Secondary Interventions for Chiari I Malformation

16

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Abstract

Patients with Chiari I malformations may present again after a foramen magnum decompression for two reasons: either they are unsatisfied with the result or new neurological symptoms have appeared. This chapter provides a systematic approach to these patients. As a general rule, revision surgery should be reserved for patients with progressive neurological symptoms. Arachnoid scarring causing obstructions of cerebrospinal fluid (CSF) flow was the commonest intraoperative finding in such revisions. Craniocervical instability in patients with basilar invagination or Klippel-Feil syndromes is the other potential mechanism leading to postoperative deterioration after a foramen magnum decompression. In such patients, a revision has to include craniocervical stabilization. Apart from these foramen magnum-related mechanisms, degenerative diseases of the cervical spine may lead to signs of a cervical myelopathy requiring early surgery. With revision surgeries, no major postoperative improvements should be expected. Stabilization of the neurological state is the realistic outlook.

Foramen magnum decompression is widely recognized as the procedure of choice for treatment of patients with Chiari I malformation (CMI) with long-term success rates well above 80 % reported in numerous reports in the literature. On the other hand, few publications deal specifically with treatment concepts for patients who develop new neurological problems after such a decompression. Furthermore, there still exists considerable disagreement, as to what a foramen magnum decompression should include: Is it necessary to open both layers of the dura? Should the arachnoid be opened and dissected? How should we deal with the cerebellar tonsils? Should a duraplasty be performed and if so, what kind of material should be used for grafting? An analysis of patients with new symptoms after a decompression may provide some answers to these questions. Other possible causes for a neurological deterioration have to be evaluated as well.

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Diagnosis

Among a series of 559 patients presenting with CMI, 107 patients had already undergone a foramen magnum decompression. Of these, 27 had also been treated by a syrinx shunt. Fifty-six of these 107 patients were not operated: in 40 patients, a revision was not recommended because the neurological status was stable or considered unlikely to be stabilized by another intervention, while 16 refused another operation. The majority of patients, in whom a revision was not recommended, presented because they were disappointed by the result of their decompression. Burning-type dysesthesias were the commonest complaint of these patients. Although many other symptoms were often improved and a syrinx had decreased, this type of pain persisted and was notoriously difficult to treat with analgesics. It is important to inform a patient before surgery that burning-type dysesthesias may not respond to an otherwise successful decompression. Bernard Williams even observed postoperative aggravations in a few patients despite regression of a syrinx (personal communication). However, this was not observed in this series. Surgery was not recommended for most patients with a history of postoperative meningitis or after multiple procedures at the foramen magnum considering the increased risks of another intervention and the reduced chances for success under such circumstances.

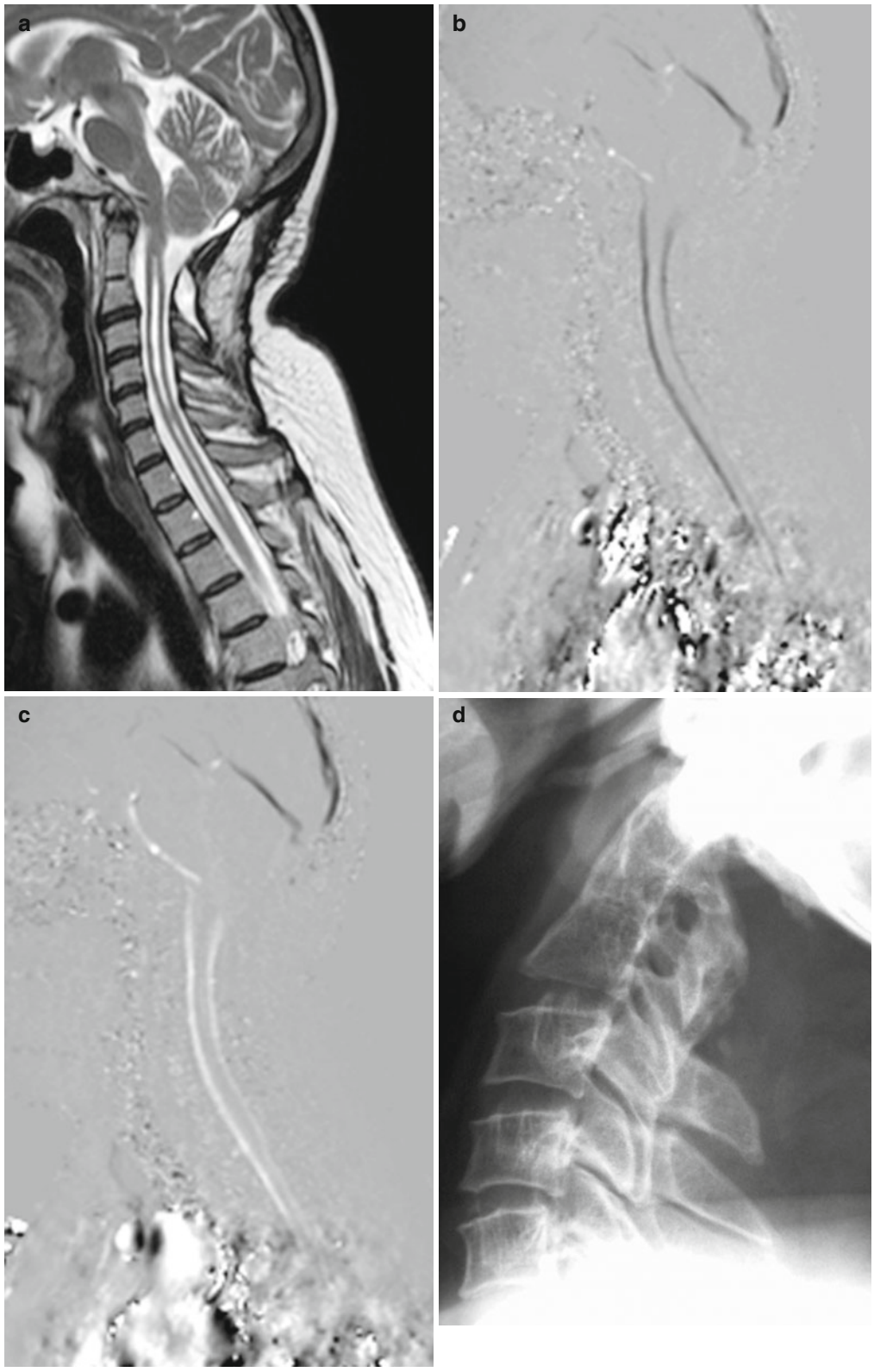
Fifty-one patients underwent another surgical procedure. The decision was based on a detailed clinical and neuroradiological analysis. Once hydrocephalus was ruled out, the evaluation started with the clinical history before the previous decompression and how preoperative symptoms responded to it. Was the neurology unchanged or improved or did symptoms progress further without an interval of stable neurologic function? If symptoms progressed without an interval of clinical stability, such a course suggested an insufficient operation. In most instances, this was related to untreated features of an associated basilar invagination, such as anterior compression by the odontoid or craniocervical instability.

