AQUEDUCT STENOSIS

CLINICAL ASPECTS, AND RESULTS OF TREATMENT BY VENTRICULO-CISTERNOSTOMY (TORKILDSEN’S OPERATION)

KENNETH W. E. PAINE, F.R.C.S., AND WYLIE McKISSOCK, F.R.C.S.
National Hospital, Queen Square, London, England

(Received for publication November 29, 1954)

Partial or complete occlusion of the sylvian aqueduct may be caused by congenital malformation, post-inflammatory ependymitis, or may result from pressure of an intracranial tumour, aneurysm or angioma. In order to overcome such an obstruction some new pathway must be made that will enable the cerebrospinal fluid to gain access to the subarachnoid space covering the cerebral hemisphere or be directed to some other absorptive space.

The obvious method would be to dilate the aqueduct or to insert a tube connecting the third and fourth ventricles. Dandy, in 1920, reported 2 cases in which he passed a tube from the fourth ventricle through the aqueduct. In both cases the tube was later removed. One patient died and the other was reported as being well 1 year after operation. Fraser and Dott reported 5 cases of aqueduct stenosis, in 4 of which the operative procedure was a cerebellar decompression and insertion of a probe through the aqueduct. One patient was stated to be cured, 1 improved, and the other 2 died. The follow-up period was not given. Leksell, more recently, used a tantalum wire coil to pass through the stenosed aqueduct. He had 2 operative deaths, and 1 death a year after operation, in a group of 10 patients. Three infants under the age of 1 year did not survive the operation.

Shelden, Parker and Kernohan described the treatment of 6 patients with aqueduct stenosis, 1 with an associated meningocele. All 6 died: 1 after lumbar puncture, 3 after cerebellar decompression, 1 after subtemporal decompression and 1 after third ventriculostomy.

Stookey and Scarff, summarising the results of treatment in reported cases of the condition, found that there were 10 deaths among patients not submitted to operation, 1 death after subtemporal decompression and puncture of the corpus callosum, 2 deaths following ventriculography, 2 deaths after suboccipital decompression, and 1 death 5 weeks after this procedure. No survivals were recorded. These authors then reported the results of their operation of third ventriculostomy. Four patients were submitted to this procedure. One died after 1 month, but the other 3 were stated to be well 6 months, 14 months and 3 years later. Here at last seems to be a method of treatment that yields successful results.

Pennybacker in 1940 reported to the Royal Society of Medicine 18 cases of aqueduct stenosis. A variety of operations had been practised on
these patients. Cerebellar decompression had been performed in 7, 1 of whom died postoperatively from a wax embolus. Another patient had a recurrence of symptoms 3 months later. Three patients were well 3 years after operation and one was well 4 years after operation. Two of these patients had been subjected to a course of irradiation. The final patient, who had had a pineal exploration in addition to the decompression, was well 4 years later. Third ventriculostomy was performed in 5 patients, 2 of whom died postoperatively whilst the other 3 were reported as being well 4 months, 1 year and 2 years later. In 2 patients the corpus callosum was split but 1 died postoperatively and the other 10 months later. The only patient subjected to a pineal exploration and irradiation was well 7 years later. One patient who had had a subtemporal decompression and a course of X-ray therapy was still symptom-free after 6 years. One patient died from an intracranial infection after ventriculography and the remaining patient died 3 years after diagnosis, having received no operation.

Drainage of the cerebrospinal fluid into the body tissues or cavities has been advocated by many, tubes of varying construction having been passed between the ventricular system above the structure to the mastoid, jugular vein, the soft tissues of the neck or scalp, peritoneum and pleura, to mention but a few. No one of these methods has proved satisfactory and the death rate has been high. The tendency of cerebrospinal fluid to produce a dural membrane around itself prevents success by many of these methods whilst drainage into such cavities as the middle ear opens a potential road for infection of the subarachnoid spaces.

Avulsion of the choroid plexus to provide a pathway from the lateral ventricle into the cisterns around the brain stem was advocated by Hildebrand in 1904. Hyndman described 1 case of stenosis of the aqueduct treated by his operation of disruption of the choroid plexus but the patient died 2 months later.

