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The effect of breath physiotherapeutic maneuvers on cerebral hemodynamics

A clinical trial

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ABSTRACT

Objective: To observe the repercussion of respiratory physiotherapy techniques on the mean arterial pressure (MBP), intracranial pressure (ICP), cerebral perfusion pressure (CPP), jugular venous oxygen pressure ($PjvO_2$) and jugular venous oxygen saturation ($SjvO_2$). **Method:** The sample consisted of 20 patients with head trauma. The protocol consisted of physiotherapy techniques application of vibrocompression (VBC), expiratory flow increase (EFI) and suction. **Results:** The results show the maintenance on variables of cerebral hemodynamics during the techniques of VBC and EFI. However, in relation to suction, there was an increase of MBP, ICP, with maintenance of CPP, $PjvO_2$ and $SjvO_2$ and return to baseline of MBP and ICP 10 minutes after the end of suction. **Conclusion:** The respiratory physiotherapy techniques (VBC, EFI) do not promote cerebral hemodynamic repercussion, unlike suction, in severe head injury patients, mechanically ventilated, sedated and paralyzed.
Key words: physical therapy, respiratory, brain.

Efeitos das manobras de fisioterapia respiratória na hemodinâmica cerebral: um ensaio clínico

RESUMO

Objetivo: Observar a repercussão das técnicas de fisioterapia respiratória na pressão arterial média (PAM), pressão intracraniana (PIC), pressão de perfusão cerebral (PPC), pressão venosa jugular de oxigênio (PjO_2) e saturação venosa jugular de oxigênio (SjO_2). **Método:** Foram incluídos no estudo 20 pacientes com traumatismo cranioencefálico. O protocolo consistiu na aplicação das manobras fisioterapêuticas de vibrocompressão (VBC), aumento de fluxo expiratório (AFE) e aspiração (ASP). **Resultados:** Os resultados mostraram a manutenção das variáveis da hemodinâmica cerebral durante as manobras de VBC e AFE. Porém, em relação à ASP, houve uma elevação da PAM e PIC, com manutenção da PPC, PjO_2 e SjO_2 e retorno aos valores basais da PAM e PIC dez minutos após o final da aspiração. **Conclusão:** As manobras de fisioterapia respiratória (VBC, AFE) não promovem alterações sobre a hemodinâmica cerebral, ao contrário da ASP traqueal, em pacientes com traumatismo cranioencefálico grave, em ventilação mecânica, sedados e curarizados.
Palavras-chave: fisioterapia, respiratória, cérebro.

Head injury (HI) is the largest cause of incapacity and death among Western nations[1]. The total number of deaths per year resulting directly or indirectly from HI are estimated in 500.000[2].

In the current knowledge status concerning treatment of patients stricken by severe HI, it is recognized the extremely outstanding role carried out by an appropriate physiotherapeutic attendance in-

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volving motor and cardio-respiratory care oriented to aspects like possible neurological sequels, bed immobilization and lung secretion accumulation. Without that specific treatment modality, most of the patients develop lung infection associated to mechanical ventilation or even acute respiratory distress syndrome rising up morbidity and mortality indexes[3].

In most of the developed nations' hospitals, physiotherapy constitutes an integral part of the intensive care unit (ICU) management procedures. Among the several techniques used by ICU physiotherapists, the most common are postural drainage, mobilization, vibration, percussion, manual hyperinsuflation, aspiration (ASP) and many types of breathing exercises. These techniques, routinely combined, focus the patient's underlying physiopathology condition intending to avoid lung complications[4].

The physiotherapeutic maneuvers of vibrocompression, expiratory flow increase (EFI) and aspiration act in the intracranial compartment's pressure through venous stream, in the same way as the positive end expiration pressure. By that, the central venous pressure can be increased, elevating intrathoracic pressure levels, obstructing and delaying the superior and inferior vena cava venous return. In addition, positive end expiration pressure can decrease cardiac output and mean blood pressure (MBP), reducing cerebral perfusion pressure (CPP) and increasing intracranial pressure (ICP)[5].

This research was designed to verify the influence of breathing physiotherapy maneuvers of vibrocompression (VBC), EFI and aspiration (ASP) in order to analyze their effects in the variables; MBP, ICP, CPP, jugular venous partial pressure in oxygen ($PjvO_2$) and internal jugular venous oxygen saturation ($SjvO_2$) in patients with HI admitted to ICU.

