Cerebral perfusion pressure, intracranial pressure, and head elevation

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Previous investigations have suggested that intracranial pressure waves may be induced by reduction of cerebral perfusion pressure (CPP). Since pressure waves were noted to be more common in patients with their head elevated at a standard 20° to 30°, CPP was studied as a function of head position and its effect upon intracranial pressure (ICP).

In 18 patients with varying degrees of intracranial hypertension, systemic arterial blood pressure (SABP) was monitored at the level of both the head and the heart. Intracranial pressure and central venous pressure were assessed at every 10° of head elevation from 0° to 50°. For every 10° of head elevation, the average ICP decreased by 1 mm Hg associated with a reduction of 2 to 3 mm Hg CPP. The CPP was not beneficially affected by any degree of head elevation. Maximal CPP (73 ± 3.4 mm Hg (mean ± standard error of the mean)) always occurred with the head in a horizontal position. Cerebrospinal fluid pressure waves occurred in four of the 18 patients studied as a function of reduced CPP caused by head elevation alone. Thus, elevation of the head of the bed was associated with the development of CPP decrements in all cases, and it precipitated pressure waves in some. In 15 of the 18 patients, CPP was maintained by spontaneous 10- to 20-mm Hg increases in SABP, and pressure waves did not occur if CPP was maintained at 70 to 75 mm Hg or above.

It is concluded that 0° head elevation maximizes CPP and reduces the severity and frequency of pressure-wave occurrence. If the head of the bed is to be elevated, then adequate hydration and avoidance of pharmacological agents that reduce SABP or prevent its rise are required to maximize CPP.

KEY WORDS - intracranial pressure - cerebral perfusion pressure - pressure wave - head elevation

Elevation of the head of the bed has been standard neurosurgical practice for management of intracranial pressure (ICP). Yet, such elevation has been shown to be capable of elevating ICP and even inducing pressure waves. More recently, we have provided laboratory and clinical evidence linking pressure waves to cerebral perfusion pressure (CPP) decrements. These latter observations have led us in the present study to examine CPP as a function of head elevation.

In our recent descriptions of the “vasodilatory cascade,” we have pointed out that decreasing CPP is a stimulus to vasodilation, and, given constant systemic pressure at head level, a cycle will be set off that results in a pressure wave. The reduction in CPP will stimulate vasodilation and an increase in cerebral blood volume that will increase ICP. The increased ICP will further lower CPP which will stimulate vasodilation, and the cycle will continue until vasodilation is maximal.

Because the rate of vasodilation increases logarithmically as CPP declines through the “threshold” range of 70 to 80 mm Hg, we need not hypothesize a change in compliance or brain stiffness to explain the abrupt increase in ICP characterizing a pressure wave. The rapid ICP increase is explained by the rapidly rising cerebral blood volume subserved by rapid increments in the rate of vasodilation. Because this phenomenon occurs in the 70- to 80-mm Hg range of CPP, and elevation of the head of the bed will induce a hydrostatic gradient of 15 to 20 torr, we asked ourselves whether the reduction in ICP seen in some patients may not be associated with a CPP reduction. We also sought to explain the occurrence of plateau waves reported by others in association with head elevation on the basis of this mechanism. Therefore, the primary hypothesis that CPP will be maximal at 0° of head elevation was tested. A secondary hypothesis was that elevation of the head will precipitate pressure waves in some patients.
Cerebral perfusion pressure, ICP, and head elevation

**Clinical Material and Methods**

The study group included 18 patients with intracranial hypertension (Table 1). Under strict monitoring their heads were elevated from 0° (horizontal) to 50° in 10° increments. Measurements of ICP (referenced to the foramen of Monro), systemic arterial blood pressure (SABP) referenced to the heart, the mid-axillary line, and the fourth intercostal space, SABP referenced to the foramen of Monro, and central venous pressure (CVP) referenced to the heart were made at each position. Cerebral perfusion pressure was taken as the difference between SABP and ICP when measured at the foramen of Monro via intraventricular cannulas (No. 5 French feeding tubes). Reduction in CPP below 50 torr was used as a criterion to abort the study in any given patient.

