doi: 10.17116/engneiro201579680-85

Prx-Monitoring Based Decision-Making About Decompressive Craniectomy in a Patient with Severe Traumatic Brain Injury. A Case Report

A.V. OSHOROV, K.A. POPUGAEV, I.A. SAVIN, A.G. GAVRILOV, A.D. KRAVCHUK, A.A. POTAPOV

Burdenko Neurosurgical Institute, Moscow, Russia

The presented case illustrates a new approach to making a decision about decompressive craniectomy in patients with severe traumatic brain injury and intracranial hypertension. The approach is based on continuous assessment of cerebral autoregulation using Prx-monitoring in addition to monitoring of intracranial pressure and cerebral perfusion pressure. Prx-monitoring enables timely detection of autoregulation failure and provides the opportunity to make a decision about decompressive craniectomy before starting such aggressive methods of intensive care as hypothermia or barbiturate coma.

Keywords: *intracranial hypertension, extended neuromonitoring, Prx-monitoring, decompressive craniectomy.*

Intracranial hypertension remains to be a leading cause of high mortality and disability among patients with severe traumatic brain injury (TBI) [1–4, 6]. Its incidence among comatose TBI patients ranges from 40 to 60% [6–8]. Neurosurgical techniques occupy a special place in the arsenal of methods of correction of intracranial hypertension. They include evacuation of intracranial hematomas, external ventricular drainage, and decompressive craniectomy [1–3, 6]. Along with elimination of secondary brain damage factors, timely diagnosis and prompt evacuation of pathological intracranial masses are important components of TBI patients' treatment. Brain imaging techniques, such as CT and MRI, are key diagnostic tools for neurosurgical problems [3—5]. Indications for neurosurgical intervention include assessment of volumetric criteria and their dynamics: volume of pathological substrate, degree of the basal cisterns compression and displacement of the midline brain structures, etc. [1, 4, 5]. The main mechanism of decompression is the creation of additional space to compensate for the increased intracranial volume [6—8, 11]. The effectiveness of decompression is believed to depend on the size of decompression window [9–11]. Intracranial pressure (ICP) monitoring is an additional factor in deciding whether to perform decompressive craniectomy. ICP above 20 mmHg that cannot be relieved by intensive therapy (sedation, anesthesia, hyperventilation, hyperosmolar solutions) requires more intense conservative therapy and/or decompressive craniectomy. ICP parameters, such as average value and duration of increase above 20 mmHg, were used in the two most recent randomized studies featuring decompressive craniectomy [12, 13]. These studies had different designs and inclusion criteria and used different limits for value and duration of intracranial hypertension to decide on decompression. These studies arbitrary divided the timing of decompression procedures into early secondary or neuroprotective and deferred secondary, in which the neurosurgical intervention was considered as the last stage of intracranial hypertension correction [14].

In our clinical case, we used pathophysiological approach to addressing the issue of decompression based on enhanced monitoring of ICP, cerebral perfusion pressure (CPP) and Prx autoregulation ratio [15]. The patient belonged to a group of TBI patients for whom the decompression was performed due to decompensation of cerebral vessels autoregulation and the development of intracranial hypertension [15]. The presence of decompensation and the lack of further options for conservative therapy were evaluated based on the dynamics of Prx ratio above 0.2. The autoregulation of cerebral vessels was assessed using Prx correlation ratio. Prx was calculated using the ICM Plus software (Cambridge, UK) and represents the correlation between the ICP and ABP. The Prx was calculated automatically as a moving average of 40 consecutive ICP and BP values, using 5-second window. Prx values of 1 to 0.2 were indicative of intact or partially intact autoregulation. Prx values of 0.2 to 1 were considered to be signs of complete autoregulation failure.

Clinical case

The first 24 h after the injury. The patient was a man (39 years) who sustained an injury in a fall from an ATV. He lost consciousness at the site of the accident and was transferred to the city hospital. At the time of admission he was in stupor (9 points on the Glasgow Coma Scale, GCS) with episodes of psychomotor agitation. Oval pupils along the midline, weak pupillary light reflex in both eyes. Left-sided hemiparesis. CT revealed bilateral lesions in the frontal lobes and in the left temporal lobe and convexital subarachnoid hemorrhage (SAH). Moderate swelling at the expense of the left hemisphere, visible cisterna ambiens. The ventricular system and subarachnoid space were narrowed. Tracheal intubation and artificial lung ventilation (ALV) due to increasing respiratory failure and the need for sedation. BP 130/80 mmHg, HR 108 bpm. The patient was ventilated and administered sedatives, decongestants, and antibiotics.

