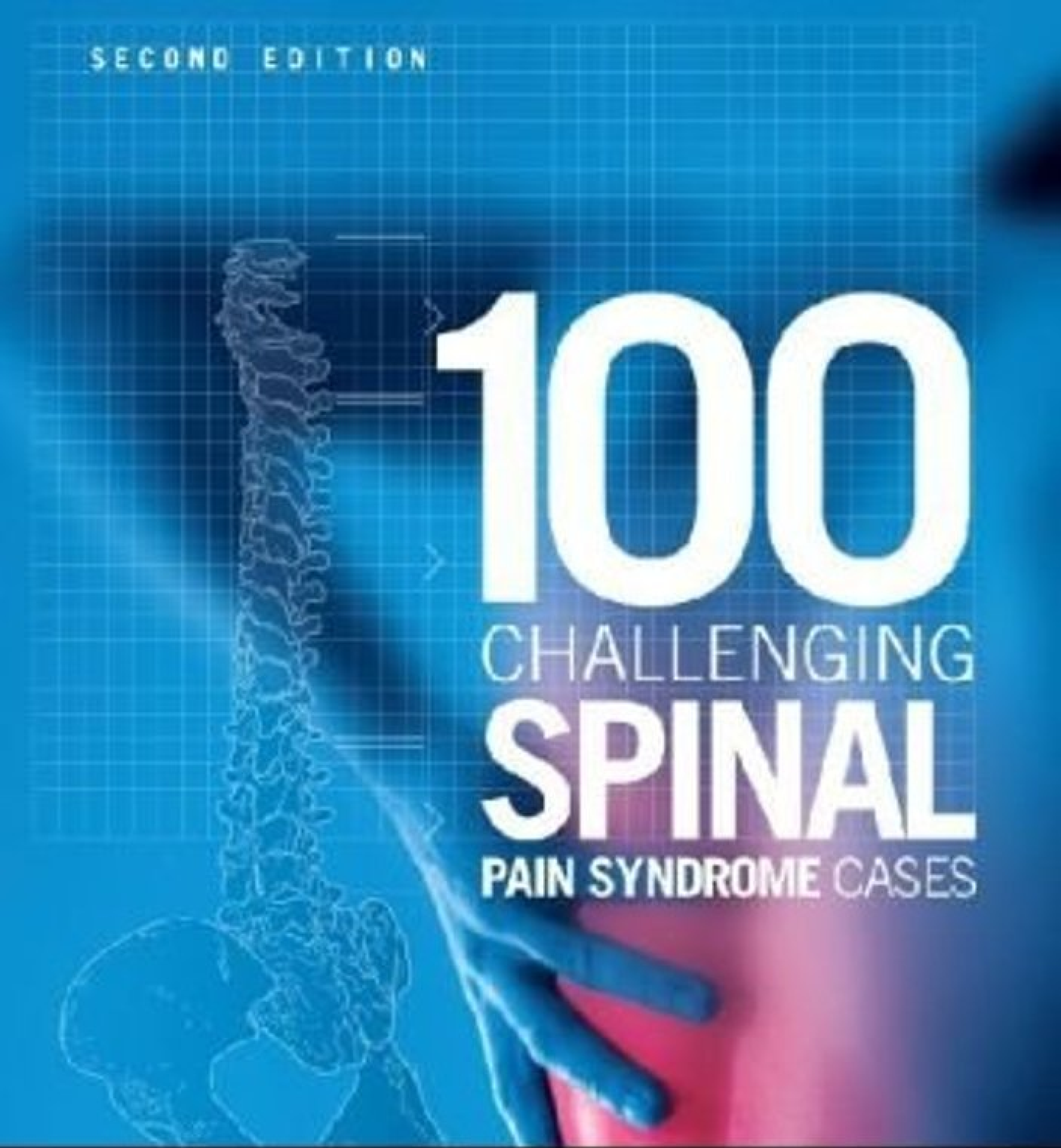
SECOND EDITION
100
CHALLENGING
SPINAL
PAIN SYNDROME CASES

CHURCHILL
LIVINGSTONE
ELSEVIER

An imprint of Elsevier Limited

First published 2009

ISBN-13: 978–0-443–067167

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging in Publication Data
A catalog record for this book is available from the Library of Congress

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Printed in China

To Jennifer

My best friend, wife and colleague for her unqualified support and painstaking efforts in helping to produce this text

Preface

Conditions causing spinal pain syndromes are many and varied and patients can present with a wide variety of symptoms. Therefore, each patient needs to be thoroughly investigated before treatment commences i.e. diagnosis must be a prerequisite to treatment.

It behoves all clinicians dealing with spinal pain syndromes to note the following:

- First, do no harm (Hippocrates, Epidemics, Bk. 1, Sect. XI).
- "If you listen carefully to the patient they will tell you the diagnosis" (Quotation of Sir William Osler 1849–1919). This statement still holds true and emphasizes the great importance of taking a good history.
- Remember that patients may present with more than one condition, so consider all possibilities.
- Get back to the basic principles (Frymoyer JW 1997 Foreword. Clinical anatomy and management of low back pain. In: Giles LGF and Singer KP, Butterworth–Heinemann, Edinburgh) e.g. be conversant with the principles of anatomy, physiology, pathology, etc.

Knowledge is ever increasing on spinal anatomy and histopathology and the possible physiological mechanisms by which pain may be generated and experienced. Therefore, in this text an introductory chapter summarizes possible pain sources based on known anatomical principles. Because spinal pain syndromes can be complex, there often is a tendency for clinicians to incorrectly label patients as being 'neurotic', or when patients are involved in litigation they may be considered to have litigation 'neurosis' as a motive. However, it should be remembered that it is not always possible to diagnose a patient's spinal pain condition because of many factors such as the limitations of imaging procedures and the specificity and sensitivity of laboratory tests. Therefore, patients should not be considered as malingerers unless there are very strong grounds for doing so. Imaging frequently only provides shadows of the truth and laboratory tests can be misleading, so it is imperative to take a careful history and to perform a thorough physical examination followed, as indicated, by appropriate imaging and laboratory procedures.

During the last few years of a 37-year career specializing in spinal pain syndromes, interesting challenging spinal pain syndrome cases have been collected, 100 of which are presented in this text. In some of the cases presented, gross anatomy and pathology, as well as some histopathology specimens, obtained from postmortem material, with changes similar to the clinical cases presented, are used to illustrate how such conditions in patients may cause spinal pain syndromes and provide a basis upon which to recommend treatment options.

In some cases patients merely wanted reassurance, based upon a thorough evaluation leading to a likely explanation for their chronic pain syndrome(s), rather than requesting treatment. The importance of providing adequate, albeit time consuming, psychological assurance should not be underestimated. Obviously, it is important to consider a particular pain syndrome in great detail while not forgetting that psychology must be taken into account for each patient, as symptoms and signs should not be isolated from the patient as a whole being.

The cases begin with the most frequently involved spinal level (lumbar spine) and conclude with the least frequently involved spinal level (thoracic spine).

The cases represent what actually takes place in day-to-day clinical practice and illustrate some of the various shortcomings of health care providers.

It should be noted that it is the responsibility of the treating clinician, relying on independent expertise and knowledge of the patient, to determine the best treatment and method of application for their individual patients.

Finally, all clinical professionals may make errors of judgement in the diagnosis and management of patients. Therefore, it is not the intention of this text to criticize any particular profession but rather to draw to the attention of health practitioners from various backgrounds what actually takes place in the health care domain in the hope that clinicians, and students embarking upon a health care career, will glean some insight into the possible difficulties that may arise with respect to individual cases.

In my opinion, multidisciplinary cooperation is essential if clinicians from different backgrounds are to best serve individuals with spinal pain syndromes and the possible sequelae of such syndromes – no one profession has all the answers to manage challenging acute and chronic spinal pain syndrome patients.

L.G.F. Giles

General introduction

INTRODUCTION

The diagnosis of spinal pain syndromes is often difficult, as the anatomy of the spine and its adjacent soft tissue structures is complex. For details of spinal anatomy including histology and histopathology see Rickenbacher et al (1982), Giles (1989), Von Hagens et al (1991), Rauschning (1997), Giles & Singer (1997, 1998, 2000), Cramer & Darby (2005), Moore & Dalley (2006). The purpose of this introduction is to provide a synopsis of possible diagnoses based on a sound anatomical foundation as well as on the clinical history, physical examination, imaging and laboratory findings. In addition, it should be noted that the neurophysiology of pain is not fully understood at this time. For example, when Slipman et al (1998) studied the mechanical stimulation of cervical nerve roots C4 to C8 in patients with cervical radicular symptoms who were undergoing diagnostic selective nerve block, to document the distribution of pain and paraesthesiae that result from stimulation of specific cervical nerve roots, and to compare that distribution to documented sensory dermatomal maps, they demonstrated a distinct difference between *dynatomal* and *dermatomal* maps. A dynatome is the distribution of referred symptoms from root irritation and this is different to the sensory deficit outlined by dermatomal maps. Slipman et al (1998) suggest that cervical dermatomal mapping is inaccurate, as the distribution of referred symptoms from cervical root irritation is different than the sensory deficit outlined by dermatomal maps. Therefore, it is reasonable to suggest that a similar neurophysiological finding may occur at other spinal nerve root levels. Thus, when considering neurological tests such as pinprick or light touch, summarized later in this text, it would be prudent to remember the work of Slipman et al (1998).

Low back pain is experienced by 80–90% of the population (Deen 1996), while 34–40% of the population experience neck and arm pain (Hardin & Halla 1995) compared to 7–14% of the population that experience thoracic spine pain (Pedersen 1994, Hinkley & Drysdale 1995). Therefore, musculoskeletal spinal pain can be a significant health problem. On analysing 1775 new patients presenting to a multidisciplinary spinal pain unit Giles et al (2003) found that, of the 949 male patients and 826 female patients (aged 10 to 91 years; average age 43 years), all of whom had some form of spinal imaging, 1% of patients had radiologically identifiable life threatening pathological processes. This percentage concurs with the serious pathology percentage of up to 1% as stated by Waddell (2004).

Pain in any structure requires the release of inflammatory agents, including bradykinin, prostaglandins and leukotrienes, that stimulate pain receptors and generate a nociceptive response in the tissue; the spine is unique in that it has multiple structures that are innervated by pain fibres (Haldeman et al 2002).

Spinal pain syndromes must be viewed in the context of (i) clearly defined pathological conditions, and (ii) the less well-defined, but much more prevalent, condition of non-specific spinal pain of mechanical origin (Stoddard 1969, Kenna & Murtagh 1989). It is imperative to distinguish dysfunctional mechanical causes of spinal pain from other causes, as patients with mechanical disorders of the spine are likely to respond dramatically to manual treatment (Kenna & Murtagh 1989).

