A Radioisotopic Test for Communicating Hydrocephalus

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Recently, there has been a rekindling of interest in hydrocephalus, due largely to increased awareness of this condition in the adult. However, in adults, for two reasons, it is often very difficult to diagnose symptomatic hydrocephalus. First, hydrocephalus in adults is usually secondary to or associated with other intracranial lesions such as tumors, meningitis, or subarachnoid hemorrhage, and, in these cases, it is difficult to differentiate the symptoms and signs of the hydrocephalus from those due to the primary lesion. Second, available tests, including measurement of the cerebrospinal fluid (CSF) pressure, dye studies, and radiography of the ventricular system and subarachnoid spaces using air or radioopaque material, often fail to establish whether the hydrocephalus is due to obstruction or atrophy. Measurement of CSF pressure may not answer this question for, as Adams, et al.,1 have pointed out, the CSF pressure may be normal in the presence of symptomatic hydrocephalus.

Radiographic studies may also be inconclusive. Hydrocephalus due to a complete block within the ventricular system is readily diagnosed by air or positive contrast studies, but when the block is incomplete and the ventricular system is only slightly larger than normal, the diagnosis may be difficult. With communicating hydrocephalus, the diagnosis is reasonably certain if the air study shows a block in the subarachnoid space either in the basal cisternae or over the cerebral convexities with failure of air to enter the sulci. However, if the cisternae and sulci are incompletely blocked and a few of them do contain air, then diagnosis is difficult. Thus, in many cases, it may be impossible to establish whether symptomatic hydrocephalus is present. Further methods for studying CSF dynamics are clearly needed.

Although several methods have been devised over the past 50 years, none has gained widespread use. In 1964, Di Chiro6–8 reported a radioisotopic scanning technique for studying CSF dynamics after the intrathecal administration of radioiodinated human serum albumin (131I-HSA). Our initial experience using this method has been reported.15 This paper describes its application to the diagnosis of communicating hydrocephalus.

Method

The tracer used was human serum albumin labelled with 131I (with a specific activity of approximately 5 μc/mg).* It was diluted with Elliott’s “B” solution and injected into the CSF by lumbar puncture with the patient in the lateral decubitus position, or by suboccipital puncture into the cisterna magna with the patient sitting. For lumbar injection, the dose was 100 μc 131I-HSA in 10 ml, and for cisternal injection 100 μc in 5 ml. After injection, the patient’s activity and posture were not restricted. Scintillation scanning was performed using a Picker Magnascanner with a 3-inch sodium iodide crystal. Anterior and lateral scans were obtained 2 hours after lumbar injection, and almost immediately after cisternal injection. Repeat scans were performed at varying intervals up to 48 hours after injection.

Because of the diminishing radioactivity in the later scans due to physical decay of the isotope, dispersion of the tracer in the subarachnoid space, and absorption of tracer, the sensitivity of the scanner was increased for scans performed at later time intervals. Therefore, the amount of radioactivity in the scans is not strictly comparable, and this factor must be considered when comparing the amount of radioactivity in the individual scans of a series. The radioactivity of serial blood samples was measured in a well-type scintillation counter in order to study the rate of transfer of tracer from CSF to blood.

* Obtained from Charles E. Frostt & Co., Montreal.
Results

Normal CSF Scans. Diagrams of the normal intracranial subarachnoid space, and the corresponding scans in which the various cisternae can be identified, are shown in Figs. 1 and 2.

When $^{131}$IHSA was injected into the subarachnoid space, a consistent pattern of flow and distribution of tracer was observed. Following injection into the cisterna magna (Fig. 3), most of the tracer flowed around the medulla to the cisterna medullaris and up into the cisterna pontis. Some flowed downward through the foramen magnum into the spinal subarachnoid space. From the cisterna pontis, there was diffusion laterally into the lateral recesses of the cisterna pontis, but the main flow was directed anteriorly and superiorly into the interpeduncular cistern. To reach above the tentorium the tracer took two different routes: posteriorly around the midbrain via the paired cisterna ambiens to reach subsequently the quadrigeminal cistern, or anteriorly into the suprasellar cistern. Usually there was good visualization of all the basal cisternae on the first scans, which were com-

Fig. 1. Diagram of a lateral view of the normal intracranial subarachnoid space (left) and a normal lateral scan performed 45 minutes after the cisternal injection of $^{131}$IHSA (right). The letters are interpreted as follows: A = cisterna magna, B = cisterna pontis, C = cisterna interpeduncularis, D = suprasellar cistern, E = cisterna ambiens, F = quadrigeminal cistern, G = callosal cistern, H = cisterna lamina terminalis. The callosal cistern did not contain radioactivity in this scan.