In the majority of patients, however, the clinical history revealed a stable interval after foramen magnum decompression with or without improvement of preoperative symptoms. It should then be noted how and when the deterioration started. The longer the interval of clinical stability before the deterioration began, the less likely the cause was related to the foramen magnum. The only clinical symptoms, which pointed to a foramen magnum problem, were occipital headaches or swallowing dysfunctions. If these did not progress or reappeared, the clinical history often not indicated, whether a foramen magnum-related cause or another pathology, had to be addressed.

Next, a careful neuroradiological assessment was essential for these patients. The area of the previous operation was evaluated comparing pre- and postoperative MRI scans. Was there any evidence for an insufficient decompression or recurrent compression? It has been reported that new bone formation may cause recurrent compression in children [1–3]. This was not observed in this series, however. No example of cerebellar ptosis [4] due to an oversized craniectomy [5] resulting in medullary compression was found either.

Was there a basilar invagination with persistent anterior compression of the odontoid? Was there an indication of craniocervical instability such as an assimilated atlas to the occiput, a Klippel-Feil syndrome of the upper cervical spine, or a pannus formation around the odontoid [6] (Fig. 16.1)?

Fig. 16.1 (**a**) This sagittal T2-weighted MRI was performed 8 years after decompression of the foramen magnum in another institution in a 46-year-old patient with Chiari I malformation, basilar invagination, and syringomyelia. The syrinx appears of small caliber, and a small pseudomeningocele is apparent. C2/C3 is fused, i.e., Klippel-Feil syndrome. The patient complained about severe neck pain, dysesthesias, and a slight gait ataxia. (**b**, **c**) The cine MRI shows no flow signals in the foramen magnum region. (**d**, **e**) Functional X-rays of the cervical spine demonstrate the laminectomy of C2 and C3 and instability at C3/C4. Revision surgery incorporated a revision at the foramen magnum with arachnoid dissection and a new duraplasty followed by occipitocervical fusion C0–C5 with lateral mass screws. (**f**) The postoperative MRI demonstrates a large cisterna magna. (**g**) The control X-ray 7 years later shows the correct positions of all implants with a good sagittal profile. Postoperatively, the patient has remained neurologically stable for 7 years with some improvement of her neck pain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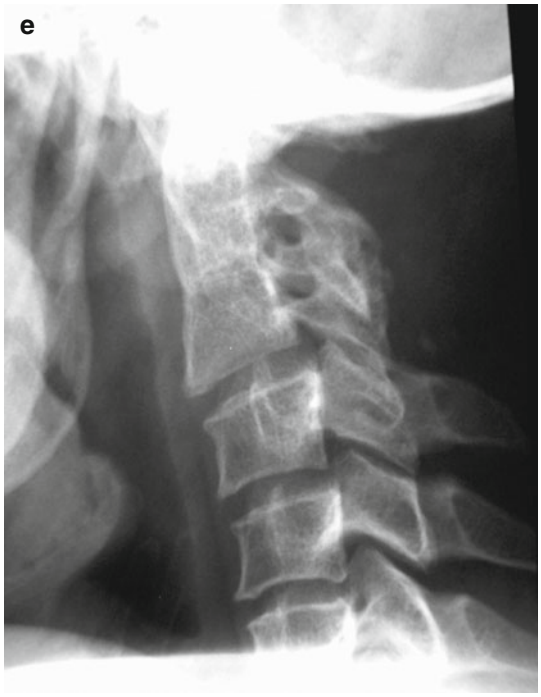
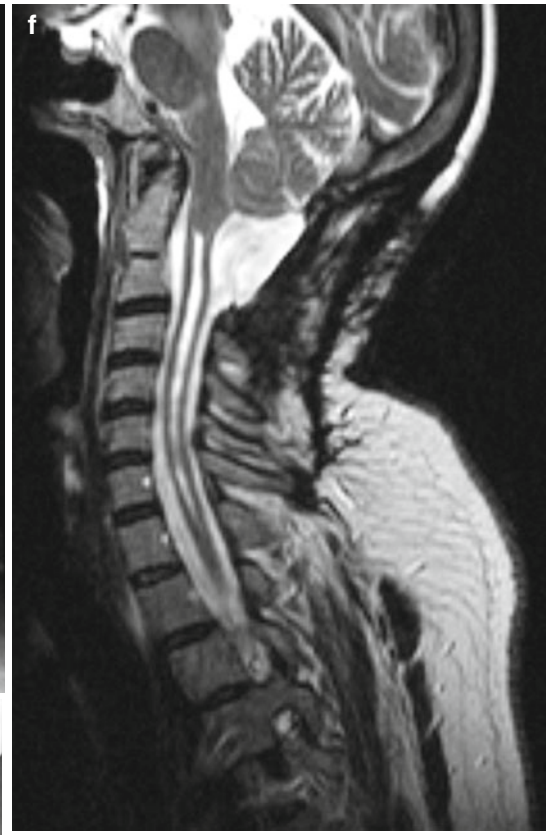




