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MR of Postoperative Syringomyelia

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Twenty-seven MR scans of 20 patients surgically treated for syringomyelia were reviewed. Thirteen patients had syringomyelia associated with the Chiari I malformation, four cases were posttraumatic, and three were idiopathic. The operations performed included syringosubarachnoid and syringoperitoneal shunts, myelotomies, and foramen magnum decompressions. Three of the foramen magnum decompressions had associated posterior fossa duroplasties, two had fourth-ventricle-to-subarachnoid shunts, and two had plugging at the obex. On 20 scans of patients in whom the syrinx cavity had been shunted, the shunt catheter was seen in 15 (75%). When adequately treated by shunting, syringes are completely collapsed and show no flow void. Nine patients were treated by foramen magnum decompression; all were well seen by MR. Three of these patients had a poor clinical result; these were the only patients in whom CSF was not seen between the foramen magnum and the neural structures of the posterior fossa on any images. The proposed mechanisms of syrinx formation and extension are discussed and related to the surgical procedures used to treat syringomyelia.

Syringomyelia is a disorder of the spinal cord characterized by the presence of fluid-filled cavities within the cord substance. When the cavity is a dilated central canal of the spinal cord, the term hydromyelia has been applied, reserving the term syringomyelia for cavities in the cord extending lateral to or independent of the central canal [1]. The term syringohydromyelia reflects this difficulty in classification [2]. Others have used the terms syringomyelia and syrinx in a general manner to represent any spinal cord cyst [3], and we shall use these terms in the same general sense in this paper.

The diagnosis of syringomyelia has been greatly facilitated by MR imaging [4–9]. The full extent of the syrinx and associated anomalies and lesions are readily and noninvasively demonstrated. The presence of a CSF flow void has been demonstrated in some syringes [10, 11].

There are many approaches to the treatment of syringomyelia [3, 12–24] that are based on the theories of pathogenesis [4, 12, 15, 25–29]. The purpose of this article is to review the various types of surgical treatments of syringes and their relation to the various proposed pathophysiologic mechanisms. The MR findings are correlated with the results of the surgery.

Materials and Methods

The MR scans of 20 patients with surgically treated syringomyelia were retrospectively evaluated for the location and appearance of shunt catheters, the presence or absence of a CSF flow void within the syrinx cavity, and change in flow void or size of the cavity from the preoperative examination (when one was present). In patients who had a foramen magnum decompression (FMD) instead of or in addition to syrinx shunting, the examinations were evaluated for the size of the CSF space at the new craniocervical junction, evidence of cerebellar compression, and a change in the appearance of the tonsils since the preoperative examination. Evidence of obex plugging or a fourth-ventricle-to-subarachnoid shunt (where

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present) or any change in the size or flow void in the syrinx from the preoperative examinations was also evaluated. The preoperative studies of all but two of these patients are included in a separate report and will not be discussed here.

Twenty-one examinations were performed on a 0.5-T whole-body superconductive magnet (Vista MR, Picker International) and six were performed on a 1.5-T, whole-body superconductive unit (Teslacon, Technicare Corp.). Standard, manufacturer-provided, single-echo, spin-echo sequences were obtained. Data were typically acquired with either 256 or 128 complex samples/view, 256 views, and four excitations using the two-dimensional Fourier transform (2DFT) method. A 256 × 256 or 128 × 256 matrix was employed. The field of view for these examinations was 30 cm for at least one sagittal sequence in all cases, although variable field diameters were also employed during many examinations. Surface coils were used in two examinations.

T2-weighted sequences were done with a TE of 60, 80, 100, or 120 msec and a TR of 1500–3000 msec. T1-weighted sequences used a TE of 30 or 40 msec with a TR of 500–800 msec. Intermediate sequences were occasionally employed. All patients were studied with T1-weighted sequences in the sagittal plane. All but two patients were studied with T2-weighted sequences. Section thickness was 5 mm for all sagittal images. Eight patients also had T1-weighted axial images for which section thickness was 10 mm.

Clinical information was obtained from patient records or from the referring physicians.

Results

Twenty-seven MR examinations were made of 20 patients who had been surgically treated for syringomyelia. Preoperative MR scans were available for comparison with 17 of the postoperative scans. The patient group was composed of 11 men and nine women ranging in age from 16 to 64 years; the average age was 43 years. Thirteen of the patients had communicating syringomyelia (associated with the Chiari I malformation); four were of traumatic origin, and three were classified as idiopathic. One patient was studied after each of three operations, a second patient was studied after each of five procedures, and a third patient after each of two procedures.

The syringosubarachnoid shunt (SSAS) was the most common procedure. Ten patients were treated with this shunt alone, while two had an FMD in association with an SSAS, one an FMD and SSAS associated with plugging at the obex, and one had an FMD and SSAS with a fourth-ventricular subarachnoid shunt. Six patients underwent syringoperitoneal shunting (SPS) alone, four had FMDs alone, one had a Gardner procedure (see discussion), one had a syringotomy, and one had a myelotomy. Three of the patients who had an FMD had a duroplasty at the foramen magnum as well.

