11 Post-traumatic and Post-inflammatory Syringomyelia

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Contents

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11.1 Introduction

Most cases of syringomyelia resulting from pathology at the craniovertebral junction are not associated with a significant amount of local scar tissue formation. In contrast, the majority of cases of syringomyelia caused by obstruction elsewhere in the spinal canal have, as the primary underlying pathology, cicatrix within the leptomeninges. This condition is often referred to as arachnoiditis, but this is something of a misnomer because, when operating upon such conditions, we do not encounter active inflammation. Rather, we see mature scar tissue, which is the end result of an earlier inflammatory process.

The most common initiator of scar tissue formation is blood shed into the subarachnoid channels. Organisation of blood clot into scar tissue, anywhere in the body, is part of the normal healing process, but it can lead to problems when excessive adhesions form, be they in the peritoneal cavity, around a lumbar nerve root or within the spinal theca. The density and extent of this scar tissue varies, according to the underlying cause. In the spinal canal, causes include subarachnoid haemorrhage, from intracranial or intraspinal sources, intradural surgery and spinal trauma. The latter need not be particularly severe or even associated with spinal cord injury.

The second important cause of inflammation, with subsequent scar tissue formation, is infection, be this pyogenic, tuberculous or due to other microorganisms. A third cause, now only seen occasionally, is chemically induced meningitis,

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caused by radiographic contrast media. Finally, we sometimes encounter thin arachnoid webs, the origin of which is not always clear. Some may be congenital in origin, but others may have resulted from undiagnosed episodes of leptomeningeal inflammation, earlier in life.

What we do not know is just how frequently arachnoid scarring leads to the formation of syrinx cavities and how often such scar tissue exists without an associated syringomyelia. Neurosurgeons are unlikely to explore a cicatrised spinal canal in the absence of associated cord cavitation, so operative experience cannot answer these questions. Experience with spinal cord injuries suggests that syrinx cavities are generated in only a minority of cases, albeit not insignificant in number (see below). If we then assume that the various pathologies mentioned above will always be associated with some scar tissue formation, we must ask ourselves what other factors determine just when a syrinx will form.

There are other noninflammatory pathologies within the spinal canal, which can lead to syrinx formation. These include intramedullary tumours and dysraphic abnormalities, but this chapter is concerned with those post-arachnoiditic conditions that lead to syringomyelia. Idiopathic syringomyelia is also dealt with in a separate chapter, as is syringomyelia caused by fibrosis at the craniovertebral junction.

The filling mechanism underlying the formation of syringomyelia cavities is discussed in detail in Chap. 6. Suffice it to say here that the normal movement of CSF throughout the spinal canal depends upon there being an uninterrupted column of fluid, outside the cord, along the entire length of the spine. Interruption of this column results in abnormal dissipation of arterial and venous pressure waves, which pass up and down the spinal canal, with the result that fluid accumulates within the cord itself.

11.2 Post-traumatic Syringomyelia

This is the most commonly encountered variety of post-inflammatory syringomyelia (Fig. 11.1) although other forms share much in common with it, as regards pathogenesis and treatment

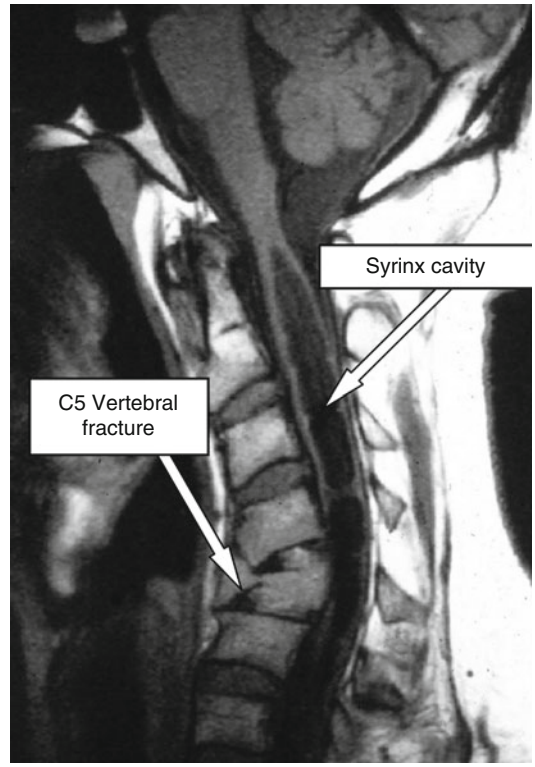

Fig. 11.1 Typical MR appearances of post-traumatic syringomyelia (Reproduced with kind permission of the Ann Conroy Trust)

options. It is generally assumed that post-traumatic syringomyelia cavities originate from local ischemic changes within the cord. Consequently, they are thought to represent "extracanalicular" syrinxes, rather than "hydromyelic" dilatations of the central canal (Milhorat et al. 1995a, b), although some histological studies suggest that post-traumatic syrinx cavities may not be entirely extracanalicular in origin (Reddy et al. 1989; Squier and Lehr 1994).

It is important to distinguish primary, post-traumatic cysts from true syringomyelia cavities. The former are more common and are seen on magnetic resonance imaging (MRI) scans in half of all spinal cord injury victims (Backe et al. 1991). They are the result of primary damage to the cord and are located at the level of the original injury. They are usually small and rounded or ovoid and do not produce a great deal in the way of cord expansion (Perrouin-Verbe et al. 1998). Post-traumatic syringomyelia, on the other hand, refers to propagating cavities, extending beyond the level of the original trauma. These

develop as a secondary, delayed consequence of the original injury.

Post-traumatic syringomyelia is important in two respects. Whereas syringomyelia remains an uncommon condition in the community as a whole, it is very common in the population of spinal cord injury victims. Quoted figures vary, but even early estimates of about 5 % represent a significant incidence, more even than common medical disorders such as diabetes or asthma. Later publications give figures that are higher still, at around 20 %. These are based on both radiological surveys (Perrouin-Verbe et al. 1998; Squier and Lehr 1994; Wang et al. 1996) and post-mortem studies (Squier and Lehr 1994). The differences in these figures reflect the fact that earlier estimates were derived when MR imaging was carried out selectively, as indicated by the onset of new neurological symptoms (El Masry and Biyani 1996). The higher estimates also depend upon a definition of syrinx cavities as being those that extend two or more segments beyond the level of the original injury. If the definition stipulated three segments, then the incidence would fall back to single figures (Pearce 1995). A reasonable working rule, therefore, would be to say that up to one in five of all spinal cord injury victims develop anatomical syringomyelia and that at least 1 in 20 will develop symptoms.

The second point to note is that most victims of spinal cord injury already have to contend, in their day-to-day life, with significant neurological disability. The devastating transformation that spinal cord injury brings about in somebody's life is very apparent, and it is a cruel irony when the development of a post-traumatic syringomyelia cavity threatens the individual with further loss of independence. Yet most patients cope remarkably well, albeit requiring a good deal of support. They are often the least complaining of all the patients who come to our clinics. Few conditions in neurosurgery challenge our skills, as surgeons and as doctors, to the same extent.

