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Effects of rapid chest compression technique on intracranial and cerebral perfusion pressures in acute neurocritical patients: a randomized controlled trial

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Abstract

Background Some studies refer to the increase in intracranial pressure (ICP) with chest physiotherapy techniques but without any randomized controlled trials that evaluate the safety of the manual rapid chest compression technique in patients with severe acute brain injuries on invasive mechanical ventilation. Our research question examines whether intracranial and cerebral perfusion pressures significantly change during rapid chest compression technique.

Methods A prospective, randomized, single-blinded controlled trial of acute neurocritical patients under mechanical ventilation was performed. The intervention group was subjected to rapid chest compression, and the control group received mechanical passive inferior limbs mobilization. The outcomes were intracranial pressure, cerebral perfusion pressure, blood partial pressure of oxygen and carbon dioxide, and inspiratory and expiratory peak flows.

Results Between May 2021 and December 2023, 50 patients (aged 56.3 years), 66% females, were randomized into two groups (25 controls and 25 interventions). The ICP and cerebral perfusion pressure (CPP) did not significantly differ between the groups at any of the studied times. Intragroup analysis revealed significant decreases in the ICP and CPP in the intervention group, with posterior recovery in both groups. The CPP significantly decreased in the control group but did not reach the preintervention values at the last measurement time. $PaCO_2$ was significantly lower in the intervention group than in the control group at the end of the study.

Conclusion The rapid chest compression technique did not increase the ICP during its application or even 30 min after it. The ICP showed a slight significant decrease during the application of the rapid chest compression technique but reached the previous values in the posterior 30 min. CPP had a similar behavior but did not completely recover in both groups.

Trial registration: NCT03609866. Registered on 08/01/2018.

Keywords Acute brain injury, Intracranial pressure, Cerebral perfusion pressure, Chest physiotherapy, Respiratory physiotherapy

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Introduction

Acute brain injuries (ABIs) are a critical group of pathologies that frequently require invasive mechanical ventilation, sedation and sometimes relaxation. The main pathologies of this group include traumatic brain injury (TBI), acute ischemic stroke (AIS), subarachnoid hemorrhage (SAH), and intracerebral hemorrhage (ICH) [1, 2].

Mechanical ventilation leads to limitation of respiratory secretion removal caused by the endotracheal tube presence, the air flow changes, and the patient positioning. Sedation and relaxation medication also can compromise ciliary function [3, 4].

The chest physiotherapy in intensive care usually includes vibration, percussion, flow modulation techniques, cough assistance and hyperinflation. These techniques can be used with the goal of preventing mucus retention, atelectasis or ventilation-associated pneumonia [5].

Some chest physiotherapy techniques employed in intensive care units aim to modulate ventilatory flows and improve mucus transport based on the concept of dual gas–liquid phase principle. Laboratory evidence supports the use of these techniques, suggesting that mucus removal is more effective when expiratory flow exceeds inspiratory flow. For example, the rapid chest compression technique (RCCT) is designed to increase expiratory flow and aid in mucus transport out of the bronchial tree [4, 6].

Several studies have investigated the effectiveness of RCCT in mobilizing secretions and enhancing peak expiratory flow. While Felipe and Reis [7] applied thoracic compression in an animal model with atelectasis but reported that it was ineffective at resolving atelectasis and mobilizing secretions. More recent research by Martí et al. [6] demonstrated that RCCT effectively mobilized secretions outward from the bronchial tree in pigs undergoing invasive mechanical ventilation [6, 7].

Studies on humans have studied expiratory rib or chest compression. While Naue et al. [8] found no difference in suctioned secretions with added pressure support, their 2014 trial showed increased secretion clearance, exhaled volume, and pulmonary dynamics when combined with higher inspiratory pressure. Similarly, Guimarães et al. [10] reported improved peak expiratory flow with rapid thoracic compression, reinforcing the effectiveness of these techniques [8–10].

Several studies have attempted to understand the safety of some respiratory physiotherapy techniques in neurocritical patients.

This is the case for the effects of manual RCCT on intracranial pressure (ICP) in patients with severe acute brain injuries. The first clinical study on this matter was carried out by Thiesen et al. [11], who studied the effects of several techniques of chest physiotherapy on ICP, among which manual thoracic compression was included with satisfactory results with respect to the control of ICP [11].

Studies on chest physiotherapy in patients with acute brain injury show mixed effects on intracranial pressure (ICP) [12]. Toledo et al. [12] and Cerqueira-Neto et al. found no significant impact of respiratory physiotherapy on ICP or cerebral perfusion pressure (CPP) [13, 14]. However, Ferreira et al. reported increased ICP with no significant CPP changes [15]. More recently, Tomar et al. found that manual chest percussion raised ICP compared to mechanical vibration [16].

