Management of Pediatric Severe Traumatic Brain Injury: What are News?

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Abstract

The management of pediatric traumatic brain injury at prehospital and hospital stages is presented. The possibilities of monitoring, intensive treatment and surgical treatment are determined. The recommendations are given as standards, options and recommendations.

Keywords: Pediatrics; Severe Traumatic Brain Injury; Monitoring; Intensive Treatment

Introduction

Traumatic brain injury (TBI) is more common in children than in adults [1,2]. Among hospitalized children with neurotrauma, the proportion of severe TBI is 6% [2]. According to the literature, mortality among children with severe TBI ranges from 12 to 30% [2,9,20].

It is known that universal pathophysiological stress reactions underlie the body's response to acute cerebral injury. However, anatomical and physiological characteristics of children of different age groups play a significant role in their implementation: high intensity of metabolic processes; low tolerance to blood loss, hypoxia and hypotension; a tendency to swelling and swelling of the brain; low tolerance to hyperthermia; tendency to generalization (convulsive syndrome, protective-adaptive reaction in the form of switching off consciousness to the level of coma); high compensatory capabilities with subsequent sudden and rapid decompensation of functions. To a large extent, the listed anatomical and physiological features in children are characteristic of the younger age group. In predicting the outcomes of severe TBI, it is necessary to take into account the high plasticity of the child's brain.

Pre-hospital stage

The prehospital stage for injured children with severe TBI is one of the most important, determined by the time factor and the optimal amount of care provided. The main goal at the prehospital stage is to assess the severity of injury and the severity of the general condition; for children with severe TBI (GCS 3-8 points), maintenance of vital functions and prevention of secondary brain damage. If necessary, resuscitation measures should be as close as possible to the place of injury [2,14,15,19]. It is necessary to organize the fastest possible delivery of the victim to a specialized hospital with appropriate diagnostic and treatment capabilities [9,19].

Initial examination of the patient

Assessing the severity of brain damage For pediatric patients, taking into account age characteristics, the Glasgow Pediatric Coma Scale (GCS) is used (Appendix 1).

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At the prehospital stage, especially in patients in a coma, it is important to evaluate brainstem pupillary reflexes (pathology: asymmetry of the pupils (anisocoria), fixed bilateral pupil dilation (mydriasis), lack of response to bright light). Depression of the level of wakefulness less than 9 points on the GCS, symmetric or asymmetric increase in the diameter of the pupils (anisocoria) with impaired response to light, symmetric or asymmetric increase in muscle tone up to decerebrational rigidity, instability of blood pressure, bradycardia, indicate a severe and extremely serious condition of the victim and the development of dislocation syndrome.

Severe TBI includes victims with a consciousness level of 3 - 8 points.

Patients with a Glasgow coma score of 9 to 12 are considered moderate TBI patients.

Patients with a level of wakefulness of 13 - 15 points have mild TBI.

Mild TBI 13 - 15 points corresponds to a concussion and mild brain contusion. Moderate (9 - 12 points) includes moderate brain contusion, subacute and chronic cerebral compression. Severe TBI (3 - 8 points) includes severe brain contusion, diffuse axonal injury, and acute brain compression [3,13].

Assessment of the general condition of the patient includes an assessment of the patency of the airways and the nature of breathing (free, obstructed, superficial, pathological, rhythmic, presence or absence of pneumo-hemothorax), the state of the cardiovascular system (the nature of the pulse on the carotid and radial arteries (in the absence of pulse on radiation and its presence on carotid systolic pressure within 50 - 70 mm Hg), heart sounds, heart rate, blood pressure), assessment of the level of wakefulness.

Moving the victim into a vehicle

Moving the victim onto a stretcher and into a medical transport is carried out with stable fixation of the cervical spine. It is preferable to use a vacuum mattress to fix the entire musculoskeletal system. In the absence of a vacuum mattress, a fixing cervical collar and splints immobilizing the injured limb are applied (in case of combined injury). In patients with concomitant trauma, movement into a vehicle is performed after preliminary anesthesia. Opioid analgesics are administered (in case of shock injury - promedol 1% solution or fentanyl 0.005% solution in an age-specific dosage) [6,11,12]. Foreign bodies and bone fragments from the cranial cavity in the case of a penetrating wound are not removed. As early as possible, carry out monitoring of blood pressure systole, Diastole, Average, Heart rate, RR, pulse oximetry. Contraindication to moving into the car is respiratory arrest and cardiac arrest, life-threatening external bleeding. Condition monitoring continues during transport. Along with the assessment of the cardiovascular and respiratory systems, special attention is paid to the dynamics of impaired consciousness, the state of the pupils, the presence and progression of movement disorders (paresis and paralysis) and muscle tone to identify a possible increase in dislocation syndrome (Appendix 1).

Restoration of oxygenation at the prehospital stage

Tracheal intubation is indicated for victims with impaired consciousness to the level of coma I (less than 9 points on the GCS) [3,6,7,13,19]. Tracheal intubation is performed after the introduction of atropine 0.01% solution at the rate of 0.1 ml/year of the child's life, the opioid analgesic fentanyl 2 μg/kg and pretreatment of the mucous membranes of the oropharynx and the endotracheal tube with a local anesthetic solution (10% lidocaine solution in the form of a spray) in conditions of a fixed cervical spine. It is advisable to use sedatives (benzodiazepines, propofol, or barbiturates) and, if necessary, non-depolarizing relaxants. If intubation is not possible, corticotomy is performed. Airway patency is ensured. If necessary, the mouth/nasopharynx is sanitized, and an air duct is installed. If there are signs of respiratory failure against the background of spontaneous breathing, breathing through an oxygen mask is provided. Adequacy is assessed by clinical data and monitoring indicators. In the presence of a tense pneumo-hemothorax, a puncture of the pleural cavity is performed. Ventilation is carried out with a respiratory rate of 20 per 1 min for children over 1 year old and 30 per 1 min for children under 1 year of age. The adequacy of mechanical ventilation is assessed by clinical data and indicators of saturation monitoring

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and capnometry.