A major difficulty involved in evaluating a patient with non-specific spinal pain of mechanical origin, with or without root symptoms, is that many causes of pain are possible. Because the painful structure, or structures, are not amenable to direct scrutiny, a tentative diagnosis is usually arrived at for an individual case by taking a careful case history and employing a thorough physical examination, with imaging and laboratory procedures as indicated. There are several main approaches to patient evaluation: (i) history taking; (ii) assessment of pain (using subjective self-report measures estimating pain severity, quality and location); (iii) investigation of personality structure, including the use of appropriate subjective questionnaires; (iv) clinical identification of signs and symptoms; (v) the use of appropriate imaging; and (v) the use of appropriate laboratory investigations. Evidence of signs and symptoms deemed excessively, or inappropriately, abnormal (Main & Waddell 1982) should be recorded. However, caution has to be exercised when making judgements on an individual's behavioural responses to examination as serious misuse and misinterpretation of behavioural signs has occurred in medicolegal contexts using such signs (Main & Waddell 1998). These behavioural signs have been questioned by Giles (2005).

There is still little consensus, either within or among specialties, on the use of diagnostic tests for patients with spinal pain syndromes, and the underlying pathology responsible for various spinal pain problems remains elusive (Videman et al 1998). Furthermore, in spite of following a thorough examination procedure, one often merely eliminates overt pathologies and the precise cause of non-specific spinal pain syndromes of mechanical origin often remains obscure (Turner et al 1998).

Specifically, diagnostic problems relate to: (a) the limitations of many diagnostic procedures, including plain film radiography, computerized tomography (CT), positron emission tomography (PET) scan, magnetic resonance imaging (MRI), myelography, discography and bone scans; (b) some diagnostic and therapeutic chemical agents being harmful, as can be the case when such chemicals injected into intervertebral discs extravasate into the epidural space (Weitz 1984, Adams et al 1986, MacMillan et al 1991) causing complications due to contact between them and neural structures (Dyck 1985, Merz 1986, Watts & Dickhaus 1986); (c) inadequacies in the precise anatomical knowledge of the spine including its nociceptive tissues; (d) anatomical complexity of the spine often making roentgenographic interpretation difficult (Le-Breton et al 1993); and (e) there sometimes being multifactorial causes of pain at a given level of the spine (Haldeman 1977, Gross 1979), e.g. injury to the intervertebral disc, the zygapophysial facet joints and to the segmental soft tissue structures. In addition, there may be several types of spinal pain that closely mimic each other (Haldeman 1977). A further important point to remember is that a *central* disc herniation may cause spinal pain alone without radiculopathy (Postacchini & Gumina 1999) whereas a posterolateral or far lateral disc herniation will, in all likelihood, cause radicular pain (Keim & Kirkaldy-Willis 1987).

The nerve root compression that occurs in lumbar disc herniation and lumbar canal stenosis often results in a range of symptoms, including low back pain, sciatic pain, and sensory disturbances and muscle weakness in the legs (Kobayashi et al 2005). Summers et al (2005) consider that the degree of back or leg pain caused by an acute disc prolapse depends, in part, on the position, size, and level of the disc prolapse.

There often is disagreement on which imaging procedures have diagnostic validity for non-specific spinal pain of mechanical origin, although it is generally agreed that, for plain film X-ray examinations, two views of the same anatomical region at right angles is the minimum requirement (Henderson et al 1994), and that erect posture radiography (Giles & Taylor 1981) and functional views (Jackson 1977) are more useful. Furthermore, Buirski & Silberstein (1993) correctly noted that MRI can only be used as an assessment of nuclear anatomy and not for symptomatology, and Schellhas et al (1996) showed that significant cervical disc annular tears often escape MRI detection. In addition, Osti & Fraser (1992) concluded that lumbar discography is more accurate than MRI for the detection of annular pathology. However, according to Shalen (1989), lumbar discography is a controversial examination that is regarded by some radiologists and spine surgeons as barbaric and non-efficacious (Wiley et al 1968, Clifford 1986, Shapiro 1986). For lumbar spine CT and MR imaging, Willen et al (1997) showed that the diagnostic specificity of spinal stenosis will increase considerably when the patient is subjected to an axial load, and Danielson et al (1998) concluded that, for an adequate evaluation of the cross-sectional area, CT or MR imaging studies should be performed with axial loading in patients who have symptoms of lumbar spinal stenosis. The advantage of MRI, which is considered to be the major medical imaging development of the century (Wong & Transfeldt 2007), is that it is a non-invasive procedure, without any recognized biological hazard, that combines a strong magnetic field and radiofrequency energy to study the distribution and behaviour of hydrogen protons in fat and water (Weir & Abrahams 2003). More recently, exciting new developments have occurred with, for example, upright, dynamic-kinetic i.e. 'functional' MRI and its ability to detect load- and motion-dependent disc herniations, stenosis, instabilities, and combinations of these pathologies not seen during recumbent imaging (Jinkins et al 2003, Elsig & Kaech 2006, Jinkins JR personal communication 2007). In addition, 3 Teslar (T) MRI units are becoming available that can provide higher-quality images than those obtained at 1.5 T (Tanenbaum 2006).

In the thoracic spine, with its particular combination of intervertebral and various synovial joints, the most common cause of thoracic spine pain syndromes is dysfunction and degeneration of spinal intervertebral joints and the associated rib articulations (Kenna & Murtagh 1989). Furthermore, Erwin et al (2000) showed that costo-vertebral joint anterior capsule and synovial tissues are innervated, demonstrating that these joints have the requisite innervation for pain production. Root compression due to posterolateral disc protrusion, with resulting signs and symptoms of intercostal radiculopathy, such as pain, paraesthesiae and sensory disturbances, has to be differentiated from overt pathological conditions such as neoplasms. Thoracic disc protrusion has long been a difficult clinical entity to diagnose (Brown et al 1992) as symptoms can vary dramatically from none at all to motor and sensory deficits resulting from spinal cord compression (myelopathy) – pain, muscle weakness, and spinal cord dysfunction are the most common clinical symptoms (Cramer 2005). On the other hand, thoracic disc protrusion can produce spinal cord compression with bladder incontinence and signs of an upper motor neuron lesion (Kenna & Murtagh 1989) and occasionally paraplegia. As the thoracic cord is immobilized by the dentate ligaments, the anterior spinal artery may be significantly compressed by a relatively small central disc protrusion (Pate & Jaeger 1996) and, because there is little extradural space in the thoracic spinal canal, a comparatively small disc protrusion may have pronounced effects on the neurology (Hoppenfeld 1977). Thoracic cord compression can also be caused by ossified ligamenta flava in Caucasians (van Oostenbrugge et al 1999) as well as in Japanese people (Otan et al 1986, Yonenobu et al 1987, Kojima et al 1992).

As it is still only rarely possible to validate a diagnosis in cases where pain arises from the spine (White & Gordon 1982) and, because it is not possible to establish the pathological basis of spinal pain in 80–90% of cases (Chila et al 1990, Spratt et al 1990, Pope & Novotny 1993), this leads to diagnostic uncertainty and suspicion that some patients have a ‘compensation neurosis’ or other psychological problem. It is also appropriate at this time to recognize the role of psychosocial factors in spinal pain. Although the complex interaction of psyche and soma in the aetiology of spinal pain is not well understood, a psychogenic component may be primary (conversion disorder), secondary (depression caused by chronic pain), contributory (myofascial dysfunction), or absent (Keim & Kirkaldy-Willis 1987). Nonetheless, clinicians must have a good understanding of possible causes of symptoms and a high suspicion of possible underlying pathology, e.g. patients presenting with symptoms of tethered cord syndrome (Yamada 1996, Giles 2003, Yamada et al 2004), or patients presenting with symptoms of arteriovenous malformations (Criscuolo & Rothbart 1992), i.e. an abnormal communication between an artery and a vein (Beers et al 2006), should not be overlooked.

It is reasonable to broadly classify acute spinal pain as being of 7–28 days or less duration, which may be followed by a sub-acute stage of up to 12 weeks; after this the pain can be considered chronic (Skoven et al 2002).

HISTORY OF SPINAL PAIN

The importance of an exhaustive case history cannot be overemphasized and it should take into account facts such as the patient’s age, occupation, onset of pain, previous injuries, medication, recreational activities, pain aggravation and characteristics, location, distribution, and any related neurological symptoms (numbness, paraesthesiae, muscle weakness) and whether compensation is involved regarding an injury. Some conditions provide reasonably characteristic patterns, while others do not. For example, pain that occurs at night, and which is relieved by aspirin, may be associated with an osteoid osteoma, that is a benign tumour of bone (Keim & Kirkaldy-Willis 1987). Night pain *per se* should be considered as being of probable serious pathological change. Likewise, night sweats may suggest a serious underlying pathology.

Taking into consideration the abovementioned issue of dermatomes and dynatomes (Slipman et al 1998), an important neurological concept that has been recognized for anatomically normal spines and which needs to be considered during the examination is that the distribution of cutaneous areas supplied with afferent nerve fibres by single posterior spinal nerve roots, i.e. dermatomes of the human body (Dorland’s *Illustrated Medical Dictionary* 1974, Barr & Kiernan 1983), has been fairly well established (Fig. i). Myotomes, a group of muscles innervated from a single spinal segment (Dorland’s *Illustrated Medical Dictionary* 1974), likewise, have been fairly well established.

According to Keim & Kirkaldy-Willis (1987), dermatomes enable deficits of a specific nerve root to be accurately localized during sensory examination. However, it is important to note that Jinkins (1993) and Slipman et al (1998) question this concept and suggest that there is, in fact, some overlap of sensation.

