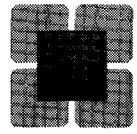

Gardner's hydrodynamic theory of syringomyelia revisited

PREM K. PILLAY, MD; ISSAM A. AWAD, MD, MSc; JOSEPH F. HAHN, MD

■ Several theories have been put forth to explain the pathogenesis of syringomyelia, the formation of longitudinal, fluid-filled cavities within the spinal cord. Chief among them is Gardner's hydrodynamic theory, widely accepted for more than two decades. Gardner attributed the genesis of syringomyelia to craniospinal pressure differentials in the setting of fourth ventricular outlet obstruction; these differentials favor cerebrospinal fluid shifts from the fourth ventricle of the brain through the central canal of the spinal cord. Gardner's theory has been questioned, and several alternative theories of syringomyelia have been proposed. Physiological data and new information from magnetic resonance imaging support many of Gardner's concepts; however, a more comprehensive elucidation of the pathophysiologic mechanisms of syringomyelia requires incorporating facets of the other theories. We propose a unified theory of the pathogenesis of syringomyelia based on recent experience with magnetic resonance imaging, and on elements of other current theories.

□ INDEX TERMS: SYRINGOMYELIA; MAGNETIC RESONANCE IMAGING; CHIARI MALFORMATION; CEREBROSPINAL FLUID PRESSURE
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THE PRESENCE of longitudinal fluid-filled cavities within the spinal cord, or syringomyelia,[1] is an anomaly seen in a number of neurologic conditions, including the Chiari malformation (congenital crowding of the posterior fossa and chronic tonsillar herniation through the foramen magnum), spinal cord trauma, neoplasms, and arachnoiditis; it also may be idiopathic.[2–4]

Several explanations for the occurrence of syringomyelia have been put forth, chief among them Gardner's hydrodynamic theory, formulated by Gardner* et al on the basis of clinical experience[6–14] and widely accepted for over two decades as the leading explanation. In recent years several other theories have reached prominence, including those of Williams, Ball and Dayan, and Aboulker.[15–20] We review these theories in the light of current physiological evidence and our experience with magnetic resonance imaging (MRI) in patients with syringomyelia, and we offer a new, unified theory synthesizing MRI observations and elements of other current theories.

From the Department of Neurological Surgery, The Cleveland Clinic Foundation.

Address reprint requests to I.A.A., Department of Neurological Surgery, The Cleveland Clinic Foundation, S80, 9500 Euclid Avenue, Cleveland, OH 44195.

* W. James Gardner, a pioneer of modern neurosurgery and founder of the Department of Neurological Surgery at The Cleveland Clinic Foundation, maintained a lifelong interest in the etiology and treatment of syringomyelia. He was also the inventor of such devices as the Gardner cervical tongs, the pneumatic suit to maintain blood pressure, the alternating air pressure mattress for the prevention of bed sores, and the pneumatic splint for fractures.[5]

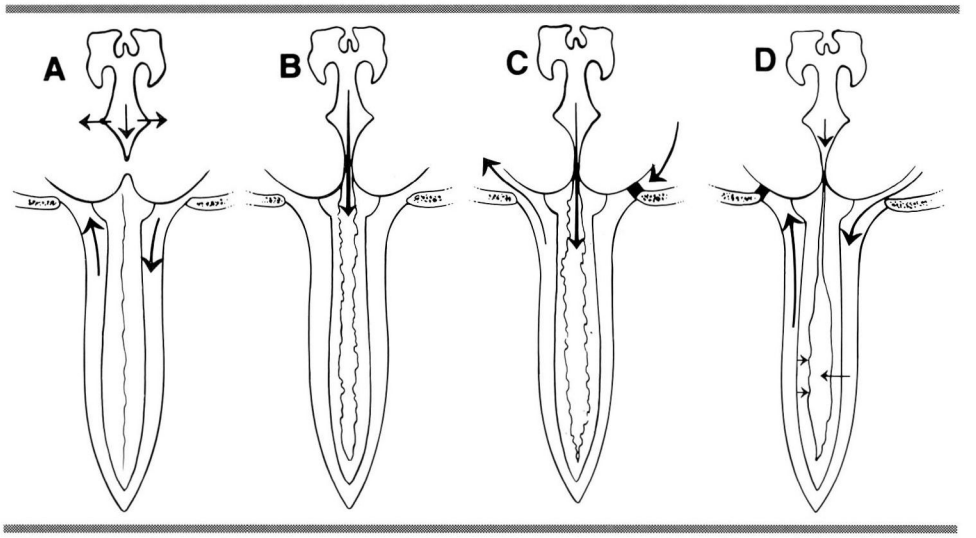

FIGURE 1. Theories of syringomyelia. *A*, normal cerebrospinal fluid dynamics, with small arrows showing free flow of cerebrospinal fluid through outlets of the fourth ventricle, and large arrows indicating free flow around the craniocervical junction. *B*, Gardner's hydrodynamic theory, in which obstruction of the fourth ventricular outlet channels the cerebrospinal fluid pulse wave through the obex into the central canal of the spinal cord, creating a syrinx. *C*, Williams' theory, in which foramen magnum obstruction leads to development of a craniospinal pressure gradient, causing hindbrain impaction and syrinx formation. *D*, the theories of Ball and Dayan and Aboulker, in which foramen magnum pathology leads to increased intraspinal pressure, forcing fluid into the spinal cord through the Virchow-Robin spaces or nerve roots.