Of the 20 studies of patients with either an SSAS or SPS, we were able to identify the shunt catheter in 15 (five of six with SPS and 10 of 14 with SSAS). In all cases, the appearance was that of a curvilinear lucency within the subarachnoid space, substance of the cord, or syrinx cavity (Fig. 1). We were unable to demonstrate the SP shunts after they exited the spinal canal in all cases but one, probably because of metal wires and sutures at the laminectomy site causing image distortion in the region where the shunt tube exited the canal. In addition, the relaxation times of the polyethylene shunt material and skeletal muscle are similar. The tube was easily seen within the cord on both T1- and T2-weighted images; it was seen more easily within the subarachnoid space on T2-weighted images where it was contrasted by the more intense signal of CSF. The outer surface of the cord bulged outward acutely at the site of shunt entry in all cases. As would be expected, the tube always entered the cord at the site of a laminectomy. In four cases, radiographs or CT scans were available to correlate with the MR images; the

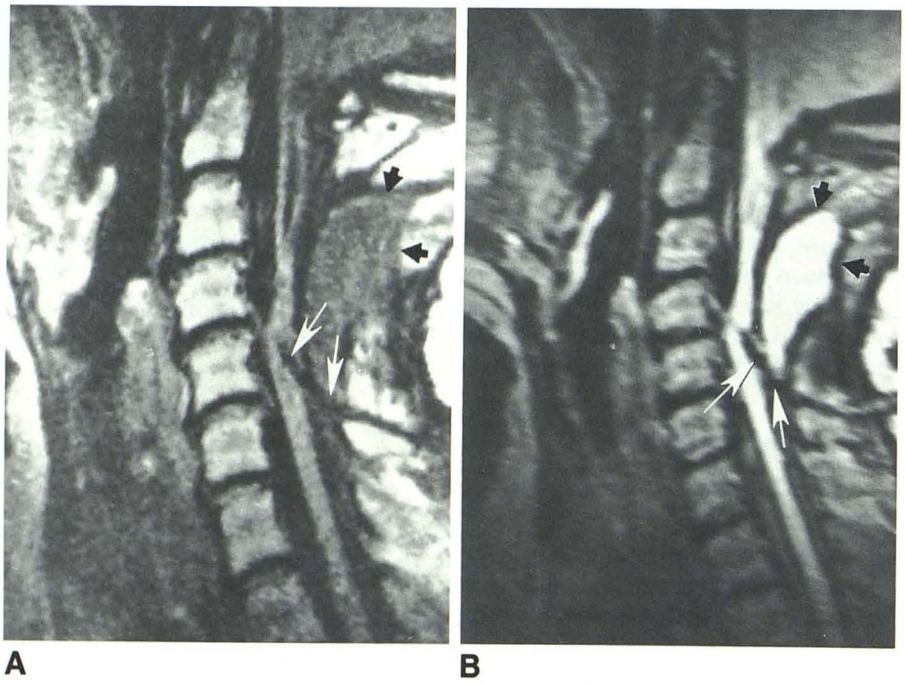

Fig. 1.—Collapsed syrinx after syringoperitoneal shunting.

A, SE 700/40. There is marked angulation of spinal cord at level of shunt tube. Shunt tubing (*white arrows*) is well seen within cord but not after it enters paraspinous soft tissues. Lobulated mass dorsal to canal (*black arrows*) is a postoperative pseudomeningocele (see text).

***B*, SE 2000/80. Shunt tubing (*white arrows*). Note increased intensity in psuedomeningocele (*black arrows*) compared with T1-weighted image (*A*).**

shunt position matched exactly on all studies. An attempt was made to differentiate patent from blocked shunts using the presence or absence of a flow void within the tube. No difference was seen between tubes that were clinically functional and those that were not.

The appearance of the craniocervical junction after FMD varied depending on whether a duroplasty was performed. In all cases, the foramen magnum was markedly widened. The new opisthion (defined as the bony posterior rim of the foramen magnum after FMD) varied from a gradually tapered to a flared bony prominence (Figs. 2 and 3). When a duroplasty was performed, there was a large amount of CSF-containing space inferior to the cerebellum, which remained above the level of the resected occiput. In three patients, the inferior cerebellum was lower than the new foramen magnum. None of these three patients had duroplasties, and in all the amount of CSF surrounding the cerebellum at the new foramen magnum was considerably less than in the other six patients. Specifically, there was no image in which CSF was seen between the cerebellum or medulla and the foramen magnum on either study; in one, the cerebellum appeared frankly indented by the bone of the foramen magnum (Fig. 4). These three patients remained symptomatic despite collapse of their syringes (see discussion).

Seven of the patients treated with FMD had the Chiari I malformation. In all seven, the cerebellar tonsils were of a normal, rounded shape postoperatively. Preoperatively scans were available for four of these patients and showed compressed, "pointed" tonsils in all cases.

Two patients had fourth-ventricular subarachnoid shunts, and two had pluggings at the obex; these were not identified by MR.