11.2.1 Presentation

Most spinal cord injury patients living in economically privileged societies are currently offered regular surveillance of their neurological and general status. Their supervising units have a low threshold for carrying out MR imaging if problems develop. Indeed, there is a case for offering routine screening for all victims of spinal cord injury, looking specifically for post-traumatic syringomyelia (Sett and Crockard 1991). Syrinx cavities are now being detected with increasing frequency, often in patients with little in the way of new neurological symptoms.

Table 11.1 Common presenting features of post-traumatic syringomyelia

Symptoms
Increasing pain
Hyperhidrosis[a]
Increasing spasms
Loss of sensation
Loss of trunk control
Reduced dexterity
Altered bladder function
Autonomic dysreflexia
Signs
Ascending sensory level
Focal motor deficits
Loss of upper limb reflexes
Horner's syndrome

[a]Glasauer and Czyrny (1994), Stanworth (1982)

Many patients do display clear indications of having developed a complication of their original spinal cord injury (Table 11.1). In most published series, pain is at the top of the list of presenting symptoms. The differential diagnosis of the symptoms of post-traumatic syringomyelia includes constipation, pressure sores and urinary tract infections, causing sweating attacks and spasms, and ulnar nerve palsies resulting from repeated pressure being placed on the elbows, during transfers. As with all forms of syringomyelia, there is a poor correlation between the size of the cavity and the magnitude of the clinical features.

An impressive feature of post-traumatic syringomyelia is the wide variation in the latent interval between the original spinal cord injury and the first onset of symptoms arising from the syrinx. The range extends from a few months to several decades. Attempts have been made to predict which spinal cord victims might go on to develop post-traumatic syringomyelia, and although individual studies have pointed to one factor or another (Table 11.2), there are currently

Table 11.2 Possible predictors of development of post-traumatic syringomyelia

Level	Cervical vs dorsal spine
Severity	Complete vs incomplete functional transection Displaced vs undisplaced fracture
Age	Older vs younger patients
Management	Conservative vs surgical management Fixation with or without bony decompression
References	Brodbelt and Stoodley (2003a), El Masry and Biyani (1996), Klekamp and Samii (2002), Levy et al. (1991), Vannemreddy et al. (2002)

For each proposed indicator listed here, the item underlined has been proposed as a possible indicator of increased likelihood of a spinal cord injury victim going on to develop post-traumatic syringomyelia

no reliable predictors. Lifelong surveillance remains the only safe option.

The generation of symptoms and neurological deficits probably relates, in part at least, to hydraulic pressure within the syrinx, leading to local ischemia, as well as stretching of decussating fibres and long tracts (Milhorat et al. 1997; Young et al. 2000). The magnitude and duration of raised pressure may account for whether or not these clinical features are reversible. Another mechanism is tethering, leading to traction on the cord during normal movement of the spinal column. Release of tethering may, in some cases, be at least as important as collapse of the syrinx, when it comes to gaining some clinical improvement (Ragnarsson et al. 1986). It has also been suggested that leptomeningeal fibrosis may lead to cord ischemia, but it is difficult to substantiate or refute such suggestions.

11.2.2 Syringomyelia Following Minor Trauma

A particular category of lesions is that of cavities detected following relatively minor injuries, such as whiplash or a fall. Victims may complain, often after an interval, of a variety of neurological symptoms, yet there may be no accompanying physical signs. MR imaging is carried out, sooner or later, and the sort of lesion revealed may consist of a short, spindle-shaped intramedullary cavity, without any associated abnormality at the craniovertebral junction and without any obvious obstruction elsewhere in the spinal canal (Fig. 11.2). Not uncommonly, medicolegal experts become involved and discussions centre on whether the cyst was caused by the accident or was pre-existing. Questions arise as to whether it is a true syrinx or something else, such as a glioependymal cyst. The latter are well documented as intracranial lesions but also occur within the spinal cord. Radiologically, they are usually short but relatively plump in size, being quite well defined but not enhancing with contrast injection

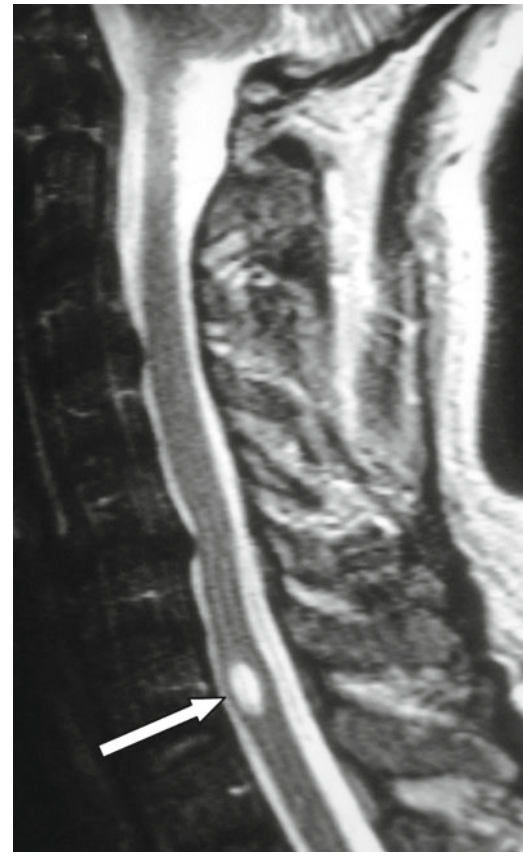

Fig. 11.2 Short, spindle-shaped cavity (*arrow*). Revealed during the course of investigation for pain and somatic sensory disturbances, following minor trauma, this could represent a small, post-traumatic syrinx or a pre-existing glioependymal cyst, rendered symptomatic by the injury

(Robertson et al. 1991; Saito et al. 2005). Legal debate continues as to whether, irrespective of its nature, the cavity was rendered symptomatic by the injury or was simply an incidental finding, having nothing to do with the claimant's symptoms. Onset of symptoms immediately after the injury suggests a pre-existing cavity, rendered symptomatic by the event. Delayed onset suggests that the cavity was caused by the trauma (Barnett 1973). To declare that the presence of a syrinx bears no relationship to a prior injury, or to suggest that neurological symptoms do not arise from the lesion, ignores the temporal association between the injury and detection of the syrinx. This legal issues are discussed in more detail in Chap. 18, but it is fair to say that there is little in the literature to guide an expert witness, beyond the fact that any series of post-traumatic syringomyelia cases may well include a significant number where the initial trauma was moderate and where no neurological deficits were evident at the time (Klekamp and Samii 2002; LaHaye and Batzdorf 1988).

11.3 Subarachnoid Haemorrhage

Relative to the overall incidence of subarachnoid haemorrhage, the occurrence of spinal arachnoid adhesions, as a complication, is low (Augustijn et al. 1989; Tjandra et al. 1989). The incidence of syrinx formation, as a further sequel, is distinctly rare, although cavities can develop following both intracranial and intraspinal subarachnoid haemorrhage (Siddiqi et al. 2005). What determines why and when this complication occurs is unknown. It may be that, if a patient remains recumbent for any period, after a subarachnoid haemorrhage, blood products pool on the spinal canal.

