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Management of mechanical ventilation in brain injury: hyperventilation and positive end-expiratory pressure

Manuseio da ventilação mecânica no trauma cranioencefálico: hiperventilação e pressão positiva expiratória final

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ABSTRACT

The study intended to make a critical review on use of pulmonary hyperventilation maneuvers and the different positive end-expiratory pressures applied to traumatic brain injury patients. As a reference were used publications in English, Spanish and Portuguese, contained in the following databases: MedLine, SciELO and LILACS, from 2000 to 2007, we included all studies about the use of pulmonary hyperventilation maneuvers and the different positive end-expiratory levels used for adult patients with brain injury at acute or chronic stage. Thirty one trials were selected, 13 about pulmonary hyperventilation, as prophylaxis, prolonged or optimized and 9 shows the levels of positive end-expiratory

pressures used, ranging from 0 to 15 cmH₂O. The prophylactic hyperventilation maneuver in the first 24 hours can lead to an increase of cerebral ischemia; the prolonged hyperventilation must be avoided if intracranial pressure did not increase; however optimized hyperventilation seems to be the most promising technique for control of the intracranial pressure and cerebral perfusion pressure; the rise of the positive end-expiratory pressure, up to 15cmH₂O, can be applied in a conscientious form aiming to increase arterial oxygen saturation in lung injury.

Keywords: Brain trauma; Intracranial hypertension; Intracranial pressure; Positive-pressure respiration; Hyperventilation

INTRODUCTION

Traumatic brain injury (TBI) is worldwide the main cause of morbidity mortality in individuals less than 45 years old with higher prevalence in the male gender. It takes place in about 40% of victims of trauma, and 20% of them die on the spot or in the first day of admission and 80% in the first seven days after the event.⁽¹⁻³⁾

TBI is a non-degenerative or congenital injury caused by an aggression or started by a process of high energy acceleration or deceleration of the brain inside the cranium which generates an anatomical damage or functional impairment of the scalp, cranium, meninges and encephalus.^(4,5) It can be caused by traffic accident, falls, aggressions, cold steel or firearm perforation, major catastrophes and sport activities.⁽⁶⁾ When appropriate, drugs and alcohol in the organism must be examined.⁽⁷⁾

Two different mechanisms determine severity of trauma (1) first insult, which takes place at the time of impact; (2) second insult which represents a pathological process subsequent to the initial clinical changes of trauma.⁽¹⁾

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Lowering of the level of consciousness is the main risk factor for bronchoaspiration and later admission to the intensive care unit (ICU) for the purpose of detecting and treating complications of the primary injury and supply a better condition for brain function recovery.⁽⁸⁾ Therefore patients with problems related to the central nervous system (CNS) often need ventilation support due to acute respiratory failure (ARF), not always caused by the neurological condition itself, such as decrease of the respiratory drive, but because of lung disease.⁽⁹⁾

Mechanical ventilation is an essential therapeutic device for patients with severe TBI, since it aims to protect the airway by endotracheal intubation and permits sedation, including curarization thus avoiding damages caused by hypoxemia and hypercapnia.⁽¹⁰⁾

Based on ventilation therapies adopted during the last years for patients with TBI, mainly for those presenting intracranial hypertension (ICH) this review intended to compare the different ventilation techniques used for management of patients with TBI and the impact on the parameters of neurological monitoring.

METHODS

A review of literature on TBI was made using as reference publications in English, Spanish and Portuguese whose keywords were traumatic brain injury, intracranial hypertension, intracranial pressure, positive end-expiratory pressure (PEEP) and hyperventilation found in the MedLine, SciELO and LILACS databases, published from 2000 to 2007. Studies that approached different levels of PEEP and use of pulmonary hyperventilation maneuvers in the adult patient with acute or chronic TBI as well as impacts on the neurological parameters were selected.

Criteria were defined to assess the studies, guarantee the works quality, such as: (a) identification of the study for type of treatment and how the technique is performed; (b) methodological characteristic of clinical trials or of review articles. When evaluating qualitative attributes, were considered decrease or increase of the intracranial pressure (ICP) as well as normalization of the cerebral perfusion pressure (CPP) and mean arterial pressure (MAP).