Third ventriculostomy, as mentioned above, was one of the early methods of treatment that gave some hope of success in the management of patients with aqueduct stenosis. Stookey and Scarff first reported 4 operations with 3 survivals. Their original procedure required the making of an opening in the lamina terminalis above the optic chiasm, the exposure being through a transfrontal craniotomy. Scarff later reported on 10 cases, in which 7 operations were performed for the relief of aqueduct stenosis. There were no operative deaths but 1 patient died a month after surgery. Another survivor died 5 years later. Three other patients were living 1, 3½ and 5 years after operation. The follow up was incomplete for the remaining patient. White gave his results of this operation on 3 patients with occlusion of the aqueduct. Two survived but 1 died 2 months after operation from pneumonia. Krayenbühl, Werner and Martin performed this operation on 17 patients, 4 of whom had stenosis of the aqueduct. Of this latter group 3 were relieved of symptoms, for 3 years in 2 cases and 4 years in 1. The fourth patient was well for 3 years but then suffered a recurrence of symptoms.
Scarff was not entirely satisfied with the results of the operation of anterior third ventriculostomy and in 1950\textsuperscript{29,30} described a modification in which the third ventricle was opened anteriorly and then a further opening was made into the floor of the ventricle, so allowing the cerebrospinal fluid to escape into the interpeduncular cistern. This was an attempt to prevent the formation of the frontal subdural collection of cerebrospinal fluid that accumulated after the original procedure. He reported 17 cases of aqueduct stenosis treated by this new method in a series of 34 patients. In this series 4 patients died postoperatively and 1 died 6 months after operation. Of the remaining 12, 11 were relieved of their intracranial hypertension for periods varying from 2 months to 14 years. Only 1 survivor was not relieved by operation.

Dandy\textsuperscript{1} had earlier experimented with the operation of third ventriculostomy, his method being to puncture the posterolateral wall of the third ventricle through a subtemporal approach. He reasoned that the falling back of the medial portion of the temporal lobe would close the cisterns around the brain stem, and so prevent the collection of subdural cerebrospinal fluid. He reported 1 operative death and 3 later deaths at 3, 5 and 6 months. Seven patients required reoperation following closure of the opening into the third ventricle. Twenty-nine patients survived more than 1 year, the longest follow up being 23\frac{1}{2} years. He also reported 63 patients under the age of 1 year with 10 hospital deaths and continued survival in 21 cases.

Ventriculo-ureterostomy has been used by Matson\textsuperscript{20} to treat 2 children with aqueduct stenosis. Both were improving 2\frac{1}{2} and 4 months after operation.

It was in 1937 that Torkildsen first performed his operation for providing a new sylvian aqueduct by passing a soft rubber tube from one lateral ventricle, through a subgaleal tunnel over the occiput, and inserting the lower end into the cisterna magna. Ten years later\textsuperscript{34} he published his results in a series of 47 cases, in 13 of which the patients were suffering from aqueduct stenosis. Four of the 13 died postoperatively, whilst 2 others, at 6 weeks and 4 months respectively, succumbed from the continuation of the raised intracranial pressure in spite of the short-circuit operation. One more patient died 2\frac{1}{2} years after operation, following ventriculography, although she had remained in good health from the time of the operation. Thus over half of Torkildsen’s original 13 patients failed to survive.

Fincher, Strewler and Swanson\textsuperscript{8} reported 5 patients treated by Torkildsen’s operation of whom but 1 had died postoperatively, whilst the rest were relieved and alive 18 to 36 months later.

Of the 4 patients described by Herlin\textsuperscript{12} 2 died postoperatively, 1 was alive but unrelieved 5 years afterwards, whilst the remaining patient was surviving and well 10 months after operation.

This paper presents a series of 25 patients treated by Torkildsen’s method for non-neoplastic aqueduct stenosis and outlines the clinical features, radiological findings and results of treatment.
The series is too small to subdivide into differing pathological groups, were it possible to do this with certainty in the absence of pathological proof. The causes, therefore, probably include many varieties of stenosis of the aqueduct of Sylvius, with the exception of tumour. These have been described as absence, obliteration, partial atresia, duplication, forking, and occlusion caused by ependymitis. The only case of a septum of the lower end of the aqueduct, seen in this clinic, has not been included, as the operative procedure consisted of rupture of this membrane through the fourth ventricle.

**CLINICAL PICTURE**

*Age at Onset of Symptoms.* In about two-thirds of the patients symptoms developed before the age of 20 years: 9 before the age of 10, and 8 between the ages of 10 and 19 years. Of the remaining 8 patients, symptoms commenced in the third decade in 5, in the fourth decade in 2 and in the fifth decade in 1 (Table 1).