Correction of arterial hypotension at the prehospital stage

Hypotension should be diagnosed and eliminated as soon as possible by restoring the volume of the circulating fluid [3,6,11-15]. Sympathomimetics are used in conditions of normovolemia. In the presence of hypotension, it is necessary to assess the presence of extracranial damage (exclude shockogenic injury).

In children, hypotension is defined as systolic blood pressure below the fifth percentile of the age norm. The lower limit of the systolic BP (fifth percentile) of the age norm can be calculated using the formula: 70 mm Hg + (2 X age in years). Venous access is performed as early as possible. If peripheral veins are inaccessible, intraosseous access to the vascular bed is used. Bradyarrhythmia, cardiac arrest is the basis for the beginning of cardiac resuscitation. A method of artificially maintaining blood circulation is a closed heart massage. The main drug used in cardiac resuscitation is adrenaline. In the absence of access to the vascular bed, endotracheal administration of drugs or once into the root of the tongue is possible.

Medication

Infusion therapy is carried out: starting solution - 6% HES (Voluven, Volulyt), Gelofusin at a dose of not more than 15 ml/kg. The rate of administration is 60 - 120 drops per minute under the control of blood pressure and heart rate. With the development or increase of hemodynamic instability with a decrease in blood pressure against the background of ongoing infusion therapy, adrenergic agonists are used in parallel (dopamine 4% 3 - 5 mcg/kg/min, if necessary, the dose is increased to 10 - 15 mcg/kgmin; and if ineffective, in a combination of adrenaline or mezaton in an age-related dose [6,7] Glucocorticoid hormones - prednisolone 5 mg/kg body weight is administered intravenously in case of shock injury.

Transportation conditions

Hardware monitoring of vital functions: mechanical ventilation under the control of pulse oximetry and capnometry; infusion therapy aimed at maintaining systolic blood pressure at the upper limits of the age norm.

Sedation and relaxation of the victim is carried out with short-acting and ultra-short-acting benzodiazepines: diazepam, midazolam in an age-specific dosage.

The head end of the stretcher is raised by 30° (in the absence of arterial hypotension). The development/growth of dislocation symptoms is monitored.

It is not recommended to administer mannitol for the prevention of edema and dislocation of the brain. The introduction of mannitol is allowed in the presence of clinical signs of the development of dislocation syndrome at a dose of 0.25 g/kg of body weight of the victim [3,6,7,11,12,20].

Normoventilation is the best option (etCO$_2$ = 37 - 39 mm Hg) for mechanical ventilation in conditions of the development of dislocation syndrome in the absence of hypotension or hypoxemia. Moderate hyperventilation (etCO$_2$ = 32 - 36 mm Hg) is permissible only in the absence of clinical signs of the development of dislocation syndrome. It is also not recommended to carry out mechanical ventilation in the hyperventilation mode (etCO$_2$ < 30 mm Hg).

Hospital stage

Children with severe TBI should be treated in pediatric specialized multidisciplinary hospitals, or in the absence of such in an adult

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multidisciplinary hospital, which has the necessary equipment and medical personnel trained to provide emergency specialized care for children (pediatric service) [7,12,13].

Transportation/transfer to a specialized hospital is recommended [7,12,13]:

- GCS < 14 points.

- Open head injury.

- Fall from a height of more than 2 meters.

- High-energy mechanism, injury.

- Long-term evacuation of the victim (more than 20 minutes) from rubble, cars, etc.

- Age < 5 years.

Affected children with impaired vital functions, hemodynamic instability, convulsions, chest injuries, open limb fractures, unstable pelvic fractures, with a GCS wakefulness level of < 13 are hospitalized through the anti-shock ward to continue/start resuscitation measures and at the same time to diagnose injuries as quickly as possible [7].

The main goal is to diagnose damage as quickly as possible and eliminate the main pathophysiological mechanism that poses a threat to life

To assess the dynamics (monitoring) of clinical manifestations, the level of wakefulness (Glasgow coma scale) and the state of the pupils (pathology: asymmetry of the pupils of one or more mm (anisocoria), fixed bilateral expansion of the pupils (mydriasis), lack of reaction to bright light) are re-evaluated.

Damage diagnostics

Computed tomography is a mandatory method of examining patients with moderate (GCS 12-9 points) and severe TBI (GCS 8-3 points). Rg-craniography is performed for all patients with a consciousness level of 15-14 GCS points in case of suspected head injury, suspected fracture of the base and calvarium, and an unclear anamnesis of trauma [7,12]. In the presence of fractures and suspicion of a fracture of the bones of the cranial vault on craniograms, the victims should be sent for CT to exclude intracranial damage. Indications for CT for head trauma with a wakefulness level of 15-13 GCS points (mild traumatic brain injury) are based on the presence of risk factors for intracranial injuries [12].