RCCT has a possible benefit in terms of secretion removal, but the real safety of its use in neurocritical patients is still unclear.

Therefore, the research questions for our single-blinded randomized controlled trial in neurocritical patients were as follows:

1. Does intracranial pressure significantly change during and immediately after rapid chest compression technique?
2. Does cerebral perfusion pressure change during and immediately after rapid chest compression?

Methods

Design

A prospective, randomized, single-blinded controlled trial of acute neurocritical patients under mechanical ventilation was performed. The trial protocol was registered (ClinicalTrials.gov NCT03609866), published [17], adapted to the CONSORT guidelines [18], and reported via the template for intervention description and replication (TIDieR) checklist [19].

The sample size was obtained with the software ENE 3.0, and on the basis of the estimations of the changes in intracranial pressure with the techniques performed in the study by Thiesen et al. [11], with a power of 80% and a level of confidence of 95%, we obtained a sample size of 25 patients in each group: control and experimental. A total of 50 subjects were included in this trial [11].

Patients with acute severe brain injury were selected from the intensive care unit of the Hospital Álvaro Cunqueiro of Vigo, Galicia, Spain, which is a tertiary hospital with neurocritical patients and the reference neurocritical center for southern Galicia. The randomization and allocation sequences were generated on the GraphPad QuickCalcs page (https://www.graphpad.com/quickcalcs/randomize2/), with no repetition and 50 subjects allocated to 2 groups (blocked randomization), with the template of allocation designed at the beginning of the

study and then placing the subjects by their entrance date in the intensive care unit. The participants were allocated to each group by the principal investigator after the acceptance and signing of the informed consent form by the next of kin. This study was approved by the Comité de Ética de la Investigación de Pontevedra-Vigo-Ourense (ethics board of the Galician public health system) with the registration number 2018/446.

We considered patients to be blinded, as all patients had a Richmond Agitation and Sedation Scale score of −5, which did not allow patients to understand if they were in the control or intervention group [20, 21].

Statistician personnel were blinded, as the data were sent for analysis without providing group information about the control or intervention [22].

Study population

We included acute neurocritical patients (TBI, AIS, SAH, and ICH) with more than 48 h of invasive mechanical ventilation, intracranial pressure monitoring, continuous arterial pressure monitoring and Glasgow Coma Scale (GCS) ≤8 before sedation or surgical procedures. The inclusion and exclusion criteria are detailed in Table 1.

Descriptive information such as age, sex, type of injury, presence of ventricular drainage, norepinephrine and relaxation drugs are presented in Table 2.

Intervention

When ventricular drainage was present, the external ventricular drain was maintained open, positioned at a height of +20 cm above the external auditory canal.

The intervention group was subjected to rapid chest compression. This technique consists of the application of rapid compression of the lower lateral area of the thorax bilaterally when the ventilator makes the transition from inspiration to expiration; the purpose is to obtain a flow bias of 33 L/m or higher, favoring expiration [4]. This was performed with the patient in the supine decubitus position with 30° elevation of the head and trunk, and the rapid chest compression technique was applied

Table 1 Eligibility criteria

Inclusion criteria	1
Intubated patients with mechanical ventilation in Volume Controlled, Pressure Controlled and Volume Controlled Pressure Regulated modes during the 48 h prior to the application of the technique	
Severe head injury with a GCS ≤8	
Hemodynamic stability (MAP >65 mmHg)	
Respiratory stability (PEEP <10 cm H_2O and FiO_2 <60%)	
Intracranial Pressure Stability (0 <ICP <20 mmHg)	
RASS sedation scale of −5 points	
Exclusion criteria	
Thoracic fractures: unstable rib, sternal, clavicular, scapular, or vertebral	
Abdominal injuries that limit the application of local manual compression	
Systemic or local changes that evolve with increased abdominal volume	
Fractures or conditions in the lower limbs that contraindicate passive mobilization	

Table 2 Sample description (ABI: acute brain injury) n = 50

	All patients		Control group		Intervention group	
Age	56		55		57	
Time since ABI (mean days)	3.3		3.3		3.3	
Sex (% females)	33	66%	14	56%	19	76%
Traumatic brain injury (TBI	12	24%	7	28%	5	20%
Acute ischemic stroke (AIS)	2	4%	1	4%	1	4%
Subarachnoid hemorrhage (SAH)	20	40%	8	32%	12	48%
Intracerebral hemorrhage (ICH)	16	32%	9	36%	7	28%
Ventricular drainage	22	44%	9	36%	13	52%
Norepinephrine	32	64%	17	68%	15	60%
Decompressive surgery	19	38%	9	36%	10	40%
Relaxation	14	28%	8	32%	6	24%

once during the transition expiratory time of each of the 3 respiratory cycles for 5 min by a physiotherapist. Safety monitoring of heart rate, blood pressure, intracranial pressure, peripheral oxygen saturation and cerebral perfusion pressure was performed by the clinical team and the principal investigator. The intervention was stopped if some of the safety values were reached or if the clinical team considered it.

