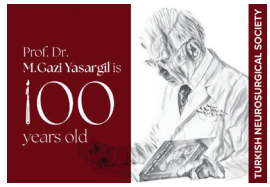
Turk Neurosurg 35(5):772-777, 2025
DOI: 10.5137/1019-5149.JTN.47457-24.1

Technical Note

Neurotrauma

Received: 29.08.2024
Accepted: 11.12.2024
Published Online: 04.09.2025



Identification of Decompressive Craniectomy Patients with Refractory ICP using Burst Suppression Ratio and Novel Subgaleal qEEG: A Technical Note

Alexander J. KIM[1], Daniel R. FELBAUM[2], Jeffrey C. MAI[2], Jason J. CHANG[3]

[1]Georgetown University School of Medicine, Washington, DC, USA
[2]Georgetown University and MedStar Washington Hospital Center, Department of Neurosurgery, Washington, DC, USA
[3]MedStar Washington Hospital Center, Department of Critical Care Medicine; Georgetown University Medical Center, Department of Neurology, Washington, DC, USA

Corresponding author: Alexander J. KIM ✉ ajk186@georgetown.edu

ABSTRACT

Decompressive hemicraniectomy (DHC) can improve outcome in patients with elevated intracranial pressure (ICP) refractory to medical therapy. However, this transition point for treating refractory ICPs with DHC is unclear as ICPs can often be controlled with escalating doses of medical management. A more individualized and precise way to monitor and define medically "refractory ICP" may be achieved with the utilization of a quantitative electroencephalography (EEG) parameter called burst suppression ratio (BSR). This technical note describes a novel device to continuously gather EEG data from subgaleal electrodes. We present two cases where BSR (i.e. an EEG-derived marker) was associated with maximal cortical suppression, indicating refractory ICP and indication for decompression. Two patients [severe traumatic brain injury (sTBI) and ruptured arteriovenous malformation (AVM)] had BSRs measured through placement of novel subgaleal EEG electrodes. Although both patients had ICPs controlled by a combination of sedation, hyperosmolar therapy, and hypothermia, the BSR over a 20-24 hour period quickly reached almost-complete EEG suppression (BSR > 90%). Each case had different reasons for delaying DHC, however both reached maximal medical therapy. Given the limit of ICP control was reached, DHC was conducted in both cases. Patient 1 failed to recover and was compassionately extubated. Patient 2 clinically recovered and was discharged to acute rehabilitation. These cases illustrate that utilization of a novel subgaleal EEG system to continuously monitor BSR in patients who are being medically managed for ICP control may be used to select appropriate candidates for surgical decompression. In our two cases, a threshold BSR value >90% (induced by medical therapy) was associated with the indication for DHC. This can be used in the future as another tool to define the limit of cortical suppression by medical therapy, thereby, indicating decompression.

KEYWORDS: Burst suppression ratio, Continuous electroencephalography, EEG, Decompressive craniectomy, ICP

ABBREVIATIONS: BSR: burst suppression ratio, **DHC:** Decompressive hemicraniectomy, **ICP:** intracranial pressure, **EEG:** Electroencephalography, **sTBI:** Severe traumatic brain injury, **AVM:** Ruptured arteriovenous malformation, **PRx:** Pressure reactivity index, **CTH:** Computed tomography head

Alexander J. KIM : 0000-0003-3355-3122
Daniel R. FELBAUM : 0000-0002-0156-4671
Jeffrey C. MAI : 0000-0002-4990-6431
Jason J. CHANG : 0000-0002-0825-3008

INTRODUCTION

Severe traumatic brain injury (sTBI) is associated with increased intracranial pressure (ICP) due to multifactorial etiologies (4). Current best-practice guidelines recommend monitoring ICPs with target goals ≤ 22 mmHg (1). However, increased therapeutic intensity level often is insufficient to control ICPs (3). In such cases of "ICP refractory to medical management (ICPref)", decompressive craniectomies can be offered as a last-tier rescue intervention (7,9).

Offering decompressive craniectomy in this setting requires a better identification of ICPref, whose definition is often opaque due to the diffuse and heterogeneous injury patterns found in sTBI. Although algorithms for medically treating ICPs exist, individualized patient physiology, metabolic clearance of drugs, and underlying brain activity make standardization of medical sedating drip rates impossible (12).