High risk factors for intracranial injury

- GCS less than 15 points

- Decrease in GCS by 2 points during clinical monitoring

- Amnesia for more than 30 minutes

- Focal neurological symptoms

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- Diagnosed skull fracture or suspected fracture

- The presence of an extensive hematoma of the scalp (in children under 2 years of age)

- Suspicion of open craniocerebral injury (profuse bleeding/suspicion of liquorrhea from the nasopharynx, ears, "glasses" symptom, bruising in the mastoid process)

- Post-traumatic convulsions

- Loss of consciousness after trauma

- Repeated vomiting for no other reason

- Intense headache after injury, general anxiety, behavior change

- Trauma to the skull in patients with coagulopathy

- A history of neurosurgical pathology

- Intoxication with alcohol or other substances that depress the central nervous system

- Falling from height

- Age up to 2 years

- High-energy mechanism of injury.

CT data make it possible to objectify the extent of damage and can be considered as predictors of outcome. Unfavorable outcomes correlate with an increase in the diffuse form of brain damage from I to IV according to the CT classification of Marshall., *et al.* the presence of blood in the basal cisterns, the presence of intracranial hematomas and foci of brain contusion that create a mass effect [9,10]. Compression or absence of basal cisternae on CT is a high-risk factor for intracranial hypertension [3,10,20]. If a craniofacial injury is suspected, a CT scan in the frontal projection is necessary. In the absence of the possibility of CT/MRI, neurosonography can be used for children 1 year of age, including for the purpose of clinical and sonographic monitoring [1].

Monitoring

The purpose of monitoring is to control the state of vital functions, maintain the physiological corridor, and prevent the development of secondary brain damage.

Victims with severe traumatic brain injury (8-3 points on GCS) are subject to monitoring of vital functions [3,5,6,11,13,20].

ICP monitoring is indicated in affected children with severe TBI (GCS scores 3 - 8) [11,12,13,15]. Neuromonitoring is optimal, which includes clinical monitoring (dynamic control of the level of wakefulness according to GCS and control of the state of pupillary reactions), control of hemodynamics (preference is given to invasive blood pressure measurement), respiration, intracranial pressure, cerebral perfusion pressure, laboratory and CT monitoring. This diagnostic complex can be expanded by ultrasound examination of cerebral vessels, monitoring of central venous pressure, determination of the acid-base state of arterial and venous blood, and tissue microdialysis [20].

Respiration monitoring

Intubation of the trachea with possible subsequent mechanical ventilation is indicated for victims with impaired consciousness to the level of coma I (less than 8 points on the GCS) [3,5,6,12,15,20]. During mechanical ventilation, hyperventilation and associated hypocapnia ($PaCO_2$ < 30 mm Hg) should be avoided. In children with TBI ≤ 8 (according to GCS), it is necessary to monitor the state of the airways to prevent hypoxemia, hypercapnia and aspiration. Oxygenation and ventilation should be assessed continuously using pulse oximetry and CO_2 monitoring, or by monitoring blood gases [15,20]. In case of loss of consciousness to the level of coma (less than 8 points on the GCS), the patient must be intubated and, if spontaneous breathing is ineffective through the endotracheal tube, invasive artificial ventilation (ALV) must be performed. To prevent episodes of desynchronization of a patient with a respirator during mechanical ventilation, causing a sharp increase in ICP, it is necessary to select ventilation modes or the introduction of short-acting muscle relaxants of a non-depolarizing type of action and sedatives. It is necessary to maintain $PaCO_2$ within 35 - 39 mm Hg. Art. To prevent cerebral hypoxia, all manipulations associated with depressurization of the ventilator circuit should be accompanied by pre- and post-oxygenation with 100% oxygen. When using brain tissue oxygenation monitoring ($Pbr0_2$), it is recommended to maintain the level above 10 mmHg.

Short-term hyperventilation can be used in the event of a sharp deterioration in neurological status (the threat of dislocation syndrome development) and an increase in ICH in the absence of the effect of sedation, muscle relaxation, cerebrospinal fluid removal from the cerebral ventricles and the use of osmotic diuretics.

Hemodynamic monitoring

It is necessary to prevent episodes of falling blood pressure [3,5,6,11-13,15,20]. Arterial hypotension is corrected by restoring the volume of circulating fluid. In children, hypotension is defined as systolic blood pressure below the 5th percentile of age or when there is clinical evidence of shock. The lower limit of systolic blood pressure (fifth percentile) of the age norm can be calculated by the formula: 70 mm Hg + (2 X age in years) If necessary, use vasopressor and inotropic support.

Recommendations

Cerebral perfusion pressure - the difference between mean arterial pressure and ICP - the pressure gradient that determines cerebral blood flow. Children with severe traumatic brain injury should maintain a cerebral perfusion pressure > 40 mmHg [11,12-15,20]. Cerebral perfusion pressure in the range of 40 - 65 mm Hg (depending on age) is optimal [20].

At all stages of the provision of care (at the scene of the accident, during transportation and in the hospital), hypotension (systolic blood pressure less than 90 mm Hg) should be immediately and carefully prevented or eliminated. The mean arterial pressure must be kept above 90 mm. rt. Art. throughout the course of intensive care. Treatment of hypovolemia and arterial hypotension should be started with the infusion of colloids and crystalloids. It is necessary to control osmolarity and sodium concentration in blood plasma. Low values of osmolarity (< 280 mOsm/L) and sodium (< 135 mmol/L) corrected upward. Hypo-osmolar solutions (for example, 5% glucose solution) are not used in the treatment of acute TBI. If the effectiveness of infusion therapy is insufficient to increase the CPP, sympathomimetics (dopamine, adrenaline, norepinephrine, mezaton) should be used. In the presence of arterial hypotension in conditions of normovolemia, dopamine is used in an age-related dosage as a starting sympathomimetic. With peripheral vasodilation, it is possible to use norepinephrine-norepinephrine (IV - 1-30 μg/min) or phenylephrine hydrochloride - mezatone (IV - 0.2 - 1.0 μg/kg/min).