The control group received mechanical passive inferior limb mobilization at a frequency of 15 cycles per minute. The patient was in the supine decubitus position with a 30° elevation of the head and trunk. The inferior limbs passive mobilization technique was applied in the middle of the knee range of motion for 5 min via a motorized cycloergometer. Safety monitoring of heart rate, blood pressure, intracranial pressure, peripheral oxygen saturation and cerebral perfusion pressure was performed by the clinical team and the principal investigator. The intervention would have been stopped if it reached some of the safety values (ICP >15 mmHg in decompressed patients or ICP >20 mmHg in non-decompressed patients; Oxygen saturation <94%; Cardiac frequency >130) or when the clinical team considered it [23–25].

Outcomes

The main and secondary outcomes were collected as described below.

Intracranial pressure (ICP)

The intracranial pressure (mmHg) data were recorded 1 h prior to the intervention, when changed the mechanical ventilation mode (if applied), 30 min prior, 5 min prior, at the beginning of the intervention and every minute since the beginning, and 5 and 30 min since the end, with the continuous intracranial pressure monitoring system. When ventricular drainage was present, the external ventricular drain was positioned at +20 cmH_2O above the external auditory canal and remained open, beginning 5 min prior to the start of the technique and continuing until its completion. Data were recorded via an Integra Camino ICP monitor and with an intraventricular placed pressure sensor.

Heart rate, blood pressure and cerebral perfusion pressure

The data from the 3 outcomes were recorded directly from vital signs monitor with the time chart as the ICP (except 60 and 30 min prior).

Expiratory and inspiratory peak flow

Data were collected over 30-s periods as in the ICP time chart (except 60- and 30-min periods). All expiratory and inspiratory peak flows were checked, and the bests were recorded from every 30 s sequence.

Blood pH, O_2 and CO_2

Recent studies have identified tight control of the lower limits of blood CO_2 partial pressure as a protective factor in cases of brain injury because high CO_2 levels induce arterial vasodilatation, as shown in laboratory studies [26]. We performed arterial blood gas analysis 5 min prior to and at the end of the intervention.

Statistical analysis

We present descriptive statistics as absolute numbers and percentages and as the means and SDs (standard deviations) or medians and IQRs (interquartile ranges), depending on normality. To assess the normality of the variables, the Shapiro–Wilk test was used.

We registered the variables at 5 min prior to the intervention (T −5) and the beginning (T0), at every minute from the beginning until the end of the intervention (T1, T2, T3, T4, T5), 5 min later (T +5) and 30 min later (T +30).

To assess whether there were differences in the values of ICP, CPP, or $PaCO_2$ between patients in the control and intervention groups at each study time point (T −5, T0, T1, T2, T3, T4, T5, T + 5, and T + 30), Student's t test or the Mann–Whitney U test was used, depending on the normality of the quantitative variables. To evaluate differences over time in ICP, CPP, or $PaCO_2$ within each group (control and intervention), repeated-measures ANOVA or the Friedman test was applied, depending on the normality of the data. Values of $p < 0.05$ were considered statistically significant. We took the T − 5 time as a reference value, as it reflects the basal status prior to any intervention and the means/medians compared with it, although we compared the means/medians with other times (supplemental material).

Results

Between May 2021 and December 2023, the recruitment process assessed 55 possible subjects; 5 were excluded, 3 did not meet the inclusion criteria, and 2 refused to participate, resulting in the allocation of 50 subjects, as shown in Fig. 1. The mean ICP was 9.40 mmHg (SD 5156) and 8.24 mmHg (SD 3756) in the control and intervention groups, respectively. There were no significant differences between the groups at any time (Fig. 2). According to the intragroup analysis, as shown in Table 3, the ICP mean was significantly lower during the rest period (T −5) at T1, T2, T3 and T5 in the intervention group and significantly inferior to T − 5 at T1 and T4 in the control group. When the preintervention rest period was compared with the late postintervention time (T +30), there were no significant intragroup differences. When