**TABLE 1**

<table>
<thead>
<tr>
<th>Age at onset of symptoms</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 to 10 years</td>
<td>9</td>
</tr>
<tr>
<td>10 to 20 years</td>
<td>8</td>
</tr>
<tr>
<td>20 to 30 years</td>
<td>5</td>
</tr>
<tr>
<td>30 to 40 years</td>
<td>2</td>
</tr>
<tr>
<td>40+ years</td>
<td>1</td>
</tr>
</tbody>
</table>

*Duration of Symptoms before Treatment.* There was a very wide variation in the duration of symptoms, so far as this could be estimated, the shortest history being only 3 weeks and the longest 46 years. Eleven patients (44 per cent) gave a history of less than 1 year and in 8 of these cases the duration of symptoms was less than 6 months. The remaining 14 cases were divided into small groups giving histories of longer periods of disability and are set out in Table 2.

The age at onset of symptoms might be a point in differentiating congenital from acquired lesions, the latter starting later in life, but we are not

**TABLE 2**

<table>
<thead>
<tr>
<th>Duration of symptoms</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 to 6 months</td>
<td>8</td>
</tr>
<tr>
<td>6 to 12 months</td>
<td>3</td>
</tr>
<tr>
<td>1 to 2 years</td>
<td>2</td>
</tr>
<tr>
<td>2 to 3 years</td>
<td>2</td>
</tr>
<tr>
<td>3 to 4 years</td>
<td>3</td>
</tr>
<tr>
<td>4 to 5 years</td>
<td>2</td>
</tr>
<tr>
<td>5 to 10 years</td>
<td>2</td>
</tr>
<tr>
<td>10 to 20 years</td>
<td>1</td>
</tr>
<tr>
<td>20 to 30 years</td>
<td>1</td>
</tr>
<tr>
<td>46 years</td>
<td>1</td>
</tr>
</tbody>
</table>
able to make any such deductions from this series. The duration of symptoms could be an indication of the degree of obstruction of the aqueduct—the less the stenosis the longer the period the patient is able to tolerate the cerebrospinal fluid obstruction. By the time these patients come to ventriculography, the stenosis appears to be almost complete in all, and so, again, we are unable to comment on this feature.

**Age of Patient at Operation.** This ranged from 18 weeks to 58 years. Four patients were under 10 years, 10 aged between 10 and 20 years, 5 between 20 and 30 years, 3 from 30 to 40 years, 2 in the fifth decade and one aged 58 years.

There is no doubt that the very young patients with aqueduct stenosis would not be included in this series, the operation of ventriculoeisternostomy not being practised in young children. Matson\(^{20} \) regards extreme youth as a contraindication to the performance of Torkildsen’s operation, stating that the subarachnoid spaces are imperfectly developed and will not allow the passage of cerebrospinal fluid through the normal channels over the vertex. Dorothy Russell’s\(^{28} \) researches into hydrocephalus have shown that many infants died from hydrocephalus caused by aqueduct stenosis. Furthermore, stenosis of the aqueduct may be associated with other congenital abnormalities and so make the problem of treatment in early life a complex one.\(^{16,19,26,27} \) Certainly the patient, in this series, who was operated upon before the age of 1 year, died in the immediate postoperative period.

**SYMPTOMS**

**Headaches** occurred in 22 patients, but in only 12 could it be said to have been the presenting symptom. In this latter group the headaches had been present for less than 7 months in 8 patients. In one instance the patient had had three severe headaches in 4 years but a persisting headache for 5 weeks before admission. Another had had a mild headache for 2 years but more severe headache in the 5 months before admission. In the other 2 the headaches had been the main symptom for 1 and 6 years respectively.

In the group in which the headache was an incidental symptom it had been present for less than 6 months in 3 cases, 9 months in 1 and 16 months in another. One patient recalled an episode for 1 week, 2 years earlier. Two patients described occasional but not severe headaches for some time and the final patient could only describe a feeling of fullness in the head associated with nausea and vomiting.

The headache usually had the features of that of raised intracranial pressure, worse in the morning on waking, aggravated by exertion, often associated with nausea and vomiting, and usually clearing during the day. The site was variously described as “all over,” bifrontal, bitemporal or vertical but never occipital. The intermittent nature of the headache was apparent in those patients with the longer histories, and the intervals between the attacks might vary from a few weeks to many months. As noted above, there were instances of many years of freedom from headaches. In such
cases the paroxysms of headache suggested temporarily increased obstruction of the cerebrospinal fluid pathways with subsequent re-establishment of the flow. Those patients with headache of but a few weeks’ or months’ duration before admission to hospital all complained of the persistent ingravescent nature of the pain suggesting that a final obstruction or failure of compensation had taken place.

