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Acute effects of physiotherapeutic respiratory maneuvers in critically ill patients with craniocerebral trauma

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OBJECTIVE: To evaluate the effects of physiotherapeutic respiratory maneuvers on cerebral and cardiovascular hemodynamics and blood gas variables.

METHOD: A descriptive, longitudinal, prospective, nonrandomized clinical trial that included 20 critical patients with severe craniocerebral trauma who were receiving mechanical ventilation and who were admitted to the intensive care unit. Each patient was subjected to the physiotherapeutic maneuvers of vibrocompression and increased manual expiratory flow (5 minutes on each hemithorax), along with subsequent airway suctioning with prior instillation of saline solution, hyperinflation and hyperoxygenation. Variables related to cardiovascular and cerebral hemodynamics and blood gas variables were recorded after each vibrocompression, increased manual expiratory flow and airway suctioning maneuver and 10 minutes after the end of airway suctioning.

RESULTS: The hemodynamic and blood gas variables were maintained during vibrocompression and increased manual expiratory flow maneuvers; however, there were increases in mean arterial pressure, intracranial pressure, heart rate, pulmonary arterial pressure and pulmonary capillary pressure during airway suctioning. All of the values returned to baseline 10 minutes after the end of airway suctioning.

CONCLUSION: Respiratory physiotherapy can be safely performed on patients with severe craniocerebral trauma. Additional caution must be taken when performing airway suctioning because this technique alters cerebral and cardiovascular hemodynamics, even in sedated and paralyzed patients.

KEYWORDS: Physical Therapy Modalities; Craniocerebral Trauma; Intensive Care.

TRIAL REGISTRATION: This trial was not registered because enrollment began prior to July 1, 2005.

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INTRODUCTION

Physiotherapeutic interventions during motor and cardiorespiratory care are considered to be important components in the management of patients admitted into intensive care units (1–3). These interventions offer short- and medium-term benefits for the patients' recovery (4).

Particularly in patients with severe craniocerebral trauma, the application of different physiotherapeutic interventions in the intensive care unit, whether singly or in combination, results in adaptations in various systems that can lead to clinically significant changes in respiratory function and cardiovascular and cerebral hemodynamics (3,5,6). The continuous monitoring of hemodynamic and respiratory parameters in critical patients provides additional safety while performing therapy and during patient follow-up, rendering therapy safer and more effective and thereby reducing the factors that can potentially contribute to adverse events (6).

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Therefore, this study focused on assessing the effects of the physiotherapeutic respiratory maneuvers of vibrocompression (VBC), increased expiratory flow manual (IEFM) and airway suctioning (AS) on hemodynamic variables (mean arterial pressure [MAP], intracranial pressure [ICP], cerebral perfusion pressure [CPP], heart rate [HR], central venous pressure [CVP], cardiac output [CO], pulmonary artery pressure [PAP] and pulmonary capillary pressure [PCAP]) and on blood gas variables (arterial oxygen tension [PaO_2], the ratio of the arterial oxygen tension to the fractional inspired oxygen [PaO_2/FiO_2], arterial carbon dioxide tension [$PaCO_2$], arterial saturation [SaO_2], jugular oxygen tension [PjO_2], jugular bulb venous oxygen saturation [$SvjO_2$], the arterio-jugular difference in the oxygen content [$AJDO_2$] and cerebral extraction of oxygen [CEO_2]) in patients with severe craniocerebral trauma admitted into the intensive care unit.

MATERIALS AND METHODS

Design

A descriptive, longitudinal, prospective, nonrandomized clinical trial was quantitatively conducted and was approved by the research and ethics committee of the Hospital de Clínicas at the Federal University of Paraná (protocol number 065EXT019/2002-11), and family members of the subjects provided informed consent.

Participants, therapists and centers

The study included consecutive male and female adult patients with severe craniocerebral trauma (Glasgow Coma Scale ≤ 8), who were selected within the first 48 hours after they were admitted into the intensive care unit. The patients included in the study presented no radiological alterations in their lungs, were sedated with propofol (Ramsay scale of 6), were paralyzed with pancuronium bromide, had their cough reflexes inhibited, were intubated and received mechanical ventilation in the assisted/controlled volume control ventilation mode (which was estimated at 8 ml/kg of body weight) with a positive end-expiratory pressure of 5 mmHg and an inspired oxygen fraction of 40%. Patients presenting any of the following criteria were excluded from the study: hemodynamic instability with a MAP of less than 60 mmHg; a CPP of less than 50 mmHg; pulmonary contusions; fractured ribs; or an undrained pneumothorax.