Those patients with both pre- and postoperative scans were evaluated for changes of syrinx size or of flow void within the syrinx. Eleven of 12 patients with syringes evaluated preoperatively with T2-weighted spin-echo sequences demonstrated a flow void within the syrinx (Fig. 5A). Postoperative scans demonstrated a flow void in 10 of 20 T2-weighted scans. In those patients having both pre- and postoperative T2-weighted scans, seven showed a smaller area of flow void or no flow void after surgery while five showed no change. All seven patients with a reduced flow void had a concomitant decrease in syrinx size (Fig. 5B), while little or no size reduction was appreciated in the patients in whom the flow void was unchanged. When the flow void was reduced, the reduction was most marked at the areas that were most completely collapsed. A lack of flow void postoperatively is thought to represent complete collapse of the

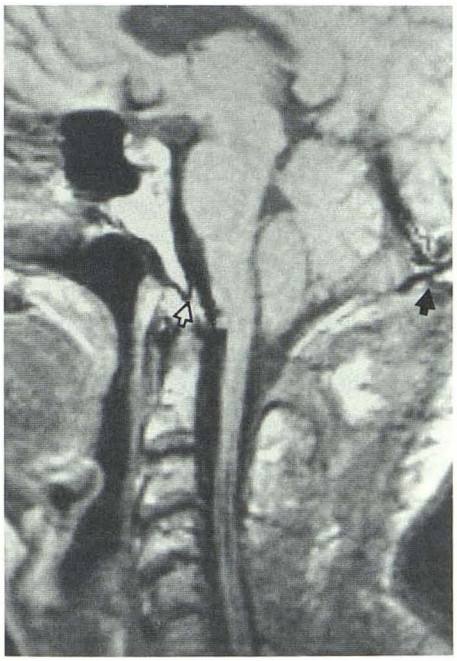

Fig. 2.—Foramen magnum decompression. SE 700/40 image shows a wide foramen magnum (FM) postoperatively with posterior lip of FM (*solid arrow*) located posterior to its normal position. There is CSF between basion (*open arrow*) at anterior FM and medulla.

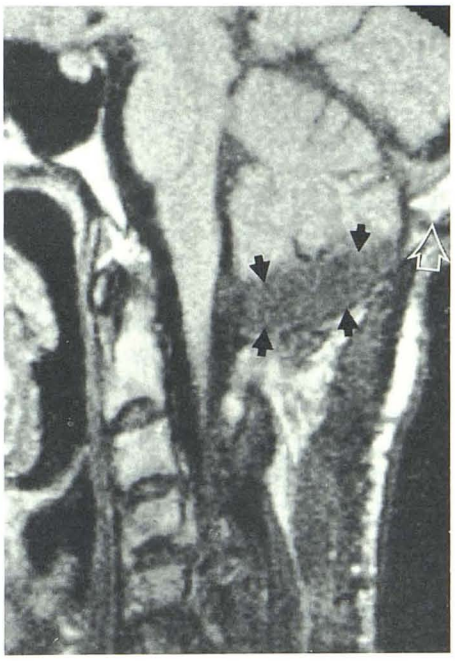

Fig. 3.—Foramen magnum decompression with duroplasty. SE 750/40 image shows increased CSF (*arrows*) between inferior cerebellum and soft tissues of upper neck as compared with Fig. 2. This patient also has some flaring of the occipital bone at margin of occipital craniectomy (*open arrow*), which was of no clinical significance.

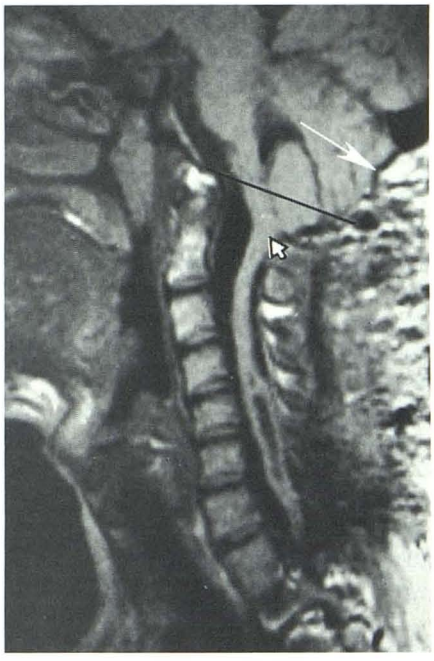

Fig. 4.—Foramen magnum decompression with "slumping" of cerebellum. SE 500/40 image reveals inferior displacement of cerebellum through surgically enlarged foramen magnum (FM). Original plane of FM is indicated by *solid line*. There is cervicomedullary kinking (*small arrow*). Cerebellum is indented by occipital bone posteriorly (*long arrow*). Residual syrinx cavity is seen in lower cervical spine but this was markedly reduced in size compared with preoperative study (not shown).