Day 2. The patient was transferred to the Burdenko Neurosurgical Institute. Diagnosis: a severe open TBI. Severe brain contusion with multiple foci of lesions (33 types) in the frontal lobes on both sides and in the left temporal lobe. Linear fracture of the occipital bone on the left with the transition to the base. Traumatic SAH. CT examination produced the same pattern **(Fig. 1)**.

© Group of authors, 2015

e-mail: agvan@nsi.ru

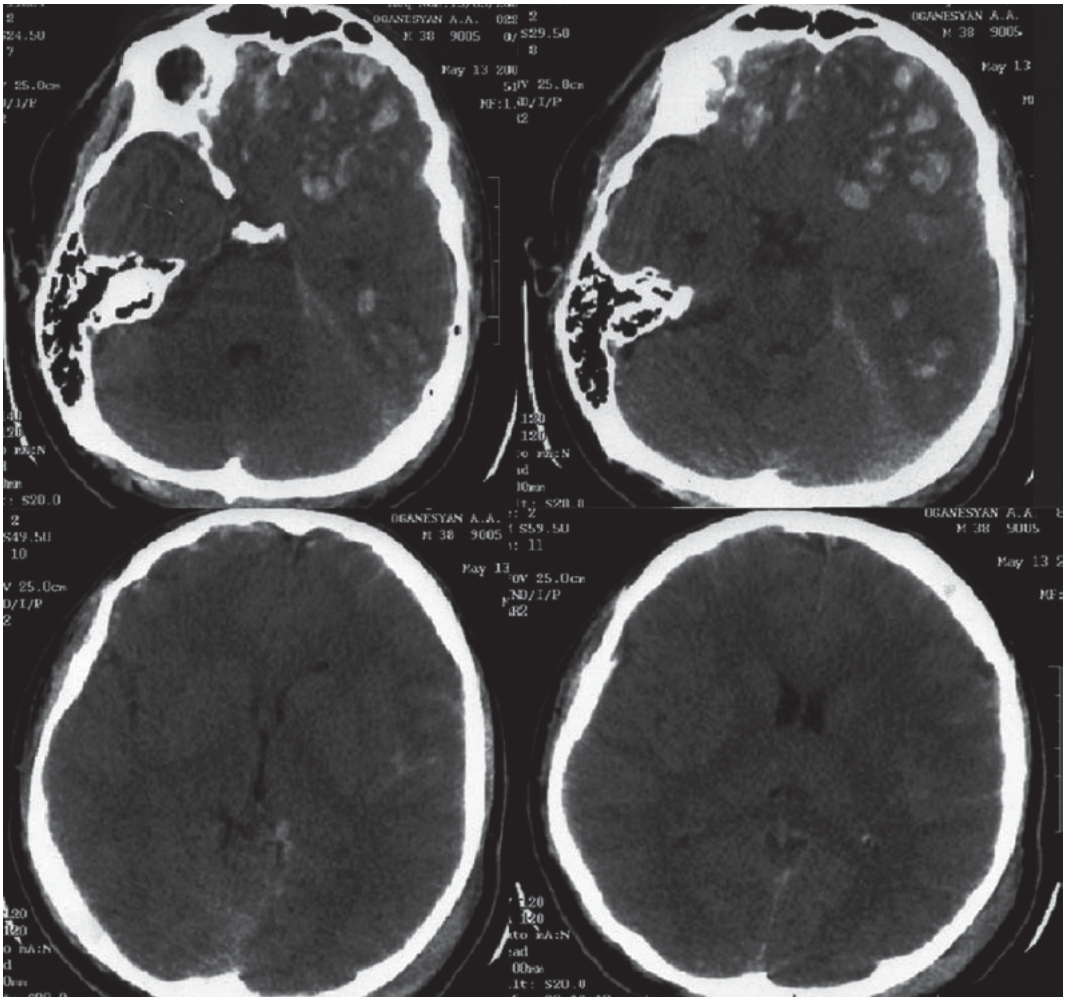


Fig. 1. **CT data on days 2–3 after the injury.**

Neurological examination: 9 points on GCS, persistent probing resulted in a hand grasp. Brisk pupillary light reflex, presence of corneal reflex, reflexive upward gaze in response to irritation. The patient was able to hold his lower jaw, brisk cough reflex. Persistent motor anxiety, stereotypical movements. BP in the range of 120—130/60—70 mmHg, HR 80—90 bpm. Mechanical ventilation in SIMV mode, RR 10, Vt 0.8 L, PS 14 cm H2O, PEEP 6 cmH2O, FiO2 40%. Taking into account the data of CT examination, acute phase of severe TBI and the need for sedation, it was decided to monitor ICP. Parenchymal sensor revealed ICP of 25—29 mmHg, and ICP amplitude above 7 mmHg. The patient was on mechanical ventilation in BTPS mode, intracranial hypertension was corrected and CPP was maintained above 60 mmHg. He was administered 100 μg/h of fentanyl and 150 mg/h of propofol. was Vasopressor support to control CPP included 1 mg/kg of body weight of mannitol, which was administered if ICP rose above 20 mmHg.