METHOD

Design of study

This descriptive longitudinal study, allowing a quantitative approach, is a prospective non-randomized clinical trial approved by the ethics and research committee of 'Hospital de Clinicas da Universidade Federal do Paraná'-065EXT019/2002-11.

Setting

The clinical trial was accomplished in the ICU for adult patients of 'Hospital do Trabalhador de Curitiba'.

Participants

The sample consisted of 20 consecutive adult patients of both genders randomly chosen on admission to ICU. The parameters of choice were; presence of severe HI (Glasgow Coma Scale score ≤8-eight) in the first 48 hours of admission, absence of lung radiological alterations, propofol sedation (Ramsay Scale score 6-six, enclosure 3), curarization with pancuronium bromide inhibiting cough reflex, intubation with mechanical ventilation (Neumovent Graph) assisted and controlled by volume estimated at 8 ml/Kg/ body weight and positive end expiration pressure of 5 mmHg with an oxygen inspired fraction of 40%. Hemodynamic unstable patients presenting MBP levels <60 mmHg with pulmonary contusion and costal arch fractures were excluded from the trial.

Description

After reading and signing Free and Informed Consent Form, all patients underwent intra-arterial catheterization for monitoring of MBP levels as well as placement of an intra-ventricular catheter for ICP gauging. The catheters then were connected to a HP multi parametric monitor (Hewlett Packard). In addiction, a catheter was placed in the jugular bulb to collect $PjvO_2$ and $SjvO_2$ gasometry data.

Interventions

The patients were positioned in dorsal decubitus with headboard at 30º. All hemodynamic variables were collected: MBP, ICP (by multi-parametric monitor) and CPP as well as $PjvO_2$ and $SjvO_2$ gasometry data and the results obtained were established as basal values.

The VBC and EFI physiotherapeutic maneuvers were performed in sequence; 5 minutes on each hemithorax, each maneuver lasting for 10 minutes. The first applied maneuver was VBC in order to modify mucous' physical properties, decreasing its viscosity by the provided thixotropism. After that, it was applied the EFI maneuver responsible for mobilize, displace and eliminate peripheral secretions towards the windpipe utilizing the increased expiratory flow. Next, ASP was undertaken preceded by 5 ml physiological saline solution (0.9%) instillation and three hyperinsuflations and hyperoxygenations with a manual resuscitation ambu connected to an oxygen source (10 l/s flow). The chosen sterile/dischargeable valve probe was number 12. The ASP procedure total time was about 10 to 15 seconds and accomplished just once.

Between each maneuver it was given a 5 minutes rest period in order to prevent reflections of the first maneuver to the second and from the second to the third one, trying this way to avoid cumulative effect of one maneuver into the other.

Statistical analysis

The database was submitted to statistical analysis software (*Statistica*) and then evaluated. Comparing all procedures in the five moments, the null hypothesis (equal averages in all of the moments) was tested against the alternative hypothesis (at least one moment with different average from the others). Evaluation was performed by

repeated analysis of variance measures. For the null hypothesis rejection, Newman-Keuls test was used, evaluating all pairwise comparisons. When comparing only two moments it was considered the Student's t test for dependent samples. Data's normal condition was evaluated by Kolmogorov-Smirnov test ($p<0.05$ indicated statistical significance).

RESULTS

Sample characterization

Twenty patients were included in the study, 18 male (90%) and 2 female (10%), age mean average of 33.5 (±11.94), Apache 26 (±4) and Ramsay 6.

All patients presented HI (100%) and admitted to ICU ('Hospital do Trabalhador de Curitiba') in the period of November 2002 to May 2004.

Analysis results

In relation to VBC, there were no significant behavioral changes in MBP, ICP and CPP values during the maneuver 10 minutes execution period (Table 1).

There were no significant changes related to MBP, ICP and CPP behavior during the EFI maneuver 10 minutes execution period as shown by additional results in Table 2.

Table 3 depicts the mean variance behavior of MBP, ICP and CPP during ASP maneuver. Comparing the moments before and after ASP, the variables MBP ($p=0.0111$) and ICP ($p=0.0004$) presented a significant change. However, by comparing the moments before ASP against 10 minutes after the final procedure, there was no statistical relevancy related to MBP and ICP, showing that the variables returned to their basal values. CPP kept unchanged during the whole protocol.

The variables $PjvO_2$ and SjO_2 did not show significant changes during MBP, ICP, CPP and 10 minutes after ASP procedures compared to basal moment (Table 4).

