Management of head injury in children

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Abstract

Objective: to make pediatricians aware of ideas about how to handle head injury in children under and over 2 years of age, to facilitate understanding and to allow a rethinking about the necessary care when attending children with head injury.

Sources: a bibliographic review of the theme based on Medline.

Summary of the findings: the following aspects were analyzed: the overall epidemiological aspects, the concept and the physiopathology of the damage caused by brain trauma. The assessment of the severity of head injury for different age ranges was discussed, as well as the opinion of various authors and the current ideas about the criteria regarding hospitalization, request for supplementary exams and therapeutic approach. The value of neuroimaging exams in cases of head injury and, finally, the prevention strategies and conclusions also were discussed.

Conclusions: in most cases, head injuries in children are rated as not being severe. Nevertheless, even though they are seen as slight, when considering the pediatric population the clinical picture is often asymptomatic presenting neuroimaging changes, the management of head injury at this age range is different from the management in adults.


Introduction

In the last few years, pediatricians and neurologists have been more and more interested in understanding the impact of head injuries in children. Head trauma (HT) is one of the most common causes of injury in children, and it is responsible for the high number of hospital admissions, with a significant rate of morbidity and mortality. In the United States, it is estimated that between 155 and 180 out of 100,000 children per year look for medical care due to closed head injuries, and 74 to 80% of the cases of HT are considered minor head injuries. In England, 5% of all pediatric admissions are due to HT, and approximately 3,000 children per year have neurological sequelae. Durkin et al. verified an annual mortality rate of six out of 100,000 children. There is a slight predominance of males, at a ratio of 1.5:1.0. Several studies performed in children with minor HT revealed the presence of headache, behavioral and cognitive changes a few days after its occurrence. Fortunately, most cases present good recovery without severe sequelae. The traumatic brain injury (TBI) is considered as closed or open head injury, with evidence of brain involvement, shown through altered level of consciousness (sleepiness, lethargy, mental confusion or coma) or signs of focal neurologic deficit. Injuries related to motorcycle or bicycle falls and accidents involving pedestrians represent 50% of the cases of HT. In children...
under three years old, falls are frequent causes of brain injury. Abuse or maltreatment is another important etiology of HT, which accounts for 24% of brain lesions in children, especially those under two years old.9

Sometimes children with HT are not seen by physicians specifically trained to manage neurotrauma. Pediatricians that perform emergency care should have some knowledge about the physiopathology and management of TBI in children.

Summary of the findings

The bibliographic review was based on Medline. Five hundred and forty-seven articles were evaluated and the references considered relevant were selected. According to the format of the study, we gave special emphasis to the use of the journal Pediatrics. Current methods and ideas regarding the management of young children presenting head trauma were mentioned. Two books were used as reference: Cuidados Neurológicos em Terapia Intensiva Pediátrica and Coma en Pediatría - Diagnóstico y Tratamiento.

Mechanisms of traumatic brain damage

Basically, the mechanism of posttraumatic brain injury occurs in two different ways: first, by the impact of the trauma; and secondly, from the effects of respiratory and circulatory insufficiency, or by increased intracranial pressure.

Primary brain injury

The extent of the initial impact of the trauma on the brain determines the severity of lesions. Brain damage may occur through several different ways such as contusions, lacerations, diffuse axonal injury and dural tear, which may be caused by acceleration and deceleration. Shortly after the head impact, there is a reduction or even the interruption of brain blood flow, and the reserves of nutrients and oxygen finish within seconds. This leads to an anaerobic metabolism of the brain tissue through ischemia, with accumulation of lactate and acidosis as a consequence. There is an unbalance due to the lack of energy supply, which establishes ionic instability with alteration of the electrolyte pumps, in the action potential of the membranes and in the calcium inflow into the cell causing irreversible cell lesion. During the phase of cerebral reflux, there is production of oxygen free radicals, which directly stimulate the lipoxygenase and cyclooxygenase pathways. Then, there is a sequence of inflammatory processes, which leads to vascular alterations (vasodilation and vasoconstriction), alteration of the vascular permeability, chemotaxis of granulocytes and platelet aggregation activating the coagulation process and occurrence of microthrombi. Regarding the body as a whole, there might occur suppression of the immune system mediated by cells, which increases the chances of secondary infections. Another physiopathological mechanism related to brain injury after TBI is excitotoxicity. Experimental studies10 verified that neurological trauma facilitates the release of excitotoxict amino acids, such as glutamate and aspartate. Both amino acids interact with the postsynaptic NMDA (N-methyl-D-aspartate receptor) and non-NMDA receptors (&-amine-3-OH-methyl-4 isoxazole receptor, AMPA), which trigger calcium and sodium influx, respectively, and establish neural damage and edema.10 The increase in these excitotoxic amino acids often cause convulsive seizures. These damages are reduced by antagonists of NMDA receptors, such as ketamine and MK-801. Changes in the cell membranes may increase cell sensitivity, and cellular lesions may increase cell sensitivity to secondary damages, such as vasospasms, edema, hypoxia and/or hypotension.11

