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Changes in Intracranial Pressure in Various Positions of the Head in Anaesthetised Patients

Vinod K Grover, MD,MNAMS* Indu Bala, MD**
Someshwar S Bandi, MD***
Rajesh Mahajan, MD*** Virender K Khosla, MS, MCh.*

Objectives: To study ICP changes during various head positions in patients undergoing ventriculoperitoneal (VP) shunt surgery.

Methods: The study was conducted on 20 randomly selected American Society of Anesthesiologists Physical Status Class I or II patients of either sex aged above five years scheduled for elective ventriculoperitoneal shunt surgery. ICP was recorded in 15° head up, 15° head down, right and left lateral bending of head, right and left rotation of head and was compared with horizontal (neutral) position.

Results: Intracranial pressure increased in all positions but significant increase was observed in right rotation (40.8%; $p<0.01$), left rotation of the head (22.2%; $p<0.001$) and head down 15° position ($p<0.05$; 6.7%). No clinically significant hemodynamic changes were observed in various positions. No significant changes were observed in oxygen saturation.

Conclusion: Rotation of head to either right or left and head down position increases the ICP significantly. Head elevation may not reduce ICP in all cases. It depends on degree of elevation, intracranial pathology and associated neck position.

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The factors influencing the intracranial pressure are of prime importance to the neuroanaesthetist, since intracranial surgery becomes difficult or hazardous in presence of uncontrolled rise in ICP. In situations where ICP is raised, the brain may be so swollen and 'tight', that it is impossible for the surgeon to operate on the structures at the base of the brain[1].

* Professor
** Additional Professor
*** Senior Resident
Departments of Anaesthesia and Intensive care & Neurosurgery
Postgraduate Institute of Medical Education & Research
Chandigarh
India

The problems which need to be tackled during neuroanaesthesia are regulation of brain volume and tension, control of hemorrhage and handling of vital structures in and near the surgical field[2]. In order to avoid the deleterious effects of raised ICP, various methods such as hyperventilation, hypothermia, CSF drainage, surgical decompression, pharmacological agents like corticosteroids, thiopentone, hyperosmolar agents (mannitol) and loop diuretics have been used to lower the ICP[3-10].

One of the common factors which significantly affects ICP is the "position of the head"[11]. Head position affects ICP by altering mean arterial pressure (MAP), jugular venous drainage and cerebral blood volume[12]. The optimum position which provides adequate exposure of the lesion as well as keeps the ICP at the lowest has to be determined. The present study was designed to study the effect of various positions of the head on the ICP, in anaesthetised patients undergoing ventriculoperitoneal (VP) shunt surgery.

METHODS

The study was carried out on 20 randomly selected American Society of Anesthesiologists Physical Status Class I or II patients of either sex aged above 5 years scheduled for elective ventriculoperitoneal shunt surgery. After approval from the institutional ethics committee, a written informed consent was obtained from all the patients or guardians before the procedure.

Patients with supratentorial tumours, preexisting cardiovascular disease, hepatic, renal or endocrine disease were excluded from the study. No premedication was given to any of the patients. After preoxygenation with 100% oxygen for 5 minutes, anaesthesia was induced with intravenous (iv) thiopentone 4-6 mg/kg. Tracheal intubation was facilitated using 0.15 mg/kg pancuronium. Anaesthesia was maintained with 66% nitrous oxide in oxygen and pancuronium. Ventilation was adjusted so as to maintain arterial partial pressure of carbon dioxide ($PaCO_2$) in range of 30-35 mmHg. At the end of surgery, neuromuscular blockade was reversed with atropine 0.02 mg/kg and neostigmine 0.05 mg/kg.

After making a burr hole in the skull, a 6 french gauge umbilical catheter was introduced in the lateral ventricles by the neurosurgeon. This catheter was connected to pressure tubing which in turn was connected to the transducer (Spectramed, Statham, PXL, USA). The whole system was filled with sterile normal saline. The pressure transducer was connected to Horizon-2000 monitor. Care was taken such that CSF leakage was avoided while connecting intraventricular catheter to the pressure tubing. Measurements were taken only after steady state was achieved (ie. when heart rate and blood pressure returned to within 10% of basal value). The transducer was fixed at the level of external auditory meatus which was taken as the zero point. Transducer was zeroed each time the position of the head was changed. ICP was recorded in head positions: (a) Horizontal (neutral), (b) 15° head elevation (up), (c) 15° head down (d), Right lateral bending of head, (e) Left lateral bending of head, (f) Right rotation, (g) Left rotation. The degree of head elevation and head down was measured using a

Goniometer. Recordings were obtained at an interval of five minutes between changes in each position.