DISCUSSION

Stiller[4] reviewing selected articles in MEDLINE and CINAHL (*Cumulative Index to Nursing and Allied Health Literature*), as well as Dean and Ross[6], verified the importance of ICP, CPP and hemodynamics monitoring procedures during physiotherapy interventions.

According to Thiesen et al.[7] physiotherapy maneuvers, when applied upon the patient's thoracic box, increase intrathoracic pressure with adverse effect in the cerebral venous return by rising the right ventricle and large vessels pressure levels, including superior vena cava.

Table 1. MBP, ICP and CPP standard deviation/mean average results for each VBC moment.

Vibrocompression maneuver			
Time (min)	MBP (mmHg) Mean average±SD	ICP (mmHg) Mean average±SD	CPP (mmHg) Mean average±SD
0:00	94.00±18.78	16.95±7.25	77.25±22.45
0:02	94.50±20.46	17.65±7.73	76.85±23.92
0:04	95.95±23.30	17.75±7.86	78.20±26.36
0:06	97.20±24.18	17.55±7.96	79.65±27.59
0:08	94.60±20.46	17.15±8.09	77.45±24.27
0:10	93.00±17.54	16.80±7.68	76.15±22.38

SD: standard deviation; MBP: mean blood pressure; ICP: increasing intracranial pressure; CPP: cerebral perfusion pressure; VBC: vebrocompression.

Table 2. MBP, ICP and CPP standard deviation/mean average results for each EFI moment.

Expiratory flow increase maneuver			
Time (min)	MBP (mmHg) Mean average±SD	ICP (mmHg) Mean average±SD	CPP (mmHg) Mean average±SD
0:00	94.70±18.17	17.75±7.78	76.90±22.95
0:02	97.10±20.93	18.10±7.93	79.20±24.98
0:04	96.90±19.92	18.25±7.89	78.55±23.98
0:06	93.05±28.65	18.50±8.08	78.30±24.75
0:08	96.85±20.27	18.70±7.77	78.10±23.86
0:10	96.65±21.61	18.80±7.35	77.65±25.80

SD: standard deviation; MBP: mean blood pressure; ICP: increasing intracranial pressure; CPP: cerebral perfusion pressure; EFI: expiratory flow increase.

Table 3. MBP mean average/standard deviation results for each moment of aspiration.

Aspiration maneuver			
Time (min)	MBP (mmHg) Mean average±SD	ICP (mmHg) Mean average±SD	CPP (mmHg) Mean average±SD
Before	94.25±18.40	19.65±8.24	74.60±21.97
Maximum	107.70±24.04	28.50±14.15	80.15±32.22
Minimum	97.25±21.14	23.15±12.07	74.85±26.86
After	100.65±22.19*	26.35±12.82*	75.15±28.77
10' later	93.10±17.62	19.65±7.88	73.45±21.30

SD: standard deviation; *p<0. 05; MBP: mean blood pressure; ICP: intracranial pressure; CPP: cerebral perfusion pressure.

Table 4. SjO_2 mean average/standard deviation results in each one of the moments.

Maneuvers		
Moments	$PjvO_2$ (mm Hg) Mean average±SD	SjO_2 (mm Hg) Mean average±SD
Basal	45.95±8.64	77.93±7.22
VBC	45.79±13.51	76.50±8.85
EFI	44.22±12.59	75.04±8.61
ASP	45.43±12.24	75.99±8.13
10' later	45.44±11.25	75.71±7.97

SD: standard deviation; SjO_2: jugular venous oxygen saturation; $PjvO_2$: jugular venous oxygen pressure; VBC: vibrocompression; EFI: expiratory flow increase; ASP: aspiration.

This study tried to observe the repercussion of respiratory physiotherapy maneuvers into cerebral hemodynamic variables. In relation to isolated VBC and EFI maneuvers, the variables MBP, ICP, CPP, $PjvO_2$ and $SjvO_2$ did not show any significant change, remaining stable during the whole 10 minutes procedure period and even after its conclusion.

The results corroborate with Ciesla *apud* Irwin and Teclin[8] who found out that percussion and vibration do not cause any adverse effect in ICP.

Thiessen et al.[7], in a study with severe HI patients, did not observe changes in MBP, ICP and CPP when costal manual vibration and manual expiratory pressure were applied, corroborating with this study results.

In terms of ASP procedure, this study observed a significant increase related to variables MBP and ICP but did not find changes in CPP, $PjvO_2$ and $SjvO_2$. The ICP increased level was gathered by an increase of MBP. This can be justified by the probable condition of limited or absent cerebral self-regulation mechanism observed in patients with severe HI.

