Cerebral and cardiovascular responses to changes in head elevation in patients with intracranial hypertension

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To establish if an optimum level of head elevation exists in patients with intracranial hypertension, the authors examined changes in intracranial pressure (ICP), systemic and pulmonary pressures, systemic flows, and intrapulmonary shunt fraction with the patient lying flat, and then with the head elevated at 15°, 30°, and 60°. Cerebral perfusion pressure (CPP) was calculated. The lowest mean ICP was found with elevation of the head to 15° (a fall of -4.5 ± 1.6 mm Hg, p < 0.001) and 30° (a fall of -6.1 ± 3.5 mm Hg, p < 0.001); the CPP and cardiac output were maintained. With elevation of the head to 60°, the mean ICP increased to -3.8 ± 9.3 mm Hg of baseline, while the CPP decreased -7.9 ± 9.3 mm Hg (p < 0.02), and the cardiac index also fell -0.25 ± 0.28 liters/min/sq m (p < 0.01). No significant change in filling pressures, arterial oxygen content, or heart rate was encountered at any level of head elevation. Therefore, a moderate degree (15° or 30°) of head elevation provides a consistent reduction of ICP without concomitant compromise of cardiac function. Lower (0°) or higher (60°) degrees of head elevation may be detrimental to the patient because of changes in the ICP, CPP, and cardiac output.

KEY WORDS: intracranial pressure · head injury · cardiac output · head position · cerebral perfusion pressure

Patients suffering an acute brain injury of traumatic or anoxic-ischemic origin frequently undergo sudden neurological deterioration. Common causes of deterioration are increased intracranial pressure (ICP) with resultant brain shift and cerebral ischemia, and acute changes in both the systemic arterial pressure (SAP) and oxygen delivery. Since significant reductions in the ICP may occur with changes in head and body position, such patients are commonly cared for in the head-up position following acute brain injury.

The benefit of postural changes on ICP control may accrue from many different integrated mechanisms. Both the SAP and the central venous pressure (CVP) are affected by postural change, and may be responsible in part for the reported beneficial effects of postural therapy on the ICP in brain-injured patients. However, cardiovascular responses secondary to postural change may theoretically vary depending upon the presence or absence of concomitant intracranial hypertension. Furthermore, the degree of postural change may have variable effects on the control of the ICP.

Because of incompletely documented reports of the results of changes in head position on intracranial and cardiovascular dynamics, in this study we examined the influence of varying degrees of head elevation on both intracranial and systemic cardiovascular dynamics, as well as on integrated cardiorespiratory function in patients with severe brain injury from trauma or anoxic-ischemic injury. We specifically sought to identify if any single head position provided optimum control of an elevated ICP, but at the same time was associated with a minimum effect on the integrated systemic cardiopulmonary adaptation to the acute brain injury.

Clinical Material and Methods

Patient Population

Eleven patients admitted to the critical care units of Victoria Hospital and University Hospital with acute brain injury and associated intracranial hypertension were studied according to criteria approved by the Human Research Committee of the University of West-
patient had been admitted to the critical care units following primary investigation and therapy of the associated acute brain injury.

All 11 patients had a Glasgow Coma Scale score of less than 8. Eight of the patients had suffered a severe head injury as a result of a motor vehicle accident, and three had sustained acute brain injury following near-drowning. No patient had undergone removal of an intracranial mass lesion, and each patient had an ICP of greater than 25 mm Hg recorded at least once immediately before initiation of the study. All patients had been hemodynamically stabilized according to standard techniques, and therefore no patient was hypertensive at the time of study.