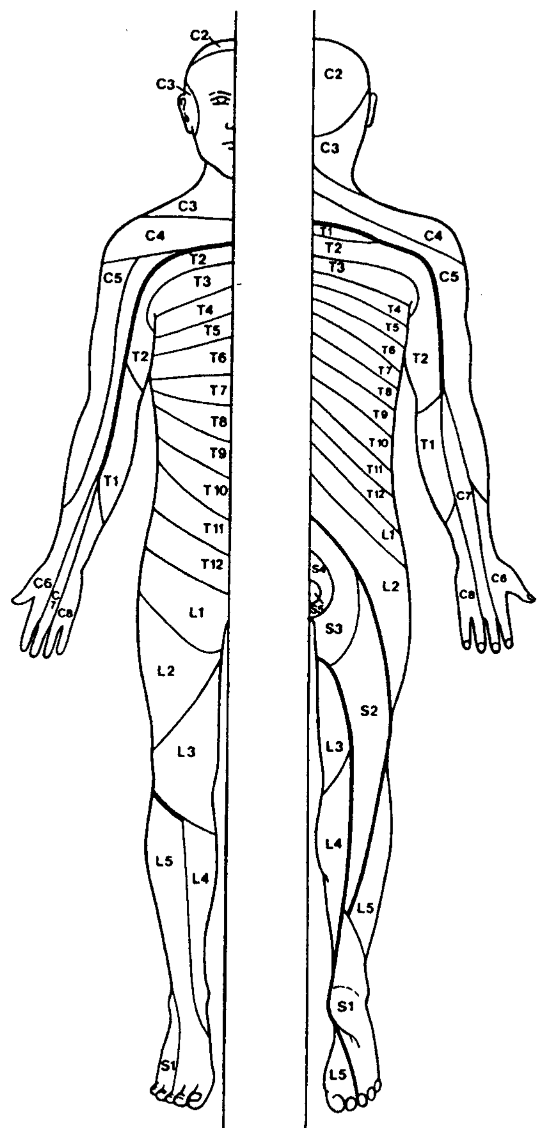


Figure i Dermatomes on the anterior and posterior surfaces of the body. Axial lines, where there is numerical discontinuity, are drawn thickly. (Modified from Wilkinson, JL 1986 Neuroanatomy for medical students. John Wright & Sons, Bristol, p 29.)

Until a thorough history has been taken and a complete physical examination and any appropriate imaging or laboratory tests have been performed, as considered necessary for a particular patient, to rule out organic disease, it is not wise to label a patient as being neurotic or a malingerer, particularly as it is thought that such patients form only a small minority of cases (Teasell 1997). Furthermore, there has long been a misconception that *all* injuries should heal after 6 weeks; however, clinical experience and follow-up studies (Mendelson 1982, Radanov et al 1994) clearly demonstrate that not *all* patients necessarily get better and that there is a significant subset who continue to suffer from chronic symptoms (Teasell 1997).

It is worth noting the sobering comment of orthopaedic Professor Ruth Jackson in her classic textbook *The Cervical Syndrome* (1977) in which she wrote:

> 'When one who is not completely versed in the symptomatology of disorders of the cervical spine has completed this portion of the examination, he may have drawn the conclusion that the patient is psychoneurotic. However, to draw such a conclusion is a reflection on the examiner's diagnostic ability and not on the patient, until proven otherwise.'

This comment applies equally well to all regions of the complex human spine.

BRIEF SUMMARY OF SPINAL INNERVATION

The overall basic pattern of innervation of the spine can briefly be outlined by looking at the anatomy of the lower lumbosacral spine as shown schematically in Figures ii and iii.

The segmental innervation of the lumbar, cervical and thoracic spines is extremely complex as partly shown in Figures iv, v and vi, respectively. Although part of the innervation of the anterior and posterior elements of the lumbar spine is shown schematically in Fig. iv, the innervation shown for the cervical and thoracic spines is less detailed in order to simplify the diagrams. However, the basic neuroanatomical principles shown for the lumbar spine are largely the same for each spinal level, bearing in mind the different osteological and soft tissue structures for the three levels of the spine; full details of spinal innervation can be found in standard textbooks such as *Gray's Anatomy* and *Moore's Clinically Oriented Anatomy*, or in anatomy texts specifically related to the spine (Cramer & Darby 2005).

Possible anatomical variations in nerves should be kept in mind in view of their associated clinical importance. For example, in the lumbar spine the furcal nerve, i.e. an accessory spinal nerve originating from the cord independently of other lumbar nerve roots that, like the latter, includes a ventral and a dorsal component; the furcal nerve can be found in all subjects, is generally single and, in most cases, its roots emerge from the spinal cord and run within the thecal sac beside the L4 roots (Kikuchi et al 1986; Postacchini & Rauschning 1999) with the furcal nerve

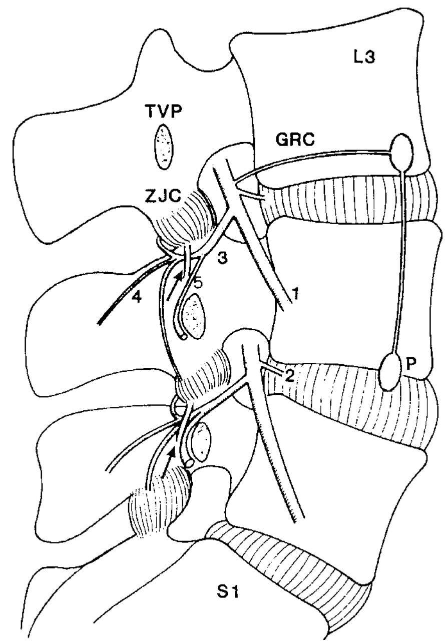


Figure ii A schematic diagram showing part of the lower lumbar (i.e. L3 to S1 levels) spinal innervation (lateral view). 1 = anterior primary ramus of the spinal nerve; 2 = anterior primary ramus branch to the intervertebral disc; 3 = posterior primary ramus of the spinal nerve; 4 = medial branch of the posterior primary ramus with an adjacent zygapophysial joint capsule (articular) branch, and a descending articular branch to the zygapophysial joint capsule one joint lower; 5 = lateral branch of the posterior primary ramus; GRC = grey ramus communicans; TVP = transverse process; ZJC = zygapophysial joint capsule; arrow = mamillo-accessory ligament. (Reproduced with permission from Giles L G F 1989 Anatomical basis of low back pain. Baltimore, Williams & Wilkins, p 60).

usually formed by the fourth lumbar root (Izci et al 2005). Occasionally, two furcal nerves may be present, i.e. L3 and L4 or L4 and L5; sometimes only one L5 furcal nerve is present. The dorsal root of the furcal nerve has a ganglion situated, as for the normal dorsal roots, at the level of the intervertebral foramen; once the nerve has left the intervertebral foramen with the roots proper of that level, with which it constitutes a single radicular nerve, it gives off three branches, contributing to form, respectively, the femoral nerve, the obturator nerve, and the lumbosacral trunk (Postacchini & Rauschning 1999). The clinical relevance of the furcal nerve is that disc herniation, or other pathological conditions, may impinge upon both the radicular nerve proper of that level and on the furcal nerve, thus causing atypical bi-radicular syndromes (Kikuchi et al 1986; Postacchini & Rauschning 1999). According to Haijiao et al (2001), MRI provides accurate information on lumbosacral nerve root anomalies.

The thoraco-lumbar junction gives rise to the cluneal nerves providing innervation of the lower lumbar region, below the iliac crest, i.e. by sensory nerves coming from the T11, T12, L1 and L2 roots; these roots give rise to a short dorsal ramus that divides into two branches – a medial branch and a

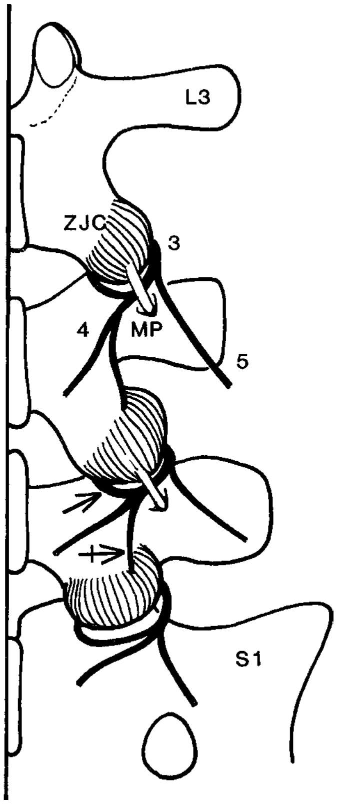


Figure iii A schematic diagram showing part of the lower lumbar spinal innervation (posterior view). 3 = posterior primary ramus of the spinal nerve; 4 = medial branch of the posterior primary ramus with an adjacent zygapophysial joint capsule (articular) branch (arrow), and a descending articular branch to the zygapophysial joint capsule one joint lower (bisected arrow); 5 = lateral branch of the posterior primary ramus; MP = mammillary process with mamillo-accessory ligament; ZJC = zygapophysial joint capsule. (Reproduced with permission from Giles LGF 1989 Anatomical basis of low back pain. Baltimore, Williams & Wilkins, p 61.)

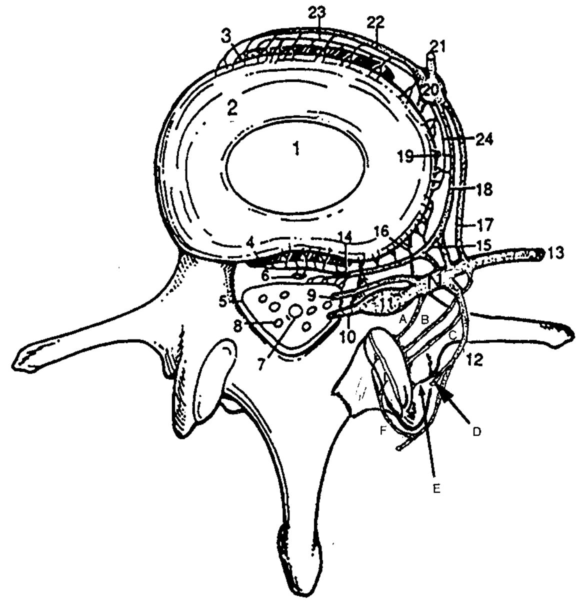


Figure iv A schematic diagram showing basic lumbar vertebral anatomy and part of the innervation of the anterior and posterior structures of the vertebral column: 1 = nucleus pulposus; 2 = annulus fibrosus; 3 = anterior longitudinal ligament/periosteum; 4 = posterior longitudinal ligament/periosteum; 5 = dural tube; 6 = epidural vasculature; 7 = filum terminale; 8 = intrathecal lumbosacral nerve root; 9 = ventral (anterior) root; 10 = dorsal (posterior) root; 11 = dorsal root ganglion (spinal ganglion); 12 = dorsal ramus of spinal nerve; 13 = ventral ramus of spinal nerve; 14 = recurrent meningeal nerve (sinuvertebral nerve of von Luschka); 15 = autonomic (sympathetic) branch to recurrent meningeal nerve; 16 = direct somatic branch from ventral ramus of spinal nerve to lateral disc; 17 = white ramus communicans (not found caudal to L2); 18 = grey ramus communicans (multilevel irregular lumbosacral distribution); 19 = lateral sympathetic efferent branches projecting from grey ramus communicans; 20 = paraspinal sympathetic ganglion (PSG); 21 = paraspinal sympathetic chain; 22 = anterior paraspinal afferent sympathetic ramus projecting to PSG; 23 = anterior sympathetic efferent branches projecting from PSG; 24 = lateral paraspinal afferent sympathetic ramus projecting to PSG. (Note: afferent and efferent sympathetic paraspinous branches/rami may be partially combined *in vivo*.) A = neural fibres from main trunk of spinal nerve (N); B = neural fibres from ventral ramus (13) of spinal nerve; C = neural fibres from dorsal ramus (12) of spinal nerve; E = neural fibres from medial branch (D) of dorsal ramus (12); F = neural fibres from lateral branch of dorsal ramus (12) of the spinal nerve. (Modified from: Jinkins JR et al 1989 American Journal of Neuroradiology 10: 219–251, American Journal of Roentgenology 152: 1277–1279; Jinkins JR 1997 Clinical anatomy and management of low back pain, p 255–272; Auteroche P 1983 Anatomia Clinica 5: 17–28.)

lateral branch that innervate the skin and are known as the superior cluneal nerve (Maigne 1980; Maigne & Maigne 1991). The cluneal nerves can be associated with low back pain in the region of the posterior iliac crest (Maigne 1980, Maigne 2000) and with pain projecting into the buttock (Maigne & Doursounian 1997).

