Considerable concern has been expressed about the effect of respiratory therapy on intracranial pressure (I.C.P.) in the acute stage of head injury. A study was performed to evaluate the effects of respiratory therapy techniques on the level of I.C.P. in neurosurgical patients. Twenty subjects were studied in both the paralysed and non-paralysed states. Their intracranial pressures were monitored during periods of no treatment (the control), during the application of individual respiratory techniques and during a complete respiratory treatment.

Analyses revealed that total treatment time is a crucial factor in the level of I.C.P. Patients with a high resting I.C.P. are more vulnerable to large increases, prolonged manual hyperinflation raises I.C.P. level and suctioning, in particular, causes dramatic increases in I.C.P.

With improved technology and ventilation, the rate of survival of neurosurgical patients has increased in recent years. It should be stressed, however, that it is the quality of survival that is important.

One of the factors affecting the prognosis of the neurosurgical patient is the level of Intracranial Pressure (I.C.P.). Normal intracranial pressure is defined as 0-15 mmHg, and many studies have demonstrated that sustained increases in I.C.P. carry a poor prognosis (Miller and Sullivan 1979, Vapalahti and Troupp 1971, Gibson et al 1975). Miller et al (1977) found that in the patient with diffuse brain injury, elevation of I.C.P. above 10 mmHg contributed significantly to abnormal neurological function.

Factors that can cause an elevated I.C.P. include cerebral oedema, intracranial haemorrhage, cerebro-spinal fluid obstruction, increases in jugular venous pressure, hypoxia, infection and increased cerebral blood volume.

The partial pressures of arterial blood gases, especially partial pressure of arterial carbon dioxide (P$_{\text{aCO}_2}$), also directly affect the level of I.C.P. by causing immediate and often sustained increases in cerebral blood volume (Beks 1978, Miller and Sullivan 1979). Small increases in P$_{\text{aCO}_2}$ may cause large increases in I.C.P. due to the dilatory effects of P$_{\text{aCO}_2}$ on cerebral arteries (Frost 1979). If the partial pressure of arterial oxygen (P$_{\text{aO}_2}$) decreases, lactic acid in the tissues of the brain increases, causing acidosis and a subsequent increase in cerebral blood flow (Shapiro 1975). If hypercarbia exists, i.e. a P$_{\text{aCO}_2$ of greater than 45 mmHg, a hypoxia of 70 mmHg can magnify the response of an increase in P$_{\text{aCO}_2$.

Not only do abnormal arterial blood gases adversely affect I.C.P., but an increased I.C.P. may cause a ventilation-perfusion imbalance (Jennett and Hoff 1978). Deleterious arterial blood gases in the acute neurosurgical patient can delay neurological recovery, extend cerebro-vascular lesions and contribute to the formation of cerebral oedema. Adequate therapy for the pulmonary system is therefore vitally important in managing patients with intracranial abnormalities.

Many authors (Gibson, Turner and Nobles 1975, Moss and McDowall 1979, March, Marshall and Shapiro 1977, Baigelman and O'Brien 1981, Lofgren 1976, Trubuhovich 1979) claim that respiratory physiotherapy results in an increase in I.C.P. This increase is attributed to such factors as systemic hypertension, increase in intrathoracic pressure, impedance of cerebro-spinal fluid flow from the cranial cavity, hypercarbia, hypoxia, or stimulation of the cough reflex.

Unfortunately, reports claiming that respiratory therapy increases I.C.P. have described studies which do not appear to be satisfactory experimentally in terms of a consistent measurement, presence of a control measure.
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measurement, or a statistically relevant number of subjects.

Because of these views, many neurological teams do not support the use of respiratory therapy in the acute stage. However, lack of respiratory therapy can lead to secondary pulmonary complications, causing an increase in I.C.P.

Due to the uncertainty surrounding the use of respiratory therapy for neurosurgical patients, a clinical study was instituted at the Department of Physiotherapy, University of Queensland, to investigate the individual and collective effects of percussion, manual hyperinflation, expiratory vibration and endotracheal suction on the level of I.C.P. in neurosurgical patients.

