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Effects of respiratory physiotherapy on intracranial pressure and cerebral perfusion pressure in severe traumatic brain injury patients

Efeitos da fisioterapia respiratória na pressão intracraniana e pressão de perfusão cerebral no traumatismo cranioencefálico grave

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Received from Improvement Program of the Faculdade de Medicina de São José do Rio Preto - FAMERP - São José do Rio Preto (SP), Brazil.

Submitted on 22 April, 2008
Accepted on 26 November, 2008

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ABSTRACT

Objective: After brain injury intracranial hypertension is the major cause of mortality, in addition to the possibility of functional, behavioral and cognitive sequels. Scarcity of studies on the effects of respiratory physiotherapy on these patients may lead to contradictory performances. This study aimed to assess the effects of customary respiratory physiotherapy maneuvers on intracranial and cerebral perfusion pressures in patients with severe brain injury.

Methods: Clinical, prospective trial with patients with severe traumatic brain injury, mechanically ventilated and with a continued measurement of intracranial pressure. The effects of manual vibrocompression maneuvers and intratracheal aspiration with or without saline infusion on the measurements of intracranial and cerebral perfusion pressures, between the first and third day after cerebral injury were evaluated.

Results: Data were collected from 11 patients, 41 years of age (median) and APACHE II of 19.5 ± 5. The manual vibrocompression maneuver did not cause an increase of intracranial pressure on any of the days assessed. Intracranial pressure significantly increased after intratracheal aspiration maneuvers in relation to the basal measurement (day1, 9.5 ± 0.9 mm Hg vs 18.0 ± 3.2 mm Hg; day 2, 10.6 ± 1.7 mm Hg vs 21.4 ± 3.8 mm Hg; day 3, 14.4 ± 1.0 vs 24.9 ± 2.7 mm Hg; $p<0.05$ for all). However, these elevations were transient (about 27 seconds) and accompanied by compensatory increases of the cerebral perfusion pressure.

Conclusion: The manual vibrocompression maneuver did not increase intracranial pressure or cerebral perfusion pressure in patients with severe brain injury. Intratracheal aspiration induced a significant and transient increase of the intracranial and cerebral perfusion pressures.

Keywords: Physical therapy modalities/methods; Intracranial hypertension/therapy; Intracranial pressure; Respiratory therapy; Intubation, intratracheal; Brain injuries

INTRODUCTION

Brain injury (BI) is worldwide the primary cause of mortality and functional disability. Every year about 1.5 million people die and hundreds of millions require emergency treatment.[1] Recognition and treatment of intracranial hypertension (ICH) must be immediate.[2,3] Secondary cerebral injuries result from systemic and intracranial causes and may lead to development of functional, psychological, behavioral and cognitive sequels, with an important onus on rehabilitation and difficulty for psychosocial and familial reintroduction of these patients.[4,5]

In the intensive care unit (ICU), the main objective of care for neurological patients is to avoid secondary injury by maintaining hemodynamic, metabolic and respiratory stability to ensure an adequate offer of oxygen and nutrients to the cerebral tissue.[5] In adult patients IHC is defined as presence of intracranial pressure (ICP) over 20 mm Hg, persisting for more than 20 minutes.[6,7]. Values of ICP lower than10 mm Hg (tolerated up to 20 mm Hg), cerebral perfusion pressure (CPP) over 70 mm Hg and mean arterial pressure (MAP) from 70 to110 mm Hg are considered normal or desirable values.[8] Increases of ICP may cause decrease of CPP, if there is no concomitant increase of MAP. [7] This decrease results in circulatory impairment with cerebral hypoxia and increase of cerebral edema, that in the more severe cases can lead to encephalic death.[1,6] Monitoring of ICP is indicated for all patients with BI with a possibility of neurological recovery, with values of the Glasgow Coma Scale (GCS) from 3 to 8 and with abnormal findings at computerized tomography (CT).[7,8]

During recovery of neurological injury some of the fundamental procedures are mechanical ventilation and deep sedation. In these conditions there is a major risk of pulmonary complications. Respiratory physiotherapy belongs to the therapeutic armamentarium with a significant role in preventing mechanical and infectious complications. Earlier studies suggest that, in insufficient sedation conditions, maneuvers of respiratory physiotherapy may determine significant increases in cardiac rate, arterial pressure, cardiac output, oxygen consumption and production of CO_2.[9] Systemic alteration of arterial pressure (AP), of intrathoracic pressure and of cough reflex, induced by respiratory physiotherapy, must have some impact on CPP. It was reported that respiratory physiotherapy may be safely used in patients with a ICP lower than 30 mm Hg, but that the intratracheal aspiration (ITA) maneuver significantly rises the ICP.[10,11] The objective of our study was to assess the effects of manual vibrocompression maneuvers and of ITA, on the measurements of ICP and CPP at the acute stage of BI.