**Results**

Figure 1 and Table 2 summarize the essential findings of this study. As head elevation progressed, there was a gradual decrement in SABP recorded at head level from approximately 95 to 81 mm Hg at 50°. This relationship

**TABLE 1**

Demography and initial ICP, SABP, and CPP in 18 patients without head elevation*

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis &amp; GCS</th>
<th>CT Findings</th>
<th>Pressure Levels (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36, F</td>
<td>hydrocephalus, GCS 15</td>
<td>noncommunicating hydrocephalus (acoustic neuroma)</td>
<td>ICP: 20 SABP: 104 CPP: 84 CVP: 3</td>
</tr>
<tr>
<td>2</td>
<td>21, F</td>
<td>aqueductal stenosis, hydrocephalus, GCS 15</td>
<td>noncommunicating hydrocephalus</td>
<td>ICP: 9 SABP: 85 CPP: 76 CVP: 0</td>
</tr>
<tr>
<td>3</td>
<td>29, M</td>
<td>head injury, GCS 3</td>
<td>small ventricles, no mass, no parenchymal lesion</td>
<td>ICP: 17 SABP: 117 CPP: 100 CVP: 18</td>
</tr>
<tr>
<td>4</td>
<td>19, F</td>
<td>head injury, GCS 4</td>
<td>small ventricles, compressed cisterns, punctate intraparenchymal lesions</td>
<td>ICP: 17 SABP: 100 CPP: 83 CVP: 4</td>
</tr>
<tr>
<td>5</td>
<td>27, M</td>
<td>traumatic ICH, GCS 6</td>
<td>frontotemporal hemorrhage, 2-cm lt→rt shift, brain edema postop small ventricles</td>
<td>ICP: 21 SABP: 114 CPP: 93 CVP: 12</td>
</tr>
<tr>
<td>6</td>
<td>12, M</td>
<td>Reye’s syndrome, GCS 7</td>
<td>small ventricles</td>
<td>ICP: 29 SABP: 84 CPP: 55 CVP: 0</td>
</tr>
<tr>
<td>7</td>
<td>58, M</td>
<td>extreme capsule hemorrhage, GCS 6</td>
<td>2.5-cm rt→lt shift postop</td>
<td>ICP: 49 SABP: 95 CPP: 46 CVP: 13</td>
</tr>
<tr>
<td>8</td>
<td>24, F</td>
<td>head injury, GCS 4</td>
<td>small ventricles, compressed cisterns, punctate intraparenchymal lesions</td>
<td>ICP: 18 SABP: 108 CPP: 90 CVP: 8</td>
</tr>
<tr>
<td>9</td>
<td>52, F</td>
<td>cerebellar &amp; frontal hemorrhage, GCS 9</td>
<td>mild hydrocephalus, small rt frontal hemorrhage postop (posterior fossa craniectomy)</td>
<td>ICP: 30 SABP: 122 CPP: 92 CVP: 0</td>
</tr>
<tr>
<td>10</td>
<td>19, M</td>
<td>acute SDH, GCS 6</td>
<td>1.5-cm lt→rt shift postop</td>
<td>ICP: 30 SABP: 108 CPP: 78 CVP: 0</td>
</tr>
<tr>
<td>11</td>
<td>62, M</td>
<td>rt parietal GBM, GCS 14</td>
<td>2.5-cm rt→lt shift</td>
<td>ICP: 6 SABP: 95 CPP: 89 CVP: 0</td>
</tr>
<tr>
<td>12</td>
<td>49, F</td>
<td>bifrontal GBM, GCS 12</td>
<td>bifrontal enhancing lesion, compressed ventricles</td>
<td>ICP: 28 SABP: 100 CPP: 72 CVP: 4</td>
</tr>
<tr>
<td>13</td>
<td>52, F</td>
<td>traumatic lt parietal intracerebral hemorrhage, GCS 6</td>
<td>1.0-cm lt→rt shift</td>
<td>ICP: 18 SABP: 70 CPP: 52 CVP: 0</td>
</tr>
<tr>
<td>14</td>
<td>34, F</td>
<td>hydrocephalus, 2° spinal tumor, GCS 14</td>
<td>communicating hydrocephalus</td>
<td>ICP: 24 SABP: 98 CPP: 74 CVP: 0</td>
</tr>
<tr>
<td>15</td>
<td>29, M</td>
<td>head injury, GCS 4</td>
<td>small ventricles, compressed cisterns</td>
<td>ICP: 30 SABP: 87 CPP: 57 CVP: 11</td>
</tr>
<tr>
<td>16</td>
<td>83, F</td>
<td>frontal meningioma, seizure disorder, GCS 6</td>
<td>brain edema, lt→rt shift postop</td>
<td>ICP: 19 SABP: 89 CPP: 70 CVP: 0</td>
</tr>
<tr>
<td>17</td>
<td>25, M</td>
<td>head injury, GCS 4</td>
<td>small ventricles, compressed cisterns</td>
<td>ICP: 19 SABP: 99 CPP: 80 CVP: 10</td>
</tr>
<tr>
<td>18</td>
<td>23, M</td>
<td>head injury, GCS 5</td>
<td>small ventricles, compressed cisterns, intraparenchymal lesions</td>
<td>ICP: 13 SABP: 78 CPP: 65 CVP: 8</td>
</tr>
</tbody>
</table>

