Brain Swelling and Ventricle Size

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SUMMARY: Cerebral ventricle size was measured in one hundred normal people, age 10 to 50 years. The measurements were obtained from computed tomographic (CT) scan examinations, using the Huckman (1975) method. Normal ventricles were significantly bigger at age 45 than at age 15. These normals were compared to groups of patients with pseudotumor cerebri, water intoxication and cerebral edema, and cerebral hypoxia and edema due to cardio-respiratory arrest.

The relative ventricular size was the same in the normal group and all three disease groups. Measurement of ventricle size from CT scan images is not even a crude index of cerebral edema in these three diseases.

METHODS AND SUBJECTS:

The scanner was an EMI, 5005, 18 second scan with a matrix of 160 x 160. Ventricle size was measured by the method of Huckman (1975), in all cases (Figure 1).

Five groups of patients were examined. The 100 patients with normal CT scans had a variety of diagnoses including concussion, syncope, migraine, intractable headache, single seizure, or personality disorder.

Twelve patients with pseudotumor cerebri (PTC) have been examined here in the past three years and followed long enough to confirm the diagnosis. All had more than one CT scan. Nine patients, under 50 years of age, with acute water intoxication and some disturbance of consciousness had CT head scans. Five patients, under 50 years of age, with acute cardiac/pulmonary arrest producing coma had CT head scans. Two patients with thrombosis of the superior sagittal sinus had CT head scans.

RESULTS:

The range of ventricle sizes, in the normal group, is shown in Table I classified by decades. There is a significant difference in size between the second decade as compared to the fifth decade and older. There is also a significant difference between the third decade and fifth decade and older. The degrees of significance between these differences are listed in the legend of Table I.

The relative ventricle sizes of the 12 patients with PTC are shown in Figure 2. They are not different from the normals.

The nine patients with acute water intoxication and plasma sodium of less than 120 meq/l had CT head scans because of obscurity of diagnosis. The scans were done within two to six hours of the finding of the low sodium. The linear measurements of relative ventricle size were normal and appropriate for age.

The five patients with cardiac/pulmonary arrest were unconscious. Their CT head scans were performed between eight and 34 hours after the ictus. They all had evidence of cerebral edema based on clinical examination, intracranial pressure monitoring, and autopsy findings. The measurements of cerebral ventricles were normal in all cases.

The two patients with superior sagittal sinus thrombosis both had normal sized ventricles. They had several CT scans, during the course of the disease, and after recovery. There was no significant change in ventricle size.

DISCUSSION

The cause of increasing cerebral ventricle size from age 10 to 50 in normal people is not known. The gradient is evident in Table I.

Pseudotumor cerebri is a disease of unknown cause. It has a mixed and conflicting background of information respecting ventricle size. Currently popular textbooks of neurology state the ventricles are "small" (Gilroy & Meyer, 1969), "smaller than normal" (Merritt, 1979), "normal or of
TABLE 1: Size of Normal Cerebral Lateral Ventricles Grouped by Decades

<table>
<thead>
<tr>
<th>Age in Years</th>
<th>Number</th>
<th>Ventricle Size in mm *</th>
<th>Mean **</th>
<th>Range</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-19</td>
<td>19</td>
<td>14.2</td>
<td>11-21</td>
<td>2.21</td>
<td></td>
</tr>
<tr>
<td>20-29</td>
<td>18</td>
<td>14.07</td>
<td>11-17</td>
<td>1.81</td>
<td></td>
</tr>
<tr>
<td>30-39</td>
<td>20</td>
<td>15.60</td>
<td>11-18</td>
<td>2.09</td>
<td></td>
</tr>
<tr>
<td>40-49</td>
<td>26</td>
<td>16.19</td>
<td>13-20</td>
<td>1.89</td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>17</td>
<td>16.54</td>
<td>15-19</td>
<td>1.13</td>
<td></td>
</tr>
<tr>
<td>Total Group</td>
<td>100</td>
<td>15.60</td>
<td>11-21</td>
<td>2.09</td>
<td></td>
</tr>
</tbody>
</table>

* as determined by Huckman (1975) measurements

** Second decade vs fifth and older decades, significant, p<.001
Third decade vs fifth and older decades, significant, p<.01
Fourth decade vs fifth and older decades, significant, p<.05

Figure 1 — Relative ventricle size as determined by Huckman et al (1975). The measurement of lateral ventricle size (as listed in Table I) is the sum of the distance between the most lateral aspects of the frontal horns of the lateral ventricles (open arrows) and the greatest width of the lateral ventricles in the region of the head of the caudate nucleus just anterior to the third ventricle (solid arrows).

Figure 2 — Distribution of relative ventricle size from 100 normal CT scan examination (closed circles and single lines) and the same measurement from 12 patients with pseudo tumor cerebri (open circles and double lines).

Reduced size” (Walton, 1977), or “small or normal size” (Adams & Victor, 1977).