Secondary brain injury

Ninety percent of the patients with HT present secondary complications. These complications occur within a few minutes, hours or days after the primary lesion and may lead to parenchymal damage, which worsens the neurological damage.12 The secondary conditions of the posttraumatic brain damage may be caused by systemic disorders and intracranial disorders. Among the systemic disorders, hypoxia and hypotension are significant factors for the complication of the brain lesion. Trauma often leads to loss of consciousness, and it may affect vital structures of the brainstem. Therefore, it may trigger long apnea, hypoventilation, hypercarbia and/or atelectasis. The alterations triggered by the loss of consciousness often contribute to the hypoxic condition, such as pneumothorax, hemotherax and pulmonary edema. Arterial hypoxemia (PaO2<60mmHg) can be related to the increase in the mortality rate from 25 to 50%.

Hypotension occurs in 35% of the cases with severe TBI.11 When hypotension occurs, there is a decrease in the brain blood flow, since brain perfusion pressure (BPP) is directly related to the mean arterial pressure (MAP), (BPP = MAP-ICP) leading to ischemia and cerebral stroke. The severity of this situation might also be increased, if the patient is anemic. Hypercarbia causes vasodilation in the cerebral venous system and, consequently, the intracranial pressure (ICP) increases. On the other hand, ICP may also be increased by the mass effect caused, for instance, by intra- or extra-axial hematomas, cerebral edema and/or cerebral hyperemia. Intracranial hypertension, in its turn, may produce ischemia or direct compression of the brain structures, leading to a transtentorial herniation.

Electrolytic abnormalities constitute another set of factors involved in the mechanism of brain injury, and disorders of calcium homeostasis should receive special attention. Hyponatremia may cause inappropriate secretion of the antidiuretic hormone, which leads to cerebral edema. Diabetes insipidus is another complication seen in severe
cases of HT. Hypoglycemia, more often observed in newborns or preterm infants, also leads to lactic acidosis and release of free radicals, which worsen the brain injury.

Hyperthermia is another factor that can worsen brain injury, since every time there is an increase of 1 °C in the body temperature, there is also an increase of 5% in the brain metabolism. Hyperthermia should be observed, for it increases the unnoticeable loss of water, which can worsen hypotension.13

Assessment of the severity of head trauma in children

For an appropriate assessment, the pediatrician should analyze the whole context of the medical care provided to the pediatric patient. Some aspects need to be observed in order to obtain success regarding the evolution of the child with HT:

– with regard to the conditions of the institution that provides the medical care, the following aspects related to infrastructure should be observed: diagnostic imaging sector (pediatric radiologist), presence of pediatrician, pediatric neurosurgeon and specialized and/or trained staff able to provide medical care to the child with HT;

– with regard to the conditions related to the child that should be assessed: patient’s age, level of consciousness (sleepiness, lethargy, coma); presence of focal neurological signs; occurrence of convulsive seizure; alterations of child’s behavior, such as irritability, agitation or severe apathy or indifference during the post traumatic period; intense headache. The anamnesis should be brief, but clear and straightforward, in order to clarify the exact conditions of how the trauma occurred. When the person reporting the episode has doubts, neglect or maltreatment should be considered. Alterations verified in the child’s skullcap or scalp must be observed, suspicion of fractures, including presence of lesions in other parts of the body.

