

# Imaging in chronic spinal cord injury—indications and benefits

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## Abstract

As most patients who have suffered spinal cord injury can now expect a normal life span, the late complications of these injuries are seen increasingly frequently. Regular surveillance of both the renal tract and the central nervous system (CNS) is important as the treatment of impending, potentially fatal complications can be implemented before damage has progressed too far. Renal tract complications are particularly dangerous as they are often clinically silent but regular surveillance to detect early deterioration in renal function, particularly from reversible causes such as reflux or obstruction can pre-empt problems. Follow-up protocols depend on the bladder management regime but most centres advocate regular ultrasound with less frequent isotope function studies. With the increasing ability to diagnose and treat the neurological complications, surveillance of the state of the spinal cord with MRI is also important and many centres now advocate checks every few years with sagittal midline T2W sections are sufficient unless changes are noted, when axial T1W sections can be added without significant examination time penalty. Imaging is critical in acute problems. In addition to suffering from the usual normal conditions, patients with spinal cord injury suffer others peculiar to, or particularly related to, the injury, which may be missed as their symptomatology is greatly altered by their paraplegic or quadriplegic status and they may often present as generally unwell but with no obvious cause. This review discusses the role of radiology in routine surveillance of the CNS and the renal tract as well as in assessing specific conditions such as deteriorating neurology or renal function, pain, spinal instability, pressure sores, ectopic ossification, muscular spasm, spinal instability, airway problems and elective operations on the renal tract. © 2002 Elsevier Science Ireland Ltd. All rights reserved.

*Keywords:* Chronic spinal cord injury; Surveillance; Spinal cord; Renal tract; Syringomyelia; MRI; Radiological investigations

## 1. Introduction

With improvements in the management of patients with spinal cord injuries (SCI) over the past 60 years, the patterns of morbidity, mortality and psycho-social outcomes have changed quite markedly [1,2]. Within the last decade a biphasic pattern of occurrence has become apparent with peaks of injuries noted in the third and sixth to seventh decades. With patients in-

jured in their youth now surviving for many decades, there is an ageing population of patients with SCI and the chronic changes in the body, particularly in the central nervous and renal systems, are becoming of greater importance. In addition there is a group of geriatric patients injured as a result of trauma exacerbating degenerative changes in the spine.

Significant recovery of neurological function rarely commences more than 1 year after the accident, particularly in complete lesions. There may, however, be a deterioration with time, the main herald symptoms being loss of function, the loss of a reflex or increasing pain or spasms. Any deterioration, however slight, in the limited function the patient possesses may make the difference between relative independence and the need for chronic care, so prompt and accurate diagnosis as well as regular surveillance are of paramount importance. Tables 1 and 4 summarise the role of radiology in the long-term follow-up of these patients. Tables 2 and 5 summarise the role of radiology in investigating other problems peculiar to patients with chronic SCI.

*Abbreviations:* AXR, abdominal radiograph; CTM, computerised tomography myelogram; DREZ, dorsal root entry zone lesion; DVT, deep venous thrombosis; EO, ectopic ossification; ERPF, effective renal plasma flow study (TC-99m MAG3); HO, heterotopic ossification; IVP/U, intravenous pyelogram/urogram; MRI, magnetic resonance imaging; PAO, para-articular ossification (osteopathy); PCNL, percutaneous nephrolithotomy; PPCM, progressive post-traumatic cystic myelopathy; PPMM, progressive post-traumatic myelomalacic myelopathy; SCI, spinal cord injury; US, ultrasound.

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Table 1  
Radiology is critical in routine surveillance and specific conditions

<i>Routine surveillance</i>	
Central nervous system	
Renal tract	
<i>Specific conditions</i>	
Deteriorating neurology	
Spinal instability	
The acutely unwell patient	
Deteriorating renal function	
Pressure sores	
Ectopic ossification	
Pain	
Muscular spasm	
Airway problems	
Elective management of the renal tract	

1.1. Cord changes post SCI

The three most important changes that have been noted in the cord are atrophy, myelomalacia (progressive post traumatic myelomalacic or non-cystic myelopathy) and cystic changes (focal cyst and syringomyelia). Understanding of all these conditions has been impeded by a general lack of agreement on their definitions, both histologically and radiologically in the various studies of their occurrence. Most studies have been performed on relatively small numbers of mostly symptomatic patients so a true incidence is essentially unknown. Post-traumatic changes in the cord are difficult to study as there is no true animal model and there are few extensive series. The most comprehensive descriptions are by Quencer [3,4] and Squier [5].

Following a cord injury, Wallerian degeneration has been noted cephalad to the lesion and, with demyelination and subsequent gliosis, is the main cause of proximal atrophy [6,7] which represents an irreversible state of degeneration manifest by a loss of spinal cord substance [8], macroscopically appearing as abnormal narrowing of the cord diameter. Other factors causing atrophy are still poorly understood and possibly relate to ischaemia [8–10], a postulated mechanism that surgeons who decompress [11,12] or graft omentum [13] or physicians who administer high dose corticosteroids within 6 h of trauma [14] are aiming to ameliorate.

In 1915, Holmes [15] described six SCI patients

whose neurological condition had deteriorated and who at surgery had intramedullary cysts extending several segments above or below the injury site. By 1977 about 32 cases had been described and two were added by Jensen [6] who described an acute (2 months post injury) and a chronic (4 years post injury) case. In both there was an extensive cavity which in the acute case contained phagocytes and myelin tissue breakdown products. Extensive narrowing of the cord was also frequently found.

At the time of injury there is an injection of crushed cord tissue from the site of maximum impact along shear planes in the cord. This tissue then undergoes necrosis and cyst formation, a process also probably involving liquefaction of parenchymal haematoma, ischaemia, the release of intracellular enzymes and mechanical damage from cord compression [4,17]. The cysts are eccentrically placed and are rarely contiguous with the central canal. They have been termed ‘contusion evolving to cavity’.

The progression to syringomyelia, i.e. a cyst extending beyond the limits of bony damage, appears to involve arachnoid adhesions, presumably secondary to blood products and inflammatory changes at the time of injury. The inability of the cord to move freely and the consequent turbulent CSF flow dynamics is thought to lead to the formation of the syrinx [5,18,19]. Animal models using kaolin irritants [20] and CSF flow assessments in patients with Chiari and post-traumatic cysts [21–34] support these views and restoration of CSF dynamics is the basis of the most successful surgical approach for treatment [35–37], shunting alone being less successful [38].

The most plausible current concept is that of a ‘pre-syringomyelic’ myelopathic state with reversible cord oedema secondary to abnormal CSF flow through the perivascular and interstitial spaces of the cord tissue [39–42]. The Miami group have described the condition of progressive post-traumatic myelomalacic myelopathy as distinct from that of progressive post-traumatic cystic myelopathy which is characterised by microcystic change but no true cysts. In both situations the patients have identical presenting symptoms and signs and at operation have adhesive arachnoid tethering of the cord and are relieved by untethering and an allograft expansile duroplasty [36,40].

Table 2  
Investigations for surgical planning

Spinal cord assessment	MRI	CTM	Intraoperative US
Skeletal stability	CT	MRI	Plain films (flexion and extension 30 min positioning)
Sacral anterior root stimulator (SARS)	US, AXR, Plain films	MRI	Cystourethrography
Intrathecal pumps	Plain films	Scintigraphy	

## 2. Reported series

### 2.1. Atrophy

Group mean values for the sagittal diameters of the cord at different levels have been determined from pathological studies [43] and CT myelography [8,44,45] although there appears to be a significant difference between the two methods. Seibert [46] and Isherwood [47] have drawn attention to the variation in measurement with CT depending on the window level settings which may explain some of the variance. Similar differences exist in measuring atrophy on T1 and T2 sequences (personal observations). From the pathological studies, a normal cervical cord does not measure less than 7.5 mm and the thoracic cord 6.5 mm [48].

Unfortunately, some authors have assessed atrophy qualitatively and it is difficult to compare rates when the criteria are not given. Using MRI, Yamashita [49], who took < 6 mm in the cervical region and < 5 mm in the thoracic cord as atrophic, found five patients in a total of 76 (7%) and Curati [50] found 16 in 87 (18%) using the same criteria. Nidecker [48] found six cases in 22 (27%) patients over 18 weeks post injury using an undefined criterion of ‘thinning’. However, as the papers do not describe the sequences or cuts used for these measurements and there is such a wide interpretation of the concept ‘chronic’, the groups are heterogeneous and comparisons are virtually meaningless.

### 2.2. Cystic change

Until recently there has been no universally agreed definition of these conditions and in the literature there is often confusion between cysts, myelomalacia and syringomyelia so that comparisons of the different series is impossible. Some authors record all abnormal cystic-looking structures as post traumatic spinal cord cysts (PTSCC) [51] but others make a variable distinction between focal cysts, syringes and myelomalacia [48–50,52,53]. Claimed incidences for ‘syringomyelia’ vary between 1, 1.4 and 3.2% [54–56] using clinical criteria in the pre-MRI era and up to 59% using MRI [57]. The term ‘myelomalacia’ has been used loosely to describe acute ischaemic changes due to vascular stasis and perivascular oedema [48] or areas of degenerative change in the cord in which there are multiple small cysts and gliosis.