The literature prior to the CT scan reveals mixed opinions about ventricle size, but the majority are reported as normal. Dandy (1937) described twenty-two patients, all of whom had gas ventriculography. He thought their ventricles were small. Foley (1955) reported 60 patients of whom 58 had ventriculography and all were normal. In his literature review he found 46 cases of “otic hydrocephalus”. The ventricles were normal in 13, smaller in 3 and enlarged in 2. In 60 other (non-otic) cases from the literature, 45 had ventriculography. The ventricles were normal in 29, small in 14 and dilated in 2. Unlike Weisberg (1975) and Reid et al (1980, 1981), Foley found that ventricle size did not correspond to the duration of the disease.

Weisberg (1975) found six of 120 patients had small ventricles and three patients with a protracted course or with recurrence of the disease had enlarged ventricles. The other 111 patients had normal ventricles. All sizes were determined by pneumoencephalography. Prior to the CT scan, the consensus of opinion on ventricle size in PTC suggested that most were normal, some were reduced in size.
and an even smaller number were enlarged. Comparative measurements with normal controls were rarely given. Infrequent mention was made of the shortcomings of pneumoencephalography as a method of demonstrating ventricle size by underfilling or overfilling and distortion (Tanaka, 1981; Boddie et al., 1974). Therefore, it seems most reasonable to conclude that by air study the ventricles were normal or enlarged. When enlarged the disease was likely to be chronic or recurrent (Weisberg, 1975) or some other diagnosis would eventually be made (i.e. meningeal carcinomatosis, posterior fossa tumor, midline tumor, meningeal granuloma) (Moffat, 1978).

Since the CT scan has been available, the opinion on ventricle size in this disease has become more uniform. They are normal. Thus, Delaney & Schellinger (1976) reported seven patients, all with normal ventricles; Weisberg et al. (1977), 31 patients all with normal ventricles; Rush (1980), 63 patients of whom 33 had CT scans and all were normal. (Five of these were commented on to have “small” ventricles, but no measurements were given). Vassilouthis & Utley (1979), reported 28 patients all with normal ventricles. Huckman et al. (1976) measured the size of the lateral ventricles in 17 patients with PTC and compared them to 27 controls. There was no significant difference.

In summary, five papers reported 116 patients with PTC in whom ventricular size was determined by linear measurement from CT scans. In all cases the ventricles were of normal size. The current study adds another twelve patients to this list.

In spite of this unanimity of opinion, linear one dimensional measurements of ventricular size are an inaccurate way of estimating ventricular volume. They are better than the vague descriptive terms of moderate, marked, or slight enlargement. Penn et al (1978) have described an interactive computer system to measure ventricle volume which was found to be highly accurate when tested on a series of phantoms. In a group of patients with obstructive hydrocephalus, their measurements correlated poorly with the Evans ratio, but more accurately with the Huckman measurement. Their method may not lend itself to accurately estimating ventricle volume when the ventricles are normal (see Penn 1978, Figure 3). Reid et al (1980) have a similar system of volume measurement. However, it has an error factor of 20% - 30% (Wyper et al., 1979). They estimated ventricular volume in 18 patients with PTC and found the volume significantly less than controls. They were able to re-examine 15 of these patients an average of 26 months later. The differences in ventricle volume from the first to the last examination are so small and there are so few of them that the information is probably not significant.

There is little to be said about the patients with water intoxication or cerebral hypoxia. These patients had CT scans because of obscurity of diagnosis. The type of cerebral edema produced by these conditions does not reveal itself in CT scans by areas of diminished density or reduction of ventricle size.

In intracranial vein and venous sinus obstruction the CT findings can be more complex because of the associated venous infarction which may be present. Kingsley et al. (1978) reported five patients with superior sagittal sinus thrombosis. All had CT scans between three and 21 days of the ictus. The ventricles were thought to be of normal size although no measurements were reported. In one patient with repeated scans, the ventricles were larger on the subsequent examinations and in retrospect the initial size of the ventricles was considered to be abnormally small. Lavin et al. (1978) reported a single case of intracranial venous thrombosis. CT scan revealed massive venous infarctions and hemorrhages in both hemispheres and small ventricles, although no measurements were given. Brant-Zawadzki et al (1982) described a single case of dural sinus thrombosis. The ventricle size was normal and a filling defect at the level of the torcular herophili indicated thrombosis which was confirmed by angiography.

Buonanno et al., 1978 reported eleven patients with cerebral vein or venous sinus occlusions. The CT findings included a host of local manifestations, i.e. hemorrhage, infarction, gyral and tentorial enhancement, as well as edema and the abnormality of the torcular herophili. Five had “small” ventricles, “like PTC”. None were measured.

In summary, the size alone of cerebral ventricles is not informative in the diagnosis of cerebral venous sinus or vein occlusion or cerebral edema associated with hyponatremia or hypoxia.

Acknowledgement
This study was funded by the Mrs. James A. Richardson Foundation. It is a pleasure to thank Gail Landry for the statistical computations, reference checking, art work and manuscript preparation.

References