Fig. 2. Diagram of an anterior view of the normal intracranial subarachnoid space (left) and a normal anterior scan performed 6 hours after the lumbar injection of $^{131}$IHSA (right). The large accumulation of radioactivity in the midline is due to superimposition in this view of many of the basal cisternae. The letters correspond to those in Fig. 1; I = Sylvian cistern, which is well demonstrated at this time.
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Completed within 1 hour of injection. From the quadrigeminal cistern the tracer flowed upwards into the callosal cistern, and from the suprasellar cistern the tracer flowed laterally into the Sylvian cisternae or forward into the cisterna lamina terminalis and then into the callosal cistern. These latter cisternae were usually well demonstrated on the second scans, which were completed within 4 hours of injection. After flowing through the basal cisternae, the tracer spread along the sulci and gyri of the cerebral hemispheres, forward under the frontal lobes, laterally over the temporal and parietal lobes, and superiorly along the medial longitudinal or interhemispheric fissure. Many of these routes lead ultimately to the parasagittal area. Twenty-four hours after injection much radioactivity was present over the convexities of the cerebral hemispheres, particularly in the parasagittal area and in the interhemispheric fissure. By 48 hours, most of the radioactivity had disappeared, except from the region of the cisterna magna which usually remained active for several days probably due to leakage of some of the tracer out of the subarachnoid space through the injection site.

When the tracer was injected into the lumbar subarachnoid space, about 2 hours were required for adequate visualization of the basal cisternae. The cisterna magna did not usually fill from this route because the tracer flowed anterior to the upper cervical cord and medulla. At 6 hours radioactivity was seen in the Sylvian cisternae and along the interhemispheric fissure, and by 24 hours there was some accumulation along the sagittal sinus. After 48 hours most of the radioactivity had disappeared. Usually much less activity was detected in the head after lumbar injection as compared with cisternal injection. In normal cases, little or no radioactivity was seen in the ventricular system.

**CSF Scans in Communicating Hydrocephalus.** Communicating hydrocephalus may be defined as a condition in which there is an obstruction to the flow of CSF in the subarachnoid space beyond the outlet foramina of the fourth ventricle, resulting in an excessive accumulation of CSF in the ventricular system, and perhaps also in that portion of the subarachnoid space proximal to the obstruction. Our nine cases have been grouped according to the site of the block in the subarachnoid space as demonstrated by the scans. In Case 1 the block was in the pontine cistern, and in Cases 2–5 at the level of the tentorial incisura, or just beyond, in the suprasellar cistern. In Cases 6–9 most of the tracer entered the ventricular system, masking the region of the basal cisterns and making it difficult to identify the exact site of the subarachnoid block.

**Case Reports**

**Case 1.** In 1958, a 36-year-old engineer complained of memory impairment, urinary incontinence, and unsteadiness of gait, which

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Fig. 3. Serial scans after cisternal injection in a normal case. Anterior and lateral views were performed within a few minutes of injection, at 3 hours, and 26 hours. The "x" markers on the anterior scans mark the external auditory meati and the vertex, and on the lateral scans mark the nasion, external auditory meatus, and inion.
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had developed gradually during the preceding year. He was found to have a poor memory, mild spastic quadripareis, and bilateral Babinski reflexes. He had sustained a head injury with brief unconsciousness followed by complete recovery 18 months previously. A pneumoencephalogram showed non-filling of the ventricles, and a ventriculogram showed grossly dilated lateral and third ventricles. No air could be manipulated into the aqueduct or fourth ventricle. Indigo carmine placed in the lateral ventricle was not recovered at lumbar puncture. A diagnosis of aqueduct stenosis was made and a ventriculocisternal shunt inserted. Preoperatively, the patient was febrile, and postoperatively a gram negative bacillus (alkaligenes metalkaligenes) was cultured from the CSF. The meningitis which ensued was very severe but responded to chloramphenicol and sulphonamides.