Usually for VP shunt, burr hole was made in the skull in the parietal region. So lateral rotation of that side could not be done (eg. in left VP shunt cases, left rotation could not be done), to avoid dislodgment of the catheter. Mannitol or furosemide were not administered in any of the cases.

Along with ICP, heart rate, systolic, diastolic and mean arterial pressure, central venous pressure (CVP), arterial blood gases and oxygen saturation were monitored.

The data was collected, tabulated and was analysed using student's paired 't' test.

RESULTS

Twenty patients posted for VP shunt surgery were taken up in the study. There were 12 male and 8 female patients aged between 5-61 years with a mean age of 22 years.

All the readings of intracranial pressure taken in various positions were compared with respect to neutral (horizontal) position. Only one rotation could be tested in each patient because the approach for VP shunt surgery was through parietal burr hole and ipsilateral rotation was avoided. A left parietal burr hole was made in 14 patients; a right parietal burr hole in 5 patients and a frontal burr hole was made in 1 patient. In the last patient recordings were obtained in all positions.

The most significant increase in ICP in our study occurred in right rotation of head (40.8%; $p<0.01$) and left rotation of head (22.2%; $p<0.001$). Significant increase in ICP (6.7%) was also seen in 15° head down position. In 15° head up position, ICP increased by 5.5% above the baseline value (Figs.1&2). The increase in ICP was not statistically significant ($p>0.05$). Right and left lateral bending of head increased ICP to a very minimal extent of 1% and 2% respectively from neutral position, which was not statistically significant.

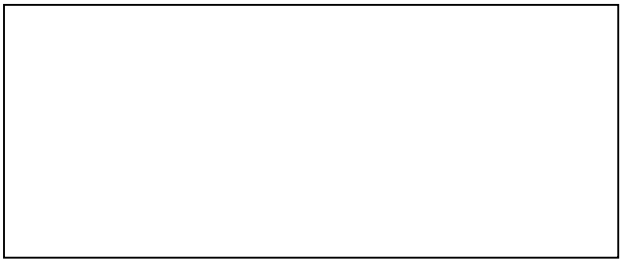

Figure 1. Mean ICP (mmHg) changes in various head positions compared to neutral position.

N = Neutral, HU = Head elevation, HD = Head down,
RL = Right lateral bending, LL = Left lateral bending,
*RR = Right rotation, LR = Left rotation. *p<0.05, **p<0.01, ***p<0.001*

Significant ($p<0.05$) decrease in heart rate was observed during 15° head down, right and left bending of head, and right and left rotation of head. No statistical significant changes were observed in mean and diastolic arterial pressure in various head positions.

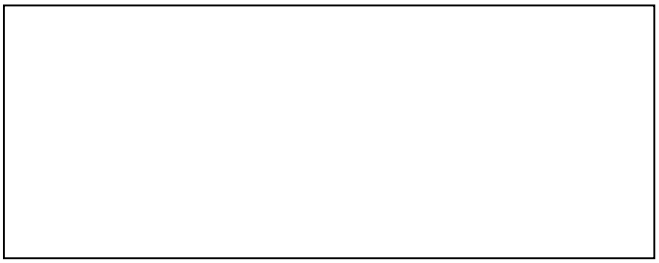

Figure 2. Percentage changes in ICP in various head positions.

N = Neutral, HU = Head elevation, HD = Head down,
RL = Right lateral bending, LL = Left lateral bending,
*RR = Right rotation, LR = Left rotation. *p<0.05, **p<0.01, ***p<0.001*

There was a decrease in CVP in all positions but significant ($p<0.05$) decrease was observed in head up and right and left bending of head and right and left rotation of head. Oxygen saturation was maintained above 95% in all positions and $PaCO_2$ between 30-35 mmHg.