GARDNER'S HYDRODYNAMIC THEORY

Gardner's hydrodynamic theory of the pathophysiology of syringomyelia and other dysraphic conditions[14] was based on several observations. In normal embryonic development, hydrodynamic mechanisms are in play, particularly in the expansion of the neural tube. Bering showed that the choroid plexus is the chief site of transfer of arterial pulsations to the ventricular fluid.[21] With each systolic pulse, a pressure gradient is transmitted throughout the cerebrospinal fluid (CSF) system that tends to force fluid out of the ventricles. This was recently confirmed by intraoperative sonographic studies in man.[22] Bering also demonstrated in hydrocephalic dogs that unilateral choroid plexectomy causes the ipsilateral ventricle to shrink while the other continues to enlarge (the choroid plexus is responsible for a large proportion of CSF secretion).[23] In other animal studies, McLaurin showed that blockage of the fourth ventricular outlets with intracisternal kaolin is associated with hydrocephalus and syringomyelia.[24] Becker et al demonstrated that syringomyelia does not develop with kaolin hydrocephalus if the central canal is occluded at the obex.[25]

Gardner suggested that the Bering effect, with its unvalved, pump-like action, not only was responsible for the embryonic development of the subarachnoid pathways, but also helped to determine the shape of the developing brain. He proposed that the balance between the pulsatile lateral ventricular choroid plexus and the fourth ventricular choroid plexus controls developmental events such as the migration of the tentorium. Gardner noted that overactivity of the anterior choroid plexus can cause a small posterior fossa and the Chiari malformation. He reasoned that if the posterior choroid plexus is overactive, then the tentorium was too high, and additional space was taken up by the ballooning rhombic roof (which may have failed to perforate), and the Dandy-Walker deformity was the consequence. He also noted that with late or inadequate rupture of the rhombic roof, the physiological outlets of the fourth ventricle (foramina of Luschka and Magendie) remained functionally closed. The pulsatile flow of CSF would then be directed through a patent obex, with a "water-hammer" pulse effect causing dilatation of the central canal and syringomyelia (*Figure 1*, *A* and *B*).[14]

WILLIAMS' THEORY

Williams concurs with Gardner that the mechanisms that force fluid into the central regions of the spinal cord are most likely related to hydrodynamic phenomena, and his theory further refines those concepts.[15] He suggested that a "suck" phenomenon is a major factor in hindbrain herniation and syringomyelia.

Using data from manometric pressure measurements, Williams demonstrated craniospinal pressure differentials in patients with hindbrain anomalies (*Figure 2*).[16] He interpreted these data to suggest that Valsalva maneuvers (associated with straining, coughing,

and sneezing) result in epidural venous engorgement. This would cause the intraspinal pressure to rise, compressing the spinal subarachnoid space and generating a pressure wave in both cranial and caudal directions. The upward pressure wave would enter the intracranial compartment with little resistance. However, when the spinal venous pressure returns to normal, flow out of the intracranial compartment would be delayed by adhesions or hindbrain herniation, leading to the observed post-Valsalva rebound that produces a craniospinal pressure differential of up to 100 mm Hg. This one-way valve effect of severe hindbrain herniation is particularly marked in babies with advanced Chiari malformation. As pointed out by Graftjik, pressure on the head of such babies will not inflate the meningocele, whereas pressure on the meningocele can inflate the head.[26] The pressure differential not only would promote further impaction of the hindbrain through the foramen magnum, but also would suck fluid into low-pressure areas of the spinal cord, causing fluid to build up in syringes (*Figure 1, C*).