ICP monitoring is recommended for children with severe TBI (GCM ≤ 8). The use of ICP monitoring can also be considered in children of the first year of life (infants) with severe TBI [1,11-16,20]. Goal: Maintain ICP < 20 mm Hg. Art. Children with severe TBI (GCS ≤ 8) have a high risk of ICH. ICH can accompany diffuse axonal brain injury and post-traumatic sinus thrombosis. At higher GCG values, ICP

monitoring can be recommended for victims with severe associated injuries requiring prolonged mechanical ventilation, sedation and relaxation, i.e. under conditions that limit the ability to conduct clinical monitoring.

Intracranial hypertension is a key pathophysiologic variable in the development of secondary injuries. A meta-analysis of 15 studies involving 857 children with TBI showed the dependence of an unfavorable outcome on an increase in ICP (> 20 mm Hg) [12]. The expediency of using ICP monitoring is confirmed by the data on the high incidence of ICP in children with severe TBI, the correlation of adverse outcomes with high ICP values, and improved treatment results in patients with controlled ICP values. Although these studies are consistent with only class III evidence, their results demonstrate an association between ICH control and neurological outcome. A number of studies have shown the dependence of an unfavorable outcome after severe TBI on ICH and/or systemic hypotension [12,13].

ICP monitoring allows, on the basis of objective indicators, to determine the optimal amount of treatment, contributing to an adequate choice of such therapy methods as hyperosmolar therapy, sedation, muscle relaxation, the use of barbiturates, and decompressive craniotomy.

The treatment of severe TBI in children, as well as in adults, is largely focused on controlling ICP and maintaining the CPP. A short-term increase in ICP with a return to normal values within < 5 minutes is acceptable, but a prolonged increase in ICP ≥ 20 mm Hg. Art for ≥ 5 minutes requires treatment. In adult patients, ICP treatment begins with a threshold value of 20 mm Hg [3-5,10]. The optimal threshold and target ICP values for TBI in children depend on age. There are practically no publications on this issue; In only one study, ICP thresholds for initiation of therapy varied with age and were 15, 18 and 20 mm Hg. in children 0 - 24 months, 25 - 96 months and 97 - 156 months, respectively [13,20].

Treatment of intracranial hypertension. Correction of ICP should be started when the threshold of 20 mm Hg is exceeded [3-5,11,12,20]. There are basic (prophylactic) and emergency therapy for intracranial hypertension.

Basic (prophylactic) therapy is aimed at preventing and eliminating factors that can worsen or accelerate the development of ICH. Specific factors that can lead to an increase in ICP include impaired venous outflow from the cranial cavity (incorrect position of the patient's head, psychomotor agitation), respiratory disorders (airway obstruction, hypoxia, hypercapnia), hyperthermia, arterial hypo- and hypertension, convulsive syndrome.

Emergency therapy for intracranial hypertension. With an increase in intracranial pressure of more than 20 mm Hg. Art. all factors that can worsen or accelerate the development of intracranial hypertension should be eliminated:

- Give the head end of the bed an elevated position with an angle from 15 ° to 45 °, gradually increasing the elevation angle. The head should be in a position that prevents compression of the cervical veins.

- Control of hemodynamics and respiration.

- Exclude airway obstruction.

- Stop hyperthermia (strive to maintain normothermia).

- Stop seizures (if any).

- Control of intra-abdominal (intra-abdominal bleeding, intestinal obstruction, etc.) and intrathoracic pressure (pneumo-, hemothorax).

- Provide deep sedation and relaxation.

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With a persistent increase in intracranial pressure of more than 20 mm Hg. Art. Recommended repeated CT examination of the brain to exclude intracranial injuries requiring urgent surgical intervention (epi-, subdural, intracerebral hematomas, occlusive hydrocephalus, development of dislocation syndrome) [3-5].

External ventricular drainage, combined with an ICP sensor, makes it possible to monitor ICP by direct measurement of ICP and, if necessary, drain the cerebrospinal fluid. Drainage of CSF can be carried out only under the control of ICP measurement [5,10]. Ventricular drainage is installed in patients with severe TBI with a GCS level of consciousness < 8 points if manipulation is possible (after neuroimaging).

Hyperosmolar solutions

The results of modern clinical studies based on the principles of evidence-based medicine (two class II studies and one class III study) make it possible to recommend hypertonic saline solution for the treatment of ICH in children [20]. Children with intracranial hypertension are advised to administer a bolus of hypertonic (3%) sodium chloride solution. The recommended dose range is 2 - 5 ml/kg for 10 - 20 minutes. Also recommended is the microjet injection of 3% sodium chloride solution at a dose of 0.1-1.0 ml/kg/h [20].

It is recommended to avoid persistent (> 72 hours) sodium level rise above 170 mEq/ml. Mannitol can be used in children with increased ICP, although the evidence base is not presented [20]. Mannitol is administered as a bolus at a dose of 0.25-1.0 g/kg of body weight. The daily dose of administered mannitol should not exceed 140 - 180g.