In one of the largest published series, Curati et al. [50] examined 87 patients who were a heterogeneous group and approximately one third had already undergone spinal surgery. They defined myelomalacia as areas of patchy low intensity on T1W images with corresponding high intensity on T2W images. A syrinx was identified as a low intensity linear or septate structure within cord tissue on T1W sections together with

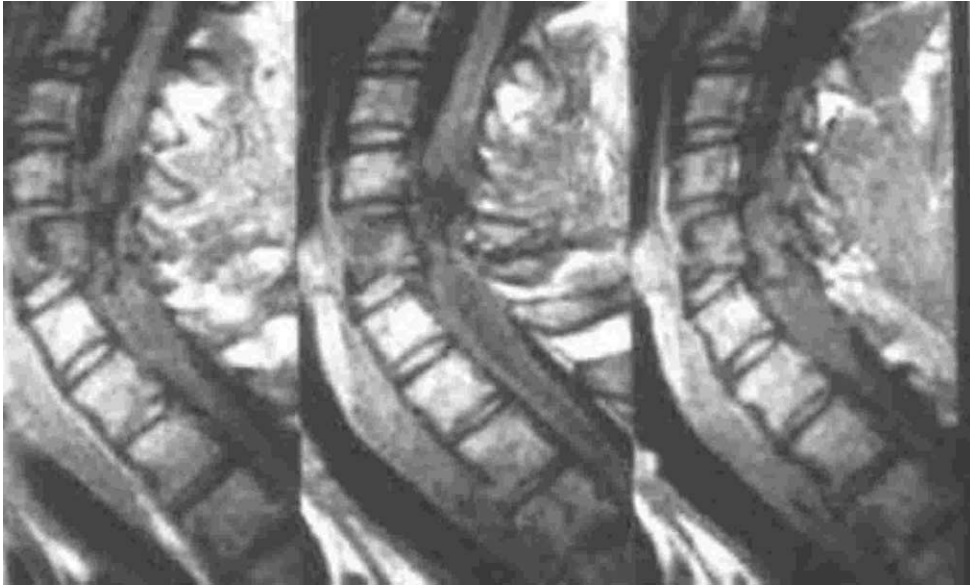
areas of low signal in at least part of the cyst on the T2W sections, a change thought to be due to fluid motion; the overall characteristics were similar to those of the surrounding CSF. No distinction between syringomyelia and a focal cyst was made. With these criteria, they found an incidence of myelomalacia in 37%, syringomyelia in 40%, persistent cord compression in 32% and atrophy in 18%.

A syrinx was twice as common in the group who had had their injury over 10 years prior to investigation compared with a group of patients less than 10 years post-injury. Cord compression was found in approximately equal frequency in patients with total paraplegia, incomplete paralysis and with minor signs only (30–40%).

Several authors [50,53] have noted cyst formation at less than 3 months post injury. However, there is still imprecision as to whether this is a focal cyst, myelomalacia or syringomyelia. Most other authors had only small numbers of true chronic cases.



Fig. 1. Sagittal T2W section showing atrophy.



(a)



(b)



(c)

Fig. 2. Progressive post-traumatic myelomalacic myelopathy. (a) Sagittal T1W sections showing changes at 3 months post injury. (b) Sagittal T2W section. (c) Sagittal T2W section showing posterior arachnoid tethering.

To date the most likely indicator of incidence is the study reported by Wang [58] where MR imaging at Stoke Mandeville Hospital of a cohort of 153 consecutive patients, regardless of their symptomatology, who had had their SCI at least 20 years previously, using the following definitions:

*Atrophy*: abnormal narrowing of the spinal cord in the sagittal plane two segments or more beyond the limits of the vertebral injury (<7 mm in the cervical and <6 mm in the thoracic cord—measured on a local normal population) (Fig. 1).

*Myelomalacia*: an area of low T1W and high T2W signal intensity that is between that of normal spinal cord and the surrounding CSF with ill defined contours and irregular shapes, occasionally appearing as a string of small cysts (Fig. 2).

*Syrinx*: an area with the same signal intensity as CSF which is usually tapered at one or both ends and which may appear loculated, with a well defined contour and which extends beyond the length of maximal bony damage (Fig. 3).

*Focal cyst*: an area having the same signal intensity and contour definition as a syrinx, but with a round or oval shape and confined to the site of maximal bony protrusion into the spinal canal (Fig. 4).

*Disruption*: the complete absence of the spinal cord signal through the traumatised area reported an incidence of atrophy in 62%, myelomalacia in 54%, syringomyelia in 22%, focal cysts in 9% and disruption in 7%.

In a stable cyst there is fluid equilibrium and in our experience, appearances may not change for many years though if there is no deteriorating neurology, few surgeons would recommend operation. Life-style modification may be suggested—e.g. the avoidance of contact sports, etc. In a hydrodynamically unstable cyst, however, there may be abnormalities in the cord tissue surrounding the cyst. Jinkins et al. have reported the correlation of increased T2W signal caudad to the top of a syrinx (Fig. 3a) with clinically progressive disease and its disappearance with resolution of the clinical problems post-operatively. It presumably represents oedema from raised intra-cystic pressure and is analogous to the flare in the periventricular oedema of hydrocephalus [59,60].

### 3. Treatment

The treatment of syringomyelia is still controversial and most authors would argue that only the symptomatic syrinx should be treated as the results are disappointing in terms of recovery of lost function so any unnecessary surgery should be avoided [19,61]. Traditionally, syringo-subarachnoid, syringo-peritoneal and syringo-pleural shunts as well as laser cut myeloto-

mies have been described [19] and if possible, drainage should occur from that part of the syrinx below the fracture level so that damage to functioning tissue during placement is minimised. Imaging to identify the levels is therefore important. Occasionally pre-operative percutaneous syringography may be required to assess the patency of a connection between a syrinx extending from below to above the fracture site. This enables drainage of the lower segment only to be performed and minimises cord trauma during catheter placement. Intra-operative ultrasound may be needed to locate the exact position of the syrinx as they are not infrequently eccentrically placed. In refractory cases, cordotomy is offered but there is general reluctance to accept this final step in the light of impending new cord treatments aimed at reconnecting the pathways and promoting regeneration of the cord [62].

Currently, however, the emphasis on treatment is changing with an understanding of the role of CSF flow dynamics and arachnoid adhesions. The most successful approach is to free the adhesions and create a wide duroplasty in an attempt to restore some degree of normality to the CSF flow dynamics [36,40]. The technique of omental transposition and grafting has been effectively discredited in controlled trials.

Post-operative assessments to confirm syrinx collapse or a return to normal of the cord can be rapidly performed in a brief examination as two or three T2W or T1W sagittal mid-line sections only are required. Occasionally shunt blockage is suspected and in this situation, Gadolinium contrast enhancement may show an area of increased signal around the catheter tip suggesting chronic inflammatory reaction and perhaps infection (Fig. 5).

#### 3.1. CT myelography

If MRI is contraindicated (Table 3) computerised tomographic myelography (CTM) can give an accurate assessment of the spinal cord [46]. The contrast is preferably run up from below but if there is a spinal block, cisternal puncture (most comfortably approached via the posterior midline with the patient in left lateral decubitus) may be needed. Some 5 mm scans at 15–20 mm intervals the length of the cord are then performed.

This technique is most useful in the assessment of atrophy (Fig. 6c) [8], but can be used to assess the presence of a myelopathy as the contrast agent diffuses into areas of myelomalacia or cystic change over several hours. Unfortunately the rate at which this occurs is variable so that scanning of the whole cord at 6, 18 and 24 h post myelography may be needed to show the characteristic contrast reversal (Fig. 6a,b). It is not appropriate to scan only the dilated areas of the cord as a significant syrinx may be present in a cord of normal



Fig. 3. Syringomyelia. (a) Sagittal T2W section showing flare at the rostral end indicating oedema. (3) Axial T1W section. (c) Sagittal T1W sections showing 'septa' that are probably decussating tracts. (d) Intraoperative US axial image showing the cyst.



Fig. 4. Sagittal T2W section showing a focal cyst at the injury site.

or even reduced calibre. There are also false positive and false negative results and it can sometimes be particularly difficult to differentiate myelomalacia from true cystic change. Its use should be limited to when MRI is contraindicated.

#### 4. Frequency of investigation

As the rate of progression of complications is variable, the frequency of investigation is controversial but sequential studies have shown that changes in the cord can progress with surprising speed and a developing syrinx may become evident within 2–3 months (Fig. 2a), ([50,63] and personal experience). At this stage it has the features of PPMM. It would seem prudent to scan at least before discharge from hospital so that a baseline is established and then at regular intervals, perhaps every 2–5 years, during the follow-up or at any other time if the neurological condition deteriorates.

The purpose of imaging in the chronic state is to assess the presence of remediable pathology. PPMM and syringomyelia are the only direct complications that can be

treated but other pathologies such as disc disease, tumours, etc. may be present and they would require as full a work-up as in any other patient.

#### 4.1. Contraindications to MRI scanning

In addition to the usual contraindications to the use of MRI such as cardiac pacemakers (and probably phrenic pacemakers, although there have been occasional inadvertent cases of scans performed on patients with phrenic pacers with no demonstrable deleterious effects), aneurysm clips and some types of metallic prosthetic valves, great care should be exercised in scanning patients with SCI. With gunshot or shrapnel wounds it may be wise to remove the fragments surgically prior to scanning unless they can be demonstrated to be firmly fixed in the tissues. This is probably safe in the chronic state but caution is always appropriate. The problems are theoretically decreased in magnets with low field and low power gradient strengths and if the patient is introduced slowly into the magnet, any abnormal reaction should be reversible. It must be remembered however that sensation is very abnormal in these patients.