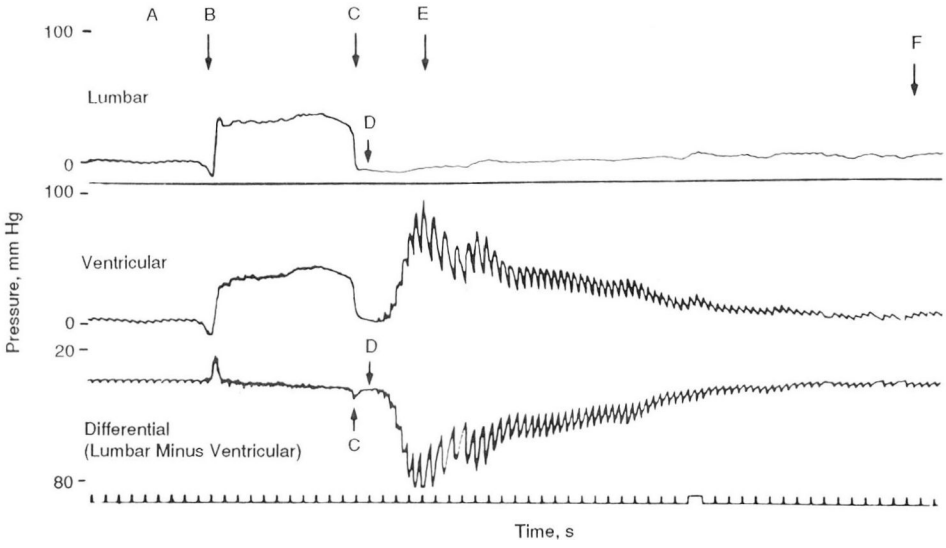

FIGURE 2. Demonstration of the suck phenomenon. Intraventricular and intraspinal pressures were measured in the sitting position. At *A*, the patient is at rest and the craniospinal differential (bottom) is zero. At *B*, the patient initiates a Valsalva maneuver; the brief upward deflection in the differential trace indicates a transient obstruction. At *C*, the Valsalva maneuver is stopped, and there is a contrary downward deflection (*D*). At *E*, the post-Valsalva rebound produces a peak pressure differential of about 100 mm Hg between the head and the spine before restabilization (*F*). Reproduced from *Neurol Res* (1986;8:130–145), with permission.[15]

Williams also introduced a second concept of "slosh" to explain the propagation of syringes within the spinal cord.[15,17,18] It refers to a form of rapid impulsive fluid movement within the spinal cord caused by the epidural venous engorgement that occurs with a Valsalva maneuver. This fluid movement may not only propagate the syrinx craniocaudally along the central canal, but may dissect new channels into the spinal cord parenchyma.

OTHER THEORIES OF SYRINGOMYELIA

Ball and Dayan[19] proposed that syringomyelia may be produced by CSF under pressure entering into the spinal cord from the subarachnoid space along Virchow-Robin spaces (which surround parenchymal vessels and are filled with extracellular fluid). Ball noted that, in autopsies of patients with syringomyelia, small arteries and veins were prominent in the walls of the syrinx. Microscopic examination revealed dilated perivascular spaces. Injection of India ink into the lumen of the syrinx followed by serial sectioning of the walls demonstrated that the ink spread centrifugally from the lumen along the Virchow-Robin spaces. Ball and Dayan proposed that during sudden increases of thoracoabdominal pressure that occur with coughing and straining, fluid forces generated due to foramen magnum obstruction (as proposed by Williams) could cause CSF to enter the spinal cord through the Virchow-Robin spaces. Small pools of extracellular fluid within the spinal cord would then eventually coalesce to form a syrinx (*Figure 1, D*).

Aboulker's theory[20] is similar to Ball and Dayan's. On the basis of animal experiments, he believed that up to 30% of CSF is produced in the spinal cord compartment. Stenosis at the foramen magnum or elsewhere along the cord would increase spinal CSF pressure. This force, perhaps aggravated by sudden