Routine care prior to study consisted of intubation and mechanical ventilation with a volume-cycled ventilator. Patients were hyperventilated to maintain a PaCO2 of 25 to 30 mm Hg, initially by adjusting the tidal volume (12 to 15 ml/kg) and then by adjusting the respiratory rate to effect the desired PaCO2. Oxygenation was provided by varying the inspired concentration of oxygen to maintain the PaO2 greater than 70 mm Hg. No alteration in this predefined respiratory care was made for 30 minutes before the study or during the period of study. In addition, on admission all patients were kept with their heads elevated 15° or 30° by flexion at the hips. Mannitol (0.25 gm/kg intravenously every 4 to 6 hours) and dexamethasone (Decadron, 0.1 mg/kg intravenously every 2 to 4 hours) were prescribed in all cases. In addition, five patients were receiving thiopental sodium (Pentothal, 2 mg/kg/hour, intravenously) both for control of intracranial hypertension and for cerebral protection, prior to study. Cerebrospinal fluid (CSF) venting by intermittent drainage from the ventricular catheter was performed by nursing personnel when the ICP exceeded 20 mm Hg despite the aforementioned neuroresuscitative therapy.

Protocol

Prior to study, all patients had had insertion of intraventricular, arterial, and flow-directed right heart catheters as part of the routine neuroresuscitative care. The ICP and SAP transducers were calibrated to the level of the foramen of Monro, and the pulmonary artery pressure (PAP) transducer was calibrated to the level of the right atrium. Each of the three catheters was connected to a Statham pressure transducer, and the pressures were recorded on a Hewlett-Packard multichannel chart recorder.*

The 11 patients underwent a total of 31 separate recording sessions. Repeated studies were performed only after a change in the patient's clinical status had been documented by one of the authors (Q.J.D). No study was repeated more frequently than once every 24 hours. One patient was recorded on six occasions, one on four occasions, five on three occasions, two on two occasions, and two on one occasion. Before each recording session, the patient was placed flat (0°) for 5 minutes. Measurements were then taken of the ICP, SAP (systolic, diastolic, and mean SAP), PAP (systolic, diastolic, and mean PAP), pulmonary capillary wedge pressure (PCWP), CVP, heart rate (HR), and cardiac output (CO). Blood was withdrawn from the arterial line and from the distal port of the right heart catheter into heparinized syringes for measurement of arterial and mixed venous gases, respectively. Oxygen tension, pCO2, and oxygen saturation (percent) were measured.

Cardiac output was determined by the thermodilution technique using iced saline as described previously. The mean of three consecutive thermodilution measurements was taken to represent the CO in this series. Cardiac index (CI) was calculated as the CO divided by body surface area and was expressed in liters per minute per square meter. All pressure measurements were taken at end-expiration, and the mean of the results obtained over three respiratory cycles was determined. Following each set of measurements, which took approximately 5 to 10 minutes, the patient's head was elevated by flexion at the hips to the next selected position (15°, 30°, or 60°). The angle of elevation was measured by a goniometer. After 5 minutes in the new position, the same measurements were repeated, beginning with the ICP obtained as previously described by averaging the results from three consecutive respiratory cycles.

The initial four patients admitted to the study (Group 1) had eight recordings taken with the head at 0°, and then elevated to 30°, and then 60°. To further delineate the head position resulting in the lowest ICP, the latter seven patients (Group 2) had 23 recordings taken at 0°, and then at 15°, 30°, and 60° of head elevation. For each of the patients, the results of the measured parameters recorded at each level of head elevation from all recording sessions were averaged. Statistical analysis of the various parameters was then performed from these averaged results to prevent weighting of the mean from patients recorded more frequently.

Results

Intracranial Pressure

The ICP of all patients increased when the patient was placed flat at the commencement of each study. Table 1 documents that the lowest ICP from 31 recording sessions occurred at 15° and 30°, and the highest ICP occurred at 0° and 60°. The averaged results from all recording sessions for each of the 11 patients showed that the ICP fell in all Group 2 patients with head elevation to 15°. All patients benefited with head elevation to 30°. Four patients had a further reduction in

*Pressure transducer manufactured by Statham Laboratories, Inc., Hato Rey, Puerto Rico; multichannel chart recorder manufactured by Hewlett-Packard Corp., San Diego, California.
Fig. 1. Mean ± standard deviation of the change in intracranial pressure (ICP) between lying flat (0°) and 15°, 30°, and 60° of head elevation in 11 patients. The ICP's at 15° and 30° are significantly less than at 0° (p < 0.001). The ICP at 60° is not significantly less.