Certain doubts or controversies still remain regarding the best approach employed to manage the medical care of a child under two years old with minor HC. When should the child be admitted to the hospital? When should the child be sent home? When should the physician ask for complementary neuroimaging exams, such as skull radiograph (SR), cranial computerized axial tomography (CAT) or nuclear magnetic resonance (NMR) of the brain? It is important to remind the physicians of the fact that children under two years old, mainly the ones under 12 months, may present a completely normal clinical-neurological exam and, even though, present brain alterations detected through neuroimaging exams.14 The American Academy of Pediatrics designed a protocol for the management of minor HT in children under two years old, published in the journal Pediatrics.15 It will be discussed later.

Severity scale of traumatic brain injury

It is important to define the degree of neurological damage of a child with HT, since it allows the ICU physician, or the pediatrician who provides emergency care, to have an idea of the patient’s initial neurological conditions and his/her evolution. It also allows the physician to design an action plan in order to find a better set of necessary and efficient measures regarding the management of patients with neurotrauma. Table 1, mentioned by Sanchez et al.16, describes the severity scale of traumatic brain injury, and it is used for older children and adults. Taking into consideration that children under two years have greater chances to present skull fracture as a consequence of HT, and they often present a different route of evolution, in addition to the difficulty of assessment, this group of patients receives special management. Quayle et al.17,18 have found 6%, and Dietrich et al.,19 5% of the children with minor HT, whose intracranial lesion was observed though the cranial CAT.

Sanches et al.16 consider a minor HT when the child or adult presents, in the initial examination after the occurrence of HT, a Glasgow scale score between 13 and 15, without occurrence of loss of consciousness and neurological

<table>
<thead>
<tr>
<th>Data</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Very severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>GCS</td>
<td>13-15</td>
<td>9-12</td>
<td>3-8</td>
<td>3-4</td>
</tr>
<tr>
<td>Coma duration</td>
<td>No</td>
<td>No</td>
<td>&gt;6h</td>
<td>–</td>
</tr>
<tr>
<td>ICD-9</td>
<td>&lt;1h</td>
<td>1-24h</td>
<td>&gt;24h</td>
<td>–</td>
</tr>
<tr>
<td>Amnesia duration</td>
<td>&lt;1h</td>
<td>1-24h</td>
<td>1-7 days</td>
<td>&gt;7 days</td>
</tr>
<tr>
<td>Posttraumatic evolution</td>
<td>Good</td>
<td>High risk for cognitive problems</td>
<td>Very high risk</td>
<td>Physical and cognitive deficit</td>
</tr>
</tbody>
</table>

GCS- Glasgow Coma Score, ICD- International Classification of Diseases - 9th issue
abnormalities. The same authors recommend the performance of a cranial CAT in every patient presenting loss of consciousness for longer than five minutes, even if he/she has a score considered as minor HT. Casado Flores & Serrano also adopt this approach, which we also consider appropriate, since recent studies have recommended cranial CAT scanning even for minor traumas, so that the patient will be sent home based on a safer decision.

Patients considered as presenting moderate HT have a high risk for neurological sequelae, such as behavioral-cognitive disorders. Severe and very severe cases may present motor or physical incapacity, which means that moderate, severe and very severe cases of HT are always hospitalized.

Assessment and management of minor HT in children under two years

Pediatricians have shown special interest in the group of children under two years, taking into consideration that 76% of the cases of HT are classified as minor HT. Even though head trauma is classified as minor, some children under two years present complications. And this explains the reason why this pediatric group has been studied as a unique group. According to the recent review of the American Academy of Pediatrics, minor HT in children was defined as those cases presenting history of scalp, skull or brain trauma, alert or awake mental state, observed on the first examination, absence of focal neurological findings, and physical absence of skull fracture evidence. Newborns, multiple traumas, penetrating lesions, hemorrhagic diathesis, preexistence of neurological involvement and/or previous neurosurgery and absence of abuse or neglect in his/her preexistence of neurological involvement and/or previous neurosurgery and absence of abuse or neglect in his/her initial examination are not included in this group. The presence of hematoma, cerebral contusion and/or cerebral edema are also defined as intracranial injury (ICI).

According to the analysis of the American Academy of Pediatrics, children with minor HT were classified in four groups of risk for ICI:
1. children with high risk for ICI, who should undergo a cranial CAT scanning;
2. children with some potential indicator of risk for ICI, who should undergo cranial CAT scanning and observation;
3. children with absence of signs and symptoms of brain lesion, although presenting some risk for skull fracture and ICI, who should undergo cranial CAT scanning, skull radiograph and observation;
4. children with low risk for ICI, who do not need to undergo neuroimaging exams.