Method

Subjects

Twenty ventilated neurosurgical patients were included in the study. Their ages ranged from twelve to sixty years, with 15 of the 20 patients, between 20 to 25 years. The conditions comprised diffuse brain injury (thirteen patients), subdural or extradural haematoma (three patients) and intracranial haemorrhage (four patients). Patients were excluded from this study if they were experiencing incipient territorial herniation, fulfilled the criteria for brain stem death, diagnosed as having senile or pre-senile dementia, receiving positive end expiratory pressure or had pre-existing respiratory disease.

Measurement of I.C.P.

Measurement of I.C.P. was by a 'Richmonds' screw and involved the threading of a small hollow bolt into the skull via a burr hole so that the tip was below the open dura (Vries, Becker and Young 1973).

This was connected through a short length of tubing to a pressure sensitive diaphragm (maintained at the level of the Richmonds screw) attached to the patient's bed. Movement of the diaphragm resulting from a change in I.C.P., was converted to electrical energy and this figure was displayed digitally. The equipment was calibrated daily.

Procedure

Neurosurgical patients are often paralysed and sedated for the first 48 to 72 hours, after which the paralysing agents are withdrawn and the patient's conscious state assessed. To determine whether the state of sedation and paralysis modifies the influence of respiratory therapy on I.C.P., the techniques were administered during the paralysed state and again when these agents were withdrawn. In each of these cases, recordings were made while the patient received no stimulation whatsoever, this measurement acting as the control. I.C.P. was then monitored during either a full respiratory treatment or a series of four individual respiratory techniques. Each patient was ventilated by a volume cycled ventilator and maintained in a state of hyperventilation, i.e. a PaCO₂ between 28-32 mmHg during the paralysed state.

In the absence of evidence relating to optimal treatment times, the duration of treatment followed in the Intensive Care Unit of Royal Brisbane Hospital was adopted for this study. Percussion of the chest wall was performed for five minutes, followed by disconnection of the patient from the respirator and connection to a manual hyperinflation bag. The patient's lungs were then hyperinflated with 100% oxygen at a flow rate of eight litres per minute, via a two litre rebreathing bag while vibrations were applied to the chest wall to coincide with the expiratory phase of manual hyperinflation. Following six breaths with the manual hyperinflation bag, the patient's endotracheal tube was suctioned. This procedure was repeated twice and the patient then reconnected to the respirator.

Each respiratory technique, that is, percussion, vibration, manual hyperinflation and suction were also performed separately to ensure that no one technique influenced the response to another. To provide a form of control in case any changes occurred in the subject over the time of application, the order of presentation of techniques was systematically rotated for consecutive patients. Unfortunately the force of percussion, vibration and manual hyperinflation could not be standardized. However, the same physiotherapist applied percussion and expiratory vibrations during the full treatment session, and each of the four techniques when applied individually.

Any disturbance or stimulation prior to treatment can result in abnormally high values of I.C.P. For this reason, patients were not disturbed or handled fifteen minutes prior to any measurement being taken. Pre-trial testing had revealed that fifteen minutes provided sufficient time for I.C.P. to return to baseline level after any disturbance.

During this study patients were positioned in right or left side lying, with the bed horizontal and no head up or head down tilt, as this was the position desired by the medical staff in charge of the Unit. Because it has been shown that rotating or flexing a neurosurgical patient's head may result in a considerable increase in I.C.P. (Shalit and Umansky 1977, Shapiro 1975), the subject's head was maintained strictly in a neutral position prior to and during treatment.

When subjects were receiving paralysing agents, treatment commenced uniformly ten minutes after intravenous administration. The amount of sedation and paralysing agent administered was dependent on the weight, age and medical condition of the patient, and was adjusted until the patient exhibited no voluntary movement or reaction to pain. If at any stage during treatment the patient showed signs of instability, (for example, tachycardia or I.C.P. rising above 30 mmHg), the treatment or technique in use was ceased immediately and the patient reconnected to the ventilator.

When collecting data for both the control period and the full respiratory treatment, I.C.P. was recorded at the
end of each thirty seconds for a total period of seventeen minutes. Time of administration for each of the individual techniques varied slightly. Percussion, vibration and manual hyperinflation were continued for seven and a half minutes. However, as suctioning is a relatively dangerous technique, it was repeated only twice over a period of two and a half minutes.