Day 3. Examination 30 minutes after turning off the sedation and narcotic analgesics revealed markedly negative dynamics: rough oral-stem syndrome which manifested as Magendie sign, oval pupils, limited reflexive upward gaze, hypersalivation. Swallowing movements and cough reflex sharply depressed. Low muscle tone in the limbs. Neurological examination was discontinued due to episodes of ICP elevation to 30—35 mmHg, which was alleviated by hyperventilation, reinitiation of sedation and analgesia in the previous doses, and additional infusion of mannitol (15%, 400 ml). It was decided to forgo the awakening test in the future. Osmolarity and sodium levels in the plasma were normal. Ventilation, SIMV, RR 14, Vt 0.78 L, PS 14 cm H2O, PEEP 6 cm H2O, FiO2 40%. Et CO2 30 mmHg. Low-grade fever up to 37.7—38 °C throughout the day. Controlled normothermia (36.5—37 °C) was performed using TropyCool external cooling system. Hemodynamics was maintained by vasopressors (phenylephrine 8—10 mg/h). BP 135—165/70—90 mmHg. According to the neuromonitoring **(Fig. 2)**, ICP 18—35 mmHg, CPP 60—95 mmHg, Prx 0.5—0.1.

CT was not repeated due to unstable hemodynamics and intracranial hypertension, which was provoked by any manipulations and changes in the patient's position. Tracheostomy was postponed until stabilization of the patient. Conservative therapy with neuromonitoring.