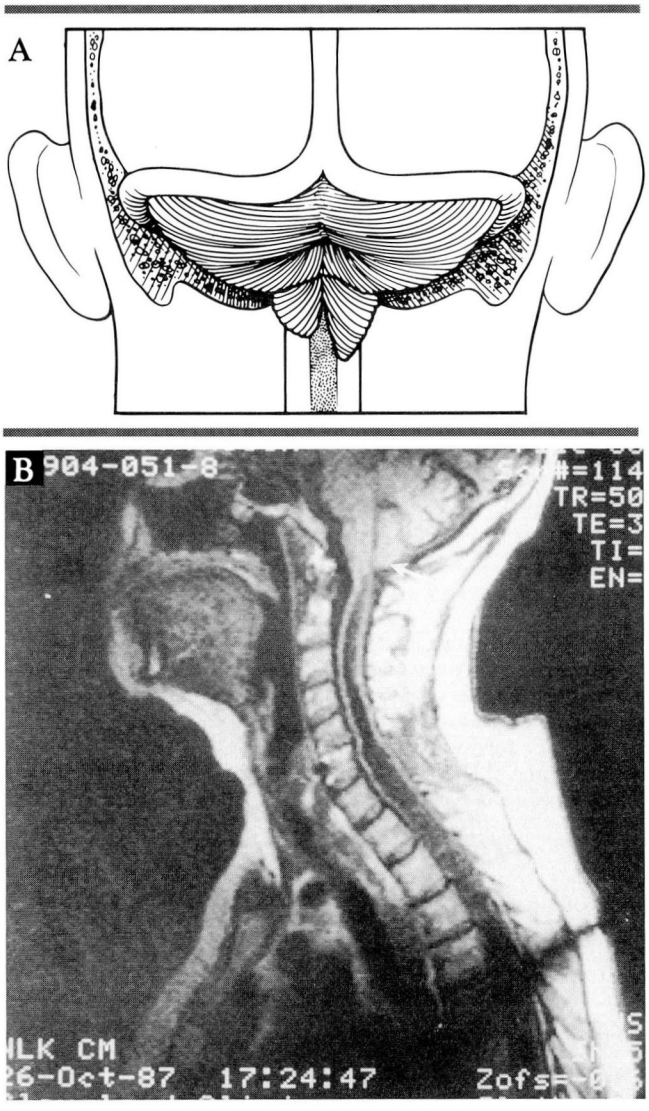

FIGURE 3. Chiari malformation with syringomyelia. *A*, deformity with tonsillar herniation. *B*, sagittal T1-weighted magnetic resonance imaging study (TR 500 milliseconds, TE 32 milliseconds) of a patient with Chiari malformation with tonsillar herniation (white arrow) and a cervicothoracic syrinx.

increases in thoracoabdominal pressure, would push CSF either directly into the spinal cord or along the nerve roots. Pooling of extracellular fluid in the cord would result in a syrinx (*Figure 1, D*).

PHYSIOLOGICAL STUDIES

Bering was among the first to conduct experiments on patients to obtain simultaneous pressure readings from the intracranial and intraspinal CSF compartments. He observed a steep pulse pressure (from the choroid plexus) in the ventricles which was delayed and dampened to a gentle curve in the lumbar subarachnoid space.[22] This was compatible with Gardner's concept of hydrodynamic forces acting in a cranial-to-spinal direction. More recently, Muraszko and colleagues demonstrated with intraoperative ultrasonography that pulsatile cervical syrinx expansion occurs in synchrony with the heartbeat and is reduced by suboccipital decompression and dural opening.[23] This would validate Gardner's hydrodynamic concepts, since the CSF pulse wave is synchronized with the heartbeat.

Williams carried out detailed measurements using intracranial and intraspinal transducers calibrated to give equal pressures at rest. In patients with syringomyelia and hindbrain anomalies, he demonstrated craniospinal pressure differentials which were accentuated during the Valsalva maneuver (*Figure 2*).[15,16,27] He showed correction of the suck phenomenon by repeating the pressure measurements in patients who had decompression of the posterior fossa.[15,18] Williams' data on significant craniospinal pressure differentials in myelodysplastic children with severe Chiari malformation has recently been questioned by Park,[28] who was unable to demonstrate a pressure gradient in his patients. However, in Park's study the conditions of measurements, anesthetic techniques, and patient pathologies were quite different from those in Williams' study.

Another area of research has addressed the question of how the fluid moves into the spinal cord parenchyma. Gardner suggested the possibility of intermittent patency of the central canal of the spinal cord and provided evidence of this in cadavers examined within minutes of death.[13,14] Park[28] and others have questioned this mechanism; however, their single radiographic studies could not exclude intermittent patency. Using horseradish peroxidase and lanthanum, Ikata et al have shown that the extracellular space provides transit for entry into the normal spinal cord and syringeal cavities.[29] This is compatible with the Aboulker and the Ball and Dayan concepts of direct entry of CSF into the spinal cord (*Figure 1, D*).

THE ROLE OF MAGNETIC RESONANCE IMAGING

Formerly, detailed study of the pathoanatomy of the craniovertebral junction and syringomyelia was hindered by limitations of imaging modalities such as gas myelography, iophendylate myelography, and computed tomographic metrizamide myelography.[30–32] The

TABLE
MRI-BASED CLASSIFICATION OF SYRINGOMYELIA PATIENTS: A COMPARISION OF TWO STUDIES

Syrinx type	Pillay et al[4] (Cleveland Clinic) (N=59)	Kokmen et al[36] (Mayo Clinic) (N=15)
Syringomyelia with Chiari malformation	28 (47.5%)	10 (66.6%)
Neoplastic	14 (24.0%)	0
Idiopathic	8 (13.5%)	4 (26.7%)
Post-traumatic	6 (10.0%)	1 (6.7%)
Syringomyelia with spinal arachnoiditis	3 (5.0%)	0

introduction of MRI has provided exquisite demonstration of the craniovertebral area and spinal cord in both sagittal and axial planes.[3,4,33,34] Wilberger found that MRI demonstrates a large number of syringes which are either poorly visualized or missed on myelography.[35]

In a recent MRI study at the Cleveland Clinic, 35 symptomatic adult patients with Chiari malformation were classified as either "A" or "B" patients[3]: Chiari A patients had syringomyelia and a less severe hindbrain herniation, whereas Chiari B patients did not have syringomyelia but were more likely to have complex and severe hindbrain and tonsillar herniation (*Figures 3* and *4*). The reduced likelihood of syringomyelia in patients with significant brain stem herniation can be explained if the energy from Gardner's hydrodynamic force or from Williams' suck phenomenon is dissipated in compacting the hindbrain into the foramen magnum rather than forcing CSF through the obex. This crowding also acts as an external plug compressing the lower brain stem and upper cervical spinal cord. This would prevent transmission of water-hammer pulses of CSF (as described by Gardner) or the suck effects of craniospinal pressure dissociation (according to Williams) from developing a syringeal cavity in the central canal of the spinal cord. In patients with less evidence of craniovertebral crowding, there would be no impediment to these forces producing a syrinx.