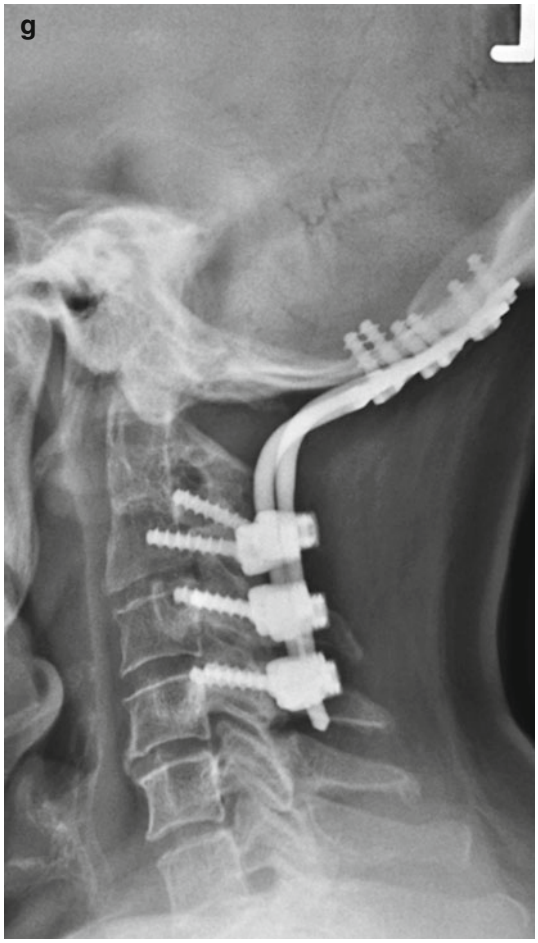


Fig. 16.1 (continued)

Was there a cisterna magna of sufficient size (Figs. 16.1, 16.2, and 16.3)? Was there a pseudomeningocele pushing the dura anteriorly [7] (Figs. 16.1, 16.2, and 16.3)?

Another important aspect was the postoperative course of a syrinx. If the syrinx decreased after surgery and remained so, it was unlikely that new symptoms were related to the foramen