The patient was improved slightly at discharge, but was re-investigated a few months later because of persisting symptoms. Ventriculography showed that the ventricles had not reduced in size. As the Torkildsen tube was found to be patent, a lumbo-peritoneal shunt was performed. Thereafter the patient’s gait and memory slowly improved, and he returned to work in 1963.

In 1966 there was a return of symptoms which slowly progressed until readmission in May, 1967. The patient showed moderate intellectual deficit, ataxia, increased tone in the limbs, and equivocal plantar responses. The CSF pressure was 175 mm H₂O at lumbar puncture, and the CSF protein was 39 mg/%. After ¹³¹IHSAn was injected by lumbar puncture, there was very slow flow of the tracer up the spinal subarachnoid space. Scans performed 4 and 24 hours after injection showed no flow of the tracer beyond the pontine cistern (Fig. 4). The cisterna magna was well outlined. The intracranial subarachnoid block seen on the scans indicates that this man had communicating hydrocephalus. It is significant that the tracer failed to enter the ventricular system as it often does in communicating hydrocephalus. This lack of ventricular filling was probably due to the persisting aqueduct stenosis. His failure to improve after the Torkildsen shunt and his improvement after the lumbo-peritoneal shunt suggest that communicating hydrocephalus was present after the episode of meningitis. His recent deterioration was due to blockage of the lumbo-peritoneal shunt. A ventriculocisternal shunt was performed in May, 1967, with almost immediate improvement in psychomotor capacity and in gait.

Case 2. A 48-year-old man had had untreated hydrocephalus since infancy. He was mentally retarded, disoriented, and had severe memory impairment. He was obese and of short stature, with a head circumference of 27 inches. His limbs were spastic with bilateral Babinski reflexes.

An intraventricular injection of ¹³¹IHSAn revealed enormous ventricles (Fig. 5). Repeat scans up to 48 hours after injection showed no detectable migration of the tracer from the ventricular system and, therefore, his diagnosis was considered to be noncommunicating hydrocephalus. However, a cisternal injection of ¹³¹IHSAn showed obstruction to the flow of the tracer at the level of the tentorium (Fig. 6), and an air ventriculogram showed some air in the suprasellar cistern, indicating that a communication between the ventricular system and subarachnoid space must have existed. It is not known why ¹³¹IHSAn injected into the ventricles was not detected in the subarachnoid space. It may be that the flow was so slow that any ¹³¹IHSAn which did leave the ventricular system was too small in amount to be detected on the scans. Therefore, it is likely that the patient had communicating hydrocephalus with a block of unknown nature at the tentorium.

Case 3. This 42-year-old woman had a
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Fig. 5. Case 2. Scans after injection into the right lateral ventricle. There was no detectable migration of the tracer from the ventricular system.

Mild head injury at the age of 7 but remained well until age 14, when she gradually developed failing vision, ataxia, and headaches. At that time a cerebellar exploration performed by Sir Geoffrey Jefferson was negative. A diagnosis of congenital stenosis of the aqueduct was made. Recovery was slow, requiring about 3 years before the patient could walk normally. She married and had two children.

In 1962, diplopia and ataxia developed. The next year, she was found to have early papilledema. A pneumoencephalogram was performed but no air entered the ventricles. There was air in the basal cisterns, but none in the sulci. A ventriculogram showed enlarged ventricles and aqueduct stenosis. A ventriculocisternal shunt was inserted, and this resulted in marked improvement in gait and vision. Because of return of symptoms in 1964, the cisternal end of the shunt was revised. Postoperatively the patient developed meningitis. A smear of the CSF showed gram-positive cocci, but CSF cultures were negative. She was treated with antibiotics, improved, and then remained well until January, 1967, when progressive ataxia and diplopia developed.