Results

The data collected in this study allowed not only the determination of any changes in I.C.P. with respiratory therapy, but also the identification of the technique responsible and the time of such influence. To achieve these objectives, a Factorial Analysis of Variance (FANOVA) was applied to the main factors in the study, that is 'technique' and 'time'. For each category of time period, computational analysis provided the means of twenty scores. As a result of this analysis, a difference in the effect of treatment on paralysed and non-paralysed subjects was revealed. In non-paralysed patients, the continuous application of a full respiratory treatment resulted in a statistically significant effect on I.C.P. (p < .01). As Figure 1a illustrates, its level rose above that of the control period once manual hyperinflation and vibration were introduced, and continued to rise as further procedures were administered (see Table 1). After thirteen minutes of the treatment regime, the I.C.P. increased to a potentially dangerous level. For paralysed patients, the overall measure of I.C.P. monitored throughout the full treatment did not increase by a statistically significant amount. Nevertheless, time proved to be an important factor for these patients also and examination of Figure 1b demonstrated that by the end of the full application of treatment, the I.C.P. of these patients had reached a level similar to the end point for non-paralysed subjects (Table 2).

On examination of the raw scores of individual subjects, it was revealed that

![Graph](image)

**Figure 1:** Comparison of intracranial pressure during periods of respiratory therapy and no treatment

<table>
<thead>
<tr>
<th>Time (Minutes)</th>
<th>No Treatment Mean</th>
<th>S.D.</th>
<th>Treatment Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5 (P)</td>
<td>13.45 (4.54)</td>
<td>13.5 (5.66)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.5 (P)</td>
<td>12.8 (5.21)</td>
<td>13.75 (4.98)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 (P)</td>
<td>13.0 (5.66)</td>
<td>12.2 (4.63)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.5 (M.H. + V.)</td>
<td>14.3 (5.27)</td>
<td>15.5 (6.24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 (M.H. + V.)</td>
<td>14.3 (5.67)</td>
<td>16.45 (6.06)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.5 (S)</td>
<td>13.0 (5.33)</td>
<td>15.0 (6.27)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 (M.H. + V.)</td>
<td>13.85 (5.18)</td>
<td>18.5 (7.20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17 (S)</td>
<td>13.85 (5.47)</td>
<td>19.0 (7.22)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

S.D. = Standard Deviation  
P = Percussion  
M.H. + V. = Manual Hyperinflation and Vibration  
S = Suction

Respiratory Therapy and I.C.P. in Ventilated Neurosurgical Patients

Table 2:
The mean intracranial pressure of twenty paralysed patients during no treatment and during respiratory physiotherapy during specified time periods

<table>
<thead>
<tr>
<th>Time (Minutes)</th>
<th>No Treatment</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>0.5 (P)</td>
<td>13.20 (5.94)</td>
<td>13.0 (5.66)</td>
</tr>
<tr>
<td>2.5 (P)</td>
<td>13.50 (6.02)</td>
<td>10.90 (4.90)</td>
</tr>
<tr>
<td>5 (P)</td>
<td>13.15 (4.70)</td>
<td>11.10 (4.59)</td>
</tr>
<tr>
<td>7.5 (M.H. + V)</td>
<td>13.80 (5.18)</td>
<td>11.80 (5.09)</td>
</tr>
<tr>
<td>10 (M.H. + V)</td>
<td>13.60 (5.26)</td>
<td>14.20 (6.03)</td>
</tr>
<tr>
<td>12.5 (S)</td>
<td>13.12 (6.58)</td>
<td>14.9 (6.60)</td>
</tr>
<tr>
<td>15 (M.H. + V)</td>
<td>13.60 (5.42)</td>
<td>17.1 (7.48)</td>
</tr>
<tr>
<td>17 (S)</td>
<td>13.4 (5.39)</td>
<td>18.8 (6.46)</td>
</tr>
</tbody>
</table>

S.D. = Standard Deviation
P = Percussion
M.H. + V = Manual Hyperinflation and Vibration
S = Suction

an increase occurred only in subjects whose resting I.C.P. was above 15 mmHg. Eight patients in the paralysed group and eleven in the non-paralysed group had a resting I.C.P. above 15 mmHg. In subjects whose resting I.C.P. was less than this, the effect of treatment tended to follow that of the control period and did not result in an increase after the fourteenth minute. Figure 2 demonstrates this difference in response. This finding could be explained by intracranial volume pressure relationships. Those patients with a lower resting I.C.P. appear to have more compensation available.