Intervention and outcome measurements

The patients were subjected to intra-arterial catheterization for MAP monitoring; arterial blood sample collection for blood gas analysis (PaO_2, PaO_2/FiO_2, $PaCO_2$, SaO_2); intraventricular catheterization to measure the ICP and calculate the CPP; and pulmonary artery catheterization (Swan-Ganz) to monitor the CVP, CO, PAP and PCAP. All of the catheters were connected to a multi-parameter monitor, through which the HR was also monitored. A jugular-bulb catheter was also placed to collect venous blood samples for blood gas analysis, to assess PjO_2 and $SvjO_2$ and to calculate $AJDO_2$ and CEO2. Each patient was then placed into a dorsal decubitus position, with his or her head elevated at 30°, and all of the variables were assessed to establish baseline values. The physiotherapeutic maneuvers of VBC and IEFM (5 minutes for each hemithorax) were subsequently applied for 10 minutes each. VBC was performed first to modify the physical properties of the mucus, resulting in reduced viscosity due to the thixotropy provided by this maneuver. The IEFM maneuvers followed and were responsible for mobilizing, moving and eliminating secretions from the periphery toward the trachea via increased expiratory flow. AS was then performed but was preceded by a 5-ml instillation of saline solution (0.9%) and three hyperinflations and hyperoxygenations using a manual resuscitator connected to an O_2 source (flow of 10 l/s). A disposable, sterile probe with a valve was used for 10 to 15 seconds during this procedure, which was only performed once. To avoid cumulative effects, an interval of 5 minutes was established between physiotherapeutic maneuvers (7). Figure 1 provides design and flow of the participants through the trial.

Data analysis

The data were analyzed using statistical analysis software (Statistica). When comparing the procedures across time points, the null hypothesis of equal means at all of the time points was tested *vs.* the alternative hypothesis of at least one time point with a different mean. Repeated measures

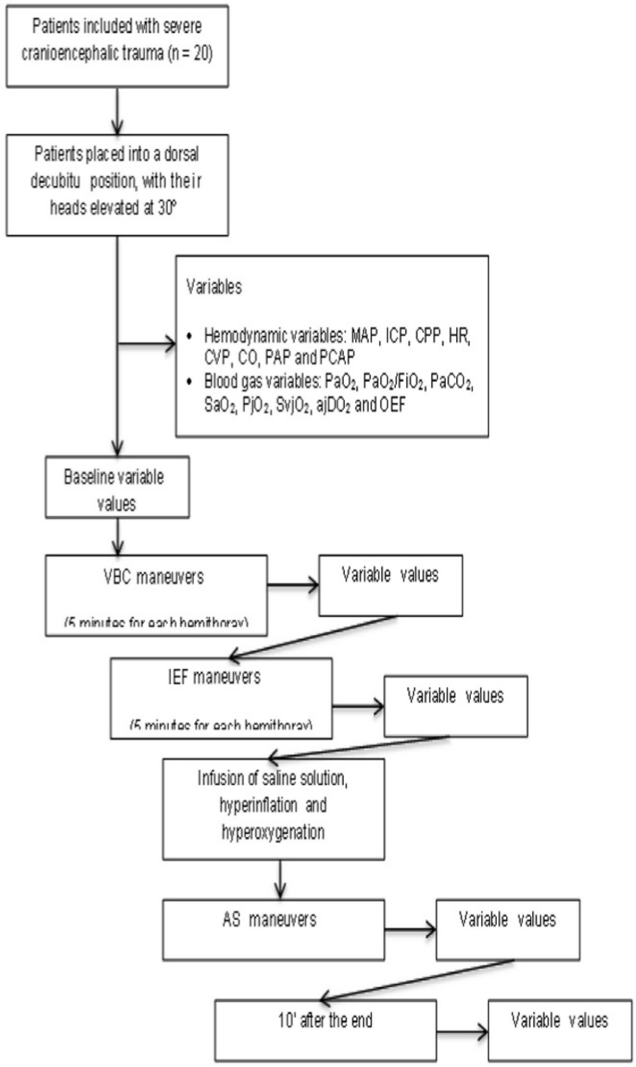
Patients included with severe craniocephalic trauma (n = 20)
Patients placed into a dorsal decubitu position, with their heads elevated at 30°
Variables
Hemodynamic variables: MAP, ICP, CPP, HR, CVP, CO, PAP and PCAP
Blood gas variables: PaO₂, PaO₂/FiO₂, PaCO₂, SaO₂, PjO₂, SvjO₂, ajDO₂ and OEF
Baseline variable values
VBC maneuvers
(5 minutes for each hemithorax)
Variable values
IEF maneuvers
(5 minutes for each hemithorax)
Variable values
Infusion of saline solution, hyperinflation and hyperoxygenation
AS maneuvers
Variable values
10' after the end
Variable values