The ICP at 60°, two patients had no change, and five had an increase in the ICP over that recorded at 30°.

The mean ICP fell -4.5 ± 1.6 mm Hg (p < 0.001) with elevation of the head from 0° to 15° (Figs. 1 and 2). This decrease in the ICP was maintained at 30° (-6.1 ± 3.5 mm Hg; p < 0.001), but this value was not significantly different from the ICP at 15°. At 60°, the change in ICP (-3.8 ± 9.3 mm Hg) was not significantly different from the baseline value recorded initially at 0°. At four different recording periods in two patients the session was terminated prematurely because of a severe increase in ICP (Fig. 3), which was immediately controlled by returning the patient to a position with 30° head elevation.

**Systemic Hemodynamics**

The mean SAP (recorded at the level of the foramen of Monro) fell with the progressive increase in head elevation (r = 0.98; p < 0.001) (Fig. 4 left), whereas the cerebral perfusion pressure (CPP) was not significantly affected by 15° or 30° of head elevation (Fig. 4 right). However, elevation of the head to 60° produced a significant reduction in the CPP when compared to baseline levels (a fall of -7.9 ± 9.3 mm Hg; p < 0.02).

![Fig. 2. Intracranial pressure (ICP) and systemic arterial pressure (SAP) recordings obtained during head elevation from 0° to 15°, 15° to 30°, and 30° to 60° in one patient. Note that the largest decrease in ICP occurred from 0° to 15°. Minimal further reduction occurred from 15° to 30°, and a slight increase occurred from 30° to 60°.](image-url)

**TABLE 1**

<table>
<thead>
<tr>
<th>Head position</th>
<th>Lowest ICP</th>
<th>Highest ICP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1</td>
<td>Group 2</td>
</tr>
<tr>
<td>0°</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>15°</td>
<td>NR</td>
<td>9</td>
</tr>
<tr>
<td>30°</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>60°</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>total sessions</td>
<td>8</td>
<td>23</td>
</tr>
</tbody>
</table>

*Four Group 1 patients had eight recording sessions and seven Group 2 patients had 23 sessions. ICP = intracranial pressure; NR = not recorded.*
Head elevation and intracranial pressure

With head elevation to 15° or 30°, no change was found in the mean CI. However, at 60°, a significant fall in the mean CI was noted (−0.25 ± 0.28 liters/min/sq m; p < 0.01) (Fig. 5). The decrease in CI was not associated with a change in the mean PCWP, HR, mean PAP, or CVP (Table 2).

A significant linear correlation (r = 0.47; p < 0.05) was noted between the change in both the ICP and change in the CVP from 0° to 60° (Fig. 6). Head elevation through the various heights had no significant effect on the arterial oxygen content or arterial-mixed venous saturation (Table 2).

Discussion

In critically ill patients with acute brain injury and associated intracranial hypertension, elevation of the head is used clinically to effect a reduction in the ICP. Since acute postural change may potentially influence the ICP through many different mechanisms, we evaluated the effect of varying degrees of head elevation upon the ICP as well as on integrated cardio-pulmonary function. We found that 15° to 30° of head

Fig. 3. Systemic arterial pressure (SAP), pulmonary artery pressure (PAP), and intracranial pressure (ICP) in a patient raised from 30° to 60° of head elevation. The rapid rise in ICP precipitated within 5 seconds of the change in head position was partially controlled by cerebrospinal fluid (CSF) venting. The patient was lowered back to 30°, and the ICP fell, but further CSF venting was required.