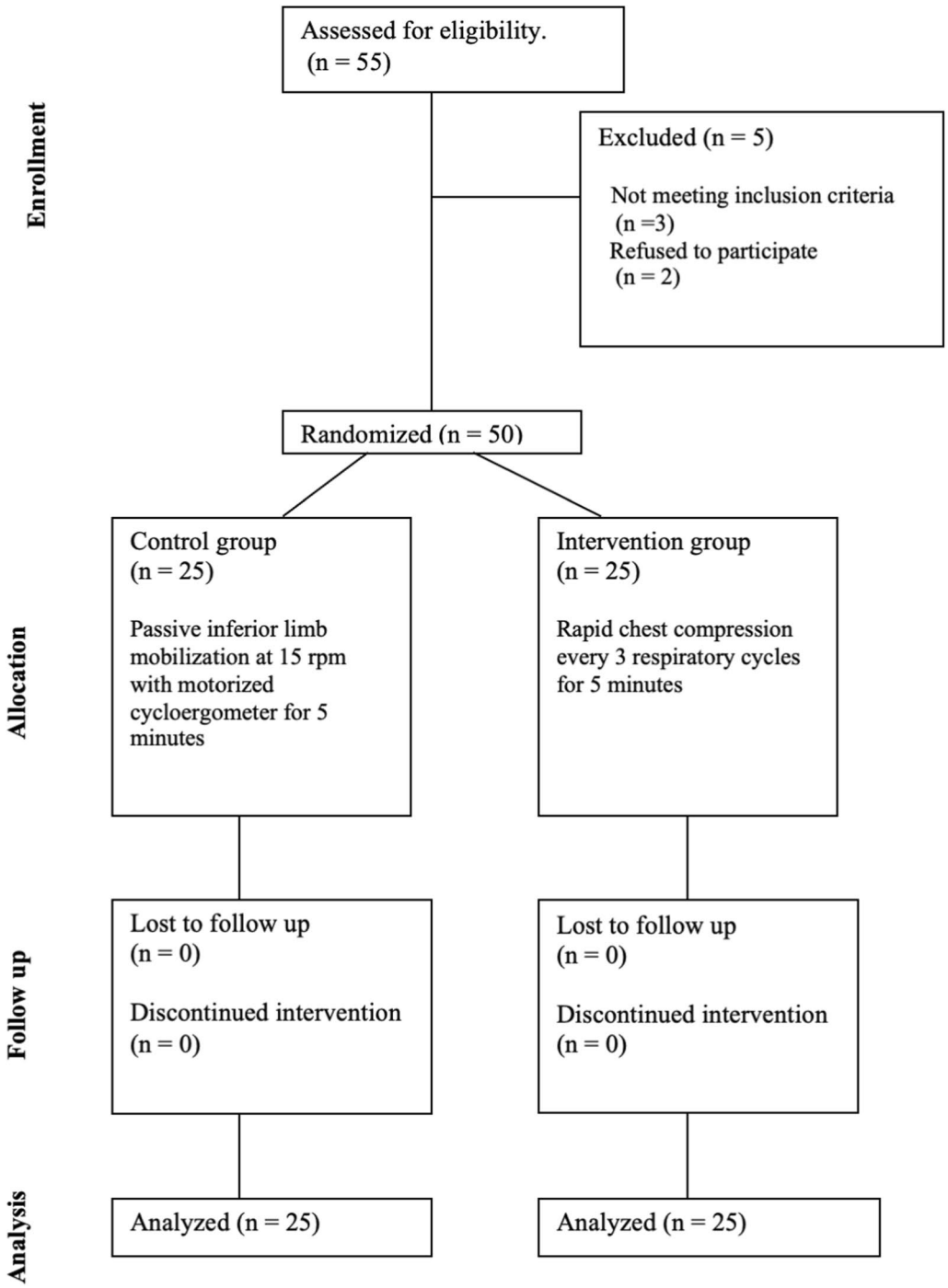

Fig. 1 Consort flow chart

analyzed the data of patients with ICP ≥ 10, as shown in Fig. 3, there was a similar behavior to the general group analysis, without significant differences at any time.

The median CPP were 76 mmHg (SD 16,927) and 80 mmHg (SD 18,127) in the control and intervention groups, respectively. No significant differences were found between the groups at any time. Compared with the preintervention rest period, the CPP was significantly inferior at T1, T2 and T4 during the intervention period and at T + 5 postintervention in the intervention group and at T3, T5 and T + 30 in the control group (Fig. 4).