Sacral anterior root stimulators are used to aid control of micturition and bowel function. A receiver is implanted subcutaneously and wires are tunneled through a laminectomy in the upper sacrum and clipped to the anterior roots of the sacral nerves S1–S4, the exact roots depending on the responses elicited on testing during the operation. This acts rather like a pacemaker and there are theoretical reasons for avoiding MRI, such as inadvertent activation of the functions and heat damage to the roots themselves [64].

At least one manufacturer (Siemens, personal communication), has now refused to support the scanning of patients with sacral anterior root stimulators on the grounds of potential damage to both patient and scanner, despite the inventor's own experience in scanners with his implanted devices. We have scanned many patients on low (0.2 T) and high (1.5 T) systems with no untoward effects apart from occasional muscular contractions that were related to the orientation of the receiver to the magnetic field and which in all but one patient were stopped by changing the position of the patient on the table, generally by rolling them onto their side. It is, however, prudent not to examine these patients in high field systems with rapid sequences using high gradients.

With the increased tendency to early stabilisation surgery, many patients will have metal fixation plates, rods, rectangles screws or wires near the spinal column. The use of titanium decreases the metal artefact and in many cases excellent visualisation of the cord and soft tissues is obtained (Fig. 7). Older and cheaper materials are associated with quite marked artefact, especially on



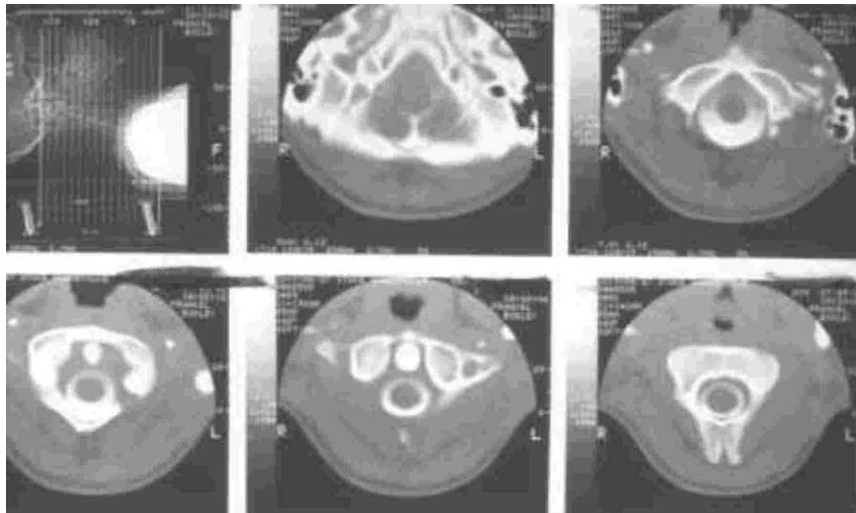
Fig. 5. Post shunt complications. (a) Sagittal T1W section pre gadolinium. (b) Section post gadolinium showing inflammatory reaction at the end of the tube which settled with antibiotics.

T2W images, which may obscure some details but in most cases that we have scanned we have gained useful information and been able to exclude or confirm major complications.

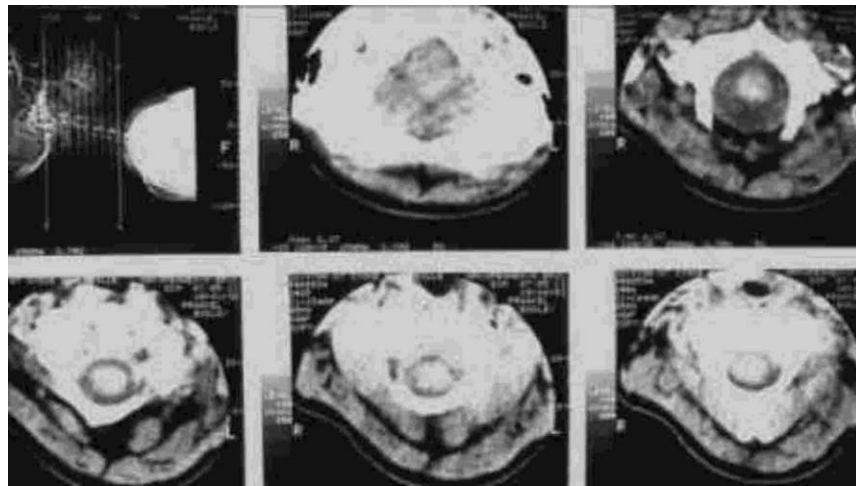
Persistent spasms are increasingly being treated by the use of implanted intrathecal drug administration systems (Baclofen being the currently used drug), that have a subcutaneous combined pump and reservoir that are either gas or electronically driven. Theoretically, the reservoir should be emptied of active drug before scanning to prevent possible overdose but in practice this has not proved a problem. Some artefact may arise from the reservoir but as this is situated on the anterior abdominal wall it rarely interferes with images of the spine. Programming should be checked after the scan.

Table 3  
Contra indications for MRI

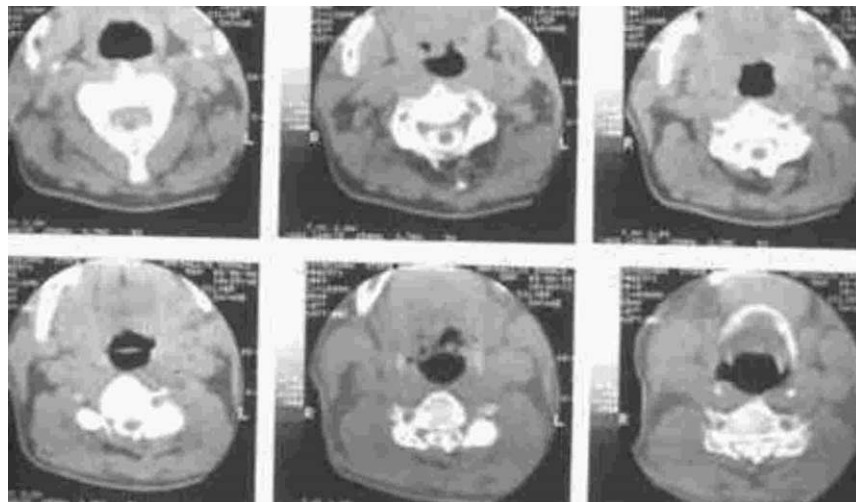
Absolute	General contra-indications
SARS	Theoretically absolute (Unsupported by manufacturers if patient or the equipment is damaged. No damage yet reported in practice, especially at low field strengths)
Intrathecal pumps	Mechanical—no problem
Spinal fixation devices	Electronic—discuss, as control settings may be altered No safety problem after 2 weeks post operatively Variable artefact



(a)

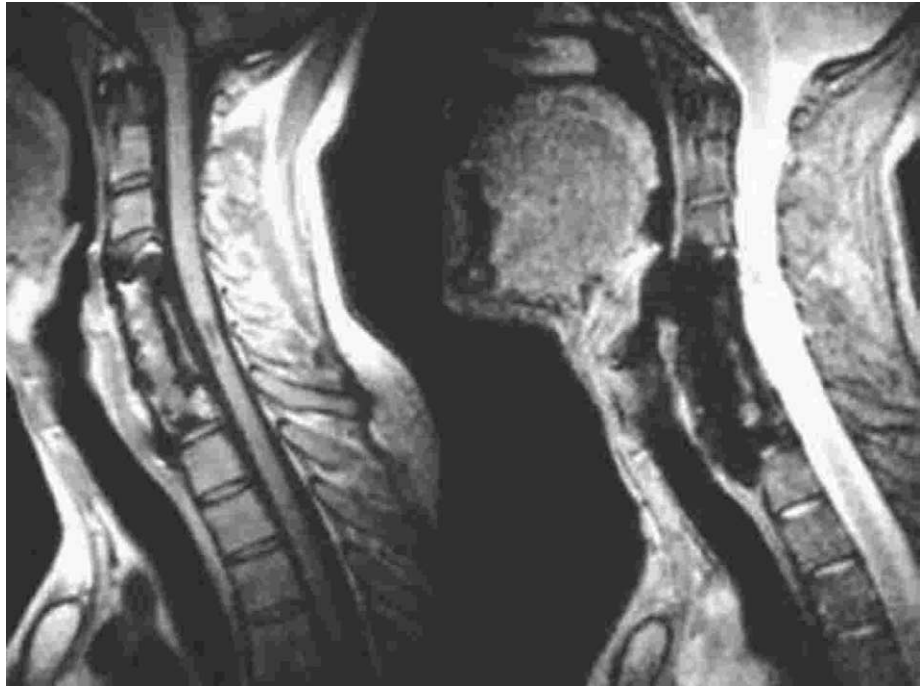


(b)



(c)

Fig. 6. CT myelography. (a) Axial sections immediately post injection—note dilated cord with surrounding contrast. (b) Sections, 18 h post injection with contrast 'reversal'. (c) Atrophy.