On admission in June, 1967, the CSF pressure was 170 mm H₂O, and the CSF contained 26 mg.% protein, no white blood cells, and 6 red blood cells per cu mm. There was residual positive-contrast material in the ventricular system from the 1963 injection, and this again revealed the aqueduct block. Scans after lumbar injection of ¹³¹I-HSA revealed obstruction to the flow of tracer at about the level

Fig. 6. Case 2. Scans after cisternal injection show a block at the level of the tentorial incisura.
of the suprasellar cistern (Fig. 7). No radioactivity was seen in the cisterna ambiens, quadrigeminal or Sylvian cisternae. However, at the later time intervals, a small amount of activity was seen over the cerebral convexities indicating that obstruction in the basal cisternae was incomplete. The subarachnoid obstruction was considered secondary to the previous meningitis. Failure of the tracer to enter the ventricular system was probably due to the aqueduct stenosis. A ventriculoatrial shunt was performed, with almost immediate relief of ataxia and diplopia.

Case 4. This 62-year-old woman was admitted on November 25, 1966, complaining of progressive deterioration of memory, instability of gait, and urinary incontinence during the past year. For 6 months she had been having almost daily headaches. A few days before admission she had a transient episode of unconsciousness. Examination revealed mental confusion, papilledema, limitation of upward gaze, spasticity of arms and legs, and bilateral Babinski reflexes. Lumbar puncture showed clear, colorless fluid at a pressure of 260 mm H₂O and containing 112 mg% protein. A pneumoencephalogram showed enlargement of the third and fourth ventricles and of the aqueduct. The right lateral ventricle was grossly enlarged, and the lateral ventricle did not fill. The pontine and interpeduncular cisternae contained air, but no subarachnoid air was seen supratentorially. A ventriculogram (Fig. 8) showed enormous dilatation of the ventricular system including the third and fourth ventricles and a large mass in the right lateral ventricle.

The scans after lumbar injection of 131I-HSA showed an obstruction to flow at the level of the tentorium (Fig. 9). The pontine cistern was well demonstrated, but only a small amount of radioactivity was present in the interpeduncular and suprasellar cisternae. No tracer entered the ventricular system. The 24-hour scan showed that a small amount of radioactivity had gone forward underneath the frontal lobes, indicating that the block at the tentorium was incomplete. No activity was seen in the quadrigeminal or callosal cisternae. A ventriculoatrial shunt was performed, followed by marked improvement in the patient's mental status, gait, and urinary incontinence. The nature of the tumor remained unknown.

These scans, together with pneumographic demonstration of enlargement of the entire ventricular system, suggest communicating hydrocephalus. There are several possible explanations for the presence of communicating hydrocephalus in this patient. One possibility is obliteration of the subarachnoid space either by the raised intracranial pressure compressing the subarachnoid spaces over the convexities, or by tentorial impaction. In this regard, Robertson has described obstruction of the subarachnoid space at the level of the tentorium due to supratentorial...
FIG. 8. Case 4. Air ventriculograms show very large lateral ventricles with a mass projecting into the right lateral ventricle.

FIG. 9. Case 4. Scans after lumbar injection show obstruction at the level of the tentorial incisura. At 24 hours some radioactivity was detected from the inferior aspect of the frontal lobes.

subdural collections, and Johnson has noted that enlarging ventricles in cases of non-communicating hydrocephalus may produce a secondary block of subarachnoid pathways at the tentorium. Another possible explanation may be obliteration of the subarachnoid space by arachnoiditis due to a toxic substance secreted into the CSF by tumor, as suggested by Dott and Gillingham.

Case 5. In January, 1965, a 61-year-old hypertensive woman had an episode of nausea and vertigo followed by diplopia, ataxia, and headache which was diagnosed as a pontine hemorrhage. Recovery was incomplete, and she remained ataxic. In March, 1966, because of persisting ataxia and the onset of dysarthria and regurgitation of fluids through the nose, angiography was done and demonstrated a huge fusiform aneurysm of the basilar artery. An air study showed minimal dilatation of the lateral ventricles. A posterior fossa myelogram with iophendylate showed a large filling defect in the pontine cistern (see ref. 16, case 2). In November, 1966, she became increasingly ataxic, confused and incontinent. She was placed in a nursing home.