METHODS

Prospective clinical, interventional, non-randomized and uncontrolled trial carried out from September, 2007 to January, 2008. The study was approved by the Research Ethics Committee of the Faculdade de Medicina de São José do Rio Preto – FAMERP (N°241/2007). Free written consent was signed by next of kin. Patients admitted at the ICU with BI, with age ≥18 years, admitted for less than 24 hours, monitored for ICP and mechanically ventilated were included. Brain death or life expectancy of less than 3 day were exclusion criteria.

Results of 330 ICP and MAP measurements from 11 patients obtained between the first and third day after cerebral injury were evaluated. The CPP was calculated using the formula CPP = MAP – ICP. Monitoring of ICP was carried out by subarachnoideal catheter with monitor and Kit Micro Externo Ventura – KOMPACTO®. MAP was monitored by means of invasive arterial pressure (AP).

The respiratory physiotherapy protocol comprised bilateral and manual vibrocompression maneuvers and ITA with and without saline infusion , performed in the morning for about 20 minutes. All physiotherapeutic interventions were performed by the same physiotherapist (CT) for all patients, who were in dorsal decubitus, with a 30^0 inclination of the headrest during the procedure. The ICP and the MAP were monitored at the following times: immediately before beginning the procedure (TO), at the end of the manual vibrocompression maneuver (T1), immediately after the first ITA (T2), immediately after the second ITA, with saline infusion (T3) and after end of the entire procedure (T4).

The results are presented as the mean ± SD. Statistical significance was determined with a *t* test or an analysis of variance for repeated measurements. A Bonferroni adjustment was used for multiple comparisons. A *P* value of < 0.05 was considered statistically significant.

RESULTS

Data from 11 patients were collected, 10 of the male gender and one of the female gender, with 41 years of age (median). GCS values, Acute Physiology and Chronic Health Evaluation *(*APACHE) II index and the main findings of the CT images are described in table 1. Values for GCS and APACHE II were 5.4 ± 1.4 and 19.5 ± 5, respectively.

The manual vibrocompression maneuver did not lead to ICP increase in any of the days assessed. On the contrary, at day 3 a significant drop of ICP after the vibrocompression maneuver was observed when compared to the basal measurement (14.5 ± 1.0 mm Hg vs 11.6 ± 1.6 mm Hg, $p<0.05$) (Figure 1).

ICP increased significantly after the ITA maneuvers (T2 and T3) in relation to the basal measurement (T0) on all days evaluated ($p<0.05$) (Figure 1). The final ICP measurement (T4) was significantly higher than the initial (T0) (day 1, 18.0 ± 3.2 mm Hg vs 9.0 ± 0.0 mm Hg; day 2, 21. ± 3.8 mm Hg vs 10.6 ± 1.7 mm Hg; day 3,

Table 1- Demographic, imaging and severity data

Patients	Gender	Age	Weight	GCS	APACHE II	Cerebral tomography
1	M	28	70	7	17	EDH SAH
2	M	42	65	7	12	Frontal Contusion
3	M	58	75	3	21	SDH, SAH, Temporal contusion
4	M	76	80	6	27	SDH temporal contusion
5	M	31	70	6	16	EDH, SAH
6	M	41	85	6	27	Temporal and mastoid bone fracture
7	M	19	80	3	16	SDH
8	M	57	130	4	21	SDH, DAÍ
9	M	27	75	6	16	EDH, SAH, DAI, pneumoencephalus, fracture of the cranial base
10	M	22	70	6	17	SDH DAI, temporal fracture
11	F	51	65	5	25	SDH

GCS – Glasgow coma scale; EDH – extradural hematoma, SDH - subdural hematoma; ASH - subaracnoid hemorrhage; DAÍ – diffuse axonal injury

24.9 ± 2.7 mm Hg vs 14.4 ± 1.0; p<0.05 for all). At the end of the physiotherapeutic procedure the ICP was above 20 mm Hg in 9 of the 11 patients, with a median of 27 mm Hg (minimum, 22 mm Hg; maximum, 47 mm Hg). Nevertheless a rapid return to the basal levels was observed (median, 27 seconds; minimum, 10 seconds; maximum, 180 seconds). The MAP rose significantly after the ICA maneuver (T2 and T3) on day 1 and, after ITA with tracheal saline infusion (T3) at every day (Figure 2). MAP at the end of the procedure (T4) was higher that the basal every day (day 1, 110 ± 5 mm Hg vs 99 ± 3 mm Hg; day 2, 104 ± 6 mm Hg vs 93 ± 3 mm Hg; day 3, 113 ± 4 mm Hg vs 97 ± 2 mm Hg vs; p<0.05 for all) (Figure 2).

CPP did not change on day 1 and 2 but was significantly higher at the end of the procedure (89 ± 3 mm Hg) in comparison to the initial measurement (82 ± 3 mm Hg) (p<0,05) (Figure 3).

