Surgical experience in 130 pediatric patients with Chiari I malformations

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Object. The aim of this study was to present the long-term findings of a surgical series of pediatric patients with Chiari I malformations.

Methods. One hundred thirty symptomatic pediatric patients with Chiari I malformations underwent posterior fossa decompression. The age in this group of patients ranged from 2 months to 20 years (mean 11 years). The length of the hospital stay extended from 2 to 7 days (mean 2.7 days), and follow up was from 3 months to 15 years (mean 4.2 years). Patients most often presented with headache/neck pain (38%) and scoliosis (18%). Examples of associated diagnoses included neurofibromatosis Type 1 (5.5%), hydrocephalus (11%), idiopathic growth hormone deficiency (5.5%), and Klippel–Feil anomaly (5%). Syringes were present in 58% of patients. Seventeen percent of patients had caudal displacement of the brainstem and fourth ventricle.

Postoperative relief of preoperative pathologies was experienced in 83% of patients. Of the most common presenting symptoms—headache/neck pain and scoliosis—12 and 17%, respectively, were not alleviated postoperatively. Complications occurred in 2.3% of this group and included the development of acute hydrocephalus postoperatively and severe life-threatening signs of brainstem compression that necessitated a transoral odontoidectomy. Nine patients have had to undergo repeated operations for continued symptoms or persistent large syringes. During surgery 10 patients (7.7%) were found to have arachnoid veins occluding the fourth ventricular outlet, and nine of these had syringomyelia. In our experience almost all syringes will stabilize or improve with posterior fossa decompression and duraplasty.

Conclusions. The authors believe this to be the largest reported series of pediatric patients who have undergone posterior fossa decompression for Chiari I malformations.

KEY WORDS • hindbrain • brain herniation • ectopia • children • posterior fossa decompression

CHIARI malformations represent varying degrees of hindbrain herniation through the foramen magnum. Professor Hans Chiari (1851–1916) made a pioneering contribution to our early understanding of these entities by developing a classification system based on the results of more than 40 detailed autopsies he conducted while a pathologist in Prague.26 The Type I malformation, described in 1891, involved caudal descent of the cerebellar tonsils inferior to the plane of the foramen magnum. In the current report we present the salient features of our surgical experience with Chiari I malformations in children.

Clinical Material and Methods

Patient Population

We present data from 130 pediatric patients with Chiari I malformations whom we encountered during our 23 years of surgical experience. Patient age ranged from 2 months to 20 years (mean 11 years); sixty-nine patients were male and 61 were female. Each patient underwent posterior cranial fossa decompression together with the removal of the posterior arch of C-1. The posterior elements of C-2 were removed in one patient. One hundred twenty-nine patients underwent duraplasty (cadaveric pericardium, pericranium, posterior atlantooccipital membrane, or nuchal ligament) at their first operation, and one patient who had previously not undergone a duraplasty underwent one at a second operation when symptoms had persisted. The first 30 patients in the entire group underwent surgery at Duke University Medical Center, Durham, North Carolina (1979–1982), whereas the remaining 100 patients underwent surgery at The Children’s Hospital of Alabama, Birmingham, Alabama (1992–2002). All surgeries were performed by the senior author (W.J.O.). The duration of each procedure was approximately 90 minutes. Each patient was monitored in the intensive care unit overnight and sent to a ward room the following day.

Results

The hospital stay for this cohort ranged from 2 to 7 days (mean 2.7 days). Preoperative imaging demonstrated the inferior border of the lowest cerebellar tonsil between the fo-
rumen magnum and C-1 in 26 patients, at C-1 in 61, at C-2 in 42, and at C-3 in one patient. Follow up in this group was 3 months to 15 years (mean 4.2 years). Patients most often presented with headache/neck pain (38%) and scoliosis (18%). Table 1 lists other common presentations. Associated diagnoses within this group included neurofibromatosis Type 1 (5.5%), hydrocephalus (11%; all patients with hydrocephalus had a verified functioning CSF diversion shunt), idiopathic growth hormone deficiency (5.5%), Sprengel deformity (2.7%), Klippel–Feil anomaly (5%), and basilar invagination (4%). Table 2 lists all associated diagnoses in this group. No single association indicated whether a patient would respond to surgery. Syringes were present in 75 patients (58%): three in the brainstem, 16 in the cervical cord, nine in the cervicothoracic cord, 12 in the thoracic cord, two in the lumbar cord, and 33 were holocord syringes. Seventeen percent of patients had caudal displacement of the brainstem and fourth ventricle in addition to tonsillar ectopia. Previously, we reported that the odontoid process in many of these patients was significantly retroflexed when compared with that in controls.\(^9\)