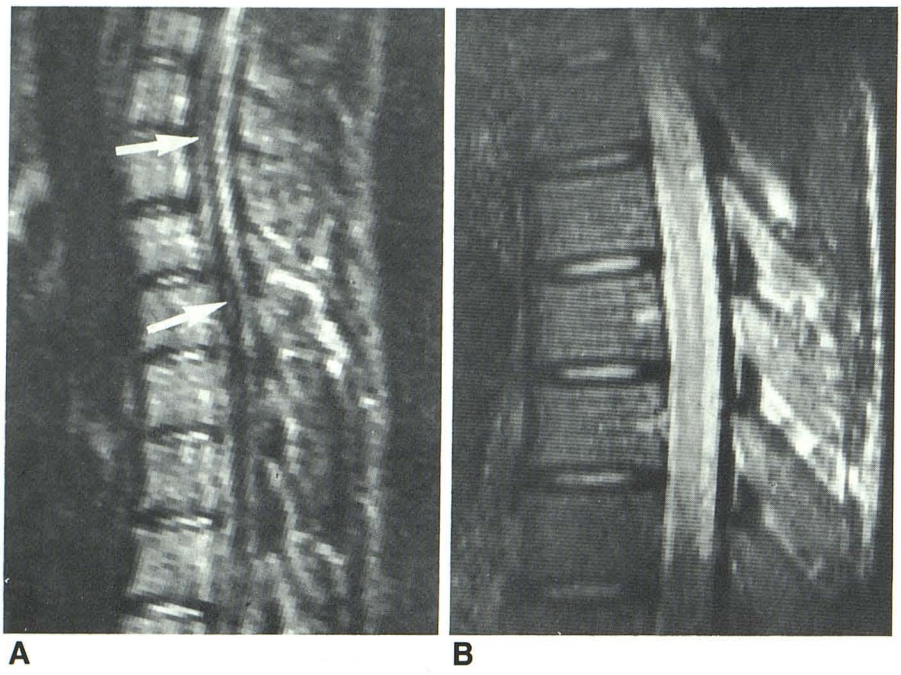

Fig. 5.—Postoperative cervicothoracic syrinx.
***A*, Body coil SE 2000/100 image obtained after syringosubarachnoid shunting reveals residual syrinx cavity (*arrows*) with a flow void.**
***B*, Surface coil 2000/100 image obtained after Gardner's surgical procedure (see text) demonstrates complete collapse of syrinx and absence of a flow void.**

syrinx such that pulsatile movements of the fluid are no longer possible.

One of three patients with SSA shunts had a decrease in syrinx size and flow void postoperatively. This was seen in two of four patients with SP shunts, two of two patients with FMD and SSAS, and in the one patient with the Gardner procedure. The patient treated with syringotomy alone showed no change postoperatively.

One patient with a traumatic syrinx in the cervicothoracic region had four shunting procedures over an 18-month period that failed to halt a progressive cranial extension of enlarging cord. Initially, no flow void was seen in the syrinx; in its absence, no extension of the cord widening was seen over a 4-month period. A flow void subsequently developed in an area that was previously bright on the T2-weighted images. At the same time, cranial extension of the widened cord was demonstrated. This newly expanded area was bright on the T2-weighted images: no flow void was seen. An extensive myelotomy at this time showed multiple loculations of CSF separated by gliotic bands in the region of long T2. Repeat MR after lysis of these bands revealed a continuous, tubular syrinx cavity with a large flow void (Fig. 6).

Three patients had postoperative fluid collections in the paraspinous soft tissues at the site of surgery. Two were in the cervicothoracic region, associated with lower cervical laminectomies; the third was at the posterior craniocervical junction in association with an FMD. In all cases, these fluid collections were sharply defined, lobulated collections that exhibited a signal intensity similar to CSF on T1-weighted images and a very intense signal (brighter than CSF) on T2-weighted images. In none of these cases did the fluid collections cause any mass effect on the spinal canal. We hypothesize that these postoperative fluid collections are pseudomeningoceles with a high protein concentration, possibly from the surgically traumatized surrounding muscle. The protein may cause some shortening of the T1 of this aqueous solution, which is manifested on the long TR images as suggested by Brant-Zawadzki et al. [30].

Pathogenesis of Syringomyelia as It Relates to Therapy

To understand the different operations used to treat syringomyelia, it is necessary to understand the theories of pathogenesis of this entity. Early authors described the cells in the walls of a syrinx cavity as being of embryonal character [31]. It was thought that these abnormal cells may be neoplastic, secreting fluid into the canal and thereby forming a cavity. Irradiation was proposed to eradicate the abnormal cells and thereby stabilize the cavity [3]. The treatment was minimally successful, the successes possibly being secondary to induction of fibrosis in the cord surrounding the syrinx, thereby making extension of the cavity more mechanically difficult.

Gardner [12, 25] proposed a unifying theory for all forms of dysraphism. He stated that the underlying problem is a failure of perforation of the roof of the rhombencephalon. This tissue layer, being permeable to CSF, permits diffusion of CSF from the fourth ventricle into the cisterna magna but does not transmit pressure waves from pulsations of the choroid plexus. These pulsations, therefore, drive CSF through the calamus scriptorius into the central canal of the spinal cord, causing hydromyelia (Fig. 7A). The lateral ventricular hydrocephalus in the fetus (from the blocked outlet foramina) is thought to cause a more inferior position of the transverse sinuses and tentorium, resulting in a small posterior fossa with tonsillar and/or vermian herniation through the foramen magnum. According to Gardner, cerebellar herniation interferes with the escape of the ventricular fluid pulse wave