Clearly, any intradural surgical procedure carried out on the spine will result in some blood and tissue products being shed into the canal. This can result in formation of arachnoid adhesions, potentially resulting in syringomyelia. Once again, such complications are rare (Cusick and Bernardi 1995; Klekamp et al. 1997).

11.4 Contrast Media

Prior to the advent of MR scanning, the principal means of investigating the spinal canal and its contents was by myelography. Originally, this involved the use of oil-based contrast media although, subsequently, water-soluble agents were developed. Once injected into the spinal canal, this oily material would not be absorbed, and in some cases, an arachnoiditic reaction followed. Once these effects were recognised, it became the normal practice to aspirate as much of the contrast medium as possible, after the radiological study was completed. Some patients, nevertheless, developed meningeal fibrosis, with disabling consequences. A curious aspect of the problem was why only a minority of people developed this complication. One suggestion was that the inflammatory response depended upon a synergistic reaction between the contrast medium and something else, such as blood or even powder from surgical gloves.

Although largely a matter of historical interest these days, newly diagnosed cases of syringomyelia still present, occasionally, in somebody who underwent myelography, with an oil-based contrast medium, many years ago (Tabor and Batzdorf 1996).

11.5 Post-tuberculous Syringomyelia

Spinal cord involvement is a recognised complication of tuberculosis, and as with tuberculosis anywhere in the nervous system, different pathological manifestations are seen. These include acute inflammation and oedema of the cord, intramedullary abscesses and intramedullary or intradural granuloma formation (Muthukumar and Sureshkumar 2007). The most common consequence, however, is late adhesive arachnoiditis. Despite this, syringomyelia is a relatively uncommon sequel. Even surgeons who deal with a lot of cases of syringomyelia are unlikely to see many tuberculosis-related syrinxes, and most reports in the literature relate to no more than a

handful of patients (Kaynar et al. 2000; Moghtaderi et al. 2006; Schon and Bowler 1990).

Most cases present at an interval, after the underlying infection has been treated. As with post-traumatic syringomyelia, this latent interval can vary widely from case to case. Early presentation has also been described and the assumption is that acute cord inflammation is the underlying mechanism rather than arachnoid adhesions (Daif et al. 1997; Fehlings and Bernstein 1992).

Post-tuberculous adhesions are likely to be extensive, making the creation of a conduit all but impossible in most cases. It is in this type of syringomyelia that direct shunting may be a preferred option.

11.6 Other Infections

Syringomyelia is also recognised as an occasional complication of other infections involving the spinal cord. Organisms which have been reported include Listeria (Nardone et al. 2003), Cryptococcus (McLone and Siqueira 1976), Syphilis (Bulundwe et al. 2000; Mebrouk et al. 2011) and Candida (Phanthumchinda and Kaoropthum 1991). Syringomyelia is also occasionally seen in association with previous epidural inflammatory pathology (Klekamp et al. 1997). All these examples are much less common than post-tuberculous cases. It should, nevertheless, be part of the routine history taking, in all cases of syringomyelia where the cause is not immediately apparent, to enquire about a past history of meningitis, spinal trauma, head injury and subarachnoid haemorrhage.

11.7 Arachnoid Webs and Cysts

When we consider what might happen as a consequence of obstruction to CSF flow within the spinal canal, we might reasonably predict a build-up of fluid on the outside of the cord rather than the more usual finding of syrinx formation. Indeed, we do sometimes see such external, "hydraulic compression" of the cord, in association with arachnoid webs (Fig. 11.3). Such webs, as well as cysts (or pouches), may be congenital lesions or may arise as the result of earlier haemorrhage (Thines et al. 2005) or arachnoiditis from other causes (Gnanalingham et al. 2006; Gopalakrishnan et al. 2010). The obstruction caused by such webs may also result in syrinx formation (Mallucci et al. 1997). The likely mechanism, once again, is that normal arterial and venous pressure waves are not dissipated normally down the spinal canal, leading to creation of the syrinx cavity (Brodbelt and Stoodley 2003a, b; Holly and Batzdorf 2006). The syrinx is the more obvious abnormality on imaging and a careful search should be made to identify the web. Indeed, there may be occasions when it is only revealed at the time of surgical exploration.

When an operation is planned, the surgeon must realise that the lesion might be an arachnoid cyst rather than a simple web. The myelopathy, be this due to the pressure within the cyst or from a resulting syrinx, might not be relieved unless both the upper and lower limits of the cyst are broached.

11.8 Surgical Management

Compared with operations for hindbrain-related syringomyelia, surgery for cavities caused by spinal arachnoid fibrosis does not yield results that are as good overall. Indeed, this category of syringomyelia is one of the most difficult conditions that neurosurgeons are called upon to treat, at least in terms of gaining consistent results. It is not surprising, therefore, that various techniques have been described over the years (Edgar and Quail 1994). Further, not all syrinx cavities progress relentlessly (Anderson et al. 1986) and it is certainly the author's experience that many seem to enter a state of "hydrodynamic equilibrium" (see also Chap. 2). For all these reasons, surgical intervention should be reserved for those cases that show clear evidence of neurological deterioration. Not surprisingly, most published series include significant numbers of patients who do not go on to surgical intervention.