(a)



(b)



(c)

Fig. 7. MRI artefact post stabilisation. (a) Titanium anterior fixation (minimal artefact). (b) Stainless steel wire posterior fixation. (c) Harrington rods—note the cord can still be usefully seen.

## 5. Urological investigations

### 5.1. Surveillance

While most discussion of the imaging of patients with SCI concentrates on the spine and the spinal cord, probably because the ‘holy grail’ of treatment is to restore function through the damaged segments and a full understanding of the processes occurring at this site is critical, from the aspect of the continuing care of the patient it is the urological system that is by far the most important (Table 4).

It is largely because of the detailed attention to the urinary tract that the life expectancy of the patients with SCI has been increased and their quality of life enhanced [16]. In the past 20–30 years this change has been so successful that deaths related to urological complications have fallen to fourth place behind cardiovascular and respiratory causes and suicide [1,65].

Bladder contraction is under parasympathetic (S2–4) and the bladder neck and sphincter under sympathetic (T10–L2) and voluntary (S2–4) control so function after damage can be complex. Spinal cord lesions above T10 will ‘disconnect’ the reflex areas from higher control giving an upper motor neurone reflexic bladder unless there is secondary ischaemic damage to the lower cord and reflex arcs. Trauma below this will damage the conus or lower motor neurones, hence the reflexes are lost giving an atonic bladder. In most cases, the sphincter control is also damaged and detrusor–sphincter dyssyn-

ergia occurs when the bladder contracts against a closed sphincter causing a rise in pressure, poor upper tract emptying, the possibility of reflux and a trabeculated, neuropathic bladder.

Infection and reflux with consequent renal damage and stone formation are common sequelae. Gupta [66] recorded morphological abnormalities in 63% overall with 3% renal and 11% bladder stones. It is now accepted that there is a need for long term surveillance of the renal tract in all patients whether or not they are symptomatic [67,68] although it may not be so critical after 10 years of normal radiological follow-up [66]. The methods used vary between the different centres and depend on the bladder management technique, which is a controversial issue and depends on the level of injury [67,69–71]; some aim for clean intermittent self catheterisation [72] and others for condom catheter drainage, a technique generally requiring a sphincterotomy to allow maintenance of a low pressure state within the bladder. With tapping and supra-pubic pressure techniques, reflux and consequent renal tract damage is a potential sequel that may be very insidious in onset. The aim is for a ‘balanced’, low pressure, regularly drained and infection free bladder [73].

Recently balloon dilatation and metal stenting of the external sphincter and prostate has been used with initial success although a long term follow-up assessment is required [74–77]. Women may prefer supra-pubic to urethral catheterisation although they do not have the same degree of problems as men with urethral catheters.

Table 4  
Routine asymptomatic surveillance

System	Technique	Frequency	Benefit and comment
CNS	MRI of the Cord	Prior to discharge then every ? 5 years	Watch for early development of progressive post traumatic myelomalacia myelopathy (PPMM) and syringomyelia. Lifestyle modifications (e.g. decreased contact sport, etc.) may be advised
Renal tract	Sag T2 Axial T1		? only treat symptomatic patients
	US	At the regular clinical review	The frequency and use will depend on the bladder management technique
	AXR (IVP not indicated)		Renal tract complications are the silent killer Monitor development of stones and hydronephrosis—useful as a baseline when symptoms suddenly change
	ERPF/MAG 3	Baseline, at 1 year then 5 yearly  (depending on departmental capacity and bladder management technique.)	Low threshold for nephrostomy if ? obstructed Regular monitoring of renal function to preempt pressure related effects from detrusor/sphincter dyssynergia Deterioration should be investigated further
	Urodynamics	Variable practice depending on the resources available	Not needed if the patient is catheterised or uses self-intermittent catheterisation Pressure-flow measurement generally sufficient Cystourethrography is rarely performed as surveillance but when there is deteriorating function, to check for reflux

As well as regular microbiological screening, annual or biennial assessment of the upper tracts with either IVU, US and plain films, or isotope studies have their advocates [78–80], although the IVU is now rarely used routinely in most centres, especially when there is renal function impairment. If the upper tracts are dilated, a post micturition view must be obtained, if necessary after a trial with an indwelling catheter, to assess whether or not this is reversible.

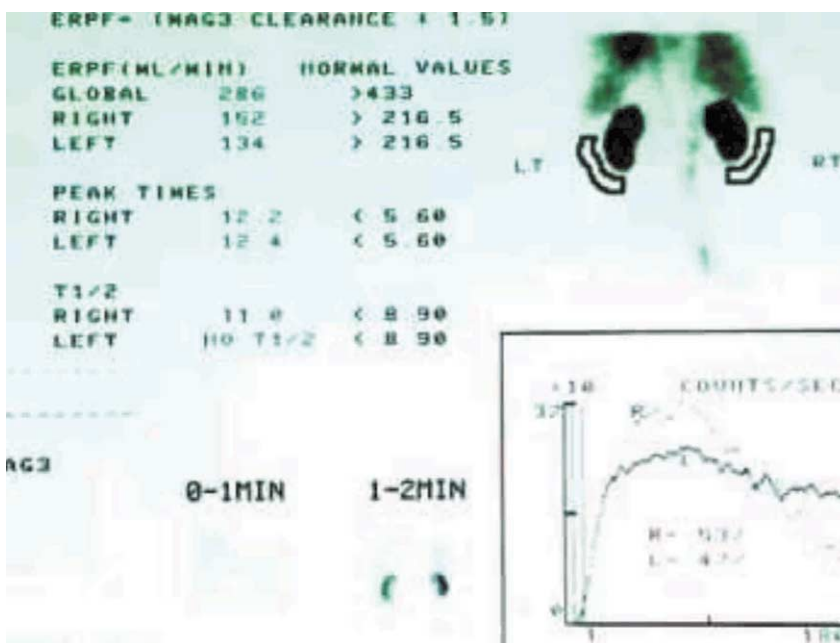
A reasonable practice would be US at 1–2 year intervals with an ERPF study as a baseline before discharge, at 1 year and every 5 years if stable. Proto-

cols vary with departmental capacity but the ERPF study is a critical method for assessing the function that US alone cannot provide (Fig. 8). Urodynamics is rarely advocated as a routine screening technique but is critical in assessing complications and planning the bladder management strategy [81].

With recumbency and inactivity, calcium metabolism changes dramatically, urine flow is often decreased and this, with infection from urea splitting organisms, leads to a high incidence of stone formation—3–6% in the upper tracts, 11% in the bladder [66,82]—which may present as early as 6 weeks post injury. These patients



(a)



(b)

Fig. 8. The contribution of renography. (a) US showing moderate hydronephrosis. (b) ERPF showing good equal function and delayed emptying. (c) Poor left renal function (but a misleading normal US).

require access to the normal range of treatments such as extra-corporeal shock wave lithotripsy, percutaneous nephrolithotomy or open surgical techniques for stone removal.

**6. Imaging in specific conditions**

*6.1. Deteriorating neurology*

MRI or CTM is needed to differentiate between evolving PPMM, syrinx and atrophy and other non SCI related causes (Table 5).

*6.2. Spinal instability*

Pain or instability, particularly if associated with changing neurology, is an indication for stabilisation. Both MRI and CT studies may be indicated to allow for planning of the surgical approach and to assess bone stock. It is crucial to liaise with the surgeon performing the operation as the studies often have to be tailored to the particular case. In some cases, instability is not evident on rapidly performed flexion and extension films and our local practice of careful positioning over pillows for at least 25–20 min before taking the film has revealed instability that was a cause of pain.

Local misalignment will lead to abnormal stresses in the compensating areas and scoliosis assessment is critical. Long cassettes are needed to compare the spine in the normal sitting or standing position with that in the recumbent position under traction to assess the correction possible.

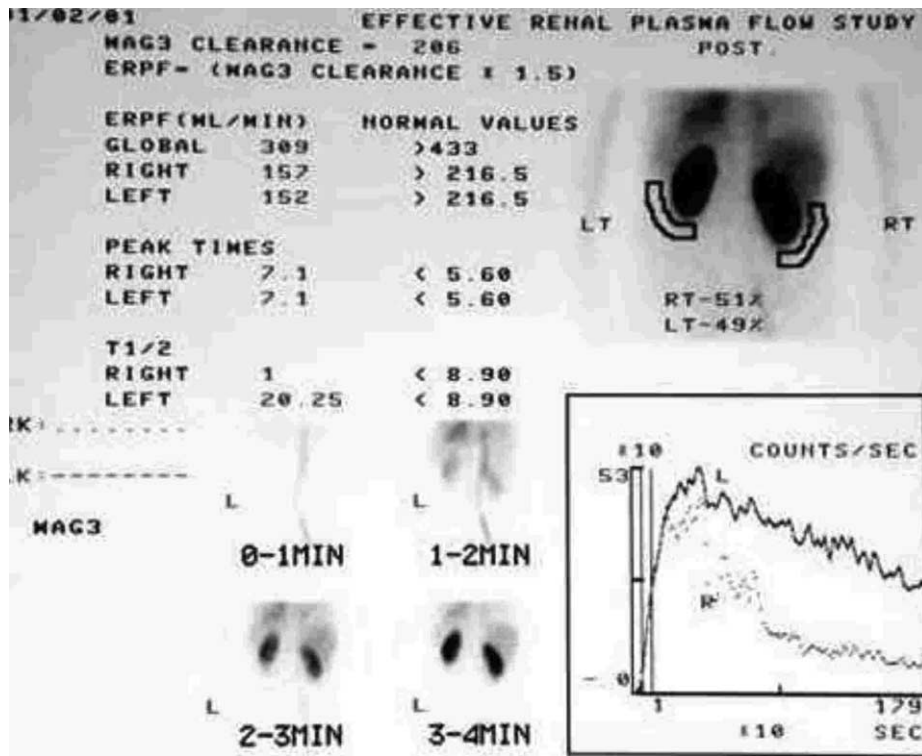
A rare complication found in the thoraco-lumbar region is Charcot arthropathy with a pseudarthrosis of the disc in which definitive exclusion of infection, e.g. tuberculosis may require vertebral biopsy.

