Progressive ventricular dilatation following pneumoencephalography

A radiological sign of occult hydrocephalus

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Eight patients with "normal pressure hydrocephalus" are presented who demonstrated radiographic and occasional clinical evidence of progressive dilatation of the ventricles following pneumoencephalography. The characteristic pneumographic signs of tentorial obstruction to the flow of cerebrospinal fluid (CSF) had been documented in the original air contrast study. The authors postulate that pneumoencephalography in patients with normal pressure hydrocephalus may result in a sudden displacement of CSF from the ventricles into the already compromised basal cisterns, leading to further obstruction of CSF outflow and progressive ventricular dilatation. Other mechanisms such as reduction in the potential resorptive capacity of the ventricular ependyma by air replacing ventricular fluid may play a part. The value of repeat radiological studies 24 and 48 hours after the original pneumogram is emphasized both as an aid in the radiological diagnosis of normal pressure hydrocephalus and as an additional parameter for studying problems in CSF flow and absorption.

KEY WORDS pneumoencephalography · cerebrospinal fluid · normal pressure hydrocephalus

Since the initial report in 1965 by Adams and coworkers describing "occult hydrocephalus with normal CSF pressure," various authors have confirmed their original observations and elaborated upon the radiographic criteria necessary for diagnosis. From the radiographic standpoint the contrast studies have revealed the picture of a communicating hydrocephalus with the area of CSF obstruction at, or close to, the tentorial notch. Confirmatory radiodiagnostic criteria have been obtained by delineation of the temporal and spatial pattern of CSF absorption following the introduction of specific isotopes into the lumbar subarachnoid space or cisterna magna. These latter studies have disclosed a characteristic brain scan picture in patients with communicating hydrocephalus, i.e., concentration of the isotope within the ventricular system, often extending over a 48-hour period, with little if
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any evidence of the isotope traversing the usual CSF pathways along the basal cisterns and over the convexities of the cerebral hemispheres.

The present paper is concerned with the demonstration of an additional radiographic criterion which we have found helpful in the diagnosis of communicating normal pressure hydrocephalus. This is a progressive enlargement of the lateral ventricles which takes place over several days after the original pneumoencephalogram has been performed. We have documented this phenomenon in eight cases of normal pressure hydrocephalus over the past several years and have not seen similar disturbances in cases with normal pneumoencephalograms or in patients with enlarged ventricles who displayed the characteristic radiographic stigmata of cerebral atrophy.

Case Reports

Case 1

A 58-year-old woman had a subtotal resection of a cystic craniopharyngioma impinging on the optic chiasm in June, 1961. Vision improved slightly after operation, but in 1968, several months before readmission, it deteriorated. She also complained of headaches, apathy, and fatigue.

Examination. On admission on September 23, 1968, the patient had bilateral optic atrophy, bitemporal hemianopsia, and diminished visual acuity. Angiography and pneumoencephalography on September 25, 1968, revealed a filling defect in the chiasmatic region compatible with either a recurrent craniopharyngioma or adhesive arachnoiditis involving the chiasm and basilar cisterns. The ventricular system showed mild dilatation with scant air in the supratentorial subarachnoid sulci above the tentorium (Fig. 1 upper left and right). For 2 days following pneumography, she became more disoriented and confused, and radiographs showed a dramatic increase in ventricular size with still no air in the supratentorial subarachnoid sulci (Fig. 1 lower left and right and Fig. 2). A lumbar puncture on September 29 disclosed a pressure of 120 mm of H_2O with clear CSF. The patient was placed on large doses of steroid medication.

Operation. On October 3, exploration of the chiasmatic region was carried out via a bifrontal craniotomy. Dense adhesions around the optic nerves and chiasm were found and were divided, allowing free flow of CSF in this region. There was no evidence of recurrent tumor.

Postoperative Course. Recovery was uneventful. The patient was brighter and more alert following surgery, and her headaches were relieved. Repeat ophthalmological examination on July 21, 1970, revealed no significant change in the visual fields.

Case 2

This 64-year-old man suffered a head injury in June, 1969.

Examination. On admission the patient was comatose without evidence of any lateralizing neurological defect. Carotid angiography on June 4, 1969, revealed generalized narrowing of the intracranial vessels on the right side, probably due to contusions and edema with evidence of slight right hippocampal herniation. He was placed on steroids and improved gradually, although he continued to be apathetic and confused. Pneumoencephalography on August 14 revealed generalized enlargement of the ventricular system compatible with communicating hydrocephalus secondary to tentorial obstruction (Fig. 3 upper left and right). A RISA cisternogram also showed tentorial obstruction. Following pneumography the patient was lethargic for 5 days, and skull radiographs repeated 2 days after the encephalogram showed progressive ventricular enlargement (Fig. 3 lower left and right). He was subsequently transferred to a nursing home.

