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PRACTICAL PEARL

Changes in intracranial pressure associated with chest physiotherapy

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Abstract
Introduction Management of intracranial hypertension is pivotal in the care of brain-injured patients.
Summary of Case We report the case of a patient with both a closed head injury and anoxic encephalopathy, who subsequently experienced episodes of refractory intracranial hypertension. The patient's care was complicated by the development of a pneumonia, which required frequent turning of the patient and chest physiotherapy. Conventional wisdom suggests that these interventions may stimulate the patient and worsen intracranial pressure, and therefore should be avoided.
Results Our observations on this patient, however, contradict this belief. This single-subject study presents data to support the use of chest physiotherapy in patients at risk for intracranial hypertension.
Conclusions Further, the evidence is compelling that a randomized-controlled trial is indicated to test the hypothesis that chest physiotherapy may actually result in short-term resolution of high intracranial pressure, and thus provide one more clinical tool in the management of elevated intracranial pressure.

Keywords Intracranial hypertensions · Brain injury · Chest physiotherapy

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Introduction

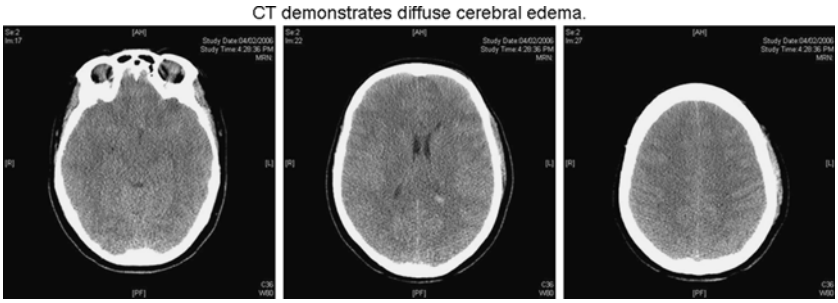
The management of intracranial pressure (ICP) is often complicated by the need to simultaneously treat organ systems and injuries, beyond just those directly affecting the central nervous system. Often, the patient who needs ICP treatment therapy also needs other systemic therapies. Therapy aimed to directly reduce or control ICP may negatively impact other body systems and vice versa.

Brain-injured patients at risk for intracranial hypertension often require mechanical ventilation. Aggressive pulmonary care in these cases is essential to promote recovery without pulmonary complications. Pulmonary care is a broad term for the many actions (turning, chest physiotherapy [CPT], oral hygiene, endotracheal suctioning, etc.) that are performed to promote ventilation and prevent ventilator associated pneumonia. Many of these procedures may either directly or indirectly stimulate the patient. The need to avoid excessive stimulation of patients at risk of intracranial hypertension is often cited, in spite of the lack of conclusive evidence supporting the hypothesis that pulmonary care procedures negatively impact ICP. In order to assess the potential impact of CPT on ICP, we conducted a single-subject study design.

Case presentation

A 41-year-old female was admitted to hospital following a severe, closed head injury (fall from a horse) complicated by hypoxia during prolonged resuscitation. Her initial Glasgow Coma Score (GCS) was 8 (E-2, V-1, M-5). Her initial brain CT scan showed a minimal left fronto-parietal subdural hematoma and diffuse cerebral edema (Fig. 1). Chest X-ray at the time revealed bilateral tension pneumothoraces.

Fig. 1 Brain CT scan on Day 1



Her poor neurological status was attributed to a combination of traumatic and ischemic cerebral injury.

Aggressive treatment of intracranial hypertension was initiated immediately. ICP monitoring and CSF drainage were facilitated by an intraventricular catheter and osmotic therapy was initiated with intravenous mannitol (25 g every 3 h). Despite efforts to maintain an ICP < 20 mm Hg and a cerebral perfusion pressure (CPP) > 60 mm Hg, the patient continued to experience episodes of sustained intracranial hypertension (ICP > 25–30 mm Hg) and as such she was put on barbiturate coma on day 2. On day 3 of her admission, hypertonic saline boluses (30 ml of 23.4% given every 6 h) were administered, but were effective for only short periods of time. Despite ongoing medical management, the patient had refractory intracranial hypertension. Further, brain CT scans showed persistent, diffuse cerebral edema, without any mass, which could be relieved by surgery. The high ICP was further complicated by the development of a ventilator associated pneumonia and difficulty with ventilation and oxygenation. Patient repositioning and CPT were now indicated to optimize her pulmonary care but concerns were raised by the nursing staff regarding the potentially adverse effects of CPT on ICP. The following study thus ensued.