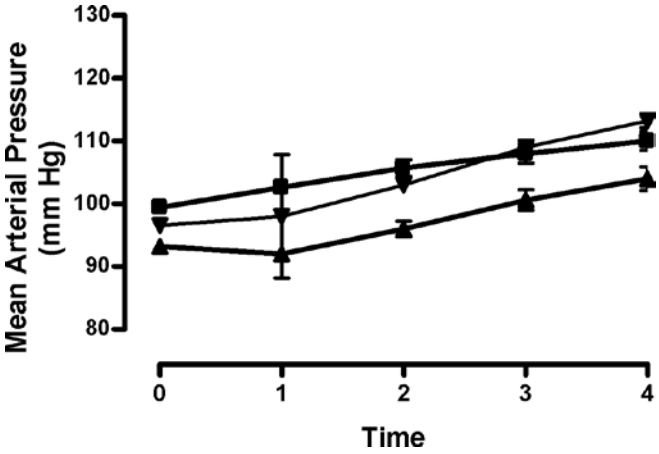

Figure 2 – Mean arterial pressure measurements at different times. At baseline (T0), at vibrocompression ((T1), after intratracheal aspiration (ITA) without tracheal saline infusion (T2), after ICA with tracheal saline infusion (T3),and endpoint (T4), on days 1(■), 2 (▲) e 3 (▼). P<0.05 for T4 vs T0, T3 vs T0, T2 vs T0 on days 1 and 3; T4 vs T0ande T3 vs T0 on day 2

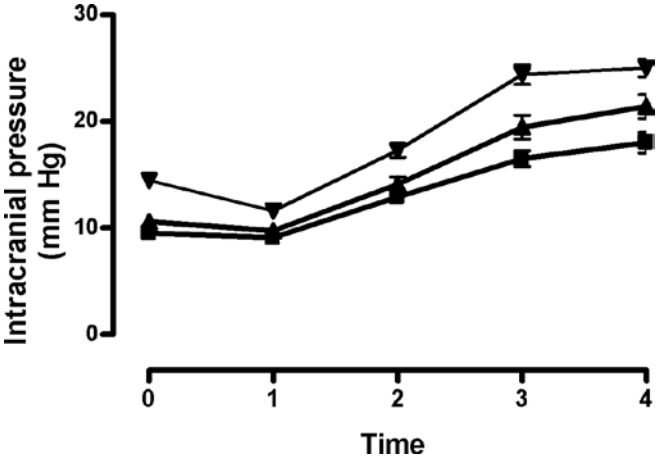

Figure 1- ICP measurements at different times. Baseline moment (T0) after vibrocompression (T1), after intratracheal aspiration (ITA) without tracheal saline infusion (T2), after TIA with tracheal saline infusion (T3), and endpoint (T4) on days 1(■), 2 (▲) e 3 (▼). P<0.05 forT1 vs T0 on day 1; T4 vs T0, T3 vs T0 and T2 vs T0 on days 1, 2 and 3.

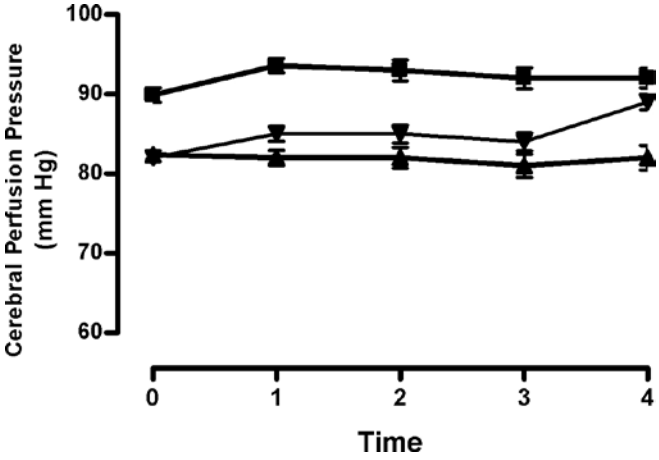

Figure 3 Cerebral perfusion pressure measurements at different times baseline (T0), after vibrocompression (T1), after ITA without tracheal saline infusion (T2), after ITA with tracheal saline infusion (T3), and endpoint (T4), on days 1(■), 2 (▲) e 3 (▼). *: P<0.05 for T4 vs T0 on day 3.

DISCUSSION

It has been reported that respiratory physiotherapeutic interventions commonly performed in critically ill patients may influence cerebral oxygen transport due to its adverse effects on cardiac output (CO). [12,13] Theoretically, respiratory physiotherapy measures applied on the chest, increase intrathoracic pressure (ITP) with drop of cerebral venous return and implies increase of ICP.[3,10]

Results of this study disclosed that the manual vibrocompression maneuver did not determine an increase of ICP or CPP on any of the assessed days. On the contrary, on the third day of intervention a significant drop of ICP followed the maneuver. However, the ICT maneuver, especially with tracheal saline infusion caused a significant increase of ICP. Yet, this increase was transitory and followed by compensatory increase of CPP and none of the patients presented with ICH.