To maintain ICP <20 mm Hg. the minimum dose should be used. Osmolarity is maintained at <360 mOsm/L [11,12,20]. Acute renal failure (ARF) is a complication of therapy with hyperosmolar solutions. Administration of mannitol is not indicated for hypernatremia (Na > 160 mmol) and hyperosmolarity (OSM > 320 mosmol/L)

The use of barbiturates

Prophylactic treatment in children is not recommended. Phenobarbital: loading dose: 10 mg/kg for 30 minutes; then 5 mg/kg every hour in three doses. Maintenance dose: 1 mg/kg/h.

Thiopental: loading dose 10 mg/kg; maintenance dose: 3 - 5 mg/kg/h

Therapeutic anesthesia with high doses of barbiturates can be used in affected children with severe TBI with stable hemodynamics and the presence of refractory ICH, resistant to the use of other methods of conservative treatment [20]. When carrying out medical barbituric anesthesia, it is advisable to control the arterio-venous oxygen difference, since there is a risk of developing oligemic cerebral hypoxia [5]. Initially, the drug is administered at a dose of 10 mg/kg per hour, followed by the infusion of 3 doses of 5 µg/kg per hour and maintaining the achieved concentration of barbiturates in the blood plasma by introducing them using an automatic perfuser at a dose of 1 mg/kg per hour. The duration of the infusion is at least 48 hours.

The use of barbiturates at high ICP values in children with severe TBI has been practiced since the 1970s. One of the first was the publication by Marshall., *et al*. [12], who reported that the use of barbiturates in patients with refractory ICH improves the outcome of severe TBI.

Clinical studies on the use of barbiturates in children are limited to the description of two cases (III class of evidence), which does not allow making clear conclusions [12]. Based on these data, it can only be assumed that barbiturates are effective in treating persistent ICH in children, however, the effect of barbiturates on survival or improvement in neurological outcome has not been established. The use of large doses of barbiturates leads to a decrease in blood pressure in both children and adults, which requires appropriate monitoring of hemodynamics [5,12,20].

Hyperventilation for severe TBI in children has been used since 1970 to rapidly reduce ICP. This approach was based on the assumption of the development of cerebral hyperemia after severe TBI. In later studies, it was found that in children, hyperemia does not occur so often, in connection with which there were doubts about the safety and advisability of using hyperventilation. A decrease in ICP with hyperventilation is due to the development of vasoconstriction (caused by hypocapnia) and a decrease in cerebral blood flow. In recent studies involving adults and children, it has been shown that hyperventilation reduces brain oxygenation and leads to the development of ischemia [5,12,20]. In a study by Stringer., *et al*. [10] the authors showed that hyperventilation simultaneously caused a decrease in cerebral blood flow in both damaged and presumably intact parts of the brain. The authors showed a relationship between the level of hypocapnia and the incidence of cerebral ischemia. The incidence of regional ischemia was 28.9% during normocapnia and increased to 59.4% and 73.1% with $PaCO_2$ 25 - 35 mm Hg and < 25 mm Hg, respectively. Experimental studies have shown that preventive hypocapnia, in addition to a decrease in cerebral blood flow, changes the buffering capacity of cerebrospinal fluid and the significance of this effect may be more important than the effect on cerebral blood flow [5].

Despite the lack of published data on the benefits of hyperventilation in children with severe TBI, it continues to be widely used. There are no randomized controlled trials of the effect of hyperventilation on various aspects of severe TBI in children, such as refractory ICH or the development of dislocation syndrome.

Recommendations

Avoid hyperventilation to $PaCO_2$ < 30 mm Hg in order to prevent the development of intracranial hypertension (especially in the first 48 hours) [12,20]. Hyperventilation is contraindicated at pCO_2 < 28 mm Hg. Art. Hyperventilation should be considered as a temporary forced measure to reduce increased ICP, for example, when transporting a patient to the operating room, with the ineffectiveness of all conservative measures and the threat of the development/increase of dislocation syndrome [5,10].

When using hyperventilation, the adequacy of oxygen supply to the brain should be monitored by determining $SvjO_2$ and/or $PbrO_2$. $SvjO_2$ values that are in the range of 55-75% are considered normal, provided that the arterial blood is sufficiently oxygenated. The $PbrO_2$ norm is 25-35 mm Hg. Art. when oxygen tension in arterial blood is 80-100 mm Hg [20].

Hypothermia

The therapeutic effect of hypothermia is achieved by reducing the metabolic requirements of the brain, the severity of inflammatory reactions, lipid peroxidation and excitotoxicity. Moderate hypothermia (32 - 33°C) is possible immediately after severe TBI within 48 hours [5,12].

It is necessary to cool the patient to the required temperature very quickly (within 30 - 60 minutes), and warm it up very slowly (0.2 - 0.3°C per hour). Cooling the patient can be accompanied by serious complications: hypocoagulation, increased urine output, electrolyte disturbances, impaired hydration of the respiratory mixture, infectious complications (from the recommendations for adults).

There is considerable doubt about the prophylactic use of hypothermia in children with TBI. A number of studies, including two studies of class II evidence, have shown that mild to moderate hypothermia in comparison with normothermia contributes to a decrease in ICH. However, the effectiveness of such therapy compared to other treatments, including first-line drugs or targeted therapy for persistent ICH, remains unclear. In addition, conflicting results have been obtained regarding the effect of hypothermia on mortality and/or outcomes.

