Relationship between Rapid Variations in Intracranial Pressure and Changes in Respiratory Pattern during Postoperative Monitoring

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Summary

Correlation between changes in epidural pressure, respiration and other related phenomena were studied in 7 patients with brain tumor by post-operative monitorings. Mutual changes in intracranial pressure (ICP) and respiratory pattern during rapid ICP variations were the main focus of our study.

Occurrence of rapid ICP variations of A and B-waves was closely related to changes in respiratory pattern. In large plateau-like waves, suppression in respiration usually initiated the pressure rise, while hyperpnea or tachypnea accounted for the pressure decrease. Analysis on B-type pressure oscillation disclosed a clear phase relation that ICP rises during hypopneic phase of periodic respiration and its reduction coincides with the onset of hyperpnea. Noticeably such pressure variations as well as respiratory oscillations appeared dominant during sleep. Thus it is indicated that, under pathologic conditions, occurrence of oscillating respiration is provoked by depression in wakefulness of patients, presumably accompanying changes in the function of respiratory centers, and affects the changes in ICP.

Key words: Epidural pressure, intracranial pressure, rapid ICP variation, respiratory pattern, periodic respiration

Introduction

Continuous recording of ICP in patients with intracranial hypertension often reveals a fluctuating pressure which is in contrast to the fairly flat curve obtained in patients with a normal ICP. Such pressure variations have been considered to be important clinical implications representing the pathophysiologcal basis of paroxysmal symptoms.3,21-22,24) As regard the pathogenesis of the rapid ICP variation, Lundberg and associates16,21) already mentioned that such rise in pressure is probably caused by a dilatation of cerebral vessels and that the associated paroxysmal symptoms may be induced by some disorder within the brain stem. Later, Risberg et al.27) proved an increase in cerebral blood volume during the spontaneous plateau waves by means of an isotope technique. However, the nature of rapid oscillating changes in pressure remains to be discussed.

Clinically it is familiar that deterioration in neurological signs sometimes occurs during the night. Källquist et al.19) and Cooper et al.8,9) noticed frequent incidences of pressure variations during sleep with associated alterations in respiratory function.

The present study was designed to examine the relationship between changes in ICP and related phenomena by means of a polygraphic observation during postoperative monitoring in
patients with brain tumor. Special attention was paid to study the role of changes in respiratory pattern on the rapid ICP variations.

**Materials and Methods**

Seven patients with brain tumor were subjected to the analysis of mutual changes in ICP, respiratory pattern and other related phenomena in the present series. The patients included 3 meningiomas and one case each of glioblastoma multiforme, reticulum cell sarcoma, huge suprasellar extension of pituitary adenoma and cerebral metastasis of lung cancer.

For the continuous recording of postoperative ICP change, epidural pressure (EDP) measurement was applied in all instances using TICP-II semiconductor pressure transducer, developed in the collaborated works of the Toyota Central Institute and our clinic. The EDP sensor was settled usually in the frontal region opposite the craniotomized side through a burr hole on the calvarium. To provide accurate pressure values, caution was paid to position the sensor at a coplanar point to the exposed dural surface. Zero level calibration was made just prior to setting the EDP sensor on the skull and re-examined at the time of taking off the sensor in the patient’s ward. Duration of EDP measurement was 89 hours in average, ranging from 32 to 104 hours.

Respiratory pattern was monitored with the aid of an impedance pneumogram (Nihon-Koden OGR-5100), by which serial changes in relative values of tidal volume were recorded. The coupled electrodes were placed usually at both preaxillary lines of the patient. When the patient was too restless, the electrodes were positioned on the chest wall with a distance of more than 20 cm to lessen artifact by body movement. Systemic arterial pressure was continuously measured through a catheter in the A. dorsalis pedis in some instances. Electroencephalogram was also recorded in the usual fashion intermittently as needed.

To evaluate movement of the patients during monitoring, changes in electromyographic potential were simultaneously recorded on the polygraph. Potentials in body movement (BM), likewise, coupled electrodes were placed on the periorbital region crossing both eyes diagonally. During these monitorings, states of wakefulness and modes of body movement, as well as neurological signs, were watched and checked correspondently through bed-side observation.

**Results**

*Changes in intracranial pressure*

Clinical features and changes in ICP are summarized in Table 1. The EDP values in the postoperative monitorings varied from 40 mmH₂O to 1200 mmH₂O, and the maximal pressure level attained were found within the third postoperative day in the present series, in all of which fairly good outcome was obtained after surgery.

