Transient abducens paresis after shunting for hydrocephalus

Report of two cases

PETER McL. BLACK, M.D., PH.D., AND PAUL H. CHAPMAN, M.D.

Neurosurgical Service, Massachusetts General Hospital, Boston, Massachusetts

In two cases of normal pressure hydrocephalus, the patients suffered transient abducens weakness following a shunting procedure.

KEY WORDS • hydrocephalus • cerebrospinal fluid shunt • abducens nerve

Shunting for hydrocephalus is a procedure with a significant rate of complications. We report here two cases of transient sixth nerve weakness following shunt placement.

Case Reports

Case 1

This 70-year-old woman, otherwise in good health and without a previous history of extraocular movement difficulty, presented to the neurosurgical service with a 9-month history of intermittent gait difficulty and increasing memory loss. Her neurological examination on admission demonstrated difficulty with retention of newly presented material and unsteadiness on tandem gait or sudden turning. Computerized tomography (CT) scanning demonstrated enlarged ventricles with little cortical sulcal enlargement. Isotope cisternography demonstrated ventricular stasis and no ascent of radionuclide over the convexities at 48 hours. The lumbar puncture opening pressure was 145 mm H2O and the cerebrospinal fluid (CSF) was acellular. A ventriculoatrial shunt was placed without apparent difficulty, using a medium-pressure Hakim valve with a pretested closing pressure of 70 mm H2O.

On the third postoperative day, the patient complained of difficulty in hearing, first in the right ear and then in the left. She also had persistent nausea and headache and pain in the right side of the face, teeth, and forehead. Cranial nerves I, II, III, IV, VIII, IX, X, XI, and XII were normal. An audiogram showed a 20 dB hearing loss bilaterally. The facial pain and nausea disappeared after 3 days. On the 7th postoperative day, she complained of diplopia, and over the next days developed a complete right sixth nerve palsy.

A lumbar puncture on the 8th postoperative day showed an opening pressure of 55 mm H2O with 14 lymphocytes and one polymorphonuclear cell per high-power field. The CSF protein was 24 mg/ml. The patient's sixth nerve palsy worsened after the lumbar puncture, but began to improve at 2 weeks and was gone at 2 months. Her memory improved with shunting, and her gait became more normal. Her hearing loss subjectively improved but not to what she considered normal.

Case 2

This 72-year-old woman was referred to the neurosurgical service with a history of progressively increasing gait disturbance over the previous 8 months. The abnormality, described as “shuffling” and loss of equilibrium, had resulted in repeated falls. During the most recent few months, her family had noted an additional mild forgetfulness and occasional confusion. There was no history of urinary incontinence. Her general health was good. Initial evaluation at the referring hospital included CT scan, which showed moderately severe symmetrical ventricular enlargement without sulcal prominence. On isotope cisternography, there was stasis of radionuclide in the basilar and Sylvian cisterns at 48 hours.

On admission to the Massachusetts General Hospital, the patient had a notable gait disturbance, with
short, shuffling steps. Muscular strength, tone, and reflexes were normal. There was a mild disturbance of immediate recall, but no abnormality of recent or remote memory. The remainder of her neurological examination, including cranial nerve testing, was normal. Subsequently, a right ventriculoatrial shunt procedure was performed using a Hakim medium-pressure valve with a pretested closing pressure of 90 mm H\textsubscript{2}O.

In the ensuing days, as she began to walk, the patient noted improvement in her gait, but complained of severe postural frontal headache with nausea. This persisted. Eight days postoperatively, she began also to complain of diplopia, initially on looking to the right but finally to either side. On examination, she was noted to have bilateral incomplete sixth nerve paresis. A CT scan on the 8th postoperative day showed that the ventricles were now only slightly enlarged and revealed a prominent sulcal pattern. There was no evidence of subdural hematoma. The abducens palsies persisted, along with her postural headache, necessitating shunt revision on the 12th postoperative day. At that time, the valve was replaced with a high-pressure Hakim valve with a closing pressure of 140 mm H\textsubscript{2}O. In addition, an antisiphon device was inserted. This relieved the patient's headache but had no immediate effect on her diplopia. At 8 weeks, there was a persistent mild left abducens palsy, although her neurological examination was otherwise normal. By 12 weeks her diplopia had resolved. She remains well to this time.

Discussion

The abducens or sixth cranial nerve is well known for its sensitivity to intracranial pressure. This is partly a result of its anatomical course.\textsuperscript{9} After leaving the brain stem at the pontomedullary junction, it runs upward, forward, and laterally in the pontine cistern. The anterior inferior cerebellar artery and labyrinthine artery clasp it as it travels lateral to the basilar artery. It pierces the dura beside the dorsum sellae and then bends sharply forward, crossing the superior border of the petrous portion of the temporal bone close to its apex. There it usually runs beneath the petroclinoid ligament, although occasionally the nerve divides into bundles that pass above and below the ligament.\textsuperscript{9} It then enters the cavernous sinus and exits from the skull through the superior orbital fissure. With increased intracranial pressure, it has been suggested that the abducens nerve becomes compressed between the pons and the basilar artery or is stretched along the sharp edge of the petrous temporal bone.\textsuperscript{11}

It appears that similar stretching can occur with a low-pressure state. Certain anatomical variations may predispose to this, including large basilar artery branches, a sharp edge in the dural entry site, a high-lying petrous apex, or a nerve bundle that runs above the petroclinoid ligament. Asymmetry in these variations may explain why the palsy may be unilateral. Lumbar puncture is one manipulation that may give chronically low CSF pressure, and abducens palsy has been reported after it; this accompanies the headache and nausea that are generally part of the low-pressure syndrome.\textsuperscript{3,7,10} It is the meningeal penetration that presumably leads to abducens weakness after spinal anesthetic or myelography. Thorsen\textsuperscript{16} estimated that abducens palsy occurs in one out of 400 cases after spinal block anesthesia; in his cases, three out of four palsies were unilateral and most had disappeared at 6 weeks. Seyfert and Mager\textsuperscript{14} presented five patients with abducens palsy following the use of a watersoluble myelography medium; they proposed that both a low-pressure state and a toxic factor were involved. Emphasizing the severely low pressure that can occur with persistent leakage is a recent case of chronic subdural hematoma after lumbar puncture.\textsuperscript{1}

Cerebrospinal fluid shunting has repeatedly been shown to result in a low-pressure condition. McCullough and Fox\textsuperscript{8} have documented the negative intracranial pressure that can follow this procedure, and subdural hematomas are well established sequelae of shunting.\textsuperscript{6,8,12}

In the two cases presented here, it appears that the abducens paresis was a result of low pressure. The time course was similar to the low-pressure syndrome that follows spinal anesthesia: both patients had headache, there was no evidence of subdural collection on CT, and, in one patient, replacement of a valve by one with a higher pressure relieved the symptoms. In the first patient, the possibility of unilateral involvement of the trigeminal and acoustic nerves might also suggest torsion through low pressure. Although our two patients both had normal-pressure hydrocephalus, this complication can undoubtedly occur regardless of the cause of hydrocephalus.

References

4. Ignelzi RJ, Kirsch WM: Follow-up analysis of ven-
Abducens palsy following CSF shunt


Manuscript received April 30, 1981.
Address reprint requests to: Peter McL. Black, M.D., Neurosurgical Service, Massachusetts General Hospital, Boston, Massachusetts 02114.