In a study quoted by Stiller[4], when evaluating several physiotherapeutic maneuvers including ASP, the observed increase in ICP was less than 10 mmHg. In the studies involving MBP, ICP and CPP measures, the increase of ICP values was accompanied by an increase of MBP, resulting this way in a less than 10 mmHg CPP elevation.

Gemma et al.[9] observed that ASP caused increases in ICP, CPP and $SjvO_2$ levels without ischaemic evidences in well-sedated patients as well as decreases of CPP and $SjvO_2$ levels in patients presenting cough reflex.

The observed ICP increase is justified by Fortune et al.[10], Kerr et al.[11], Knobel[12] and Carvalho[13] as a result of the intrathoracic pressure elevation, due to carinal stimuli, leading to venous return impairment what elevates cerebral blood flow (CBF) with a secondary increase of the arterial pressure.

Ersson et al.[14] found more ICP variations in the group where cough reflex was not inhibited and lesser ICP increases associated to groups where the reflex was abolished. The largest pressure levels variation were verified in the first minute after procedures conclusion. At the end of the fifteenth minute, pressure levels presented stable with values similar to basal.

The care procedures involving airways, performed in the previous study, can increase ICP values at least by two different manners. In ASP and *bag squeezing* the intrathoracic pressure is momentarily elevated by cough and lung insufflation respectively. Consequently, there will be an increase in central venous pressure level, decreasing cerebral venous drainage and elevating ICP. This sequence occurs during the procedures' initial period.

Another mechanism explains the ICP variation as a result of CPP changes due to the CBF reduction derived from sympathetic branches vagal activity due to tracheal stimuli. The rise of ICP can be a consequence of an increased cerebral blood volume due to a CPP decreased level below certain limits (70-80 mm Hg) when vasodilatation is induced by the CBF self-regulation mechanism.

Applying the findings of Ersson et al.[14] in this study, increased ICP levels can occur derived from an elevation of intrathoracic pressure due to pulmonary hyper-insufflation (prior to ASP) as well as through carinal stimuli. In addiction, the likely presence of pain during the pro-

cedure may contribute to the rising of ICP since the patients were sedated merely by a hypnotic drug.

In order to investigate the early administration efficacy of neuromuscular blocking agents (NMB), related to ICP management, in patients with severe HI by trauma, Hsiang et al.[15] and White et al.[16] concluded that the use of NMB must be administrated only in cases presenting intracranial hypertension. Other HI patients may be managed by sedation once the use of NMB do not show benefits. On the contrary, it delays hospitalization period and increases extra-cranial adverse effects associated to pharmacological paralysis as: accidental disconnection, pulmonary complications linked to the ventilation/perfusion relationship, atelectasis, embolism and cardiovascular side effects.

The present study agrees with White et al.[16] assertion that isolated NMB administration do not provide necessary protection since it was not able to inhibit MBP rises. In addiction, in the event of absent cerebral self-regulation mechanism, NMB can also induce increases in ICP. The quoted authors suggest that NMB combined with a topic anesthetics could bring benefits, preventing ICP and MBP risings associated to endotracheal ASP.

Kerr et al.[11] reported ICP elevations in patients not treated with NMB during hyperinsuflation, hyper-oxygenation and in the course of the endotracheal ASP catheter insertion procedure. The increases were concomitant with MBP elevations. Otherwise, patients managed with NMB did not present increased ICP during the ASP catheter insertion; however, NMB was unable to prevent ICP rising during hyperinsuflation and hyper-oxygenation.

Unni et al., Werba et al. (1991) and Klezl et al. (1991) *apud* Kerr et al.[11] suggest that NMB, by inducing vasomotor tonus changes as well as intercostals muscles and diaphragmatic paralysis, can mitigate the expected rising in ICP during endotracheal ASP.

Venous pressure elevations caused by hyperinsuflation combined with arterial pressure rising, both related to CBF augments, can hinder the ICP return to basal levels. This mechanism may explain the reason why it takes 10 minutes or more for ICP to return at basal values.

Oertel et al.[17], Oertel et al.[18] and Pinaud el al.[19] corroborate with Knobel[12] asserting that, in terms of HI patients, relaxation and curarization decrease ICP levels, maintaining arterial pressure stable.