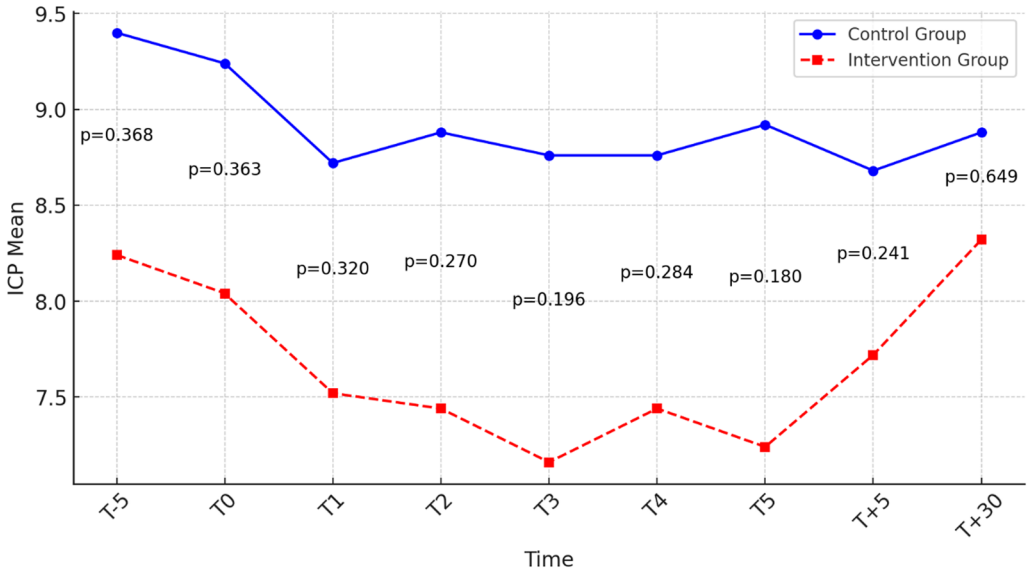

Fig. 2 ICP means at the measured times and *p* values between groups

Table 3 Intragroup mean or median values compared with the rest period (T − 5) and the respective *p* values

		T − 5	T0	T1	T2	T3	T4	T5	T + 5	T + 30
ICP	Mean	9.40	9.24	**8.72**	8.88	8.76	**8.76**	8.92	8.68	8.88
Control	SD	(5.16)	(5.12)	**(4.70)**	(5.05)	(4.73)	**(4.59)**	(4.79)	(4.53)	(4.61)
	p value		.701	**.035**	.119	.100	**.029**	.191	.077	.188
ICP	Mean	8.24	8.04	**7.52**	**7.44**	**7.16**	7.44	**7.24**	7.72	8.32
Interv	SD	(3.76)	(4.06)	**(3.69)**	**(4.01)**	**(3.86)**	(4.00)	**(3.90)**	(3.68)	(4.01)
	p value		.307	**.013**	**.038**	**.013**	.079	**.046**	.063	.819
CPP	Median	76	75	77	78	**78**	78	**77**	75	**75**
Control	IQR	(68–89.5)	(69–83)	(70–85)	(71.5–86)	**(71–86)**	(70.5–87)	**(70.5–88.5)**	(65.5–84.5)	**(69–87.5)**
	p value		3.52	.727	.167	**.040**	.36	**.048**	.670	**.050**
CPP	Median	80	75	**75**	**75**	77	**75**	76	**77**	77
Interv	IQR	(66–95)	(65–94)	**(70–94.5)**	**(66.5–95)**	(68–95.5)	**(67.5–94.5)**	(67.5–94)	**(65.5–93)**	(67.5–91)
	p value		.667	**.000**	**.043**	.167	**.048**	.727	**.033**	.667
$PaCO_2$	Median	39						39		
Control	IQR	(37.5–41)						(37–41.5)		
	p value							.540		
$PaCO_2$	Median	39						**38**		
Interv	IQR	(37–41)						**(36.5–40.5)**		
	p value							**.013**		

The mean arterial pressure (MAP) did not significantly differ between the control and intervention groups but was significantly lower in the intervention group (T − 5 to T5), which recovered at T + 30 (Fig. 5).

In terms of $PaCO_2$, no significant differences between the groups were found (means of 39.28 and 38.08 at T5, respectively). This variable showed a significant difference in the means between T − 5 and T5, 38.64 mmHg and 38.04 mmHg, respectively, in the intervention group as seen in Fig. 6.

The cardiac frequency was not significantly different between the groups, with no significant changes at the studied time points in any group.

pH and PaO_2 were not significantly different between the groups or in the intragroup analysis, without changes in the evaluated points.