Standard scalp electroencephalography (EEG) and quantitative EEG (qEEG) can quantify background frequencies and detect early-onset ischemia (5). However, scalp qEEG is often insufficient to evaluate background rhythms in patients with ICPref given the suppressed backgrounds in the setting of high doses of sedation required to control ICPs. Using an FDA-approved, novel, neuromonitoring device that uses subgaleal electrodes, we quantified ICPref in sedated patients using a qEEG marker called "burst suppression ratio" (BSR). BSR ranges from 0 to 100% with 100% indicating full suppression of cortical activity.

We describe two patients who had subgaleal electrodes placed and developed ICPref. In these two patients, higher BSR (> 90%) due to escalating doses of medical management was associated with ICPref aiding the decision for decompression.

MATERIAL and METHODS

We describe a novel subgaleal EEG system, iCEWav (iCEWav Neuromonitoring Platform, iCE Neurosystems, Washington, DC, USA).

Subgaleal EEG via iCEWav

One subgaleal electrode consists of eight leads with electrical charge differences measured over leads 1-5, 2-6, 3-7, and 4-8. Each electrode is inserted via a tunneled trochar into the subgaleal space over a parasagittal plane located at the level of the outer conjunctiva. This parasagittal site is thought to best approximate the watershed zone between the middle cerebral and anterior cerebral arteries.

Raw EEG patterns in left side (L1-L5, L2-L6, L3-L7, L4-L8) and right side (R1-R5, R2-R6, R3-R7, R4-R8) (Figure 1) are then converted into qEEG (Figure 2) that incorporate similar quantitative variables as used in traditional scalp EEG—BSR, compressed spectral array (CSA), alpha-delta-ratio (ADR), TPW (total power)—and can incorporate traditional hemodynamic variables—cerebral perfusion pressure (CPP), mean arterial pressure (mART), ICP, brain oxygen (P4).

Patient Treatment Protocol

The treatment regimen adhered to standard, tiered protocols for managing elevated ICP (6). Parenchymal ICP monitors (i.e. bolt) were used (Neurovent-PTO, Raumedic, Mills River, NC, USA). In both cases, medical management for ICP control was initiated. The customized clinical decision – incorporating data from this subgaleal qEEG – for surgical decompression was made by the treating neurosurgical and neurocritical teams.

RESULTS

Clinical presentation

Case 1

A 67-year-old man with hypertension, chronic kidney disease, atrial fibrillation on anticoagulation was transferred from an outside hospital for sTBI after an unwitnessed fall (Figure 3A). The patient had a Glasgow Coma Scale of 12 on admission but quickly deteriorated and was intubated. A left frontal bolt and subgaleal EEG electrodes were placed for monitoring.

Given his bihemispheric contusions, conservative management was deemed reasonable. However, over the next 20

