Severe head trauma following a road traffic accident.
Continuous monitoring of cerebral autoregulation

Colour coded autoregulation (AR) index PRx
- AR intact
- AR impaired

Courtesy of Peter Smielewski
Conclusions

• In order to introduce a treatment we have to understand the pathobiology
• We also have to understand pathobiological processes evoked by/ operant at an individual level
• Treatment plans and decisions should be tailored to the actual patient
A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury

Randall M. Chesnut, M.D., Nancy Temkin, Ph.D., Nancy Carney, Ph.D., Sureyya Dikmen, Ph.D., Carlos Rondina, M.D., Walter Videtta, M.D., Gustavo Petroni, M.D., Silvia Lujan, M.D., Jim Pridgeon, M.H.A., Jason Barber, M.S., Joan Machamer, M.A., Kelley Chaddock, B.A., Juanita M. Celix, M.D., Marianna Cherner, Ph.D., and Terence Hendrix, B.A.

ABSTRACT
BEST:TRIP trial
n = 324

Randomization to:
  Pressure monitoring group (n=157)
  Imaging - clinical examination group (n=167)

Power calculation: 80% power to detect an increase of 10 percentage points in the % of patient with a favorable outcome (GOSE)

Primary outcome measure: composite outcome at 6 months
Testing Two Protocols

Brain Trauma Foundation
ICP Monitoring

Imaging & Clinical Examination
No ICP Monitoring

Courtesy of R. Chesnut
Conclusions: BEST:TRIP

Apparently sound hypothesis and design

Groups comparable

Care focused on maintaining monitored ICP ≤ 20 mm Hg was not shown to be superior to care based on imaging and clinical examination
BEST:TRIP
Critical comments...

Conceptual: Any differences in outcome will be related to type and intensity of treatment, and not to the monitoring itself.

Both groups received ICP targeted treatment.

Statistical: Power calculations should NOT be based on the total number of patients but on the (expected) number with raised ICP.

Median/mean % of ICP > 20 mmHg: 7 and 20%.

In every fourth patient at the ICP group there was no raised ICP at all!!
BEST:TRIP

Critical comments...

- Composite outcome measures: half of them are neuropsychological
- GOSE: 5% benefit for ICP-group
- Confounding effects of:
  - more aggressive treatment in the cons. group,
  - longer transfer times with no documentation on hypoxia/hypoperfusion
What is the problem?
What should ICP monitoring provide to us?

- timely detection of space occupying lesions...
- user friendly, reliable, cost-efficient tool with minimal complication rate, based upon evidence based medicine...
Does ICP monitoring help…

...to identify those patients who are at risk for late deterioration?
Deterioration is not exclusively caused by enlargement of the ICH ...

several studies have demonstrated that cerebral infarction, measured by brain tissue oxygen monitoring, can occur despite normal ICP readings. Increased ICP detection is only responsible for half the episodes of cerebral ischaemia.


low PbO2 was associated with normal CPP, indicating that CPP could be an inadequate estimate of regional CBF in focal ischaemic areas.

44y ♂ car accident, driver
EO 1, VR 2, MR 4: GCS: 7, pupils equal, reactive
left hemiparesis
severe pulmonary and mild liver contusion, unstable rib cage
2007.09.19 5:00-21:00

Part.O2

PbrO2
MMSE = 97/100, IQ (TONI-3) = 108

full recovery

back to work

practically unaffected social functions

normal endocrine checkups
Does ICP-monitoring improve outcome?
In order to prove that ICP monitoring per se improves outcome, assuming a 9% mortality reduction, a prospective randomized study including approximately 768 patients would be required...
On the basis of our present knowledge this is an irrelevant question as...

... We do not have pathobiology driven therapeutic targets/decisions to make


...we do not have surrogate markers to compare with
General considerations about neuromonitoring
ICP monitoring: the rationale
ICP monitoring: the debate
Current practice/future directions
Rethinking our approaches to ICP monitoring/treatment

• ICP monitoring should NOT be discarded

• Think in terms of understanding what is going on

• Think in terms of strategies
Trend - Time - Multimodality

• Trend is more important than a single, actual value

• Raised ICP in patients “awakening” is normal

• The more widespread, relevant physiological information we gather is the best!
CPP Management

The concept:
Increase flow by increased driving force

CPP = MABP - ICP

Limitation: not indicated if auto regulation severely disturbed

PRX and CPP opt.

Courtesy of A. Maas
Compared an “ICP Dose” based on correlation with disturbed autoregulation (PRx) versus “standard” ICP > 20 mmHg in predicting clinical outcome.

Our findings become further pertinent in view of the recent publication of the randomized controlled trial of ICP monitoring in severe TBI by Chesnut et al. This was the first such trial to compare management of intracranial hypertension based on monitoring and treatment of ICP above the fixed threshold of 20 mm Hg, versus a protocol based on clinical examination and neuroimaging. This trial has been a significant step forward in the field of neurocritical care and has shed light on the importance of individualized approaches to ICP management.

In conjunction with the aforementioned DECRA trial, we believe that an important aspect in interpreting the results should be the limitation of using fixed, universal ICP thresholds and thus disregarding patient-specific injury patterns, individual pathophysiology, and response to treatment interventions.
Goal directed therapy in Neuro ICU

ICP < 20
CPP > 60
PBrO2 > 15
SjO2 > 55%

Understanding what is going on

Courtesy of AAO Neuro
While recognizing certain limitations of ICP monitoring, the most important message for the near future is:

• Maintain ICP monitoring as a cornerstone of treatment
• Keep up the critical approach and pathophysiology-driven decision making with information gathered from multimodal monitoring
Thank you for your attention!