Besides the changes in basal level of EDP, sudden pressure oscillations were observed during the monitoring. These rapid variations in ICP were classified according to Lundberg: large plateau-like waves, occurring usually with the duration of 5 to 20 minutes (A-wave) and rhythmic oscillations with a dominancy of 1/2-2 per minute (B-wave). These ICP variations of A and B-types were observed in 4 of 7 patients, with predominance of B-wave.

The A-type pressure wave was distinctive in sudden pressure rise and plateau forming with markedly increased pulse amplitude. In occasions the pressure elevated more than 100 mmH₂O from its basal level. The B-type pressure oscillation appeared usually with a trend of rhythmic grouping. In occasions, it continued for a long period of more than 30 minutes. Otherwise, it appeared in combination with plateau waves, particularly in the later phase of long-lasting pressure elevation. Occasionally there was a transitional form of pressure variation with the duration from 3 to 5 minutes. In this form the pressure increased relatively gradually and decreased rapidly. The A-type pressure waves appeared at a relatively high pressure level of 440 mmH₂O in average, whereas the rhythmic B-type waves occurred at both considerably high and low pressure levels.

*Rapid pressure variation and respiratory pattern*

Occurrence of ICP variations of both A and B types was related to changes in respiratory
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Operative Diagnosis</th>
<th>Surgical Intervention</th>
<th>Range in EDP (mmH₂O)</th>
<th>Duration of Monitoring Hours</th>
<th>Rapid ICP Variation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. N.T.</td>
<td>37</td>
<td>M</td>
<td>falx meningioma</td>
<td>lt-parietooccipital craniotomy, total removal</td>
<td>60–1260</td>
<td>42</td>
<td>+</td>
</tr>
<tr>
<td>2. S.O.</td>
<td>42</td>
<td>F</td>
<td>falx meningioma</td>
<td>lt-parietooccipital craniotomy, total removal</td>
<td>100–680</td>
<td>114</td>
<td>+</td>
</tr>
<tr>
<td>3. H.K.</td>
<td>50</td>
<td>F</td>
<td>rt-sphenoidal ridge meningioma</td>
<td>rt-frontotemporal craniotomy, subtotal removal</td>
<td>50–350</td>
<td>88</td>
<td>-</td>
</tr>
<tr>
<td>4. C.W.</td>
<td>64</td>
<td>F</td>
<td>glioblastoma multiforme</td>
<td>bifrontal craniotomy, partial removal</td>
<td>40–520</td>
<td>140</td>
<td>-</td>
</tr>
<tr>
<td>5. M.N.</td>
<td>27</td>
<td>M</td>
<td>reticulum cell sarcoma</td>
<td>lt-frontotemporal craniotomy, temporal lobectomy</td>
<td>40–360</td>
<td>134</td>
<td>-</td>
</tr>
<tr>
<td>6. Y.K.</td>
<td>61</td>
<td>F</td>
<td>recurrent pituitary adenoma</td>
<td>rt-frontotemporal craniotomy, partial removal</td>
<td>40–320</td>
<td>75</td>
<td>-</td>
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</tbody>
</table>
patterns. As shown in Fig. 1, the initial rise of ICP in large plateau-like wave fairly corresponded to a suppressed tidal volume. During the plateau of the elevated pressure, respiratory pattern was revealed to be rather hypopneic and uniform. Corresponding to the phase of sudden decrease in ICP, hyperpnea and/or tachypnea were usually accompanied. Fig. 2 shows another plateau-like wave. Relatively regular and hypopneic breathings were maintained during the marked elevation of ICP and in the later phase of fluctuating pressures. These fluctuations in pressure usually corresponded to the small oscillations in tidal volume.

In B-type pressure oscillation, changes in ICP were related more closely to the alterations in respiratory pattern as shown in Figs. 3 and 4. Usually the pressure elevation corresponded to respiratory suppression and the pressure reduction to hyperpnea. When there was a clear

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**Fig. 1** A large plateau-like wave. Suppression in respiration corresponds to pressure rise, while tachypnea or hyperpnea to pressure reduction.

**Fig. 2** A serial recording of long-lasting pressure elevation. Rather small and regular respirations continue during the plateau of high pressure and later phase of fluctuating pressure. Concomitant with changing pattern into large and irregular respirations, pressure levels are decreased rapidly.
periodicity in respiration, forming apneic and hyperpneic cycle, the pressure rose during the apneic phase and the elevated pressure was reduced coincidentally with the onset of hyperpnea. As the patient became awake and began to move, such oscillation in breathing disappeared and pressure returned to the previous level.