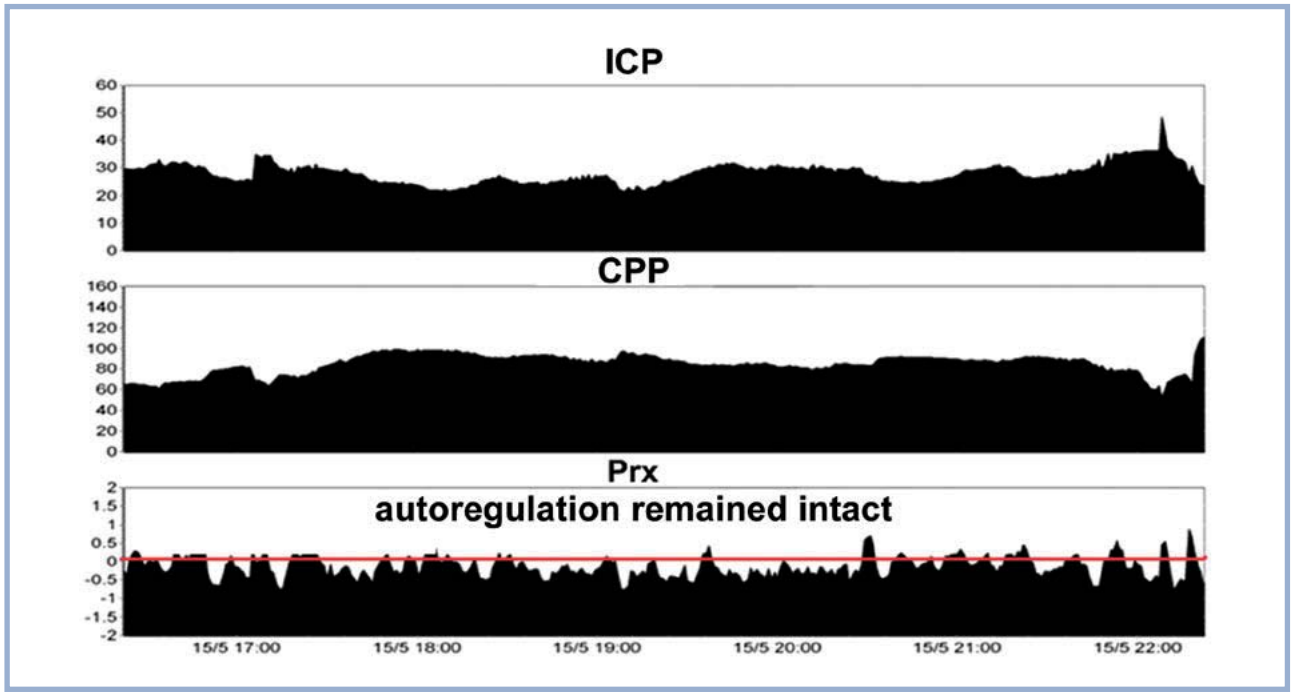


Fig. 2. **Extended neuromonitoring data on day 3 after the injury.**

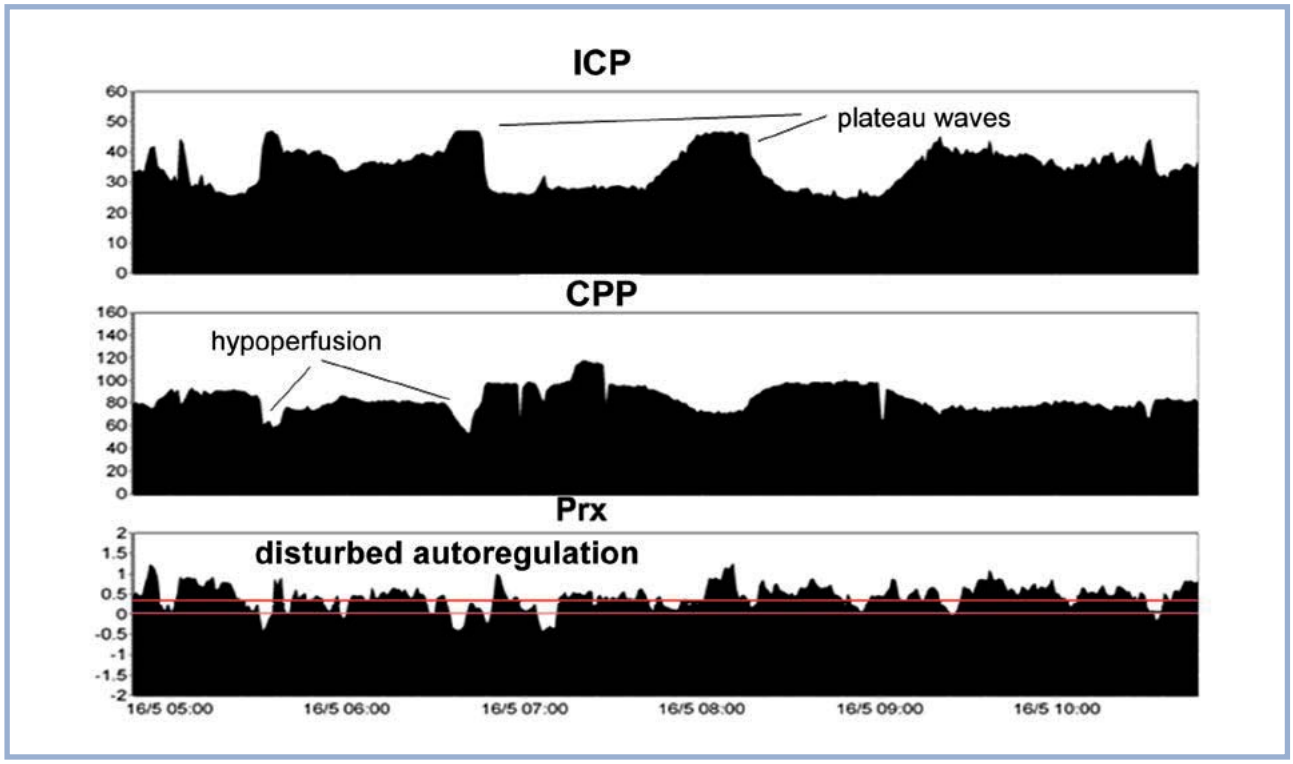


Рис 3. **Extended neuromonitoring data on day 4 after the injury.**

Day 4. Repeated episodes of intracranial hypertension involving a plateau of ICP waveforms up to 40 mmHg, a short-term CPP drop below 50 mmHg during the plateau. Dynamic monitoring revealed a steady trend towards an increase in Prx above 0.2, indicating a failure of cerebral autoregulation **(Fig. 3)**.

