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REVIEW ARTICLE

Advances in the management of head injury

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Head injuries have a profound societal impact given that they are the leading cause of death in persons under 45 years of age and the third leading cause overall. To this, we must add the family, social, and financial burdens of survivors who suffer sequelae. Most pathophysiologic changes in cerebral metabolism and blood flow occur in the first few hours after injury. Early management by emergency services and in hospital emergency departments therefore plays a key role in determining prognosis. Although the primary injury cannot be undone, we can limit its progression by minimizing secondary brain damage. From the moment of first response to the patient, once basic resuscitation measures have been taken, the main goal should be to stabilize the patient, especially preventing hypotension and hypoxia, which are independent risk factors for morbidity and mortality. Patients with severe head injury must be transported in stable condition to a tertiary care hospital with a neurosurgery department and an intensive care unit that can monitor such neurologic variables as intracranial pressure and cerebral oxygenation. This review of head injury management addresses 2 aspects that are essential for treatment. We first describe pathophysiologic concepts that influence cerebral blood flow and metabolism and intracranial pressure, and we also cover types of primary and secondary injuries. We then discuss evaluation and management more thoroughly, including measures in prehospital and emergency department settings, as well as intensive care unit treatment involving multimodal neurologic monitoring techniques. [Emergencias 2009;21:433-440]

Key words: Head injury Pathophysiology. Multimodal nerve stimulation.

Introduction

More than one million patients per year in the U.S. visit emergency services with traumatic brain injury (TBI)[1]. Eighty percent are considered slight, with a Glasgow Coma Score[2] (GCS) of 14-15 after resuscitation. Moderate TBI (GCS = 9-13) occurs in 10% of patients and the remaining 10% have severe head injury (GCS ≤ 8). Each year in the U.S., 52,000 patients die and 70,000-90,000 are left with neurologic sequelae after traumatic brain injury[3].

All this, in addition to the cost of human lives and resulting social problems, involves costs to society estimated at approximately 3,000 million dollars[3]. TBI is therefore a serious public health problem, requiring preventive solutions and specific treatment.

From the point of view of emergency department physicians, their mission is to detect patients with moderate or severe TBI, proceed with stabilization, prevent secondary brain damage (mainly hypoxia and hypotension), and transport the patient in the best possible condition to a hospital able to offer definitive care. A better understanding of the pathophysiological changes in TBI obtained in the last two decades has highlighted that, although we can not act on the primary lesion produced at the moment of impact, the development of further damage can be avoided in the following minutes, hours or days. This brain damage, called secondary, may lead to increased mortality and severe disability, as demonstrated in the epidemiological study carried out by the Brain Trauma Foundation in 1995[4].

Mortality due to severe head injury has declined from 50% to 30% in the last 30 years, without increasing the number of patients with serious neurological sequelae[5,6]. The reason for this decline is largely due to improved response by pre-hospital emergency services, based on better knowledge of the pathophysiology of TBI and improvement in the specific care and treatment later provided in hospitals[8].

Prognostic data for these patients have been analyzed and many variables identified that determine prognosis, such as the mechanism of traumatic injury, age, pupil reactivity, the GCS score after resuscitation and the type of lesion as shown by neuroimaging[9].

Concepts of pathophysiology in head injury

Alterations in cerebral blood flow

Thanks to the peculiarities of the arterial circle of Willis, with its numerous collateral pathways, cerebral blood flow (CBF) is maintained constant despite fluctuations in mean arterial pressure (MAP), provided this is in the range of 60-140 mmHg. CBF is calculated as the quotient of cerebral perfusion pressure (CPP) with respect to cerebral vascular resistance (CVR).

According to clinical practice guidelines[8], cerebral perfusion pressure [CPP = MAP – intracranial pressure (ICP)] should be maintained between 60-70 mmHg.

CVR also influences CBF, and more specifically arteriolar reactivity to $paCO_2$ changes. This has very important therapeutic implications; a large decline of $PaCO_2$, although reducing ICP by arteriolar vasoconstriction, can compromise cerebral blood flow and oxygenation, resulting in cerebral ischemia, especially in the early hours of TBI when blood flow is weaker[10,11].