Nausea and Vomiting. These, the next most common complaints, were present in 13 of the 25 patients, and in 12 of the 13 were associated with the bouts of headache although they did not necessarily occur with every attack of pain in the head. In the remaining case there was no complaint of headache at all, vomiting being an isolated symptom.

Visual failure was a spontaneous complaint in 11 cases and in only 1 other patient was there visual deterioration on examination. Visual failure had been noticed for 2 weeks in 2 patients, 2 months in 2, 4 months in 1 and 18 months in 2 others. One patient had had deterioration for 2 years, another for 3 years and another for 4 years. The final patient complained of failing vision for 20 years. Two patients had early papilloedema, 3 moderate swelling, 3 severe swelling and 3 had secondary optic atrophy. Except that the patient with a 20-year history had secondary atrophy, there appeared to be no relation between the length of history and the degree of papilloedema.

Mental Defect or Deterioration. This condition was present in 11 cases but was never the primary complaint of patients or relatives. Mental deficiency was of minor degree in 5 patients and moderate or severe in the other 6. The mental change in 2 of these 6 patients was not in evidence until after a head injury which was severe enough in 1 instance to produce a hemiplegia.

Unsteadiness occurred in 10 patients and clinical examination in 9 of these revealed incoordination or ataxia of the limbs suggestive of cerebellar dysfunction. In the tenth case nystagmus was present with minimal pyramidal signs. In 4 instances the unsteadiness was the presenting symptom and had been present for 2 weeks in 1, 2 years in 1, 6 years in 1 and for an unstated number of years in the fourth.

Increasing weight was complained of by 6 patients and in 2 of these there were associated endocrine changes.

Endocrine changes occurred in 5 patients, 2 females having irregular periods, 2 males being sexually underdeveloped, and menstruation not having occurred in the fifth, a girl of 15. Endocrine changes may well be associated with hypothalamic disturbances produced by the dilated third ventricle as are known to occur in patients with tumours of the posterior part of the third ventricle or with obstruction of the ventricular system more distal to this.

Attacks of unconsciousness occurred in 5 patients. In 1 case such attacks were the presenting symptom and were sometimes associated with generalised convulsive movements: this patient had suffered from headache and vomiting for a number of years. In another instance the attacks appeared to be epileptiform following a severe head injury at the age of 18 months. In a
third case the attacks also appeared to be those of post-traumatic epilepsy. The other 2 patients had intermittent attacks of unconsciousness, 1 having had two attacks in 2 to 3 weeks only, and the other a number of “faints.” In only one instance, therefore, were these attacks suggestive of an acute hydrocephalic episode.

Diplopia was a complaint of 5 patients, all with severe headache of brief duration, and in 3 instances was one of the presenting symptoms. Three patients had a unilateral sixth nerve palsy but no cause could be found in the other 2.

Epilepsy was an associated symptom in 4 patients, 2 of whom have been mentioned above. The other 2 had attacks of dissociation suggestive of temporal lobe seizures.

Giddiness was complained of by 4 patients and in 1 was associated with unsteadiness and cerebellar signs. None of the other 3 had clinical evidence of cerebellar or brain stem dysfunction. In 1 instance the vertigo was a presenting symptom.

A large head was remarked upon in 3 cases, and in 1 child aged 18 weeks was the only symptom.

A weak arm or leg as part of a hemiparesis was present in 3 patients but in all could be attributed to natal or postnatal trauma.

Incontinence was present in 3 patients.

Paraesthesiae of the arms or legs were present in 3 cases, in only 1 of which could the sensation be attributed to epilepsy.

Dysarthria was present in 2 patients, both with well marked cerebellar signs.

“Bad nerves” had been complained of by 2 patients for a varying length of time.

Difficulty in opening her mouth for the previous 4 months was the surprising symptom presented by 1 patient, a nurse.

