Variations of intraventricular pressure during pneumoencephalography

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Intraventricular pressure was studied in eight patients during and after diagnostic pneumoencephalography. In cases with normal initial pressure and normal cerebrospinal fluid (CSF) dynamics, variations in pressure were moderate, immediate, and disappeared at the end of the examination. In cases of normal-pressure hydrocephalus, there was a slow but relatively important elevation that continued for at least 24 hours. In cases with intracranial hypertension, there was a rapid significant increase; return to normal depended principally upon the flow from a large CSF compartment.

Key Words · intraventricular pressure · pneumoencephalography · normal pressure hydrocephalus · cortical atrophy

Isotope cisternography has recently allowed a new approach to the problem of cerebrospinal fluid (CSF) dynamics in humans. The subarachnoid infusion test has also provided interesting results concerning CSF absorption.6,7,9,13

We are reporting a study of changes in intraventricular pressure (IVP) during pneumoencephalography conducted as a diagnostic procedure in patients both with and without disturbed CSF dynamics as shown by clinical examination and isotope studies.

Clinical Material and Methods

The IVP was recorded in eight patients in whom pneumoencephalography was being used as a diagnostic procedure. The basic clinical diagnoses were: cortical atrophy (Cases 1 and 2), brain-stem tumor without intracranial hypertension (Cases 3 and 4), normal-pressure hydrocephalus (Cases 5 and 6), high-pressure hydrocephalus with external CSF drainage (Case 7), and intracranial hypertension from a thalamic tumor (Case 8).

The IVP recordings were made by placing a catheter in the frontal horn of the lateral ventricle and connecting it to a strain gauge.* Variations were registered continually during the injections. A mean pressure was obtained in the supine position by recording during several hours before the x-ray procedure, and in the sitting position prior to the air injection. Pressure variations were then continuously recorded during the injections and until a normal pressure was

*Statham P23 DB strain gauge manufactured by Statham Laboratories, Inc., Huerto Rey, Puerto Rico 00919.
Fro. 1. Continuous recording of variations in IVP during PEG in a case of cortical atrophy. Arrows indicate time of air injection. Average change in pressure is indicated by the curve. Time in minutes (abscissa).

re-established; in some instances this required several hours, and occasionally 2 to 3 days. Thus, it was possible to compare differences in pressure at the beginning and end of the pneumoencephalography, variations in the average pressure (considered as the average between maximum and minimum pressure following each injection), and immediate changes resulting from a single injection (Fig. 1).

The usual pneumoencephalography technique was followed. In particular, no drugs having an effect on intracranial or arterial pressures were administered. Every 3 minutes, 5 cc of air were injected, the total amount reaching 30 to 40 cc. In one patient with normal-pressure hydrocephalus, the intraventricular pressure (IVP) reached 60 mm Hg, and the injection was stopped at 25 cc.

Results

Three groups were differentiated in terms of pressure variations.

Group 1: Moderate Variations
In IVP (Cases 1-4)

In these four cases (Table 1) the IVP showed an increase of 5 mm Hg or less at the end of pneumoencephalography (PEG). Two of the patients had atrophy (Cases 1 and 2) and two, brain-stem tumors (Cases 3 and 4).

Variations in the average change in pressure during air injection were less than 35 mm Hg in each of these four cases: 22 mm Hg (Case 1), 30 mm Hg (Case 2), 29 mm Hg (Case 3), 35 mm Hg (Case 4). The IVP increased rapidly during the first four injections (by 20 to 30 mm Hg), fell to between 25 and 20 mm Hg, leveled off during the last injection at around 30 mm Hg, and returned to normal in the minutes following the last injection (Fig. 2).

Group 2: Significant and Persistent Variations in IVP (Cases 5 and 6)

The differences in IVP before and after encephalography were respectively 35 mm Hg (Case 5) and 55 mm Hg (Case 6). This high pressure remained at the same level for

<table>
<thead>
<tr>
<th>Intraventricular Pressure (IVP)</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>before PEG (mm Hg)</td>
<td>7</td>
<td>10</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>after PEG (mm Hg)</td>
<td>12</td>
<td>10</td>
<td>8</td>
<td>8</td>
</tr>
</tbody>
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48 hours in Case 5 and 72 hours in Case 6. The findings in both cases in this group were consistent with those in patients with normal-pressure hydrocephalus. Variations in the average pressure change during air injection were similar in the two cases (Fig.

3). There was a rapid increase in the IVP with the first injection, although less notable than in the first group. During the three or four injections that followed, stabilization or a slight increase in the IVP was noted. At the end of pneumoencephalography, a further important increase occurred before the IVP reached a plateau at a stable and high level.

Group 3: Significant and Transitory Variations of IVP (Cases 7 and 8)

In these two cases the pressure at the end of pneumoencephalography was higher than that at the beginning but returned to normal quite rapidly (in 1 hour in Case 7, in 24 hours in Case 8). During air injection, in both cases an important increase in IVP was noticed at the onset of pneumoencephalography before stabilization at a high level (Fig. 4). After the last injection in Case 7 (with external CSF drainage), the pressure returned to its previous level in 1 hour, while in Case 8 this occurred after 24 hours (Table 2).
TABLE 2
Cases with significant and transient change in IVP at the end of pneumoencephalography (PEG)