Alterations in intracranial pressure

The intracranial vault consists of four components: brain parenchyma, interstitial area, blood and cerebrospinal fluid (CSF). Thus the total intracerebral volume is the sum of them all. This implies that under normal conditions and to maintain physiological balance, with IPC values between 10 ± 5 mmHg, any alteration in the volume of one component implies a similar alteration in volume of the rest. This is called intracranial compliance, which reflects the relationship of volume changes with respect to the resulting change in ICP (Figure 1).

Alterations of cerebral metabolism

Cerebral metabolic rate of oxygen consumption ($CMRO_2$) equals the product of CBF and the difference in arterioyugular oxygen. When CBF declines, $CMRO_2$ initially remains within the limits of normality at the expense of increasing oxygen consumption (the difference in arterioyugular oxygen increases). But if the flow continues to decline, the compensatory effect is insufficient and ischemia appears.

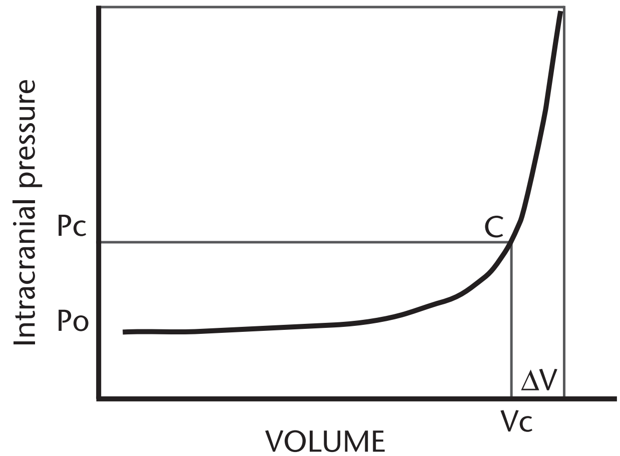

Figure 1. Cerebral compliance curve (C = critical point; Po = Initial pressure, Pc = critical pressure, Vc = critical volume; ΔV = volume increment).

Types of structural lesions

Two main types of lesions have been described. Primary damage occurs immediately after impact. The impact may cause focal or diffuse lesions; the latter includes diffuse axonal injury (DAI) by direct axonal rupture, and swelling, where axonal damage occurs after some hours or days. Clinical manifestations range from concussion (i.e. unconsciousness during less than 6 hours) to severe DAI (i.e. unconsciousness for more than 24 hours with signs of brainstem dysfunction). Then there is secondary damage leading to loss of cerebrovascular self-regulation, blood-brain barrier alterations, intra or extracellular edema and ischemia. This occurs in response to the primary damage and some systemic events[12]. Depending on etiology, the following should be noted:

1. Intracranial Complications: vascular lesions (intracranial hematoma, vasospasm, revascularization events, and cerebral venous thrombosis), hydrocephalus due to imbalance between production and absorption of CSF, intracranial infection, epilepsy and cerebral edema.

2. Systemic causes: In this type of secondary brain damage, intervention by emergency teams at the scene of the accident and those in hospitals can play a vital role and help reduce the morbidity and mortality of these patients.

2.1. *Blood pressure:* Low blood pressure increases severe TBI mortality from 27% to 50%[13-18]. The correct MAP values are 80-100 mm Hg. On the other hand, hypertension (HT) also increases ICP when self-regulation is lost.

2.2. *Positive end expiratory pressure (PEEP):* This may increase ICP through decreased systemic ve-

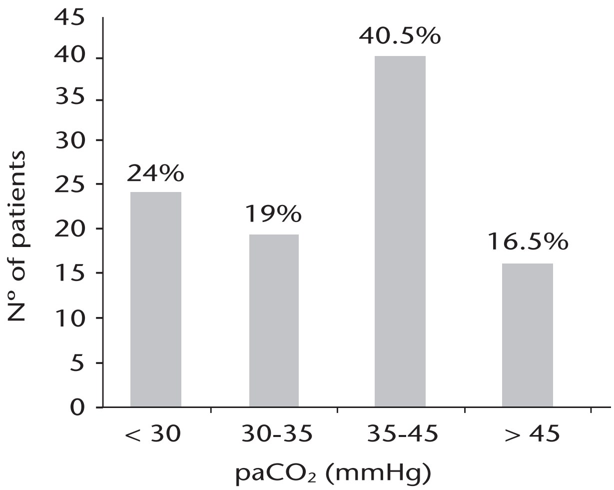

Figure 2. Pre-hospital mechanical ventilation in 75 patients with severe head injury: $paCO_2$ values determined on arrival at the hospital.