The current guidelines for adults note that although hypothermia is often used in ICUs when ICP rises in many centers, the scientific literature does not contain unambiguous data on the positive effect of hypothermia on mortality and morbidity [16]. A meta-analysis

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of data on the use of hypothermia in adult patients with severe TBI [16] showed that the total risk of mortality reduction did not differ significantly in the groups with hypo- and normothermia, but hypothermia was accompanied by a 46% increase in the chances of a good outcome (relative risk 1.46; 95% CI 1.12 - 1.92).

Decompressive craniotomy can be recommended in case of threat/increase in dislocation syndrome or in the development of refractory ICH in children with severe TBI [14-16]. Decompressive craniotomy is the most aggressive treatment for intracranial hypertension. Decompressive craniectomy is used in the last turn when conservative therapy measures are ineffective. The main goal of decompressive trepanation is to increase the intracranial volume, thereby reducing ICP and improving the functional state of the brain. Decompressive trepanation can be used as a concomitant procedure when removing injuries causing a mass effect (intracranial hematomas, contusion foci), with a threat of increasing cerebral edema (secondary decompressive trepanation).

Contraindications for decompression craniotomy

Atonic coma (GCS score 3 + bilateral mydriasis + muscle atony and areflexia) without subsequent improvement on the background of resuscitation measures and associated

- With a primary contusion of the brain stem according to CT (MRI) with a clinic of irreversible persistent damage (GCS 3 points from the moment of injury)

- With secondary ischemic damage to the brainstem on the background of dislocation syndrome with a clinic of persistent irreversible damage 3 GCS scores + bilateral mydriasis + muscle atony and areflexia)

- With reverberating blood flow (or lack of blood flow) through the main cerebral arteries of the base of the brain from both sides with TCD in combination with the clinic of atonic coma

- With the absence of blood flow in four basins of the main arteries of the brain according to MRI angiography in combination with the clinic of atonic coma

- Absence of acoustic evoked response potentials on both sides in combination with reverberant (or absent) cerebral blood flow and the clinical picture of atonic coma.

Prevention and treatment of pulmonary complications

Prevention of aspiration of the contents of the oropharynx and stomach

It is necessary to conduct early tracheal intubation and maintain the required pressure in the endotracheal tube cuff (20 - 25 cm H_2O). Continuous supra-cuff aspiration is used to prevent aspiration. If the duration of mechanical ventilation is more than 5 days, tracheostomy is necessary.

For the prevention of gastroesophageal reflux, the victims should be given a lateral position with an elevated head end of the bed and enteral nutrition through a nasojejunal tube. In the later stages of treatment of severe brain lesions, if the patient has signs of dysphagia, it is possible to carry out a percutaneous endoscopic gastrostomy. Prevention of cross-contamination and colonization through the hands of personnel: it is necessary to carry out thorough processing of ventilator equipment, fiberoptic bronchoscopes and conduct regular monitoring of bacteriological contamination of ventilators after sterilization. The order of individual use of aspiration devices should be maintained and the reuse of sanitation catheters should be avoided. Whenever possible, special closed systems for sanitation of the tracheobronchial tree and combined breathing filters should be used. The tracheobronchial tree must be sanitized with sterile gloves.

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After any manipulations with the patient, hands and gloves should be treated with special alcohol-based disinfectant solutions. Use disposable paper towels or tissues to dry your hands after washing with running water. When prescribing antibiotic therapy, one should take into account the pharmacokinetic properties of antibiotics, select doses of drugs taking into account their minimally suppressive concentrations and carry out a planned rotation of drugs.

Prevention and treatment of intracranial purulent complications

To prevent meningitis and ventriculitis, you need:

- Timely sanitation of the paranasal sinuses in the presence of purulent contents in them. Patients with TBI during the postoperative CT scan of the head should also examine the paranasal sinuses. If sinusitis is suspected, the patient should be examined by an otolaryngologist.

- Preoperative and intraoperative antibiotic prophylaxis during emergency surgery.

- Observance of the rules of asepsis during neurosurgical manipulations in intensive care units (dressings and lumbar punctures).

- Observance of the rules of asepsis by the personnel of the neuroresuscitation department.

The main routes of administration of antibacterial drugs in the development of intracranial purulent complications are parenteral and intrathecal. Intrathecal administration is carried out with a lumbar puncture, through a lumbar drain, or through a ventricular catheter. Lumbar drainage should be installed if cytosis exceeds 400-500 cells/mm3. In order to avoid dislocation of the brain, lumbar puncture and installation of lumbar drainage are not performed if there are signs of axial or pronounced transverse dislocation according to computed tomography of the brain. In the presence of clinical and CT signs of ventriculitis, catheters are inserted into the anterior horns of both lateral ventricles. Antibiotic therapy is prescribed according to cerebrospinal fluid culture. Antibacterial drugs for drainage are administered 2 - 6 times a day. The introduction of antibiotics is carried out by a neurosurgeon. Intrathecal administration of antibiotics is carried out in dilution with physiological sodium chloride solution. It is preferable to use Vancomycin in a single dose of 5 mg (daily dose 10 mg), Amikacin in a single dose of 100 mg (daily dose 300 mg), Meropenem in a single dose of 10 mg (daily dose 40 mg). The rest of the time, the drainage is set to passive outflow. When conducting ventricular or lumbar drainage, cerebrospinal fluid over drainage should be avoided by installing a drainage system "knee" at the level of the Monroe's orifice or using special closed systems that allow simultaneous measurement of CSF pressure and a fixed discharge of cerebrospinal fluid. Parenteral antibiotic therapy is discontinued 3 to 4 days after complete regression of the symptoms of meningitis or ventriculitis.