The patient was readmitted on October 10 because of pneumonia, which responded to antibiotics. At this time his neurological deficit was one of dementia, essentially unchanged from that of the previous examination.

He was brought back to the hospital May 5, 1970, for further evaluation. Lumbar puncture disclosed a pressure of 70 mm of H_2O. A pneumoencephalogram on May 11 disclosed persistent ventricular enlargement similar to that in the examination of August 14, 1969. A ventriculogram on May 25 revealed moderate enlargement of the ventricular system with little air in the cerebral
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Fig. 1. Case 1. Upper Left and Upper Right: Anteroposterior and lateral pneumoencephalograms showing mild symmetrical dilatation of the ventricular system. Little air has gone beyond the chiasmatic cistern. Lower Left and Lower Right: Anteroposterior and lateral films 24 hours later showing marked progressive dilatation. There is still no air over the cerebral convexity.

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Fig. 1. Case 1. Upper Left and Upper Right: Anteroposterior and lateral pneumoencephalograms showing mild symmetrical dilatation of the ventricular system. Little air has gone beyond the chiasmatic cistern. Lower Left and Lower Right: Anteroposterior and lateral films 24 hours later showing marked progressive dilatation. There is still no air over the cerebral convexity.

...sulci, and 24-hour films revealed further enlargement in ventricular size.

Operation. A ventriculopleural shunt with a low-pressure Spitz-Holter valve was performed on June 1, 1970. At the time of discharge on June 26 there had been only slight improvement in his mental status.

Case 3

This 28-year-old man entered the hospital on February 5, 1969, because of progressive difficulty in walking for 2 years. He had had meningitis (probably tuberculous) at the age of 16 as well as the onset of grand mal seizures at the age of 19.

Examination. The patient displayed mental retardation and spasticity of both legs with hyperactive reflexes and an unsteady gait. He was not incontinent. Radiographs of the skull and spine as well as a brain scan were negative. A pneumoencephalogram on March 6 revealed a dilated ventricular system, compatible with communicating hydrocephalus (Fig. 4 left). Films obtained 24 hours later demonstrated a further increase in the ventricular volume (Fig. 4 right). Injection of RISA into the lumbar sac on March 26 disclosed no mobilization of the isotope cephalad to the site of instillation in the lumbar subarachnoid space. Cervical...
myelography on April 14 revealed diffuse spinal arachnoiditis and partial block at T-2.

**Operation.** On April 26 a ventriculopleural shunt with a medium-pressure Spitz-Holter valve was inserted in the hope that relief of the hydrocephalus would improve the patient's mental status. Although no significant improvement in cerebral function was noted, a follow-up ventriculogram on May 9 demonstrated smaller ventricles than seen prior to the bypass procedure.

**Case 4**

This 60-year old man was found unconscious in the street on December 24, 1968.

**Examination.** The patient was deeply comatose with bilateral decerebrate responses stertorous respirations, papilledema, hyperactive deep tendon reflexes, and bilateral Babinski signs. Radiographs of the skull revealed a linear fracture in the right parieto-occipital region.

**First Operation.** A right temporoparietal trephination on the day of admission revealed a 2-cm-thick solid acute subdural hematoma which was evacuated.

After surgery the patient responded to noxious stimuli and began to move all extremities spontaneously, especially the left side. On January 1, 1969, his condition deteriorated and coma deepened. The trephine button was removed and xanthochromic subdural fluid collection was aspirated. He again improved to a point where he was able to mumble a few words although he could not obey a command. Several lumbar punctures during this period disclosed opening CSF pressure varying between 70 to 130 mm H₂O. The patient remained in a state of akinetic mutism thereafter. A pneumoencephalogram on March 23, 3 months after injury, demonstrated large ventricles with no air in the subarachnoid space over the cerebral convexities. Repeat radiographs 4 days after the pneumoencephalogram revealed a definite increase in the size of the lateral ventricles. One week after the pneumoencephalogram there was still considerable air within the ventricular system, and the temporal horns appeared especially dilated.