Rudy et al.[20] showed that patients with severe and closed HI (Glasgow ≤8) show the risk of presenting intracranial hypertension peaks during endotracheal ASP procedure. In this type of patient, endotracheal ASP is necessary but raises potential adverse effects. To prevent intracranial hypertension setup, the ASP maneuver time must be lower than 10 seconds and limited to one or at maximum two procedures. Due to ICP rising, observed later than 10 minutes after ASP conclusion (recognized to provoke mean ICP and MBP increased values), this rest period must be prolonged.

Opposing to the latter, this study observed the return of ICP basal values, as well as CPP levels maintenance, on the tenth minute after ASP conclusion with no signs of cerebral ischemia or hyperemia (evaluated by $PjvO_2$ and $SjvO_2$ data). Even by that, we are unable to assure the absent of cerebral vasodilatation and CBF levels risings (the most probable causes of increased ICP during endotracheal ASP). In order to confirm this hypothesis, it is necessary to carry on transcranial Doppler evaluation.

Cruz[21] reported that ASP increased MBP and ICP levels along with SjO_2 elevation but with no observed risings in systemic arterial and venous oxygenation. This study suggests that the elevations induced by ASP in MBP also increased CBF values. Fortune et al.[10] reported MBP, CPP and $SjvO_2$ increasings accompanied by a decrease of the oxygen extraction associated to ICP augmentation. The study also assures that the observed risings, induced by ASP, were resulted from CBF elevation due to vasodilatation accompanied by $PjvO_2$ maintenance.

Fortune et al.[10] found dramatic increases in $SjvO_2$ associated to ASP while Kerr et al.[22] did not observe $SjvO_2$ fluctuations once, as in this study, some aspects of the ASP protocol, more likely to influence ICP elevations and/or cerebral oxygenation changes, were controlled (e.g.: hyper-oxygenation, ASP period duration, FiO_2 and catheter type).

Although hypoxemia induced by ASP was quoted to justify MBP risings, there were clear evidences pointing to the absence of hypoxemia in these patients with elevated MBP levels as it was found in this study and in Kerr et al.[23], Rudy et al.[20], Kerr et al.[22,23]. In all the quoted studies, the ASP induced hypoxemia in HI patients was prevented by hyper-oxygenation, previous to ASP (well documented by its benefic effects in other acute injured patients' samples). These studies point to the fact that in the absence of hypoxemia, ICP elevations appear synchronically with MBP elevations. In this study, we found ICP and MBP risings while the hypoxemia, ASP induced, was controlled by previous hyper-oxygenation as recommended in Skelley et al. and Urban and Weitzner *apud* Keer et al.[23].

Kerr et al.[23] depicted that ASP, preceded by hyper-oxygenation, was associated to blood flow velocity and arterial pressure risings. Significant decrease in the pulsatility index suggests that vasodilatation plays important part as a mechanism to maintain cerebral oxygenation in the presence of ICP raisings.

Therefore, it can be concluded that transient ICP and MBP elevations, associated with ASP procedure preceded by cerebral oxygenation, do not seem to provoke hazardous effects in cerebral oxygenation once $PjvO_2$, $SjvO_2$ and CPP maintain its desirable levels.

By preserving pulmonary alveoli function, one can guarantee optimized ventilation efficacy. Losses of gas trade zones compromise $PaCO_2$ controlling, raising critical features to the treatment mainly in the event of cerebral hyperemia. Consequently, in terms of beneficial effects, respiratory physiotherapy maneuvers must be managed in HI patients.

Thus, the respiratory physiotherapy, through VBC, EFI and ASP maneuvers, seems to be a required condition since attention is given to ICP. In effort to aloud optimal tracheobronquial hygiene, physiotherapy procedures must be performed in patients submitted to maximum sedation, always avoiding any other unnecessary stimulus. By not performing the procedure, secretions accumulation may lead to hypoventilation.

In summary, VBC and EFI respiratory physiotherapy maneuvers do not cause alterations in cerebral hemodynamics related to the MBP, ICP, CPP, $PjvO_2$ and $SjvO_2$ variables. Consequently, the procedures can be safely accomplished in sedated and curarized patients presenting HI and submitted to mechanical ventilation.

The tracheal ASP procedure provokes changes in cerebral hemodynamics leading to MBP risings with concomitant ICP elevation and maintenance of CPP, $PjvO_2$ and $SjvO_2$ levels. By this way, ischaemic events due to ASP can be avoided since MBP and ICP return to its basal values after 10 minutes of tracheal ASP conclusion.

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