In the neck, the vertebral nerve that originates from near the superior pole of the stellate ganglion, and rarely from the inferior cervical ganglion, is usually a single filament and, in essence, is a deeply placed long gray ramus communicans from the 6th or 7th cervical nerves that connects the stellate or inferior cervical ganglia to the lower cervical spinal nerves (Tubbs et al 2007). Many of the fibres from this ramus (C6) become incorporated in the sympathetic plexus surrounding the vertebral artery (Axford 1928) and branches from the vertebral nerve join the perivertebral nerve plexus that accompanies the basilar and superior cerebellar arteries intracranially (Lang 1993). Articular and meningeal branches were sometimes identified by Tubbs et al (2007) emanating from the stellate ganglion; Tubbs et al (2007) concluded that this may be important in treating medically intractable neck pain by allowing neural blockade of the vertebral nerve.

Among various other structures that may be involved in a patient's symptoms are the transforaminal ligaments

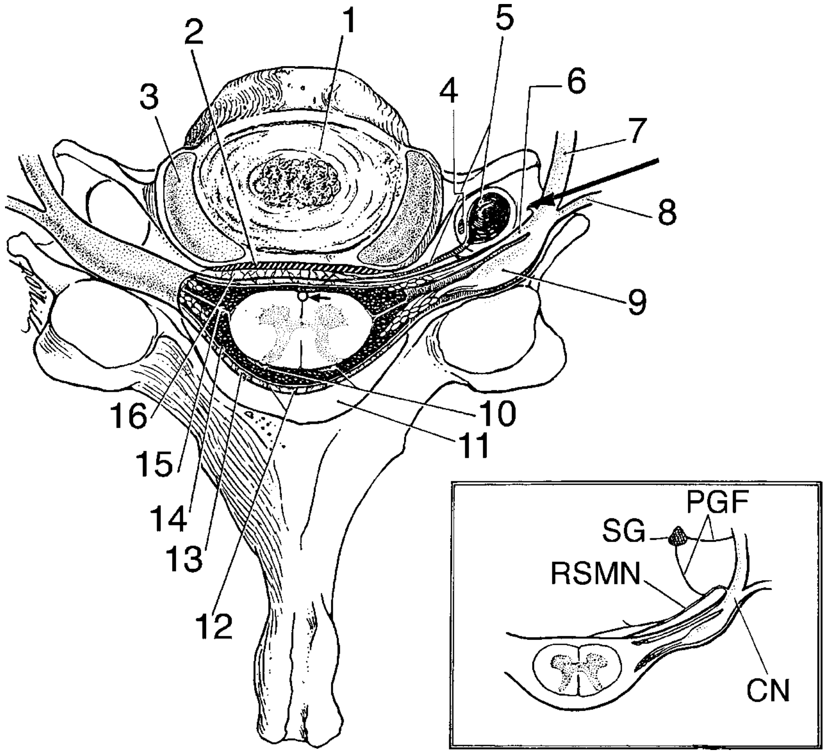


Figure v A schematic diagram of a typical cervical vertebra showing basic cervical vertebral anatomy and part of the neuroanatomy within the spinal and intervertebral foramen canals. The long arrow points to the origin of the recurrent spinal meningeal nerve and the insert shows this nerve in detail. The short arrow points to the anterior spinal artery. The diagram does not show the peripheral innervation of the intervertebral disc or of the zygapophysial (facet) joints. 1 = intervertebral disc; 2 = posterior longitudinal ligament; 3 = uncovertebral joint; 4 = deep sympathetic chain; 5 = vertebral artery and spinal branch; 6 = anterior nerve root; 7 = anterior primary ramus; 8 = posterior primary ramus; 9 = posterior nerve root and ganglion of C5; 10 = posterior spinal arteries; 11 = spinal canal; 12 = subdural space; 13 = dura; 14 = arachnoid; 15 = dentate ligament; 16 = epidural space; CN = cervical nerve; RSMN = recurrent spinal meningeal nerve; SG = sympathetic ganglion; PGF = post ganglionic fibres. (Modified and reproduced with permission from: Jackson R 1977 The cervical syndrome, 4th edn. Charles C Thomas, Springfield.)

(TFLs) (Amonoo-Kuofi et al 1988, Giles 1992a, Cramer et al 2002) that reduce the space available for the spinal nerve root within the intervertebral foramen (Min et al 2005) and thus may play a role in giving rise to severe pain and paraesthesiae along the distribution of a nerve due to direct mechanical pressure on the neural complex (Amonoo-Kuofi & El-Badawi 1997). TFLs can successfully be imaged with low-field-strength MRI; if a trained radiologist identifies a TFL, there is an 87% chance that one is present and, if a trained radiologist does not identify a TFL in an intervertebral foramen, there remains a 51% chance that one is present (Cramer et al 2002).

Another structure to be considered, and alluded to above, is that of spinal arteriovenous malformations (AVMs) i.e. dural arteriovenous fistulas, intradural arteriovenous malformations, and extradural venous varices (Criscuolo & Rothbart 1992, Mascalchi et al 2001) that, although rare, may occur in any region of the spine (Royston 2007) including the sacrum (Schaat et al 2002). The spinal level of the arteriovenous malformation will determine the symptoms in a particular patient. Symptoms associated with AVMs include pain, sensory abnormalities, sphincter disturbances and paresis; pain may be present in various forms and may take the form of low back pain or neurogenic claudication or, if subarachnoid haemorrhage occurs, then severe headaches, photophobia, and meningism may be present (Rothbart et al 1997). A spinal dural arteriovenous fistula, where a direct connection between the radicular branch of a radicular artery and the corresponding radicular vein at the dorsal root sleeve, results in signs of a progressive myelopathy (van der Meulen et al 1999, Jellema et al 2007).

MRI of the spinal cord plays a very important role in the diagnosis of AVMs. For example, high T2 signal MRI of the spinal cord is the most sensitive imaging finding in spinal dural arteriovenous fistula (Gilbertson et al 1995).

Because of the lack of pathognomonic symptoms and signs associated with spinal arteriovenous malformations, the differential diagnosis is extremely important because the associated progressive myelopathy may be confused with other, more common, neurological conditions (Rothbart et al 1997). For a full appreciation of the various types of AVMs, their symptoms, signs and a differential diagnosis work-up, see Rothbart et al (1997). Essentially, neuroradiological studies should demonstrate the presence and precise location of the

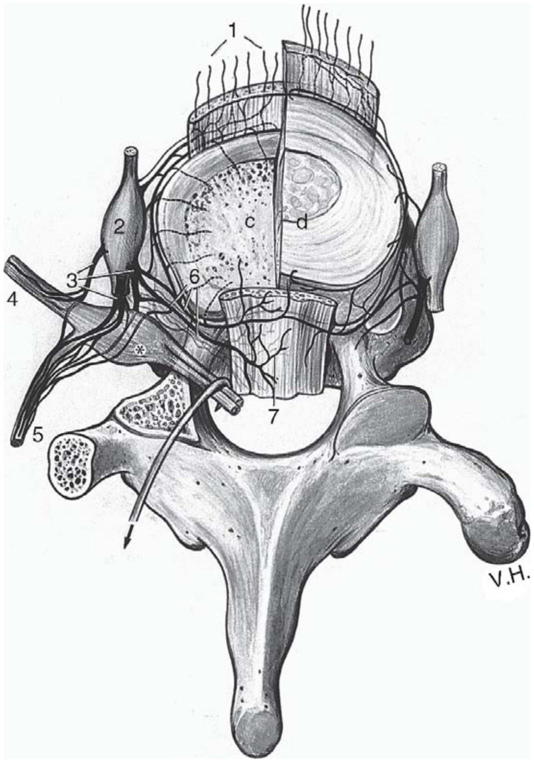


Figure vi A schematic diagram showing basic thoracic vertebral anatomy and the nerve supply of the thoracic ventral compartment at the level of the vertebral body (c) and at the level of the intervertebral disc (d). The ventral and dorsal roots of the spinal nerve are retracted dorsally (arrow). Bundles of nerve fibres originating from rami communicantes (3) pass cranial and caudal to the spinal nerve and the dorsal root ganglion (G) towards the dorsal ramus of the spinal nerve (5). Large and small sinuvertebral nerves (6) derive from the rami communicantes. 1 = anterior longitudinal ligament nerve plexus; 2 = paraspinal sympathetic ganglion; 3 = rami communicantes; 4 = ventral ramus of the spinal nerve; 5 = dorsal ramus of the spinal nerve; 6 = sinuvertebral nerves; 7 = posterior longitudinal ligament nerve plexus. The diagram does not show the innervation of the intervertebral disc or the zygapophysial (facet) joints and other structures. (Reproduced with permission from Groen G J et al 1990 Nerves and nerve plexuses of the human vertebral column. Am J Anatomy 188(3): 282–296, Wiley-Liss, Inc., a subsidiary of John Wiley & Sons, Inc.)

vascular nidus of the AVM, define the angioarchitecture, determine the operability of the malformation, and aid in the safe obliteration of the lesion by defining the vascular anatomy of the malformation and the normal spinal cord (Rothbart et al 1997). According to Xia et al (2007), intraoperative angiography for AVMs has been found to result in favourable clinical results by ensuring safe and accurate occlusion of the fistula.

From the above, it is clear that clinicians need to be familiar with both normal and abnormal anatomy in order for them to think laterally when confronted with challenging spinal pain syndromes.