Risk 1 Group: children that present the following symptoms are considered as having high risk for ICI:
- alteration of the level of consciousness (patient with great difficulty to stay awake);
- presence of focal neurological abnormalities, sign of depression of the skullcap, clinical signs of skull fracture or fracture detected through the SR, irritability while being examined, bulging fontanelle. Occurrence of convulsions and/or persistent vomiting, even though the presence of vomiting is quite common in children. The occurrence of more than five episodes of vomiting in six hours should be considered;
- the risk factor inversely proportional to the child’s age is also important or, in other words, special care should be provided to breastfed infants under three months of age.

Groups 2 and 3: intermediate risk
a) Children with clinical indicators of potential ICI:
- children with three-four episodes of vomiting;
- transient loss of consciousness > 1 minute;
- history of lethargy or irritability (improved up to the examination);
- alterations of behavior;
- skull fracture observed after a period of 24 hours.

Cranial CAT should be requested, if more than one of the factors mentioned above are present, if the loss of consciousness lasts longer than 15-30 seconds and if the change in the pattern of behavior lasts longer than 30 minutes, or if the patient is very young. In case of cranial CAT impossibility, the patient should remain under continuous observation for four to six hours after the trauma; in the presence of the clinical indicators mentioned above, CAT scanning should be performed. If those clinical indicators are not present during the observation period, the child should be sent home.

b) Children presenting trauma with unknown mechanism, or whose physical examination findings suggest the possibility of skull fracture:
- situation of strong impact, such as crashes or motorcycle fall while in movement, with child ejection and one meter (three-four feet) fall;
- fall on hard surfaces (concrete, wooden floor);
- scalp hematomas, especially in the temporo-parietal region. Frontal hematomas present lower risk of complication;
- vague or unclear report of the trauma occurrence, in this case, it is important to consider the possibility of maltreatment and neglect.

Depending on the factors mentioned above, a cranial CAT scanning might be performed. One of the difficulties is that, in order to obtain a CAT in this age group, usually sedation and/or general anesthesia is necessary. However, this possibility is not always available in pediatric medical care centers. If it is not possible to perform a cranial CAT, observation of the child for six hours is recommended, and in case the symptoms mentioned above are not present, the patient might be sent home after the observation period.
c) Children with low risk for ICI:

In this group of children, the risk of neurological complications is rather insignificant. This category includes children with trauma caused by mechanism of low impact (low energy), under one meter falls, and who do not present posttraumatic clinical manifestations for, at least, two hours.

After appropriate assessment/observation and/or after undergoing neuroimaging exams all children may be sent home, if:
- the child does not present any condition of potential ICI;
- the child remains awake with normal neurological examination;
- there is not suspicion of abuse or neglect;
- the child lives near a health care clinic, or near the hospital where he/she was seen, in case the child needs to return for an appointment.

Every time a patient is sent home, relatives or guardians should receive information regarding possible alterations that might occur, such as excessive sleepiness, change in behavior patterns and/or persistent vomiting, especially in young breastfed infants. The immediate return to the medical care center is recommended, if such abnormalities occur after the patient was discharged from hospital.

Use of neuroimaging in HT

Skull radiograph (SR) has low predictive value regarding neurological complications, and its performance is controversial. SR is highly recommended if there is suspicion of skull fracture, mainly for linear fractures, and in young children.

Computerized axial tomography (cranial CAT) is the exam of choice for HT and for the assessment of ICI. The only obstacle is that it is usually necessary to sedate the child and/or use general anesthesia, which may produce symptoms such as sleepiness and vomiting after anesthesia. Such symptoms may be mistaken for HT symptoms and, consequently, they might cause longer observation period and length of stay in hospital. CAT detects abnormalities that may define the therapeutic management in the first hours, and draw physicians’ attention to quick neurological deterioration (small extradural hematomas, bilateral frontal contusion or unilateral temporal mass), in addition to being an auxiliary prognostic tool. Table 2 describes the classification of the most frequent tomographic findings in children according to Marshall et al.25

The nuclear magnetic resonance (NMR) of the brain should be performed, when necessary, after CAT scanning. It offers better image resolution than the cranial CAT for small hemorrhages, ischemic disorders, microthrombi, late hemorrhages and posterior fossa lesions can be better observed.26,27 However, the performance requires more specialized centers, and it also requires a longer sedation. Thus, this procedure remains restricted to more specific and later assessments, depending on the clinical evolution of the patients, or whether the pediatric neurosurgeon considers it appropriate.