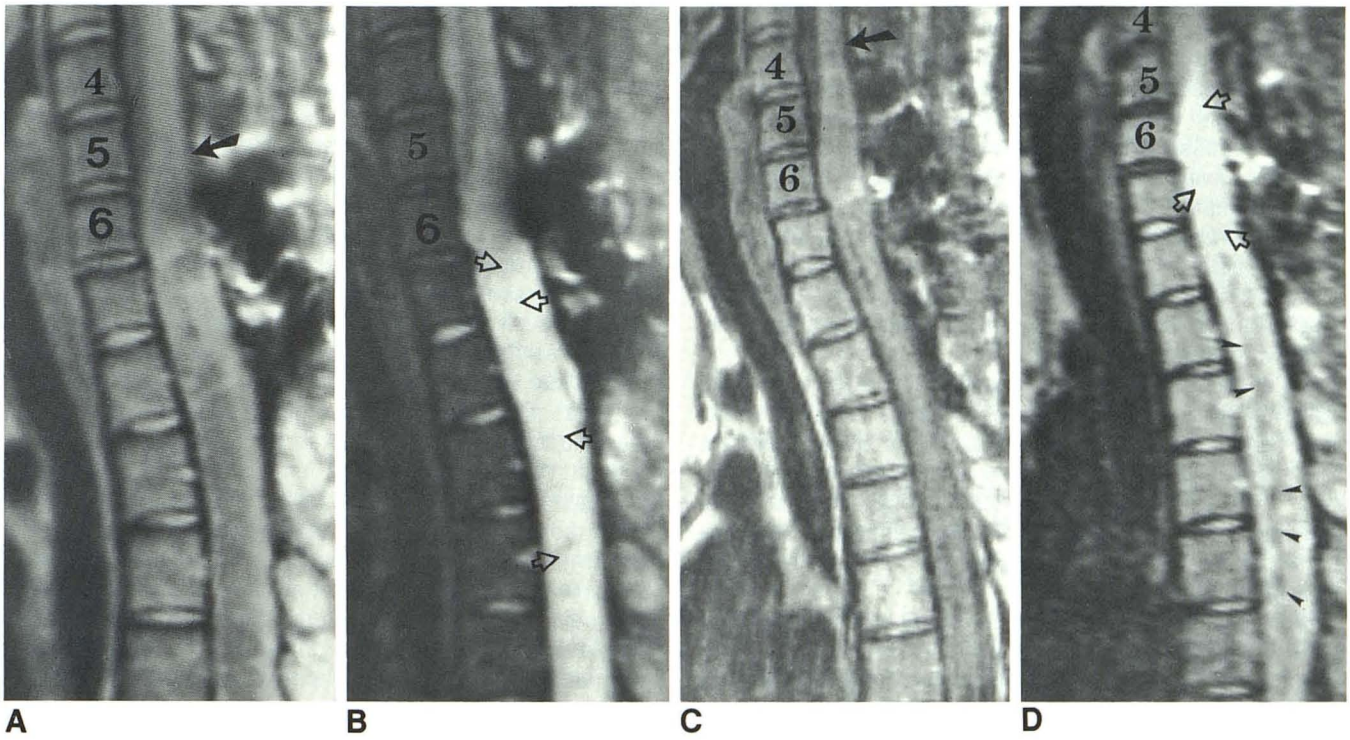

Fig. 6.—Extension of a posttraumatic syrinx. (*C* and *D* were obtained 3 months after *A* and *B*).
A, SE 2000/25. Syrinx cavity in enlarged cord extends to inferior C5 level (*arrow*). Cavity is irregular rostrally. Enlarged spinal cord fills spinal canal.
B, SE 2000/70. Note increased intensity in syrinx (*arrows*).
C, SE 500/40. Cord enlargement has progressed up to mid-C4 level (*arrow*).
D, SE 2000/80. Newly expanded area of cord has high-intensity signal (*open arrows*). Flow void is now present in syrinx (*arrowheads*). At surgery, multiple loculated cavities were present. Gliosis and small cysts were present in rostral area of increased signal.