Postoperative relief of preoperative pathologies occurred in 83% of patients. Of the most common presenting symptoms, headache/neck pain and scoliosis were not alleviated postoperatively (12 and 17%, respectively). Stents extending from the fourth ventricle to the cervical subarachnoid space were placed mainly in the first 30 cases (26 patients). Patients in whom stents were inserted were less likely to experience resolution of their preoperative presenting symptoms (p < 0.05, Student t-test). Cervical instability was noted in one patient preoperatively, and although another patient (13 years old) was noted to have a predental space of 4 mm in flexion preoperatively, this same patient has had no clinical symptoms indicative of cervical instability to date. Postoperatively, one patient demonstrated symptoms referable to brainstem compression, which was treated with a transoral odontectomy. This patient had severe retroflexion of the odontoid process with significant ventral compression.

Complications occurred in 2.3% of the entire group. Two patients developed extraxial subdural fluid collections and acute hydrocephalus postoperatively. These patients were treated with extraventricular drainage for approximately 1 week until intracranial pressures normalized and the hydrocephalus resolved.\(^16\) As mentioned earlier, one patient developed severe, life-threatening signs of brainstem compression following surgery, which were addressed with a transoral odontoidectomy and occipital cervical fusion 8 days later. This patient recovered, although a left mild esotropia persisted. Unilateral tonsillar coagulation was performed in 22 patients (17%), eight of whom underwent repeated operation for continued syringomyelia. Nine patients have undergone repeated operations for continued symptoms or persistent large syringes; eight surgeries were performed for continued syringomyelia and one for continued headache that was not relieved following posterior fossa decompression without duraplasty. Indeed, the latter patient’s headaches were relieved immediately postoperatively following duraplasty. Of the eight cases of continued syringes, all but one in which tonsillar coagulation had occurred was resolved with repeated operation. The one patient in whom resolution did not occur was treated with the insertion of a syringopleural shunt and only then had diminishment of his syrinx. All patients with persistent syringes at the second operation did not demonstrate spontaneous egress of CSF from the fourth ventricle. Ten patients (7.7%) were found to have an arachnoid veil occluding the fourth ventricular outlet at surgery and these vela were all transected. Nine of these 10 patients with vela had associated syringomyelia. For duraplasty, we have used cadaveric pericardium and autologous pericranium, muncal ligament, and posterior atlantooccipital membrane. No form of meningitis or cerebellar ptiotis was observed in this group of 130 patients. In addition, no operative or postoperative complications have resulted in a patient’s death.

### TABLE 1

<table>
<thead>
<tr>
<th>Sign/Symptom</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>head/neck/back pain</td>
<td>55</td>
</tr>
<tr>
<td>scoliosis</td>
<td>23</td>
</tr>
<tr>
<td>apnea/bradycardia</td>
<td>11</td>
</tr>
<tr>
<td>irritability</td>
<td>16</td>
</tr>
<tr>
<td>drop attacks</td>
<td>3</td>
</tr>
<tr>
<td>rage attacks</td>
<td>2</td>
</tr>
<tr>
<td>hoarseness</td>
<td>7</td>
</tr>
<tr>
<td>upper-extremity pain/weakness/numbness</td>
<td>22</td>
</tr>
<tr>
<td>chronic hiccups</td>
<td>2</td>
</tr>
<tr>
<td>dysphagia</td>
<td>20</td>
</tr>
<tr>
<td>facial numbness</td>
<td>11</td>
</tr>
<tr>
<td>dyspnea</td>
<td>3</td>
</tr>
<tr>
<td>ataxia</td>
<td>12</td>
</tr>
<tr>
<td>lower-extremity hyperreflexia/weakness</td>
<td>7</td>
</tr>
<tr>
<td>chronic enesis</td>
<td>13</td>
</tr>
<tr>
<td>nasal speech</td>
<td>15</td>
</tr>
<tr>
<td>C-2 dysesthesia</td>
<td>26</td>
</tr>
<tr>
<td>migraine-like headache</td>
<td>28</td>
</tr>
<tr>
<td>urinary incontinence</td>
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</tr>
</tbody>
</table>

* Many patients exhibited more than one sign or symptom; hence the total does not equal 130.