IMAGING

Routine radiographs of the spine, and when indicated by the history and symptoms, the chest, should be taken to establish a baseline and to rule out metabolic, inflammatory, and malignant conditions (Keim & Kirkaldy-Willis 1987), bearing in mind the limitations of plain X-ray examinations. As long as proper coning of the X-ray beam is used in conjunction with high speed screens and films, and appropriate filtration of the X-ray beam, there is minimal X-radiation to the patient while obtaining maximum quality X-ray films. These radiographs should be taken in the erect posture, whenever possible, using carefully standardized procedures; for example, to accurately determine whether possibly significant leg length inequality is present with corresponding pelvic obliquity causing postural (compensatory) scoliosis in the spine (Giles & Taylor 1981, Giles 1984, 1989). In the cervical spine, functional flexion and extension views may show instability; sagittal plane displacement between two adjacent cervical vertebrae of more than 3.5 mm, or relative sagittal plane angulation greater than 11°, is considered to represent cervical segmental instability (White et al 1975) – that is, horizontal or angular instability (Dai 1998).

Bogduk's (1999) 'modified criteria for the use of plain films in low back pain' which are based on the work of Deyo & Diehl (1986) are of concern when viewed in the light of some of the following cases from over 37 years experience in the diagnosis of spinal pain syndromes. Bogduk (1999) states 'plain films may be used as a screening test for "red flag" conditions if a patient presents with any of the following features': history of cancer, significant trauma, weight loss, temperature >37.6 °C, risk factors for infection, neurological deficit, minor trauma in patients (over 50 years of age, known to be osteoporotic or taking corticosteroids), and no improvement over a 1 month period. Clearly, it is better for a patient to undergo plain film radiography when indicated by the history (unless there is a contraindication such as pregnancy) at the onset of symptoms, rather than to risk misdiagnosis and mismanagement, both of which would be disadvantageous to patients. This is particularly important when treatment by spinal manipulation is considered, as the application of mechanical forces to a spine that may have degenerative changes, or overt pathological changes, could be dangerous. In my opinion, not looking at the spine and pelvis in all cases prior to using mechanical treatment may well explain the occurrence of some adverse events, even though most authors have reported a low rate of adverse events associated with spinal manipulation.

Further imaging procedures may be necessary. These include (i) magnetic resonance imaging, which can provide very good detail of soft tissue structures in and about the spinal column without the need of radiation or of contrast, (ii) CT scans, which are particularly good at showing bony structures and are useful for some neural problems,

(iii) myelography or post-myelography CT scans for demonstrating lesions of the spinal cord and canal such as neoplasm and other space occupying lesions, for example disc herniation, (iv) bone scans when tumour, infection or small fracture(s) are suspected, and (v) discography when indicated, to show tears in the intervertebral disc with internal disc disruption. The usefulness of a positron emission tomography (PET) scan, used heavily in clinical oncology, should not be underestimated. When invasive imaging is being considered, the possible complications of such a procedure should always be considered.

Unfortunately, all the preceding procedures have some limitations, for example plain film radiographs will not show an osseous erosion until approximately 40% decrease in bone density has occurred (Michel et al 1990, Perry 1995), and Schellhas et al (1996) and Osti & Fraser (1992) found that discography is more accurate than MRI for the detection of annular pathology in the cervical and lumbar spines respectively. Therefore, these limitations show, as stated above, that imaging procedures may only give a 'shadow of the truth'; this important fact should be remembered. This is particularly true when a patient's physical examination and imaging studies are not remarkable and do not pinpoint the cause of symptoms, as imaging cannot show all tissue changes (Giles & Crawford 1997a). The limitations of present diagnostic imaging procedures (Finch 2006) in not being able to show all soft tissues are an unfortunate but obvious fact. The following comments are of interest with respect to some limitations of imaging procedures.

With respect to the lumbar spine, and with the same principle applying to the cervical and thoracic regions, Vernon-Roberts (1980) wrote: 'It may be a minor consolation to clinicians and others who have to deal with the problem of low back pain to know that even clinically and radiologically 'normal' spines can have pathological changes which, until proved otherwise, could be a cause of much stress to both doctor and patient'.

According to Osti & Fraser (1992), a normal MRI does not exclude significant changes in the peripheral structure of the intervertebral disc that can produce spinal pain. Furthermore, Schellhas et al (1996) demonstrated that 'significant cervical disc annular tears often escape magnetic resonance imaging detection, and magnetic resonance imaging cannot reliably identify the source(s) of cervical discogenic pain'. The same principle is likely to apply to all spinal disc levels.

Several MRI studies have shown that 20–76% of asymptomatic adults exhibit abnormalities of lumbar discs (Boden et al 1990a; Buirski & Silberstein 1993, Jensen et al 1994, Deyo 1994, Boos et al 1995, Jarvik et al 2001) in other words, 24–80% of *symptomatic* adults exhibit abnormalities of lumbar discs on MRI. Kleinstuk et al's (2006) lumbar spine MRI study showed that *symptomatic* adults with chronic, non-specific low back pain appear to have an overall higher prevalence of structural abnormalities than previously reported for *asymptomatic* individuals. Haldeman et al (2002) agree, stating that even the most severe degenerative changes can occur in the absence of symptomatology, but back pain is more common in individuals who demonstrate these degenerative changes. The specificity difficulties encountered with MRI studies to date may relate to the fact that recumbent MRI technology was used; new technology allowing for comparisons between upright, weight-bearing, dynamic, positional MRI and traditional supine MRI has shown that there is a very significant difference between the pathology visualized between the two MRI procedures; therefore, the diagnostic information that can be derived from routine supine MRI studies is extremely limited (Jinkins JR, personal communication, 2007).

It should be noted that another limitation of MR imaging is the resolving power (i.e. ability to distinguish small or closely adjacent structures) of the MRI machine; this has to be taken into account when considering what cannot be seen. I am advised that the latest MRI scanners have a resolving power of 100 to 250 microns i.e. 1/10th to 1/4 of a millimetre, so any lesion smaller than this would not be seen. For this reason, it is not uncommon for imaging reports to fail to indicate injuries that may be the cause of pain.

It is of interest to note that cervical spine investigations with plain radiography, myelography, and computed tomography have shown that degenerative disease frequently occurs in the absence of clinical symptoms (Boden et al 1990b) while Gore (1999) stated that degenerative changes of the cervical spine, as seen on plain roentgenograms and more sophisticated studies, are common in both symptomatic and asymptomatic patients.

Nonetheless, various studies have shown a significant association between some structural abnormalities and the presence (Parkkola et al 1993, van Tulder et al 1997, Paajanen et al 1997, Luoma et al 2000), frequency (Videman et al 2003, Videman & Nurminen 2004) or severity (Videman et al 2003, Peterson et al 2000) of low back pain.

Using flexible fibrescopes (external diameter of 0.6–1.5 mm) Tobita et al (2003) stated, with respect to the entire spine: 'Although the diagnosis of spinal disease has been greatly improved by computed tomography (CT) and magnetic resonance imaging (MRI), there are still many conditions that are difficult to diagnose by these means as pathological changes were seen by fibrescopic examinations in patients in whom no abnormal changes were found by MRI or CT'.

The abovementioned findings raise questions about the morphology-based understanding of pain pathogenesis in patients with disc abnormalities (Boos et al 2000). Furthermore, Karppinen et al (2001) found that MRI scans from 180 patients with unilateral sciatic pain suggested that a discogenic pain mechanism other than nerve root entrapment generates the subjective symptoms among sciatic patients.

A further difficulty is that the nomenclature and classification of lumbar disc pathology is not standardized

(Fardon & Milette 2001), although Pfirrmann et al (2001) have suggested a method for grading disc degeneration on T2-weighted MRI.

When nerve root dysfunction is suspected, electromyography (EMG) and nerve root conduction studies can be helpful (Hoppenfeld 1977).

It is important to note the following comments regarding imaging shown in this text:

- Most plain film anteroposterior radiographic images of the spine and or pelvis are printed as if the clinician were looking at the patient's back; i.e. a marker showing 'R' indicates the patient's right side.
- Spinal axial CT scans are viewed, as usual, from 'below'; i.e. remember that the clinician 'looks up' the patient's spinal canal with the patient supine, so the patient's right side is marked 'R' on the left side of the axial CT scan figure(s).
- For spinal axial MRI scans, the same principle as for CT axial scans is applied; i.e. the patient's right side is on the left side of the figure(s).
- MRI T1-weighted images produce essentially a *fat image* in which structures containing fat (bone marrow, subcutaneous fat) appear bright, while structures containing water (oedema, neoplasm, inflammation, cerebrospinal fluid, sclerosis, large amounts of iron) appear dark (Yochum & Barry 1996).
- MRI T2-weighted images produce essentially a *water image* in which structures containing predominantly free or loosely bound water molecules (neoplasm, oedema, inflammation, healthy nucleus pulposus, cerebrospinal fluid) appear bright, while substances with tightly bound water (ligaments, menisci, tendons, calcification, sclerosis or large amounts of iron) appear dark (hypointense) (Yochum & Barry 1996).
- Patient identification details have been blacked out to maintain patient confidentiality.

LABORATORY TESTS

When bony pathology is suspected, useful laboratory tests that may be helpful in detecting bone disease include serum calcium, phosphorus, acid phosphatase, and alkaline phosphatase (particularly alkaline phosphatase isoenzyme determination by electrophoresis, which differentiates alkaline phosphatase of osteoblastic origin from alkaline phosphatase from other sources) (Brown 1975). On electrophoresis, liver isoenzyme levels usually range from 20 to 130 U/L; bone isoenzyme levels from 20 to 120 U/L; and intestinal isoenzyme levels (which occur almost exclusively in individuals with blood group B or O and are markedly elevated 8 hours after a fatty meal) from undetectable to 18 U/L (Brabson 2001). The five alkaline phosphatase isoenzymes of greatest clinical significance originate in the liver (includes kidney and bile fractions), bone (may also include bile fraction), intestine, and placenta (Brabson 2001). Evaluation of enzymes more specific to the liver, 5′-nucleotidase or γ-glutamyltranspeptidase (GGT), can differentiate hepatic from extra-hepatic sources of alkaline phosphatase (Beers et al 2006). Early inflammatory changes may be detected by an increase in C-reactive protein (CRP) and/or an increase in the erythrocyte sedimentation rate (ESR). A full blood count can be helpful, for example, in cases where there is suspicion of primary haematological disorders and for some infections (Henderson et al 1994). Immunoelectrophoresis of serum and urinary proteins may also be useful diagnostic procedures in the diagnosis of multiple myeloma (Brown 1975, Beers et al 2006). Examples of other tests that should be considered, when indicated, are: (i) latex flocculation for rheumatoid spondylitis; (ii) serum and urine amylase and lipase for chronic pancreatitis (Collins 1968, Schroeder et al 1992); and (iii) blood culture and sensitivity, and for genitourinary tract infections urine culture and sensitivity. In addition, it may be necessary to assess bone density using a dual energy X-ray absorption (DEXA) bone densitometer in osteoporotic patients. In males, prostate-specific antigen (PSA) should be considered if there is any suspicion of prostate malignancy (Kingswood & Packham 1996).