Sedation was deepened: propofol 250—300 mg/h, fentanyl in the previous dose, deep hyperventilation to EtCO2 of 28 mmHg under the control of jugular oximetry (SvjO2 no lower than 50%). Sodium 145—149 mmol/l. Osmolarity 310 mOsm/l. The 15% mannitol solution was used four times during the day (1600 ml in total). A decision to perform decompressive trepanation was taken after a joint discussion with neurosurgeons. Surgery: decompression craniectomy of the fronto-parietal-temporal area on both sides. Removal of subdural hy-

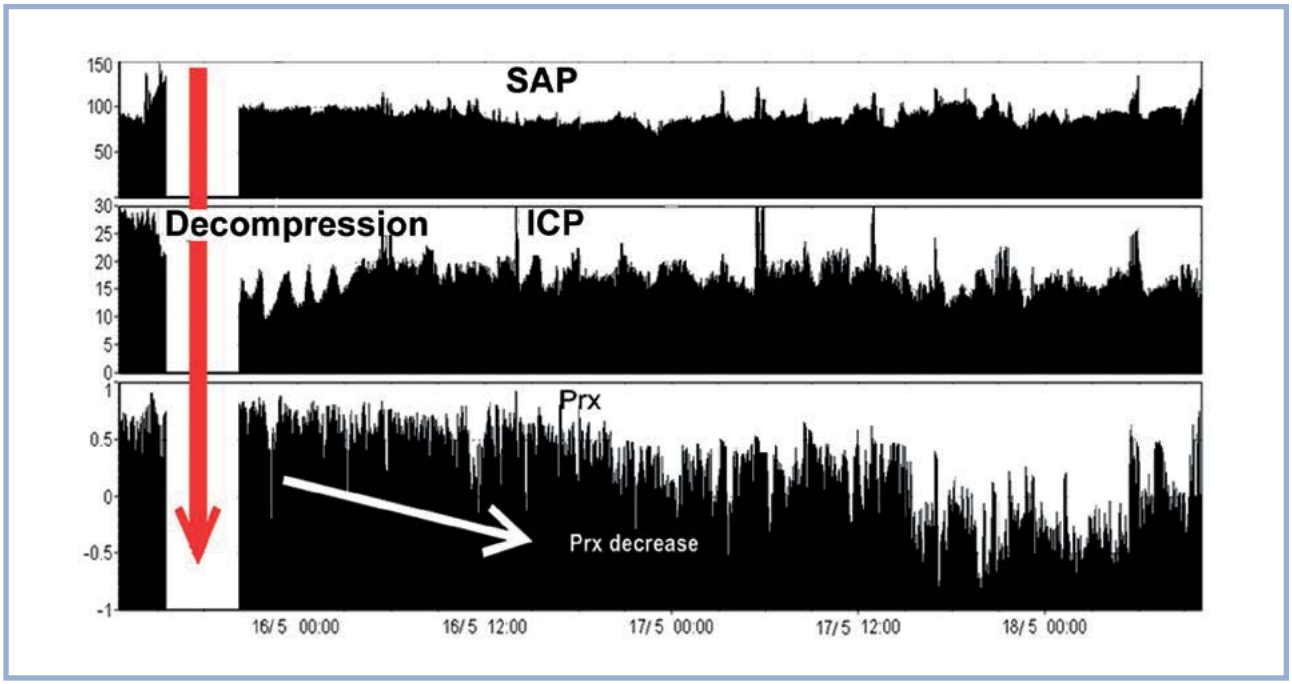


Fig. 4. **Extended neuromonitoring data after the decompression.**

groma on the left (30 ml). Dura mater was repaired with periosteal flap on the left.

Days 5—6. Stabilization of ICP at 5–12 mmHg, CPP at 60—65 mmHg, and Prx >0.2 was observed in the postoperative period **(Fig. 4)**.

Propofol dose was reduced to 80 mg/h, fentanyl was discontinued, phenylephrine dose was reduced to 5 mg/h. Controlled normothermia was discontinued, low-grade fever of 37.8 °C. Examination without sedation: 6 points on GCS, right arm adduction with flexion, without a distinct localization, slight right leg adduction. Passive opening of the eyelids revealed eyeballs fixed in the central position, oval pupils, weak pupillary light reflex, weak corneal reflex on both sides, rare blinking movements. The lower jaw droops, excessive salivation from the mouth, tongue protrudes beyond the line of incisors, edematous. Presence of lower jaw reflex. BP 120—145/85—80 mmHg, HR 65–90 bpm, sinus rhythm. Ventilation, SIMV RR 14, Vt 0.75 L, PS 12 cmH2O, PEEP 7 cmH2O, FiO2 40%. Symmetrical bilateral lung breathing, rigid, slight bilateral weakening in posterior basal section. Increase in inflammatory markers in the blood: leukocytosis with left deviation, CRP 178 mg/l. X-ray: bilateral pneumonia. Antibiotic therapy, taking into account the sensitivity of the flora. Transcutaneous dilated tracheostomy.