**Second Operation.** A ventriculoatrial shunt with a low-pressure Spitz-Holter valve was inserted on March 30, 1970. Following the operation, the patient appeared slightly brighter, although he remained mute and hypokinetic. He died 3 months later without further change in his neurological status.

**Clinical Material and Technique**

Our series includes eight patients, seven male and one female, ranging in age from 28 to 65 years (Table 1). The etiology of the communicating hydrocephalus was diverse. Two patients (Cases 1 and 3) had basal arachnoiditis and one (Case 7) apparently had a spontaneous intracerebral hematoma. The remaining five patients all had sustained severe head trauma sufficient to require the evacuation of bilateral subdural hematomas in two (Cases 6 and 8), an acute subdural hematoma in one (Case 4), and a large frontal intracerebral hematoma in another (Case 5). One patient (Case 6) had evidence of severe bilateral cerebral contusions but no signs of a specific mass lesion requiring surgical evacuation.

**Radiological Technique**

Pneumoencephalograms were performed on these eight patients 3 weeks to 12 years after the original cerebral insult. A standard fractional-filling technique was used, with 30 to 50 cc of room air added in increments of 5 to 10 cc in the upright position. After adequate ventricular filling, special efforts were made to secure the passage of air into the basal cisterns and over the cerebral convexities. Standard brow-up, brow-down, and

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**Fig. 2. Case 1.** Overlay drawing of the lateral projections of two brow-up films, one taken directly after pneumoencephalography, the other 48 hours later. The coarsely stippled shadow represents the marked increase in ventricular size in the second examination.
FIG. 3. Case 2. **Upper Left and Upper Right:** Anteroposterior and lateral pneumoencephalograms showing generalized moderate enlargement of the ventricular system with no air distal to the tentorial notch. **Lower Left and Lower Right:** Anteroposterior and lateral films 48 hours later showing marked progressive ventricular enlargement.

FIG. 4. Case 3. **Left:** Encephalogram, lateral view, showing ventricular dilatation and no air over the cerebral convexity. **Right:** Encephalogram 24 hours later showing increase in the ventricular volume and still no air over the cerebral convexity.
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**Fig. 5.** *Left:* Schematic representation of the normal situation during pneumoencephalography. A volume of CSF equivalent to the volume of air is displaced out of the ventricles into the basal cisterns and past the tentorial notch. With no obstruction to the extraventricular pathways, there is no significant enlargement of the ventricular system. *Right:* Schematic representation of normal pressure hydrocephalus secondary to tentorial obstruction. The CSF, displaced from the ventricular system by the air, is prevented from passage at the tentorial notch. This results in an acute increase in the volume of the ventricular system (lightly stippled area) which is already dilated.

temporal horn views were obtained, and the patients were returned to their rooms and observed closely for any change in their clinical condition. Repeat brow-up AP and lateral radiographs were usually performed 24, 48, and occasionally 72 hours after the original pneumoencephalogram. At the time of the repeat radiographic studies, special efforts were made to keep the x-ray tube and film distances and the angles of the radiographic projections constant.

**Evaluation**

Comparison of the original and repeat pneumoencephalograms were performed using the radiographs obtained in the brow-up lateral and AP projections. Estimations were made of the comparative size of the frontal horns by measuring the height and breadth of the anterior cranial cavity occupied by the air-filled frontal tips in both AP and lateral projections.

**Results**

All eight patients had the characteristic radiographic picture of communicating hydrocephalus, i.e., an enlarged ventricular system, primarily demonstrated in the frontal and temporal horns of the lateral ventricles, but also involving to a lesser extent the third ventricle, aqueduct, and fourth ventricle. Basal cisterns were usually enlarged, and in six of the eight cases no air could be seen within the sulci over the cerebral convexities up to 72 hours after the original radiographs. In two patients small amounts of air were seen in the region of the Sylvian fissures 72 hours after the pneumoencephalogram had been performed, but, except for one or two shadows indicative of subarachnoid air over the convexities, there was no evidence of dilated cerebral sulci. The frontal and temporal horns were ballooned, with rounded angles especially at the superior and lateral aspects of the frontal tips. Comparison of repeat radiographs (24 hours) with the original pneumograms revealed the frontal and temporal horns to have dilated even more in all eight patients (Table 2). This post-pneumographic progressive ventricular enlargement was minimal in two patients, moderate in three, and marked in the remaining three. Ventricular dilatation continued to progress in several patients where both 24- and 72-hour studies were performed. It was our impression that maximal or nearly maximal ventricular dilatation was observable in the 24-hour post-pneumographic radiographs although further ballooning of the ventricles may have taken place thereafter. Significant amounts of air persisted within
TABLE 1

