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Perioperative management of patients with traumatic brain injury

Julio Cruz-Santos, MD;* Jorge Froylán Cuenca-Dardón, MD**

* Intensive Anesthesiologist of the «Anesthesia Course on the Patient Major Polytraumatized». Hospital General y de Urgencias Traumatológicas «Xoco» SSDF.
** Intensive Anesthesiologist. Professor of the Postgraduate Course for Medical Specialist «Anesthesia on the patient major politraumatized». Department of Anesthesiology. Hospital General «Xoco», SSDF.

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SUMMARY

Objective: To review the current recommendations involved in the management of traumatic brain injury. **Material and methods:** To review in ovid data bases all the articles related with the management of traumatic brain injury between march of 2001 to march of 2006. **Results:** The traumatic brain injury has incidence of 2% in the world population, and is the principal cause of death and severe disability in young population. Represent the 26% of all the patient with multiple injuries. 75% of all the patients that died presented ischemic lesions. The incidence of intracranial hypertension in severed head injuries is above 53 a 63% with abnormal CT scan, and 13% with normal study. The time of the patient in the Emergency Room should be less than 10 minutes. The time employed in the first evaluation should be performed in 120 seconds. The cardiopulmonary resuscitation algorithm: A: airway control with stabilized of cervical spine. All patient with severe head injury and Glasgow coma scale < 8 should be intubated. A rapid sequence intubation is recommended to avoid increases in intracranial pressure. B: breathing: a «low» normocapnia is recommended, maintain $PaCO_2$ between 4 to 4.5 kPas. The hyperventilation to low levels is associated with worst prognosis. Meanwhile the PaO_2 should be > 13 kPas. C: circulation, is maintained above 90 mm Hg with fluids and inotropics. The principal objective is to keep the euvolemia. The permissive hypotension is not indicated in severe head trauma. D: neurologic deficit, evaluating the pain response and the unconsciousness state. Exposure: to search other lesions. The following of these recommendations is associated with better outcomes, and can not be substituted for sophisticated therapies in critical care. The platinum 10 minutes and golden hour concepts are still valid in the management of these patients. The intracranial hypertension and diffuse axonal lesions are the principal causes of death in patients with severe head injury. **Conclusions:** The traumatic brain injury is one of the principal causes of death and disability in population in productive age. The injury mechanism and the following of the principles of reanimation are related with the outcome and good prognosis in patients with severe head injury.

Key words: Head injury, intracranial hypertension, hypocapnia.

RESUMEN

Objetivo: *Revisar las recomendaciones actuales involucradas en el manejo de los pacientes con traumatismo craneoencefálico.* ***Material y métodos:*** *Revisión en las bases de datos del sistema ovid de todos los artículos relacionados al manejo del traumatismo craneoencefálico a partir de marzo del 2001 hasta marzo del 2006.* ***Resultados:*** *El TCE tiene una incidencia anual del 2% de la población y constituye la causa más importante de muerte y discapacidad*

severa entre la población joven. Es la causa de defunción del 26% de los pacientes politraumatizados. Hasta 75% de los pacientes fallecidos por TCE presentaron lesión cerebral por isquemia. La frecuencia de hipertensión intracraneana en los traumas severos es de 53 a 63% con TAC anormal y 13% TAC normal. El tiempo ideal en emergencia deberá ser menor de 10 minutos, realizando una primera revisión en no más de 120 segundos. Imponiéndose el algoritmo de la reanimación cardiopulmonar: A. Vía aérea permeable con control de la columna cervical. Todo paciente con lesión de cráneo y ECG < 8 debe ser intubado. Recomendándose una inducción de secuencia rápida para evitar incrementos en la presión intracraneana que puedan ser fatales. B. Respiración: ventilación-oxigenación. Debe optimizarse a una normocapnia «baja», es decir una $PaCO_2$ de 4-4.5 kPa (30-34 mmHg). La hiperventilación a niveles más bajos de $PaCO_2$ se asocia con peores pronósticos, mientras que se debe mantener una PaO_2 mayor de 13 kPa (98 mmHg). Se recomienda el uso de oximetría de pulso y capnografía en lo que se encuentran disponibles los gases arteriales. C. Circulación: se mantiene a base de líquidos e inotrópicos para mantener una PAM de 90 mmHg. El objetivo inicial del tratamiento de un paciente con lesión cerebral con o sin trauma es el mantenimiento de la euvolemia. El concepto de «hipotensión permisiva» no se aplica en los casos asociados a TCE severo. D. Deterioro neurológico: evaluación de la respuesta al dolor e inconsciencia. E. Exposición completa del paciente para buscar y tratar lesiones asequibles con control y tratamiento de la hipotermia. Se ha reconocido que el seguimiento de estos principios básicos es vital para un buen pronóstico y no pueden ser sustituidos por sofisticados tratamientos de cuidados críticos o balas mágicas para detener el efecto de la lesión inicial. Estos son los «10 minutos de platino» y la «hora dorada» de la intervención, la hipertensión intracraneana y la lesión axonal difusa las principales causas de la defunción de estos pacientes. ***Conclusiones:*** *El TCE es una de las principales causas de muerte y discapacidad en personas en edad productiva. El mecanismo de lesión y el seguimiento de los principios básicos de la reanimación son factores muy importantes relacionado con el buen pronóstico de estos pacientes.*