DISCUSSION

An elevated intracranial pressure is one of the most common and difficult problems encountered by anesthesiologists and neurosurgeons. An elevated ICP may lead to further brain damage by promoting ischemia and subsequent brain shifts and herniations.[1,2] Elevated ICP can produce worsening of neurological symptoms, reduction of cerebral perfusion pressure, impairment of cerebrovascular regulatory mechanism, difficulty in operating due to bulging of brain and difficulty in closure of duramater[1].

Management of patients at risk for intracranial hypertension, or indeed patients with established intracranial hypertension traditionally involves regimen consisting of hyperventilation, drugs and surgical decompression[3,4,7-9]. Since any postural change of head position may potentially influence the ICP[11,12], we evaluated the effect of various positions of head on ICP as well as on integrated cardiopulmonary function, in anesthetised patients undergoing ventriculoperitoneal shunt surgery.

The most significant increase in ICP in our study was observed in right rotation of the head and left rotation of the head. ICP increased by 40.8% in right rotation of the head and 22.2% in left rotation of the head above the basal level. These values were statistically significant ($p<0.01$ and $p<0.001$ respectively). Similar result was also found in a study by Emery et al where head rotation to right increased ICP significantly.[13] The study by Mavrocordatos et al concludes that rotation of head, both right and left causes statistically significant increase in ICP in horizontal position and

also in head up position[11]. This increase in ICP is probably due to obstruction to jugular venous flow on the side to which head is turned. Thus extreme caution should be exercised during patient positioning so that jugular venous outflow obstruction can be avoided. There has been report by Sherman, who in his study concluded that excessive head rotation can produce cerebral ischaemia by compression of the carotid or the vertebral arteries[14].

Significant increase in ICP (6.7%) was seen in 15° head down position. This finding was similar to the results obtained by Mavrocodatos et al and Emery et al where head down position increased ICP[11,13]. The increased ICP in dependent position may be due to the hydrostatic effect of the dependent position, causing relative reduction in jugular venous outflow, leading to pooling of blood in cerebral veins causing reduced cerebral compliance.

We found that 11 out of 20 patients manifested a rise in ICP during 15° head up position. Mean rise was 1.3 mmHg but this was statistically not significant ($p>0.05$). In the studies conducted by Rosner et al and Magnes et al also ICP was found to increase in some cases inspite of head elevation[15,16]. The rise in ICP on 15° head elevation in our study was mainly seen in patients with posterior fossa tumours. The mechanism for this rise in ICP may be (a) reduction of cerebral perfusion leading to generation of plateau waves, (b) minor degree of neck flexion during 15° head elevation leading to venous engorgement, (c) posterior fossa tumour mass might have minor position change leading to reduced craniospinal outflow of cerebrospinal fluid producing raised ICP. These observations are in contrast to the study by Emery et al, Durward et al and Feldman et al who found reductions of ICP by elevation of head[13,17,18].

Right lateral bending of head and left lateral bending of head increased ICP to a very minimal extent (1% and 2% respectively) from the neutral position, which was statistically not significant ($p>0.05$). This change may be due to minor pressure effects on the jugular venous system.

There was a decline in heart rate by 0.76% to 5.6% from the basal value in all head positions. Except for the increase in heart rate observed in 15° head up position, all other positions caused statistically significant ($p<0.05$) reduction in heart rate. But this heart rate reduction never exceeded 6 beats/minute, which occurred in 15° head down position. Similar results were obtained by Van Bredore et al[19]. They suggested that immediate heart rate changes after active change in posture are due to muscle-heart baroreceptor reflex.

No statistical significant changes were observed in mean arterial pressure in various head positions. Upon 15° head elevation, the systolic blood pressure reduced significantly ($p<0.05$). Durward et al and Lee et al in their studies also found a progressive fall in arterial pressure in head up position, but cerebral perfusion pressure was not significantly affected by 15° to 30° head elevation[17,20].

There was a decrease in CVP in all positions but significant ($p<0.05$) decrease was observed in 15° head up, right and left lateral bending of head and left rotation of head. The fall ranged between 0.12 cm to 1.08 cm of saline. No significant changes were observed in oxygen saturation in various positions of head.

CONCLUSIONS

Rotation of head either to the right or left and head down positions increase ICP significantly. Head elevation may not reduce ICP in all the cases. This depends on degree of elevation, intracranial pathology and associated neck position.

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