### TABLE 2

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>neurofibromatosis Type 1</td>
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</tr>
<tr>
<td>idiopathic growth hormone deficiency</td>
<td>7</td>
</tr>
<tr>
<td>Sprengel deformity</td>
<td>4</td>
</tr>
<tr>
<td>hydrocephalus</td>
<td>14</td>
</tr>
<tr>
<td>basilar invagination</td>
<td>5</td>
</tr>
<tr>
<td>Klippel–Feil anomaly</td>
<td>7</td>
</tr>
<tr>
<td>cervical rib</td>
<td>2</td>
</tr>
<tr>
<td>corpus callosum lipoma</td>
<td>1</td>
</tr>
<tr>
<td>mega cisterna magna</td>
<td>2</td>
</tr>
<tr>
<td>Erb palsy</td>
<td>1</td>
</tr>
<tr>
<td>diabetes insipidus</td>
<td>1</td>
</tr>
<tr>
<td>Down syndrome</td>
<td>2</td>
</tr>
<tr>
<td>epilepsy</td>
<td>5</td>
</tr>
<tr>
<td>mental retardation</td>
<td>3</td>
</tr>
<tr>
<td>Crohn disease</td>
<td>1</td>
</tr>
<tr>
<td>caudal regression syndrome</td>
<td>1</td>
</tr>
<tr>
<td>cerebral palsy</td>
<td>2</td>
</tr>
<tr>
<td>metopic ridging</td>
<td>20</td>
</tr>
<tr>
<td>middle fossa arachnoid cyst</td>
<td>1</td>
</tr>
<tr>
<td>dolichooodontoid</td>
<td>1</td>
</tr>
<tr>
<td>cloacal exstrophy</td>
<td>1</td>
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</table>
Chiari I malformations in pediatric patients

Discussion

Among the theories for the pathogenesis of the Chiari I malformation, the development of a differential cranio-spinal pressure gradient across the foramen magnum most adequately accounts for the observed clinical and anatomical findings.1,5,12,22,35,37,48,54,59 Altered CSF circulation at the foramen magnum prevents instantaneous pressure equilibration between the intracranial and spinal subarachnoid space. Presumably, an intermittent vector of force then develops with each Valsalva maneuver, which can lead to the progressive downward movement of developing tissue through the foramen magnum. If the impediment to CSF equilibration occurs after the tonsils have formed, the pressure gradient favors tonsillar herniation and may be responsible for the initiation of syringomyelia formation.50–52 The aforementioned data support the response seen in the current treatment of a Chiari I malformation, that is, posterior fossa decompression.

Regarding a cause of the Chiari I malformation, Schady, et al.,41 found that the volume of the posterior fossa was 23% smaller in patients with the malformation when compared with that in control patients. Other anomalies that further restrict the movement of the posterior fossa contents, such as occipital dysplasia, are seen in as many as 76% of patients.42 With rapid growth in the neonatal period during which the cerebellum grows to 80% of its adult weight in the 1st year of life,14 a delay or arrest in the expansion of the osseous posterior fossa may contribute to the caudal herniation of the cerebellar tonsils through the foramen magnum.27,30,55 An associated diagnosis in our group included idiopathic growth hormone deficiency (5.5%), which we have hypothesized as a potential cause of a smaller-than-normal posterior fossa in some patients with Chiari I malformations.31