Palabras clave: *Traumatismo craneoencefálico, hipertensión intracraneana,*

Accidents are the leading cause of mortality during the first four decades of life and constitute 7% of all deaths[1].

Thus we have that deaths secondary to trauma may be due to the following reasons[1]:

1. 10% patients died *in situ* by lesions incompatible with life.
2. 75% of patients died in the early hours.
3. 15% of patients died a week by multiorgan failure.

The traumatic brain injury (TBI) is defined as a head injury with the presence of at least one of the following: alteration of consciousness and/or amnesia due to trauma, neurological or neurophysiological changes or diagnosis of skull fracture or injuries attributable to trauma; the occurrence of death resulting from trauma including diagnoses of head injury and/or traumatic brain injury among the causes that resulted in death[2].

The TBI has an annual incidence of 2% of the population and is the leading cause of death and severe disability among young people[3]. It is the cause of death of 26% of polytraumatized patients. Up to 75% of patients who died by TBI presented brain injury by ischemia. The frequency of intracranial hypertension in severe trauma is 53 to 63% with abnormal CAT and 13% with normal CAT[1].

Kinematics. Based on the mechanism, TBI is classified in[4]:

I. Open mechanism: defined by the penetration of the dura mater commonly due shrapnel or injuries, it is associated with a higher mortality compared with the closed mechanism (88 vs 32%)[5], there are differences between the properties of military and civilian weapons determining the difference in the magnitude of the injury. The projectiles from military weapons are of high energy and can reach between 600 and 1500 m/sec, while the civilian weapons are of low energy and do not exceed 180 m/sec. On the other hand, the explosive splinters can reach nearly 900 m/sec[6]. The distance at which a low-energy projectile is fired plays an important role: a short distance can enter and exit the skull[4]. Once inside of the skull, run may be irregular y have deviations of its pathway, injuring multiple structures. Part of the projectile energy is absorbed by bone and the remain-

ing energy determines the degree of brain injury. The shock of brain tissue generates an expansive lesion often causing a lesion away from the projectile path, thus it forms a transient cavity higher with a diameter higher than one of bullet lasting for milliseconds, producing bleeding along the path of the projectile. Also contusion areas, cerebral edema, hemorrhage and haematoma are generated. Death occurs by the sudden and significant elevation of intracranial pressure[4].

II. Closed mechanism: being the motor vehicle accidents the most frequent cause of it. Falls and direct trauma have a lower incidence. The generation of acceleration-deceleration forces produces tangential forces in the brain, causing diffuse axonal injury that is characterized by loss of consciousness. Furthermore, the acceleration forces may cause contusion and damage to the brain tissue. The severity of diffuse injury determines the duration and depth of loss of consciousness and post-trauma amnesia. Direct trauma may cause fracture of the skull, lesion of meninges or vessel therefore, and generate the formation of epidural haematoma[4].