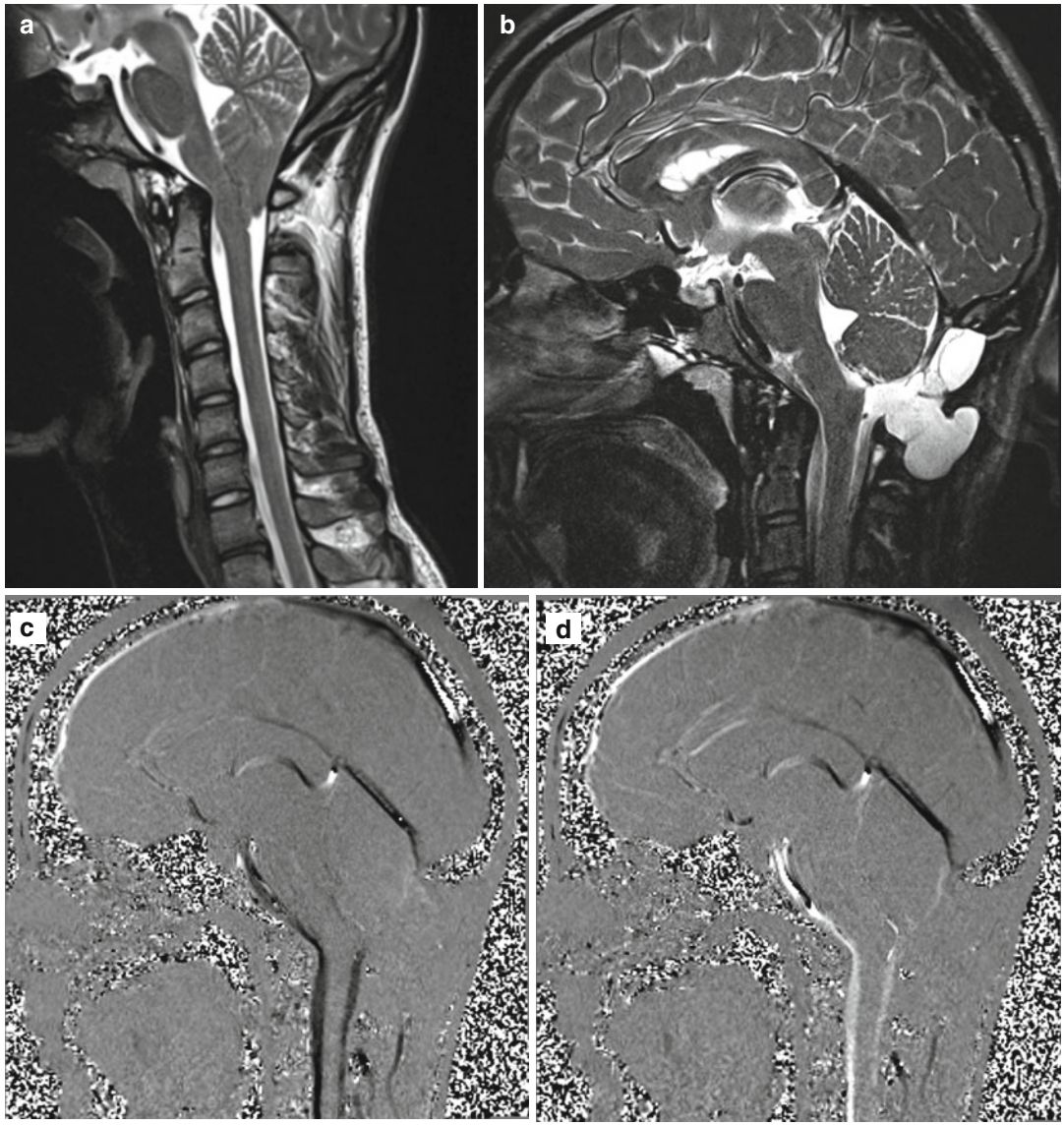


Fig. 16.2 (**a**) This sagittal T2-weighted MRI shows a classical Chiari I malformation without syringomyelia in a 15-year-old boy with occipital headaches. (**b**) After decompression of the foramen magnum with resection of both tonsils in another institution, the postoperative scan demonstrates a large pseudomeningocele. There appears to be a membrane obstructing the foramen of Magendie. (**c**, **d**) The cine MRI shows no flow signals in the area of the foramen magnum. The patient no longer complained about occipital headaches but reported quite severe local discomfort. At reoperation 2 years later, a large defect in the suture line for the duraplasty was evident. After removal of the duraplasty, profound scarring at both tonsillar stumps was detected. Both posterior inferior cerebellar arteries were embedded in this scar tissue which also obstructed the foramen of Magendie. The foramen was not opened to avoid any vascular injuries and a new duraplasty inserted. (**e**) The postoperative scan shows a free CSF passage across the foramen magnum with normal soft tissue healing. The patient made a full recovery

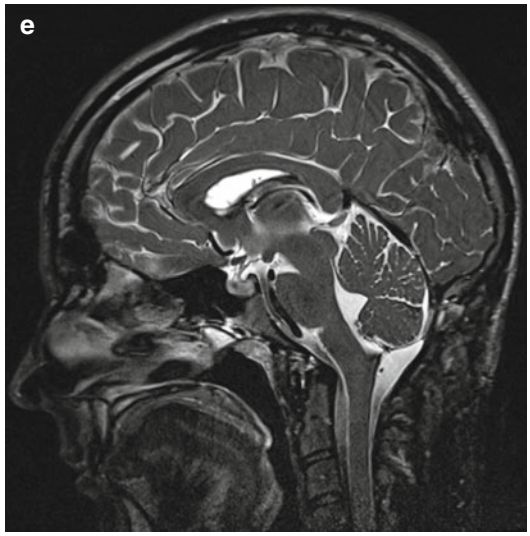
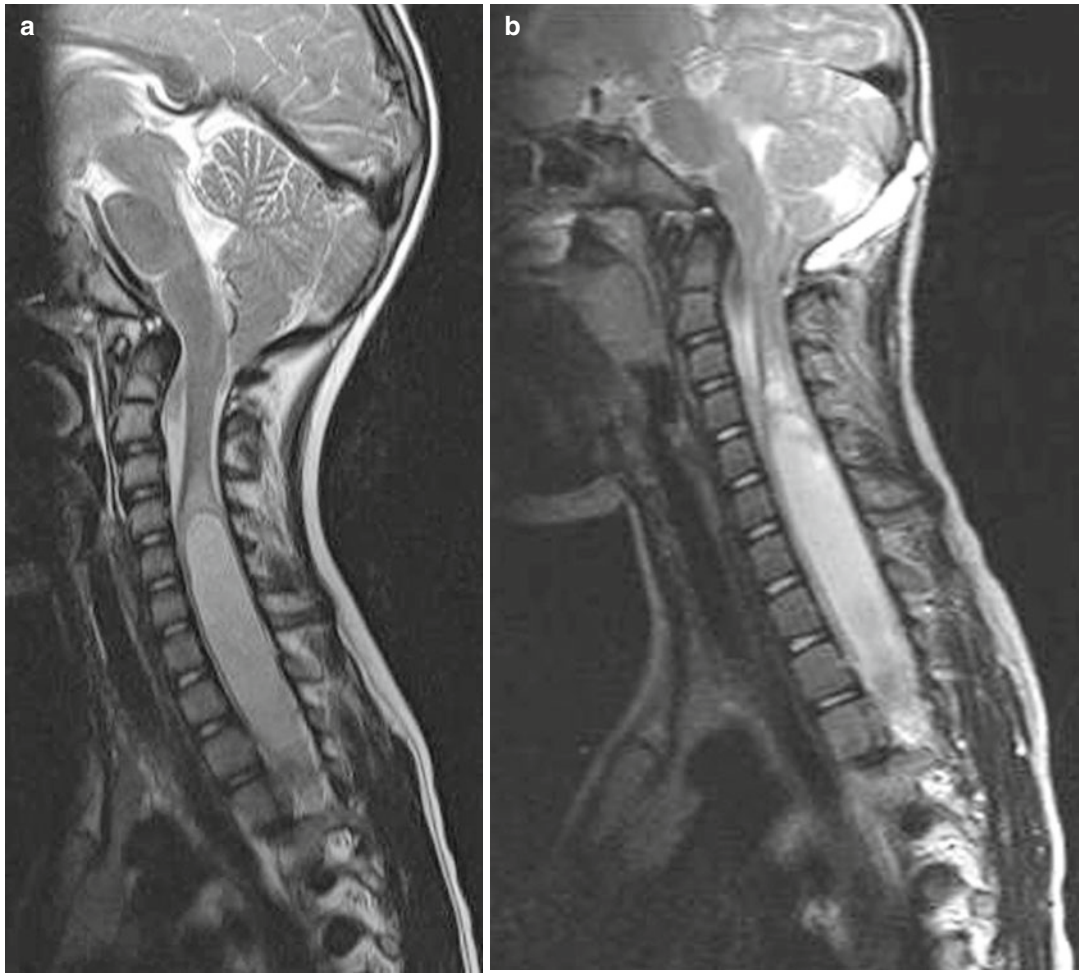