The high incidence of cervical syringes in our series,[3] the absence of solitary thoracic or lumbar syringes, and the presence of the upper end of most syringes between the foramen magnum and C_{2-3} provide further support for the theories of Gardner and Williams.

In a second MRI study of 59 symptomatic patients with syringomyelia,[4] we confirmed previous reports that the Chiari malformation is the most frequent associated condition[36] (*Table*). Neoplastic syringes were the next most common category, followed by

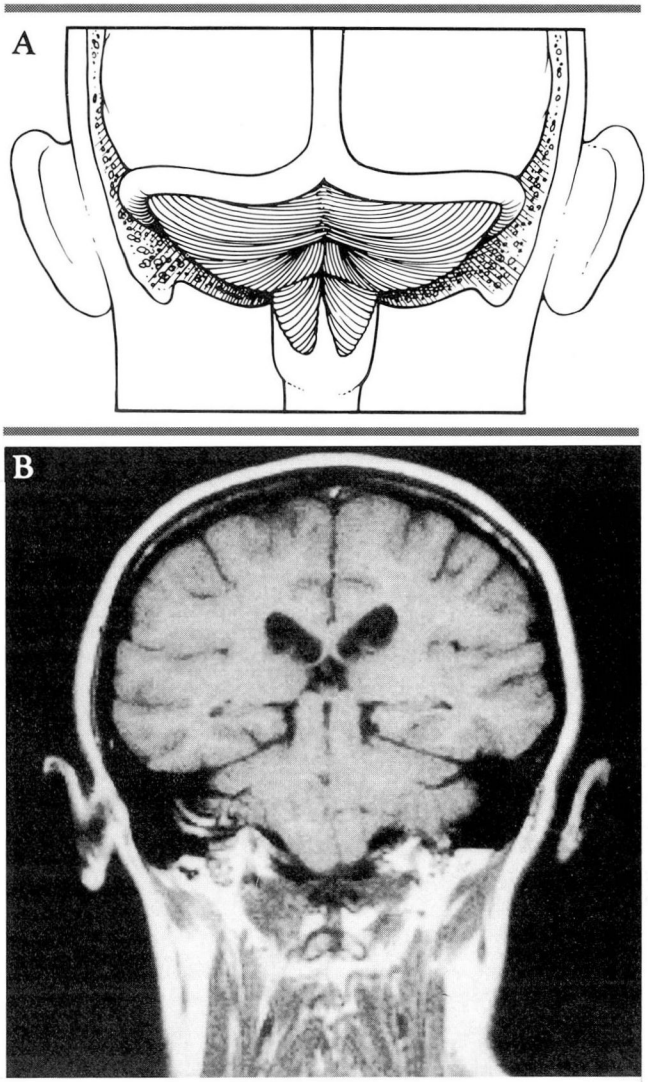

FIGURE 4. Chiari malformation without syringomyelia in an adult. *A*, tonsillar herniation and an inferiorly displaced brain stem. *B*, the Chiari malformation as seen in a coronal T1-weighted magnetic resonance imaging (MRI) study (TR 500 milliseconds, TE 17 milliseconds). An MRI study of the spinal cord showed no evidence of a syrinx.

idiopathic syringomyelia.[4,36]

Idiopathic syringomyelia appears in many instances to be a forme fruste of syringomyelia in association with the Chiari malformation (*Figure 5*, *A* and *B*). In our study, these patients were more likely to have cervical or holocord syringes. At surgery, they exhibited adhesions obstructing the fourth ventricular outlet and the craniovertebral subarachnoid space. We believe the syringes of these patients formed predominantly