Nutrition for victims with severe TBI

There is insufficient evidence to recommend immunomodulators to improve outcome in severe TBI [9,16-18]. Hyperglycemia should be avoided as post-traumatic hyperglycemia may be associated with poor outcomes [5,13-16].

The use of parenteral nutrition in children with TBI is of great importance. As in adults, children with severe TBI need to be provided with energy requirements to effectively repair damage, function and prevent other pathological conditions initiated by trauma.

Children have higher energy requirements for normal growth and development than adults. The decision to use parenteral nutrition, including the timing, amount, method and composition of such support, can significantly affect short-term and long-term outcomes.

Despite numerous studies on the timing, volume and composition of parenteral nutrition in children with severe TBI, only one of them met the requirements of a class II randomized controlled trial. The results of the study showed no difference in the outcomes of children

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with standard nutrition and nutrition that includes immunomodulators. Hyperglycemia should be avoided as there is evidence that post-traumatic hyperglycemia is associated with poor outcome.

For an accurate assessment of the energy demand of patients, it is necessary to use the method of indirect calorimetry. In the absence of a metabolograph, the energy requirements of patients are calculated using the formulas. Nutrition can be carried out both enterally and parenterally. The advantages of enteral nutrition over parenteral nutrition are the lower risk of developing hyperglycemia and infectious complications. For enteral nutrition, a naso-or orogastric tube is inserted. If the gastric nutritional option is ineffective for more than 2 days, a small intestinal probe is installed. In this case, special semi-element mixtures should be used for feeding patients. If long-term enteral tube feeding is required for more than 4 weeks, a gastrostomy tube may be applied. Where possible, enteral nutrition should be given priority. Children should provide from 130 to 160% of metabolic costs [15].

Post-traumatic convulsions

Phenytoin (diphenin) can be recommended for the prevention of early post-traumatic seizures in children with severe TBI [20].

Post-traumatic seizures include seizures that develop within 7 days after injury, or later, 8 days after coming out of a coma. The risk factors for the development of post-traumatic seizures include the severity of the injury, the location and nature of the injury, the presence of bone fragments or foreign bodies in the brain substance, a depressed skull fracture, focal neurological deficit, loss of consciousness, decreased wakefulness on the Glasgow coma scale <10, duration of post-traumatic amnesia, presence of subdural or epidural hematoma, penetrating injury, chronic alcoholism and age. Infants and young children have a lower seizure threshold.

For the diagnosis of post-traumatic epilepsy, the most important are computed tomography, electroencephalography and magnetic resonance imaging (Angeleri F, Majkowsky J, Cacchio G, et al. Posttraumatic epilepsy risk factors: one year prospective study after head injury. Epilepsia. 1999; 40: 1222 - 1230, Verellen RM, Cavazos JE. Post-traumatic epilepsy: an overview. Therapy. 2010; 7 (5): 527-531). These methods of neuroimaging in the acute period of traumatic brain injury make it possible to identify lesions corresponding to risk factors for the development of post-traumatic epilepsy.

In an EEG study, attention is paid to the presence of patterns of epileptic activity or epileptic seizures according to the criteria for detecting epileptiform activity. It is important to note that with the development of epileptic seizures, it is necessary to conduct CT (MRI) of the brain and EEG studies.