nous and cerebral return. Recommended PEEP values are 5-10 cmH_2O. However, risk/benefit must be weighed, because if we do not get optimal PaO_2 with increased FiO_2, we resort to increased PEEP.

2.3. *Hyperthermia:* This causes increased brain metabolism, but unless prolonged, fever has not been shown to influence the prognosis of TBI patients.

2.4. *Exchange of gases:* According to results of the Traumatic Coma Data Bank, hypoxemia increases the mortality rate by approximately 2%, and 25 times more so when associated with hypotension[19]. Changes in $PaCO_2$ have a profound impact on CBF due to vasoconstriction. Hyperventilation can be used therapeutically in the absence of ischemia, seeking to maintain values of 33-37 mmHg. Lower values are dangerous because of ischemia due both to hypocapnia vasoconstriction and the Bohr effect (influence of CO_2 on the oxygen dissociation curve of blood). In a study performed by our group in the hospital emergency department (ED), we found that a high percentage of patients receiving mechanical ventilation at the accident site had, on arrival at the ED, hypocapnia values compatible with cerebral ischemia and less than half had $PaCO_2$ values within the normal range (Figure 2).

2.5. *Hyperglycemia:* this contributes to brain damage by increasing osmolarity with consequent osmotic swelling and at the same time promoting metabolic acidosis.

2.6. *Hypernatremia/Hyponatremia:* Hypernatremia can result from diabetic coma, dehydration, diuretics, diabetes insipidus, panhypopituitarism or treatment with hyperoncotic hypertonic saline which produces cellular dehydration. Hyponatremia is usually secondary to excessive hypotonic fluid intake in the acute phase.

Assessment and management of TBI

Initial assessment of severe brain injury

A severe brain injury in isolation is an unlikely finding: over 60% of TBI are associated with severe injury of another organ[20]. Recent multicenter data[6] show that the association of severe TBI with systemic damage ranges between 25% and 88% of cases. Moreover, the rachis is frequently involved in multiple trauma patients and therefore inadequate movement may trigger or aggravate a spinal cord injury.

It is for these reasons that all patients with severe TBI should be considered multiple trauma patients until proven otherwise and evaluated and treated as outlined in the Advanced Trauma Life Support (ATLS) guidelines[21]. After resuscitation and stabilization, use of the GCS remains the best tool for evaluating trauma severity, although it should be noted that the GCS is not a neurological examination, but measures the responsiveness of patient to a stimulus. Currently, the prognosis of patients should not only be based on the GCS or CT scan on arriva, but also the motor response to stimuli (motor GCS motor), pupil reactivity, age, pre-TBI condition, and especially the degree of secondary brain damage during the acute phase[22].

Prehospital stabilization of TBI patients

Regarding TBI, after performing advanced life support (ALS) procedures, the isolation of the airway is necessary in all patients with GCS < 8 points and all moderate TBI with associated injuries or agitation that require sedation[23-25]. Orotracheal intubation (OTI) and Sellick's technique (for possible basilar skull fracture and spinal injury) should be performed. Sedative-analgesic regimes[26-28] are outlined in Table 1. The concentration of oxygen (FiO_2) should be that required to achieve arterial oxygen saturation ($SatO_2$) higher than 95%. Special care must be taken with manual ventilation which may

Table 1. Sedation and analgesia regimes

Morphine chloride	Bolus 5 mg/iv	Cp: 5-10 mg/h/iv
Fentanyl	Bolus 1-2 mcg/Kg	Cp: 1-1.5 mcg/Kg/h
Remifentanil	Bolus 1 mcg/Kg	Cp: 0.5-1 mcg/Kg/min
Propofol	Bolus 0.5-4 mg/Kg	Cp: 0.125-0.25 mg/Kg/min
Midazolam	Bolus 0.1-0.2 mg/Kg	Cp: 5-40 mg/h/ev