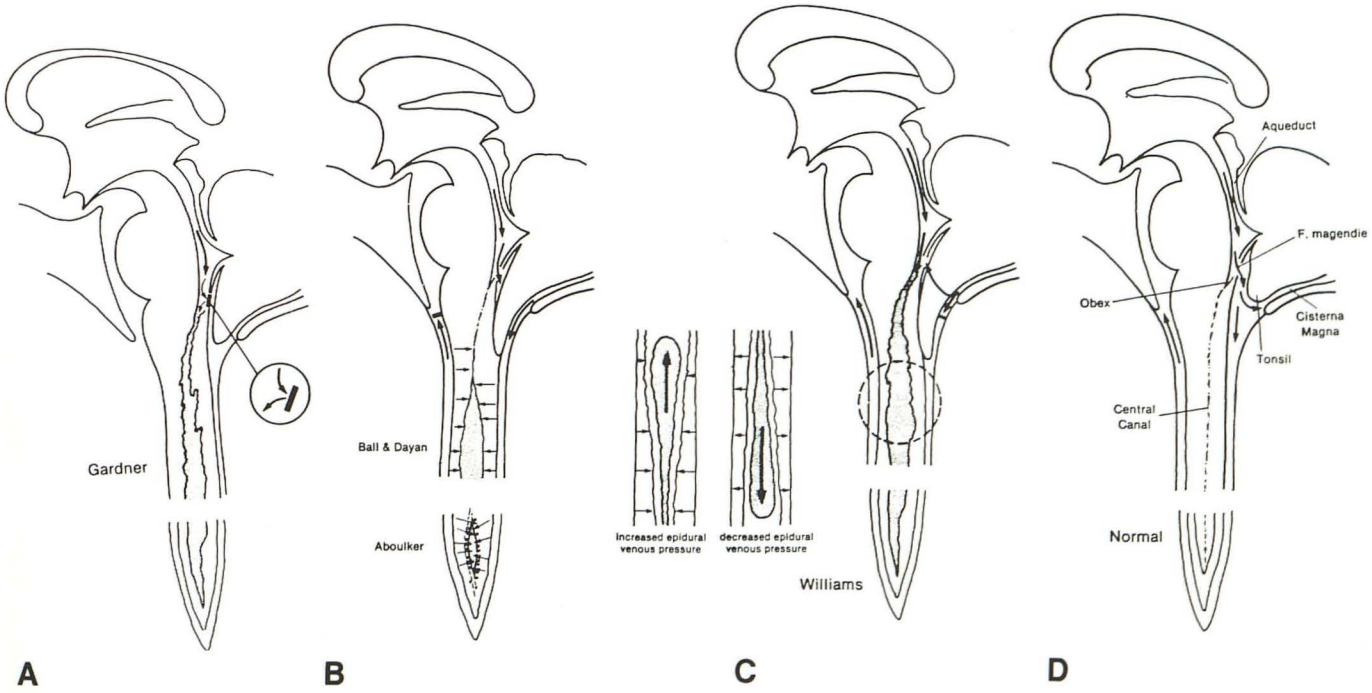

Fig. 7.—Diagrams representing various theories of syrinx development. See text for full explanation.
A, Gardner's theory emphasizes block at foramen of Magendie.
B, In Ball and Dayan's theory, CSF passes into central canal of cord via Virchow-Robin spaces. Aboulker believes CSF is forced into spinal cord but remains separate from central canal.
C, Williams' theories emphasize ball-valve effect at foramen magnum and "sloshing" of syrinx fluid.
D, Normal diagram for comparison.

and creates a subarachnoid block; both these factors exaggerate the pulse wave and direct it into the central canal. Gardner's operation was designed to decompress the foramen magnum via suboccipital craniectomy and relieve the outlet obstructions by surgically creating fourth-ventricular outlet foramina (by perforating any obstructing membrane) and inserting a fourth-ventricular-to-subarachnoid shunt. The calamus scriptorius is then occluded at the obex with a cone-shaped Dacron or muscle plug. Additionally, many surgeons now perform a posterior fossa duroplasty using Dacron or pericranium to enlarge the volume of the posterior fossa. This is known as Gardner's procedure.

Ball and Dayan [26] have theorized that CSF enters the central canal via the perivascular spaces of Virchow-Robin. They believe that increased pressure in the spinal subarachnoid space is generated by a foramen magnum lesion acting as a one-way valve, allowing CSF egress from the basal cisterns into the spinal canal but blocking its return. They feel that this increased pressure then drives CSF into the central canal (Fig. 7B).

Aboulker [28] proposed a theory somewhat analogous to Ball and Dayan. He referred to animal experiments [32] that demonstrate that 30% of CSF is produced in the central canal of the cord. Stenosis at the foramen magnum or elsewhere in the canal is thought to inhibit CSF flow toward intracranial areas of resorption and cause increased spinal CSF pressure. CSF, driven by high intraspinal pressure, then filters into the spinal cord, either through the parenchyma or along the posterior roots. This longstanding spinal cord edema may eventually cause cavitation within the cord parenchyma (Fig. 7B).

Williams [24, 29] believes that the valvular action at the foramen magnum works in the opposite direction, allowing fluid to move upward but not downward. This pattern of fluid movement leads to relatively decreased pressure in the spinal canal when compared with the basilar cisterns, since fluid is accelerated upward by a milking action of the spinal canal by distended epidural veins (see below). This causes a craniospinal pressure dissociation. He proposed that this pressure differential also causes CSF to be "sucked" into the central canal of the cord, initiating the syrinx and causing downward extension of the cavity in communicating syringohydromyelia. He also proposed that syrinx cavities extend by "sloshing" of the syrinx fluid, this sloshing being secondary to engorgement of epidural veins by increased thoracoabdominal pressure. These veins compress the subarachnoid space and spinal cord and force fluid upward within the syrinx cavity (Fig. 7C); if enough fluid is forced upward with sufficient force, the cavity will be extended. A rapid upward acceleration of contrast within syrinx cavities and the subarachnoid space has been demonstrated with coughing, valsalva, and respiration [29, 31, 33]. A downward acceleration of fluid after drainage of the veins relieves the compression could then lead to a caudal extension of the cavity.

Traumatic syringes may also extend by "suck" and "slosh," the pressure dissociation in this case being caused by canal narrowing secondary to either spondylosis or a fracture. The "slosh" wave would be generated in the same manner described above.

Williams believes that the two main goals of surgery are to relieve the "suck" and the "slosh." The FMD will eliminate the valvular action at that level and should relieve many of the aforementioned causes of syrinx formation or extension. Shunting of the syrinx should eliminate the "slosh" by leaving too little fluid to generate a wave of sufficient magnitude to extend the syrinx cavity.

Syringes are still occasionally treated by laminectomy and syrinx aspiration, the goal being elimination of the "slosh" effect. The site of puncture of the cavity might be identified as a focus of long T2 secondary to a small amount of glial reaction at the puncture site if scanner resolution were sufficient. More likely, any gliosis at the puncture site would be averaged with the glial tissues known to line syrinx cavities [1]. In fact, we saw no evidence of a syringotomy site in the patient treated in this manner.

Other attempts to eliminate the "slosh" effect include opening of the syrinx to either the subarachnoid space or areas of low pressure such as the pleural or peritoneal spaces using procedures such as myelotomy, myelotomy with an SSAS or SPS, and terminal ventriculostomy [3, 16–19, 22]. We were unable to identify any defect from a simple myelotomy in the one patient treated in this manner, probably for the same reason mentioned for the syringotomy. None of the patients examined in our series had a terminal ventriculostomy, but since this is essentially a myelotomy (at the conus medullaris), we would not expect to see the defect with MR.