The incidence of post-traumatic seizures in children with TBI is approximately 10%. Based on the results of one Class III study, prophylactic anticonvulsant therapy with phenytoin may be considered a method for reducing the incidence of early post-traumatic seizures. Given the high likelihood of metabolic disorders in patients with severe TBI, it is recommended to monitor the drug level.
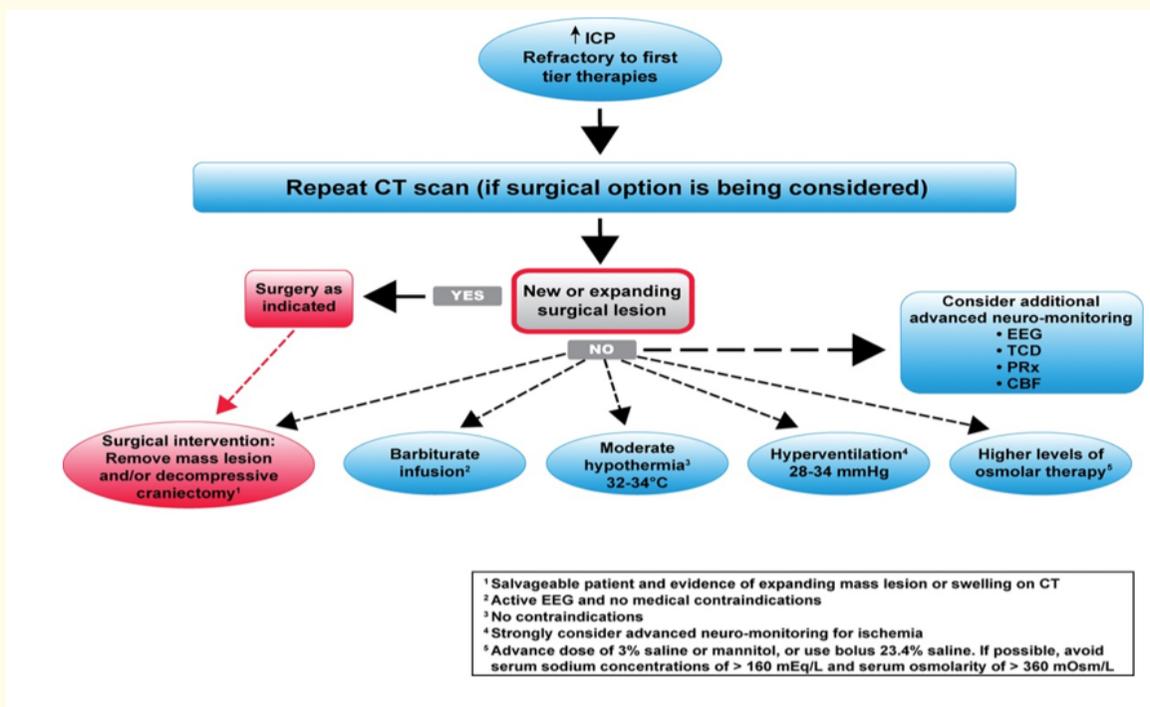
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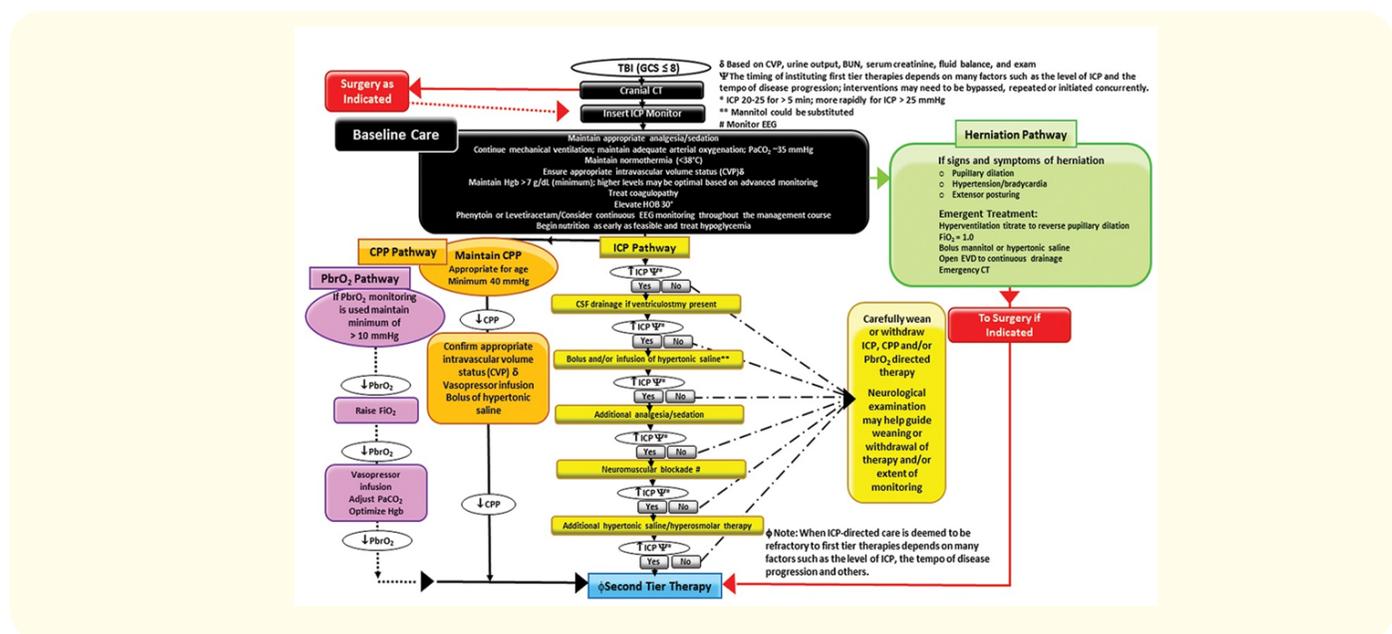
Figure 1: *Algorithm for diagnosis and treatment tactics in children with severe TBI (Guidelines for the Management of Pediatric Severe Traumatic Brain Injury, Third Edition: Update of the Brain Trauma Foundation Guidelines, 2019).*

Conclusion

In the presence of risk factors for the development of post-traumatic seizures, as well as the presence of unprovoked paroxysms and epileptic activity on the EEG, it is recommended to prescribe anticonvulsants in initial therapeutic doses. When choosing anticonvulsants for prophylactic anticonvulsant therapy, preference should be given to drugs with a neuroprotective effect (hydantoins, barbiturates, gamma-aminobutyric acid preparations, etc).

Appendix 1: Glasgow paediatric coma scale (GCS)

		Older than 1 year	Younger than 1 year	
Eye opening	1	Spontaneous	Spontaneous	
	2	Per voice command	Per voice command	
	3	For pain	For pain	
	4	No answer	No answer	
Best motor response	1	Command	Execution	
	2	Localization of pain	Localization of pain	
	3	Flexion - withdrawal	Normal flexion	
	4	Pathological flexion (decortication rigidity)	Pathological flexion (decortication rigidity)	
	5	Extension (decerebral rigidity)	Extension (decerebral rigidity)	
	6	No answer	No answer	
		Over 5 years old	2-5 years old	0-23 months
Best verbal response	1	Oriented and contact	Age-appropriate speech production	Gulps, smiles, or shows displeasure
	2	Incoherent speech confusion	Incoherent speech confusion	Episodic scream, cry
	3	Isolated words in response to irritation or spontaneous	Screaming and/or crying	Constant screaming or crying
	4	Inarticulate sounds on irritation or spontaneous	Moan	Moan
	5	No answer	No answer	No answer

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