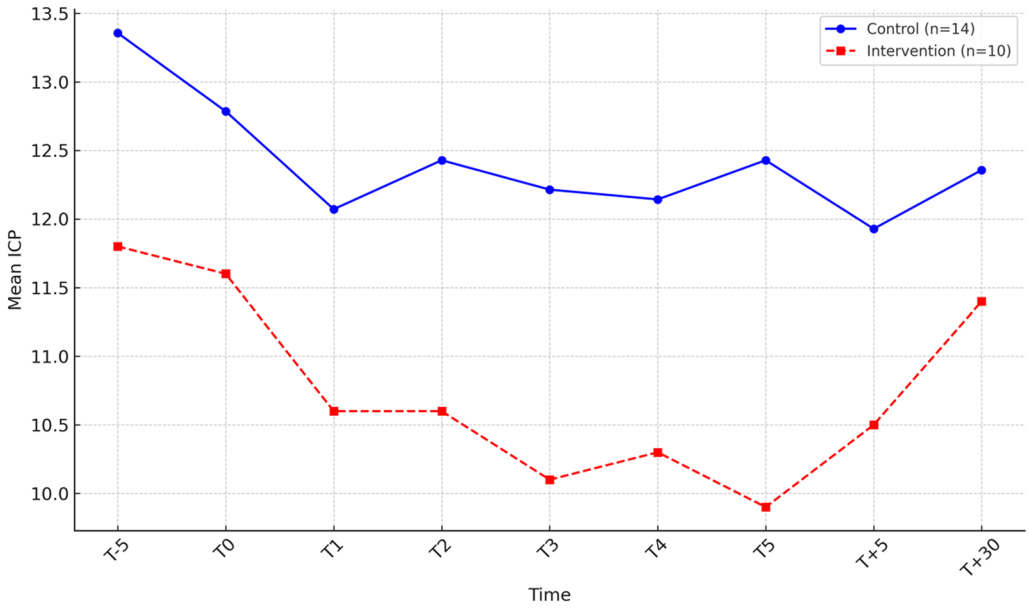

Fig. 3 ICP means at the measured times. Individuals totals with ICP ≥ 10 at T − 5

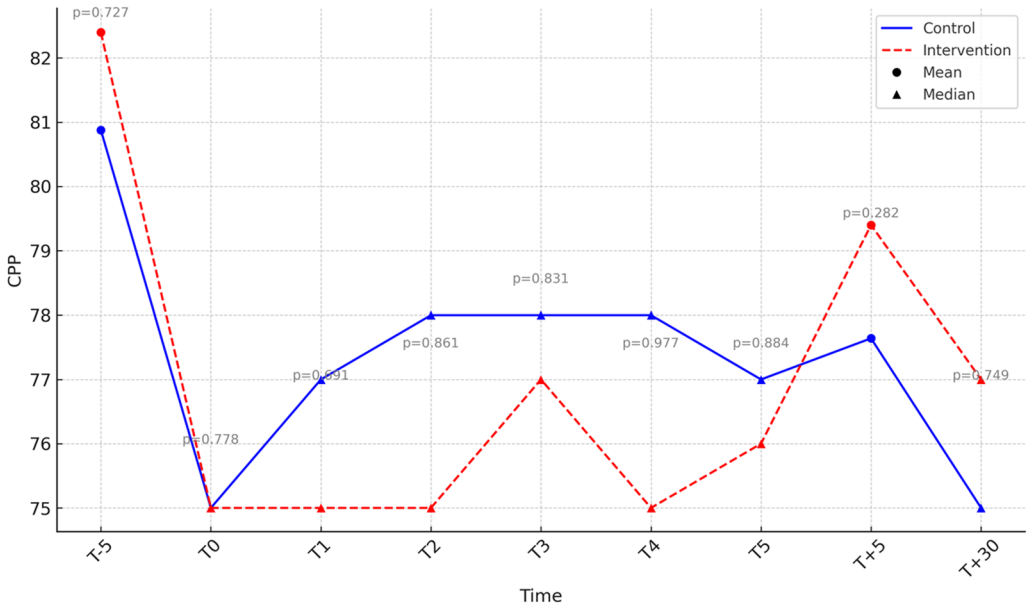

Fig. 4 CPP means/medians at the measured times and *p* values between groups

Flow bias was used to ensure that the theoretical effectiveness of the rapid chest compression technique was more than 33 L/m at all the points checked during its application, approximately 50 L/m in the intervention group and approximately 9 L/m in the control group were reached.

Discussion

With this randomized controlled trial, we avoided mixing the techniques that are usually referred to as respiratory or chest physiotherapy, as they can lead to significant contraindications that are not clinically apparent when the techniques are applied separately. The most recent