* ICP = intracranial pressure; SABP = systemic arterial blood pressure; CPP = cerebral perfusion pressure; CT = computerized tomography; CVP = central venous pressure; GCS = Glasgow Coma Scale score; SDH = subdural hematoma; GBM = glioblastoma multiforme.

Fig. 1. Composite graph showing that intracranial pressure (ICP) does decline as head elevation progresses, but that the hydrostatic decrement in systemic arterial blood pressure (SABP) at head level is greater. Therefore, cerebral perfusion pressure (CPP) declines. Only the tendency for SABP to increase prevents a greater CPP decrement. CVP = central venous pressure; NS = not significant. Values are means ± standard error of the means (sem).
FIG. 2. Case 18. The intracranial pressure (ICP) gradually decreased from 14 to 6 mm Hg at 50° head elevation; however, cerebral perfusion pressure (CPP) was lower throughout, and only the systemic arterial blood pressure (SABP) response kept CPP as high as it was. CVP = central venous pressure.

was described by the equation \( y = -0.29x + 94 \) (\( p < 0.001 \)). The ICP similarly declined from a mean of 22 mm Hg to approximately 16 mm Hg for the same degree of head elevation (\( y = -0.1x + 21 \), 0.05 < \( p << 0.1 \)). Because SABP recorded at head level declined faster than ICP (see Fig. 4), there was a net decrement in CPP which was described by the equation \( y = -0.18x + 73 \) (\( p < 0.05 \)); the average drop was from 73 mm Hg to approximately 65 mm Hg. Central venous pressure dropped slightly as head elevation continued. Systemic arterial blood pressure at heart level rose slightly from 96 to 99 mm Hg at 50° head elevation. The SABP response, increasing on average, prevented a greater decline in CPP.

Linear regression analysis yielded significant relationships between SABP at the head, CPP, and CVP. The equation describing the relationship between ICP and head elevation has a statistical significance of \( p < 0.10 \) to 0.05. Nine of 18 patients demonstrated a consistent fall in ICP with head elevation which contributes substantially to the "near significance" of this relationship; Fig. 2 demonstrates this pattern. It should be noted that no patient experienced a lasting improvement in CPP as a result of head elevation, regardless of whether the ICP decreased or not. The remaining patients were distributed between varying patterns of response. One patient (Case 10) had virtually no change in his ICP (Fig. 3 left), one (Case 11) demonstrated a rise in ICP followed by a fall (Fig. 3 center), and the last (Case 9) demonstrated varying patterns of response with the ICP terminating at the same level or even higher than the initial value (Fig. 3 right).