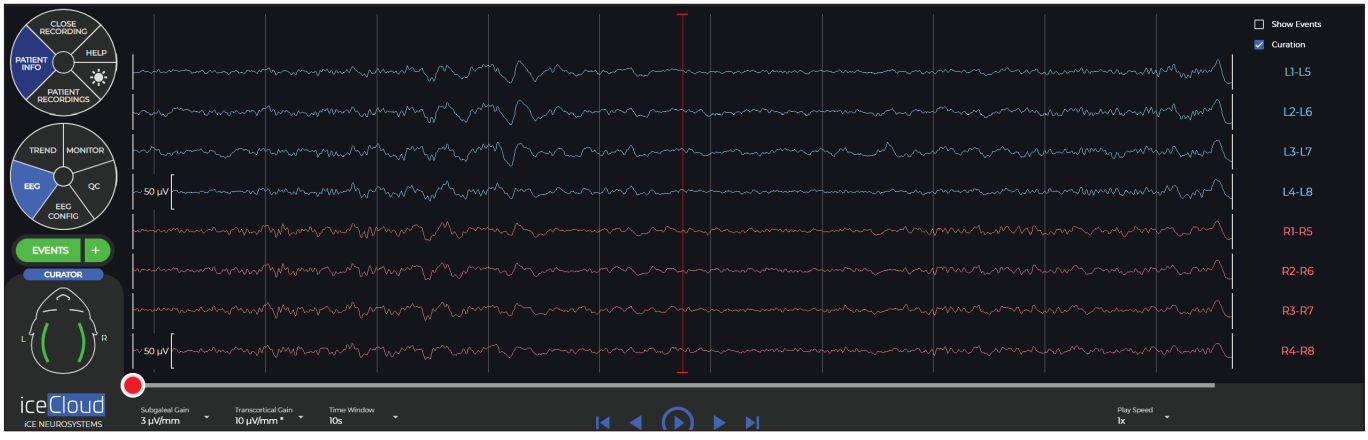


Figure 1: Image from iCEWav demonstrating an example of the raw electroencephalography (EEG) patterns in left side (L1-L5, L2-L6, L3-L7, L4-L8) and right side (R1-R5, R2-R6, R3-R7, R4-R8).

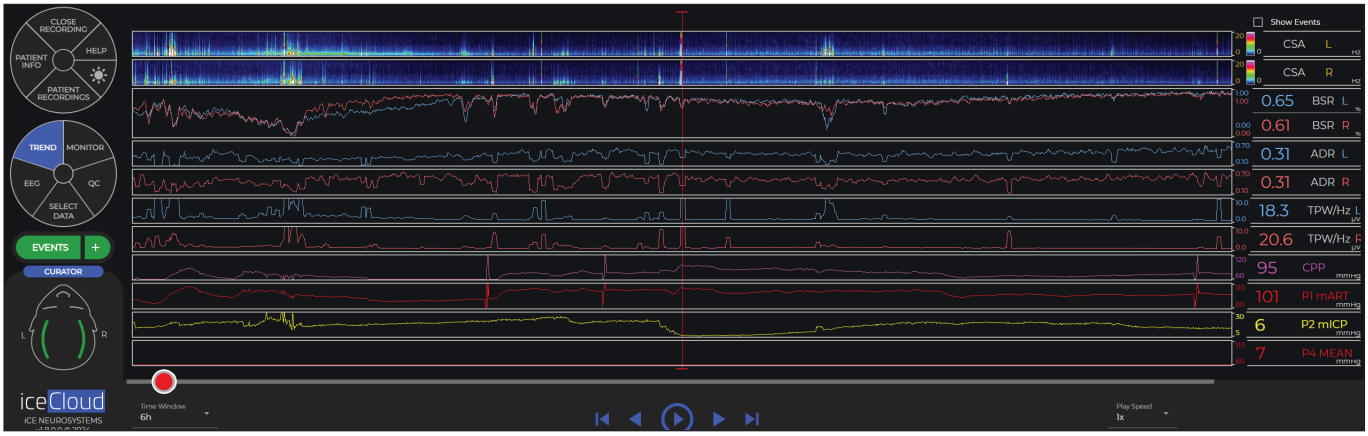


Figure 2: Image from iCEWav demonstrating an example of the quantitative electroencephalography (EEG) values converted from the raw EEG patterns that the system collects. These incorporate similar quantitative variables as used in traditional scalp EEG. **BSR:** Burst suppression ratio, **CSA:** Compressed spectral array, **ADR:** Alpha-delta-ratio, TPW (total power)—and can incorporate traditional hemodynamic variables— **CPP:** Cerebral perfusion pressure, **mART:** Mean arterial pressure, **ICP:** Intracranial pressure, **P4:** Brain oxygen.