*6.3. The acutely unwell patient*

Such patients present a diagnostic challenge as there is often severe infection present but because of the abnormal sensation and autonomic function compromise, localising the problem is very difficult. Often a non-specific trawl is necessary though the most common causes are the renal tract (see below), pressure sores and orthopaedic sepsis.

*6.4. Deteriorating renal function and renal tract complications*

Any change in dilatation of the upper tracts should be investigated fully with US and ERPF/MAG3 studies, particularly if it is acute and accompanied by symptoms ranging from a vague feeling of being unwell



(c)

Fig. 8. (Continued)

Table 5  
Indications for specific investigations

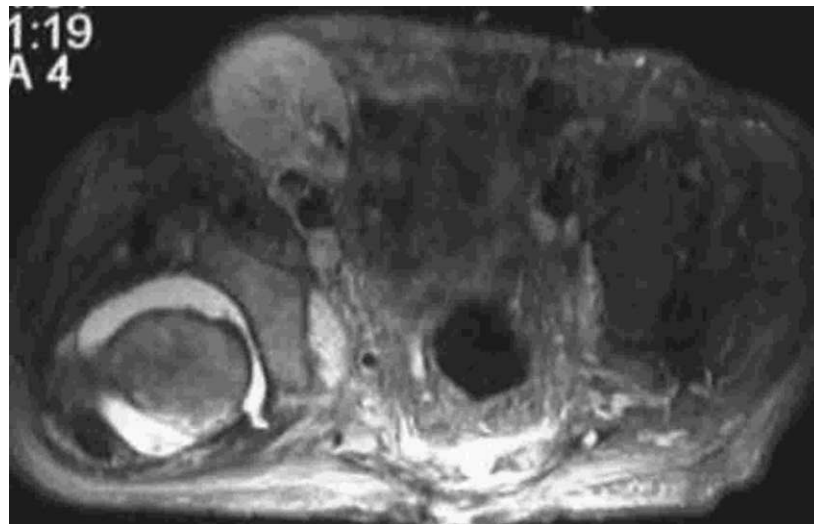
Condition	Investigation	Benefits and comment
Deteriorating neurology	MRI Sag T1, T2, axial T1	Differentiation of PPMM and syrinx (treatable) from atrophy and other non treatable causes
	CTM with delayed scanning	Exclude non neurological but treatable causes, e.g. disc, spinal stenosis
	US (intraoperative)	Confirmation of syrinx cavity size and location
Spinal instability	Plain films (flexion and extension, 30 min positioning)	Assessment of mechanical instability
	MRI CT Biopsy	Long cassettes for scoliosis monitoring Planning for choice of surgical options, e.g. pedicle screw size May need guided biopsy to rule out infection, e.g. tuberculosis
Acutely unwell	Routine CXR and AXR US abdomen—especially renal tract evaluation	It must be remembered that advanced sepsis often presents as non specific malaise, increased spasms, autonomic dysreflexia, etc.
	CT chest, abdomen and pelvis nephrostomy	A newly demonstrated hydronephrosis, especially if associated with stones, needs a nephrostomy to exclude infection
	PCNL	
Deteriorating renal function	AXR, US	Rule out obstruction, stones, etc.
	ERPF/MAG3 (IVP is rarely indicated)	Split function and handling assessment
	Urodynamics	Urodynamics for abnormal split function or high residual volumes in reflux voiders
	Cystourethrography	Assessment of reflux
Pressure sores Hip and joint problems	Plain films	Extent of involvement
	MRI T1 ± gadolinium STIR Sinography ± CT	Assessment of subcutaneous tissue damage (often conical and deep), osteomyelitis or septic arthritis Delineation of sinus or fissure tract
Ectopic ossification	US	Ultrasound is the earliest indicator of muscle and soft tissue perturbation which is a precursor to EO. DVT can also be excluded in the same examination
(HO, PAO)	Bone scintigraphy (isotope bone scan)	Bone scintigraphy is indicated where ultrasound skills are not available. It is probably most useful in monitoring activity prior to excision
	CT	CT is more sensitive than plain radiography, which is not positive until 10+ days after the start of the process
	Plain Film	Aim is to start treatment as soon as possible—diphosphonates or radiotherapy (ideally within 5 days of onset)
Pain Nociceptive Spine Visceral Neuropathic	Plain film (flexion/extension)	To assess stability of spine or visceral problems
	CT, MRI	To assess the state of the spine, e.g. ? bone infection To assist in planning of surgery
	MRI	Size morphology and extent of PPMM or syrinx and its relation to the fracture. To look for arachnoid tethering, CSF flow abnormalities, etc.
CNS		To confirm level of the lesion prior to laminectomy for DREZ lesion or rhizotomy surgery
Muscle spasms	US, AXR	General health assessment
	MRI	Cord assessment (rule out central cause or identify the site for laminectomy for posterior rhizotomy)
	Scintigraphy Cysternography Pump function	Assessment for intrathecal therapy (CSF pathway integrity) Level of blockage if pump fails
Airway patency	AP and lateral tomography CT (very low dose spiral) with multi planar reconstruction	Assessment of degree and level of tracheal stenosis
Elective surgery of the renal tract	US, AXR, Plain films	To exclude ureteric reflux, stones, etc.
	IVP	Exclude spinal anomalies
SARS, augmentation cystoplasty, artificial sphincter	Urodynamics Cystourethrogram	Aid surgical planning

to overt infection. Our unit's experience of over 60 nephrostomies in SCI patients feeling 'unwell' but not objectively very ill in whom a dilated upper tract was noted as a new finding, is that an obstructed kidney with urinary tract infection was invariably present and in approximately half, a frank pyonephrosis was found, reflecting the length of time from onset to presentation and the potentially fatal masking of symptoms. Stones are generally the cause and access to a full range of stone treatment techniques will be needed.

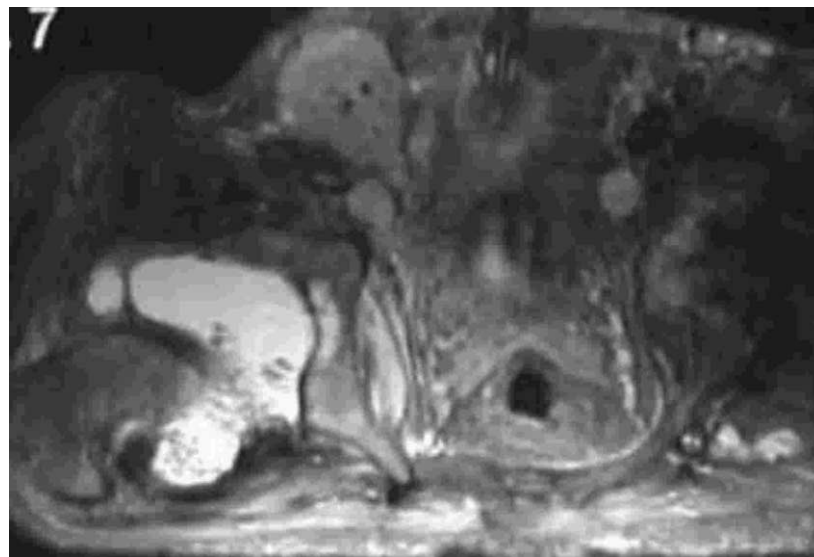
Cystography to assess for reflux may be required but autonomic dysreflexia is a hazardous complication of over distending the bladder, particularly in quadriplegics. It is advisable not to instill more than 250–350 ml with the investigation being terminated as soon as reflux is noted. In unstable patients where dysreflexia is likely, investigations should be terminated

and the bladder drained as soon as headaches and/or sweating is experienced. In patients where this is a possibility, an automatic blood pressure recorder should be used and sublingual nifedipine or a GTN patch may be required to reverse the massive hypertension occasionally suffered [73].

Urodynamics is now accepted as having a pivotal role in bladder management [68,70,81]. Multi-channel analysers that measure intra-vesical and intra-abdominal pressures to give detrusor muscle activity, combined with pelvic floor electromyography and video cysto-fluoroscopy are now used to assess detrusor function. In most centres, the decision to perform a sphincterotomy is based on urodynamic assessment together with the presence of other factors such as vesico-ureteric reflux, upper urinary tract abnormalities, symptomatic urinary infection or autonomic symptoms.



(a)



(b)

Fig. 9. Deep pressure sore—osteomyelitis. (a,b) Large joint effusion, no evidence of osteomyelitis.