Fig. 4. Mean ± standard deviation of the change in systemic arterial pressure (SAP) and cerebral perfusion pressure (CPP) between lying flat (0°) and 15°, 30°, and 60° of head elevation in 11 patients. Left: To measure the SAP, the pressure transducer was calibrated to the level of the foramen of Monro. The three mean measurements are all significantly less than the pressure at 0°, with a linear correlation coefficient of 0.984 (p < 0.001). Right: The CPP is significantly less with the head elevated at 60° compared to at 0° (p < 0.02). The CPP's at 15° and 30° are not significantly less than at 0°.
elevation was associated with a fall in the ICP with maintenance of the CPP in a majority of patients, while head elevation to 60° was frequently associated with adverse influences on both the ICP and the CPP.

Brain swelling with consequent elevation of the ICP often occurs in patients who sustain severe acute brain injury. An elevated ICP may lead to further brain damage by promoting ischemia and causing subsequent brain shift and herniation. Management of the patient at risk for intracranial hypertension, or indeed the patient with established intracranial hypertension, traditionally involves a regimen consisting of hyperventilation, osmotic diuresis, and high-dose glucocorticoids. With established intracranial hypertension, intermittent CSF venting, barbiturates, lidocaine, and even decompression are utilized. In addition, both in patients at risk and in patients with established intracranial hypertension, elevation of the head to varying levels is used to minimize the ICP.

We found that the majority of patients studied manifested a significant fall in ICP with an elevation of the head from 0° to both 15° and 30°. Elevation of the head to 60° resulted in an ICP lower than at 0° in some patients, but more importantly was associated with increases in the ICP in others (Table 1). The variability in the change of the ICP from 0° to 60° has also been noted by Ropper, et al., and Magnaes. However, such variability was not recorded by Kenning, et al., quite possibly because their patient population was not as severely injured as ours.

In 12 of the recording sessions from five of the patients, we found that the ICP was greater at 60° than that recorded at 0°. Indeed high pressure waves caused the immediate discontinuance of four of the recording sessions in two patients. In each of these recording sessions, a reduction in the ICP occurred immediately after the patient was returned to 30° of head elevation.

### TABLE 2

<table>
<thead>
<tr>
<th>Parameter Recorded</th>
<th>Head Elevation</th>
<th>0° to 15°</th>
<th>0° to 30°</th>
<th>0° to 60°</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ PaO₂ (mm Hg)</td>
<td></td>
<td>+5.7 ± 12.3</td>
<td>+1.4 ± 13.3</td>
<td>−1.0 ± 16.8</td>
</tr>
<tr>
<td>Δ %O₂ Satn (mm Hg)</td>
<td></td>
<td>+0.21 ± 4.1</td>
<td>−0.5 ± 1.1</td>
<td>+0.3 ± 2.9</td>
</tr>
<tr>
<td>Δ PCWP (mm Hg)</td>
<td></td>
<td>+0.5 ± 1.6</td>
<td>−0.2 ± 1.5</td>
<td>+1.4 ± 2.4</td>
</tr>
<tr>
<td>Δ HR (beats/min)</td>
<td></td>
<td>0 ± 6</td>
<td>0 ± 8</td>
<td>+1 ± 4</td>
</tr>
<tr>
<td>Δ PAP (mm Hg)</td>
<td></td>
<td>+1 ± 2</td>
<td>0 ± 2</td>
<td>+1 ± 2</td>
</tr>
<tr>
<td>Δ CVP (mm Hg)</td>
<td></td>
<td>+1 ± 1</td>
<td>0 ± 1</td>
<td>0 ± 3</td>
</tr>
</tbody>
</table>

* Values are means ± standard deviations. % O₂ Satn = percentage arterial-mixed venous oxygen saturation; PCWP = pulmonary capillary wedge pressure; HR = heart rate; PAP = pulmonary artery pressure; CVP = central venous pressure. None of the changes was significant.