Figure 1 - Design and flow of participants through the clinical trial.

Table 1 - The results of the cerebral hemodynamic variables, reported as means and standard deviations.

	VBC			IEFM			AS		
	Before	After	p	Before	After	p	Before	After	*p*-value
MAP (mmHg)	94.00±18.78	93.00±17.54	0.451	94.70±18.17	96.65±21.61	0.209	94.25±18.40	100.65±22.19	0.011*
ICP (mmHg)	16.95±7.25	16.80±7.68	0.877	17.75±7.78	18.80±7.35	0.107	19.65±8.24	26.35±12.82	<0.001*
CPP (mmHg)	76.90±22.95	76.15±22.38	0.475	76.90±22.95	77.65±25.80	0.634	74.60±21.97	75.15±28.77	0.833

Before = pre-maneuver; After = post-maneuver; MAP = mean arterial pressure; ICP = intracranial pressure; CPP = cerebral perfusion pressure; VBC = vibrocompression; IEFM = increased expiratory flow manual; AS = aspiration; Student's t test, * = $p<0.05$.

analysis of variance was used for this analysis. If the null hypothesis was rejected, the time points were subjected to pair-wise comparisons using the Newman-Keuls test. When comparing only two time points, Student's t-test for analyzing dependent variables was used. The normality of the data was assessed using the Kolmogorov-Smirnov test. Statistical significance was defined as $p<0.05$.

RESULTS

Twenty patients were included in the study, including 18 men and two women with a mean age of 33.5±11.9 years old, an average Apache score of 26±4 and a mean Ramsay score of 6.

The VBC and IEFM maneuvers did not produce any significant changes in the cerebral hemodynamic variables of MAP, ICP or CPP compared to their values before and after these maneuvers. However, significant variations in the MAP and ICP values were identified immediately after the AS maneuver (see Table 1). There were no statistically significant differences in MAP ($p = 0.85$) or ICP ($p = 1.00$) before AS or 10 minutes after the final procedure, indicating that these variables had returned to baseline. The cerebral perfusion pressure remained unchanged throughout the protocol.

Table 2 presents the data on the behavior of the cardiovascular hemodynamic variables at baseline; after performing the VBC, IEFM and AS maneuvers; and 10 minutes after the end of the protocol. Statistically significant changes were specifically identified for the HR, PAP and PCAP variables when comparing the AS, VBC and IEFM values to baseline, and the variables returned to baseline 10 minutes after the end of AS. No significant differences were observed for arterial or jugular bulb blood gas variables at any time during the protocol (see Table 3).

DISCUSSION

The results indicate that physiotherapeutic maneuvers do not alter cardiovascular hemodynamics. Rib cage compression can increase intrathoracic pressure, which increases the pressure in the right ventricle, reducing the venous return (7). However, no increases were noted in pressure (CVP, PAP and PCAP) in this study, suggesting that venous return and cardiac preload and afterload were maintained throughout the VBC and IEFM maneuvers. Discrepancies between studies could be related to differences in the rate of vibration and the force applied during the maneuvers, among other features (8,6).

In addition, in our study, all of the patients were sedated and paralyzed, which might explain the absence of hemodynamic effects. Another possible difference might be related to the lack of change in decubitus, the evaluation of the maneuvers in isolation and the prior analysis of the variables before suctioning, which did not occur in the previous studies.