PHYSIOPATHOLOGY OF TRAUMATIC BRAIN INJURY

Mechanism of TBI causes disruption of the hematoencephalic barrier permitting plasma components

to easily cross this barrier towards the neural tissue (vasogenic edema). Hypoxia (secondary insult) affects the sodium-potassium ATPase of the cell membrane, promoting intracellular accumulation of sodium and subsequent water flow to the cell by osmotic gradient. As such, a cytotoxic edema occurs, however, at the sub acute and/or chronic stage. Therefore, the vasogenic edema, accrued by eventual localized areas of hemorrhage with mass effect, is primarily responsible for appearance of intracranial hypertension. Such mechanisms reach their peak in about three to five days.^(11,12)

Changes in the brain flow, inflammation and edema are components of the pathogenesis of brain tissue alterations. The brain is contained in a rigid, not compliant structure in which a relatively low level of swollen tissue can increase the ICP. It also has a special self-regulatory system of the cerebral blood flow (CBF) maintained in normal conditions, even with MAP ranging from 50 to 140 mmHg. Self-regulation of the CBF is achieved by the rapid constriction and relaxation of the cerebral arterioles and venules in response to chemical and endothelial factors and to release of neurotransmitters from adjacent neurons.⁽⁸⁾

CBF relies on the difference of arterial pressure and cerebral venous pressure, being inversely proportional to the cerebral vascular resistance. CPP is calculated by the difference between MAP and the ICP. A CPP of 60 mmHg is commonly accepted as the minimal value needed for adequate cerebral perfusion.⁽¹¹⁻¹³⁾ ICP is determined by pressure of the cerebral parenchyma, cerebral blood volume and fluid volume.⁽³⁾ Increase of ICP is common after TBI when intracranial compliance is unable to accommodate volume increase. The normal value of ICP in adults is 10 mmHg, values over 20 mmHg require therapeutic intervention. Values from 10 to 20 are considered slightly increased and between 20 and 40 mmHg moderately high. Over 40 mmHg are the severe cases of intracranial hypertension, when herniations of nervous tissue may occur.^(8,14)

Hyperventilation may reduce ICP by hypocapnia that induces cerebral vasoconstriction with subsequent reduction of CBF. Routine induction of vasoconstriction by hypocapnia may cause an accidental decrease of the CBF, which would exacerbate perfusion deficit leading later to brain ischemia.^(9,15-17)

PEEP increases functional residual capacity (FRC) and may reduce incidence of mechanical ventilation induced injury. However, it may have deleterious effects on the brain compartment by increase of in-

trathoracic pressure that will increase central venous pressure (CVP) influencing return flow of blood to the heart. Finally, the cardiac output (CO) is reduced, with subsequent decrease of MAP and CPP.⁽¹⁸⁾

LITERATURE REVIEW

In this study 31 publications were selected from the year 2000 to 2007, 11 articles on utilization of pulmonary hyperventilation,^(10,13,15-17,19-24) 7 on utilization of PEEP^(4,18,26-30), two encompassing both subjects^(14,25) and 11 general articles on TBI.^(1-3,5-9,11,12,31) Only one article about pulmonary hyperventilation was clinical⁽²⁰⁾ while five were found about PEEP level.^(18,26-29)

Pulmonary hyperventilation

Regarding pulmonary hyperventilation all nine articles mentioned prophylactic hyperventilation and agreed that it is not recommended in the first 24 hours, since CBF is reduced at this time after trauma^(10,13,16,19,21-25). Six articles conclude that prolonged pulmonary hyperventilation must be avoided in the absence of high ICP as sustained vasoconstriction reduces CBF to deleterious levels and could generate brain ischemia.^(10,14,19,21,23,24) There is a consensus about optimized hyperventilation for a short time with high ICP and the causal factor for increase of ICP must be sought and efforts be made to treat it. There is also a consensus about the cerebral vasoconstrictor effect of hypocapnia, generated by hyperventilation, which reduces CBF and ICP (Chart 1).

