Effect of intermittent pneumatic leg compression on intracranial pressure in brain-injured patients

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Objective: To evaluate the effect of intermittent pneumatic leg compression on intracranial pressure and cerebral perfusion pressure in brain-injured patients.

Design: Prospective, sequential patient study.

Setting: Surgical/trauma ICU of a community hospital providing regional trauma care.

Patients: Twenty-four adult, brain-injured patients (mean Glasgow Coma Scale score = 6) who required hemodynamic and intracranial pressure monitoring.

Interventions: Placement of intermittent sequential pneumatic leg compression devices for prevention of venous thrombosis.

. *Measurements:* Mean arterial pressure (MAP), heart rate, central venous pressure, and intracranial pressure were measured at baseline, and at 0, 10, 20, and 30 mins of intermittent pneumatic leg compression. Cerebral perfusion pressure was calculated as the difference between MAP and intracranial pressure.

Results: No significant changes in MAP, central venous pressure, or intracranial pressure occurred during the study interval. Calculated cerebral perfusion pressure remained unchanged. A total of 23 of 24 study patients had intracranial pressure controlled by hyperventilation or pharmacologic measures within the normal range at the time of study.

Conclusion: Intermittent pneumatic leg compression results in no significant changes in intracranial pressure or cerebral perfusion pressure in stable, brain-injured patients who have intracranial pressure controlled by medical means. (Crit Care Med 1993; 21:224-227)

Intermittent pneumatic compression of the legs effectively reduces the occurrence rate of deep venous thrombosis in a variety of patients (1). The brain-injured patient in coma is at particular risk for deep venous thrombosis and, because of the potential for catastrophic intracranial bleeding, anticoagulant methods of deep venous thrombosis prophylaxis are usually avoided. Intermittent pneumatic compression has been recommended in this setting, and has been shown to reduce the frequency of venous thrombosis in neurosurgical patients (2, 3):

When a patient is suffering from head injuries, routine nursing procedures such as turning side to side and endotracheal suctioning may result in an increase in intracranial pressure. The stimulus of intermittent pneumatic leg compression might also cause increased intracranial pressure, but no studies have investigated this possibility. However, a case (4) has been reported in which intermittent pneumatic leg compression was associated with cyclic increases in arterial BP and central venous pressure in a patient with preexisting cardiac disease. If similar hemodynamic changes were to occur in brain-injured patients with reduced intracranial compliance, this circumstance might result in adverse changes in intracranial pressure.

The aim of this study was to prospectively evaluate the influence of intermittent pneumatic compression of the legs on intracranial hemodynamics in severely brain-injured patients.

MATERIALS AND METHODS

Patients admitted to the surgical/trauma ICU of Sharp Memorial Hospital were eligible for study if they had placement of intracranial pressure monitors for management of head injury, indwelling radial artery catheters, and intermittent pneumatic compression devices as part of their routine care. No diagnostic categories were excluded, but patients were accepted

for study only after a brief period of stable intracranial pressure was achieved. This study was approved by the hospital Institutional Review Committee, and the requirement for individual informed consent was waived.

All patients had been volume resuscitated to the point of hemodynamic stability and were mechanically ventilated. Medical therapy to stabilize intracranial pressure included the provision of mechanical hyperventilation; neuromuscular blocking agents; diuretic and osmolar therapy; and barbiturates, depending on the individual patient status. The authors made no attempt to control the medical regimen; however, data were collected after the initial placement of intermittent pneumatic compression devices during a period of stable hemodynamics and intracranial pressure. Baseline values for heart rate, mean arterial pressure (MAP), intracranial pressure, and central venous pressure (when available) were recorded before placement of the pneumatic compression device. MAP was obtained from a radial artery catheter, and central venous pressure from a central venous catheter placed in the superior venacava, utilizing disposable fluid-filled transducers (Baxter Edwards Critical-Care, Irvine, CA) leveled to the phlebostatic axis. Intracranial pressure was measured either by ventriculostomy or subarachnoid screw using fluid-filled transducers or fiberoptic catheters (Camino Laboratories, San Diego, CA). Ventriculostomy transducers were leveled to the outer canthus of the eye and subarachnoid screws leveled to the insertion site. All transducers were leveled and calibrated before data collection. Cerebral perfusion pressure was recorded as the difference between MAP and intracranial pressure.