Summary of observations in patients with post-pneumoencephalographic ventricular dilatation

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Etiology of Hydrocephalus</th>
<th>Clinical State</th>
<th>Time Between Cerebral Insult &amp; PEG</th>
<th>Deterioration Following PEG</th>
<th>Positive RISA Cisternogram</th>
<th>Improvement following Shunt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>58</td>
<td>F</td>
<td>arachnoiditis after excision of cranio-pharyngioma</td>
<td>headaches, hypopituitarism, chiasmal + optic nerve compression</td>
<td>8 yrs</td>
<td>moderate</td>
<td>not done</td>
<td>no shunt</td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>M</td>
<td>trauma, diffuse cerebral contusion</td>
<td>hypokinetic dementia, ataxia</td>
<td>3 mos 11 mos</td>
<td>moderate moderate</td>
<td>positive</td>
<td>not done</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>M</td>
<td>meningitis with diffuse arachnoiditis</td>
<td>seizures, spastic paraparesis lowered mental acuity</td>
<td>12 yrs</td>
<td>mild</td>
<td>no mobilization above lumbar subarach., space</td>
<td>mild</td>
</tr>
<tr>
<td>4</td>
<td>60</td>
<td>M</td>
<td>trauma, left subdural hematoma</td>
<td>akinetic, mute</td>
<td>3 mos</td>
<td>mild</td>
<td>not done</td>
<td>mild</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>M</td>
<td>trauma, left frontal hematoma</td>
<td>apathetic mute, rt. hemiparesis</td>
<td>10 wks</td>
<td>mild</td>
<td>not done</td>
<td>marked</td>
</tr>
<tr>
<td>6</td>
<td>52</td>
<td>M</td>
<td>trauma, bilateral subdural hematomas</td>
<td>confusion, lethargy, ataxia, incontinent</td>
<td>14 wks</td>
<td>mild</td>
<td>positive</td>
<td>mild</td>
</tr>
<tr>
<td>7</td>
<td>48</td>
<td>M</td>
<td>intracerebral hematoma, left temporal</td>
<td>mute, lethargic, incontinent</td>
<td>5 wks 4 mos</td>
<td>mild mild</td>
<td>positive</td>
<td>moderate</td>
</tr>
<tr>
<td>8</td>
<td>50</td>
<td>M</td>
<td>trauma, bilateral subdural hematomas</td>
<td>confused, disoriented, lethargic</td>
<td>3 wks</td>
<td>mild</td>
<td>positive</td>
<td>mild</td>
</tr>
</tbody>
</table>

the dilated frontal and temporal horns for as long as 2 weeks after the original pneumo- graphic study. Although the intraventricular air shadow remained, it appeared to occupy progressively decreasing portions of the ventricular system, suggesting that while intraventricular air tended to be resorbed rather slowly, it was indeed being resorbed and probably was not responsible for expansion of the ventricular system (see Discussion). There was no tendency for this intraventricular air to eventually find its way into the subarachnoid sulci.

In addition to the obvious enlargement of the frontal horns, it was apparent that the anterior tips of the temporal horns also demonstrated progressive dilatation and ballooning in the post-pneumoencephalographic radiographs. These temporal tips became large and sausage-like, often giving the impression of a lobulated porencephalic cyst. This tendency for temporal horns to be dilated in patients with communicating hydrocephalus in comparison with the relatively normal-sized temporal tips in patients with cerebral atrophy ex vacuo has been commented upon by Sjaastad, et al., and was confirmed in the present study. Since our post-pneumoencephalographic radiographs were taken primarily in the brow-up projections, we have little data relative to the degree of dilatation of the rest of the ventricular system.

Discussion

It is quite conceivable that the clinical deterioration that follows pneumoencephalog-
TABLE 2