**PHYSICAL SIGNS**

*Fundus Examination.* Bilateral papilloedema was present in 16 patients, unilateral papilloedema in 1 and secondary optic atrophy in 4. Thus in 21 cases there was evidence that raised intracranial pressure was present or had been present. The papilloedema was described as early in 5 cases, and thus in 16 patients incontrovertible fundus changes were present. Two patients were said to have primary optic atrophy and the remaining 2 to have normal fundi. These 2 were aged 18 weeks and 10 years with histories of 5 weeks and 14 months respectively.

*Visual Acuity.* Seven patients had an acuity of 6/60 or less in one eye, 1 patient had a bilateral cataract and 1 was too young for visual assessment.

*Cerebellar Signs.* Ataxia of the arms or legs occurred in 14 patients. Nine of these had complained of the unsteadiness as a symptom. In the other 5 the cerebellar signs were of slight degree only.

*Nystagmus* was present in 7 patients and was unassociated with other
cerebellar signs in only 2. One of these made a spontaneous complaint of giddiness.

*Pyramidal signs*, either as a manifest hemiplegia or as a reflex change only, was present in 11 cases. In 1 case an extensor plantar response was the only abnormality and in 2 other cases, an increase in the tendon reflexes on the abnormal side. In 1 instance the hemiplegia dated from birth, in another from a head injury at 18 months, and in 1 it followed an illness at the age of 18 months (? encephalitis). The hemiplegia in the remainder of the patients was only slight.

*Facial weakness* as the only sign of an upper motor neurone lesion was present in 3 other patients.

*Obesity* was an observed feature in 12 patients and included all those with endocrine disturbances.

*A large head* was recorded in 8 cases, although it had been commented upon by the relatives in 3 only. In 7 of these patients the onset of symptoms was before the age of 12 years. The other patient was 30 years old on admission and her history was of 2 years’ duration.

*A sixth nerve palsy* was unilateral in 5 cases and bilateral in 1. Five of these patients had severe papilloedema and 1 had secondary optic atrophy.

*Deafness was present in 3 cases.*

*Visual field defects* were present in 2 patients, nasal in one eye in 1 patient and bitemporal in the other. Constriction of the visual fields and enlargement of the blind spot were common findings.

*Café au lait patches* were present in 3 patients, 1 of whom had generalised neurofibromatosis. Pennybacker reported a similar patient.

*Hyposmia, diminished corneal reflexes and ptosis* were each recorded once only in the clinical examination of the patients of this series.

*Loss of position sense*, other than in association with a severe hemiplegia, occurred in the arm and leg of the same side of 1 patient.

*An occipital naevus* was present in 1 case.

*Comment.* The clinical picture of aqueduct stenosis is not, therefore, clearly defined and, indeed, many writers in the past have brought attention to the confusing array of symptoms and signs suggesting a diagnosis of cerebellar tumour. To us there appears to be a group of patients in whom a preventriculographic diagnosis of aqueduct stenosis can be entertained. It is composed of children of 10 to 15 years of age, usually a little slow at school and overweight, who complain of increasing headaches over a period of a year to 18 months. They may or may not have noticed visual deterioration; the parents may comment on the size of hat the child takes. Examination discloses a head on the large side of normal with a high-pitched percussion note. Bilateral papilloedema of slight to moderate degree is present, a convergent without demonstrable weakness of eye muscles is common. There is a fine tremor of the outstretched hands with a little ataxia of movements. Balancing tests are not well performed. In these patients the history is too long and the signs are too slight to make a diagnosis of cerebellar tumour,
the slightly large head and the abnormal percussion note indicate that the hydrocephalus has been advancing for a longer period than the history suggests, and one is faced with a patient of preadolescent age with a benign obstructive hydrocephalus without any certain localising signs but with questionable cerebellar dysfunction. Smallness of the posterior fossa on palpation will weigh heavily in favour of the diagnosis of aqueduct stenosis. Of course diagnosis can be made by pneumography with that certainty necessary for operation.

RADIOLOGICAL EXAMINATION

Plain roentgenograms of the skull demonstrated raised intracranial pressure in 20 instances. Erosion of the dorsum sellae and digital markings in the vault were the common findings (Fig. 1). In 4 instances the posterior fossa was small and suggested the diagnosis of aqueduct stenosis (Figs. 2 and 3). Dandy stated that the inion is situated at a relatively lower level in those patients who have hydrocephalus caused by aqueduct stenosis, than when the hydrocephalus is produced by any other cause. It is probably more nearly correct to say that a benign obstruction of the ventricular system between the third ventricle and the fourth ventricle, commencing in childhood, is associated with a greater relative enlargement of the supratentorial por-

![Fig. 1. Roentgenogram showing suture diastasis, erosion of the dorsum sellae, and increased convolutional markings in the skull of a patient with aqueduct stenosis.](image-url)
Fig. 2. Roentgenogram of the skull of an adult patient with aqueduct stenosis, showing the small posterior fossa.