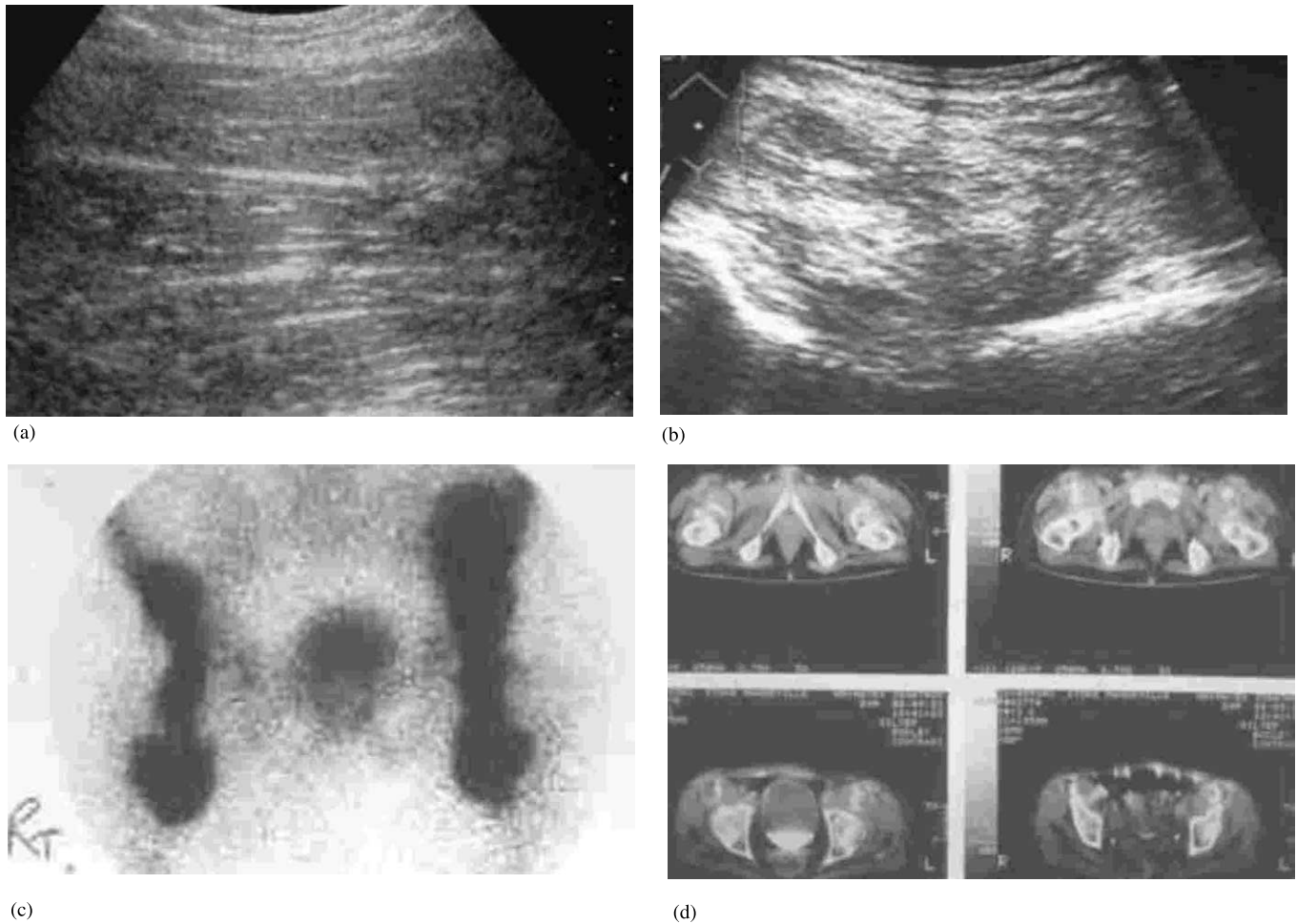


Fig. 10. Ectopic ossification. (a) US normal muscle. (b) US abnormal 'chaotic' muscle structure 1 day after clinical suspicion. (c) Tc HMDP bone scintigram showing extent of abnormality 7 days after clinical suspicion. (d) CT of the hips showing calcification in the quads.

With self intermittent catheterisation commonly practised, traumatic urethral complications are associated with sometimes less than dextrous manipulation of anaesthetic tissues. Urethrography, sometimes with catheter placement under screening control is required in this situation.

### 6.5. Pressure sores

Anaesthetic skin in a paralysed patient is particularly prone to pressure sores so thin cushions and prolonged waits must be avoided (2 h immobility on a stretcher may take 2 days to recover.) Once established, the depth of the injury is often much greater than expected from the surface. Secondary infection is common and septic arthritis and osteomyelitis must often be excluded. STIR sequences are invaluable (Fig. 9) and sinography followed by CT will frequently be necessary.

### 6.6. Ectopic ossification

Ectopic, heterotopic or para-articular ossification is an area of abnormal bone formation occurring in the denervated limb muscles, probably secondary to micro-trauma, commonly around the hips and knees, which generally presents in the second to fourth months post injury as a thick, swollen leg, probably secondary to trauma. Differentiation from deep venous thrombosis is critical and this can be readily performed using Doppler ultrasound when assessment of the muscles can also be made [83–85].

Ectopic ossification has a characteristic chaotic echo texture that replaces the normal lamellar structure of striated muscle (Fig. 10) and this finding is sufficient diagnostically to initiate treatment—radiotherapy [86], bisphosphonates, non-steroidals, etc.). Where the skill is available, this technique has been found to be more convenient, practical and cheaper for early diagnosis than the three phase isotope bone scan [87–91].

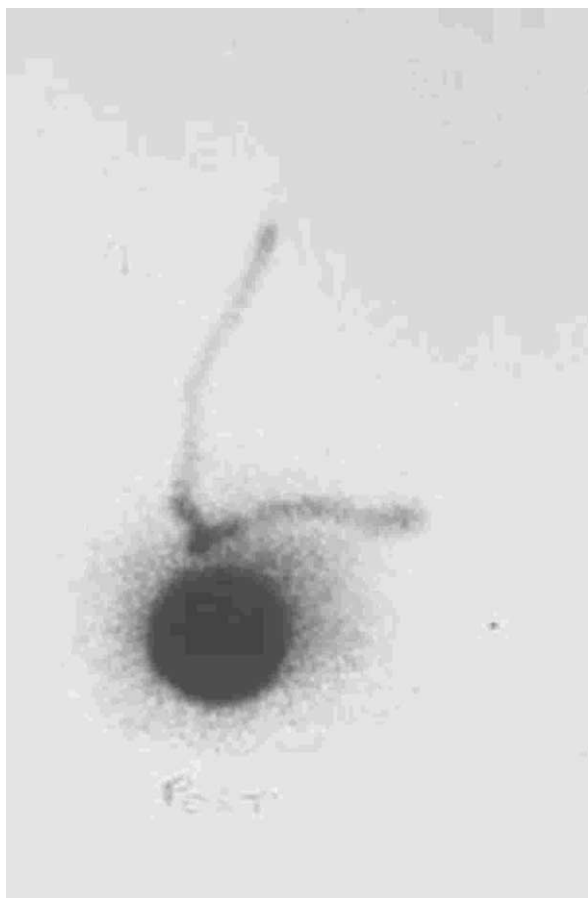


Fig. 11. Isotope pump study showing a blockage at the tip of the tube.

### 6.7. Pain

This can be considered as nociceptive when a spinal or visceral cause should be sought, or neuropathic, when a central cause is likely [92–94]. A full work-up of the whole patient including the spine and cord MRI may be necessary, with a special search for infection.

Surgery to the dorsal roots and pain pathways in the cord (DREZ lesions) may be offered and MRI is needed for level identification.

### 6.8. Muscular spasms

These can be a response to pain and a full pain assessment may be needed. If no underlying cause can be found then intrathecal antispastic drugs can be given via an implanted pump and catheter. The most useful technique for assessing possible pump failure is scintigraphy [95] (Fig. 11).

### 6.9. Airway problems

As well as problems that affect the lungs of normal people, patients with SCI have increased risks of tra-

cheal stenosis after tracheostomy and of infections if their neurological level compromises normal respiratory activity. Low dose CT of the trachea is more informative than tracheal tomography.

### 6.10. Elective management of the renal tract

Sacral anterior root stimulators, artificial sphincters, sphincter stents, augmentation cystoplasties, etc. all require a careful urological workup to exclude anatomical abnormalities, reflux, renal disease, stones.

## 7. Conclusion

With appropriate rehabilitation, a patient with a spinal cord injury can live an extremely fulfilled and useful life with the minimum of dependency on others. Regular surveillance and high quality imaging of the neurological and urological systems is critical to this goal but unwell patients are a diagnostic challenge that require complex imaging and thought to resolve.