Chart 1 – Results of use of hyperventilation

Reference	Study	Method	Results	Conclusion
Belda FJ. 2004 ⁽¹⁰⁾	R	Prophylactic hyperventilation and Prolonged hyperventilation	Both may compromise CPP.	Prophylactic hyperventilation must be avoided ($\text{PaCO}_2 \leq 35$ mmHg) in the first 24 hours and prolonged hyperventilation ($\text{PaCO}_2 \leq 25$ mmHg) if there is no high ICP.
Stocchetti N et al. 2005 ⁽¹³⁾	R	Induction of hypocapnia by hyperventilation.	Decrease of ICP by a cerebral vasoconstriction and decrease of CBF. Low CBF may cause ischemia.	Short term, careful hypocapnia controls decrease of ICP, meaning that it is a useful therapy. Prophylactic hyperventilation seems inappropriate.
Deem S. 2006 ⁽¹⁴⁾	R	Correlation between respiratory management in TBI and its physiology.	Hyperventilation effect on ICP is quickly lost, however effect on CBF can be sustained.	It is prudent to avoid long periods of hyperventilation, as it can exacerbate brain ischemia, mainly in the first 24 hours.
Vincent JL, Berré J. 2005 ⁽¹⁵⁾	R	Management of ICH, TBI and brain edema. Besides control of ICP, divided into 3 interventions.	Periods of hyperventilation may induce some degree of brain ischemia.	Hyperventilation was kept for cases of brain herniation because decrease of the CBF is considered priority for the purpose of avoiding an excessive intervention of ICP.
Brain Trauma Foundation. 2007 ⁽¹⁶⁾	R	Hyperventilation to reduce ICP.	Reduction of ICP by cerebral vasoconstriction and decrease of CBF. Risk of ischemia in aggressive hyperventilation.	Prophylactic hyperventilation ($\text{PaCO}_2 \leq 25$ mmHg) is not recommended and should be avoided in the first 24 hours, due to previous decrease of CBF. Hyperventilation is recommended with monitoring of ICP.
Ghajar J. 2000 ⁽¹⁷⁾	R	Hyperventilation technique in the control of ICP.	Prophylactic or prolonged hyperventilation generates a worse prognosis.	Aggressive technique produces sites of brain ischemia. If after liquor drainage ICP remains between 20 to 25 mmHg, $\text{PaCO}_2 < 30$ mmHg, must be maintained between 30 to 35 mmHg. In ICH refractory to drugs and to surgical procedures $\text{PaCO}_2 < 30$ mmHg should be maintained with monitoring of SjO_2 and CBF.
Andrade FC, Andrade Jr FC. 2000 ⁽¹⁹⁾	R	Hyperventilation maneuver in prevention and treatment of ICH.	Causes brain ischemia in patients with severe TBI.	Prophylactic and continued hyperventilation maneuver must be avoided in the first five days after severe TBI and especially prohibited during the first 24 hours.

Continue...

Chart 1 – Continuation

Reference	Study	Method	Results	Conclusion
Falcão ALE, et al. 2000 ⁽²⁰⁾	T	Hyperventilation optimized at ICP > 20 mmHg, controlling ECO ₂ and CPP.	Ideal PaCO ₂ is that which maintains ICP < 20 mmHg, ECO ₂ between 24% and 42% and CPP > 60 mmHg.	Value of aCO ₂ is dynamic and relies on the stage of TBI and priority must be given to maintenance of an adequate ECO ₂ to avoid brain ischemia.
Marik PE, Varon J, Trask T. 2004 ⁽²¹⁾	R	Current management of TBI.	Prophylactic hyperventilation was associated with worse prognoses in relation to normocapnia..	Prophylactic and prolonged hyperventilation are not recommended. Initial PaCO ₂ between 35 and 40 mmHg. Short hyperventilations as initial measure, to reduce ICP in patients with brain deterioration.
Valadka AB, Robertson CS. 2007 ⁽²²⁾	R	Approach to the use of current "positive" techniques in TBI.	Worse prognosis in severe TBI routinely treated with hyperventilation.	Prophylactic use of hyperventilation is not recommended, becoming the last choice in ICH or at normal or high levels of CBF at the onset of ICH, as well as in the cranial mass lesions.
Vender J. 2000 ⁽²³⁾	R	Aggressive, prophylactic or prolonged hyperventilation in control of ICP.	Cerebral vasoconstriction, reduction of CBF and cerebral blood volume may quickly reduce ICP..	Prolonged hyperventilation (PaCO ₂ < 25 mmHg) must be avoided if there is no elevation of ICP, as well as prophylactic hyperventilation in the first 24 hours. However, hyperventilation is maintained as choice therapy for treatment of high ICP.
Brain Trauma Foundation. 2000 ⁽²⁴⁾	R	Management and prognosis of severe TBI.	Hyperventilation may compromise cerebral perfusion in areas of reduced CBF.	When ICP is not high, prolonged hyperventilation (PaCO ₂ ≤ 25 mmHg) and prophylactic (PaCO ₂ ≤ 35 mmHg) in the first 24 hours of severe TBI should be avoided. Hyperventilation may be needed in acute brain deterioration or in long periods of ICP refractory to sedation, paralysis, drainage of cerebrospinal fluid and osmotic diuretics.
Helmy A, Vyzaychipi M, Gupta AK. 2007 ⁽²⁵⁾	R	Discussion on the intensive management of severe TBI.	Reduction of PaCO ₂ causes cerebral vasoconstriction and reduction of CBF which reduces ICP.	Prophylactic hyperventilation is not recommended during the first 24 hours, if necessary it should be performed in the intensive care unit.