Signs and Symptoms

In adults, the predominant clinical symptoms are pain and headache, which occur in 15 to 75% of these patients.11,13 Valsalva-induced headache was observed in 73% of our patients with this complaint. Dysesthesia in the distribution of the greater occipital nerve was observed in approximately 7% of our patients. Interestingly, seven of our patients presented with frontal headache and in all but one this symptom was relieved postoperatively.45,46 More recently, because of the ease of diagnosis and increased clinical awareness, cases in pediatric patients are increasingly reported.10,11,14 Younger children, who have difficulty localizing pain, may simply be more irritable, which obviously could have numerous other causes.28 This change in temperament may be their only symptom, and often other clinical signs may not be present. In the cohort in the current study, 17 children were 3 years old or younger. Among this younger group, patients who were verbal presented with headache/neck pain (41%) and those who were nonverbal presented with opisthotonus/irritability (29%), apnea (11.7%), scoliosis (6%), and drop attacks (11.7%).12,27,30 Frequently, lower cranial nerves are affected, although sudden death, recurrent ataxia, ocular motor disturbances, vertigo, syncope, stridor, and drop attacks appear less frequently in children.11,13,17,19,32,59 Neurogenic dysphagia was demonstrated in five of our patients.25,43 Infrequently, asymptomatic children with Chiari I malformations may present with acute spinal cord injury or death following minor trauma.9,47 Two patients in our group initially underwent imaging after the onset of symptoms that were experienced after trauma. The extent of tonsillar displacement may correlate with symptoms.6 In the current study, the degree of tonsillar ectopia did not correlate significantly (p > 0.05, Student t-test) with either the presence of symptoms or their postoperative resolution.

With the ease of an MR imaging–based diagnosis, patients, especially children, are presenting with fewer signs and increasingly subtle symptoms. In this situation, differentiating between minor or early Chiari malformation–related problems and unrelated symptoms can be difficult. The clinician should strive for the earliest possible recognition of this diagnosis, but realize that not every child with 3 mm of tonsillar herniation will need immediate surgical intervention. The clinician should also make every effort to exclude all other non-Chiari origins of a patient’s symptoms.

Occurrence of Syringomyelia

Syringomyelia occurs in 50 to 76% of patients with Chiari I malformations.13,33 Periodic Valsalva with resultant elevation in subarachnoid space pressure may cause a surge into the cyst cavity.38,48,50 On occasion, this is associated with a stepwise worsening of symptoms as the cavity enlarges or moves, and this was demonstrated in four of our patients (3%). Commonly, cavities due to a Chiari I malformation spare the C-1 area and involve the middle and lower cervical spine.33,57 These skipped areas have been observed in 92% of our patients with syringomyelia. Back/shoulder pain and scoliosis, which occurred in 10 and 18% of our population, respectively, may have been the initial complaints in a significant proportion of patients.18 Scoliosis, which is present in 25 to 50% of patients with Chiari I malformations and in 18% of our patient population, can develop before skeletal maturity in association with syringomyelia.7,12,22 Interestingly, scoliosis and an underlying holocord syrinx were the presenting symptoms in a 2-year-old boy from our group.

Magnetic Resonance Imaging

The position of the tonsils is the key MR imaging criteria for the diagnosis of a Chiari I malformation. Nonetheless, we have reported on a subgroup of patients with the Chiari malformation, who do not have frank tonsillar ectopia but have syringes that respond to posterior fossa decompression.50 Barkovich, et al.,6 in a study of 200 healthy adult and pediatric patients, revealed that normal tonsils lie 1 mm above the foramen magnum, with a standard deviation of 1.9 mm; virtually all symptomatic patients had tonsillar herniation beyond the 3- to 5-mm range. Some researchers have observed that normal tonsils may prolapse up to 6 mm during the first decade of life and gradually ascend with patient age.15,31

This raises the question of whether the surgeon should defer surgically correcting Chiari I malformations in mildly symptomatic children and wait for chronic manifestations of this disease with the hope that the tonsils will ascend and symptoms will resolve without surgical intervention. A prospective randomized trial developed to answer this question is not likely to gain approval by most
institutional review boards because many patients would be exposed to potential neurological compromise. Note, however, that parents of most patients request, if not demand, surgical intervention for symptomatic children even after being informed of the risks of the procedure. Complications occurred in 2.3% of our cases. For diagnostic purposes, most authors consider a Chiari I malformation to be present if tonsils descend more than 5 mm inferior to the foramen magnum, as demonstrated on T1-weighted MR imaging.6 In our patients, preoperative imaging exhibited the inferior border of the lowest cerebellar tonsil to be situated between the foramen magnum and C-1 in 26 patients, at the level of C-1 in 61, at the level of C-2 in 42, and at the level of C-3 in one patient. The tonsils are very frequently asymmetrically displaced.30 In patients with “benign tonsillar ectopia,” the diagnosis of a Chiari I malformation may be justified if there is an associated syrinx or loss of the normal rounded shape of the inferior tonsils.5,30 Pointed tonsils were visualized in 97% of our surgical population with a Chiari I malformation. Preoperative imaging should include neuroimages of the neck in flexion and extension to assess the stability of the cervical spine and the position of the dens.37 The surgeon should also remember while positioning the patient for surgery that tonsillar herniation is exaggerated and the odontoid moves more posteriorly with cervical flexion.49