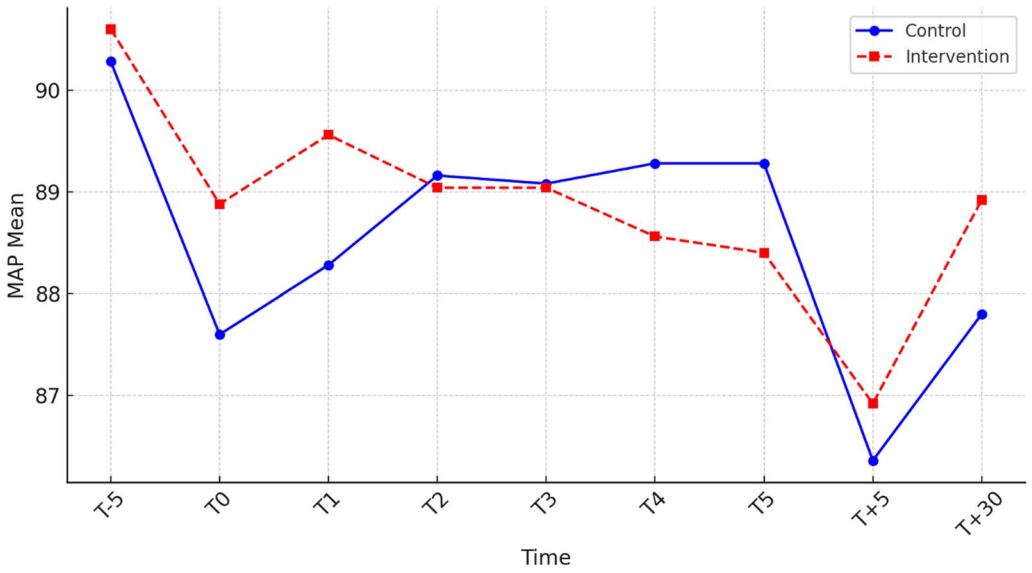

Fig. 5 MAP means of control and intervention groups at the measured times

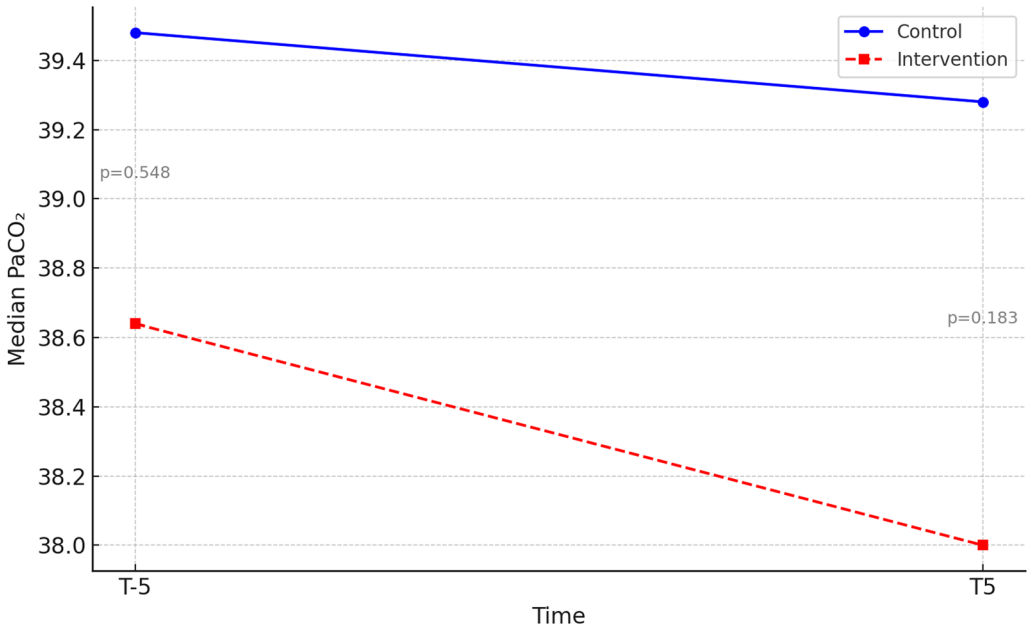

Fig. 6 $PaCO_2$ at the measured times and *p* values between groups

studies concerning the effects of chest physiotherapy techniques on the ICP performed by Tomar et al. [16] revealed that increased values were observed when manual percussion was applied, and the systematic review of Ferreira et al. [15] also revealed that the ICP significantly increased with techniques. While the previous studies from Thiesen et al. [11], Toledo et al. [12], Cerqueira-Neto et al. [14] and Cerqueira Neto et al. [13], focused mainly on descriptive and/or mixing techniques, thus reinforcing the need for RCTs studying the safety of respiratory physiotherapy interventions in neurocritical patients (12–17).

The ICP was the main variable of interest in this trial, and its behavior was within the safety limits, with no significant differences between the groups. According to the intragroup analysis during rapid chest compression, the ICP was significantly lower than T − 5 at 4 of the 6 checked points, and the same behavior was observed

in the mobilization group at 3 points. When we analyzed ICP changes in patients with higher ICP values (10 mmHg or more), the behavior was similar to that of the global group, as seen in Fig. 3. It is acceptable to assume the safety of both procedures in the acute set, and the lower values during the intervention can be explained by the release of the chest, post rapid chest compression in the intervention group and by the abdominal movement in the control group. Both can be explained based on the negative intrathoracic pressure or pressure gradient between intrathoracic and intracranial compartments on the return of blood from the brain and the consequent decrease in ICP [27, 28]. The $PaCO_2$ was significantly lower at T5 (p 0.013), which also explains the significantly lower ICP pressure in the intervention group [26]. In our sample 5 patients had ICP values higher than 15 mmHg (3 in the control group and 2 in the intervention group), that is a small size to obtain a sound subgroup conclusion. Nevertheless, the intervention was not stopped as the ICP values stayed within safety limits. However, future studies should assess this subgroup more thoroughly.