## References

- [1] Whiteneck G, Charlifue S, Frankel H, Fraser M, Gardner B, et al. Mortality, morbidity and psychosocial outcomes of persons spinal cord injured more than 20 years ago. *Paraplegia* 1992;30:617–30.
- [2] Weitzenkamp DA, Jones RH, Whiteneck GG, Young DA. Ageing with spinal cord injury: cross-sectional and longitudinal effects. *Spinal Cord* 2001;39(6):301–9.
- [3] Quencer RM, Bunge RP. The injured spinal cord: imaging, histopathologic clinical correlates and basic science approaches to enhancing neural function after spinal cord injury. *Spine* 1996;21(18):2064–6.
- [4] Schwartz ED, Falcone SF, Quencer RM, Green BA. Post traumatic syringomyelia: pathogenesis, imaging and treatment. *Am J Reoentgenol* 1999;173(2):487–92.
- [5] Squier M, Lehr R. Post traumatic syringomyelia. *J Neurosurg Psychiatry* 1994;57:1095–8.
- [6] Jenson F, Reske-Nielsen E. Post traumatic syringomyelia. *Scand J Rehab Med* 1977;9:35–43.
- [7] Terae S, Taneichi H, Abumi K. MRI of Wallerian degeneration of the injured spinal cord. *J Comput Assist Tomogr* 1993;17(5):700–3.
- [8] Holtzman RNN, Yang WC. Chapter 9: Spinal cord atrophy. In: Holtzman RNN, Stein BN, editors. *Surgery of the Spinal Cord. Potential for Regeneration and Recovery*, 1992, pp. 165–196.
- [9] Fujii H, Kazunori Y, Sakou T. Magnetic resonance imaging study of experimental acute spinal cord injury. *Spine* 1993;18(14):2030–4.
- [10] Ohshio I, Hatayama A, Kaneda K, Takahara M, Nagashima K. Correlation between histopathologic features and magnetic resonance images of spinal cord lesions. *Spine* 1993;18(9):1140–9.
- [11] O'Beirne J, Cassidy N, Raza K, Walsh M, Stack J, Murray P. Role of magnetic resonance imaging in the assessment of spinal injuries. *Injury* 1993;24(3):149–54.
- [12] Silberstein M, Brown D, Tress B, Hennessey O. Suggested MRI criteria for surgical decompression in acute spinal cord injury. Preliminary observations. *Paraplegia* 1992;30:704–10.

- [13] Sgouros S, Williams B. A critical appraisal of pediculated omental graft transposition in progressive spinal cord failure. *Br J Neurosurg* 1996;10(6):547–53.
- [14] Bracken M, Shepard M, et al. A randomised, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury. *N Engl J Med* 1990;322:1405–11.
- [15] Holmes G. Goulstoman lecture on spinal injury of warfare. *Br Med J* 1915;2:769.
- [16] Geisler WO, Jousse AT, Wynne-Jones M, Breithaupt D. Survival in traumatic spinal cord injury. *Paraplegia* 1983;21:364–73.
- [17] Yang L, Jones N, Stoodley M, Bloombergs P, Brown C. Excitotoxic model of post traumatic syringomyelia in the rat. *Spine* 2001;26(17):1842–9.
- [18] Bonafe A, Aubin M. Syringomyelia. In: Manelfe C, editor. *Imaging of the Spine and Spinal Cord*. New York: Raven Press, 1992.
- [19] Williams B. Post traumatic syringomyelia, an update. *Paraplegia* 1990;28:296–313.
- [20] Cho KH, Iwasaki Y, Imamura H, Hida K, Abe H. Experimental model of post traumatic syringomyelia: the role of adhesive arachnoiditis in syrinx formation. *J Neurosurg* 1994;80(1):133–9.
- [21] Tobimatsu Y, Mihei R, Kimura T, Suyama T, Tobimatsu H. A quantitative analysis of CSF flow in post traumatic syringomyelia. *Seikeigeka Gakkai Zasshi* 1991;65(8):505–16.
- [22] Greitz D, Wirestam R, Franck A, Nordell B, Thomsen C, Stahlberg F. Pulsatile brain movement and associated hydrodynamic studies with magnetic resonance imaging. The Monro-Kellie doctrine revisited. *Neuroradiology* 1992;34(5):370–80.
- [23] Tobimatsu Y, Nihei R, Kimura T, Suyama T, Kimura H, Tobimatsu H, Shirakawa T. A quantitative analysis of cerebrospinal fluid flow in post-traumatic syringomyelia. *Paraplegia* 1995;33(4):203–7.
- [24] Brugieres P, Iffenecker C, Hurth M, Parker F, Fuerxer F, Idy-Peretti I, Bittoun J. Dynamic MRI in the evaluation of syringomyelic cysts. *Neurochirurgie* 1991;1(45 Suppl):115–29.
- [25] Heiss JD, Patronas N, DeVroom HL, Shawker T, Ennis R, Kammerer Edisath A, Talbot T, Morris J, Eskioğlu E, Oldfield EH. Elucidating the pathophysiology of syringomyelia. *J Neurosurg* 1999;1991(4):553–62.
- [26] Levy LM. Towards an understanding of syringomyelia; MR Imaging of CSF flow and neuraxis motion. *Am J Neuroradiol* 2000;21(1):45–6.
- [27] Hoffmann E, Warmuth-Metz M, Bendszus M, Solymosi L. Phase-contrast MR imaging of the cervical CSF and spinal cord: volumetric motion analysis with Chiari I malformation. *Am J Neuroradiol* 2000;21:151–8.
- [28] Hirai T, Korogi Y, Shigematsu Y, Sugahara T, Takahashi M, Ushio Y, Uemura S. Evaluation of syringomyelia with three-dimensional constructive interference in a steady state (CISS) sequences. *J Magn Reson Imaging* 2000;11(2):120–6.
- [29] Bakshi R, Caruthers SD, Janardhan V, Wasay M. Intraventricular CSF pulsation artifact on fast fluid-attenuated inversion recovery MR images: analysis of 100 consecutive normal studies. *Am J Neuroradiol* 2000;21(3):503–8.
- [30] Castillo M. Further explanations for the formation of syringomyelia; back to the drawing table. *Am J Neuroradiol* 2000;21:1788–9.
- [31] Brugieres P, Idy-Peretti I, Iffnecker C, Parker F, Jolivet O, Hurth M, Gaston A, Bittoun J. CSF Flow measurement in syringomyelia. *Am J Neuroradiol* 2000;21:1785–92.
- [32] Loss F, Yardimci MA, Alperin N. Hydrodynamic modelling of the CSF motion within the spinal cavity. *J Biomech Eng* 2001;123(1):71–9.
- [33] Freund M, Adwan M, Kooijman H, Heiland S, Thomsen M, Hahnel S, Jensen K, Gerner HJ, Sartor K. Measurement of CSF flow in the spinal canal using MRI with an optimized MRI protocol: experimental and clinical studies. *Rofo Fortschr Geb Röntgenstr Neuen Bildgeb Verfahr* 2001;173(4):306–14.
- [34] Inoue Y, Nemoto Y, Ohata K, Daikokuya H, Hakuba A, Tashiro T, Shakudo M, Nagai K, Nakayama K, Yamada R. Syringomyelia associated with adhesive spinal arachnoiditis; MRI. *Neuroradiology* 2001;43(4):325–30.
- [35] David P, Tadie M. Treatment of syringomyelia. *Neurochirurgie* 1999;1(45 Suppl):130–7.
- [36] Falci SP, Lammertse DP, Best L, Starnes CA, Prenger EC, Stavros AT, Mellick D. Surgical treatment of post traumatic cystic and tethered spinal cords. *Spinal Cord Med* 1999;22(3):173–81.
- [37] Ohata K, Gotoh T, Matsusaka Y, Morino M, Tsuyuguchi N, Sheikh B, Inoue Y, Hakuba A. Surgical management of syringomyelia associated with spinal adhesive arachnoiditis. *J Clin Neurosci* 2001;8(1):40–2.
- [38] Ronen J, Catz A, Spasser R, Gepstein R. The treatment in dilemma in post traumatic syringomyelia. *Disabil Rehabil* 1999;21(9):455–7.
- [39] Falcone S, Quencer R, Green B, Patchen S, Post MJD. Progressive post traumatic myelomalacic myelopathy; imaging and clinical features. *Am J Neuroradiol* 1994;15:747–84.
- [40] Fischbein MJ, Dillon WP, Cobbs C, Weinstein PR. The 'presyrinx' state; a reversible myelopathic condition that precedes syringomyelia. *Am J Neuroradiol* 1999;20(1):7–20.
- [41] Quencer R. The pre syringomyelic myelopathic state; a plausible hypothesis. *Am J Neuroradiol* 1999;20(1):1–2.
- [42] Milhorat TH. Is reversible enlargement of the spinal cord a pre syrinx state? *Am J Neuroradiol* 1999;20(1):21–2.
- [43] Nordqvist L. The sagittal diameters of the spinal cord and subarachnoid space in different age groups—a roentgenographic and postmortem study. *Acta Radiol (Suppl)* 1964;227:1–96.
- [44] Thijssen H, Keyser A, Horstink M, Meijer E. Morphology of the cervical spinal cord on computed myelography. *Neuroradiology* 1979;18:57–62.
- [45] Nakada T, Kwee I, Palmaz J. Computed tomography of spinal cord atrophy. *Neuroradiology* 1982;24:97–9.
- [46] Seibert CE, Barnes J, Dreisback JN, Swanson WB, Heck RJ. Accurate CT measurement of the spinal cord using metrizamide. *Am J Roentgenol* 1980;136:777–80.
- [47] Isherwood I, Fawcitt RA, Nettle JR, et al. Computer tomography of the spine. A preliminary report. In: duBoulay GH, Moseley IF, editors. *Computerised Axial Tomography in Clinical Practice*. Berlin: Springer, 1977:322–35.
- [48] Nidecker A, Kocher M, Maeder M, Gratz O, Zach G, et al. MR imaging of chronic spinal cord injury. Association with neurologic function. *Neurosurg Rev* 1991;14:169–79.
- [49] Yamashita Y, Takahashi M, Matsuno Y, Sakamoto Y, Oguni T, et al. Chronic injuries of the spinal cord: assessment with MR imaging. *Radiology* 1990;175:849–54.
- [50] Curati W, Kingsley E, Kendall B, Moseley I. MRI in chronic spinal cord trauma. *Neuroradiology* 1992;35:30–5.
- [51] Backe H, Betz R, Mesgarzadeh M, Beck T, Clancy M. Post-traumatic spinal cord cysts evaluated by magnetic resonance imaging. *Paraplegia* 1991;29:607–12.
- [52] Gebarski SS, Maynard FW, Gabrielsen TO, Knake JE, Latack JT, Hoff JT. Posttraumatic progressive myelopathy. Clinical and radiologic correlation employing MR imaging, delayed CT metrizamide myelography and intraoperative sonography. *Radiology* 1985;157:379–85.
- [53] Quencer R, Sheldon J, Post M, Diaz R, Montalvo B, et al. Magnetic resonance imaging of the chronically injured cervical spinal cord. *Am J Neuroradiol* 1986;7:457–64.
- [54] Barnett JM, Botterell E, Jouse A, Wynn Jones M. Progressive myelopathy associated with traumatic paraplegia. *Brain* 1966;89:159.
- [55] Gardner WJ, 1973. Syringomyelia following traumatic paraplegia. In: *The Dysraphic States*. Amsterdam: Excerpta Medica Offices, Chapter: 5, pp. 67–73.