Figure 6. Relationship between the change in central venous pressure (Δ CVP) and the change in intracranial pressure (Δ ICP) at head elevation from 0° to 60°. The linear correlation coefficient is 0.467 and is significant (p < 0.05).
Head elevation and intracranial pressure

(Fig. 3). The development of a high ICP in some of our patients at 60° probably represents the Pattern B of ICP change described by Magnaes. The cause for the significant increase in the ICP seen in some patients at 60° is ill-defined, but may include changes in the intracranial intravascular volume consequent upon autoregulatory attempts to maintain the cerebral blood flow in the presence of a falling systemic blood pressure. Alternatively, it might lead to the triggering of plateau waves in patients with impaired autoregulation caused by transient CSF pressure waves at the time of head elevation. Theoretically, the impaction of the intracranial contents into the foramen magnum that occurs with the patient in the sitting position may prevent egress of CSF from the intracranial compartment into the spinal canal and lead to an increase in the ICP.

We found a direct correlation between changes in the ICP and CVP, suggesting that an increase in the CVP occurring at 60° in some patients might effect intracranial venous back-pressure and hence lead to an increase in the ICP. Although not measured in this study, the increase in the CVP at 60° may have been due to increased intrathoracic pressures possibly caused in part by the abdominal contents being pushed upward with placement of the patient in the sitting position. We do not feel that cervical venous obstruction, which can cause ICP elevation when the head is flexed at the neck, occurred in these patients. Special care was taken during all recording sessions to maintain the head in a neutral, nonflexed position with reference to the torso.

Although we demonstrated that the SAP, measured at the level of the foramen of Monro, fell in a linear fashion with head elevation, the concomitant decrease in the ICP at 15° and 30° resulted in a constant CPP in these positions. In the group as a whole, however, elevation of the head to 60° did effect a significant fall in the CPP, likely due to both a fall in the mean SAP as well as the tendency for the ICP to rise again at 60°, as discussed previously.

It is possible that barbiturate coma may influence the systemic hemodynamic response to changes in body posture in acutely brain-injured patients. However, we were not able to demonstrate any difference in response in the effect of head elevation on ICP and CPP in the patients treated with barbiturates.

We also found that a significant fall in the CI accompanied an increase in head elevation to 60°. Although the cause of this fall in systemic blood flow is not immediately apparent, there was no associated change in the PCWP, CO, or HR. When analyzed by Frank-Starling myocardial function curves, a fall in the mean CI without a change in the mean PCWP implies an associated decrease in left ventricular contractility at this level of head elevation where increases in the ICP were frequently noted. However, since the PCWP, although representing left ventricular end diastolic filling pressure, does not specifically reflect the left ventricular preload, we cannot discount that the decrease in CI may have been solely due to posturally induced changes in left ventricular preload.

The systemic arterial oxygen tension and the change in percentage arterial-mixed venous saturation difference appeared not to be significantly affected by the alterations in head position. These results, however, may be open to criticism because of the short periods of time (10 minutes) that the patients were maintained at each level.

Conclusions

In this study, we found that four parameters critical to cerebral and cardiac function in the acutely brain-injured patient, the ICP, mean SAP, CPP, and CI, were significantly altered by the degree of head elevation. Placing the patient flat results most frequently in the highest ICP. Head elevation to 15° or 30° results most consistently in the lowest ICP while maintaining the CPP and CI. Elevation to 60° usually results in a fall in the CPP and CI, and a variable response of the ICP. Some patients in this study did manifest further decreases in the ICP while maintaining a constant CPP at 60°. We suggest, therefore, that on failure to control the ICP in the patient nursed at 30° of head elevation, therapeutic trial of a further increase in elevation to 60° may be warranted. Furthermore, investigative procedures, such as chest radiography, which require changes in position of the patient should be performed cautiously, and preferably attempted at the degree of head elevation which has resulted in the lowest ICP.

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