Clinical management

Children with HT need to be continuously monitored regarding their level of consciousness, respiratory pattern, hemodynamic control, and they also need adequate technological resources and a quiet environment. Most of the time, there is no necessity for surgical intervention, since minor HT predominates in the pediatric population. Basically, the management of these children aims at controlling intracranial components (brain, liquor and blood). This means to keep the brain blood flow stable, to guarantee a good oxygenation in order to provide recovery of the brain tissue, avoiding brain herniation. The head of the bed should be slightly raised, from 30 to 45 degrees.

Oxygen support is recommended for patients with HT, mainly for those patients with symptoms of brain dysfunction presenting decreased level of consciousness, with Glasgow score below eight. In the most severe cases, endotracheal intubation and ventilation are necessary, even though the prophylactic hyperventilation is not recommended, since it may cause cerebral vasoconstriction.28,29

Table 2 - Classification of diffuse brain injury based on CT

<table>
<thead>
<tr>
<th>Category</th>
<th>Definition</th>
</tr>
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<tbody>
<tr>
<td>I</td>
<td>Absence of any visible intracranial injury on CT</td>
</tr>
<tr>
<td>II</td>
<td>Presence of cisterns Midline shift from 0 to 5 mm or dense injury or both Absence of high-density or mixed-density lesion &gt;25 ml Includes bone fragments and foreign bodies</td>
</tr>
<tr>
<td>III</td>
<td>Compressed and absent cisterns Midline shift from 0 to 5 mm Absence of high-density and mixed-density lesion &gt;25 ml</td>
</tr>
<tr>
<td>IV</td>
<td>Midline shift &gt;5 mm Absence of high-density and mixed-density lesion &gt;25 ml</td>
</tr>
</tbody>
</table>
The use of corticoids in HT is not generally recommended. Steroids are recommended for the vasogenic cerebral edema, around tumoral masses or brain abscesses as well as in the medullary injury. Recent studies have not shown significant changes in the mortality and morbidity rates of patients treated with corticoids soon after the head trauma. Therefore, their use does not present confirmed benefit, and its recommendation has not been stimulated.30,31

The use of anticonvulsivant drugs, such as diphenylhydantoin and phenobarbital, are recommended for severe traumas, which may potentially lead to convulsive seizures.32 Barbiturates were broadly used in the past, today their recommendation is restricted to invasive procedures that cause severe increase in the intracranial pressure, such as status epilepticus and refractory intracranial hypertension. Their prophylactic use is not recommended.33

The use of diuretics in the management of TBI is based on the reduction of intravascular volume and potentiation of the beneficial effects of fluid restriction and, consequently, it reduces ICP. The diuretic often employed is furosemide, and it has been used for the prevention of hyperemic cerebral edema and in the treatment of patients with cerebral edema with subacute or chronic evolutions.

The dose is 0.5 to 1 mg/kg, and it may be administered every four or six hours, the effect lasting for 30-60 minutes. Electrolytes should be monitored. Mannitol has been consistently used for the control of intracranial hypertension. It reduces the intracranial hypertension and improves brain perfusion.34 The dose administered is from 0.25 to 1 g/kg, in intravenous bolus, it may be repeated every two or six hours. Its effect lasts for four hours. Its use is limited by hyperosmolarity and electrolytic disorders, and in the depletion of intravascular volume. Uncontrolled use of mannitol can produce some complications, such as hemorrhage and ischemia in cases of hyperosmolarity, hemodynamic overload with heart failure, dehydration, renal insufficiency and deterioration of the cerebral edema, if the serum osmolarity is higher than 310 mOsm/L. Other authors do not agree with the use of mannitol.

Another commonly used diuretic is acetozolamide, which inhibits carbonic anhydrase. It is recommended for cerebral edema, although its use should be restricted in severe cases with intracranial hypertension. The use of acetozolamide for a long period of time may establish systemic acidosis and deleterious effects on the CNS.

The management of fluids and electrolytes is essential, uncontrolled water intake may intensify hyperemia and severe cerebral edema. Hypovolemia, in patients with multiple traumas presenting hemorrhage, may contribute to the cerebral ischemia. Therefore, monitoring the cardiac function is absolutely necessary.