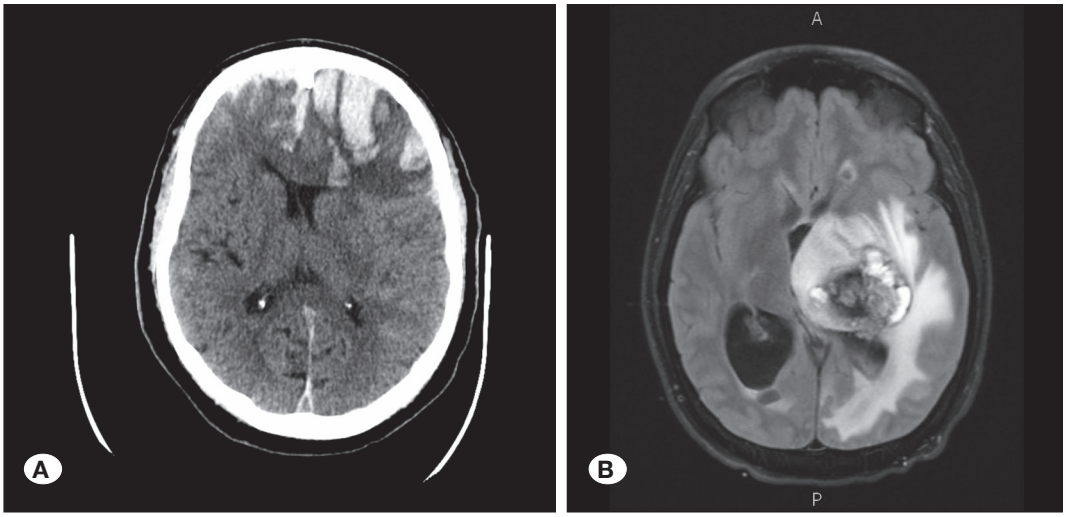


Figure 3: Imaging of **A)** case 1 severe traumatic brain injury demonstrating bifrontal traumatic contusions (left greater than right) and **B)** case 2 demonstrating vasogenic edema and mass effect occurring after a subacutely ruptured arteriovenous malformation.

hours, escalating doses of medical therapy were given for ICP control. He arrived with a body temperature of 32-33°C and this temperature was initially maintained. His medical management included versed drip at 10mg/hr, ketamine drip at 40mg/hr, propofol drip at 50mcg/kg/min, hypertonic saline at 30ml/hr (several boluses were given with a goal serum Na > 160), fentanyl drip at 200mcg/hr, and several vecuronium pushes. His subgaleal electrodes demonstrated extreme burst suppression with a BSR 0.95. On exam, his HR was 49 bpm and sinus rhythm on low-dose levophed to maintain a goal cerebral perfusion pressure (CPP) > 60 mmHg; his pupils remained sluggishly reactive. Despite these treatments, the patient's ICP remained elevated at 22 mmHg. The subgaleal EEGs also demonstrated a steady increase in BSR to 90-100% concurrently with the escalating doses of medication required for ICP control-likely indicating ICPref (Figure 4A). Given the persistently elevated ICPs, the neurosurgical team performed a left-sided decompressive hemicraniectomy. Post-decompression, he remained comatose. A right-frontal bolt was placed post-decompression to evaluate for further blossoming. Despite the decompression, he remained sedated to control his elevated ICPs until post-operative day 6. His exam would unfortunately remain comatose despite achieving adequate ICP control. After discussion between the treating teams and family, a decision for compassionate extubation was made, and he passed away on hospital day 15.

Case 2

A 27-year-old man with a remote known history of a left-sided arteriovenous malformation (AVM), embolized nine years prior and followed by radiosurgery, had been doing well until presenting with headache, nausea, vomiting, and right-side weakness. He remained awake with a Glasgow Coma Score of 14. Outside hospital computed tomography head (CTH) demonstrated a left thalamic hemorrhage, measuring 4.3 x 3.2 cm, with surrounding vasogenic edema and associated ventriculomegaly. Cerebral angiography on hospital day

1 showed no recurrent aneurysm or underlying AVM. The etiology was unclear with a differential of delayed radiation necrosis or malignancy.

He remained clinically intact until hospital day 10 when he was intubated secondary to seizures. He had hydrocephalus and an external ventricular device (EVD) was placed. Despite CSF drainage, his ICPs continued to escalate. Imaging (Figure 3B) showed a stable lesion and extensive vasogenic edema. However, due to persistent and more frequent ICP elevations, a bolt and subgaleal leads were placed. An initial sedation regimen consisting of versed drip, propofol drip, and fentanyl drip was initiated for more consistent ICP control.

The patient continued to have intermittent, but manageable, ICP spikes until he developed a Cushing reaction. His bradycardia persisted, accompanied by worsening shock, requiring intermittent atropine pushes. While he hemodynamically deteriorated, his ICPs continued to increase; he eventually required versed drip at 15mg/hr, propofol drip at 100 mcg/kg/min, fentanyl at 200mcg/hr, targeted temperature goal of 32-33°C, and hypertonic saline at 100ml/hr (intermittent boluses were given with a goal serum Na > 155). Low-dose pentobarbital infusion was also started. BSR increased to 90-100% (Figure 4B). His BSR demonstrated possible ICPref. Due to the persistently elevated ICPs, the neurosurgical team performed a hemicraniectomy on hospital day 15.