Fig. 16.2 (continued)

magnum with one exception: craniocervical instability still had to be ruled out.

If all these points were excluded by conventional MRI scans, a cardiac-gated cine MRI was performed to evaluate the CSF passage at the foramen magnum. This modality is the most sensitive method to detect or exclude arachnoid scarring and adhesions that may have formed after the first decompression [8–11]. If such a study demonstrated CSF flow at the foramen magnum and the neuroradiological evaluation had excluded all the other above-mentioned possibilities, then the clinical deterioration had to be caused by a process unrelated to the previous decompression.

In patients with syrinx shunts, the shunt catheter might have caused tethering of nerve roots or spinal cord [12] leading to radicular or myelopathic symptoms, which were often provoked by



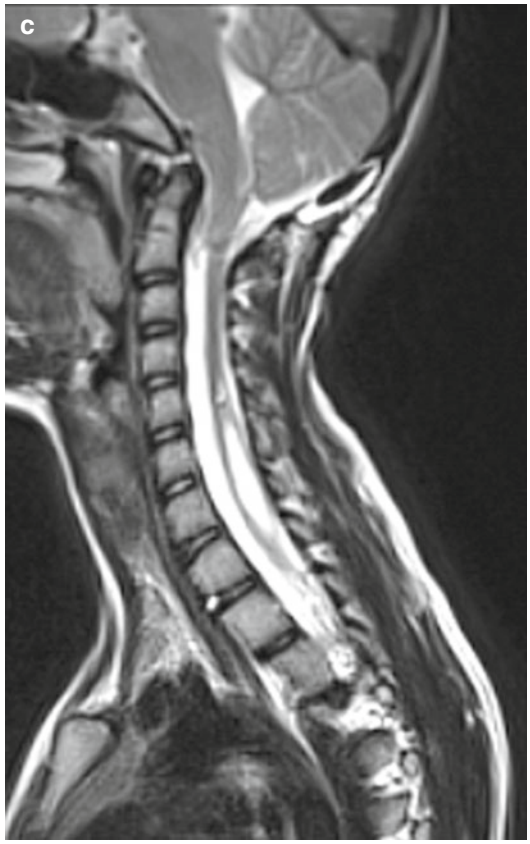


Fig. 16.3 (continued)

neck or arm movements. The MRI in these patients showed adherence of the cord to the dura at the level of the shunt.

If this had been excluded as well, degenerative changes of the cervical spine were evaluated next (Fig. 16.4). Many patients with a well-treated Chiari malformation and a collapsed syrinx demonstrated a considerable amount of spinal cord atrophy as the result of the long-standing syringomyelia. Therefore, MRI scans often gave the impression that a slight or moderate degree of cervical stenosis may not be clinically relevant. However, this is a very dangerous assumption. Such patients had very little functional reserve in their spinal cord as a consequence of their former syringomyelia. Any additional affection – even a minor one – may be enough to cause significant new deficits. It has even been suggested that Chiari patients may be particularly prone to degenerative problems of the cervical spine [13]. Signs of hypermobility of cervical segments should be looked for in particular by X-rays in ante- and retroflexion (Fig. 16.4).

Patients requiring a foramen magnum revision were significantly younger (40 ± 17 years vs. 48 ± 14 years; *t*-test, $p = 0.046$) with trends for a shorter interval between previous decompression and onset of new symptoms (40 ± 34 months vs. 63 ± 49 months; *t*-test, $p = 0.06$) and a longer history before the secondary operation (52 ± 98months vs. 34 ± 65 months; *t*-test, $p = 0.2$). Table 16.1 gives an overview on symptoms at presentation for unoperated patients and patients operated again at the foramen magnum or elsewhere in the spinal canal. The percentage of patients suffering from neuropathic pain was equal in all three groups. For the remaining symptoms, unoperated patients were less severely affected compared to the surgical groups. Patients undergoing a foramen magnum revision presented occipital pain and swallowing problems more commonly, whereas hypesthesia and sphincter disturbances were less common in this group. Otherwise, the neurological courses of patients with either a new foramen magnum problem or a cervical myelopathy were indistinguishable.

