The Effect of Head Elevation on Intracranial Pressure in Intracranial Hypertension

SHIH-TSENG LEE and JYI-FENG CHEN

ABSTRACT

To understand the effect of head elevation on increased intracranial pressure, studies were performed in 22 patients with cerebral edema due to head injury. Intracranial pressure was measured with the patient in four different positions; head at 0 degree, head elevate 30 degrees, 45 degrees and 60 degrees, without turning the head. Intracranial pressure results were as follows: at 0 degree, 24.73 ± 1.50 (mean \pm SE) mmHg; head elevate 30 degrees, 23. 64 ± 2.40 mmHg; head elevate 45 degrees, 19.14 ± 1.92 mmHg; head elevate 60 degrees, 17.18 ± 1.70 mmHg. Intracranial pressures were lower in all head elevate position but only head elevate 45 degrees or 60 degrees reach the statistical significance (P < 0.001). The data suggest that the head elevate 45 degrees or 60 degrees have a greater chance to reduce the increased intracranial pressure in patients with intracranial hypertension. The elevation of head to 45 degrees or 60 degrees might be the first, simplest treatment to intracranial hypertension.

Key words: Head elevation, intracranial hypertensin, intracranial pressure.

Control of increased intracranial pressure remains a major challenge in the management of neurological and neurosurgical patients. One of the simplest way has been the elevation of patients' head which is presumed to reduce cerebral venous pressure or and hence ICP. Many previous reports noted that moving the patient from the supine to sitting position causes significant reduction of intracranial pressure⁽¹⁻⁵⁾, but most studies were performed

on patient with normal intracranial pressure, after craniotomy. In order to determine the optimal head position for patients with raised intracranial pressure we studied the effect of several head positions on intracranial pressure by using the subarachnoid screw to monitor ICP continuously.

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MATERIAL AND METHOD

This study was performed in 22 patients with cerebral edema cause by head injury without intracranial hematoma. The groups consisted of 15 males and 7 females, ranging in age from 21 to 63 years with a mean of 32.54 yeares (Table I). All patients were admitted to the intensive care unit with a frontal subarachnoid screw for continuous monitoring of intracranial pressure. The subarachnoid screw was connected by a sterile catheter (Cobe Laboratories, Inc., Lakewood, Colo) to a transducer (Hewlett Packard, Andover, Mass.). The pressure transducer was calibrated and kept at the level of the patient's foramen of Monro for each pressure recording. A continouous digital display of mean intracranial pressure was obtained. The following positions were studied: supine with bed at the horizontal setting (0 degree), supine with head of the bed up 30 degrees, up 45 degrees

Table I. Summary of Demographic Features of 22 Patients

Age (years)	
Range	21-36
$Mean \pm SEM$	32.54 ± 2.86
Sex	
Male	15
Female	7
Coma Scale	
8	6
7	7
6	5
5-	3
4	1
Cause of Injury	
Motorcycle	14
Car	5
Pedestrian	3

and up 60 degrees. The neck of patients was maintained in all positions to prevent jugular vein compression. Intracranial pressure measurements for each position were taken every 10 seconds for 1 minute after a 5 minute stablization period. The mean of intracranial pressure measurements was used for statistical comparisons between the four groups. Student's t test was used for statistical significance in the data interpretation.

RESULTS

Each position was compared with the standard head position, of supine, 0 degrees elevation. The mean intracranial pressure for each position compared to the mean intracranial pressure at 0 degree position, 24.73 ± 1.50 (mean ± SEM) mmHg, and statistical significance of the difference determined. For the group as a whole, the intracranial pressures were decreased after elevation of the head (Fig. I): head elevated 30 degrees, 23.64 ± 2.4 mmHg (P>0.05), head elevated 45 degrees, 19.14 \pm 1. 92 mmHg (P<0.001), and head elevated 60 degrees, I7.18 \pm I.70 mm Hg (P < 0.001). However, some patients had a different changes. Seven patients with 30 degrees head up, three patients with head up 45 degrees, and two patients with head up 60 degrees showed increased intracranial pressure as compared with their own standard position (Fig. 2-4). The change in mean intracranial pressure from supine 0 degree to the different elevated positions were as follows: head up 30 degrees, -I.I mm Hg, head up 45 degrees, -5.6 mmHg and head up 60 degrees, -7.2 mmHg.

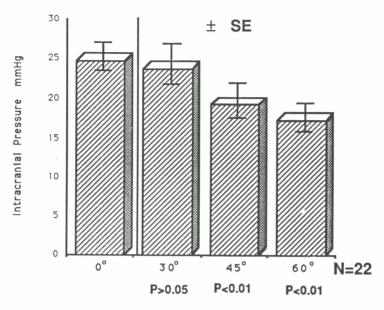


Fig. I. Effects of head elevation on intracranial pressure

30°: head up 30 degrees. 45°: head up 45 degrees. 60°: head up 60 degrees.