Cp = continuous infusion.

cause hypocapnia. MAP must be maintained at 90 mmHg and isotonic solutions infused, not hypotonic (Ringer lactate or glucose). If these measures fail, and other lesions are ruled out, infusion with norepinephrine is recommended, as it has no effect on CBF. Finally, any multiple trauma patient with severe, moderate or mild TBI should be transferred to a third level hospital centre.

Management of TBI in the emergency department

Systemic parameters should be evaluated, using hematological and biochemical tests, EEG, radiography of the skull, neck, chest, spine and pelvis. All patients with moderate or severe TBI should receive CT brain scan, and cervical scan, since plain X-ray may fail to reveal fractures of vertebrae or facet joints in as many as 60% of cases. In addition, abdominal ultrasound or CT should be performed, depending on physical examination findings[25]. Cranial CT should be repeated if there is a fall of two points in the GCS not attributable to extra-cranial causes or if there are changes in motor or pupil response.

Once severe head injury is confirmed, short-acting sedation-analgesia should be administered, to create neurological windows if necessary. For manoeuvres (related with chills or aspiration of secretions) that may increase IPC, neuromuscular blockers (cistracurio, rocuronium or vecuronium) should be administered.

In patient management, the fundamental principle is to try to maintain overall equilibrium. This will require strict control of the following general measures:

1. Avoid cerebral hypoxia: The objective is to maintain PaO_2 between 80 and 120 mmHg and $PaCO_2$ between 35 and 38 mmHg. Do not use prophylactic hyperventilation as this increases cerebral ischemia.

2. Control of cerebral compliance: the head of the patient should be inclined at 30 degrees above the horizontal plane in the absence of cervical lesions that contraindicate this position. Avoid endotracheal tubes around the neck, and ensure neck-chest alignment.

3. Treatment of edema: prophylactic treatment with osmotic diuretics is contraindicated.

This would be correct in pre-hospital and/or in the emergency department, if pupil or motor response changes appear or if GCS deteriorates. The drug most commonly used initially is 20% mannitol[29]. The dose should be the minimal effective dose, ranging from 0.25 to 1 mg/kg/iv and not exceeding 6 g/kg/day. Initially, mannitol increases plasma volume and reduces blood viscosity produced by increased CBF; this effect is followed by cerebral vasoconstriction and decreased CBF.

Table 2. General measures in the management of head injury in the emergency department

Head position	30º
PaO_2	90-120 mmHg
$PaCO_2$	35-38 mmHg
Mean arterial pressure	90 mmHg
pH/lactate	7.3-7.45/≤ 2.2 mlosm/l
CVP	10-15 mmHg
Hb	≥ 10 mg/dl
Ion control	Non-hypotonic fluids
Glycemia	80-120 mg/dl/7.37
Temperature	36-36.5ºC

Currently, the most accepted osmotic agent, due to fewer side effects, is SSH. Generally, 7.5% saline solution with starch at 6% is administered, 2 ml/kg/iv in 10 minutes, which reduces ICP in a more intense and lasting manner than mannitol in equi-osmotic doses with volumes 6 times lower. In addition, SSH does not produce secondary vasoconstriction as mannitol does, nor severe polyuria.

Regarding the use of corticosteroids, prospective studies with random assignment have shown no benefit[30] and adverse side effects (GI bleeding, hyperglycemia, cerebral vascular thrombosis, immunosuppression).

4. Maintain adequate hemodynamic stability: the CPP recommended by the Brain Trauma Foundation[8] is 60-70 mmHg; values above 70 mmHg are associated with an increased incidence of acute respiratory distress syndrome (ARDS) and increased mortality[31]. Optimum MAP values should be higher than 90 mmHg. Initially hypotension should be treated with volume (crystalloid and colloid), but if this fails to restore the GPA, resort to amines such as norepinephrine. The technique of small volume resuscitation with SSH can be used in situations of TBI with shock[32]. Permissive hypotension is not permitted in multiple trauma with severe head injury[33].