The presence of cranial vault fracture forces to reject the presence of intracranial haematoma[7]. One of the factors determining higher severity of the injury is the fact that the cerebrospinal fluid (CSF) is 4% more dense than brain tissue and acts as a shock. At the moment of trauma, CSF moves in the direction of the stroke in front of the brain. If the deceleration force is sufficient will cause to the brain moves in the opposite direction to the hit and CRL and impacts against the skull. The contrecoup injury is more frequently located in the frontal lobes, specifically in the orbital frontal surface and in the anterior of the temporal lobes[4].

The TBI can be classified based on the Glasgow Coma Scale (GCS) in[1]:
Mild: Glasgow from 13 to 15 points.
Moderate: Glasgow from 9 to 12 points.
Severe: Glasgow 8 points or less.

The time of the patient in the Emergency Room should be less than 10 minutes, making an initial review at no greater than 120 seconds. Imposing the cardiopulmonary resuscitation algorithm[1]:

A. Permeable airway with control of the cervical spine. All patient with head injury and Glasgow coma scale < 8 should be intubated. A rapid sequence intubation is recommended to avoid increases in intracranial pressure that can be fatal[4].
B. Breathing: Ventilation-oxygenation. It must be optimized to a "low" normocapnia, ie a $PaCO_2$ of 4-4.5 kPa (30-34 mmHg). The hyperventilation to low levels is associated with worst prognosis [9], meanwhile a PaO_2 greater than 13 kPa (98 mmHg) must be maintained[6]. It is recommended the use of pulse oximetry and capnography in what is available arterial blood gases[11].
C. Circulation: It is maintained on the basis of liquids and inotropic agents to maintain a MBP of 90 mmHg[8]. The main goal of treating a patient with brain injury with or without trauma is the maintenance of euvolemia. The concept of "permissive hypotension" is not applicable in cases associated with severe TBI[6].
D. Neurological impairment: evaluation of response to pain and unconsciousness.
E. Full presentation of the patient to find and treat approachable injuries with control and treatment of hypothermia.

Has been recognized that the follow-up of these basic principles is vital for a good prognosis and they can not be replaced by sophisticated critical care treatments or magic bullets to stop the effect of the initial injury. These are the "platinum 10 minutes" and the "golden hour" of the intervention[6].

To understand the TBI management is important to understand some pathophysiological mechanisms. Edema formation is common after the initial brain injury, considering both the vasogenic (because of decreased blood-brain barrier permeability and extravasation of fluid) and cytotoxic edema (as a result of a massive increase in osmolarity due to the rupture of cell structures and loss of cellular ability to regulate their ionic gradients[7]).

The primary or initial injury causes neuronal damage and death. Caused damage can be from simple hematomas to more complex and diffuse lesions. In any event, a cascade of molecular events leading to secondary injury is triggered, including the effects of hypoxia, release of endogenous excitatory amino acid, production of proinflammatory substances and free radicals, which results in more destruction[11].

It should be noted that the cranial vault is a rigid structure and that the edema and hemorrhage increase the intracranial pressure to critical levels. There are several mechanisms attempting to compensate this increase. All these mechanisms are associated with cerebrospinal fluid causing a decrease in its production, increase in its absorption or diversion of circulation. However, once these compensatory mechanisms become unable to compensate the increase in intracranial pressure, reducing cerebral perfusion and possibly causing cerebral ischemia. If it perpetuates, the cycle can result in irreversible neurological damage[13].

The most recent studies have suggested the possibility that mitochondrial failure plays a crucial role in triggering some of the mechanisms of injury and production of proinflammatory substances and generation of free radicals, in-

ducing a more destructive chain reaction with spread of brain damage after severe brain TBI[14].

The increase in intracranial pressure (ICP) can move the brain to areas of high pressure to those of low pressure, thus presenting the various syndromes of herniation. The most common and greater clinical significance herniations are: uncal, transtentorial and infratentorial herniation. After all, these herniations result in brain compression, bradycardia, hypertension and disturbances of breathing resulting in apnea[12].

Brain injury in humans is a heterogeneous disease with different pathological processes: ischemia, excitotoxicity, energy failure, neuronal death cascade, inflammation and brain edema[12].

A good neurological evaluation is essential in patients with severe TBI, this based on the repeated application of the GCS and the pupillary diameter[4]. Some clinicians prefer to use the recommendations of the APACHE II in which the score of 1 is assigned for absence of verbal response, and score of 5 is assigned for a normal verbal response, and score of 5 is assigned for questionable verbal response[15].