Secondary Surgeries in the Cervical Spine

In 15 instances, a mechanism independent from the foramen magnum region had caused a myelopathy (Table 16.2; Fig. 16.4). In all these

Fig. 16.3 (**a**) This sagittal T2-weighted MRI shows a Chiari I malformation with a substantial syrinx and scoliosis in a 5-year-old girl. (**b**) After decompression, a pseudomeningocele had formed pushing the duraplasty anteriorly, obstructing CSF flow. Consequently, the syrinx did not resolve. Seven years after the first operation, the scoliosis deteriorated and the decision was made to revise the foramen magnum. (**c**) After this revision which included arachnoid dissection and insertion of a new duraplasty, the CSF pathway is free and the syrinx has started to decrease. There has been no further progress of her scoliosis

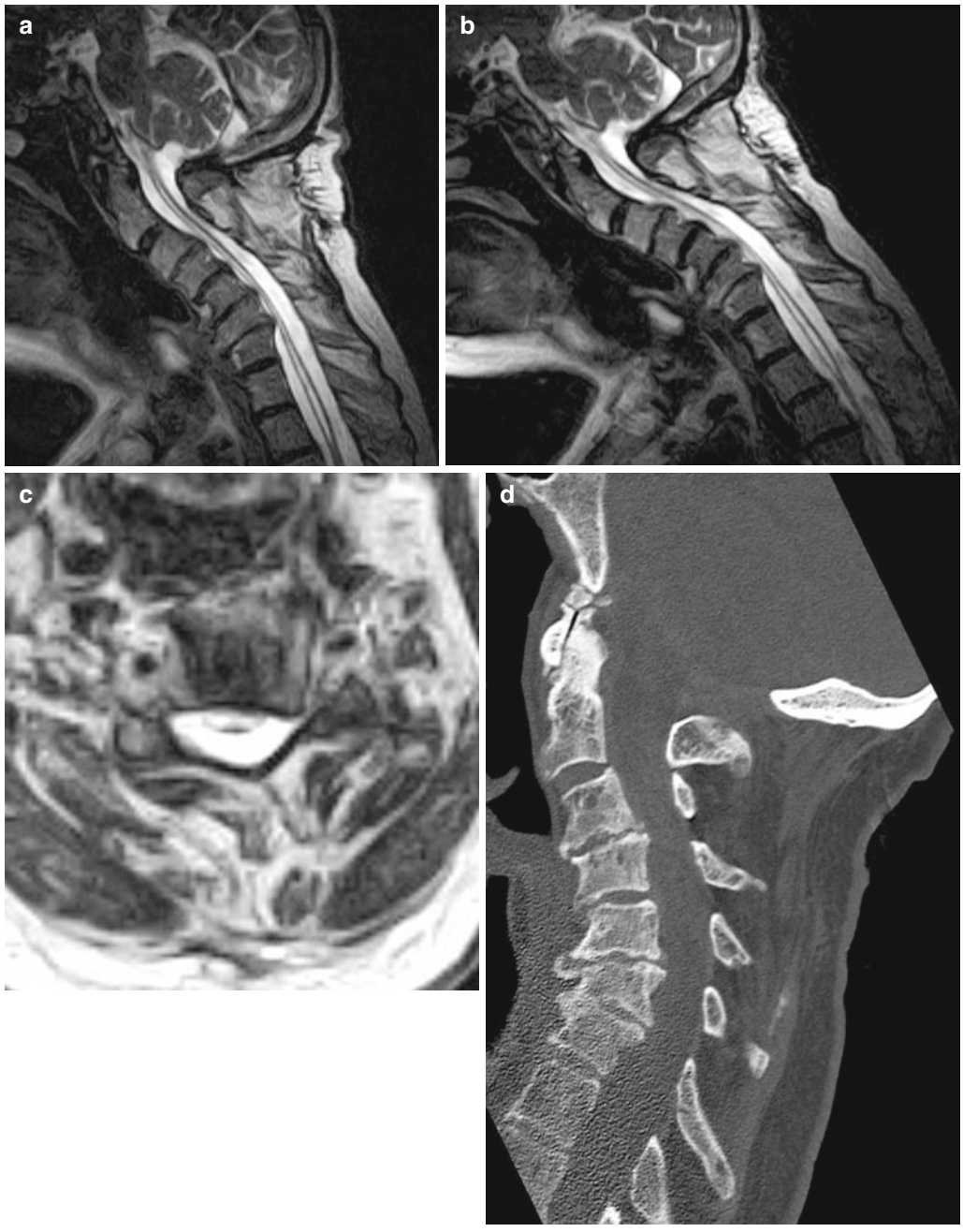
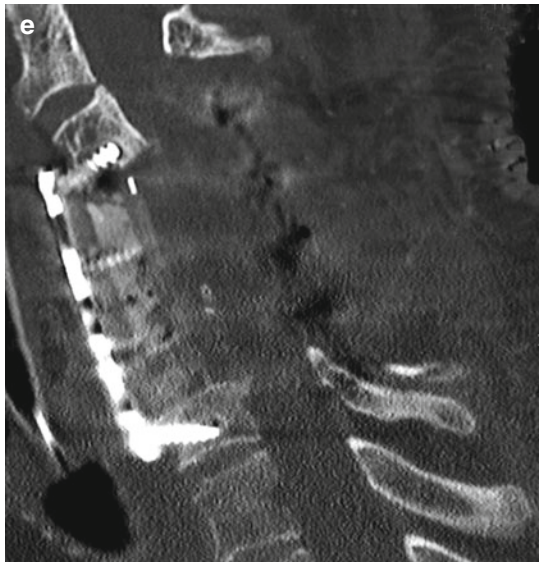


Fig. 16.4 (**a**) This sagittal T2-weighted upright MRI was taken 9 years after decompression of the foramen magnum in neutral position and demonstrates profound spinal cord atrophy, a collapsed syrinx, a free CSF passage at the foramen magnum, and a multilevel osteochondrosis in his cervical spine with a kyphotic deformity in a patient now 74 years of age. He suffered a progressive tetraparesis confining him to a wheelchair with increasing weakness of his respiratory muscles and loss of upper extremity functions. (**b**, **c**) With inclination of his head, the compression of the cord by osteophytes is evident. (**d**) The sagittal CT reconstruction shows the multiple osteochondroses and the swan neck deformity. The patient underwent a combined decompression with corpectomies C4–C6, reconstruction and ventral fusion C3–C7 followed by posterior decompression C3–C6, and fixation with lateral mass screws C3–C7. The postoperative CT reconstruction (**e**) and lateral X-ray (**f**) demonstrate a good sagittal profile with decompression of the cervical cord. Postoperatively, he made a slow recovery. Four months after surgery, he is able to walk again for about 20 m and is gaining strength and coordination skills in his hands. Respiratory functions have improved only slightly