R- review; T - clinical trial; CPP – cerebral perfusion pressure; PaCO₂ – partial carbon dioxide pressure; ICP – intracranial pressure; ICH – intracranial hypertension; CBF – cerebral blood flow; TBI – traumatic brain injury; SjO₂ – jugular venous oxygen saturation; ECO₂ – Cerebral oxygen extraction.

In the first hours after TBI, absolute values of CBF match those of an ischemic event and in this case the hyperventilation maneuver does not always reduce ICP to improve CPP. The ideal value for PaCO₂ is the one that keeps ICP < 20 mmHg and cerebral extraction of oxygen (CEO₂) between 24% and 42% to avoid brain ischemia.⁽²⁰⁾ PaCO₂ must be kept at 35mmHg and 40 mmHg while hyperventilation is reserved for cases with cerebral herniation.^(15,21)

Prophylactic and prolonged hyperventilation maneuvers are not recommended during the first 24 hours,^(10,13,14,16,17,19,22-24) or out of the intensive care unit.⁽²⁵⁾ Hyperventilation is recommended whenever there is monitoring of ICP⁽¹⁶⁾ being indicated for a short time in episodes of its elevation. In prolonged hyperventila-

tion, jugular venous oxygen saturation (SjO₂) and partial oxygen pressure in the brain tissue (PbrO₂) must be monitored.⁽¹⁴⁾

Clinical data showed worse prognosis in severe TBI, routinely treated with hyperventilation. Hyperventilation becomes appropriate in two situations: (1) treatment of difficult to control ICH; (2) CBF at normal level or high at onset of ICH. Also when brain deterioration with suspicion of intracranial mass lesion has occurred.⁽²²⁾

Hyperventilation may be needed at times of acute brain deterioration or in long periods of ICH refractory to sedation, paralysis, drainage of cerebrospinal fluid and osmotic diuretics.⁽²⁴⁾

In an aggressive form hyperventilation may produce sites of brain ischemia. If, after fluid drainage, ICP re-

mained between 20 to 25 mmHg, hyperventilation must be used in an effort to maintain PaCO₂ between 30 to 35 mmHg. In presence of ICH refractory to drugs and surgical procedures, PaCO₂ < 30 mmHg must be maintained with monitoring of SjO₂ and CBF.⁽¹⁷⁾

Utilization of positive end-expiratory pressure

Among the articles on PEEP, five include levels ranging from 0 to 15 cmH₂O and suggest that PEEP may be used, whenever statistically significant increases of ICP have not occurred.^(4,14,27,29,30) Videtta et al. observed a sig-

nificant increase of ICP in PEEP of 10 to 15 cmH₂O without significant changes of CPP.⁽²⁸⁾ Gamberoni et al. disclosed changes in respiratory mechanics due to TBI⁽²⁶⁾ and three articles showed that, in patients with poor pulmonary compliance, use of PEEP up to 12 cmH₂O, caused insignificant increase of ICP (Chart 2).^(14,18,25)

Moderate levels of PEEP, as 15 mmH₂O or even higher, can be safely used in patients with cerebral lesions, mainly in those with low pulmonary compliance.⁽²⁶⁾ It was observed and suggested that PEEP levels up to 12 cmH₂O resulted in a non-significant rise in ICP.⁽²⁷⁾