Fig. 3. Roentgenogram of the skull of a child with aqueduct stenosis, showing the enormous hydrocephalic enlargement of the vault but without enlargement of the posterior fossa.
tion of the skull than if the obstruction lies in or below the fourth ventricle. A posterior fossa tumour of benign nature will be associated with an expansion of the posterior fossa caused by the presence of the tumour, as well as enlargement of the supratentorial portion of the skull vault caused by the hydrocephalus. The inion thus remains in a relatively high position. One skull in this series, that of a young child, showed a very great increase in the size of the head, separation of the sutures and enlargement of the fontanelles. One other patient had an associated platybasia.

Another feature of a nonspecific nature, frequently seen in the plain roentgenograms, is the presence of large diploic channels in the occipital bone over the posterior fossa. Longstanding raised intracranial pressure is the probable cause of the opening up of these venous pathways. Fig. 4 shows a skull with these diploic channels, in this instance over the supratentorial portion of the skull as well.

Ventriculography was performed in 24 cases, the contrast media being air only in 12, myodil only in 5, and both air and myodil in 7. It is our practice at the present time to use myodil alone or myodil plus 10 cc. of air into the ventricles of a patient suspected of suffering from aqueduct stenosis. If at ventriculography the lateral ventricles are large, then 1 cc. of myodil (pantopaque, ethiodan) and 10 cc. of air are introduced into the ventricles. If the ventricles are small, then our suspicions were probably ill founded and we proceed to ventriculography with air in the usual manner. Another method which we have found useful is to introduce only 10 to 15 cc. of air into

![Fig. 4. Roentgenogram of the skull of a patient with aqueduct stenosis showing the large diploic channels in the occipital bone over the posterior fossa.](image-url)
the ventricles if they are large. This air can then be manipulated into the third ventricle and aqueduct and will not be obscured by overlying air in the temporal horns, as would be the case if larger amounts of air were used. Verbiest\(^5\) obviously found the same difficulty for he suggested manipulating air into the third ventricle and then replacing the air in the lateral ventricles with saline before taking the X-rays!

Ventriculographic Appearances. The first and obvious feature is the enlargement of the lateral and third ventricles. This at once suggests that the obstruction of the ventricular system is at or below the posterior portion of the third ventricle. We have found a number of different appearances of the stenosed portion of the aqueduct:

(1) Partial Occlusion. Fig. 5 shows a narrowing of the aqueduct just after its origin from the third ventricle, occurring in a child 5 years old.

(2) Funnelled Aqueduct. Fig. 6 demonstrates a similar appearance of the proximal portion of the aqueduct, but in this case myodil could not be made to pass through the distal portion. The pointed ending is well shown.

(3) Bulbous Type. Fig. 7 shows an obstructed aqueduct ending in a bulbous fashion as though enlarged by the occlusion at the lower end. Fig. 8 is an air ventriculogram showing a similar appearance.

(4) Atresia of the Aqueduct. Fig. 9 shows the appearances of the myodil ventriculogram when none of the opaque material can be passed through the

---

**Fig. 5.** Myodil ventriculogram showing partial occlusion of the sylvian aqueduct.
AQUEDUCT STENOSIS

Fig. 6. Myodil ventriculogram to show funnelling of the aqueduct of Sylvius.

aqueduct. The projection of the third ventricle toward the mouth of the aqueduct is to be seen in the upper blob of myodil.

No anteroposterior projections are shown, but are necessary to confirm that the posterior portion of the third ventricle and the aqueduct and fourth ventricle are not displaced to one side. Projections into the third ventricle as from pineal tumours or thalamic tumours should be easy to distinguish. Tumours of the posterior fossa will show kinking with or without lateral displacement of the aqueduct and may show displacement of the fourth ventricle. Brain stem tumours do not cause obstruction of the aqueduct without deformity of that structure. The first three types of ventriculo-

Fig. 7. Myodil ventriculogram showing the bulbous proximal portion of the aqueduct.
Fig. 8. Air ventriculogram to show enormously dilated lateral ventricles and the bulbous ending of the occluded aqueduct.