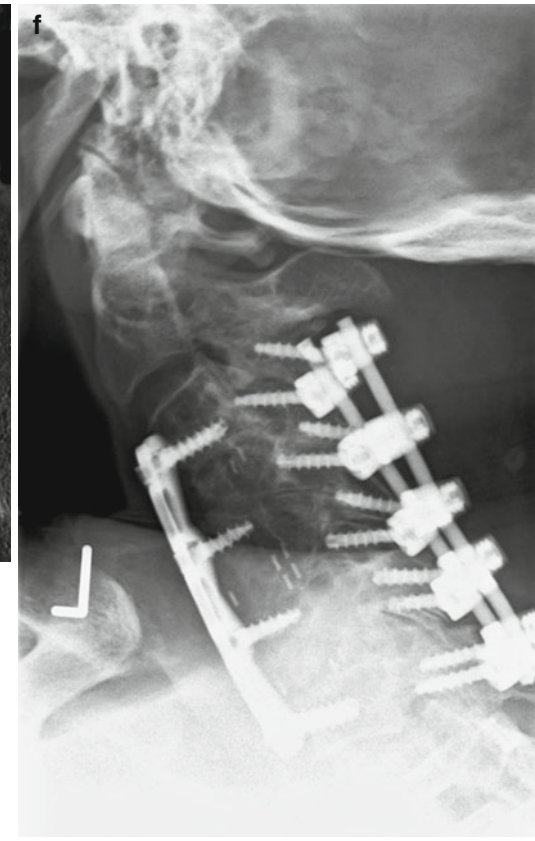


Fig. 16.4 (continued)

Table 16.1 Clinical symptoms for patients presenting after foramen magnum decompression

Group	Occ. pain (%)	Neurop. pain (%)	Hypesthesia (%)	Gait (%)	Motor power (%)	Sphincter function (%)	Swallowing function (%)
No surgery	72	38	58	58	51	5	17
FM group	84	35	73	81	62	19	27
Spinal group	75	38	94	75	69	31	12
Total	77	37	69	69	68	19	20

Abbreviations: *Occ*. occipital, *Neurop*. neuropathic, *FM* foramen magnum

Table 16.2 Operations for patients after foramen magnum decompressions

Group	Ventral fusion	Posterior dec. + fusion	Catheter removal	FM revision	FM revision + fusion	VP shunt
FM group				35	10	1
Spinal group	8	2	5			

Abbreviations: *Dec* decompression, *FM* foramen magnum, *VP shunt* ventriculoperitoneal shunt

patients, the Chiari malformation had been adequately treated with collapse of the syrinx and a free CSF passage at the foramen magnum. Five syrinx shunt catheters were removed to release a postoperative tethering of either nerve roots or spinal cord. In each of these patients, pain and dysesthesias were provoked with certain body movements. The tethering had not caused reappearance of the syrinx in any of them.

For removal of a syrinx catheter, the sharp microsurgical dissection concentrated on untethering nerve roots and spinal cord first. Once this was achieved, the catheter could be removed in most instances. If it was stuck in the cord, it was transected right at the entry point into the spinal cord.

For patients with degenerative disc disease, it was the general policy to restrict one- or two-level ventral fusions to patients with radicular symptoms, whereas multilevel posterior decompressions and fusions were preferred for patients with a progressive myelopathy. This strategy was based on observations that patients with a progressive cervical myelopathy almost always displayed a profound spinal cord atrophy due to the former syringomyelia and often demonstrated multilevel hypermobilities of the cervical spine. The intention was to prevent future deteriorations from adjacent levels in patients with a significantly reduced functional reserve.

Six patients underwent seven ventral fusions for single- or two-level disc disease of the cervical spine. One of these underwent an additional posterior cervical decompression and fusion 10 years later when she developed a progressive myelopathy. One patient underwent a combined anterior and posterior decompression and fusion for a swan neck deformity (Fig. 16.4). Finally, one patient received a posterior decompression and fusion only. Posterior decompressions consisted of laminectomies C3 to C6 with lateral mass fixation (Table 16.2).

There were no surgical morbidity or complications in the spinal group. Looking at individual symptoms, a trend for improvements of pain, sensory disturbances, and dysesthesias was observed. Other neurological signs such as motor weakness or gait problems tended to remain unchanged. At 3 months postoperatively, three of five patients reported improvement after syrinx catheter removals. All but one ventral fusion of the cervical spine resulted in some clinical improvement at that time. Posterior decompressions and fusions were followed by improvement after both operations.

In the long term, two patients developed adjacent-level disease in the cervical spine after ventral fusions and underwent another ventral or posterior operation each, which stabilized the status. After catheter removal, one patient experienced another deterioration due to postoperative scar formation 4 months after surgery. Her past history had been complicated by meningitis after the initial foramen magnum procedure and no further operation was undertaken.