5. Metabolic Control: primarily aimed at removing metabolic acidosis and hyperglycemic states. The aim is to achieve blood glucose levels in the range of 80-120 mg/dl, using regular subcutaneous or intravenous bolus of insulin.

6. Maintenance of normothermia (core temperature 36-36.5°C). Initially we resort to physical measures, with infusion of cold fluids or intravascular cooling methods.

7. Hemoglobin level: to ensure adequate cerebral oxygen supply, hemoglobin levels should be ≥ 10 mg/dl[34].

8. Anticonvulsants should not be routinely used. Their use may be justified for 7-10 days in patients with risk factors for early seizures[36]. The indications for use are depressed skull fracture, intracerebral or subdural sylvian hematoma, penetrating head

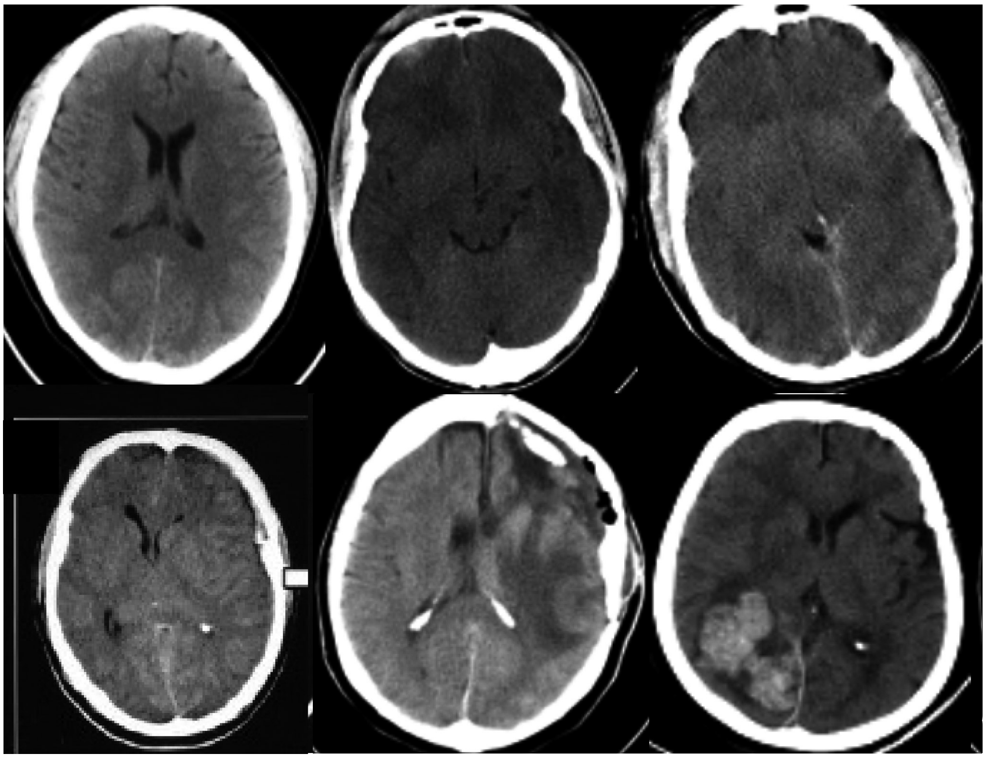

Figure 3. Marshall classification[38]. (DBI = Diffuse Brain Injury).

wound and seizure within 24 hours after injury. The drug of choice is phenytoin with a loading dose of 18 mg/Kg in 30 minutes, then after 24 hours followed by 1.5-2.5 mg/Kg/8h/iv.

9. Diagnostic imaging techniques: Brain CT is the technique of choice for diagnosis, prognosis and controlling initial TBI lesion progression[37]. According to the Marshall classification[38], the radiological patterns are:

9.1. *Diffuse brain injury type 1 (DBI I):* No intracranial pathology visible on CT.

9.2. *Diffuse injury type 2 (DBL II):* cisterns present with or without midline shift of structures (≤ 5 mm).