Thigh-length multicompartment sequential pneumatic compression devices (Kendall, Mansfield, MA) were placed over thigh-length Kendall thromboembolism deterrent stockings. The head of the bed was maintained elevated 30°. Intermittent pneumatic compression was initiated when heart rate and intracranial pressure were within 10% of baseline values, allowing for resolution of any hemodynamic changes due to movement during placement of the pneumatic compression devices. Measurements were then recorded when the intermittent pneumatic compression device was turned on (time 0) and at 10, 20, and 30 mins into therapy. The sequential pneumatic compression devices utilized consist of a series of six compartments inflated serially from the ankle to the mid-thigh with an 11-sec compression phase followed by 60 sees of deflation. The initial ankle pressure was adjusted to 45 mm Hg in all subjects. All pressure recordings were obtained at the start of the deflation cycle, correlating with completion of leg compression, when femoral vein emptying and leg compression should be maximal. No

changes in position were made and no endotracheal suctioning was performed during the study period, and no effort was made to coordinate timing of compression or data collection with the respiratory cycle.

A total of 28 patients were initially entered into the study. Four patients were excluded; two patients had arousal from sedation and required bolus administration of sedative drugs; two patients were excluded for protocol violations due to incomplete data collection. A total of 24 patients completed the studies; 21 had central venous catheters allowing central venous pressure monitoring.

Statistical Analysis. Statistical comparison of the data at baseline and at subsequent intervals was performed using BMDP (BMDP Statistical Software, Los Angeles, CA, 1990) univariate analysis of variance (ANdVA) with repeated measures. Ap of $< .05$ was considered significant. Adjustment for sphericity was performed using the Greenhouse-Geiser method. (5)

RESULTS

Patient population characteristics are shown in Table 1. Most patients (21 of 24) were being therapeutically hyperventilated, as indicated by a $Paco_2$ of <33 torr (<4.4 kPa) at the time of study. No patient had a clinical deterioration or adverse event felt to be due to the study.

Figure 1 represents the average values, relative to baseline, for intracranial pressure, central venous pressure, cerebral perfusion pressure, and MAP. There were no clinically significant changes in the values in individual cases or in group means during the study. No transient brief increases of intracranial pressure were noted in any patient between data collection periods. The mean baseline intracranial pressure was 11.1 mm Hg. Only one of 24 patients was entered into the study with an intracranial pressure of >20 mm

Table 1. Patient characteristics

Etiology of Brain Injury (n = 24)	
Closed-head trauma	**19**
Penetrating trauma	**3**
Intracerebral bleed	**2**
Male/female	**20/4**
Hyperventilated	**21**
Glasgow Coma Scale Score	
Mean	**6**
Median	**3**
Mean Baseline Pressures (mm Hg)	
Central venous	**5.5 ± 4°**
Mean arterial	**85 ± 13**
Intracranial	**11.1 ± 8**
Cerebral perfusion	**74 ± 18**

•Mean ± sn.