Discussion

Ollivier [34] first gave the name syringomyelia to cysts within the spinal cord in 1827. Sixty-five years later, Abbe and Coley [35] described the first surgical treatment of this entity. In spite of nearly 100 years of surgical experience with this condition, syringomyelia remains a frustrating entity for the clinician; diagnosis has been difficult and an effective treatment has been elusive [3, 14, 18, 19, 22, 24, 36].

In the classic case, symptoms of syringomyelia begin in late adolescence or early adulthood and progress irregularly with long periods of stability. In traumatic syringomyelia, a latent period of 20–30 years is not uncommon before the onset of symptoms [37]. The clinical picture is variable, depending on the cross-sectional and vertical extent of cord destruction. Patients show segmental weakness and atrophy of the hands and arms with loss of tendon reflexes and segmental anesthesia of the dissociated type (loss of pain and temperature sense with preservation of the sense of touch) over the neck, shoulders, and arms [38]. Pain is often severe and may be most intense in an analgesic limb (anesthesia dolorosa). In practice, these findings are often unilateral, confined to the lower extremities [19, 22], or absent. Indeed, Schlesinger et al. [22] report 10% of 60 cases of syringomyelia to have a normal sensory examination, 10% with normal deep tendon reflexes, and 13.3% with normal motor strength.

The clinical course of syringomyelia is equally unpredictable. Anderson et al. [14] followed 44 patients with syringomyelia for a median interval of 10 years. Of the 24 who were treated surgically, eight (33%) showed long-term improve-

ment, while of the 20 managed conservatively, seven (35%) remained stable. Many authors have reported improvement in the symptoms of their patients after a variety of surgical procedures, including decompressive laminectomy alone or decompression laminectomy with needle aspiration of the syrinx [14, 16–23]. Aboulker [28], Logue and Edwards [19], and Levy et al. [18] note, however, that with long-term follow-up, many of these patients once again show sporadic deterioration. There are several possible explanations for this clinical deterioration. The initial improvement may be a placebo effect; the laminectomy may initially relieve pressure effects in a cord that is markedly expanded and ischemic due to the pressure and gliosis, as Netsky [44] has suggested, and eventually, extension of the syrinx due to the "slosh" effect may cause recurrent symptoms; or the syrinx may be adequately shunted initially but shunt malfunction then causes reaccumulation of fluid within the syrinx cavity that eventually precipitates recurrent symptoms.

Adding to this confusion is the association of foramen magnum lesions, such as Chiari I malformations, arachnoiditis, and foramen magnum tumors with syringomyelia. Levy et al. [18], Saez et al. [39], and Logue and Edwards [19] have pointed out the difficulty in differentiating symptoms of syringomyelia from those of compression of the brainstem due to the foramen magnum lesion. When symptoms persist after syrinx shunting, the cause may therefore not be clear. Moreover, Williams [24] has discussed the phenomenon of the cerebellum "slumping" into the enlarged foramen magnum after FMD, causing compression of the cerebellum and brainstem. Therefore, even after FMD, the cause of symptoms is not clear.

MR is now the procedure of choice for diagnosing syringomyelia [4–9]. Our experience indicates that it is equally valuable in evaluating syringes postoperatively. We were able to identify shunt catheters and their location within the syrinx in 15 of 20 cases where shunts were present (Fig. 1). Most of the cases in which the shunt was not identified were early in our experience, when imaging was done with body coils, decreasing the signal-to-noise ratio. In the near future, as slice thicknesses of less than 5 mm and smaller interslice "gaps" become available, visualization should become still better.

MR can visualize the syrinx cavity directly, demonstrating whether adequate drainage has been achieved. One patient, a 29-year-old man, was initially treated by an SSAS then by an SPS. Follow-up MR both times demonstrated a residual syrinx cavity with marked flow void, unchanged from the preoperative appearance. For his third operation, a Gardner procedure was performed. Follow-up at this time showed complete collapse of the syrinx cavity, which mirrored his clinical improvement (Fig. 5).

The presence or absence of a flow void within the syrinx cavity may be significant in evaluating response to therapy. Sherman and Citrin [40] have demonstrated that in regions of rapid CSF flow a lack of signal is noticeable on T2-weighted images. Rubin and Enzmann [11] demonstrated that this phenomenon can be generated by pulsatile flow using 2DFT pulse sequences. In view of the work of DuBoulay [33] and Williams [29] demonstrating the pulsatile motion of Myodil within syrinx cavities, it is reasonable to assume that the lack of signal seen within some syrinx cavities on T2-weighted images is due to this pulsatile flow. If one accepts Williams' theory of extension of syrinx cavities due to "sloshing," it would seem reasonable that a syrinx is not adequately treated until a flow void is no longer seen. Half (10 of 20) of the patients in our series demonstrated a flow void postoperatively. Although most of these patients are stable clinically at this time, the average follow-up time has been less than 1 year. Long-term follow-up on these and other patients will be the subject of a future report. The single patient in whom a flow void was not demonstrated preoperatively had multiple loculations and septations that presumably prevented CSF pulsations. It is interesting to correlate this case with both Aboulker's and Williams' theories on the pathogenesis and extension of syringes. The initial posttraumatic MR in this patient and a second scan 4 months later demonstrated an enlarged cord in the cervicothoracic region with a high signal intensity on the T2-weighted image extending upward to approximately the C5–C6 level (Fig. 6). SSASs, first at the T2 level and then at the C6 level, gave no clinical improvement. Three months later, another scan (done because of progression of symptoms) demonstrated a flow void in the upper thoracic-lower cervical area and a new area of cord widening and long T2 extending upward to the upper C4 level (Fig. 6). At this time, a myelotomy was done, which showed the gliosis and multiple loculations in the cervical cord. After the myelotomy, a continuous cavity with a large flow void was demonstrated on MR (not illustrated). The implication from these observations is that there was initial myelomalacia in the cord that subsequently underwent liquification, as Gebarski et al. [4] have reported. CSF pulsations within the syrinx (as demonstrated by the flow void) may have then traumatized the cord immediately superior to the cavity, causing edema and gliosis in this region, which then began to liquify and cavitate as well, a demonstration of the extension of a syrinx by MR.