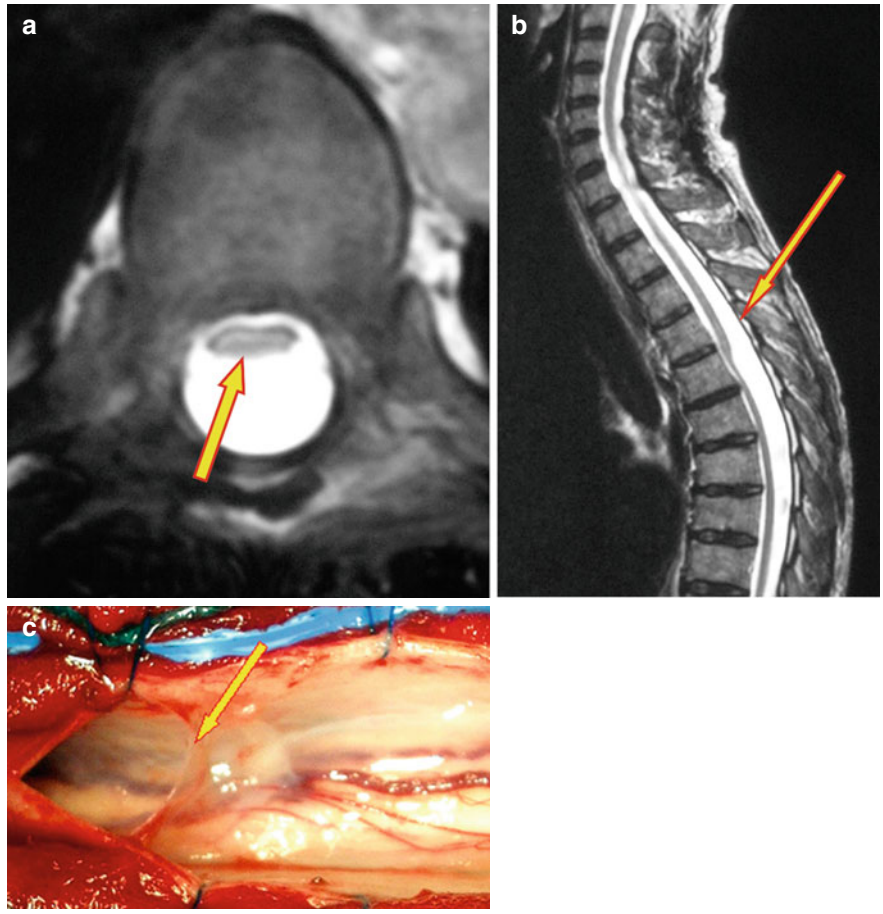

Fig. 11.3 Spinal arachnoid webs. (**a**) T2-weighted MRI, transverse section: the cord is clearly compressed flat, in the anterior half of the spinal canal (*arrow*). (**b**) T2-weighted sagittal MRI: the same feature is evident in the sagittal plane, where the level of the focal point of obstruction to CSF flow is very evident (*arrow*). (**c**) Operative appearances of the lesion. With the dura opened, a free edge forms along the dorsal aspect of the web (*arrow*). Extending anterior to this can be seen the thin membrane, which occupies the full cross-sectional area of the thecal sac

Table 11.3 Treatment options

Creation of a conduit for CSF flow
Direct drainage of the syrinx cavity
Lowering the overall CSF pressure
Conservative management

11.8.1 Creation of a CSF Conduit

There are, broadly speaking, three types of surgical manoeuvre that we can offer to a patient with syringomyelia (Table 11.3). Clearly, it makes sense to treat the underlying cause if possible. So, when we can identify a focal point of obstruction to CSF movement, the logical approach is to try and relieve that blockage and create a new conduit for CSF flow. This is, of course, a major operation, involving the removal of several laminae, and it requires microsurgical technique for the intradural part of the procedure. In most cases of post-traumatic syringomyelia, the scar tissue encountered is relatively limited in extent, and breaking it down is not unduly difficult. The offending cicatrix is clearly recognisable by its typical milky-white appearance and can be readily distinguished from normal, translucent arachnoid. Dissection dorsal and dorsolateral to the cord

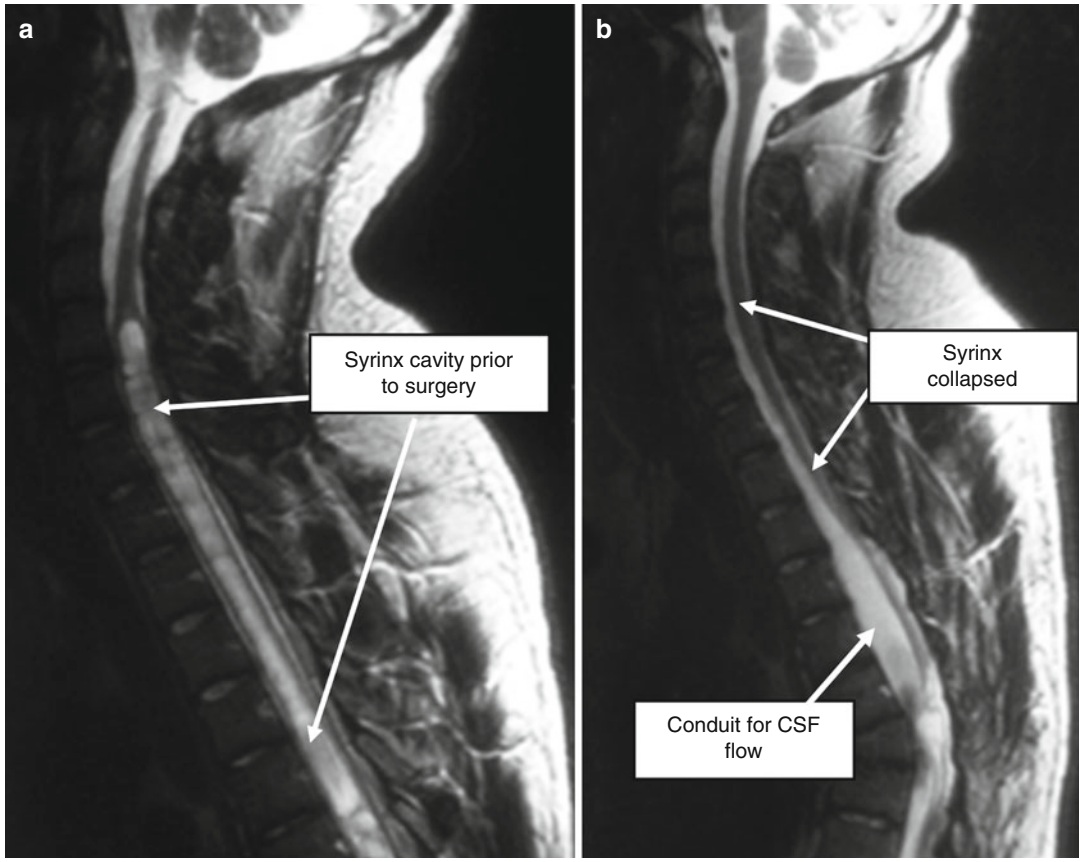

Fig. 11.4 Effective surgery for post-traumatic syringomyelia. Laminectomy, with release of intradural adhesions and creation of an artificial conduit for CSF flow, can produce very rewarding anatomical results, with arrest of clinical deterioration and sometimes recovery of lost function. (**a**) Prior to surgery. (**b**) Following laminectomy, release of scar tissue and creation of pseudomeningocoele, to act as an artificial conduit for CSF flow

will eventually result in it falling anteriorly, into the spinal canal. At this point, the syrinx has usually collapsed and the cord may begin to pulsate. It is unnecessarily hazardous for the dissection to be carried anteriorly, although opinions vary as to the value of dividing dentate ligaments. The exposure also needs to be of sufficient length to allow free, rhythmic movement of CSF, up and down the spinal canal.

The anatomical results from this sort of surgery can be very satisfying (Fig. 11.4). The problem is that blood products and muscle proteins, which are inevitably "spilt" into the laminectomy site, may go on to organise into scar tissue once more, and this leads to recurrent obstruction of the spinal subarachnoid channels (Fig. 11.5). Early postoperative imaging is, therefore, of limited value; it is very likely to show a reduction in the volume of the syrinx, but this by no means guarantees long-term anatomical improvement. If, however, the cavity remains collapsed at 6 months and beyond, then it is likely that a good result will have been achieved in the long term, in anatomical terms at least and probably in terms of function as well.