Days 7—11. Stabilization of hemodynamics, discontinuation of vasopression and sedation. Low-grade fever persisted. BP 120—130/60—70 mmHg, HR 80—90 bpm. ICP 5—7 mmHg, CPP 60—65 mmHg, Prx values 0 to –0,1, indicating recovery of autoregulation. Neuromonitoring was stopped on Day 11.

Day 12—20. Regression of infectious and inflammatory manifestations, normalization of body temperature, resolution of bilateral pneumonia. Positive dynamics in neurological signs: 10 points on GCS, slight right-sided hemiparesis. The patient was able to follow certain instructions: shake hands on demand, actively gesticulate. Stable hemodynamics, respiratory rehabilitation, transferred to the CPAP 5 cm H_2O, FiO_2=25%, PS 10cm H_2O.

Day 30. The patient regained consciousness and was disconnected from the ventilator. Stable hemodynamics. The patient successfully underwent the repairmen of dura mater in the left fronto-parietal-temporal region 22 months later due to appearance of wound liquor. Later, ventriculo-peritoneal shunt was installed on the right due to development of hydrocephalus. The patient was discharged from the hospital after 5 months. Neurological status: slight right-sided hemiparesis, sensory-motor aphasia. Swallowing movement was restored and the cannula was removed. The patient subsequently underwent rehabilitation and recovery courses up to 2 times a year. After 12 months, the outcome was assessed as moderate disability based on the Glasgow Outcome Scale.

Discussion

The presented clinical example demonstrates that in addition to the traditional of ICP and CPP monitoring, the monitoring of cerebral vessels autoregulation enable timely decision to perform decompressive craniectomy. In the presented clinical case, the persistent failure of autoregulation on Day 4 demonstrated futility of further pharmacological control of mean arterial blood pressure and therefore inability to ensure CPP above 60 mmHg. Taking this into account, any attempt to improve the ABP would lead to passive increase in ICP and further reduction in CPP. Since there was still an option of using hyperosmolar solutions, and, moreover, ICP was successfully reduced by deepening sedation and safe deepening of hyperventilation controlled by jugular oximetry, we can only assume that in the absence of autoregulation monitoring, the intensive care would have proceeded along the lines of further increase in aggressiveness. However, in the given clinical situation, osmotic diuretics could not be considered as a mean of prolonged intra-

cranial hypertension correction, since there was a tendency towards hypernatremia, and mannitol had already been administered in close to maximum doses. Other options to intensify intracranial hypertension treatment were hypothermia and barbituric coma. Barbiturates can effectively remove intracranial hypertension; however, the number of adverse events, caused by their use, outweighs their positive effect on ICP [16, 17]. One of the serious complications is hypotension, which is a powerful disturbing factor for the injured brain and significantly worsens outcomes in TBI [16, 17]. It can be assumed that the use of barbiturates in the patient would have required higher doses of vasopressors and aggressive fluid therapy, which in itself could have lead to various complications: pulmonary edema, renal failure, heart failure, cardio-vascular events, etc [18, 19]. Today, barbiturates are regarded as the last stage of aggressive intracranial hypertension therapy, and their use does not improve outcomes in TBI [1, 4, 6, 7, 16, 17].

Hypothermia has proven to be an effective method of correction of intracranial hypertension. However, international multi-center studies have not demonstrated improved outcomes with the use of hypothermia in comparison with traditional methods of intensive care [18]. It was noted, however, that the number of adverse events, associated with the use of hypothermia, is as high as that for barbiturates [19, 20]. Decompressive craniectomy is not a panacea, either, but in our clinical case it demonstrated its effectiveness in correction of intracranial hypertension and CPP stabilization. In addition, decompression made it possible to reduce the aggressiveness of intensive therapy by reducing the dosages of sedatives and catecholamines. We were able to discontinue narcotic analgesics, stop hyperventilation, discontinue administration of hyperosmolar solutions, and achieve normalization of sodium levels and recovery of autoregulation.

Therefore, we have demonstrated the possibility of using a new approach to making the decision to perform decompressive craniectomy in patients with traumatic brain edema and intracranial hypertension. The essence of the new approach is continuous assessment of cerebral vessels autoregulation using Prx values, in addition to such important neuromonitoring parameters as ICP and CPP. Prx-monitoring enables timely recognition of the cerebral vessels autoregulation failure and allows making the decision to perform decompressive craniectomy prior to escalating aggressive methods of intensive care (barbituric coma and hypothermia).

Authors declare no conflict of interest.