<table>
<thead>
<tr>
<th>Intraventricular Pressure (IVP)</th>
<th>Case 7</th>
<th>Case 8</th>
</tr>
</thead>
<tbody>
<tr>
<td>before PEG (mm Hg)</td>
<td>40</td>
<td>30</td>
</tr>
<tr>
<td>after PEG (mm Hg)</td>
<td>45</td>
<td>90</td>
</tr>
<tr>
<td>24 hours later</td>
<td>40</td>
<td>35</td>
</tr>
</tbody>
</table>

Variations in IVP after
A Single Air Injection

Single air injection was studied only in Groups 1 and 2. In Group 1, the difference in pressure ranged between 30 and 50 mm Hg; the return to a normal pressure was more rapid in cortical atrophy (less than 2 minutes) than in brain-stem tumors (2 to 4 minutes). In Group 2, the pressure difference was less (10 to 30 mm Hg), but there was no subsequent return to a normal pressure, and stabilization occurred at a level higher than the original pressure. The transformation of these values to semilogarithmic coordinates is shown in Fig. 5.

Discussion

Theoretical Considerations

The variations in intracranial pressure after the introduction of an isotonic solution into the subarachnoid lumbar space have been known since the study of Foldes and Arrowood. Air injection during pneumoencephalography may be compared to the foregoing in that an excess volume is introduced into the subarachnoid space. However, in several ways it is different. The injections are intermittent and involve relatively large quantities during a short period of time. The gas is only slightly soluble, and its diffusion outside the ventricle is minimal, especially if one considers the duration of the examination.

Variations in IVP during pneumoencephalography have been studied before but in particular cases of patients having intracranial hypertension. Moreover, the significant elevation in ICP probably brought about vasomotor phenomena, which would explain the plateau waves.

There are theoretical limits to the significance of IVP variations in our cases. There is the possibility of vasomotor phenomena. The variations observed in our results (rapid elevation and decline of IVP) are quite different from those described as plateau waves, at least in cases without initial intracranial hypertension. Thus, it seems reasonable to consider that the vasomotor phenomena are insignificant at low levels of ICP (pressure below 35 mm Hg). Likewise, in cases in which the ICP is maintained at a higher level, the long-lasting variations cannot be easily explained by a vasomotor factor. On the other hand, the resorption of CSF forms the main slow compensatory mechanism, for it has a linear increase with intracranial pressure while CSF formation is independent of that of pressure. If the injection is given relatively slowly, the ICP rises gradually until CSF resorption balances the increase in volume, that is, the injected amount plus secretion; this part represents the plateau in the perfusion test. But, for higher rates of infusion, the possibilities of resorption are no longer sufficient, and ICP will increase regularly. Katzman and Hussey reported that the CSF absorption in normal subjects may reach 6 to 8 times the CSF formed, which means that for an infusion rate higher than 3.5 cc/min, CSF reabsorption will always be insufficient.

Distention of subarachnoid space must
also be considered. The injection of a certain amount of air into the expandable CSF compartment produces an immediate rise in IVP, with a consequent increase in the volume of the subarachnoid space. The rapid compensatory phenomena involve a compression of the vascular bed, especially its venous part. The latter, because of its own elasticity, will exert a counterpressure, causing a CSF outflow. It is then possible for the CSF pressure to reach its normal level, while the vascular volume returns to its initial value. These changes in volume, as related to elasticity, must be kept in mind, at least from a theoretical point of view. It seems to us that this factor would not prevent the differentiation between our clinical groups and eventually the diagnostic value of IVP variations.

Clinical Results

In patients without an evident defect in CSF dynamics (cortical atrophy, brain-stem tumors), variations in IVP correspond with theoretical considerations: the initial rise with compression of the vascular space causes CSF outflow and return to normal pressure.

In patients with normal-pressure hydrocephalus (as far as they can be recognized by clinical signs, isotopic studies, and morphological aspects of the ventricle), IVP modifications seem to be well correlated to the potential of CSF absorption. Lorenzo, et al., have identified certain characteristics of the absorption capacity. There is normal absorption at low pressure, but there may be resistance that contributes to raising the pressure, or there may be a high opening pressure that initiates the absorption process and is followed by a normal resistance. There may be a combination of both anomalies.

In our present study, IVP variations showed a moderate initial rise followed by a stationary period before a secondary rise which terminated at the end of the injections. After termination of the air injections the IVP remained high for several hours and even days because of insufficient resorption capacity. This persistence of elevated intracranial pressure is certainly responsible for the continued ventricular dilatation and the clinical deterioration commonly observed in normal-pressure hydrocephalus patients after pneumoencephalography.

To summarize, in patients with normal-pressure hydrocephalus, IVP changes during and after pneumoencephalography show a special pattern comparable to the results obtained by Nelson and Goodman. Normal subjects can be differentiated from patients suspected of having communicating hydrocephalus by the different rates of increase of CSF pressure following different infusion rates. These results have been substantiated by statistical analysis.

In cases with an initial intracranial hypertension, the injection of a supplementary volume into the subarachnoid space produces an important and immediate rise in IVP. This may be explained by two mechanisms. Langfitt emphasized the special relationship between pressure and volume as shown by the ascendant portion of the pressure/volume curve. Cronqvist, et al., rely on vasomotor phenomena (plateau waves) which correspond to cerebral vasodilatation and a rise in cerebral blood volume during the plateau waves, as demonstrated by Risberg, et al.

Intracranial pressure may return to normal after a CSF outflow has brought about the cessation of circulatory disturbance. If this CSF outflow is increased by external drainage, the IVP becomes normal more rapidly. This is true, of course, only in cases where the high pressure is related to ventricular dilatation and not to cerebral edema.

References


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