In January, 1967, the patient was readmitted to hospital following an episode of unconsciousness. Examination showed mental confusion, spastic quadriplegia, more marked on the right, and bilateral upgoing toes. She was incontinent of bowel and bladder. A lumbar puncture revealed clear, colorless fluid, and the CSF pressure was 180 mm H₂O. The CSF contained 66 mg% of
protein and no cells. An air study showed striking further enlargement of the lateral ventricles in comparison with the 1966 films. Very little air was present in the sulci at this examination, in contrast to the earlier study which showed good filling of sulci. The pontine cistern was enlarged, especially in its right cerebellomedullary recess which showed an air-fluid level.

Serial scans following the cisternal injection of $^{32}$P-IHSA showed good filling of the pontine cistern (Fig. 10). The left cerebellomedullary recess of this cistern filled early and remained visible in the later scans. No radioactivity was detected above the tentorium. These scans plus the serial pneumograms established the diagnosis of communicating hydrocephalus.

The patient remained demented and quadriparetic and died in a chronic care hospital on March 20, 1967. The cause of the communicating hydrocephalus remained unknown. Although subarachnoid hemorrhage was a possibility, blood had never been found in the CSF. Arachnoiditis secondary to iophendylate has been described. The aneurysm measured approximately 3×6 cm and may itself have partially occluded the basal cisternae at the level of the tentorium.

Case 6. In July, 1962, a 55-year-old man underwent subtotal removal of a left cerebellopontine angle epidermoid cyst. In October, 1963, he developed fever, headaches, and vomiting. The CSF contained 1010 leucocytes per cu mm, mostly neutrophils, presumably due to aseptic meningitis secondary to a recurrent epidermoid cyst which was demonstrated by air study. A large amount of recurrent tumor and capsule was removed in November, 1963. One month later he became progressively more ataxic, with dizziness, and vomiting, going on to coma. The CSF pressure by lumbar puncture was 230 mm H$_2$O. An air study revealed gross enlargement of all ventricles. Air was seen in both cerebellopontine cisterns. Communicating hydrocephalus secondary to aseptic meningitis was diagnosed and treated by a ventriculoatrial shunt in January, 1964. The patient made a dramatic recovery and returned to work.

In July, 1964, headaches, vomiting, and ataxia returned, and the ventricular catheter was found to be non-functioning. Insertion of a new ventricular catheter resulted in complete recovery.

A further episode of meningitis occurred in September, 1965. The CSF contained 14,900 leucocytes per cu mm, mostly polymorphonuclear cells. The patient was treated by pumping the Pudenz shunt several times daily and by repeated lumbar punctures; he improved.

The patient then remained well until about 2 months prior to his present admission in April, 1967, when he developed progressive nausea, vomiting, and ataxia. His symptoms subsided almost completely on daily pumping of the Pudenz shunt. However, CSF pressures by lumbar puncture were 160, 210, and 200 mm H$_2$O. The CSF protein was 34 mg% and there were no cells. A pneumoencephalo-gram showed that the ventricular system was slightly more enlarged than previously. Air was seen in the pontine cistern and in the cerebellomedullary recesses, but no subarachnoid air appeared in the interpeduncular cistern or above.

Scans after the lumbar injection of $^{32}$P-IHSA showed that most of the radioactivity had entered the ventricular system (Fig. 11). There was scant filling of the basal cisternae. The Sylvian cisternae failed to fill, and there was no spread of the tracer over the cerebral hemispheres. This failure of the intracranial subarachnoid space to fill was probably due to its obliteration by the previous meningitis.
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Fig. 11. Case 6. Scans after lumbar injection show that most of the radioactivity has entered the ventricles. There is very little radioactivity in the basal cisternae and none over the cerebral convexities.

(In this case there was less radioactivity in the basal cisternae than in any of the 22 cases studied.)

Because the CSF pressures were in the high range even though the shunt was pumped daily, and because the air study revealed further ventricular enlargement, it was decided to revise the shunt. When a new venous catheter could not be inserted into the right internal jugular vein, a ventriculoperitoneal shunt was performed. After discharge the patient was asymptomatic.

Case 7. A 53-year-old woman was found unconscious on the day of admission, in January, 1967. Three years previously she had been investigated in Hungary because of a personality disorder of several years duration and the development of an acromegalic appearance; a pituitary tumor had been diagnosed but not treated.