How the dura is handled is likely to have a major influence on the success or otherwise of this operation. With craniovertebral decompression for hindbrain-related syringomyelia, we have discussions about dural opening – full, partial or not at all – reduction or excision of cerebellar tonsils, use of dural grafts and the role of cranioplasty. We have similar debates about the best methods of exposure and closure, when it

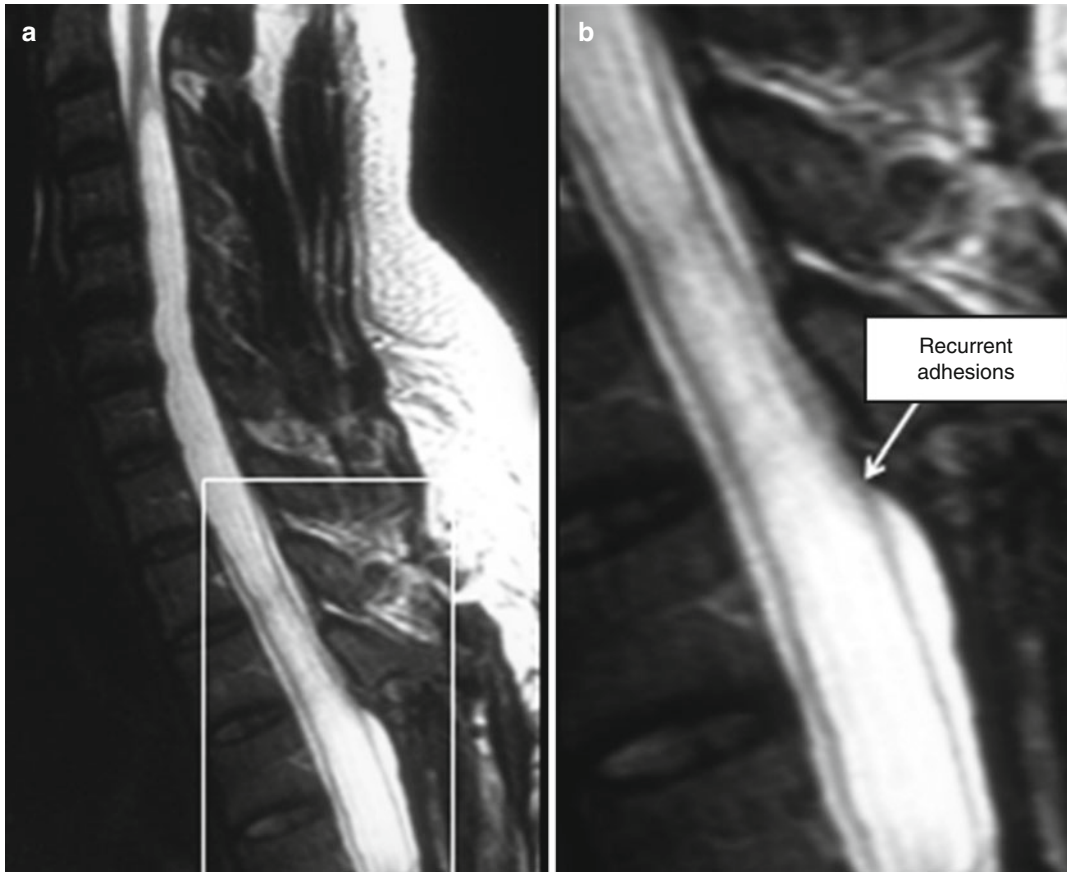

Fig. 11.5 Recurrence of syringomyelia after laminectomy. (**a**) Despite the initial operative creation of a good conduit for CSF flow, the syrinx cavity has refilled after an interval. (**b**) The *enlarged inset* from "a" reveals that recurrent adhesions at the upper aspect of the laminectomy have once again obstructed normal CSF flow

comes to operating for post-arachnoiditic syringomyelia. Dural patch grafts might be expected to limit the amount of blood and muscle products entering the spinal theca, from the surrounding tissue planes. This, we might expect, would lessen the amount of postoperative scar tissue formation. Many surgeons therefore feel more comfortable reconstituting the thecal sac, usually augmenting its volume at the same time by suturing in place a patch graft. Autografts are readily obtained from nearby muscle fascia, but synthetic materials are now preferred by most surgeons, mainly because they provoke less in the way of postoperative adhesions. The risk, however, with any form of patch graft, is that CSF may leak around its edges and collect extradurally. This may lead to a build-up of hydrostatic pressure, which pushes the graft onto the cord. This can encourage adhesions to develop between the graft and the cord. This constricting effect becomes more pronounced as healing progresses and defeats the object of opening up CSF channels in the first place. The resultant obstruction to CSF flow may be worse than preoperatively, leading to refilling of the syrinx. Many surgeons therefore make a point of hitching the dural patch graft upwards, to try to prevent such adhesions forming. The author's preference has been to leave the dura widely open, using lateral retaining sutures. With a good closure of the long paraspinal muscles, a pseudomeningocoele forms between the short muscles and this provides a conduit for CSF flow. It is important to pay close attention to the cranial and caudal aspects of the thecal opening,

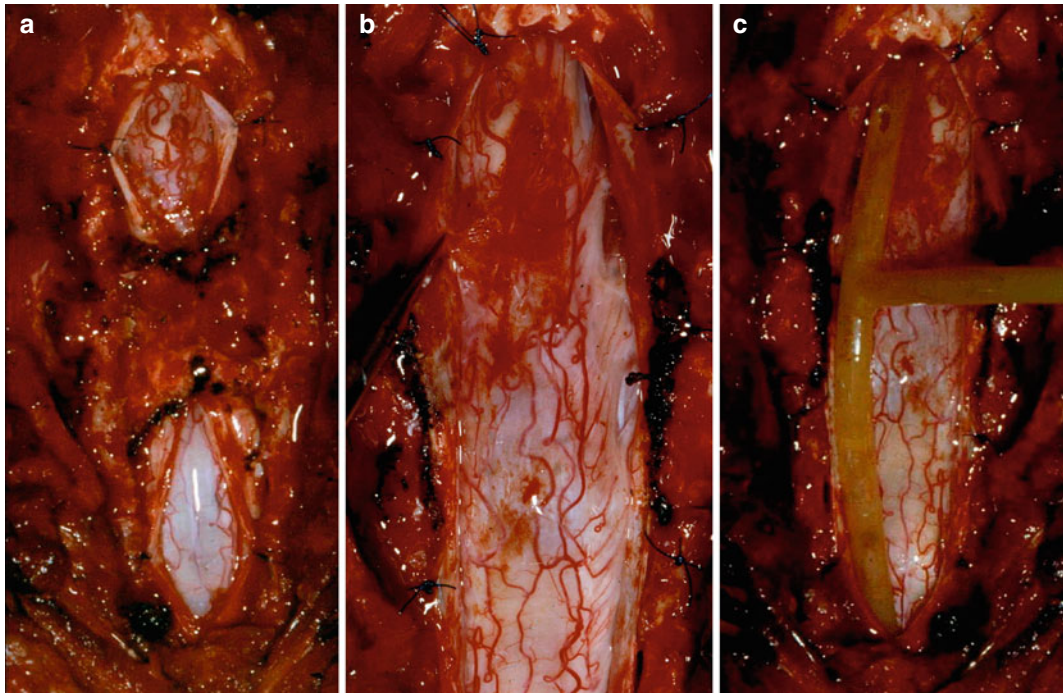

Fig. 11.6 Operative exposure of post-traumatic syringomyelia. (**a**) Initial dural opening, above and below the level of intradural fibrosis. Note the relative anaemia of the cord in the lower part of the exposure, caused by tension within the syrinx cavity. (**b**) Following completion of the dural opening and release of arachnoid adhesions. The syrinx has collapsed and the cord is now better perfused. (**c**) Optional placement of a soft tube, to allow drainage of blood-stained CSF, for 48 h after surgery

taking care to suture up the dura under the laminae at the upper and lower aspects of the exposure, to ensure that adhesions do not form at these sites. Leaving the dura open also provides an opportunity for placement of temporary subdural stents or drains (Williams and Page 1987). A soft drainage tube, left in place for no more than 48 h, not only serves as a stent but also allows CSF to "auto-irrigate" the subarachnoid channels and drain blood products away (Fig. 11.6). One study did note, however, a higher syrinx recurrence rate if the dura was left open in this way, although the difference between the two groups did not reach statistical significance (Klekamp et al. 1997).