REFERENCES

1. Potapov A, Likhterman L, eds. *Dokazatel'naya neirotravmatologiya*. M.: Antidor; 2003:517. (In Russ.).
2. Konovalov A, Likhterman L, Potapov A, eds. *Klinicheskoe rukovodstvo po cherepno-mozgovoi travme*. M.: Antidor; 1998. (In Russ.).
3. Potapov A, Krylov V, Likhterman L, Tsarenko S, Gavrilov A, Petrikov S. Current guidelines for the diagnosis and treatment of severe brain injury. *Voprosy neirokhirurgii im. N.N. Burdenko*. 2006;1:3-8. (In Russ.).
4. Potapov A, Zakharova N, Pronin I, Kornienko V, Gavrilov A, Kravchuk A, Oshorov A, Sychev A, Zaitsev O, Fadeeva L, Takush S. Prognostic value of ICP, CPP and regional blood flow monitoring in diffuse and focal traumatic cerebral lesions. *Voprosy neirokhirurgii im. N.N. Burdenko*. 2011;3:3-18; (In Russ.).
5. Zakharova N, Kornienko V, Potapov A, Pronin I. Neuroimaging of Traumatic Brain Injury. *Springer International Publishing*. Switzerland 2014.

   doi: 10.1007/978-3-319-04355-5.
6. Maas AI, Dearden M, Teasdale GM, Braakman R, Cohadon F, Iannotti F, Karimi A, Lapierre F, Murray G, Ohman J, Persson L, Servadei F, Stocchetti N, Unterberg A. EBIC-guidelines for management of severe head injury in adults. European Brain Injury Consortium. *Acta Neurochir (Wien)*. 1997;139(4):286-2–94. PubMed PMID:9202767.
7. Stocchetti N, Zanaboni C, Colombo A, Citerio G, Beretta L, Ghisoni L, Zanier ER, Canavesi K. Refractory intracranial hypertension and ««second-tier»» therapies in traumatic brain injury. *Intensive Care Med*. 2008 Mar;34(3):461-467. Epub 2007 Dec 8. PubMed PMID: 18066523.

   doi:10.1007/s00134-007-0948-9.
8. Miller JD, Becker DP, Ward JD, Sullivan HG, Adams WE, Rosner MJ. Significance of intracranial hypertension in severe head injury. *J Neurosurg*. 1977 Oct;47(4):503-516.

   doi:10.3171/jns.1977.47.4.0503.
9. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, Servadei F, Walters BC, Wilberger JE. Surgical Management of Traumatic Brain Injury Author Group. Surgical management of acute epidural hematomas. *Neurosurgery*. 2006Mar;58(3)Suppl:1-60; discussion Si-iv. Review.

   doi:10.1097/00006123-200603001-00006.
10. Menon DK, Matta BF. Intensive care after acute head injury. In: Matta B, Menon D, Turner J, eds. *Neuroanesthesia and Neurointensive Care*. London: Greenwich Medical Media. 2000;301-317.

    doi:10.1016/b978-141604653-0.10034-2.
11. Munch E, Horn P, Schurer L, Piepgas A, Torsten P, Schmidek P. Management of severe traumatic brain injury by decompressive craniectomy. *Neurosurgery*. 2000;47:315-323.

    doi:10.1097/00006123-200008000-00009.
12. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, Kossmann T, Ponsford J, Seppelt I, Reilly P, Wolfe R. DECRA Trial Investigators, Australian and New Zealand Intensive Care Society Clinical Trials Group (2011) Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med*. 2011;364:1493-1502.

    doi:10.1056/nejmoa1102077.
13. Hutchinson PJ, Corteen E, Czosnyka M, Mendelow AD, Menon DK, Mitchell P, Murray G, Pickard JD, Rickels E, Sahuquillo J, Servadei F, Teasdale GM, Timofeev I, Unterberg A, Kirkpatrick PJ. Decompressive craniectomy in traumatic brain injury: the randomized multicenter RESCUEicp study (www.RESCUEicp.com). *Acta Neurochir Suppl*. 2006;96:17-20.

    doi:10.1007/3-211-30714-1_4.
14. Kolias AG, Li LM, Guilfoyle MR, Timofeev I, Corteen EA, Pickard JD, Kirkpatrick PJ, Menon DK, Hutchinson PJ. Decompressive craniectomy for acute subdural hematomas: time for a randomized trial. *Acta Neurochir (Wien)*. 2013 Jan;155(1):187-188.

    doi:10.1007/s00701-012-1531-x.
15. Oshorov V, Savin I, Goryachev A, Popugayev K, Potapov A, Gavrilov A. The first experience in monitoring the cerebral vascular autoregulation in the acute period of severe brain injurya. *Anaesthesiology and Reanimatology*. 2008; 2:61-64.