On examination she was drowsy, with a right hemiparesis. The blood pressure was 140/100. She appeared acromegalic. A lumbar puncture revealed bloody fluid at a pressure of 165 mm H₂O. The sella turcica was enlarged. A left carotid arteriogram and a pneumoencephalogram showed a mass, considered to be an intracranial hematoma, in the left basal ganglia region. The entire ventricular system was markedly enlarged. There was good filling of the pontine and interpeduncular cisternae, but no air was seen over the sella turcica. Only an occasional sulcus contained air.

Scans following the cisternal injection of 131I-HSA showed a large amount of radioactivity in the ventricular system (Fig. 12). There was good visualization of all the basal cisternae. No radioactive material reached the Sylvian cisternae. There was marked delay in filling of the subarachnoid space over the cerebral convexities, and even at 48 hours after injection, with the scanner adjusted to maximum sensitivity, only a small amount of peripheral activity was detected.

The patient slowly recovered, the right hemiparesis almost completely disappeared, and she became alert, but still confused. The cause of her communicating hydrocephalus is unknown, although it may have been related to the subarachnoid hemorrhage. The patient refused treatment of the pituitary tumor but agreed to a ventriculoatrial shunt. After the shunt was inserted in March, 1967, the patient was less confused and more cheerful. She was discharged to a chronic care hospital.

Case 8. A 72-year-old woman with a 3-year history of progressive dementia was admitted on June 29, 1966. For the previous year she had suffered from staggering gait, and for the preceding few weeks had been incontinent. Eight years previously, she had had a brief illness characterized by fever,
headaches and stiff neck; although no CSF studies were done, this illness was probably meningitis.

Examination showed the patient to be severely demented, with marked ataxia of the legs. The tendon jerks were hyperreflexic, and there were bilateral Babinski reflexes. Lumbar puncture revealed clear colorless fluid at a pressure of 135 mm H2O, with no cells and a protein content of 24 mg%. An air study showed diffuse ventricular dilatation (Fig. 13). The cisterna pontis and suprasellar cisternae were large, but only a small amount of air was seen in the insular regions and only a few sulci contained air. Scans following lumbar injection of 131I-HSA showed that much radioactive material had entered the ventricular system (Fig. 14). There was good filling of the basal cisternae, but no migration outward into the Sylvian cisternae and sulci. Thus the diagnosis of communicating hydrocephalus was confirmed.

On July 18, a ventriculooatrial shunt was performed. Postoperatively the patient had a transient left hemiparesis, which cleared in a few days. Otherwise, her neurological status remained unchanged. On September 21, the CSF pressure was 92 mm H2O. Because of lack of improvement, a lumboperitoneal shunt was performed on October 5. The ventriculooatrial shunt was not revised because of the transient hemiparesis which had occurred. On October 24, the CSF pressure was 50 mm H2O. The patient’s clinical status remained unchanged, and she was discharged to a chronic care hospital.

Case 9. In November, 1963, a 59-year-old hypertensive man had a subarachnoid hemorrhage from a small anterior cerebral-anterior communicating aneurysm. Physical examination was normal. Twelve days later the aneurysm was clipped under hypothermia. This required occlusion of both common carotid arteries in the neck for about 5 minutes. A postoperative extradural hematoma was removed and a tracheotomy performed. Five days later the swollen necrotic left frontal pole was removed and the bone flap left out. The patient slowly regained consciousness, but had a moderately severe intellectual deficit, reduced visual acuity in both eyes, and urinary incontinence. Tantalum cranioplasty was performed before discharge.

Although there was slight improvement in the patient's mental status during the next 2 years, he remained seriously disoriented. He was readmitted in April, 1967, because of increasing confusion, urinary incontinence, and seizures. He had bilateral optic atrophy with visual acuity of 20/200 in both eyes, a right homonymous hemianopia, and equivocal Babinski reflexes. A pneumoencephalogram showed moderate ventricular dilatation, the left lateral ventricle being larger than the right. Air was seen in the basal cisternae around the midbrain, but no subarachnoid air was seen supratentorially. CSF pressures were 110, 70, and 160 mm H2O. CSF protein averaged 37 mg% and contained 4 white cells per cu mm.