Regarding blood gas variables, several studies have shown that VBC and IEFM maneuvers, alone and in combination with endotracheal suctioning, did not alter the oxygenation or ventilation of patients (9–11), corroborating our data.

Regarding cerebral variables following VBC and IEFM maneuvers alone, studies have shown that percussion and vibration do not adversely affect ICP, and manual rib-cage vibration and manual expiratory pressure maneuvers do not alter the MAP, ICP or CPP of patients with severe craniocerebral trauma (7,12). Thus, our study confirms the safety of applying physiotherapeutic respiratory maneuvers in patients with severe craniocerebral trauma, with no significant effects on MAP, ICP, CPP, PjO_2, $SvjO_2$, $AJDO_2$ or CEO_2, thereby demonstrating that there were no signs of cerebral ischemia or hyperemia during these procedures.

The significant increases in the ICP and MAP during AS could be explained by two factors. First, carinal stimulation by the AS catheter promotes vagal stimulation, which results in cerebral vasodilation and a consequent increase in ICP. Second, given the absence of analgesics, the likely presence of pain during the procedure might have contributed to the ICP elevation in this study due to an increase in sympathetic activity with a reduction in cerebral blood flow, resulting in changes in CPP (13). A third, less likely, factor is related to hyperinflation and hyperoxygenation

Table 2 - The results of the cardiovascular hemodynamic variables, reported as means and standard deviations.

	BASAL	VBC	IEFM	AS	10' AFTER	*p*-value
HR (beats/min)	92±17.95	93±18.36	94±18.69	99±20.75°	94±20.49	0.004*
CVP (mmHg)	14.25±3.91	14.25±4.30	14.80±4.89	15.25±4.48	14.90±3.49	0.508
CO (L/min)	9.72±3.20	11.24±4.22	10.70±3.48	10.97±3.69	10.35±3.52	0.060
PAP (mmHg)	25.00±4.94	24.80±4.74	25.50±5.68	27.40±5.53°	25.65±5.32	0.002*
PCAP (mmHg)	15.25±3.43	14.55±3.27	14.85±3.95	17.00±4.07°	15.50±3.12	<0.001*

HR = heart rate; CVP = central venous pressure; CO = cardiac output; PAP = pulmonary artery pressure; PCAP = pulmonary capillary pressure; BASAL = before physiotherapy maneuvers; VBC = vibrocompression; IEFM = increased expiratory flow manual; AS = aspiration; 10' AFTER = 10 minutes after the end of the AS; ANOVA, * = $p<0.05$; Newman-Keuls ° = $p<0.05$.

Table 3 - The results of the blood gas variables, reported as means and standard deviations.

	BASAL	VBC	IEFM	AS	10' AFTER	*p*-value
PaO_2 (mmHg)	107±25.12	105±42.09	111±46.79	100±41.79	107±40.84	0.479
PaO_2/FiO_2	262±74.43	253±116.24	266±129.59	235±116.90	261±110.07	0.216
PCO_2 (mmHg)	30±6.54	30±7.00	30±8.18	31±9.25	32±8.31	0.437
SaO_2 (%)	97±1.95	97±2.60	97±2.54	97±2.08	97±2.76	0.456
PjO_2 (mmHg)	45±8.64	45±13.51	44±12.59	45±12.24	45±11.25	0.913
SjO_2 (%)	78±7.22	77±8.85	75±8.61	76±8.13	76±7.97	0.355
$AJDO_2$ (ml/dl)	2.68±0.77	2.80±0.99	3.03±1.03	2.82±1.07	2.88±1.03	0.414
CEO_2 (%)	19±5.95	20±7.75	22±8.21	21±8.03	21±7.85	0.467

Arterial oxygen tension (PaO_2), the ratio of the arterial oxygen tension to the fractional inspired oxygen (PaO_2/FiO_2), arterial carbon dioxide tension ($PaCO_2$), arterial saturation (SaO_2), jugular oxygen tension (PjO_2), jugular bulb venous oxygen saturation ($SvjO_2$), the arterio-jugular difference in the oxygen content ($AJDO_2$), and cerebral extraction of oxygen (CEO_2); BASAL = before physiotherapy maneuvers; VBC = vibrocompression; IEFM = increased expiratory flow manual; AS = aspiration; 10' AFTER = 10 minutes after the end of the AS; ANOVA, $p>0.05$.

prior to AS, which induce increases in intrathoracic pressure, impair cerebral venous return and thus elevate ICP. However, the CVP and CO values were maintained, suggesting that the intrathoracic pressure did not generate hemodynamic effects (13).