Fig. 9. Myodil ventriculogram showing the opaque medium in the posterior portion of the third ventricle but with none passing through the aqueduct.
graphic appearances mentioned above are pathognomonic of stenosis of the aqueduct provided that there is no anteroposterior or lateral displacement of that structure seen on the ventriculograms. The fourth type may give doubt in diagnosis as without visualisation of the aqueduct or fourth ventricle it is impossible to exclude a tumour as the cause of the occlusion.

In this series, the only patient not submitted to ventriculography was one in whom a posterior fossa tumour was suspected clinically. After a negative cerebellar exploration, a Torkildsen’s operation was performed.

OPERATIVE PROCEDURE

Ventricular Drainage. In 4 cases, catheter drainage of one lateral ventricle was established, 2, 4, 5 and 6 days respectively, before operation. These patients were all deemed to be bad operative risks because the severity of the headache was suggestive of an acute hydrocephalic attack. Albeit 2 of these patients died after the ventriculocisternostomy and we are not able to say whether this was in spite of, or because of, the preliminary decompression.

Anaesthesia. General anaesthesia was used in every case in the series and differed in no way from that in use in the departments concerned with most major neurosurgical procedures. The induction has been that appropriate to the age and condition of the patient followed by nitrous oxide and oxygen with such adjuvants as were thought desirable or necessary.

Operation. All patients were operated upon in the sitting table position, using a special operating table.

Through a midline incision from the inion to the midecervical region a small bony opening is made in the occiput including the posterior rim of the foramen magnum, measuring in all some 4 cm. in diameter. A small linear opening is then made in the dura mater over the cisterna magna just to one side of the midline and the occipital sinus and on the side of the posterior parietal burr hole made for ventriculography. If the diagnosis is correct the cistern should be of large size and it is only rarely that a tonsillar herniation has been found. The arachnoid over the cistern should not be opened at this stage. The posterior fossa wound is covered with a wet pack and a subgaleal tunnel is made between the upper aspect of the wound and the previously made posterior parietal incision, this latter incision having been reopened. A No. 3 or No. 4 soft rubber catheter is passed through the parietal burr hole and into the lateral ventricle so that some 4–6 cm. will lie in the ventricle. The distal end of the catheter is passed into the posterior fossa wound via the subgaleal tunnel. It is unnecessary to make any side openings into the catheter inserted into the lateral ventricle.

Attention is now turned to the lower end of the catheter, which is cut so as to reach about 1 cm. below the lowest end of the dural opening over the cistern. The cut is made on the bevel so that the opening points inwards. A small opening is then made in the arachnoid over the cisterna magna and through this arachnoid opening the distal end of the tube is passed. The catheter is held in place with one black silk suture which holds the distal end close to the posterior aspect of the cistern. The dura mater is closed with interrupted black silk sutures to make a watertight closure.
and is reinforced with Oxyel or Gelfoam to prevent leakage. The wound is then closed with multiple rows of interrupted silk sutures.

The patients are nursed in the sitting position and if all proceeds well no special aftertreatment is necessary. On occasions it may appear that the tube has become blocked and then it may be necessary to aspirate the tube through the skin in the occipital region to re-establish drainage. If such is done then it is probably wise to perform daily lumbar punctures to encourage the flow of cerebrospinal fluid through the tube. If cerebrospinal fluid leaks around the tube it may be necessary to aspirate the subgaleal space and should leakage continue a temporary ventricular drain in the opposite lateral ventricle may tide the patient over the initial postoperative phase.

Small Variations in Technique in the 25 Patients. A full curved cerebellar incision was made in 4 patients in the belief that a posterior fossa tumour was present. After a negative exploration a Torkildsen's short circuit was performed as described above. The arch of the atlas was removed in 12 patients, the arches of the atlas and axis in 1, and the laminae of C1–3 in 1 patient. This last patient had a condition which appears from the operative description to be an Arnold-Chiari malformation. There appears to be no difference in the results with or without removal of the atlas. That tonsillar herniation was not the indication for removal of the arch of the atlas in all such cases is evidenced by the fact that in only 4 cases were the tonsils below normal and in 3 of these were described as herniated.

In 1 patient a circular opening was made in the occipital bone leaving the posterior rim of the foramen magnum intact. This patient died post-operatively.

In 1 instance a polythene catheter was used but proved very difficult to insert.