Post-decompression, the sedation was weaned and the patient was rewarmed. On hospital day 35, the patient had a tracheostomy and percutaneous gastrostomy tube placed. He underwent right parieto-occipital ventriculoperitoneal shunt placement on hospital day 45. Slowly his exam improved and on hospital day 87, the patient discharged to acute rehabilitation.

DISCUSSION

Our findings suggest that utilization of BSR may provide a quantitative, functional measure that neurosurgeons can utilize to evaluate for necessity of surgical craniectomy in patients with ICPref. BSR, as a functional marker that evaluates cortical suppression, is important because ICP has more subtle meanings than simply being a targeted number (10). By targeting ICP thresholds with protocolized, medical management but ignoring individualized, cortical activity, clinicians may introduce severe levels of sedation that introduce an irreversible, iatrogenic coma and subsequent shock leading to multi-organ failure while offering little improvement in cortical suppression. The precision-based, individualized evaluation of cortical activity via BSR is also important, particularly in sTBI, because oftentimes the neurological injury is diffuse, and, therefore, the benefits of surgical decompression without a targeted mass lesion is less certain.

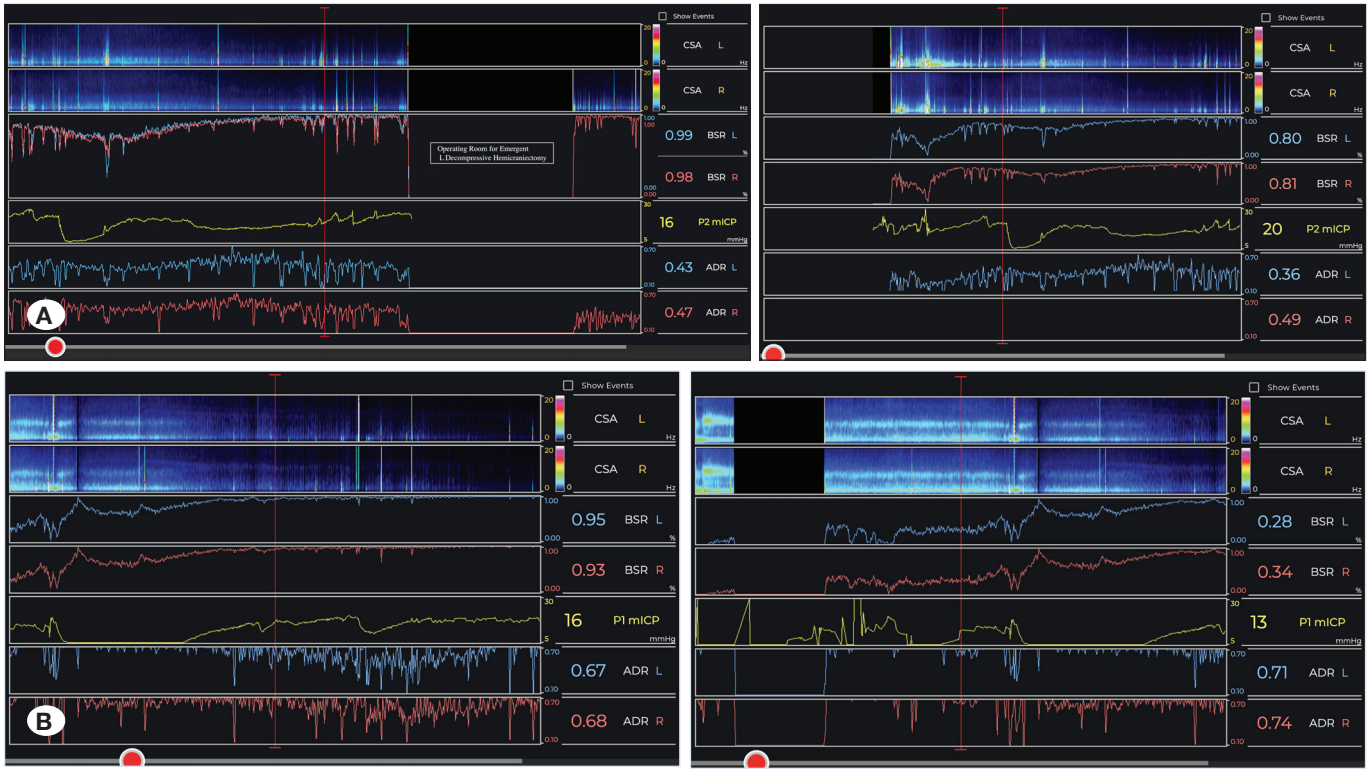


Figure 4: (A) Patient 1: Escalating sedation regimen for increasing intracranial pressure (ICP) correlates with burst suppression ratio (BSR). During this time, patient was on versed drip, propofol drip, fentanyl drip, vecuronium drip, and low-dose norepinephrine drip to achieve a BSR of 0.99 on the left, 0.98 on the right, and intracranial pressure (ICP) of 16 mmHg. **(B) Patient 2:** Escalating sedation regimen for ICP controlled correlated with increasing BSR. During this time, patient was on propofol drip, fentanyl drip, vecuronium drip, low-dose norepinephrine drip to achieve a BSR of 0.95 on the left, 0.93 on the right, and ICP of 16 mmHg. On the figure, **CSA =** compressed spectral array, **BSR =** burst suppression ratio, **P2mICP =** ICP (mmHg), **ADR =** alpha-delta ratio.

In our two cases, we utilized qEEG and BSR – derived from a novel device utilizing continuous subgaleal EEG monitoring – to identify futile levels of sedation and ICPref. This allowed us to identify the time point when medical management had become exhausted and when surgery would need to be offered to prevent further herniation.