In two patients the study was aborted when their CPP dropped below 50 mm Hg. The studies were completed 48 to 72 hours later. In four patients, at 50° of head elevation the ICP declined and a significant increase in SABP similar to a Cushing response or ischemic response combined to yield a net increment in the CPP of about 5 to 7 mm Hg. However, these were transient phenomena and were followed by increases in ICP as the hypertensive response decayed.

**Discussion**

Our results suggest that if CPP is to be maximized, then the optimal results are obtained by nursing patients with intracranial hypertension flat in bed. We found no case in which CPP was improved by head elevation, even in the 50% of our patients whose ICP did decrease. The CPP effect clearly relates to the hydrostatic decrease in SABP noted at cranial level as the head is raised above the heart (Fig. 4).

The results obtained by Durward, et al., also suggest that CPP declines as head elevation increases, although these investigators did not consider the decline in CPP to be statistically significant until 60° elevation was reached. They also gave an example of a pressure wave precipitated by head elevation. Maintenance of CPP as head elevation proceeded may have been due to SABP increasing in response to decreasing CPP. We noted this pattern in a few patients in our series, and it was most effective in CPP maintenance for short time periods with minor degrees (10°) of head elevation. However, if the SABP increase occurred as the result of ischemia or increasing "stress" on the injured brain, it is important to note and alleviate that adverse stimulus.

**TABLE 2**

<table>
<thead>
<tr>
<th>Head Elevation</th>
<th>SABP (heart)</th>
<th>SABP (head)</th>
<th>ICP</th>
<th>CPP</th>
<th>CVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>0°</td>
<td>95.6 ± 3.1</td>
<td>94.8 ± 3.1</td>
<td>22.2 ± 2.3</td>
<td>73.0 ± 3.4</td>
<td>8.0 ± 1.1</td>
</tr>
<tr>
<td>10°</td>
<td>97.6 ± 2.5</td>
<td>90.8 ± 2.9</td>
<td>20.3 ± 2.4</td>
<td>70.4 ± 3.4</td>
<td>9.2 ± 1.1</td>
</tr>
<tr>
<td>20°</td>
<td>98.4 ± 2.8</td>
<td>88.8 ± 2.8</td>
<td>18.8 ± 2.2</td>
<td>69.8 ± 3.2</td>
<td>8.8 ± 0.7</td>
</tr>
<tr>
<td>30°</td>
<td>96.2 ± 3.4</td>
<td>85.2 ± 2.4</td>
<td>17.6 ± 2.3</td>
<td>67.2 ± 2.8</td>
<td>6.8 ± 1.0</td>
</tr>
<tr>
<td>40°</td>
<td>96.4 ± 3.0</td>
<td>80.6 ± 3.1</td>
<td>16.2 ± 2.5</td>
<td>64.3 ± 3.2</td>
<td>6.0 ± 0.8</td>
</tr>
<tr>
<td>50°</td>
<td>99.2 ± 3.0</td>
<td>81.2 ± 3.1</td>
<td>16.2 ± 2.3</td>
<td>64.8 ± 2.6</td>
<td>5.7 ± 1.1</td>
</tr>
<tr>
<td>y</td>
<td>0.03x + 96</td>
<td>-0.29x + 94</td>
<td>-0.1x + 21</td>
<td>-0.18x + 73</td>
<td>-0.1x + 9</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>p &lt; 0.001</td>
<td>0.05 &lt; p &lt; 0.1</td>
<td>0.01 &lt; p &lt; 0.05</td>
<td>0.001 &lt; p &lt; 0.01</td>
</tr>
</tbody>
</table>

* Values (in mm Hg) are means ± standard errors of the means. SABP = systemic arterial blood pressure; ICP = intracranial pressure; CPP = cerebral perfusion pressure; CVP = central venous pressure; NS = not significant. Mean CPP = mean SABP - mean ICP.