    doi:10.14412/1995-4484-2008-8.
16. Cormio M, Gopinath SP, Valadka A, Robertson CS. Cerebral hemodynamic effects of pentobarbital coma in head-injured patients. *J Neurotrauma*. 1999 Oct;16(10):927-936.

    doi:10.1089/neu.1999.16.927.
17. Roberts I, Sydenham E. Barbiturates for acute traumatic brain injury. *Cochrane Database Syst Rev*. 2012 Dec 12;12:CD000033.

    doi: 10.1002/14651858.CD000033.pub2.
18. Robertson CS, Valadka AB, Hannay HJ, Contant CF, Gopinath SP, Cormio M, Uzura M, Grossman RG. Prevention of secondary ischemic insults after severe head injury. *Crit Care Med*. 1999 Oct;27(10):2086-2095.

    doi: 10.1097/00003246-199910000-00002.

19. Sydenham E, Roberts I, Alderson P. Hypothermia for traumatic head injury. *Cochrane Database Syst Rev*. 2009 Apr 15;(2):CD001048.
doi: 10.1002/14651858.CD001048.pub4. Review. PubMed PMID: 19370561.

20. Polderman KH. Mechanisms of action, physiological effects, and complications of hypothermia. *Crit Care Med*. 2009 Jul;37(7 Suppl):186-202.
doi:10.1097/CCM.0b013e3181aa5241. Review.

Commentary

The clinical case described by the group of authors presents new opportunities for neuromonitoring of patients with severe TBI. Modern software make it possible to continuously monitor the status of cerebral blood flow autoregulation using Prx values (pressure reactivity index), in addition to traditional monitoring of BP, ICP, and CPP [1].

The papers published in the last 5––7 years confirm that this neuromonitoring parameter is one of the leading and most actively used ones in decision-making about intensive care strategy in patients with acute cerebral injury. The interest in monitoring of cerebral blood flow autoregulation has significantly increased for several reasons. First of all, the method based on computer analysis of slow-wave fluctuations in BP and ICP is both widely available and relatively. This fact ensured wide spread of the method of autoregulation assessment in neurointensive therapy. Secondly, the importance of autoregulation monitoring has been highlighted in the International Recommendations for the management of TBI patients [2]. Thirdly, there was a need to tailor intense therapy to individual patients based on multiparameter neuromonitoring, taking into account the severity of the injury and the time that has passed since cerebral injury, age of the patient, concomitant somatic pathology, secondary brain injury factors etc. [3, 4].

Timely provision of surgical assistance, no doubt, remains the leading factor, defining the outcome in TBI patients. Decompressive craniectomy is a method of intracranial hypertension correction for this category of patients, especially in case of development of intracranial hypertension. Unfortunately, the timing of decompressive craniectomy is not defined. The use of variety of strategies, early "neuroprotective" or deferred decompression, did not demonstrate convincing benefits and did not produce the expected improvement in TBI outcomes.

The present work demonstrates a completely new approach that can be called pathophysiological and that is based on the assessment of autoregulation. According to the authors, the failure of cerebral blood flow autoregulation is an indication for decompressive craniectomy. It seems quite reasonable, since in the absence of autoregulation it becomes very challenging to maintain CPP against the backdrop of intracranial hypertension. By using this clinical case as an example, the authors were able to demonstrate clinical significance of using Prx in patients with severe traumatic brain injury. The results are clearly presented and thoughtfully discussed in the context of recent publications on this topic.

It seems reasonable to further study Prx autoregulation ratio and implement it into the complex of neuromonitoring measures in the most severe category of critically injured patients and to use it as a reference point for targeted therapy.

M.Yu. Kirov (Arkhangelsk, Russia)

REFERENCES

1. Czosnyka M, Miller C. Monitoring of cerebral autoregulation. *Neurocrit Care*. 2014 Dec;21 Suppl 2:95-102.
2. `Guidelines for the management of severe traumatic brain injury. IX. Cerebral perfusion thresholds. *J Neurotrauma*. 2007;24:Suppl 1:59-64.
3. Saatman KE, Duhaime AC, Bullock R, et al. Workshop Scientific Team and Advisory Panel Members. Classification of traumatic brain injury for targeted therapies. *J Neurotrauma*. 2008 Jul;25(7):719-738.
4. Le Roux P, Menon DK, Citerio G, et al. The international multidisciplinary consensus conference on multimodality monitoring in neurocritical care: evidentiary tables: a statement for healthcare professionals from the neurocritical care society and the European society of intensive care medicine. *Neurocrit Care*. 2014 Dec;21:Suppl 2:297-361.