AVERAGE MEAN ARTERIAL PRESSURE

AVERAGE INTRACRANIAL PRESSURE

AVERAGE CENTRAL VENOUS PRESSURE

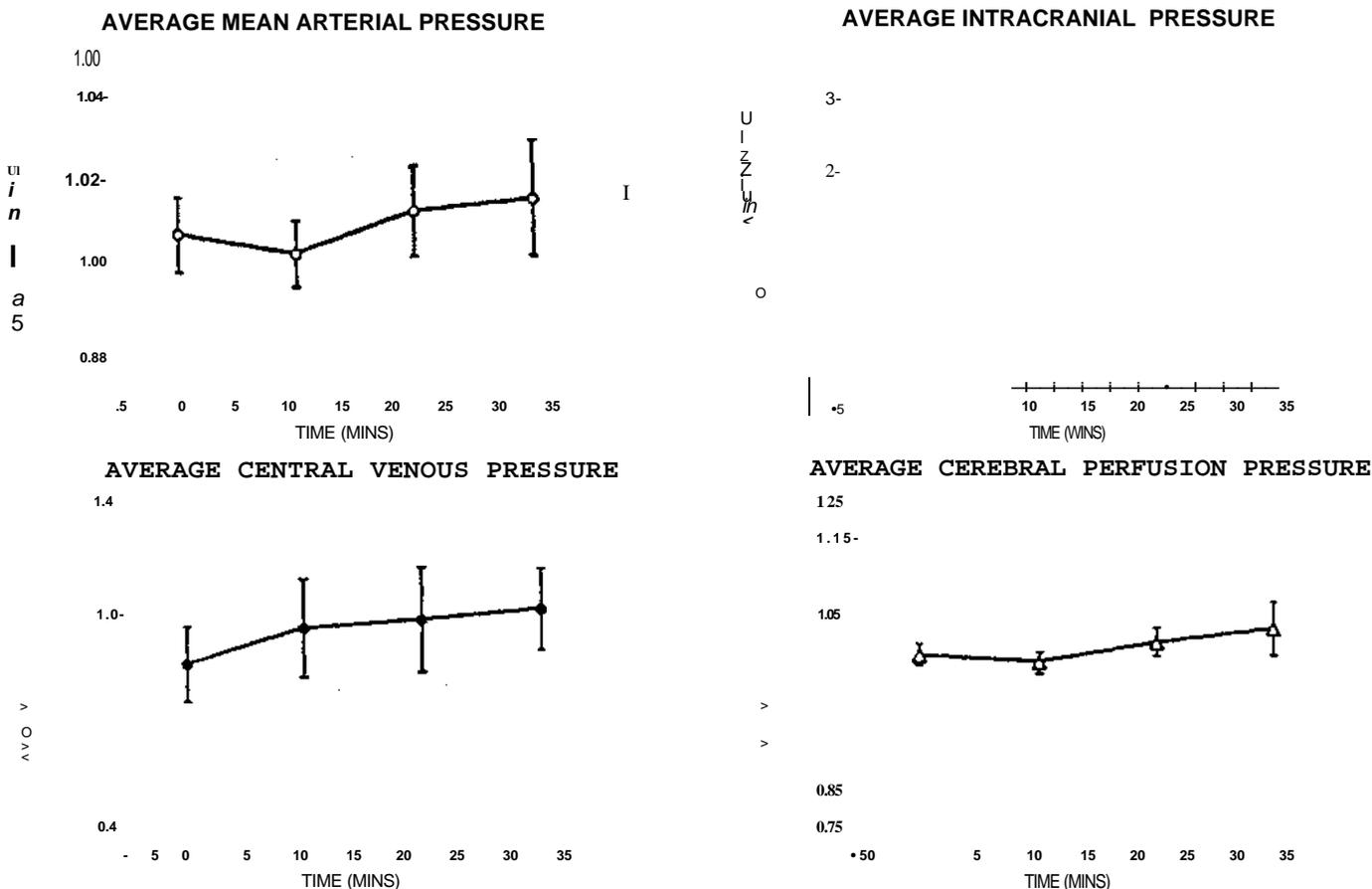**AVERAGE CEREBRAL PERFUSION PRESSURE**

Figxtre 1. Hemodynamic pressures during pneumatic leg compression. Values at each time interval represent the average pressure divided by baseline pressure (± SEM). *MAP,* mean arterial pressure; *ICP,* intracranial pressure; CVP, central venous pressure; *CPP,* cerebral perfusion pressure.