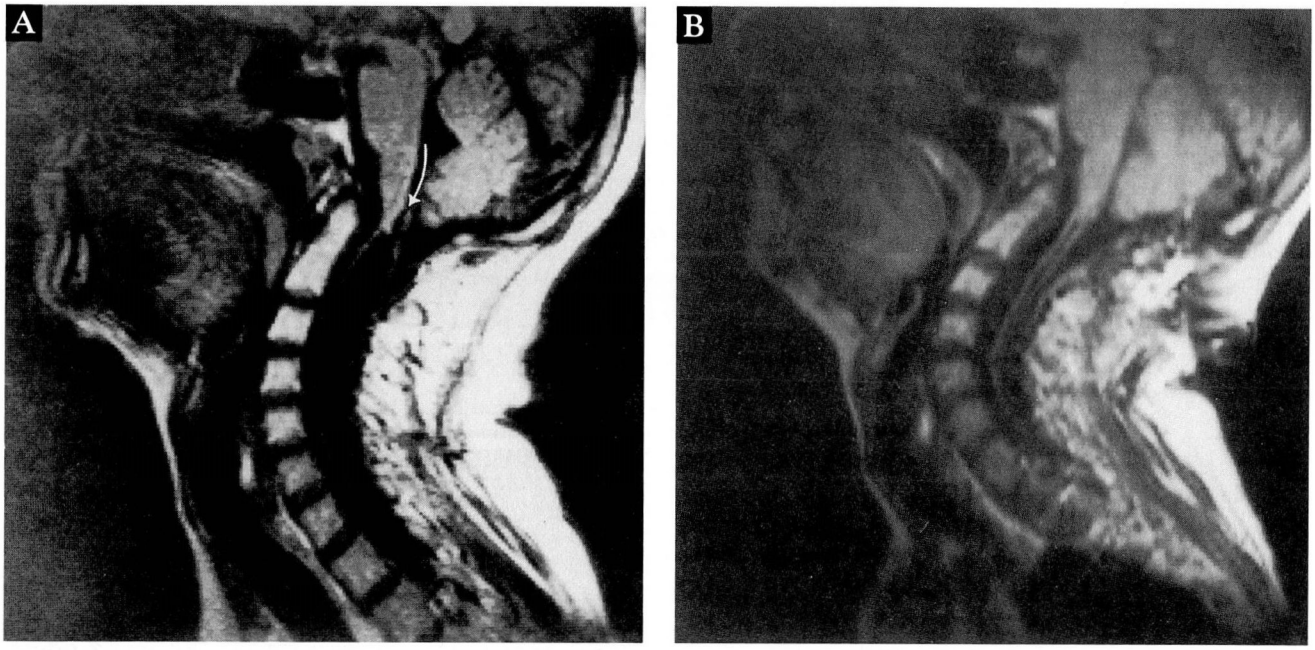

FIGURE 5. Idiopathic syringomyelia. *A*, a T1-weighted magnetic resonance image (TR 500 milliseconds, TE 38 milliseconds) of a patient with a holocord syrinx extending from the obex (white arrow) to the conus. No hindbrain pathology was found in this patient. *B*, after craniocervical decompression, opening of the fourth ventricular outlet, and obex plugging (Gardner procedure), the syrinx has resolved (TR 500 milliseconds, TE 32 milliseconds). Many cases of idiopathic syringomyelia respond well to the Gardner procedure, indicating that these may represent a less severe type of Chiari malformation.

according to Gardner's theory, as no obvious foramen magnum pathology was observed which could give rise to craniospinal pressure dissociation. The Ball and Dayan and the Aboulker theories cannot account for all these aspects of syringomyelia.

An important criticism of Gardner's theory has been its failure to demonstrate a communication between the fourth ventricle and the syringeal cavity. However, we were able to demonstrate this communication in some patients using MRI (*Figure 5, A*). In others, the communication may be intermittent or too small to demonstrate, or it may have sealed off. Another factor that needs to be considered is that communication between the fourth ventricle and a cervical syringeal cavity can occur across the extracellular spaces of the intervening spinal cord parenchyma. Hydrodynamic mechanisms or suck effects may play a role in forcing CSF through these spaces to expand the syrinx (*Figure 6*).

Neoplastic syringes probably develop through mechanisms that include interference with spinal cord blood flow and the breakdown of ischemic areas into cavities, the presence of spinal cord edema, or the coalition of microcystic areas to form a syrinx.[37–39] These syringes often have fluid with a high protein content and show a focal area of gadolinium enhancement on MRI, characteristics that distinguish them from the other types of syringes.

Syringes associated with arachnoiditis and trauma may develop as a consequence of delayed spinal cord ischemia[40] or transcordal fluid shifts. The subarachnoid space is compartmentalized by scarring. Pressure differentials can be generated in these compartments by epidural venous congestion. This may promote syrinx formation by transcordal fluid shifts, either along the nerve roots (Aboulker) or via the Virchow-Robin spaces (Ball and Dayan). Propagation and multiloculation may be promoted by Williams' slosh mechanism. MRI studies have demonstrated pulsatile movements of CSF and a flow-void signal in some syringeal cavities compatible with the slosh phenomenon.[41]

A UNIFIED THEORY OF SYRINGOMYELIA

Based on the observations discussed above, we propose a unified theory of syringomyelia (*Figure 6*). Gardner's water-hammer CSF pulse wave directed through the obex may expand the central canal of the