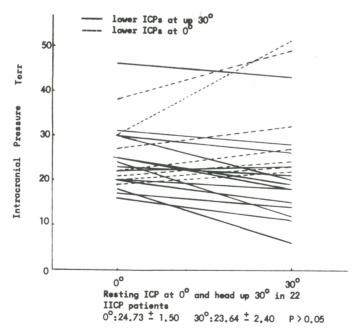


Fig. 2. Individual response of 22 patients' intracranial pressure on change position from O degrees to head up 30 degrees.

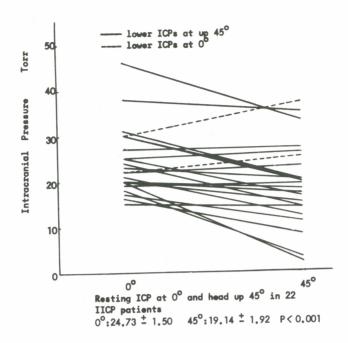


Fig. 3. Individual response of 22 patients' intracranial pressure on change position from 0 degrees to head up 45 de4grees.

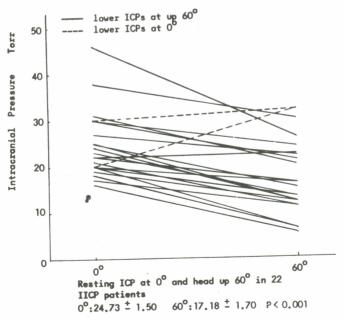


Fig. 4 Individual response of 22 patients' intracranial pressure on change position from 0 degrees to head up 60 degrees.

DISCUSSION

Many well known factors contibute to intracranial hypertension in head injury patients, such as development of intracranial mass lesions, change of blood pressure, internal jugula vein compression due to malposition, positive pressure ventilation, displacement of CSF $etc^{(1-6)}$. All of these would be expected to have a reduction of intracranial pressure after elevation of the head. For patients without intracranial pathology the head up position may lower intracranial pressure due to caudal movement of cerebrospinal fluid⁽⁷⁾. Kennig et al.⁽⁵⁾ have demonstrated that in the sitting or semisitting position the intracranial pressure will marked diminish in patients with intracranial hypertension but the sitting position is rather difficult to maintain for a long period of time during the care of prolong comatous patients. In our results for patients as a whole head up 30 degrees only a slight decrease in intracranial pressure was observed, but head up 45 degrees or 60 degrees showed significant reduce of intracranial pressure in patients with intracranial hypertension as was found in other studies $^{(1-5)}$. The possible mechanism may be that the further the head is elevate the more additional drainage of cerebrospinal fluid and cerebral venous flow causing more decline in intracranial pressure as indicated by the Monro-Kellie doctrine.

Ropper et al.⁽⁸⁾ proposed that brain tissue shift toward the foramen magnum impeding CSF outflow may raise rather than reduce intracranial pressure as the head is elevated. There are several studies demonstrating that in with normal condition when the patient is sitting or

semisitting the dural sinus pressures are reduced to negative values (1,9), but those studies were performed with patients without intracranial hypertension and with some extent of craniectomy and without concomitant measurement of intracranial pressure. Iwabuchi et al. (9) also noted that jugular bulb pressure did not always reflect the dura confluences sinus pressure. Increased intracranial pressure may cause secondary compression to the basal dural sinuses. If the elevation of the head causes a caudal shift of the intracranial content and compressed of the basal dural sinuses it may cause further venous stasis and hence an increase in intracranial pressure. Whether increased intracranial pressure will cause secondary basal dural sinuses compression after head elevation needs further study.

Although in general, the result from our study suggested in patients with intracranial hypertension head up 45 degrees or 60 degrees is better than head up 30 degrees in reduce intracranial pressure, individual responses were variable.

The authors recommend elevation of the head to 45 degrees or 60 degrees as a first simple treatment of intracranial hypertension. Continue monitoring of intracranial pressure is of great importance because individual response to head elevation vary. When intracranial pressure monitoring is not available, head up 45 degrees or 60 degrees is suggested.

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顱內壓升高病人頭部抬高後對顱內壓之影響

李石增 陳志豊

為了瞭解頭部抬高後對顱內壓增高病患降低顱內壓的效應,我們將22位因頭部受傷併有顱內壓增高的病患予頭部抬高不同角度(30°,45°,60°)。將各種不同頭部抬高角度後顱內壓的變化與平躺時(0°)之顱內壓加以比較。結果顯示顱內壓於平躺時(0°)為24.73±1.50(平均值±標準差)毫米汞柱,頭部提高30°時為23.64±2.40毫米汞柱,抬高45°時為19.14±1.92毫米汞柱,抬高60°時為17.18±1.70毫米汞柱。在各種頭部提高角度與平躺時(0°)比較,發現提高45°與60°可明顯的降低顱內壓(P<0.001)但是頭部抬頭30°則對降低顱內壓不具功效(P>0.05)。

本研究顯示將頭部抬頭 45°至 60°爲治療顱內壓增高的一種簡單,有效的方法。