The inability to differentiate between patent and blocked shunts by the presence or absence, respectively, of a flow void within the shunt was probably not surprising in view of the fact that the size of the tubing is approximately equal to the 5-mm slice thickness of the images. Most likely, volume averaging with the polyethylene walls of the tube obscured any signal from CSF within the tube. This is another area where thinner slices, when available, may be helpful.

MR was also extremely helpful in the evaluation of FMDs. These were easily recognized on sagittal sections through the craniocervical junction as a widened foramen magnum with truncation of the occipital bone posterior to the normal position of the opisthion (Fig. 2). In all cases but one, the new opisthion was tapered in appearance but in one case it appeared forked, or flared (Fig. 2B). This patient manifested no symptoms referable to this flaring. In the six patients who were clinically stable and symptomatically improved after FMD, the syrinx was collapsed and there was CSF seen between the bone of the foramen magnum and the cerebellum or the brainstem on at least one sagittal MR image. Two patients who remained symptomatic or showed progression of symptoms after FMD also had completely collapsed syringes, and the third had a syrinx markedly diminished in size

but the cerebellum and brainstem were abutting the bony foramen magnum on every image. Moreover, in one patient, the cerebellar hemisphere was frankly impressed upon by the occipital bone (Fig. 4). We believe that these cases are examples of "slumping" of the cerebellum into the foramen magnum as described by Williams [24], causing compression of the posterior fossa structures.

FMDs are frequently accompanied by a duroplasty, in which a piece of Dacron or dura is sutured into the divided dura surrounding the cerebellum in order to enlarge the volume of the posterior fossa. Patients who had undergone duroplasty were easily recognized on MR by the large collection of fluid inferior to the cerebellum (Fig. 3). This fluid collection is differentiated from a pseudomeningocele by the signal intensity, which is identical to CSF on both T1- and T2-weighted images.

The shape of the cerebellar tonsils was evaluated in the nine patients who underwent FMD. All had tonsils with a normal, rounded configuration postoperatively, including the two patients with a worsening clinical picture. Scans of the four patients who had both pre- and postoperative MR demonstrated a change in the tonsillar shape from the "pointed" tonsils characteristic of the Chiari I malformation to the rounded shape. This was noted in a previous article [41] with the hypothesis that the change in configuration represented alleviation of the tonsillar compression. The present findings support this hypothesis; in the symptomatic patients with "slumping" of the posterior fossa contents, the compression is now at a higher level of the cerebellum.

Our inability to identify "plugs" at the obex may reflect the similar MR signal characteristics of the muscle tissue used for the plug and the surrounding brain, combined with the extremely small size of the structures involved. Moreover, the muscle plugs are known to migrate from the obex area with time [3, 24], thus making their detection nearly impossible. The inability to detect the fourth-ventricular subarachnoid shunts in the two patients so treated who were scanned is more likely secondary to the small number studied and our consequent inexperience with their normal appearance. In one of the two patients, a curvilinear area of low intensity was seen superimposed upon the tonsil in the midline image, which may have represented the shunt. With further experience and advances in MR technology, detection of these should be facilitated.

A group of fluid collections that developed postoperatively in proximity to the surgical site have not been verified surgically, but they are identical in appearance to proven postoperative pseudomeningoceles that we have observed in other patients after spinal surgery. The lobulated, sharply defined appearance in the surgical bed is characteristic of these collections. An explanation for the isointense signal on T1-weighted images (relative to CSF) and hyperintense signal on T2-weighted images was provided by Brandt-Zawadzki, et al. [30], who noted these characteristics in proteinaceous aqueous fluid collections. They reasoned that the T1 of water is so long that slight shortening of the T1 of water protons (as is known to occur in protein solutions) [42, 43] could not be detected on short TR spin-echo sequences. Only when TRs in the range of 2 sec or greater are used will these effects be seen. We therefore hypothesize that protein from the surrounding soft tissues, traumatized by surgery, accumulates within the pseudomeningocele and shortens the T1 of the aqueous protons. This leads to the bright signal on the long TR image.

In summary, MR is extremely valuable in the evaluation of surgically treated syringomyelia: (A) Shunt tubes can be seen and their location within the syrinx cavity and subarachnoid space accurately determined in most cases. (B) The most important factors in the evaluation of treated syringes are diminution of syrinx size and elimination of the flow void within the cavity. (C) If clinical symptoms persist despite adequate shunting of the syrinx by MR criteria, the foramen magnum region should be evaluated to look for compression at that level. (D) After FMD, CSF should be visible between the foramen magnum and the neural structures of the posterior fossa on at least one sagittal image. If none is seen, "slumping" of the posterior fossa elements into the foramen magnum should be suspected.

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