Although there is a very reasonable correlation between the severity of the coma and the risk of intracranial hypertension, the ECG has been accepted as a simple way to estimate the prognosis of a patient. The funduscopy is not very reliable for identifying intracranial hypertension, especially in acute conditions, because the signs appear late[15].

The evaluation of patients with severe TBI usually starts with a tomographic study of the skull as an emergency procedure for[16]:

- Identifying intracranial lesions that may require surgical correction.
- Identifying obstructions to the flow of cerebrospinal fluid (hydrocephalus).
- Appreciating the severity of brain edema and possible brain damage.
- Performing a diagnosis.

In 1991, Marshall et al.[19] developed a system now widely used to classify head injuries according to the changes observed in computed tomography:

I. Diffuse lesion without visible pathology: No demonstrable intracranial pathology in tomographic study.
II. Diffuse lesion: Cisterns are observed, there is a deviation from the midline of 0-5 mm. No lesions of high or mixed density > 25 mL.
III. Diffuse lesion: There is partial compression or absence of the cisterns of the base with a deviation from the midline of 0-5 mm. No lesions of high or mixed density > 25 mL.
IV. Diffuse lesion: There is deviation from the midline > 5 mm in the absence of basal cisterns. No lesions of high or mixed density > 25 mL.
V. Evacuated lesion: Any surgically removed lesion.
VI. Non-evacuated lesion: Any not surgically removed lesion of high or mixed density > 25 mL[18].

Magnetic Resonance is being increasingly used to better assess the injuries, including injuries of posterior fossa.

INTRACRANIAL PRESSURE MONITORING

The intracranial pressure monitoring is indicated as a general rule in patients with ECG <8; however in these patients the obtained results in the CAT can influence the decision. In patients with normal CAT, monitoring must be considered in the following cases[19].

- Age > 40 years.
- unilateral or bilateral motor deficit.
- Systolic blood pressure < 90 mmHg.

And to some extent, the need to sedate the patients can influence this decision.

The standard method of monitoring is the placement of an intraventricular catheter[19]. Although there are risks of erroneous placement, local bleeding, obstruction and especially infection; intraventricular catheter allows drainage of cerebrospinal fluid, by which diagnosis and treatment can be carried out[19]. An alternative is the placement of catheters with fiber optic or needle-type transducer, these catheters have the advantage of easy placement, but are expensive. The epidural and subarachnoid-type catheters have less risk of infection; however, are more prone to artifacts[19]. The benefit of prophylactic antibiotic use has not been demonstrated by randomized controlled studies; however, a retrospective study showed no effect on infection rates[20]. Normal intracranial pressure is <15 mmHg.

JUGULAR VENOUS SATURATION

The jugular venous saturation is generally less than mixed venous blood saturation, whose normal value is of approximately 65%, consequently a saturation <60% indicates an inadequate cerebral blood flow in relation to the requirements of oxygenation of the brain, providing useful information on global cerebral oxygenation[21]. Therefore the placement of a catheter at the venous bulb allows to assess this parameter. In the case of a low SjO_2, management with liquid or vasopressor agents must be considered in order to increase MAP and CPP. But also other conditions such as hypoxia, hypocapnia and high ICP must be discarded[13].

MEASUREMENT OF CEREBRAL BLOOD FLOW

Cerebral blood flow can be measured by the Kety-Schmidt method, dilution with X^{133}, the jugular thermodilution, or more sophisticated methods such as angiography - magnetic resonance or positron emission tomography, although these methods are not commonly used. The transcranial Doppler allows evaluation of flow velocity, but without estimating the size of the vessel, accordingly it can not assess directly the CBF. However, it is particularly useful for assessing vasospasm[13].

LOCAL CEREBRAL OXYGENATION AND METABOLIC MEASUREMENTS

Monitoring of local brain tissue oxygenation and measurements of local metabolites are relatively recent developments in monitoring patients with severe TBI and may be particularly useful in identifying cerebral ischemia and evaluating the effectiveness of established treatment[13]. Generally devices implanted in the brain parenchyma are used; these devices obtained, through microdialysis, measurements of lactate, pyruvate and markers of inflammation, although these data are dependent on the site of catheter placement and still at an experimental stage[13].