9.3. *Diffuse injury type 3 (DBL III):* The midline is almost centred (≤ 5 mm of displacement) but cisterns are compressed or absent, reflecting the presence of swelling.

9.4. *Diffuse injury type 4 (DBL IV):* Midline shift (≤ 5 mm) in the absence of focal lesions > 25 ml.

9.5. *Evacuated focal lesion (Marshall V):* Any surgically excised lesion.

9.6. *Non-evacuated focal lesion (Marshall VI):* Surgical focal lesions ≥ 25 ml.

10. Decision on hospitalization of patients with slight head injury. Slight TBI (GCS: 14-15 points) can be discharged with instructions for home neurological monitoring during 48 hours, in the absence of any of the following characteristics: decreased level of consciousness, headache, peritraumatic or focal neurologic amnesia, fractured skull, suspected intoxication, risk factors (age ≥ 70 years, anticoagulation, liver disease, stroke and/or previous TBI), living alone or far from the hospital, and inadequate intellectual level of companions. If any of the above features are present, a CT brain scan should be performed and the patient kept under observation for 48 hours. All moderate or slight TBI, with normal CT and without associated systemic damage, may be managed in a neurosurgery ward with CT scan performed 12-24 hours after admission. All patients with severe or moderate TBI and associated lesions, or slight TBI and potentially serious lesions, should be admitted to intensive care.

Management of TBI patients in the intensive care unit (ICU)

Before describing ICU management of TBI patients, we offer some basic advice on multimodal

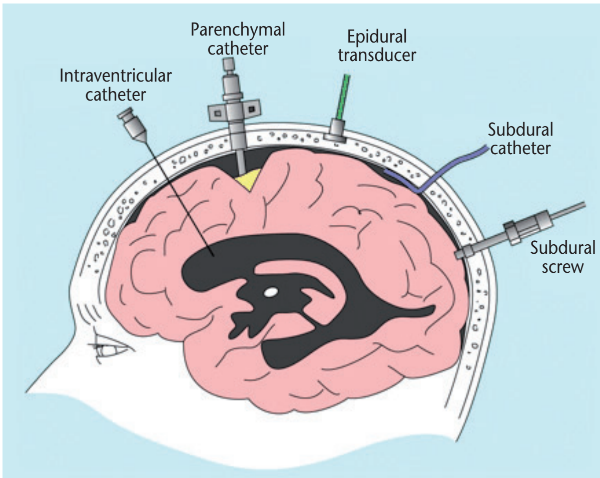

Figure 4. Methods of measuring intracranial pressure[13].

monitoring used in our ICU based on the Brain Trauma Foundation recommendations published in 2007[8].

Monitoring of cerebral perfusion

1. *Intracranial pressure monitoring:* usually performed with fibre optic systems located at the ventricular or parenchymal level. The most commonly used, due to lower risk of infection, are intraparenchymal catheters[39], inserted in the most affected hemisphere or, in the case of diffuse lesions, in the right hemisphere. Normal ICP values are 0-20 mmHg in adults. Clinical guidelines[4,8], 38 recommend monitoring of severe TBI with abnormal CT scan; severe TBI with normal CT scan and two or more of the following characteristics: age > 40 years, abnormal unilateral or bilateral motor responses abnormal (motor ≤ 4) and MAP < 90 mm Hg; space-occupying traumatic complex regardless of level of consciousness. Moderate TBI with non-surgical space-occupying lesion (SOL) or intra-axial compression of base cisterns. Moderate TBI with associated extracranial injuries require deep sedation and analgesics.

2. *Spectral transcranial Doppler ultrasound (TDU) and color coded TDU (CCDTU):* used to assess the state of the larger vessels of the arterial circle of Willis and therefore, cerebral blood circulation. They provide data on CBF velocity (systolic, diastolic and mean) and pulsatility index (PI) to detect the presence of vasospasm, intracranial stenosis and brain death, as well as the response to treatment[40].

Monitoring of oxygen extraction

1. *Jugular oximetry:* this assesses CBF n accordance with the values of mixed venous saturation (SjO_2) at the gulf of the internal jugular vein, which oscillates between 55% and 71%. Values of 50% indicate severe ischemia and values above 75% indicate hyperemia[41,42]. However, jugular oximetry is being replaced by tissue oxygen pressure.