Serial scans performed after cisternal injection of 131I-HSA showed much of the
radioisotope in the ventricles as well as in the basal cisternae (Fig. 15). The Sylvian cisternae showed no activity. Later scans showed minimal diffusion of tracer into the subarachnoid space over the cerebral hemispheres. A ventriculostial shunt was performed, and, when the patient was assessed 2 months later, there was slight improvement in his mental status.

Although the patient had communicating hydrocephalus, it was felt that most of his neurological deficit was due to the events associated with clipping of the aneurysm. The communicating hydrocephalus was considered secondary to obliteration of the subarachnoid space by subarachnoid hemorrhage.

**Discussion**

Pneumoencephalography, ventriculography, and positive contrast techniques are the conventional methods of demonstrating hydrocephalus. In noncommunicating hydrocephalus they provide an estimate of ventricular size and usually show the site of block in the ventricular system. In communicating hydrocephalus, these techniques also provide an estimate of ventricular size, but often fail to give sufficient information about two important functional aspects of the subarachnoid pathways of great importance in this disorder. The first is that of CSF transmission from the ventricular system to the site of CSF absorption, and the second is CSF absorption. With respect to CSF transmission, pneumoencephalography shows which of the subarachnoid pathways are patent. However, it is the functional capacity of the remaining pathways that determines whether hydrocephalus will occur; pneumoencephalography provides little information on this account. Nor does it tell anything about another function of the subarachnoid pathways, that of CSF absorption.

Over the years, several methods for differentiating between communicating and noncommunicating hydrocephalus have been devised. Dandy and Blackfan injected phenolsulfonphthalein into the CSF by lumbar puncture and examined the ventricular fluid 20 minutes later. If the fluid was colored by dye, then the block must be in the cisternae and the hydrocephalus of the communicating type. If the ventricular fluid remained colorless, then the block must be in the ventricular system and the hydrocephalus of the noncommunicating type. The test has also been performed by injecting the dye into the ventricles and retrieving it by lumbar puncture. However, these tests may be misleading, and they provide incomplete information regarding the functional adequacy of the CSF pathways. They also have the disadvantage of requiring ventricular puncture. Dandy also devised a test whereby phenolsulfonphthalein injected via lumbar puncture was measured in the urine. If 40% was recovered in the urine within 2 hours, then the cisternae must be patent. If less than 10% was recovered, then the cisternae or foramen magnum must be blocked.

Radioactive tracers have also been used for differentiating the type of hydrocephalus. In 1957, Bell injected 131I-HSA into the lateral ventricles and measured with a Geiger-Müller counter the radioactivity over the back of the head and down the spine to the lumbar
region. He could detect complete blocks in the ventricular system, but not partial blocks or blocks beyond the ventricular system. Similar tests have been performed with brain scanning. Di Chiro and colleagues used \(^{131}\)IHS, and Spoerri and Rösler used radioiodinated Hippuran and Hypaque injected directly into the ventricles, and were able to demonstrate the site of the block in cases of noncommunicating hydrocephalus. In incomplete blocks, this test may have certain advantages over conventional radiographic procedures, but in most cases of noncommunicating hydrocephalus the diagnosis can be made more easily by air or positive contrast ventriculography. The same authors have speculated that intrathecal injection of these tracers might be useful in the detection of communicating hydrocephalus.

The results reported here indicate that intrathecally administered \(^{131}\)IHS is useful in the diagnosis of communicating hydrocephalus. It provides information about two important functional aspects of the subarachnoid space, the transmission and absorption of CSF. It will be noted that most of the cases reported in this paper were extremely complex; some were complicated by the presence of both noncommunicating as well as communicating hydrocephalus, some had had several shunting procedures, and, in some, more than one disease process was causally related to the hydrocephalus. Isotope cisternography was an extremely useful adjunct.

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**Fig. 14.** Case 8. Scans after lumbar injection show good filling of the basal cisternae and ventricles. No radioactivity is seen in the Sylvian cisternae or hemispheral subarachnoid space.