Several studies have attested that sedation combined with neuromuscular blocking (NMB) is inversely correlated with increases in ICP (14–16). Neuromuscular blocking paralyzes the intercostal muscles and diaphragm and could be responsible for attenuating the expected increases in ICP that occur during endotracheal AS (17). Moreover, stabilization occurred, and ICP returned to baseline 10 to 15 minutes after the maneuvers, in agreement with the findings of this study (13).

Other studies have shown that increases in MAP were not prevented by neuromuscular blocking and that increased ICP can occur in the absence of cerebral self-regulation (18,19). These observations support the results of this study, in which increases in MAP occurred during and immediately after endotracheal AS. The cerebral perfusion pressure was not significantly affected, given the concomitant increases in MAP and ICP.

When assessing PjO_2, $SvjO_2$, $AJDO_2$ and CEO_2 in this study, no signs of cerebral ischemia or hyperemia were observed. Nevertheless, we cannot confirm the absence of cerebral vasodilation and increased cerebral blood flow, which would constitute reasons for increased ICP during endotracheal AS. A transcranial Doppler evaluation would be required to confirm this hypothesis (20).

Previous studies have observed changes in the behavior of PjO_2 and $SvjO_2$, combined with increased MAP and ICP, either with or without changes in CPP and cerebral blood flow (20–22). These results might be explained by the various AS protocols used by the authors.

Although AS-induced hypoxemia has been suggested to explain the increase in MAP, there is clear evidence that hypoxemia did not occur in patients with increased MAP; hyperoxygenation prior to AS assured cerebral oxygenation and provided beneficial effects. However, even in the absence of hypoxemia, increases in ICP remain, combined with synchronous elevations in MAP, as observed in previous studies (10,12,20,23).

The increase in HR observed in this study had no clinical significance, and HR returned to baseline after 10 minutes. These findings agree with other studies in which a slight increase in HR was observed during AS in sedated and paralyzed patients that immediately resolved 2 to 5 minutes after the end of AS (10,24,25).

Regarding the transient increases in PAP and PCAP during AS, three facts should be taken into account (26). First, the manual hyperinflation performed prior to AS increases pulmonary vascular resistance and can generate significant pulmonary hypertension (27,28). Conversely, hyperinflation compresses the heart between the expanded lungs, increasing the right ventricular pressure (reflected by PAP) and left atrial pressure (reflected by PCAP). The second factor is related to hypoxemia. However, due to hyperoxia being performed prior to AS, all of the patients stably maintained their oxygen levels during the protocol (29). The third and most likely factor to have changed PAP and PCAP was the presence of pain and subsequent sympathetic activation and vasoconstriction because of the absence of an analgesic, unlike in previous studies (30).

Therefore, we suggest that in patients with severe craniocerebral trauma, AS should be performed in combination with sedation, analgesia, and neuromuscular paralysis; additionally, AS should be carefully applied via a clinical evaluation and ventilatory mechanics (impedance) to avoid accumulating additional hemodynamic changes (even transient changes) in these patients.

The physiotherapeutic respiratory maneuvers of VBC and IEFM did not result in acute changes in cerebral hemodynamics, cardiovascular variables or blood gas variables. Therefore, these maneuvers can be safely performed on sedated or paralyzed patients with CET who are receiving MV.

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■ AUTHOR CONTRIBUTIONS

Neto ML conceived and designed the research, acquired the data, analyzed and interpreted the data and drafted the manuscript. Cerqueira TC analyzed and interpreted the data and provided critical revisions of the manuscript for important intellectual content. Aquim EE and Moura AV conceived and designed the research and analyzed and interpreted the data. Oliveira MC conceived and designed the research and acquired the data. Rea-Neto A, Silva Júnior VM and Santana-Filho VJ provided critical revisions of the manuscript for important intellectual content. Scola RH conceived and designed the research, analyzed and interpreted the data and provided critical revisions of the manuscript for important intellectual content.

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