11.8.2 Direct Syrinx Drainage

If the attempt to create a conduit fails, or is not suitable for some other reason, then the option of draining the syrinx is available. Indeed, this was a principal means of treatment for some years, and results were considered to be reasonable (Tator et al. 1982; Williams and Page 1987). About half of the patients treated by direct drainage of the syrinx will obtain useful relief (Batzdorf et al. 1998; Sgouros and Williams 1995). The method, however, has a number of drawbacks. In the first place, a myelotomy is required, with an incision through the dorsal columns. Some loss of proprioception is almost inevitable, and this is often more disabling for patients than one might expect, despite the individual retaining good motor power. Further, septa within larger syrinx cavities may obstruct passage of the shunt tubing. The concern then is that the cavity will not drain adequately although, in truth, this is not always a problem. The main difficulty with syrinx drainage tubes is that, in common with all shunt systems, there is a distinct likelihood that they will eventually block. Even if a shunt continues to function and the syrinx remains collapsed, there is a chance that a new cavity may form alongside, simply because the

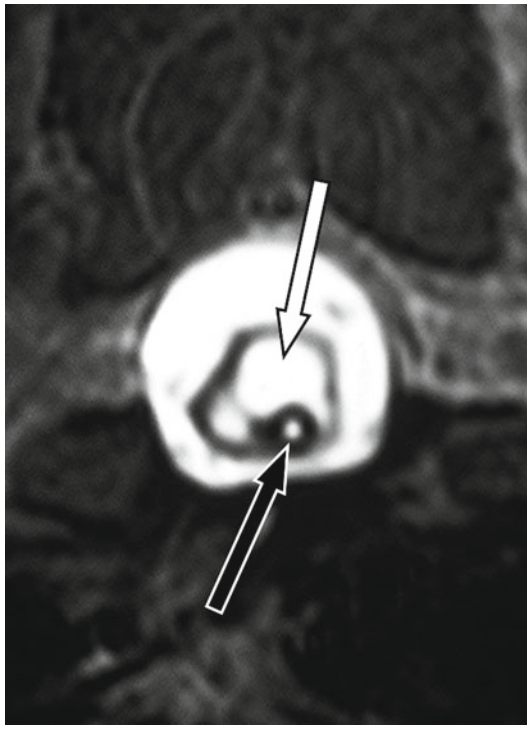

Fig. 11.7 Formation of new syrinx cavity following insertion of a syringopleural shunt. This example shows a syrinx which was initially drained effectively, after placement of a syringo-peritoneal shunt (*black arrow*). Unfortunately, the return of symptoms at a later date heralded the formation of a new cavity (*white arrow*), indicating that the underlying filling mechanism had not been disabled by the surgery

underlying filling mechanism has not been disabled (Fig. 11.7).

Direct drainage requires the surgeon to choose carefully the optimum site for insertion of the shunt tubing. Lower down the cord is preferable to higher up, provided that the conus is avoided. As to the circumferential point of entry, a myelotomy away from the median sulcus may be preferable because a midline incision risks damage to dorsal columns on both sides. If one leg is already affected more than the other, then it makes sense for the myelotomy to be placed on the same side. Appearances at surgery will, of course, have a major influence. If the syrinx presents under the pia at one location, with no intervening neural tissue, then this will be the preferred point of entry. As regards the size of catheter used, it might be supposed that bigger is better, but some surgeons prefer smaller catheters (Lohlein et al. 1990; Tator et al. 1982). Either way, it is sensible to anchor the tube to the pia, to prevent displacement.

Table 11.4 CSF receptacles for direct drainage of syrinx cavities

Pleural cavity
Peritoneal cavity
Spinal subarachnoid channels
Via shunt tubing
Cord transection
Terminal ventriculostomy

Creation of a conduit and direct drainage may be combined. The exposure required to insert a drainage tube into a syrinx may well involve microdissection and arachnolysis, sufficient in its own right to create an adequate channel for CSF flow. Equally, a surgeon may elect to drain the syrinx as a supplementary manoeuvre to creating a conduit. Whereas the shunt may not function indefinitely, it may keep the syrinx cavity collapsed sufficiently long for the deep layers of the wound to heal, with the CSF channels remaining open. Thus, the planned primary procedure may have been different in each case but the end result is the same.

Several "receptacles" are available to receive the contents of a drained syrinx cavity (Table 11.4). We cannot be dogmatic as regards whether to use the peritoneum, the pleura or the spinal subarachnoid channels. The latter are preferred by many and are often the easiest to access. A syringo-subarachnoid shunt may certainly function adequately in a case with localised fibrosis, but with more extensive fibrosis, such as seen with postinfective syringomyelia, an extra-spinal receptacle may be better. The pleural cavity is convenient when operating with the patient in the prone position, but some surgeons prefer the peritoneum, on the basis that absorption is probably more reliable in the long term. A dual system of drainage can sometimes be effected by bringing the tubing out from the syrinx and along the spinal subarachnoid channels, with side holes cut in this section, before routing the distal end to its extra-spinal receptacle (Brodbelt and Stoodley 2003a, b). A potential drawback is development of low-pressure headaches, necessitating the subsequent placement of an anti-siphon device.

It is worth remembering that syringo-subarachnoid drainage can also be achieved by cord transection and terminal ventriculostomy. The former manoeuvre sounds rather drastic, especially when explained to patients. Many spinal cord injury victims understandably hold on to hopes that techniques for inducing spinal cord regeneration may be developed in their lifetime. Whilst, however, there is little prospect of such advancements in treatment, it would be wrong to leave a completely paraplegic individual to develop progressive upper limb motor and sensory deficits, for the sake of a procedure which could both drain the syrinx and untether the cord, with beneficial results (Durward et al. 1982; Ewelt et al. 2010; Kasai et al. 2008; Laxton and Perrin 2006). Terminal ventriculostomy may be an option in those occasional cases where the syrinx passes right down to the tip of the conus (Williams and Fahy 1983).

Finally, it is worth noting that percutaneous drainage of a syrinx cavity is perfectly feasible, as a test of whether or not a patient's symptoms might respond to surgery. Relief of symptoms may last for several months and the method may therefore have therapeutic value, in those considered unsuitable for surgery (Levy et al. 1991; Sudheendra and Bartynski 2008).