In this book it is not necessary to list every spine-related condition with its possible abnormalities in serology, haematology, urinalysis and other laboratory tests, as these have been well documented in numerous clinical diagnosis texts. In some publications, particular reference to spinal pathological conditions and related pathology tests have been summarised (Haldeman et al 1993, Henderson et al 1994).

A large range of laboratory evaluations are important when the clinician suspects metabolic disturbance, malignancy, infection or one of the arthritides such as ankylosing spondylitis or rheumatoid arthritis. Nonetheless, it should be noted that various tests have different levels of *accuracy*, as calculated from their *sensitivity* (proportion of individuals with the condition whose tests are positive) and *specificity* (proportion of individuals without the condition whose tests are negative (Bloch 1987, Nachemson 1992, Henderson et al 1994)).

CONDITIONS ASSOCIATED WITH SPINAL PAIN SYNDROMES

A summary of the chief conditions causing spinal tenderness is shown in Table i. In addition, it is necessary to list some possible causes of non-specific spinal pain of mechanical origin, with or without radicular pain, as briefly summarized in Table ii, which provides a summary of some literature references over the years in order to give a historical background to the complex issue of non-specific spinal pain of mechanical origin. As Waddell (2004) stated, most back pain is 'ordinary backache'. This often is referred to as 'mechanical', 'non-specific' or 'idiopathic' spinal pain. In a large number of these cases, it may not

Table i Summary of chief conditions causing spinal tenderness

1. Diseases of the overlying skin and subcutaneous tissue. These are usually clinically obvious and include potentially serious conditions such as melanoma
2. Diseases of the vertebral column
   - Inflammatory
     - Pott's disease
     - Staphylococcal spondylitis
     - Typhoid spine
     - Spondylitis ankylopoietica (Spondylitis Ossificans Ligamentosa/ankylosing spondylitis)
     - Actinomycosis
     - Hydatid cyst
     - Paget's disease
   - Degenerative
     - Spondylosis
     - Osteochondritis (rare)
     - Nucleus pulposus herniation and other soft tissue injuries
   - Neoplastic
     - Primary tumour
     - Secondary deposit
     - Myelomatosis
     - Leukaemic deposits
   - Traumatic
     - Fracture
     - Dislocation
     - Nucleus pulposus herniation
   - Erosion by aortic aneurysm
3. Diseases of the spinal cord and meninges
   - Metastatic epidural abscess or tumour
   - Meningioma
   - Neurofibroma
   - Herpes zoster
   - Meningitis serosa circumscripta
   - Tumour of the spinal cord
   - Syringomyelia
   - Arachnoid calcifications (Wijdicks and Williams 2007)
4. Hysteria and malingering: compensation neurosis
5. Metabolic disorders: osteoporosis, osteomalacia, hyperparathyroidism

Modified from Mackenzie I 1985 Spine, tenderness of. In: Hart FD (ed) French's index of differential diagnosis, 12th edn. Butterworth & Co. Ltd, p 788.

Table ii Some possible causes of non-specific spinal pain of mechanical origin with or without radicular pain

Nerve root conditions
- Adhesions between dural sleeves and (a) the joint capsule with nerve root fibrosis (Sunderland 1968, Jackson 1977, Wilkinson 1986) and (b) intervertebral disc herniation (Wilkinson 1986).
- Intervertebral disc degeneration and fragmentation (Schiotz & Cyriax 1975), or nucleus pulposus extrusion (Mixter & Ayer 1935) causing nerve root compression (Kobayashi et al 2005), or nerve root 'chemical radiculitis' (Marshall & Trethewie 1973).

Zygapophysial joint conditions
- Joint derangement (subluxation) due to ligamentous and capsular instability (Hadley 1964, Cailliet 1968, Jackson 1977, Macnab 1977, van Norel & Verhagen 1996).
- Joint capsule tension with encroachment upon the intervertebral foramen lumen (Jackson 1977).
- Joint degenerative changes, e.g. 'meniscal' incarceration (Schmorl & Junghanns 1971), traumatic synovitis due to 'pinching' of synovial folds (Giles 1986, Giles 1987, Giles & Harvey 1987, Giles & Taylor 1987a & b), synovial fold tractioning against the pain-sensitive joint capsule (Hadley 1964), and osteoarthrosis (Jackson 1977).
- Joint effusion with capsular distension which may (a) exert pressure on a nerve root (Jackson 1977), (b) cause capsular pain (Jackson 1966), or (c) cause nerve root pain by direct diffusion (Haldeman 1977).
- Joint capsule adhesions (Jackson 1977, Farfan 1980, Giles 1989).

Intervertebral disc conditions
- Disc protrusion into the spinal and intervertebral canals.
- Disc/dural adhesions (Parke & Watanabe 1990)
- Spondylosis (Young 1967, Jackson 1977).

Miscellaneous conditions
- Spinal and intervertebral canal (foramen) stenosis (Young 1967, Jackson 1977, Epstein & Epstein 1987, Rauschning 1992).
- Intervertebral canal (foramen) venous stasis (Giles 1973, Sunderland 1975).
- Myofascial genesis of pain (trigger areas) (Travell & Rinzler 1952, Bonica 1957, Simons & Travell 1983).
- Baastrup's syndrome (Reinhardt 1951, Bland 1987)
- Osseous spinal anomalies, e.g. bilateral cervical ribs, block vertebra (Jackson 1977).
- Idiopathic scoliosis (Ramirez al 1997)

be possible to identify the precise pain generator. However, an orthopaedic surgeon well versed in spinal pain syndromes, Emeritus Professor Ruth Jackson, stated that the word idiopathic is used by defeatists to signify 'I don't know the cause' and that, if one searches diligently for the cause of any painful or abnormal condition, the answer can usually be found (Jackson 1977).

As there are many putative causes of such spinal pain, some gross anatomical and histopathology examples are provided from human postmortem material to illustrate some possible causes of such pain (Figures vii.1–32). Most examples are from the lumbosacral spine as low back pain is experienced by the majority of spinal pain syndrome patients.

To prepare the large (7 × 8 cm) histopathology sections from osteoligamentous blocks of human cadaver spinal material took up to seven months, for each specimen, using a modification of the technique designed for smaller blocks by Giles & Taylor (1983).

SOME ILLUSTRATIONS SHOWING EXAMPLES OF POSSIBLE CAUSES OF NON-SPECIFIC SPINAL PAIN SYNDROMES OF MECHANICAL ORIGIN

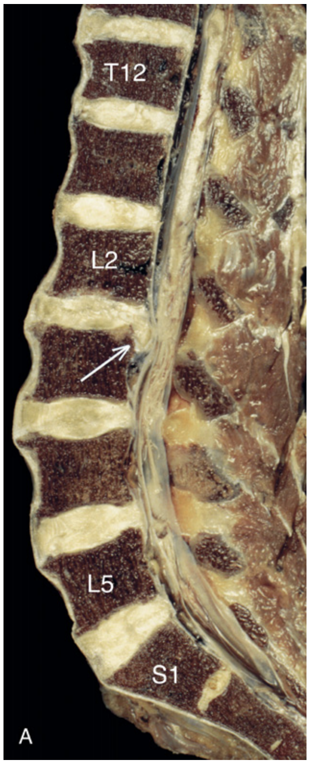
Lumbar spine

Intervertebral disc broad-based posterior protrusion (Fig. vii.1A and B)

Clinical relevance

This may cause low back pain and radicular symptoms due to:

1. Pressure upon the pain sensitive anterior surface of the dural tube (Summers et al 2005).
2. Pressure upon the recurrent meningeal nerve between the protrusion and the dural tube.
3. Pressure upon blood vessels causing ischaemia (Giles 1973, Sunderland 1975) and vascular damage (Hoyland et al 1989, Jayson 1997) between the protrusion and the dural tube.
4. Pressure upon a nerve root (Mixter & Barr 1934, Crock 1976, Summers et al 2005).



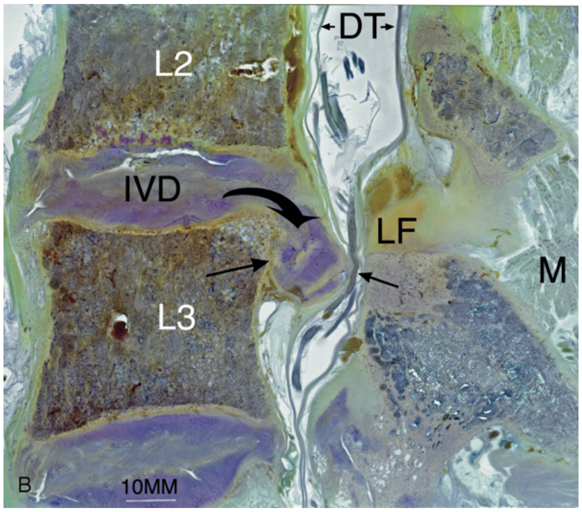


Figure vii.1A and B (A) Part of a spinal column extending from T11 to S2 is shown bisected in the sagittal plane. The intervertebral discs appear to be relatively normal for a 62-year-old male apart from the large posterior midline herniation of the second lumbar (L2) intervertebral disc (arrow) that causes compression of the dural tube and cauda equina (C), resulting in some stenosis of the spinal canal at this level. A histopathology section showing the intervertebral disc protrusion is presented in Fig. vii.1B. See also colour plate section. (B) A 200-micron thick histopathology parasagittal section from a 62-year-old male cadaveric spine. The large arrow shows protruded disc material compressing the dural tube (DT) and a nerve root (small black arrow) between the protrusion and the ligamentum flavum (LF) posteriorly. As the section is 200 microns thick (1/5th of a millimetre thick) only one nerve root is seen being compressed. However, in a broad based disc protrusion a number of nerve roots would be compressed, thus causing the cauda equina syndrome. IVD = intervertebral disc. M = muscles. (Ehrlich's haematoxylin and light green counterstain.).

Intervertebral disc central posterior protrusion (Fig. vii.2)

Clinical relevance

This is likely to cause low back pain without radicular symptoms.