Other bone abnormalities associated with Chiari I malformation include a small posterior fossa, occipitalization of the atlas, fusion of cervical vertebrae, Klippel–Feil anomaly, basilar invagination,30,31 and possibly metopic ridging.31 Approximately 5% of our patients had associated bone anomalies. Hunter, et al.,29 reported on three patients with Chiari I malformation and midbrain deformity, a feature seen more often in Chiari II malformation. In the current study population, surprisingly, 17% were noted to have tonsillar ectopia as well as caudal displacement of the brainstem and fourth ventricle, the so-called Chiari I.5 malformation.37 Note that neurofibromatosis Type 1 was demonstrated in 5.5% of our patients, with only one patient having an intracranial manifestation (swelling of the optic nerve) of this disease and none exhibiting hydrocephalus.

Treatment and Outcome

The natural history of asymptomatic Chiari I malformation is not known. A number of patients with cerebellar tonsils below the foramen magnum are frequently revealed on MR imaging studies obtained for unrelated signs. In symptomatic patients with or without hydroxy-syringomyelia, posterior fossa decompression is indicated. In the majority of patients, improvement or stabilization of symptoms occurs following decompressive surgery.34,35,40 Patients expressing symptoms for fewer than 2 years have better outcomes after surgery, and 80% are likely to experience pain relief.19 In our experience, headache (95%), sleep apnea (100%), and scoliosis (43%) are more predictably improved compared with motor or sensory deficits. This phenomenon has also been the experience of others.37

As experience accumulates, indications for surgery in patients with Chiari I malformations are evolving. Caudal descent of the tonsils 5 mm inferior to the foramen magnum associated with appropriate symptoms is considered to be abnormal by almost all authors. Patients with this degree of herniation should be deemed candidates for decompression. Caudal descent of between 0 and 5 mm must be considered in the clinical context of the individual patient. Marginal descent of the tonsils in association with hydroxy-syringomyelia without other cause may be physiologically and clinically significant. The natural history of patients with significant syringomyelia is one of progressive loss of neurological function over years or decades.2 Consideration of surgical intervention at an early stage in this group is warranted. The likelihood of a serious, irreversible injury as a result of the procedure should be minimal. Data from several large series have demonstrated no serious complications from the procedure in experienced hands.12,34

The natural history and indications for surgery in patients with syringomyelia are better understood today. If the patient has a syrinx and is symptomatic, the chances for improvement are favorable following surgical intervention.21,22,36 Two thirds of the patients with syringomyelia and scoliosis can have a reduction in the Cobb angle; those patients with a Cobb angle of less than 50˚ should be spared spinal fusion until there is clear evidence of progression.24 We have observed that asymptomatic patients with extensive cavitation consisting of 50% or more of the cross-sectional diameter of the spinal cord are likely to progress and should be considered surgical candidates. In our experience almost all syringes will stabilize or improve with posterior fossa decompression and duraplasty. Although we have not performed prospective randomized trials in which outcome with or without duraplasty was compared, in many of our patients with syringomyelia exhibiting veils at the fourth ventricular outlet we would encourage the performance of this step. To date we have had no case of viral, bacterial, or chemical meningitis associated with duraplasty.

In our patients, scoliosis involving curvature of greater than 40˚ was less likely to improve with posterior fossa decompression, even with a decrease in the size of the syrinx (16 patients). Nagib reported that among 10 patients with Chiari I malformations and scoliosis, six with preoperative Cobb angles of less than 30˚ improved and four with preoperative angles greater than 30˚ stabilized after suboccipital craniectomy. Isu, et al.,24 reported an improvement or stabilization of scoliosis in six patients with syringes and Chiari I malformation in six patients with preoperative Cobb angles of less than 40˚.