11.8.3 Lowering CSF Pressure

The third surgical option is to lower the pressure throughout the CSF pathways as a whole, using a theco-peritoneal shunt (Bret et al. 1986; Vassilouthis et al. 1994; Vengsarkar et al. 1991). This strategy may be adopted when the other methods cannot be carried out or fail in the first place. If there is hydrocephalus associated with the syringomyelia, then shunting of the ventricles is the more appropriate first procedure. This may well lead to collapse of the syrinx as well, obviating the need for further procedures (McLone and Siqueira 1976).

Lowering CSF pressure is, for the most part, empirically based, but one can propose a reasonable theoretical explanation for any response seen. A length of silastic tubing, inserted above the blockage in the spinal canal and fed round to the peritoneal cavity (a theco-peritoneal shunt), may reduce hydrostatic tension within the theca and lessen the magnitude of arterial and venous pressure waves within the enclosed column of CSF. The forces driving CSF into the syrinx will, as a result, be reduced. A similar shunt system but with the shunt tube placed below the obstruction may act differently, by inducing mass flow of interstitial water through the cord, leading to reduction in hydrostatic pressure within the syrinx. Lumboperitoneal shunts, inserted below the blockage, should normally take the form of an unvalved tube, in order to maximise the flow of interstitial fluid. Shunts placed above the blockage need a flow-regulating device, either terminal slit valves or apparatus designed to compensate for changes in posture.

Success rates are always likely to be limited with this form of treatment. Operative risks, however, are generally low, particularly with a lumboperitoneal shunt. This is a relatively minor procedure, not needing extensive laminectomy and not requiring myelotomy. About a third of patients treated with lumboperitoneal shunts are likely to improve, with reduction in syrinx size in some of cases (Oluigbo et al. 2010). This simple procedure may be suitable for frail patients, those who do not wish to undergo major surgery or those in whom a conduit or shunt has failed.

11.8.4 Omental Grafts

Based on its angiogenic properties and capacity to induce outgrowth of neurites in cell culture, vascularised omental pedicles have been used in an attempt to promote functional recovery in victims of spinal cord injury. Whilst radiographic changes may follow and some subjective improvements have been reported, objective evidence of any real value for this procedure is lacking (Clifton et al. 1996). The author's experience of reexploring one such case revealed an anaemic cord and extensive fibrosis, of a degree that could only increase the likelihood of syrinx formation.

11.8.5 Skeletal Stability and Alignment

Most patients with post-traumatic syringomyelia who come to surgery will long since have fused the skeletal elements in their spinal column. Concern about post-laminectomy instability is, therefore, usually unfounded, but if the vertebral injury has not healed adequately, it will obviously need to be managed appropriately. In such cases, a surgeon may prefer to achieve stability with a preliminary operation, prior to opening up the CSF channels posteriorly. This may well involve an anterior decompression and fusion, which raises the question as to whether correction of any extradural narrowing of the spinal canal could lead to decompression of the syrinx. It has been reported that syringomyelia may be more likely to develop if the diameter of the spinal canal is severely compromised by a bony injury (Perrouin-Verbe et al. 1998), and improvement in function has been recorded following anterior extradural decompression, although such procedures may need to be supplemented by posterior intradural surgery (Holly et al. 2000). This latter finding is not surprising, given that the pathology underlying post-traumatic syringomyelia consists of leptomeningeal fibrosis.

A related consideration is the role of surgery at the time of the original injury, in particular decompression and stabilisation of the vertebral column. Although of value in allowing early mobilisation of the patient, particularly when damage is confined to the skeletal elements of the spine, there are potential drawbacks when the cord is injured. In the early stages after injury, the spinal cord is in a very unstable physiological state. It is particularly vulnerable to falls in blood pressure that may accompany general anaesthesia or episodes of hypoxia and sepsis that may follow surgery. Further, any damage to the theca, occurring during surgery, may increase the likelihood of intradural adhesion formation and obstruction of the CSF pathways. Vertebral instability can almost always be handled, instead, by external forms of immobilisation. On the other hand, it is fair to argue that prolonged recumbency may lead to stasis of blood products shed into the spinal CSF channels, thereby increasing the chances of local scar tissue formation. The role of early surgical intervention, as a means of preventing the formation of post-traumatic syringomyelia, remains unclear (Bonfield et al. 2010).

11.8.6 Radiological Localisation

With most cases of post-traumatic syringomyelia, the level to target is obvious, being the site of the original injury, which in most cases will be marked by the bony injury. It is here that the cicatrix will be encountered, whether the syrinx cavity has since propagated in a cephalad direction, a caudal direction or both. In cases without bony injury, there may be MR features to guide the surgeon, such as focal compression or distortion of the cord. Constructive interference steady-state (CISS) or fast imaging employing steady-state acquisition (FIESTA) sequences may demonstrate an arachnoid web, which may not have been apparent on routine T2 sequences (Fig. 11.8). Cine MRI may reveal restricted CSF flow. When no such clues are present, then comparison of images taken at intervals may show progressive longitudinal enlargement of the syrinx. This will usually be in one direction, away from the point of obstruction to CSF flow, which will therefore be at the "static" end of the cavity (Klekamp and Samii 2002). This rule is not infallible, especially in cases that may have propagated in both directions by the time of initial diagnosis. Other clues are that cavities tend to be largest close to the site of obstruction and that turbulence may be seen inside the syrinx, close to its point of origin. Finally, there may be clinical clues, in the form of localising features noted at the time of the patient's initial presentation.

CT myelography may still have a role to play, if other investigations prove negative. With advances in MR technology, however, the likely additional yield from myelography is now quite low. The clinician may, therefore, reasonably declare "MR-negative" cases as being idiopathic