EVOKED POTENTIALS

Evoked potentials are electrical signals generated by the nervous system in response to sensory stimuli and ever more frequently used to assess neurological damage in severe TBI. The median nerve is the most commonly used for this evaluation by applying short electrical stimuli, while responses are recorded by EEG electrodes of contralateral sensory field[13].

MANAGEMENT OF INTRACRANIAL HYPERTENSION

There are two very important concepts related to the management of intracranial hypertension:

1. The Monroe-Kellie principle states that the volume inside the skull is the sum of the brain, CSF and blood flow. This volume alters by pathological processes increasing the amount of any of these components (hydrocephalus, for example) or the appearance of a new (tumors). None is really understandable and if the volume of one increases, the other two should do a space, so the decrease in ICP can be achieved by reducing the space occupied by any of the other components[13]:
   - Decreased brain size (usually by edema): mannitol and other hypertonic substances.
   - Decreased CSF: drainage.
   - Decreased amount of blood: hyperventilation to achieve vasoconstriction.
   - Surgical removal of pathological processes (tumors, haematomas).
2. Rosner's conjecture. Which says that the brain lesions secondary to ischemia are a consequence of systemic (hypotension, hypoxemia, fever and hypothermia) or brain factors (high CIP, low CPP, edema, lesions occupying space and seizures[13].

EARLY REVIVAL

The BIV mnemonics covers the basic principles of management of these patients[22].

Ventilation: the airway must be ensured using mechanical ventilation if necessary. Hypoxaemia worsens the prognosis, and oxygen administration must be generous to maintain $SpO_2 \geq 95\%$. The routine use of hyperventilation is not indicated, as the decrease in the CBF can worsen ischemic injury; it has been shown that even short-term hyperventilation may induce sosme degree of cerebral ischemia[23]. The $PaCO_2$ must be maintained around 35 mmHg and hyperventilation must be reserved for cases with brain herniation in which the decrease in the flow is seen as a priority to avoid an excessive increase of the ICP[24].

Infusion: as a rule, the hypotension is attributed to hypovolemia and must be corrected with fluid administration and control of the cause. Both Hartmann and normal saline fluids may be used as first line fluids.

Pump: hypoxemia and hypotension are the main enemies to be overcome during resuscitation of severe TBI. If the TBI is severe, systolic blood pressure must be maintained > 120 mmHg (MAP > 90 mmHg)[26]. When persistent hypotension despite the administration of fluids, vasopressor and inotropic agents such as dopamine or norepinephrine may be combined in order to maintain blood pressure. But they must be used with caution, because vasoconstriction may alter the local CBF despite the improvement in CPP. The vasopressor of choice in these conditions is the norepinephrine[27].

Another aspect to consider in the early resuscitation is the position of the head; it must be raised on a routine basis at 30° to improve venous return and decrease the ICP[28]. A higher elevation may decrease the CBF and CPP. In severe hypotension, CBF and CPP must be a priority and the patient must remain in neutral position[29].

CONTROL OF INTRACRANIAL HYPERTENSION

There are three steps for the management of intracranial hypertension, continuing to the next step only if the pressure remains uncontrolled.

First step: if the pressure remains <20 mmHg, mild hyperventilation is initiated or hypocapnia must be avoided at least, with a $PaCO_2$ around 35 mmHg. The decreased $PaCO_2$ associated with hyperventilation increases the pH of CSF, causing vasoconstriction, which increases cerebral vascular resistance, decreases blood flow and cerebral volume and ICP. If there are ventricular drainage systems, CSF must be drained when the ICP increases above values of 15 to 20 mmHg[30].

Second step: The use of mannitol in ranges of 0.25 to 0.5 g/kg or 7.5% hypertonic saline at a rate of 2 mL/kg body is indicated. So far there is no reason to prefer one over another, however the intermittent bolus administration is more preferable than continuous infusion[31]. On the other hand, the plasma osmolality must not exceed 320 mOsm/kg because the risk of kidney failure increases[31]. Recent randomized controlled studies have suggested that early use of high doses of mannitol (1.4 g/kg) may be more effective than low doses in controlling the spontaneous intracerebral hemorrhage (ICH) and improving forecasts in certain groups of comatose patients with severe TBI[32], although more studies are still needed to define the type of patients who may benefit from this therapy. Hypertonic saline solution reduces ICP without affecting the hemodynamic status[33] and can have a beneficial effect on the excitatory neurotransmitters and the immune system[34].