An analysis of the effect of individual techniques (using a Factorial Analysis of Variance) revealed that 'treatment' was significant in increasing I.C.P. (p < .01). A 'Tukey' test, which is designed to determine which pairs of sample means are significantly different, was applied and this demonstrated that suction was the technique responsible for increasing I.C.P. in both paralysed and non-paralysed subjects (Wright 1976).

Figure 3 compares the levels of I.C.P. recorded in non-paralysed patients during the application of individual techniques, and the early and potentially detrimental effect of suctioning can be clearly seen on this graph. Other individual effects of respiratory techniques can also be observed. For example, percussion lowered I.C.P., especially after a period of five and a half minutes, while manual hyperinflation increased I.C.P. with time.

The statistical analyses demonstrated that while similar trends occurred for both paralysed and non-paralysed patients, the increases in I.C.P. as a result of manual hyperinflation and suction were more marked when the subject was in the non-paralysed state.

Discussion

These results do indicate certain trends and provide implications for respiratory therapy in neurological patients. The following points should be made:

a) Analyses demonstrated the importance of treatment time on the level of I.C.P. and showed that respiratory therapy applied to neurosurgical patients for the seventeen minute duration considered standard at some hospitals is likely to produce potentially dangerous levels of I.C.P. in some patients. Reducing respiratory therapy treatment to a total of no greater than thirteen minutes and substituting more frequent but shorter treatments for the previous prolonged treatments would seem advisable while still ensuring adequate respiratory function.

b) Since patients with a starting I.C.P. level of 15 mmHg or higher are more vulnerable to increases in I.C.P. with respiratory therapy, it is recommended that physiotherapists monitor these patients carefully and, should I.C.P. approach 18 mmHg, delay res-
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Figure 3: The effect of individual respiratory techniques in non-paralysed patients

Respiratory therapy until I.C.P. falls to a lower level, unless it is felt that poor respiratory status is contributing to the neurological state.

d) Applications of manual hyperinflation should be kept brief (less than 3.5 minutes) in patients vulnerable to increases in I.C.P. as this technique was shown to increase I.C.P. with time. An explanation for this increase may be increased intrathoracic pressure due to a Valsalva manoeuvre. The 100% oxygen in the rebreathing bag should have had a slightly vasoconstrictive effect on cerebral arteries, decreasing cerebral blood volume and so decreasing I.C.P.

d) Suction was shown to be a relatively dangerous procedure causing a dramatic rise in I.C.P. Previous studies have suggested several mechanisms for this, including hypercarbia, hypoxia, systemic activation and stimulation of the trachea (Baigelman and O'Brien 1981, Shapiro 1975, Lofgren 1976, Fisher, Frewin and Swedlow 1982). Donegan and Bedford (1979) demonstrated that intravenous lidocaine administered prior to suctioning can prevent an increase in I.C.P. As suction of the endotracheal tube is a necessary procedure, either lidocaine or additional sedation could be provided prior to suction.

c) Non-paralysed patients should be treated with caution, as the study showed that for these patients, a continuously progressing full treatment did produce a significant increase in I.C.P. It may be even more important in non-paralysed patients than for those who are paralysed to wait until the patient's I.C.P. is below 15 mmHg before commencing treatment.

Patients in this study did not have arterial blood gases measured frequently enough to correlate with I.C.P. measurements. This study would have improved had P_co2 been measured. A further study including measurement of this variable is under way.

Conclusions

By accurately documenting the effects of respiratory care on I.C.P., this study has provided useful information for respiratory treatment of neurosurgical patients. Although this paper makes no attempt to explain changes in I.C.P., knowledge of this important area could be further expanded by collecting such data as arterial blood gases, especially P_co2, mean arterial blood pressure and cerebral perfusion pressure. In the meantime, it is hoped that implementation of the recommendations emanating from this study may help to enhance the management of this group of acutely ill patients.

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