Chart 2 – Results of utilization of positive end expiratory pressure

Referência	Estudo	Método	Resultados	Conclusão
Dutton RP, Mccunn M. 2003 ⁽⁴⁾	R	Use of PEEP with monitoring of ICP and CPP.	The classic teaching of not using or use low levels of PEEP to prevent rise of ICP is inadequate, since correction of hypoxemia may fail.	PEEP does not rise ICP, nor depresses CPP, a reduction of ICP may take place due to improved brain oxygenation when in an adequated pulmonary volume.
Deem S. 2006 ⁽¹⁴⁾	R	Decrease of Vt to 4-6 mL/Kg plateau pressure < 30 cmH ₂ O, and a change in ICP, CPP and PaCO ₂ was. observed.	PEEP increase intracranial pressure and reduces venous flow to the heart resulting in increase of the jugular venous pressure and thus ICP, CPP and PaCO ₂ become highs.	PEEP must be carefully applied to patients with acute TBI, however must not be avoided in view of the need to maintain adequated oxygenation.
Caricato A, et al. 2005 ⁽¹⁸⁾	T	Comatose patients with severe TBI or subarachnoid hemorrhage with monitoring of ICP, under MV with PEEP, divided into 2 groups. (A) 13 patients with normal lung compliance and (B) 8 with low compliance. PEEP Of 0, 5, 8 and 12 cmH ₂ O were applied and measured jugular pressure, CVP, ICP, CPP, cerebral compliance, VmMCA and SjO ₂ .	In group A rise of PEEP from 0 to 12 cmH ₂ O, significantly increase the jugular pressure but reduced MAP, CPP and VmMCA. In B there were no significant variations. ICP and cerebral compliance did not change in both groups. However, there was a reduction of the SjO ₂ .	In patients with low pulmonary compliance there was no effect in systemic and cerebral hemodynamics. Monitoring of pulmonary compliance may help to avoid harmful effects of PEEP on the intracranial system in patients with normal compliance.
Helmy A, Vyzaychipi M, Gupta AK. 2007 ⁽²⁵⁾	R	Discussion of intensive management of severe TBI.	There is no absolute contraindication to use of PEEP in hypoxemic patients, unless intrathoracic pressure rises the ICP.	PEEP is indicated for patients with lung injury..
Gamberoni C, et al. 2002 ⁽²⁶⁾	T	Analysis of the changes in respiratory mechanics and effects of PEEP in 10 healthy individuals, 10 patients with brain injury and lung injury and 10 patients with brain injury, without lung injury.	Elastance of the respiratory system, thoracic wall and maximum lung resistance in brain injury, regardless of lung injury. In brain injury and lung injury, PEEP of 15 cmH ₂ O increased elastance of the respiratory system, thoracic wall and maximum lung resistance.	In brain injury, respiratory mechanics suffer changes. PEEP has no effect on improvement of the respiratory function in lung injury caused by ventilator associated pneumonia.

Continue...

Chart 2 – Continuation

Reference	Study	Method	Results	Conclusion
Georgiadis D, et al. 2001 ⁽²⁷⁾	T	Increase of PEEP (4, 8, 12 e 4 cmH ₂ O) in 20 patients for 5 minutes, in each value and MAP, ICP respiratory rate and VmMCA were measured..	CPP varied significantly. In 15 patients parameters remained stable. In 7, a slight increase in ICP was reported in MAP. In 3, increase of PEEP generated decrease of MAP and VmMCA, with ICP stable or in slight decrease.	Increment of PEEP up to 12 mmHg has no significant influence on the values of ICP. Application of PEEP is safe, ensuring maintenance of MAP.
Videtta W, et al. 2002 ⁽²⁸⁾	T	Investigate ICP and CPP at different levels of PEEP. 20 patients under MV with monitoring of ICP, 10 with severe TBI, 5 with intracerebral spontaneous hemorrhage and 5 with subarachnoid hemorrhage. , PEEP was raised from 5 to 15 cmH ₂ O, by 5 to 5 cmH ₂ O. After, at least 10 minutes of stay, ICP and CPP were measured.	PEEP from 10 to 15 cmH ₂ O produced a significant increase of ICP (mean of +11.6 mmHg and +14.6 mmHg respectively), however insignificant changes in CPP.	Rise of ICP is a normal effect of increment of PEEP; attention must be given to changes in CPP.
Huynh T, et al. 2002 ⁽²⁹⁾	T	Gradual increment of PEEP in 3 randomized groups of patients with severe TBI and pulmonary dysfunction: (1) 0 to 5 cmH ₂ O (2) 6 to 10 cmH ₂ O (3) 11 to 15 cmH ₂ O	There was a decrease in ICP of: (1) 5.9 ± 0.1 mmHg (2) 8.3 ± 0.2 mmHg (3) 12.0 ± 0.3 mmHg	Strategy of PEEP increment is not associated with commitment of O ₂ transport nor with exacerbation of ICH .
Mascia L, Majorano M. 2000 ⁽³⁰⁾	R	Ventilation management of acute TBI and effects of PEEP.	Effects on CBF rely on intracranial compliance and the absolute value of ICP, in addition to the hemodynamic with improvement of the lung injury.	ICP will not change when increment of CVP by PEEP will remain below the ICP value