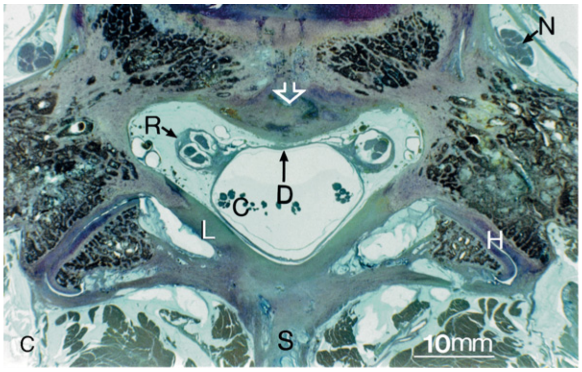


Figure vii.2 A 200-micron thick histopathology section from a cadaver with a similar, but less extensive, central posterior disc protrusion; this is to orientate the reader to the various anatomical structures. R = nerve root sleeve budding off from the dural tube (D) that contains small nerve roots arising from the cauda equina (C). H = hyaline articular cartilage on the zygapophysial joint facet surfaces. L = ligamentum flavum; N = spinal nerve. Open white arrow = intervertebral disc protrusion that is central and just indents the anterior surface of the dural tube. (Ehrlich's haematoxylin and light green counterstain.)

Intervertebral disc protrusion anteriorly and posteriorly with internal disc disruption (Fig. vii.3A and B)

Clinical relevance

Intervertebral disc pressure upon the pain sensitive anterior surface of the dural tube, the intervening blood vessels and the recurrent meningeal nerves can cause low back pain. In addition, internal disc disruption can cause low back pain (Cooke & Lutz 2000). A gross anatomical example of internal disc disruption is shown in Fig. vii.3A and a histopathology example (Fig. vii.3B) shows an intervertebral disc with internal disc disruption and some disruption of the outer annulus fibrosus fibres.

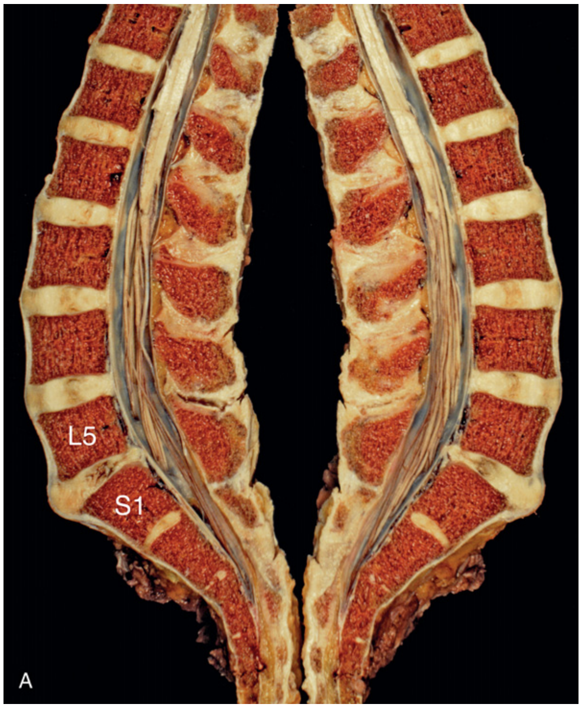


Figure vii.3A Anatomical specimen of a sagittally sectioned spine. Note the approximation (black arrow) of the elongated fifth lumbar spinous process and the adjacent sacral spinous tubercle, in spite of the normal lumbar lordosis in this 70-year-old female. The L5 intervertebral disc shows degenerative changes which include (i) anterior 'bulging' of the disc, and (ii) posterior protrusion of the disrupted nucleus pulposus which has elevated the posterior longitudinal ligament above and below this disc level. (Reproduced with permission from Giles L G F Miscellaneous pathological and developmental (anomalous) conditions. In: Giles L G F, Singer K P (eds) 1997 Clinical anatomy and management of low back pain. Butterworth-Heinemann, Oxford, p 196–216.)

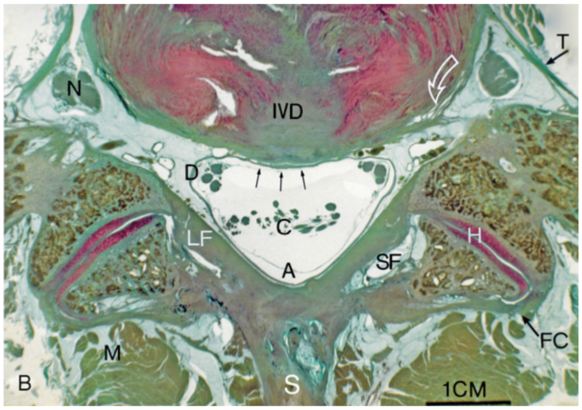


Figure vii.3B A 200-micron thick horizontal histopathology section from a 51-year-old female postmortem specimen. Note the internal disc disruption within the intervertebral disc (IVD) with some radial tears seen as white spaces and there is some disruption of the outer annulus fibrosus fibres (curved white arrow). There is some central posterior bulging of the disc which effaces (small black arrows) the anterior part of the dural tube (D) which contains the cauda equina (C) that is surrounded by cerebrospinal fluid and the arachnoid membrane (A). The neural structures (N) passing through the lateral part of the intervertebral foramen are in close proximity to transforaminal ligaments (T). S = remains of spinous process. SF = synovial fold adjacent to the ligamentum flavum (LF). H = hyaline articular cartilage surfaces on the facets of the zygapophysial joints. FC = fibrous joint capsule posterolaterally. M = multifidus muscle. (Ehrlich's haematoxylin and light green counterstain.)

Posterior migration and leakage of nuclear material (Fig. vii.4A and B)

Clinical relevance

This can cause pain due to chemical irritation of adjacent neural structures, i.e. chemical radiculopathy (Marshall & Trethewie 1973, Marshall et al 1977, Goupille et al 1998, 2006). When a disc suffers an annular tear causing nuclear material to leak out, that material can cause inflammation of the adjacent nerve roots resulting in discogenic back pain and sciatica (Grönblad & Virri 1997).

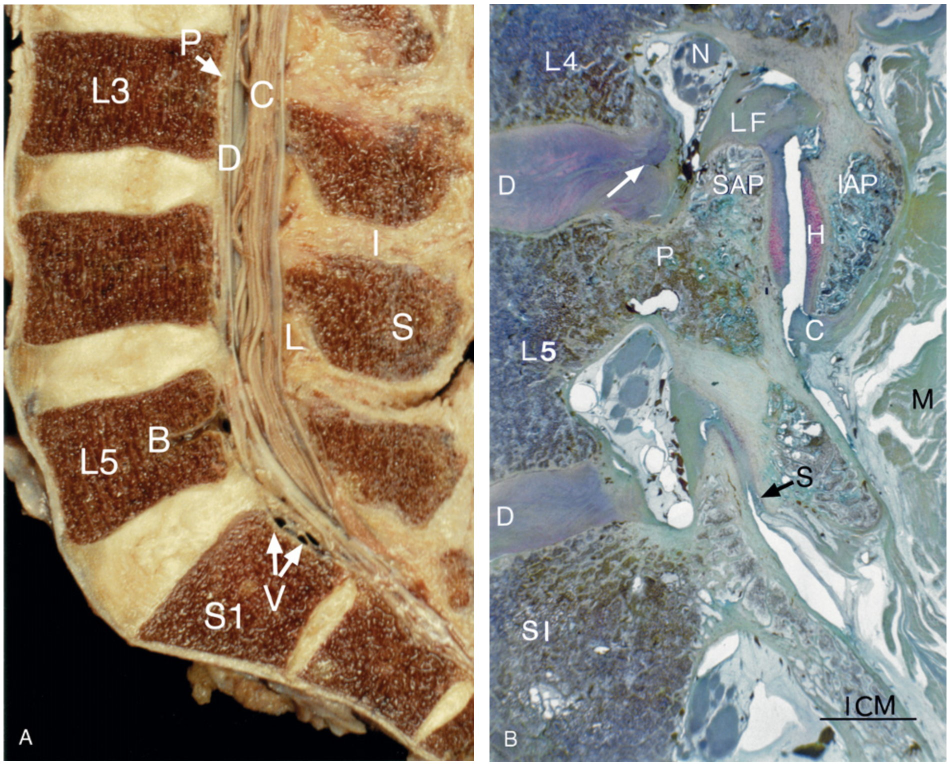


Figure vii.4

(A) Part of a spinal column extending from L3 to S2 is shown bisected in the sagittal plane. The intervertebral discs appear to be relatively normal for a 78-year-old male However, a light microscopy section showed a tear within the L4–5 intervertebral disc (Fig. vii.2B). B = basivertebral vein; C = cauda equina; D = dural tube; I = interspinous ligament; L = ligamentum flavum; P = posterior longitudinal ligament; S = spinous process; V = blood vessels within the spinal canal. (B) A 200-micron thick histopathology parasagittal section showing a tear within the L4–5 intervertebral disc with retrograde movement of nuclear material in this disc (white arrow). C = capsule (inferiorly) of the L4–5 zygapophysial joint; D = intervertebral disc; H = hyaline articular cartilage on the facet of the inferior articular process of the L4 vertebra; IAP = inferior articular process of the L4 vertebra; L4 = part of the fourth lumbar vertebral body; L5 = part of the fifth lumbar vertebral body; LF = ligamentum flavum; M = muscle; N = neural structures within the nerve root sheath located in the pear-shaped intervertebral canal; P = pedicle of the L5 vertebra; S = synovial fold projecting into the inferior recess of the zygapophysial joint at the L5–S1 level; SAP = superior articular process of the L5 vertebra. (Ehrlich's haematoxylin and light green counterstain)

Multifactorial degenerative changes may be present at one level of the spine (Fig. vii.5A, B and C)

Clinical relevance

Such changes independently and collectively can cause low back pain, with or without radicular symptoms.