Hg. This patient's intracranial pressure declined from 38 to 32 mm Hg during the study interval. The mean central venous pressure was 5.5 mm Hg, indicating that patients were not significantly hypovolemic at the time of study. Although not part of the study protocol, no clinically detected deep venous thromboses became evident in the study population.

DISCUSSION

Intermittent pneumatic compression decreases the caliber of lower extremity veins and augments venous emptying by increasing femoral vein blood velocity (6). The intermittent sequential pneumatic compression device which we used results in more complete emptying of the femoral vein and a higher femoral vein blood velocity, as compared with isolated calf compression (6). We chose this type of device rather than an isolated calf compression device, anticipating that potentially greater hemodynamic effect would occur.

Some similarities of intermittent pneumatic leg compression to military anti shock trousers led to our investigation. Both techniques involve external application of circumferential pressure to the lower extremities, albeit at different inflation pressures and duration of inflation (7). Early clinical studies (8) of military antishock trousers compression in Vietnam demonstrated significant hemodynamic support of patients in
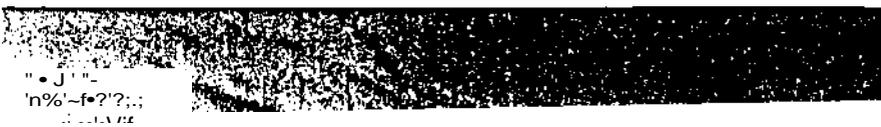
shock with military antishock trousers inflation pressures of 30 to 40 mm Hg, well within the range of compression pressures utilized currently with intermittent pneumatic compression devices (up to 45 to 55 mm Hg). Although intermittent pneumatic leg compression has not been advocated for hemodynamic support, a case has been described in which a patient had significant increases in central venous pressure, pulse pressure, and pulmonary artery pressure coinciding with the compression phase of sequential leg compression (4). This patient, with underlying cardiovascular disease, had more pronounced pressure changes during periods of relative hypovolemia.

In the past, concern had been raised over possible adverse effects of military antishock trousers suits on intracranial pressure in traumatic head injury. One postulate (9) held that military antishock trousers suit eounterpressure, by increasing central venous pressure, could restrict venous return from the head, thereby raising intracranial pressure. A series of canine experiments (9-11) to investigate this hypothesis showed that military antishock trousers compression could result in significant increases in MAP without clinically significant increases in intracranial pressure, and without adverse effects on cerebral perfusion. Gardner et al. (12) evaluated intracranial pressure, MAP, and cerebral perfusion pressure during military anti-shock trousers compression in 12 brain-injured humans, and found small incremental increases in MAP, central venous pressure, and intracranial pressure. The net effect of the increase in MAP and intracranial pressure was to increase cerebral perfusion pressure. In light of these studies, military antishock trousers compression is not contraindicated in shock patients with concomitant head injury (7).

In our study, no statistically significant changes in MAP, central venous pressure, intracranial pressure, or cerebral perfusion pressure occurred. Additionally, no individual patient was seen to have clinically important increases in intracranial pressure or declines in cerebral perfusion pressure. A number of considerations may have decreased the likelihood that intermittent pneumatic compression would influence intracranial hemodynamics in this study. First, this mode of compression utilizes transient leg compression followed by a prolonged period of deflation. Thus, the majority of the time is spent without external counterpressure that may mitigate any potential effect on central hemodynamics. Also, our patients were felt to be euvolemic, and were studied in the 30° head-up

position. In a study of healthy normovolemic adults, Gaffney et al. (13) found that the 60° head-up position minimized the effect of military antishock trousers compression of the extremities on MAP and peripheral vascular resistance compared with supine positioning. Finally, 23 of 24 patients had near-normal intracranial pressure at the time of entering the study, related to the therapy they had received before study. Thus, an influence on intracranial pressure and cerebral perfusion pressure in patients with more severely increased or unstable intracranial pressure is possible and cannot be excluded by this study.

We found no evidence to contraindicate the use of intermittent pneumatic compression in this population of patients, who need effective prophylaxis for venous thrombosis.

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