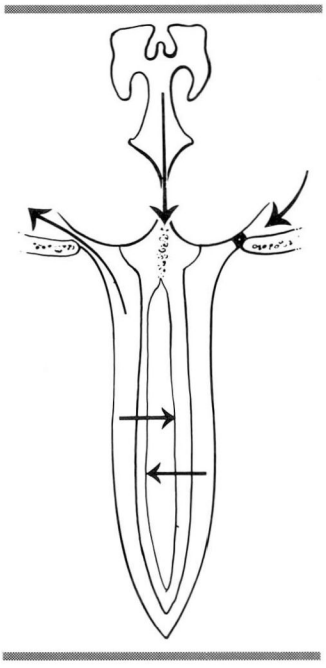

FIGURE 6. Proposed unified theory of syringomyelia includes Gardner's hydrodynamic forces magnified in the setting of foramen magnum obstruction as suggested by Williams. Transparenchymal fluid migrations (as advocated by Ball and Dayan and by Aboulker) might account for coalescence and propagation of syringes, as well as for focal post-traumatic syringomyelia not associated with hindbrain pathology.

spinal cord into a syrinx. Fourth ventricular outlet or foramen magnum obstruction would contribute to craniospinal pressure dissociation, as proposed by Williams, promoting syrinx formation and progression. In the absence of a patent obex, other mechanisms, as proposed by Ball and Dayan and Aboulker, might explain transcordal CSF flow from the fourth ventricle into a cervical syrinx. In many instances of syringomyelia these various mechanisms might operate simultaneously. In some pathophysiological situations, one or more of these mechanisms may predominate. The hydrodynamic theory is likely to play a dominant role in explaining the pathophysiology of syringomyelia associated with the Chiari malformation and idiopathic syringomyelia. Focal fluid shifts across the substance of the spinal cord may play a role in the propagation and multiloculation of syringes, as well as in the genesis of post-traumatic syringes associated with focal arachnoiditis.

Gardner's hydrodynamic theory, with refinements introduced by Williams, remains valid in the light of present physiological data and MRI studies. We continue to advocate the Gardner procedure—decompression of the posterior fossa with plugging of the obex—for patients with syringomyelia and the Chiari malformation.[3,4] This reduces the effects of suck and provides a barrier to the hydrodynamic and transcordal forces tending to push ventricular CSF into a cervical syrinx. Postoperative MRI studies have demonstrated adequate collapse of these syringes with associated favorable clinical response (*Figure 5, B*). The development of new imaging techniques and the use of dynamic real-time cine-MRI may provide further insight into the complex pathogenesis of syringomyelia.

REFERENCES

1. Ollivier d'Angers CP. Traite de la Moelle Epiniere et de ses maladies. Paris: Chez Crevot, 1827:178.
2. Barnett HJM, Foster JB, Hudgson P. Syringomyelia. Philadelphia: WB Saunders, 1973.
3. Pillay PK, Awad IA, Little JR, Hahn JF. Symptomatic Chiari malformation in adults: a new classification based on MRI with clinical and prognostic significance. Neurosurgery 1991; **28:**639–645.
4. Pillay PK, Awad IA, Little JR, Hahn JF. Surgical management of syringomyelia: a five year experience in the era of magnetic resonance imaging. Neurol Res 1991; **13:**3–9.
5. Hartwell SW. "...to act as a unit": the story of the Cleveland Clinic. Philadelphia: WB Saunders, 1985:94.
6. Gardner WJ, Goodall RJ. The surgical treatment of Arnold-Chiari malformation in adults: an explanation of its mechanism and importance of encephalography in its diagnosis. J Neurosurg 1950; **7:**199–213.
7. Gardner WJ, Abdullah AF, McCormack LJ. Varying expressions of embryonal atresia of fourth ventricle in adults. Arnold-Chiari malformation, Dandy-Walker syndrome, arachnoid cyst of cerebellum and syringomyelia. J Neurosurg 1957; **14:**591.
8. Gardner WJ, Angel J. The cause of syringomyelia and its surgical treatment. Cleve Clin Q 1958; **25:**4–17.
9. Gardner WJ. Anatomic anomalies common to myelomeningocele of infancy and syringomyelia of adulthood suggest common origin. Cleve Clin Q 1959; **26:**118–120.
10. Gardner WJ. Anatomic anomalies common to the Arnold-Chiari and Dandy-Walker malformations suggest a common origin. Cleve Clin Q 1959; **26:**206–208.
11. Gardner WJ. Myelomeningocele, the result of rupture of the embryonic neural tube. Cleve Clin Q 1960; **27:**88–100.
12. Gardner WJ. Rupture of the neural tube. Arch Neurol 1961; **4:**1–17.
13. Gardner WJ. Hydrodynamic mechanisms of syringomyelia: its relation to myelocele. J Neurol Neurosurg Psychiatry 1965; **28:**247–256.
14. Gardner WJ. The dysraphic states from syringomyelia to anencephaly. Amsterdam: Excerpta Medica, 1973.
15. Williams B. Progress in syringomyelia. Neurol Res 1986; **8:**130–145.
16. Williams B. Simultaneous cerebral and spinal fluid pressure recordings. 2 Cerebrospinal dissociation with lesions at the foramen magnum. Acta Neurochir 1981; **59:**123–142.
17. Williams B. The distending force in the production of "communicating syringomyelia". Lancet 1969; **2:**189–193.
18. Williams B. A critical appraisal of posterior fossa surgery for communicating syringomyelia. Brain 1978; **101:**223–250.
19. Ball MJ, Dayan AD. Pathogenesis of syringomyelia. Lancet 1972; **2:**799–801.
20. Aboulker J. La syringomyelie et les liquides intra rachidiens. Neurochirurgie 1979; **25:**1–44.
21. Bering EA. Choroid plexus and the arterial pulsation of the cerebrospinal fluid; demonstration of choroid plexuses as the generator of the force flow of fluid and ventricular enlargement. Arch Neurol Psychiat 1955; **73:**165–168.
22. Muraszko K, Shawker T, Oldfield E. Intraoperative ultrasonography to monitor success of surgery in patients with Chiari I malformation [Abstract]. In: Proceedings of the Congress of Neurological Surgeons. Los Angeles: Congress of Neurological Surgeons, 1990:82.