Surgical Procedure

The surgical procedure for treating Chiari malformations consists of bone decompression with or without dural expansion.29 The degree of intradural exploration will vary with the surgeon and the anatomy of the patient. A midline incision is made from just inferior to the inion to the spinous process of the axis to allow for a C-1 laminectomy and occipital craniectomy, both of which are performed on the midline extending 4 to 5 cm. We strongly believe that the lateral removal of bone is not necessary and places the patient at greater risk. Moreover, we have yet to encounter a patient with postoperative cerebellar ptosis. After removing the posterior arch of C-1, a dense constrictive band frequently causing intradural compression and arachnoid adhesion is excised or dissected free with the posterior atlanto-occipital membrane. We have encountered these bands in roughly one third of our patients. With long-term follow up of patients, we have found that the removal of the posteri-
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or elements of C-1 does not cause instability. In contrast, instability of the cervical spine has been reported in up to 7% of patients following posterior fossa decompression. We also make a special effort to avoid disrupting the musculature attachments into C-2 (for example, semispinalis cervicis and multifidus muscles). Anecdotally, we believe that patients experience greater pain postoperatively with the excessive removal of muscle. The dura mater is then opened in the midline and retracted laterally; the arachnoid is opened off the midline and clipped with the retracted dura, allowing an intact arachnoid to help form a water-tight dural seal. In addition, given our experience with two patients in this series in whom postoperative acute hydrocephalus developed, we now routinely clip the arachnoid to the dura so that potential CSF cannot dissect into the subdural plane and, through mass effect, potentially kink the cerebral aqueduct with resultant hydrocephalus. Since the introduction of this method we have not experienced this complication again.

Arachnoid adhesions are next dissected to establish spontaneous CSF flow from the fourth ventricle. This may necessitate unilateral tonsillar coagulation. Posterior fossa scarring, inadequate foramen magnum decompression, cerebellar ptosis, or failure of fourth ventricle outflow are possible reasons for surgical failure. We have had no occurrence of cerebellar ptosis and attribute this to bone removal over the midline occiput only. In symptomatic patients with syringomyelia one must establish normal intracranial pressure followed by a posterior cranial fossa decompression, as in those patients without syringomyelia. Posterior fossa decompression relieves symptoms of syringomyelia in more than 75% of patients (in our series ~80% at first operation and >95% at second operation) and is preferred over syrinx shunt placement. To months postoperatively, if a syrinx persists and the patient remains symptomatic, the insertion of a syringopleural or a syringoperitoneal shunt could be considered as well as reasons why the posterior fossa decompression failed. We believe that intradural exploration is essential, with almost 8% of our patients demonstrating an arachnoid veil that occluded the foramen of Magendie. In all but one of these patients an associated syringomyelia was present. Finally, since our experience with a girl who developed life-threatening symptoms of brainstem compression following posterior fossa decompression, we have developed the pB–C2 line (a line from the basion to the inferior posterior border of C-2, with a perpendicular line drawn from the tip of the dens to the anterior spinal dura mater), which aids in preoperative defining patients with significant ventral compression (>9 mm) that might necessitate ventral decompression in addition to posterior fossa decompression.

Conclusions

The Chiari I malformation with or without an associated syringomyelia represents a complex anatomical and clinical challenge. As more is discovered about the natural history of this disease, its management will continue to evolve. The trend toward earlier operations in selected patients with milder symptoms seemingly leads to improved long-term neurological outcomes. We believe that it is essential to decompress the posterior fossa on the midline only and to open the dura mater and explore for patency of the foramen of Magendie to ensure that there is egress for CSF, which may necessitate tonsillar coagulation. For an unknown reason, our early experience with the placement of stents was associated with poorer outcomes compared with patients in whom stents were not placed. During long-term follow up we have found no problem with instability of the cervical spine following a C-1 laminectomy. In addition, inherent risks of nonautologous grafts are obviated with the use of a pericranial graft, which during long-term follow up has shown no complication in our group. Complications from surgical intervention are minimal and results good when appropriate patients are chosen. In our experience posterior fossa decompression in the symptomatic pediatric patient with Chiari I malformation offers success in the majority of cases with minimal complications.

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