2. *Tissue oxygen pressure ($PtiO_2$):* Carried out using a polarographic catheter inserted together with the intraparenchymatous ICP catheter in apparently healthy white matter. Continuous monitoring of $PtiO_2$ value indicates cerebral oxygen availability and indirectly the balance between oxygen supply and demand. Therefore, any increase in oxygen consumption (inadequate sedation, hyperthermia, seizures, etc.) or decreased CBF [hypotension, hypoxemia, hypocapnia, vasospasm, edema with intracranial hemorrhage (ICH), etc.] lead to declines in $PtiO_2$. Conversely, increased oxygen supply or a decrease in consumption lead to increased $PtiO_2$. Normal values are in the range of 15-30. Although the threshold of ischemia is not well defined, prolonged low values of $PtiO_2$ are related with poor prognosis[43,44].

Therapeutic measures

If these general measures fail and there is ICH not attributable to surgery, a second line of measures may be used, including ventricular drainage, osmotic diuretics and hyperventilation (Pa CO_2 of 30-35 mmHg).

Finally, last resort measures may be required, such as:

Drug Coma: induced by barbiturates. Furthermore, they suppress seizure activity and reduce cerebral oxygen consumption, the main effect being cerebral vasoconstriction. Given their serious side effects (hypotension, decreased CSF and depressed T cell activity, thus favouring the development of infection and septic shock[8,45]), the use of barbiturates is currently losing ground.

Decompressive craniectomy: with HIC refractory to the above-mentioned measures and excluding those cases with bilateral lesions or with paralytic bilateral mydriasis, extensive craniectomies have been performed on both frontotemporoparietal and occipital regions as well as duraplasty, with encouraging results[46-48].

Moderate hypothermia (32-34ºC): this is associated with immunosuppression, hypokalemia and severe coagulation disorders. Although not favoured by meta-analyses[49], there are methodological deficiencies in many of the studies reporting on this technique: new analyses of studies with appropriate methodology may revive interest in the possibility of using hypothermia once again[50].

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Avances en el traumatismo craneoencefálico

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El traumatismo craneoencefálico (TCE) produce un gran impacto en nuestra sociedad al ser la primera causa de muerte en personas menores de 45 años y la tercera a cualquier edad, a lo que hay que añadir el problema familiar, el social y el financiero de los que sobreviven con secuelas. Dado que la mayoría de los cambios fisiopatológicos en el metabolismo y en el flujo sanguíneo cerebral ocurren en las primeras horas del traumatismo, el manejo inicial de estos pacientes por parte de los servicios de emergencias y en la sala de urgencias de los hospitales juega un papel fundamental en su pronóstico final. Si bien no podemos evitar en ese momento la lesión primaria, sí podemos actuar limitando su progresión mediante la minimización del daño cerebral secundario. Desde el primer contacto con el paciente y tras las medidas básicas de reanimación, el objetivo fundamental debe ser mantener una estabilidad global que evite principalmente la hipotensión y la hipoxia, que son factores pronósticos independientes de morbi-mortalidad. Además, los pacientes con traumatismo craneal grave deben ser trasladados en condiciones estables a un hospital de tercer nivel que disponga de servicio de neurocirugía y medicina intensiva con técnicas de neuromonitorización, como la presión intracraneal y la oxigenación cerebral. En esta revisión haremos varios apartados que son básicos para el actual manejo del TCE: inicialmente describiremos los conceptos fisiopatológicos en el traumatismo que inciden sobre el flujo sanguíneo cerebral, la presión intracraneal y el metabolismo cerebral, así como los tipos de lesiones primarias y secundarias; en segundo lugar, y de forma más extensa, la evaluación y el manejo pre-hospitalario, en la sala de urgencias y, por último, el tratamiento en la unidad de medicina intensiva basado en técnicas de neuromonitorización multimodal. [Emergencias 2009;21:433-440]

Palabras clave: Traumatismo craneoencefálico. Conceptos fisiopatológicos. Técnicas de neuromonitorización multimodal.