Table 16.3 Pathological findings in 45 foramen magnum revisions

Feature	Number of operations
Pseudomeningocele	8
Adhesion of graft to cerebellum	24
Adhesion of graft to spinal cord	5
Slight arachnoid scarring	7
Severe arachnoid scarring	24
History of meningitis	3
Obstruction of foramen of Magendie	20
Ventricular dilatation	7
Anterior compression by odontoid	1
Craniocervical instability	10

As multiple features were often found in a single operation, the total sum is higher than the number of revisions

Secondary Surgeries at the Foramen Magnum

Apart from one patient in this group requiring a ventriculoperitoneal shunt for late postoperative hydrocephalus, the patients in this subgroup demonstrated either an untreated or new instability of the craniocervical junction, an insufficient decompression, or an obstruction of CSF flow at this level (Figs. 16.1, 16.2, and 16.3). CSF flow obstructions were related to arachnoid scarring or compression of the cisterna magna by a pseudomeningocele. Combinations of these different mechanisms were common (Table 16.3).

In a previous publication, the lack of effect of syrinx shunts in patients with a Chiari I malformation and syringomyelia was demonstrated [14]. Therefore, such shunts were never considered

for patients after a failed decompression. If a syrinx had not regressed or reappeared, the reason had to be looked for and treated at the foramen magnum. This required a revision with opening of the dura exchanging the duraplasty, arachnoid dissection with establishment of a free outflow from the foramen of Magendie, and insertion of a new duraplasty using alloplastic rather than autologous material. Several authors have mentioned the importance of opening this foramen during foramen magnum decompressions [1, 15, 16] and especially in revisions [16, 17].

Forty-five revisions at the foramen magnum were performed (Figs. 16.2 and 16.3), of which ten were combined with a posterior craniocervical fusion (Fig. 16.1), where one revision included transoral resection of the odontoid and posterior decompression and fusion in a second operation. In four instances, the revision was restricted to craniocervical fusion only as no CSF flow obstruction was detectable on preoperative imaging and intraoperatively using ultrasound (Table 16.2).

Severe arachnoid scarring was the commonest feature in patients demonstrating a CSF flow obstruction [18–22] and detected in 31 instances (94 %) in the form of adhesions either between dural graft and cerebellum and spinal cord in 29 operations (88 %) or obstruction of the foramen of Magendie in 20 revisions (61 %) (Table 16.3). Whereas the adherence of the dura graft to underlying nervous tissue was due to either pseudomeningocele formation pushing the dura graft anteriorly [7], the suture material, autologous graft material, or insufficient arachnoid dissection at the first operation, the most severe arachnoid scarring at the foramen of Magendie was encountered after obex plugging, resection of tonsils (Fig. 16.2), or in patients with a history of meningitis [14, 18, 23].

The major problem of preoperative evaluation was the severity of arachnoiditis. The more extensive and dense the arachnoid pathology, the less the probability that a revision may produce a lasting benefit and the higher the risk of surgery. Unless there was a history of meningitis or a clear description of severe arachnoid changes in the operation notes, it was almost impossible to foresee exactly what would be discovered after opening of the dura. Thus, it is difficult to judge the prognosis for a patient before revision surgery. This needs to be discussed with the patient. Reexploration of the foramen magnum is to some degree a diagnostic procedure in order to find out why the first operation did not provide the desired result. Depending on the intraoperative findings, a surgical strategy had to be adopted which improved CSF flow but minimized the risk of postoperative arachnoid scarring, which may again lead to CSF flow obstruction and prevent a long-term benefit. Limiting the arachnoid dissection to the midline with sharp transection of arachnoid adhesions obstructing the foramen of Magendie and the posterior spinal subarachnoid space was all that was required. Blunt dissection or preparation of arachnoid adhesions laterally carries the risk of damage to small perforating arteries and caudal-cranial nerves and should be avoided. Finally, a spacious dura graft using alloplastic material provided reasonable protection against postoperative arachnoid scarring which may otherwise cause another clinical recurrence.

Complications were encountered in 22.2 % of foramen magnum revisions with CSF fistulas being the most common after four operations (8.9 %). Surgical morbidity was observed after four revisions (8.9 %) and encountered exclusively among patients who had undergone their first decompression at other institutions. Compared to patients undergoing a first foramen magnum decompression, the overall complication rate for foramen magnum revisions was similar [24], even though the rate of CSF leaks was higher.

A postoperative improvement after 3 months was reported after 65.1 % of operations, while 23.3 % resulted in no postoperative change and neurological worsening was evident after 11.6 % of revisions. Looking at individual symptoms in the first postoperative year revealed improvements for pain, sensory disturbances, and gait. The remainder of symptoms tended to be left unchanged. Improvements tended to be marginal and of little functional significance. Similar experiences have been made for patients with

severe foramen magnum arachnoiditis of other causes [25]. The realistic outlook for patients undergoing a foramen magnum revision was clinical stabilization of the previously progressive course.

Long-term results determined by Kaplan-Meier statistics revealed a recurrence rate of 34 % within 10 years. Two patients with such a recurrence underwent a third foramen magnum operation without long-term success. Both had been accompanied by severe arachnoid pathology related to obex plugging with muscle or previous meningitis, respectively.

Conclusions

Patients presenting with progressive neurological symptoms after a foramen magnum decompression for Chiari I malformation require a detailed clinical and radiological work-up to identify the responsible mechanism. Not only does the foramen magnum area need a careful analysis, but degenerative diseases of the cervical spine should also be taken into account. Particularly important are signs of instabilities as the often atrophic spinal cords of these patients may be extremely vulnerable to hypermobile segments. Multilevel decompressions and fusions may stabilize the course in such patients. Foramen magnum revisions are indicated in patients with evidence of CSF flow obstruction, cord compression, or instabilities at this level. They carry a higher surgical morbidity and are less likely to produce significant neurological improvements compared to a primary decompression. However, about 66 % can be stabilized with such a revision for at least 10 years.

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