R – review; T – clinical trial; CPP – cerebral perfusion pressure; PEEP – positive end-expiratory pressure; Vt – tidal volume; MV – mechanical ventilation; PaCO₂ – partial carbon dioxide pressure; CVP – central venous pressure; MAP – mean arterial pressure; ICP - intracranial pressure; ICH – intracranial hypertension; CBF – cerebral blood flow; TBI – traumatic brain injury; SjO₂ – jugular venous oxygen saturation.

Application of PEEP at 10 and 15 cmH₂O levels significantly increased ICP, without significant change in CPP, in patients with acute lung injury (ALI).⁽²⁸⁾ Increased in levels of PEEP from 0 to 12 cmH₂O generated a decrease of MAP in patients with normal compliance and these same values in patients with poor compliance did not bring about significant variations. Therefore, normal respiratory compliance is one of the factors assisting transmission of harmful effects of PEEP to the intracranial system.⁽¹⁸⁾

Careful control of plateau pressure, up to 30 cmH₂O must be a rational practice with due surveillance of ICP and CPP when there is elevation of PaCO₂.⁽¹⁴⁾ To use or not low levels of PEEP to avoid elevation of ICP is inadequate as it does not correct hypoxemia, which could reduce ICP by better cerebral oxygen-

ation.^(4,25) Effect of PEEP on brain circulation relies on intracranial compliance and on the absolute value of ICP. ICP will not be affected while it remains above CVP generated by PEEP.⁽³⁰⁾

Because of methodological problems there are few epidemiological studies on TBI and this is a hindrance also found in more developed countries.⁽³¹⁾ Ventilation management based upon scientific knowledge is needed for these patients and may interfere in their prognosis.

CONCLUSION

Based upon scientific studies, as well as on better understanding of the pathophysiology of TBI and its aftermaths, prophylactic hyperventilation in the first

24 hours to achieve a decrease of ICP by cerebral vasoconstriction may lead to an increase of the injured cerebral area due to tissue hypoperfusion. Prolonged hyperventilation must be avoided if ICP is not high. However, optimized hyperventilation in short periods seems to be the most promising technique for control of ICP and CPP.

Elevation of PEEP, limited to 15 cmH₂O, may be applied in an responsible way to improve alveolar oxygenation and increase of SaO₂ in lung injury, ensure an improvement of lung compliance.

RESUMO

O objetivo desse artigo foi realizar uma revisão de literatura sobre a utilização de manobras de hiperventilação pulmonar e os níveis de pressão positiva expiratória final empregados em pacientes com trauma craniocéfálico. Foram utilizados como referências publicações em inglês, espanhol e português, contidas nas bases de dados: MedLine,

SciELO e LILACS, de 2000 até 2007, tendo como critérios de inclusão: manobra de hiperventilação pulmonar e o nível de pressão positiva expiratória final aplicado ao paciente adulto com trauma craniocéfálico, agudo ou crônico. Foram selecionados 31 estudos, sendo 13 sobre a utilização da hiperventilação pulmonar, como profilática, prolongada ou otimizada e 9 abordavam o emprego da pressão positiva expiratória final, em níveis que variaram de 0 a 15 cmH₂O. A hiperventilação profilática nas primeiras 24 horas pode levar a um aumento da isquemia cerebral; a hiperventilação prolongada deve ser evitada na ausência de elevada pressão intracraniana; já a hiperventilação otimizada parece ser a técnica mais promissora no controle da pressão intracraniana e da pressão de perfusão cerebral. A elevação da pressão positiva expiratória final até 15 cmH₂O pode ser aplicada, de forma consciente, com o objetivo na elevação da saturação arterial de oxigênio em presença de injúria pulmonar.

Descritores: Traumatismos craniocerebrais; Hipertensão intracraniana; Pressão intracraniana; Respiração com pressão positiva; Hiperventilação

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