The CPP values were similar in both groups, but the differences were not significant. According to the intragroup analysis, the values were similar before and after rapid chest compression, with some significantly lower values during the procedure but not when comparing pre technique value with 30 min postintervention. In the control group, the CPP value did not recover to pre technique values, with significant difference. Zink et al. did not find significant changes in the CPP values in their study, but our results revealed significantly lower values at 30 min post mobilization, suggesting that an effect that must be taken into account when CPP values are stricter, such as in vasospasm situations [29].

Both the ICP and CPP are affected by $PaCO_2$, which is a cerebral vasodilator; therefore, we controlled the ventilator settings to avoid autotriggering with the thoracic and abdominal pressure changes and performed rapid chest compression every 3 cycles to avoid derecruitment, as there is no volume compensation in volume control mode. However, these measures need stronger control, as they were not completely effective, because $PaCO_2$ was significantly lower in the intervention group (p 0.013) at T5 [26, 30].

The MAP decreased in both groups during both techniques. We related this event to the intrathoracic pressure gradient when the thorax was released in the intervention group or to the mobilization of the abdomen with hip flexion in the control group [29].

The flow bias showed the expected response with a significant difference between the control and intervention groups (− 9.09 L/m and − 47.75 L/m, respectively) at T1 and at all times during the intervention phase, revealing the effectiveness of rapid chest compression in increasing the flow bias toward expiration and increasing it to more than 33 L/m, as referred to in the literature as needed, to mobilize secretions outside the bronchial tree [4].

Our study intended to clarify the effect and safety of RCCT, as most of the studies in neurocritical patients presented results of chest physiotherapy protocols with secretions suction as part of them, and some concluded that chest or respiratory physiotherapy is not safe in neurocritical patients because it increases the ICP, a factor that we relate to suction, as reported in several studies [31, 32].

As shown in the results discussed previously, neither the ICP, CPP, MAP nor the cardiac frequency significantly changed, nor stopped the technique's application or went out of range, allowing us to refer the safety of rapid chest compression in acute neurocritical invasively ventilated patients.

It is also important to note that the ventricular drainage remained open during the procedures. However, since ICP values did not exceed 20 mmHg, there was no risk of increased drainage, allowing the techniques to be applied safely without closing the drain, just elevating it.

We found some limitations in our study. One limitation was the inclusion of 38% of patients with decompressive craniectomy and two individuals in the control group with calvarial fractures who did not undergo decompressive surgery, situations that may lead to less accurate ICP changes. Other limitation, that could lead to gender bias, was the inclusion of 66% females in the total sample and 76% of females in the intervention group. Also, the presence of ventricular drainage (36% in control group and 52% in the intervention group) could lead to less accurate ICP changes, but as we placed it at 20 cm of height from the ear canal, this should minimize this bias. Regarding ventricular drainage, we did not quantify the cerebrospinal fluid drained after returning the drain to its original position; this limitation should be addressed in future studies.

Our study's aim was also to relate different brain pathologies with possible changes in ICP and CPP, but with our sample size, we were not able to obtain a robust result; nevertheless, with this mix of brain injuries, we obtained a sound result.

Conclusion

The rapid chest compression technique does not increase the ICP during its application or even 30 min after it has been performed. The CPP slightly decreased during the application of the rapid chest compression technique and the passive mobilization of the inferior limbs.

In our study, we found that the rapid chest compression technique was safe, but multicenter, double-blind, randomized controlled trials are needed to reinforce the results and to evaluate the effects on brain oxygenation and perfusion.

Abbreviations

ABI	Acute brain injuries
ICP	Intracranial pressure
CPP	Cerebral perfusion pressure
TBI	Traumatic brain injury
AIS	Acute ischemic stroke
SAH	Subarachnoid hemorrhage
ICH	Intracerebral hemorrhage
RCCT	Rapid chest compression technique
SD	Standard deviations
IQR	Interquartile ranges
MAP	Mean arterial pressure

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13054-025-05405-8.

Additional file 1

Additional file 2

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Author contributions
RMRG followed the protocol, recorded the data, and wrote the manuscript. APC analyzed and interpreted the data, and co-wrote the manuscript. RMR and MGG reviewed the manuscript.

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Availability of data and materials
The data will be provided as supplementary material.

Declarations

Ethics approval and consent to participate
Approved by the Comité de Ética de la Investigación de Pontevedra-Vigo-Ourense (ethics board of the Galician public health system) with the registration number 2018/446. Informed consent to participate was obtained and signed by the next of kin, who received written information about participating in the trial. Relatives were informed they could withdraw consent at any time and contact the Spanish Agency of Data Protection if their rights were not upheld. Given the slow evolution of severe acute brain injuries, patient consent was not expected.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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