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**Left:** Case 10. Head elevation had little effect upon intracranial pressure (ICP), but because systemic arterial blood pressure (SABP) did not respond adequately, cerebral perfusion pressure (CPP) declined. **Center:** Case 11. Elevation of the head of the bed up to 20° resulted in a systemic pressor response that maintained head-level SABP and CPP. Beyond 20°, SABP could not be maintained, CPP fell, and ICP rose, probably as a result of the low CPP. This phenomenon occurred in 20% of our patients. **Right:** Case 9. The ICP increased slightly at 10° elevation, but ultimately fell to 22 mm Hg before rising again. The SABP at heart level changed little and declined by 18 mm Hg at head level. The CPP never improved over that at 0° elevation.

This form of analysis may also change the conclusions reached by other investigators.

It is important to note that seemingly minor CPP decrements of 10 to 20 mm Hg may be adequate to precipitate major intracranial pressure waves. Our previous observations have suggested that SABP decrements of as little as 10 mm Hg were adequate to precipitate CPP decrements and initiate the vasodilatory cascade. Twenty percent of our patients had pressure waves induced by this maneuver; two patients had ICP increases associated with profound CPP decrement which led to temporary termination of the study. Durward, et al., Magnaes, and Ropper, et al., have observed just this effect. The latter authors very carefully noted that, of their 20 patients, two experienced the lowest ICP while in the horizontal position, 10 had

*Fig. 3.* Left: Case 10. Head elevation had little effect upon intracranial pressure (ICP), but because systemic arterial blood pressure (SABP) did not respond adequately, cerebral perfusion pressure (CPP) declined. Center: Case 11. Elevation of the head of the bed up to 20° resulted in a systemic pressor response that maintained head-level SABP and CPP. Beyond 20°, SABP could not be maintained, CPP fell, and ICP rose, probably as a result of the low CPP. This phenomenon occurred in 20% of our patients. Right: Case 9. The ICP increased slightly at 10° elevation, but ultimately fell to 22 mm Hg before rising again. The SABP at heart level changed little and declined by 18 mm Hg at head level. The CPP never improved over that at 0° elevation.

*Fig. 4.* Segments of continuous tracings in a patient with the head of the bed (HOB) elevated 0° (left) and 30° (right). The effects of head elevation are seen on systemic arterial blood pressure (SABP, recorded at head level), intracranial pressure (ICP), and cerebral perfusion pressure (CPP). With the patient lying flat the ICP was higher but the CPP was stable at 80 to 90 mm Hg. In the patient whose head was elevated 30°, the SABP was not maintained, the CPP is reduced, and ICP waves are seen.
lower ICP with the head elevated to 60°, and seven were
una rational conclusion that positioning should be individu-
alized for each patient based upon the ICP data.

The only study suggesting that ICP reduction occurs
in all patients was that of Kenning, et al., they used
extreme degrees (45° and 90°) of head elevation. No
data were offered concerning CPP, nor were continuous
records of ICP or SABP offered. Our results suggest
that very significant CPP reduction will occur with this
positioning, and this conclusion is consistent with the
CPP reduction noted by Durward, et al. 1

The mechanism for CPP reduction is twofold. With
progressive elevation of the head above the heart, the
hydrostatic pressure at the cranial level declines. In
addition, cardiac preload is also reduced as the heart is
raised further above the hydrostatic indifferent point
(venous). 2 The results of Durward, et al., 1 in this patient
population suggest that the latter effect is small until
more extreme positions are reached. Still, the effect is
toward reduction in cardiac output and reduction in
SABP recorded at the brain level. It is this latter effect
that may be very important in some patients, especially
in those who are dehydrated, who are septic, who are
on ventilators with or without positive end-expiratory
pressure, and/or who have cardiac disease; these pa-
tients may experience greater than the expected hydro-
static drop in SABP at head level.