Today, besides the usual therapeutic interventions, there are some studies regarding the beneficial pharmacological effect of some drugs on the traumatic brain injury caused by ischemia and reperfusion. Some of these drugs include calcium antagonists, calmodulin inhibitors, iron and free radicals chelators, lipid antioxidants, hormonal aspects of growth and antagonists of excitatory amino acids and of endogenous opioid peptides.35 Even though there are countless therapeutic possibilities, deeper investigations are necessary in order to define which the most efficient and less toxic agents are.

Surgical management

Surgical treatment for HT can be useful in the treatment of decompression, in order to reduce the ICP, or for the treatment of epidural, subdural and intraparenchymatous hematomas. Epidural hematomas occur in 6% of traumatic brain injuries during childhood, and usually their manifestation happens 24 to 72 hours after the trauma. Evacuation is always necessary in surgical drainage of significant epidural hematomas, with volume greater than 30 ml, density greater than 15 mm and a midline shift greater than 5 mm. However, surgical approach is controversial in minor asymptomatic hematomas.36

Subdural hematomas are associated with severe traumas. They may be caused by brain concussion or venous injury and are seldom associated with fractures. They usually are bilateral, and the occurrence is more frequent in children over one year. Children with decreased level of consciousness, bulging and tense fontanelle, and suture spacing should undergo bilateral subdural drainage by mean of subdural needles. The treatment of interhemispheric subdural hematomas in asymptomatic patients is conservative.

TBI prevention

Prevention is certainly the best kind of treatment. Since children are still growing and developing, they are prone to different kinds of head trauma. Traumas that range from birth trauma to car accidents and, especially in young children, often these children are subjected to maltreatment and neglect caused even by members of their own family. Some strategies of prevention mentioned by Fonseca11 are described below.

During sport practice or playing
- Use of helmets while cycling, skateboarding, roller skating and horseback riding.
- Smooth surfaces and light toys in playgrounds.

Falls
- Installation of safety nets or rails on the windows.
- To avoid baby walkers.
- To avoid high cement slabs or clear spans.
- To avoid gates near staircases.
**Motor vehicles**
- Use of helmets by motorcycle drivers and riders.
- Use of safety belt in cars, and belts adapted to children in the back seat.
- Airbags as mandatory equipment in the vehicles.
- Prevention and combat of the use of illegal drugs during childhood and adolescence.

**Conclusions**

The Committee of Neuropediatrics of Sociedade Paranaense de Pediatria designed a practical manual of neuropediatrics that presents useful recommendations for the medical care of children with head trauma. As a conclusion, see the suggestions below with slight alterations.

1. Never underestimate anamnesis and physical examination. No complementary exam will show as much information as the personal medical care.

2. Try to organize the data according to a time sequence, so that you are able to consider if state of consciousness is getting worse.

3. Try to reassure the child and the parents, and establish an adequate environment of dialogue and confidence.

4. Injuries are considered as minor head traumas when there is no loss of consciousness, and the Glasgow score adapted to children is 15. And when the period of amnesia does not last for more than five minutes, and neurological exam is normal. This criteria is valid even for older children.

5. Glasgow score below 12 in children, or a period of loss of consciousness longer than five minutes in older children, and longer than one minute in children under two years, requires surveillance and hospital admission.

6. Cranial computerized axial tomography is the exam of choice regarding head trauma, in order to detect brain injury. Skull radiograph is useful in young children with suspicion of head fracture, however it is not significant in order to detect brain injury.

7. Do not use sedatives or antiemetic drugs excessively in children with HT. The most adequate manner to protect children with vomiting recurrence is to allow them to rest and to provide oral fractionated hydration. When there is persistent vomiting situations together with dehydration, endovenous hydration may be performed, avoiding hyperhydration. Movement and transportation produce vomit. Parents should be made aware of it.

8. Try to provide parents with information regarding the necessity of fasting or diet fragmentation, in cases of children with persistent vomiting after head trauma.

9. Bear in mind the possibility of spanked child syndrome when dealing with cases of breastfed infants presenting lesions that are incompatible with the trauma history.

10. In newborns and breastfed infants, severe intracranial hemorrhages or extensive cephalohematomas may be the cause of shock, although it is less frequent in older children and adults.

**References**