Postoperative Complications. There were 5 postoperative deaths. In 3 cases the cause of death was not established post mortem but the diagnosis of aqueduct stenosis was confirmed. One patient died on the third and 2 on the fifth postoperative days. One patient died from gastroenteritis 14 days after operation. The fifth patient died from a coliform meningitis and ventriculitis 6 weeks after operation. This was the only proven infection in the series although many patients showed evidence of meningeal irritation in the postoperative period presumably caused by presence of the rubber catheter.

In 2 patients a hemiplegia developed as a result of operation and in neither case have we adequate reason to explain it.

One patient required a revision of the Torkildsen's operation after 31 months and her result below is assessed from the time of the second operation.

LATE RESULTS OF OPERATION

One patient died after discharge from hospital and 7 months after operation from what seemed to have been the effects of a continued rise of intra-
craniocerebral pressure (i.e., failure of operation). One patient was obviously deteriorating 3 months after operation.

Two patients were worse, physically, 14 and 39 months after operation although their intracranial pressure was restored to normal.

Three patients, 18, 19 and 32 months after operation, appear to have had their symptoms arrested, but, as regards vision and ability to work, are no better than before operation. These patients have been saved from a worse future.

Nine patients have done well and are at school or full work although one of them is blind. Two of these patients have been followed for 1 1/2 years, 1 for 2 years, 1 for 2 1/2 years, 2 for 3 years, 1 for 3 1/2 years, and 2 for 4 years.

Four patients operated upon 1, 2, 6 and 8 months ago are improving. These results are summarised in Table 3.

**TABLE 3**
Results of ventriculocisternostomy

<table>
<thead>
<tr>
<th>Type of Outcome</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Died as a result of operation or consequences of the primary condition</td>
<td>6</td>
</tr>
<tr>
<td>Deteriorating as a result of failure of operation</td>
<td>1</td>
</tr>
<tr>
<td>Made worse by operation although the hydrocephalus was arrested (hemiplegia produced)</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total failures</strong></td>
<td><strong>9</strong></td>
</tr>
<tr>
<td>Condition arrested by operation but patient not improved economically</td>
<td>3</td>
</tr>
<tr>
<td>Condition arrested and patient in full-time occupation</td>
<td>9</td>
</tr>
<tr>
<td>Too short a follow up although patient improving when last seen</td>
<td>4</td>
</tr>
<tr>
<td><strong>Total successes</strong></td>
<td><strong>16</strong></td>
</tr>
</tbody>
</table>

We thus have evidence that the short-circuit operation has proved satisfactory in 18 cases (73 per cent) although successful economic results were obtained in only 9 out of 21 followed for more than a year (43 per cent).

Failing vision is often a presenting symptom and it is of interest to investigate the changes in vision following a short-circuit operation.

In 12 cases the vision remained the same as at the time of the initial examination. In 4 instances there was improvement in vision, the actual changes being from 6/12 or 6/18 to 6/9 or better. No patient with a vision of less than 6/18 was improved.

In 8 patients there was a deterioration of vision after operation. One patient with 6/12, 6/18 vision before operation is now blind 47 months later, but he has no symptoms of raised intracranial pressure. Another with 1/60, 6/36 is almost blind 14 months later and a third with 6/12 and light perception now has 6/60 and nil 2 months after operation. All have secondary optic atrophy. The history of the last patient was of 3 years' duration and surely examination would have disclosed the papilloedema which must have been
present during most of this time. The history of the other 2 patients was brief and the papilloedema was severe on the first examination.

It appears from our results that although we are able to arrest hydrocephalus caused by aqueduct stenosis and so to relieve the symptoms of raised intracranial pressure, visual deterioration is unlikely to be reversed by a short-circuit operation and indeed may decline further with the appearance of secondary optic atrophy. The earlier the diagnosis can be made and treatment undertaken, the better will be the late results of surgery, for it has been shown that over three-quarters of the operations are successful so far as the short-circuiting procedure is concerned.

SUMMARY

A review of operative procedures and the results of operative treatment of patients with aqueduct stenosis is given.

The clinical picture of patients suffering from aqueduct stenosis is summarised in a series of 25 personal cases.

The ventriculographic appearances of the obstructed aqueduct are shown to be of 4 types.

The operation of ventriculocisternostomy as performed in this clinic is described, and the early and late results of this procedure are given for a consecutive series of 25 patients with non-neoplastic stenosis of the aqueduct. The operation is successful in nearly three-quarters of the patients.

REFERENCES

AQUEDUCT STENOSIS