23. Bering EA. Circulation of the cerebrospinal fluid. Demonstration of the choroid plexuses as the generator of the force flow of fluid and ventricular enlargement. J Neurosurg 1962; **19:**405–407.
24. McLaurin RL, Bailey OT, Schurr PH, Ingraham FD. Myelomalacia and multiple cavitations of spinal cord secondary of adhesive arachnoiditis; experimental study. Arch Pathol 1954; **57:**138–140.
25. Becker DF. CSF production: contribution of ependyma and CNS tissue. Presented at the Society of Neurological Surgeons, February 1971.
26. Graftjik VH. Over hydrocephalus [Thesis]. University of Leiden, The Netherlands, 1932.
27. Williams B. Cerebrospinal fluid pressure-gradients in spina bifida cystica, with special reference to the Arnold-Chiari malformation and aqueductal stenosis. Dev Med Child Neurol 1975; **17:**138–150.
28. Park TS, Cail WS, Broaddus WC, et al. Lumboperitoneal shunt combined with myelotomy for treatment of syringohydromyelia. J Neurosurg 1989; **70:**721–727.
29. Ikata T, Masaki K, Kashiwaguchi S. Clinical and experimental studies on permeability of tracers in normal spinal cord and syringomyelia. Spine 1988; **13:**737–741.
30. Logue V. 14th Crookshand Lecture. Syringomyelia: a radiodiagnostic and radiotherapeutic saga. Clin Radiol 1971; **22:**2–16.
31. Bosley TM, Cohen DA, Schatz NJ, et al. Comparison of metrizamide computed tomography and magnetic resonance imaging in the evaluation of lesions at the cervicomedullary junction. Neurology 1985; **35:**485–492.
32. Li KC, Chui MC. Conventional and CT metrizamide myelography in Arnold-Chiari I malformation and syringomyelia. AJNR 1987; **8:**11–17.
33. Sherman JL, Barkovich AJ, Citrin CM. The MR appearance of syringomyelia: new observations. AJR 1987; **148:**381–391.
34. Samuelsson L, Saaf J, Wahlund LO, Thuomas KA, Bergstrom K. Evaluation of syringomyelia and Chiari malformations using ultra-low magnetic resonance imaging. Magn Reson Imaging 1990; **8:**123–129.
35. Wilberger JE, Maroon JC, Prostko ER, Baghai P, Beckman I, Deeb Z. Magnetic resonance imaging and intraoperative neurosonography in syringomyelia. Neurosurgery 1987; **20:**599–605.
36. Kokmen E, Marsh WR, Baker HL. Magnetic resonance imaging in syringomyelia. Neurosurgery 1985; **17:**267–270.
37. Russell DS. Capillary haemangioma of spinal cord associated with syringomyelia. J Pathol Bacteriol 1932; **35:**103–105.
38. Feigin I, Ogata J, Budzilovich G. Syringomyelia: the role of edema in its pathogenesis. J Neuropathol Exp Neurol 1971; **30:**216.
39. Russell DS, Rubinstein LJ. Pathology of tumors of the nervous system, 5th ed. [In] Rubinstein LJ, ed. Baltimore: Williams and Wilkins, 1989:647.
40. Caplan LR, Norohna AB, Amico LL. Syringomyelia and arachnoiditis. J Neurol Neurosurg Psychiatry 1990; **53:**106–113.
41. Enzmann DR, O'Donohue J, Rubin JB, et al. CSF pulsations within nonneoplastic spinal cord cysts. AJR Am J Roentgenol 1987; **149:**149–157.