Relation to depression in awakefulness
Long-term polygraphic observation refined correlations between EDP change, respiratory pattern and patient’s state. The occurrence of ICP variations as well as changes in respiratory pattern was revealed to be in close relationship to the depressed wakefulness of the patients. As shown in Fig. 5, periodic breathing and long-lasting suppression in breathing occurred during the period of apparently resting potentials in BM and EM, in which the patient lay quiet and looked “asleep”. When the patient opened his eyes and began to move in bed, accompanied by consistent positive potentials of BM and EM, rhythmic changes in respiration as well as in ICP disappeared and the level of ICP usually dropped.

Discussion
As mentioned in the present results, rapid variations in ICP appeared in close relationship to the changes of respiratory pattern. During the pressure changes of large plateau-like waves, hypopnea usually initiated the rapid rise in pressure and hyperpnea accounted for the rapid fall in pressure. B-type pressure oscillations were
more intimately related to the changes in breathing pattern. ICP rose during hypopneic or apneic phase of cyclic respiration and fell concomitantly with the beginning of hyperpnea. These findings seemingly indicate that hypopnea and subsequent respiratory acidosis provoke an impairment of venous return as well as dilatation of cerebral vessels, resulting into the elevation of ICP in pressure waves.

Lundberg and his collaborators, in a great series of observations, had mentioned the close correlation between these pressure variations and changes in breathing pattern. However, they posed a question whether respiratory changes consist of the cause of pressure variations, stating that typical plateau waves also appeared under artificial respiration with constant minute volume and constant alveolar pCO₂. Recently, Malik et al. reported that cerebral blood flow values examined by microspheric technique increased during elevated ICP in spontaneous breathing animals, showing a clear contrast to the consistent decreases in blood flow in artificially ventilated group. They emphasized that blood gas and pH alterations in spontaneous breathing may be a mechanism which serves to maintain cerebral blood flow during the elevated ICP. Although a possibility of any neurogenic mechanism influencing both respiratory control and cerebral vasomotor tone must still be kept in mind, it is conceivable that respiratory disorder plays an important role on the pressure change.

In the present study it was noteworthy that the variations in ICP appeared apparently dominant in the stage of depressed wakefulness of the patients. This is in accordance with the observations of Cooper and Hulme. They assumed from their studies that such increased incidence of pressure wave during sleep is related to the altered metabolic activity of the brain, and that vasodilatation due to CO₂ accumulation probably consist of the primary cause of pressure waves. Presumably changes in respiratory pattern, concomitant with the depression of wakefulness, effect on the pressure elevation and such effects are manifested more markedly under the conditions of persistent intracranial hypertension, in which intracranial volumetric buffering capacity is eliminated.

Since Stevenson et al. described an elevation of ICP during sleep in 1929, occurrence of periodic respiration as well as some pressure change was reported as a related phenomenon to the sleep state. Particularly REM-sleep has been regarded as the stage of frequent occurrence of periodicity in respiration, the phenomenon being discussed in combination to increased cerebral oxygen consumption and cerebral blood flow in REM-stage. Un-
under situations of postoperative monitoring, however, it is practically impossible to distinguish whether the patient is awake, asleep or unconscious. Furthermore, it is almost impossible to determine the sleeping stage of the patients who show marked EEG abnormalities after surgery. During monitoring of this series, polygraphic observation including measurements of body movement and ocular movement was applied for estimation of transition in patient’s wakefulness and proven to be available. This measuring system was much useful for the retrospective analysis of long-term recording data.

As mentioned above, periodic breathing consists of a causal mechanism of rhythmic oscillation in pressure. Hoff and Breckenridge assumed that periodic respiration is induced by a depression of higher centers which normally exert a suppressive influence on the lower respiratory center. According to Mitchell and Berger, the degree of impairment of rhythmic breathing also depends on the extent of the brain lesion. Several investigators had noticed that loss of regularity in respiration may occur, in particular, in the patients with extensive bilateral cerebral lesions. Further, occurrence of periodic respiration has been discussed in relation to the change in sensitivity of respiratory centers to CO₂, which occurs concomitant with depression of wakefulness. Under the pathologic conditions, as suggested by Mitchell et al., respiration may be increased by voluntary effort while the patient is at arousal and decreased if the patient falls into sleep or is impaired in sensorium. This will be a possible explanation for the mechanism of the oscillating changes in respiration as well as in pressure, which appeared apparently dominant in the states of depressed wakefulness in postoperative patients.

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