The mechanism for ICP reduction with head eleva-
tion appears related to potentiation of venous outflow.
It is known that CVP and jugular venous pressures
decline markedly with head elevation; 2 presumably this
leads to reduction in the cerebral venous blood volume,
and a decrease in ICP follows.

It is of some importance to consider that displace-
ment of venous volume is probably the first event to
occurs as the intracranial mass (consisting of edema or
hemorrhage) accumulates. Thus, the patient with the
highest ICP will likely have the smallest amount of
venous blood to displace, suggesting that the patient
who benefits least from head elevation is the one who
might, superficially, appear to need it the most. Con-
versely, those patients with the greatest decrement in
ICP have the greatest venous volume available for efflux
and need ICP reduction least of all. It would appear
that this mechanism may act to lower ICP in only 50%
of neurosurgical patients at best.

Another potential mechanism for ICP reduction re-
lates to states where perfusion pressure is below its lower
limits (that is, where vasodilation is already maximal).
If the head is raised, SABP will decline further and a
passive decline in ICP will occur as the vasculature
collapses. 5,6,11 This phenomenon, the direct variation
of ICP as a function of SABP, occurs at very low
perfusion pressures (generally < 40 mm Hg) and has
been exhaustively studied by Langfitt and colleagues. 6
Clearly, a decline of ICP as a function of head elevation
in this circumstance is more ominous than reassuring
and further complicates the interpretation of studies of
head elevation without concurrent SABP data. 3

A final mechanism for ICP reduction by head eleva-
tion has been suggested by Kenning, et al. 3 They sug-
gested the possibility that hydrostatic displacement of
cerebrospinal fluid (CSF) into the spinal subarachnoid
space from the cranium might lower CSF pressure. The
possibility is intriguing, but, as they suggest, most of
the CSF buffer capacity has already been used and
significant CSF displacement may not be feasible. Like-
wise, in states of very high ICP, the increased pressure
differential between the cranial and lumbar CSF spaces
(ΔP) occurring as a result of head elevation would be
relatively small compared to the ΔP expected when CSF
pressure was lower. Obstruction of the basal CSF spaces
would potentiate the problem. Again, the patients most
in need of ICP reduction would appear to be the ones
least likely to benefit.

The mechanism for ICP increase with elevation of
the head appears to be identical to that for genera-
tion of ICP or plateau waves. 9,10,11 When CPP reduction
occurs, vasodilation follows which increases cerebral
blood volume; then ICP increases which further lowers
CPP and continues the cycle. The ICP continues to
increase until vasodilation is maximal or until CPP is
restored, usually by a Cushing or ischemic response
with rapid elevation of the SABP.

This mechanism is potentiated by the "non-linear"
rate of vasodilation occurring in the vasculature. Most
vasodilation occurs below a CPP of 80 mm Hg. There-
fore, most of the cerebral blood volume changes occur
below this CPP level. At this CPP level ICP waves have
been precipitated. In contrast, if a CPP-SABP change
occurs at 100 mm Hg or above, there is little effect on
ICP because very little vasodilation or constriction takes
place at high perfusion pressures. Thus, a relatively
wide variation in CPP is tolerated at these levels.

Conclusions

From this study of 18 patients with intracranial
hypertension, we can draw the following conclusions.

1. Cerebral perfusion pressure was maximal with
patients horizontal in bed even though ICP was usually
at its highest point. No other position resulted in im-
proved CPP.

2. If adequate CPP is viewed as more desirable than
an absolute level of ICP, then 0° head elevation (hor-
izontal) is the optimal position to achieve this goal.

3. Cerebral perfusion pressure analysis of the effect
of head elevation upon ICP enables us to explain: 1) 
why head elevation in some patients precipitates ICP
increases; 2) why patients may transiently have lower
ICP, but clinically worsen; and 3) why many patients
show little change in ICP.

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