Methods

A single-subject study design was used to prospectively explore the association between changes in ICP and the onset and duration of multiple episodes of CPT. Episodes of CPT were programmed into a bed having the ability to deliver automated CPT consisting of mechanical percussion (SPORT^{tm} bed by Hillrom^{tm}); therapeutic sessions were set to last for 10 min with the percussion mode set on 'high' at a rate of 5 beats per second. ICP values were recorded each minute for 10 min prior to the onset of CPT, each minute during CPT, and each minute for 10 min after CPT. A convenience sample of five episodes of CPT were analyzed over the course of two days, each coinciding with episodes of refractory intracranial hypertension. These 5 episodes were selected because they were performed by the same nurse using the same automated CPT settings with no changes in head of bed position (maintained at 30 degrees per physician's orders), no changes in the rate of sedative administration, and each CPT episode had a minimum of 10 min before, during, and after CPT in which there were no other ICP interventions (e.g. mannitol or hypertonic saline administration), and no interventions that may otherwise alter ICP (endotracheal suctioning or CSF drainage). No other CPT episodes were recorded or analyzed. ANOVA was used to explore differences in ICP and mean arterial pressure (MAP) values before, during and after CPT using SAS v9.1 (SAS Corporation, Cary NC).

Results

Mean values (Table 1) for ICP (mm Hg) before (25.63, SD 8.4), during (23.0, SD 9.57), and after (17.30, SD 7.15) CPT were found to be statistically ($F = 12.39$, $P < 0.0001$) and clinically significant. Mean values (mm Hg) for MAP before (91.82, SD 12.26), during (91.73, SD 10.48), and after (90.08, SD 9.95) CPT were not significant ($F = 0.40$, $P > .6724$).

Discussion

After an acute brain injury, critical care therapy is aimed at preventing secondary injury, which may result from the

Table 1 ICP and mean arterial blood pressure values before, during, and after 10 min of chest physiotherapy

	N	Infracranial pressure (mm Hg)			Mean arterial pressure (mm Hg)		
		Mean	S.D.	95% C.I.	Mean	SD	95% Cl
Before chest PT	46	25.63	8.40	2.49	91.82	12.26	3.48
During chest PT	48	23.00	9.57	2.77	91.73	10.48	3.04
After chest PT	50	17.30	7.15	2.03	90.08	9.95	2.83

cascade of events that follows primary injury [1, 2]. Key prevention strategies include optimizing ICP and ventilation management to promote cerebral tissue perfusion [2–4]. Pulmonary care is a group of procedures designed to improve oxygenation and ventilation [5, 6]. Procedures may be as simple as positioning the patient (e.g., elevating the head of the bed), or more complex, such as CPT. The vibrations from CPT open alveoli, loosen secretions and increase the surface area for gas exchange to occur. Turning the patient aids in the prevention and resolution of pneumonia and atelectasis [7, 8]. The decision not to provide pulmonary care must be examined within the holistic view of providing care to the entire patient at that point in time, rather than just considering a single system [9].

Given the wealth of information to support care of the mechanically ventilated patient, the lack of attention to pulmonary care issues for the neurocritically ill patient is arguably negligent. The Centers for Disease Control and Prevention recommendations include elevation of the head of the bed to prevent aspiration pneumonia as a category II recommendation, and frequent repositioning remains a category IB (strongly recommended) recommendation [10]. A review article of randomized controlled trials for kinetic therapy and continuous lateral rotational therapy (CLRT) found that both resulted in a decrease in nosocomial pneumonia, and patients who were turned >40 degrees had the greatest risk reduction [11]. Clinical trials specific to neurologically compromised, mechanically ventilated patients are scant [12], and it has been suggested that more appropriate respiratory care may result in improved outcomes for neurocritically ill patients [13]. In a small study including both mechanically ventilated patients as well as spontaneously breathing patients Koch et al. [14] demonstrated that passive rotation did not increase ICP. Endotracheal suctioning and ‘‘bag ventilation’’ on the other hand have been shown to increase systemic and intracranial pressure, the former demonstrating prolonged effects unless sedation was initiated prior to the interventions [15]. Randomized prospective studies evaluating the effects of pulmonary physical therapy on ICP are currently lacking.

Limitations

The exact mechanisms by which CPT may alter ICP are not described in literature. For this study, we did not collect or explore the large number of theoretical confounding variables and covariates that may be involved. Depth of sedation (Ramsay 4, BIS < 60) was maintained throughout the study periods; the effects of CPT may vary with sedation. Changes in respiratory mechanics (e.g. tidal volume, end-tidal CO2, compliance) were not measured during the study periods and the change in ICP may be a secondary pathway resulting from a change in one or more of these variables. Measures of intrathoracic pressure (CVP) were obtained only before or after CPT; monitoring for acute changes in CVP may be useful in exploring the pathophysiology of any change in ICP.

Conclusions

This case highlights the case of a patient with refractory intracranial hypertension in which CPT provided at least a temporizing effect on intracranial hypertension without decreasing MAP and compromising cerebral perfusion pressure. Although CPT is associated with improved outcomes related to hemodynamic and infectious etiologies, there is an unwritten assumption that CPT negatively impacts ICP. Little evidence exists linking changes in intracranial dynamics with specific pulmonary care procedures. A single-subject study is, however, inadequate to provide the impetus to change practice. We believe this case presents compelling evidence to support the need for more comprehensive exploration into the effects of specific pulmonary care interventions on ICP.

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