A literature search in PubMed does not show any peer-reviewed publication clinically using subgaleal EEG monitoring for evaluation of BSR.

Several limitations exist before considering BSR more routinely as a functional marker for ICPref. First, there is a lack of precision when defining and measuring "BSR", including how these models capture mechanistic versus phenomenological aspects of burst suppression (2,11). Furthermore, BSR as defined in the clinical literature always refers to traditional scalp EEG and not the novel subgaleal EEG that we utilized. We found scalp EEG consistently more suppressed due to the greater distance separating scalp electrodes from cortex. Third, the etiology of burst-suppression is debatable: sedation-induced or due to disease burden (8). We would argue Figure 4 shows that BSR rapidly increased and its sustained elevation (>90%) within a 24-hour span correlated with the escalating sedation regimen. Fourth, the utilization of BSR to quantify "refractory ICP" and initiation of surgical intervention is not necessarily associated with improved functional outcome. However, this limitation really underlies the problem with the multifactorial etiologies that influence clinical outcome and has prevented any surgical decompression trial from demonstrating improved functional outcome (1,3,4). In fact, a more precise definition of "refractory ICP" as aided by subgaleal BSR may help future trials dissect out criteria for when surgical decompression should be offered.

CONCLUSION

We conclude the pathophysiology of BSR and its association with medically refractory ICP needs further study. Once BSR is further characterized, neurosurgeons may use BSR to better define medically "refractory ICP" enabling a better discussion of the risks and benefits of introducing salvage, surgical interventions.

Declarations

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Availability of data and materials: The datasets generated and/or analyzed during the current study are available from the corresponding author by reasonable request.

Disclosure: The authors declare no competing interests.

Ethical Considerations: The study protocol did not need any IRB approval.

AUTHORSHIP CONTRIBUTION

Study conception and design: AK, JC
Data collection: AK, JC
Analysis and interpretation of results: AK, JC
Draft manuscript preparation: AK, JC
Critical revision of the article: AK, DF, JM, JC
Other (study supervision, fundings, materials, etc...): JC
All authors (AK, DF, JM, JC) reviewed the results and approved the final version of the manuscript.