Hyperventilation can be used (by increasing minute ventilation) to keep $PaCO_2$ initially around 30-35 mmHg (in severe cases 28-30 mmHg), however a decrease <25 mmHg is potentially dangerous due the reduction of cerebral blood flow and the risk of ischemia. Hyperventilation can be used more aggressively to control ICP in conjunction with monitoring the SjO_2[35].

Step Three: If ICP remains high, there are two additional options that can be used individually or in combination: barbiturates and decompressive craniectomy. Although there no controlled clinical studies have been performed to assess the effect of barbiturates and their role in the prognosis of patients, there are certain facts that support its use[13]:

- The ICH is associated with a poor prognosis, especially if the CPP can not be maintained.
- Barbiturates decrease ICH, decreasing the metabolic oxygen consumption and therefore the CBF and the brain volume, they may also limit cell injury mediated by free radicals.

Used barbiturates should be short term such as sodium thiopental, a loading dose of 5-10 mg/kg followed by an infusion at a rate of 3-5 mg/kg/h. Knowing that the use of this drug has side effects such as reducing blood pressure, the use of fluids or vasopressor or inotropic agents must be carefully monitored[13]. With regard to unilateral or bilateral decompressive craniectomy, no controlled studies have been performed, there are only case reports and personal experiences that have suggested that patients undergoing early craniotomy may have a better prognosis[35].

ADDITIONAL MEASURES

Control of seizures. The seizures complicate 20% of cases of severe TBI, so it is advisable to use prophylaxis, but does not prevent seizures in the long term, and it is not recommended to extend thje prophylaxis beyond one week after the injury[36]. Phenytoin is the most widely used drug, although some clinicians prefer the use of carbamazepine.

Control of hypertension. The hypertension may be occurred and is faced with a risk of worsening the edematous lesions by excessive intravascular pressure, especially if the self-regulation is altered. Hypertension should be treated if the MAP is> 120 mm Hg. If pharmacological intervention is required, beta-blockers are the drug of choice (if there is no contraindication to its use), as they does not increase ICP, which is the potential problem with vasodilator agents such as nitroprusside and hydralazine. However not yet defined the ideal drug[37].

Nutritional support. Should be initiated without delay, preferably by enteral route. All patients with TBI have increased caloric and protein needs. A heat load of 25 kcal/kg is tolerated in an appropriate manner[38].

Complications must also be prevented and treated. Sepsis is a common complication and should be handled appropriately; as well as prophylaxis must be used in stress ulcers. Heparin administration is usually contraindicated in early trauma. Although a recent retrospective study showed no difference in bleeding episodes among patients with severe TBI who received heparin within the first 72 hours of the event and those who received 72 hours after injury[39].

Hyperglycemia may have deleterious effects on brain function. Unless it is known that the patient is at risk of hypoglycemia, solutions containing glucose must be avoided in the initial stages of resuscitation in order to minimize the risk of hyperglycemia, requiring the administration of insulin to keep blood glucose <150 mg/dL. On the other hand, hypoglycemia should be corrected as soon as detected[40].

Fever (core temperature > 38ºC) should be treated aggressively, as it increases cerebral metabolism and vasodi-

lation. Antipyretic agents and cold blankets must be used. Intravascular cooling techniques can facilitate the reduction of temperature[41].

Steroids have not shown improvement in the prognosis of patients with TBI. The CRASH study -a randomized, controlled- showed no reduction in mortality at 2 weeks, so now it is not recommended to use steroids in these patients[42].

In conclusion, the prognosis of a patient with severe traumatic brain injury will depend on several factors: the type of injury, the initial care, the control of intracranial pressure, adequate detection and treatment of complications. Therefore, coordination between a team of specialists is needed in the management of these patients such as emergency physician, radiologists, neurosurgeons, anesthesiologists and internal medicine specialists and others.

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