- [56] Rossier AS, Werner A, Berney J. Contribution to the study of late cervical syringomyelic syndromes after dorsal or lumbar traumatic paraplegia. *J Neurol Neurosurg Psychiatr* 1968;31:99.
- [57] Sett P, Crockard A. The value of magnetic resonance imaging (MRI) in the follow-up management of spinal injury. *Paraplegia* 1991;29:396–410.
- [58] Wang D, Bodley R, Gardner B, Frankel H. A clinical-MRI study of the traumatised spinal cord more than 20 years following injury. *Paraplegia* 1994;34(2):65–81.
- [59] Jinkins JR, Reddy S, Leite CC, Bazan C, Xiong L. MRI of parenchymal spinal cord signal change as a sign of active advancement in clinically progressive post traumatic syringomyelia. *Am J Neuroradiol* 1998;19:177–82.
- [60] Quencer RM, Can MR. Help predict enlargement of post traumatic spinal cord cysts? *Am J Neuroradiol* 1998;19(1):192.
- [61] Silberstein M, Hennessy O. Cystic cord lesions and neurological deterioration in spinal cord injury; operative considerations based on magnetic resonance imaging. *Paraplegia* 1992;30:661–8.
- [62] Johnston L. Human spinal cord injury: new and emerging approaches to treatment. *Spinal Cord* 2001;39:609–13.
- [63] Quencer RM, Green BA, Eismont FJ. Post-traumatic spinal cord cysts: clinical features and characterisation with metrizamide computed tomography. *Radiology* 1983;146:415–23.
- [64] Dempsey MF, Condon B. Thermal injuries associated with MRI. *Clin Radiol* 2001;56:457–65.
- [65] Le CT, Price M. Survival from spinal cord injury. *J Chron Dis* 1982;35:487–92.
- [66] Gupta S, Chawla J. Review of upper tract abnormalities in 100 patients with spinal cord paralysis. *Paraplegia* 1994;32:531–9.
- [67] Lloyd KL. New trends in urologic management of spinal cord injured patients. *Cent Nerv Syst Trauma* 1986;3(1):3–11.
- [68] Wyndaele JJ. Neurourology in spinal cord injured patients. *Paraplegia* 1992;30:50–3.
- [69] Jamil F. Towards a catheter free status in neurogenic bladder dysfunction: a review of bladder management options in SCI. *Spinal Cord* 2001;39:355–61.
- [70] Wyndaele JJ. Correlation between clinical neurological data and urodynamic function in spinal cord injured patients. *Spinal Cord* 1997;35:213–6.
- [71] Perrouin-Verbe B, Labat JJ, Mathe JF. Bladder-sphincter disorders in patients with spinal cord injuries. *Rev Prat* 1995;45(16):2011–6.
- [72] Guttman L, Frankel H. The value of intermittent catheterisation on the early management of traumatic paraplegia and tetraplegia. *Paraplegia* 1966;4:63–83.
- [73] Karlsson AK. Autonomic dysreflexia. *Spinal Cord* 1999;37:383–91.
- [74] Chancellor M. Placement of a wire mesh prosthesis in the external urinary sphincter of men with spinal cord injuries. *Radiology* 1993;187:551–5.
- [75] McFarlane IP, Foley SJ, Shah PJ. Long-term outcome of permanent urethral stents in the treatment of detrusor-sphincter dyssynergia. *Br J Urol* 1996;78(5):729–32.
- [76] Foley SJ, Edhem I, Shah PJ. Use of Memokath temporary urethral stent in treatment of detrusor-sphincter dyssynergia. *J Endourol* 1997;11(6):485–8.
- [77] Kastler EJ, Thomas L, Bussel B, Chancellor MB, Richard F, Denys P. Feasibility of a temporary urethral stent through the striated sphincter in patients in the early phase (6 months) of spinal cord injury. *Eur Urol* 2001;39(3):326–31.
- [78] Lloyd KL, Dubovsky E, Bueschen A, Witten D, Scott J, Kuhle-meir K, Stover S. Comprehensive renal scintillation procedures in spinal cord injury: comparison with excretory urography. *J Urol* 1981;126:10–3.
- [79] Rao K, Hackler R, Woodlief R, Ozer M, Fields W. Real-time renal sonography in spinal cord injury patients: prospective comparison with excretory urography. *J Urol* 1986;135:72–7.
- [80] Klingensmith WC, Lammertse DP, Briggs DE, Smith WI, Roberts JF, Froelich JW, Sutherland JD. Tc 99m MAG3 renal studies in spinal cord injured patients: normal range, reproducibility and change as a function of duration and level of injury. *Spinal Cord* 1996;34:338–45.
- [81] Watanabe T, Rivas DA, Chancellor MB. Urodynamics of spinal cord injury. *Urol Clin North Am* 1996;23(3):459–73.
- [82] Gardner BP, Parsons KF, Soni BM, Krishnan KR. The management of upper urinary tract calculi in spinal cord damaged patients. *Paraplegia* 1985;23:371–8.
- [83] Thomas EA, Cassar-Pullicino VN, McCall IW. The role of ultrasound in the early diagnosis and management of heterotopic bone formation. *Clin Radiol* 1991;43(3):190–6.
- [84] Cassar-Pullicino VN, McClelland M, Badwan DA, McCall IW, Pringle RG, el Masry W. Sonographic diagnosis of heterotopic bone formation in spinal injury patients. *Paraplegia* 1993;31(1):40–50.
- [85] Bodley R, Jamous A, Short D. Ultrasound in the early diagnosis of heterotopic ossification in patients with spinal cord injury. *Paraplegia* 1993;31:500–6.
- [86] Colachis SC 3rd, Clinchot DM. The association between deep venous thrombosis and heterotopic ossification in patients with acute traumatic spinal cord injury. *Paraplegia* 1993;31(8):507–12.
- [87] Sautter-Bihl ML, Liebermeister E, Nanassy A. Radiotherapy as a local treatment option for heterotopic ossifications in patients with spinal cord injury. *Spinal Cord* 2000;38(1):33–6.
- [88] Naraghi FF, DeCoster TA, Moneim MS, Miller RA, Rivero D. Heterotopic ossification. *Orthopedics* 1996;19(2):145–51.
- [89] Banovac K, Gonzalez F. Evaluation and management of heterotopic ossification in patients with spinal cord injury. *Spinal Cord* 1997;35(3):158–62.
- [90] Subbarao JV, Garrison SJ. Heterotopic ossification: diagnosis and management, current concepts and controversies. *J Spinal Cord Med* 1999;22(4):273–83 Review.
- [91] Snoecx M, De Muynck M, Van Laere M. Association between muscle trauma and heterotopic ossification in spinal cord injured patients: reflections on their causal relationship and the diagnostic value of ultrasonography. *Paraplegia* 1995;33(8):464–8.
- [92] Milhorat TH, Kotzen RM, Mu HT, Capocelli AL, Milhorat RH. Dysaesthetic pain in patients with syringomyelia. *Neurosurgery* 1996;38(5):940–7.
- [93] Bowsher D. Central pain following spinal and supraspinal lesions. *Spinal Cord* 1999;37:235–8.
- [94] Siddall PJ, Loeser JD. Pain following spinal cord injury. *Spinal Cord* 2001;39:63–73.
- [95] Le Breton F, Davies JC, Monteil J, Vidal J, Munoz M, Dugognon P, Salle JY. Radioisotopic control for baclofen pump catheter failure. *Spinal Cord* 2001;39:283–5.