REFERENCES

1. Carney N, Totten A, O'Reilly C, Ullman J, Hawryluk G, Bell M, Bratton S, Chesnut R, Harris O, Kissoon N, Rubiano A, Shutter L, Tasker R, Vavilala M, Wilberger J, Wright D, Ghajar J: Guidelines for the management of severe traumatic brain injury, fourth edition. Neurosurgery 80:6-15, 2017. https://doi:10.1227/NEU.0000000000001432
2. Chemali J, Ching S, Purdon PL, Solt K, Brown EN: Burst suppression probability algorithms: State-space methods for tracking EEG burst suppression. J Neural Eng 10:056017, 2013. https://doi:10.1088/1741-2560/10/5/056017
3. Chesnut R, Temkin N, Carney N, Dikmen S, Rondina C, Videtta W, Petroni G, Lujan S, Pridgeon J, Barber J, Machamer J, Chaddock K, Celix J, Cherner M, Hendrix T: A trial of intracranial-pressure monitoring in traumatic brain injury. N Engl J Med 367:2471-2481, 2012. https://doi:10.1056/NEJMoa1207363
4. Cooper D, Rosenfeld J, Murray L, Arabi Y, Davies A, D'Urso P, Kossmann T, Ponsford J, Seppelt I, Reilly P, Wolfe R: Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med 364:1493-502, 2011. https://doi:10.1056/NEJMoa1102077
5. Foreman B, Claassen J: Quantitative EEG for the detection of brain ischemia. Crit Care 16:216, 2012. https://doi:10.1186/cc11230
6. Hawryluk G, Aguilera S, Buki A, Bulger E, Citerio G, Cooper D, Arrastia R, Diringer M, Figaji A, Gao G, Geocadin R, Ghajar J, Harris O, Hoffer A, Hutchinson P, Joseph M, Kitagawa R, Manley G, Mayer S, Menon D, Meyfroidt G, Michael D, Oddo M, Okonkwo D, Patel M, Robertson C, Rosenfeld J, Rubiano A, Sahuquillo J, Servadei F, Shutter L, Stein D, Stocchetti N, Taccone F, Timmons S, Tsai E, Ullman J, Vespa P, Videtta W, Wright D, Zammit C, Chesnut R: A management algorithm for patients with intracranial pressure monitoring: The seattle international severe traumatic brain injury consensus conference (SIBICC).Intensive Care Med 45:1783-1794, 2019. https://doi:10.1007/s00134-019-05805-9
7. Hofmeijer J, Kappelle LJ, Algra A, Amelink GJ, Gijn Jv, Worp HBvd: Surgical decompression for space-occupying cerebral infarction (the Hemicraniectomy After Middle Cerebral Artery infarction with Life-threatening Edema Trial [HAMLET]): A multicentre, open, randomised trial. Lancet Neurol 8:326-333, 2009. https://doi:10.1016/S1474-4422(09)70047-X

8. Hogan J, Sun H, Nour HA, Jing J, Tabaeizadeh M, Shoukat M, Javed F, Kassa S, Edhi M, Bordbar E, Gallagher J, Junior V, Ghanta M, Shao Y, Akeju O, Cole A, Rosenthal E, Zafar S, Westover M: Burst suppression: Causes and effects on mortality in critical illness. Neurocrit Care 33:565-574, 2020. https://doi:10.1007/s12028-020-00932-4
9. Hutchinson P, Kolias A, Timofeev I, Corteen E, Czosnyka M, Timothy J, Anderson I, Bulters D, Belli A, Eynon C, Wadley J, Mendelow A, Mitchell P, Wilson M, Critchley G, Sahuquillo J, Unterberg A, Servadei F, Teasdale G, Pickard J, Menon D, Murray G, Kirkpatrick P: Trial of decompressive craniectomy for traumatic intracranial hypertension. N Engl J Med 375:1119-1130, 2016. https://doi:10.1056/NEJMoa1605215
10. Lazaridis C, DeSantis S, Smielewski P, Menon D, Hutchinson P, Pickard J, Czosnyka M: Patient-specific thresholds of intracranial pressure in severe traumatic brain injury. J Neurosurg 120:893-900, 2014. https://doi:10.3171/2014.1.JNS131292
11. Mendelow AD, Gregson BA, Rowan EN, Murray GD, Gholkar A, Mitchell PM, STICH II Investigators: Early surgery versus initial conservative treatment in patients with spontaneous supratentorial lobar intracerebral haematomas (STICH II): A randomised trial. Lancet 382:397-408, 2013. https://doi:10.1016/S0140-6736(13)60986-1
12. Okonkwo D, Shutter L, Moore C, Temkin N, Puccio A, Madden C, Andaluz N, Chesnut R, Bullock M, Grant G, McGregor J, Weaver M, Jallo J, LeRoux P, Moberg D, Barber J, Lazaridis C, Diaz-Arrastia R: Brain oxygen optimization in severe traumatic brain injury phase-II: A phase II randomized trial. Critical care medicine. Crit Care Med 45:1907-1914, 2017. https://doi:10.1097/CCM.0000000000002619