**Fig. 15.** Case 9. Scans after cisternal injection show good filling of the basal cisternae and ventricles. At 22 and 48 hours, a small amount of radioactivity is seen in the frontal regions near the midline.
in the clarification of these complex diagnostic problems. In each patient with communicating hydrocephalus there was demonstrable obstruction to the flow of tracer in the subarachnoid space, and the site of the obstruction could generally be identified.

In most instances, the results of the isotope tests confirmed the pneumoencephalographic findings, but usually the isotope test provided much more diagnostic information about the functional capacity of the subarachnoid space. For example, in Cases 5 and 8, some air did reach the subarachnoid sulci over the cerebral hemispheres, and there was therefore some doubt about the diagnosis of communicating hydrocephalus on the basis of the air study. The CSF scans in these cases showed definite obstruction to the flow of tracer in the subarachnoid space. It is in this type of case, with incomplete subarachnoid obstruction, that the isotope method has an advantage over air studies.

The isotope test clearly differentiates communicating hydrocephalus from hydrocephalus-ex-vacuo. In the former, the scans show restriction of the subarachnoid flow of the tracer, whereas in the latter, the scans show that the subarachnoid pathways are widely patent (Fig. 16).

In the diagnosis of communicating hydrocephalus, cisternal rather than lumbar injection is preferred. Scans can be performed immediately after cisternal injections, avoiding the 2-hour delay after lumbar injection. The cisternal route produced better definition of the subarachnoid pathways because more radioactivity was present, and also demonstrated the cisterna magna, which was not seen in normal cases after lumbar injection.

In the present study there have been no complications from intrathecally injected $^{131}$IHA. Others have reported one case of pyrexia after an intraventricular injection of $^{131}$IHA of unspecified protein content, and two cases of aseptic meningitis following lumbar injection of $^{131}$IHA containing 6% albumin. It is suggested that a preparation of highest possible specific activity and therefore low protein content be used. The radiation dose to the central nervous system has been estimated to be 1 rad, and the whole body dose to be 100 millirads after the intrathecal injection of 100 $\mu$C of $^{131}$IHA. These doses are within acceptable limits.

It is possible that $^{131}$IHA may not be the ideal tracer for demonstrating the subarachnoid pathways. Spoerri and Rössler preferred radiolabeled Hippuran and Hypaque because they were absorbed more quickly from the CSF and eliminated more quickly from the body. Di Chiro also used Technetium 99m as the pertechnate ion but found that the isotope was absorbed locally and failed to be distributed throughout the subarachnoid space when injected by lumbar puncture or intraventricularly. Rieselbach, et al., injected colloidal gold by lumbar puncture and found that this compound was well distributed throughout the subarachnoid space. However, it moved slowly through the subarachnoid
space and became fixed to surrounding tissue. Future studies of other tracers may prove them to be more useful.

**Summary**

This paper describes the use of a radio-isotopic technique for the diagnosis of communicating hydrocephalus. Radiodinated human serum albumin was injected into the CSF of patients either by lumbar or cisternal puncture, and serial scintillation scans of the head were made. In normal cases one can recognize a characteristic pattern of tracer flow through the basal cisternae and subarachnoid space of the cerebral hemispheres, whereas in communicating hydrocephalus there was obstruction to flow of the tracer at varying levels in the intracranial subarachnoid space. Of the nine cases of communicat-
ing hydrocephalus presented, one showed a block in the cisterna pontis and four showed a block at the tentorial incisura or just beyond in the suprasellar cistern. In the remaining four cases, most of the tracer entered the ventricular system, and although this tended to mask the exact site of block in the basal cisternae, there was total or almost total ob-
struction of the subarachnoid space over the cerebral hemispheres including the Sylvian cisternae.

This technique is a useful adjunct to con-
vventional radiological procedures in patients with hydrocephalus. Its most important fea-
ture is that it differentiates communicating hydrocephalus from hydrocephalus-ex-vacuo (fluid outside the brain or outside the normal cerebrospinal fluid pathways): in communi-
cating hydrocephalus the scans show obstruc-
tion to the flow of the tracer, and in hydro-
cephalus-ex-vacuo the scans reveal patency of the subarachnoid pathways.

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