Radiographic changes

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Time Interval</th>
<th>Size of Frontal Horn*</th>
<th>Size of Temporal Horn*</th>
<th>Air Over Convexity</th>
<th>RISA Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24 hrs</td>
<td>++++</td>
<td>+</td>
<td>none</td>
<td>not done</td>
</tr>
<tr>
<td>2</td>
<td>24 &amp; 48 hrs</td>
<td>++++</td>
<td>+</td>
<td>little</td>
<td>+ (delayed activity over convexity after 48 hrs)</td>
</tr>
<tr>
<td>3</td>
<td>24 hrs</td>
<td>+++</td>
<td>no change</td>
<td>little</td>
<td>no mobilization from lumbar region</td>
</tr>
<tr>
<td>4</td>
<td>72 hrs 1 wk</td>
<td>+</td>
<td>+</td>
<td>none</td>
<td>not done</td>
</tr>
<tr>
<td>5</td>
<td>48 hrs</td>
<td>+++</td>
<td>+</td>
<td>none</td>
<td>+ (little uptake on surface even after 72 hrs)</td>
</tr>
<tr>
<td>6</td>
<td>1st–2nd PEG 48 hrs</td>
<td>++++</td>
<td>++</td>
<td>none</td>
<td>+ (no uptake over convexity after 48 hrs)</td>
</tr>
<tr>
<td>7</td>
<td>1st–2nd PEG 24 hrs</td>
<td>++++</td>
<td>+</td>
<td>none</td>
<td>+ (delayed activity over convexity)</td>
</tr>
<tr>
<td>8</td>
<td>24 hrs</td>
<td>+</td>
<td>not filled</td>
<td>none</td>
<td>+ (no uptake in subarachnoid space after 48 hrs)</td>
</tr>
</tbody>
</table>

* += 0 to 4 mm; ++ = 5 to 9 mm; +++ = 10 to 14 mm; ++++ = 15 to 19 mm.

raphy in certain patients having hydrostatic hydrocephalus is in some way linked to the phenomenon of progressive ventricular dilatation as demonstrated by post-pneumoencephalography radiographs. Six of the eight patients in the present series showed some degree of increasing neurological deficit beginning soon after the pneumoencephalogram and persisting for 2 to 13 days thereafter. This deficit was usually manifested by increasing confusion and apathy as well as failure to initiate speech or perform coordinated movements spontaneously. Only two patients did not display specific indications of clinical deterioration concomitant with progressive ventricular dilatation; in one of these (Case 4) the neurological deficit before the air study was so profound as to mask any further subtle signs of increasing deterioration.

Several authors16,21,58,29 have observed and discussed the phenomenon of increasing ventricular size following pneumoencephalography. The correlation between the patient's disease and the presence of post-encephalographic ventricular dilatation was not firmly established by these authors although there seemed to be a definite preponderance of positive responses in post-traumatic cases and in patients with "cerebral atrophy."

It is possible that in earlier reports there were patients classified as having "cerebral atrophy" whose radiographic findings if reviewed under present criteria would be identified as having normal pressure hydrocephalus.21 This should not be surprising. Although Foltz and Ward,11 Kibler, et al.,17 and Shulman and coauthors10 had described the phenomenon of external hydrocephalus following subarachnoid hemorrhage as early as 1956, there was an almost universal tendency, before the report of Adams, et al.,1 in 1965, to classify all patients with open, albeit dilated, ventricular systems with normal CSF pressure as having "cerebral atrophy."

It is conceivable that the progressive ventricular enlargement and neurological deterioration seen after pneumography may be related to the increased stress that ensues when an already compromised CSF outflow system is suddenly taxed by the exigencies of the air injection. When air is introduced into the ventricular system from below, CSF is displaced out of the ventricles and forced distally into the basal cisterns toward the
major areas of CSF absorption over the cerebral convexities (Fig. 5). This sudden efflux of CSF can probably be accommodated in the normal patient without significant change in ventricular size. In the patient with normal-pressure hydrocephalus, whose CSF outflow systems are already badly compromised, the added increment of CSF that presents itself over a short period of time at the area of obstruction may so overwhelm the conducting and absorptive pathways that they rapidly decompensate. Acute dilatation of the ventricular system proximal to the site of blockage results. Recent animal experiments with outflow occlusion by Milhorat, et al., have demonstrated that acute ventricular dilatation may take place as early as 1 to 3 hours following total ventricular obstruction.

An alternative hypothesis is that the acute ventricular dilatation may simply occur as a consequence of a significant volume of air within the ventricular system and occupying space usually filled with ventricular fluid. There are data suggesting that in patients with "stable" or "arrested" external hydrocephalus a degree of compensation is reached through ventricular dilatation and transependymal resorption of CSF. It is reasonable to suspect that in such patients if enough air is added to the ventricular system the gas may abut upon ependymal areas currently being used for fluid resorption. This in turn might result in a diminution of ventricular fluid resorption, and as a consequence the ventricles might enlarge to provide additional ependymal surfaces to compensate for transfer of the remaining ventricular fluid across its walls.

References

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