Our findings differ from those of other authors on some points and must portray differences in the methodology used or in the population studied. Thiesen et al. reported increased ICP after the ICA maneuver in patients with severe BI, albeit with no increase of MAP and maintenance of CPP at adequate levels. [10] While Nemer et al. reported that unilateral chest compression as well as ITA may significantly change the ICP in patients with BI and stroke.[11] In that study there were no reports of CPP nor about the time when ICP remained elevated, hindering comparison with our study.

In general, findings of these studies suggest that ITA causes small and temporary increases of ICP; if MAP increases in response to the maneuver, the CPP also increases. [9] It is desirable to maintain CPP levels above 70 mm Hg to assure adequate oxygen transport to the brain.[7] Increases of MAP, of jugular venous oxygen tension, velocity of the middle cerebral artery flow are described, which together suggest a protective compensatory response, maintaining the cerebral oxygen delivery during and after ITA.[12,13] Increase of arterial pressure apparently is a compensatory response to the procedure-induced hypoxia.[10] Other authors suggest that the ITA may increase ICP as a response to the cough reflex and to hypercapnia with consequent cerebral vasodilatation.[11,14] Contrariwise, a study that assessed the effects of using positive end expiratory pressure (PEEP) on ICP reported decreases of MAP related to lower levels of central venous pressure (CVP) suggesting that in hypovolemic patients, increase of pressure on the chest may have a significant hemodynamic effect.[3]

The reason for the decline of ICP after the manual vibrocompression maneuver, perceived on the third day, remains unknown**.** It may possibly be the outcome of amelioration of pulmonary ventilation and drop of $PaCO_2$ caused by the intervention. [9] We know that elevation of ICP may be attenuated by small periods of hyperventilation, prior to aspiration.[12,15,16] This mechanism must be better assessed in future studies.

CONCLUSIONS

The manual vibrocompression maneuver did not induce increase ICP or CPP in patients with severe BI. However, ITA led to a significant and transient ICP increase, accompanied by parallel increase of CPP. Such maneuvers are safe in BI patients, as long as they are adequately performed and under surveillance. Other studies with a higher number of patients are required to corroborate our results.

RESUMO

Objetivos: Após um traumatismo cranioencefálico, a hipertensão intracraniana representa a maior causa de mortalidade, além da possibilidade de seqüelas funcionais, comportamentais e cognitivas. A escassez de estudos sobre os efeitos da fisioterapia respiratória nestes pacientes pode levar à condutas contraditórias. O objetivo deste estudo foi avaliar os efeitos de manobras usuais de fisioterapia respiratória sobre a pressão intracraniana e a pressão de perfusão cerebral em pacientes com traumatismo cranioencefálico grave.

Métodos: Ensaio clínico, prospectivo, em pacientes com traumatismo cranioencefálico, ventilados mecanicamente e com medida contínua da pressão intracraniana. Foram avaliados os efeitos das manobras de vibrocompressão manual e aspiração intratraqueal sem e com instilação de soro fisiológico, sobre as medidas de pressão intracraniana e de pressão de perfusão cerebral, entre o primeiro e o terceiro dia após a lesão cerebral.

Resultados: Foram obtidos os dados de 11 pacientes com idade de 41anos (mediana) APACHE II de 19,5 ± 5. A manobra de vibrocompressão manual não determinou aumento da pressão intracraniana em nenhum dos dias avaliados. A pressão intracraniana aumentou significativamente após manobras de aspiração intratraqueal em relação à medida basal (dia 1, 9,5 ± 0,9 mm Hg vs 18,0 ± 3,2 mm Hg; dia 2, 10,6 ± 1,7 mm Hg vs 21,4 ± 3,8 mm Hg; dia 3, 14,4 ± 1,0 vs 24,9 ± 2,7 mm Hg; $p<0,05$ para todos). Contudo, estas elevações foram transitórias (em torno de 27 segundos) e acompanhadas de aumentos compensatórios da pressão de perfusão cerebral.

Conclusões: A manobra de vibrocompressão manual não determinou aumento da pressão intracraniana ou da pressão de perfusão cerebral em pacientes com traumatismo cranioencefálico grave. A aspiração intratraqueal levou a aumento significativo e transitório da pressão intracraniana e da pressão de perfusão cerebral.

Descritores: Modalidades de fisioterapia/métodos; Hipertensão intracraniana/terapia**;** Pressão intracraniana; Terapia respiratória; Intubação intratraqueal; Traumatismos encefálicos

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