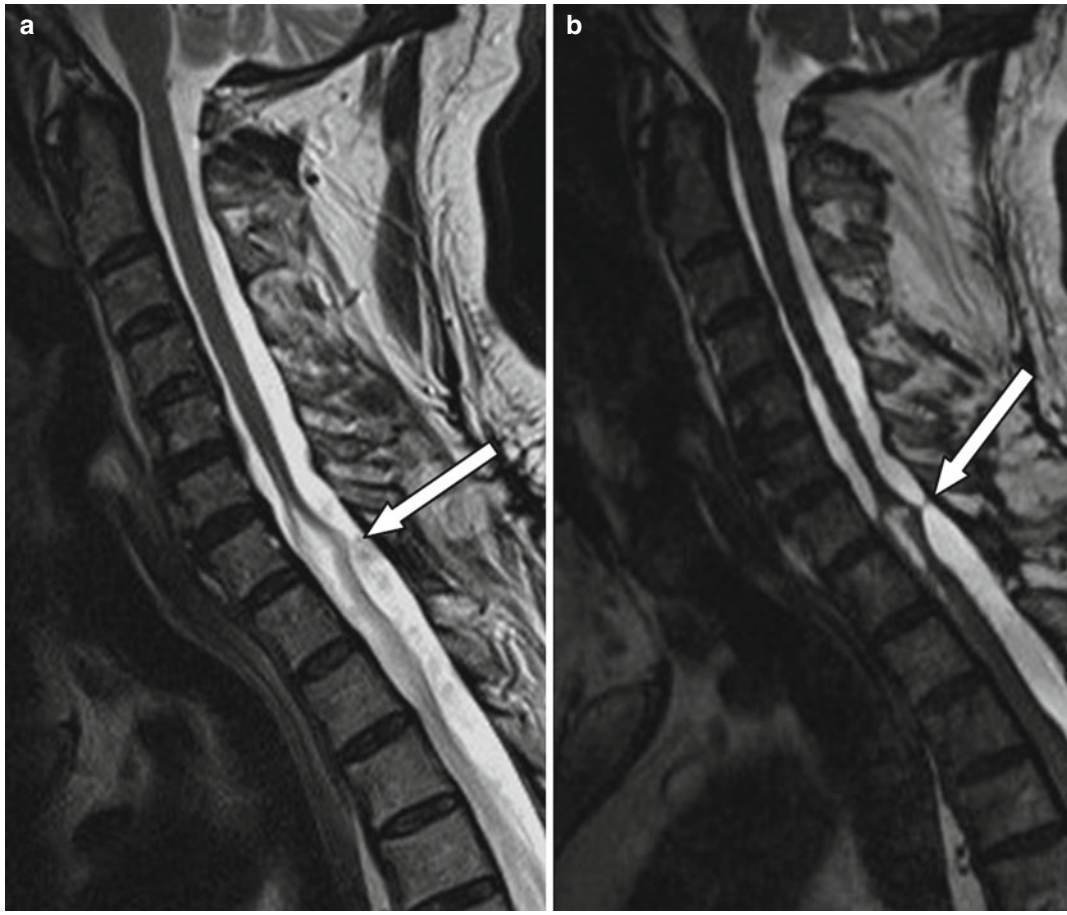

Fig. 11.8 The value of constructive interference steady-state (CISS) imaging. (**a**) Standard T2-weighted sagittal MRI shows turbulence in the CSF dorsal to the cord and oedema within the cord (*arrow*) but no clear underlying obstruction to CSF flow. (**b**) CISS sequences reveal an arachnoid web dorsal to the cord (*arrow*)

and manage them as such, by shunting the syrinx or lowering the overall CSF pressure. There may, occasionally, be a case for surgical exploration of a syringomyelia cavity, looking for a focal point of scar tissue formation, in the absence of prior radiological identification.

Finally, it is worth noting that intraoperative ultrasound may be a useful tool, to help locate the syrinx cavity, if it is not immediately obvious under the operating microscope (Aschoff et al. 1993; Lee et al. 2001; Schwartz et al. 1999).

11.8.7 Outcome and Prognosis

The natural history of syringomyelia in general is poorly defined. Deterioration is not inevitable in all cases. It would be reasonable to suggest that 50 % of patients experience functional decline and 50 % remain stable. Spontaneous collapse of a syrinx cavity is rare (Vinas et al. 2001) as is clinical progression to a state of quadriplegic helplessness, something which is feared by many patients. Post-arachnoiditic syringomyelia is probably no different in these respects, although sudden deterioration may occasionally follow episodes such as a bout of coughing or a period of straining (Balmaseda et al. 1988). Ageing will, of course, exacerbate the effects of any existing neurological disability, including any caused by syringomyelia.

Results following surgery for post-arachnoiditic syringomyelia are inconsistent and not all of the aims will be achieved (Table 11.5). Many patients may need to undergo more than one procedure (Klekamp et al. 1997). Difficulties

Table 11.5 Aims of treatment

Prevention of progressive motor deterioration
Relief of pain
Control of sensory disturbances
Reduction of sweating and spasms
Restoration of lost function

relate in part to the narrow calibre of the canal in the thoracic region, as opposed to the wider "funnel-shaped" morphology of the craniovertebral junction. More important is the extent of fibrosis that has developed, and outcome following surgery is inversely proportional to this, which in turn is largely dependent upon the underlying pathology. Focal arachnoid webs may be relatively straightforward to treat, but extensive post-tuberculous adhesions present a considerable challenge.

Whilst collapse of the syrinx is always an encouraging result following surgery, it will not always predict a good functional result. Nor does failure of the syrinx to collapse mean that no gain will follow. In either case, improvement in function is seldom substantial. Even so, modest gains, or simply just the arrest of deterioration, may be of great benefit to a patient, who may well already bear a substantial physical disability. In this respect, a patient's perception may better reflect functional outcome than might attempts at objective assessment (Falci et al. 2009; Kramer and Levine 1997; Ushewokunze et al. 2010). Figures vary from one series to another, but it would be reasonable to quote figures of roughly one third of patients improving, one third stabilising and one third continuing to deteriorate (Sgouros and Williams 1996; Ushewokunze et al. 2010).

Regrettably, most publications on the outcome of surgery for syringomyelia do not adhere to any standards of data presentation. Klekamp and Samii have stressed the value of Kaplan-Meier analyses, as the only meaningful way of presenting outcome data (Klekamp and Samii 2002).

11.8.8 Pain Control

Of all the symptoms of syringomyelia, pain is perhaps the most difficult to control (Milhorat et al. 1996). Published results suggest very variable responses to surgery, but this may reflect a failure, on the part of authors, to differentiate between pains of different types or to use standard nomenclature. Pain induced by Valsalva manoeuvres is likely to be caused by transient rises in intra-syrinx pressure and can be expected to improve if the cavity collapses after surgery. Likewise, pain caused by movement may be related to cord tethering and may well improve if the cord is adequately released at surgery. Pain which is not modified by such influences and which has the characteristics of central, neuropathic pain will very likely persist, despite an anatomically successful operation (Klekamp and Samii 2002).

11.9 Summary

Syringomyelia remains an uncommon disorder and cases caused by leptomeningeal fibrosis are relatively rare. Post-traumatic syringomyelia, however, is very common amongst the population of spinal cord injury victims, with an incidence of about one in five and producing symptoms in 1 in 20 patients. The condition can cause significant additional disability, and surveillance should be the norm for all spinal cord injury patients. Surgery is not mandatory in all cases as many syrinx cavities remain stable for many years. Further, the outcome following surgery for post-traumatic as well as other forms of post-arachnoiditic syringomyelia is variable and cannot be predicted in an individual case. The major determinant of outcome is the extent of scar tissue that has formed, which limits surgical options in some circumstances, such as post-tuberculous syringomyelia.

Broadly speaking, there are four management options: (1) release of the scar tissue and creation of an artificial conduit for normal CSF movement; (2) direct drainage of the syrinx cavity into the pleura, peritoneum or elsewhere in the spinal canal; (3) lowering of the overall CSF pressure with a thecal or ventricular shunt; and (4) conservative management. Of the surgical options, the first is the best in terms of maintaining a good long-term result. Unfortunately, it is not always possible to achieve, in which case one of the other methods may be appropriate. Management must be tailored to the individual patient and his

or her circumstances. It should be made clear that any functional improvement following surgery is likely to be limited. At the same time, even modest gains, or simply the arrest of deterioration, may be very welcome from the patient's perspective.

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