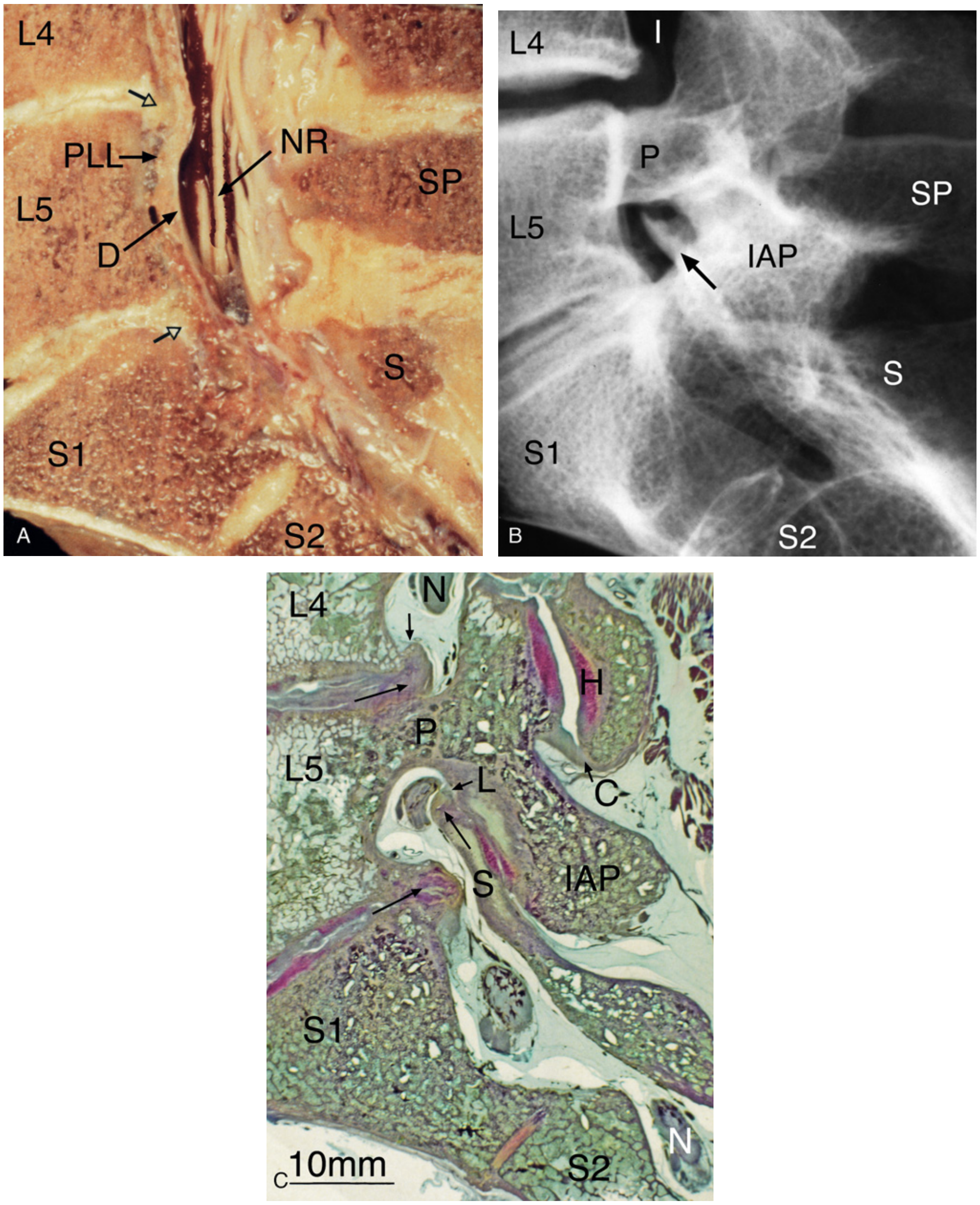


Figure vii.5 (A) Part of a spinal column extending from L4 to S2 from a 59-year-old woman is shown bisected in the sagittal plane. The L4–5 and L5–S1 intervertebral discs show *posterior protrusions* (open headed arrows) with *loss of intervertebral disc height*. The posterior longitudinal ligament (PLL), dural tube (D), nerve roots (NR), spinous process (SP) and the median sacral crest (S) are seen. (B) Plain X-ray showing a lateral view of the block of spinal tissue seen in Fig. vii.5A. Note the *loss of intervertebral disc height space* at L4–5 and at L5–S1 levels. P = pedicle of L5 vertebra; SP = spinous process; S = sacral superior articular process; I = intervertebral foramen; IAP = inferior articular process of L5 vertebra. The superior articular process of S1 (arrow) *projects into the L5–S1 foramen*. (C) A 200-micron thick histological section cut in the parasagittal plane from the specimen shown in Fig. vii.5A that shows parts of the fourth lumbar (L4) to second sacral (S2) spinal segments. The large neural structures, i.e. the nerve roots and ganglion (N), are shown within the L4–L5 and L5–S1 intervertebral canals and within the sacral canal. Posterolateral intervertebral disc *protrusions* (arrows) with *thinning of* the L4–5 and L5–S1 intervertebral discs is demonstrated; this has resulted in approximation of the vertebral bodies and *imbrication* (*subluxation*) of the opposing facets of the zygapophysial joints with their hyaline articular cartilages (H). The zygapophysial joint imbrication appears to have caused some *tractioning of the L4–L5 joint capsule* (C) inferiorly and '*buckling*' of the ligamentum flavum at the L5–S1 level. This 'buckling', together with early osteophytosis of the superior articular process (S) of the sacrum, has caused *deformation and compression* of the adjacent neural structure. There is *subchondral sclerosis* of each facet at the L5–S1 level in particular with *osteoarthrotic* changes. Note the considerable blood supply to the neural structures particularly at the L5, S1 and S2 levels. IAP = inferior articular process of L5 vertebra; L = ligamentum flavum; M = muscle; P = pedicle of L5 vertebra. (Ehrlich's haematoxylin and light green counterstain.)

Adhesion formation between a dural sleeve and an intervertebral disc protrusion (Fig. vii.6)

Clinical relevance

Adhesions between dural sleeves and an intervertebral disc protrusion are a source of pain (Wilkinson 1986).

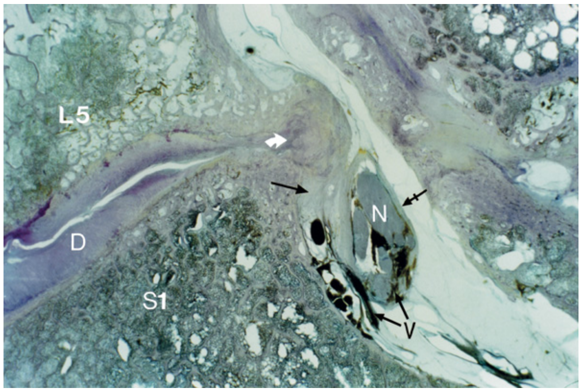


Figure vii.6 A 200-micron thick histopathology section, cut in the parasagittal plane through the lumbosacral intervertebral disc of a 59-year-old cadaver, showing a disc protrusion (small curved white arrow) with perineural adhesions (arrow) between the protrusion and the adjacent dural sleeve (tailed arrow) containing neural structures (N). D = intervertebral disc; L5 = fifth lumbar vertebral body; S1 = first sacral segment. Note the extensive vascularity (V) posterior to the sacrum and within and around the neural structures. (Ehrlich's haematoxylin and light green counterstain.)

Intra-articular synovial folds

The following histopathology figures (Figs. vii.7 to vii.9 and vii.14 to vii.16) show various forms of synovial folds within the zygapophysial joints.

Clinical relevance

Synovial fold nipping or pinching between bony surfaces can cause traumatic synovitis (Giles 1986, Giles 1987, Giles & Taylor 1987a) with pain as synovial folds have nociceptive nerves (Giles & Harvey 1987; Grönblad et al 1991) (Fig. vii.9E).

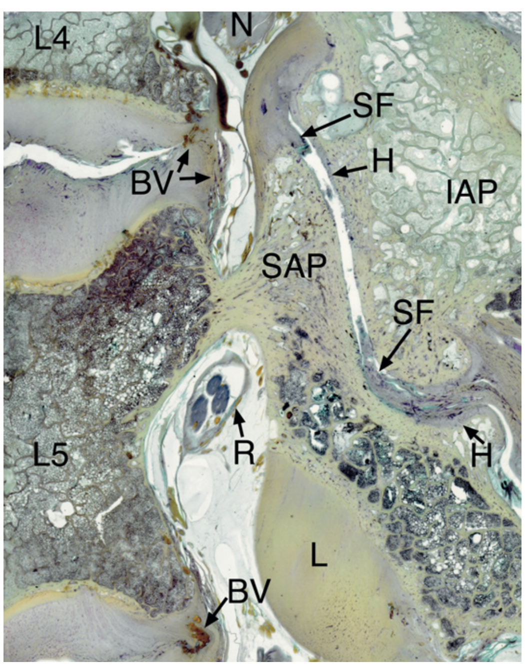


Figure vii.7 A 200-micron thick histopatholgy section cut in the parasagittal plane through the right L4–5 and L5–S1 intervertebral foramina levels in a 62-year-old male. Note the synovial folds (SF) in the superior and inferior poles of the L4–5 zygapophysial joint that is formed by the inferior articular process (IAP) of the L4 vertebra and the superior articular process (SAP) of the L5 vertebra. H = hyaline articular cartilage (osteoarthrotic) on the facet surfaces (arrow); L = ligamentum flavum; L4 = fourth lumbar vertebral body; L5 = fifth lumbar vertebral body; N = neural structure; NV = neurovascular structures in the intervertebral foramen; R = root sleeve containing neural and vascular structures. There are small blood vessels (BV) in the posterior parts of the intervertebral discs. (Ehrlich's haematoxylin and light green counterstain.)

A sagittally cut histological view of an intra-articular synovial fold (Fig. vii.8).

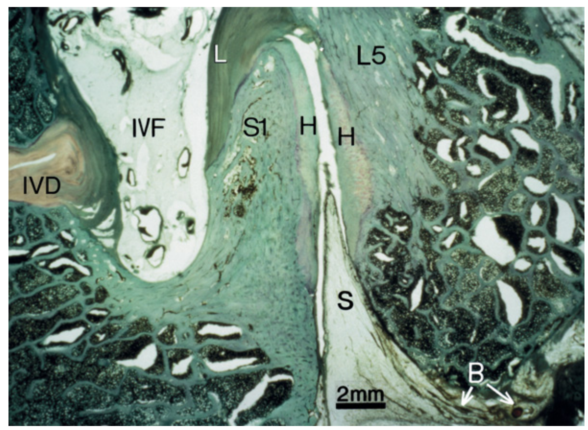


Figure vii.8 A 150-micron thick histopathology section cut in the parasagittal plane through the lumbosacral zygapophysial joint and intervertebral foramen. Note the large highly vascular intra-articular synovial fold (S) with a small fibrotic tip within the inferior recess of the lumbosacral zygapophysial joint from a 56-year-old male cadaver. B = blood vessels; H = hyaline articular cartilage on the facet surfaces; IVD = intervertebral disc of the lumbosacral joint; IVF = intervertebral foramen; L = ligamentum flavum; L5 = inferior articular process of the fifth lumbar vertebra; S = intra-articular synovial fold; S1 = superior articular process of the sacrum (Ehrlich's haematoxylin and light green counterstain.) (Reproduced with permission from Giles L G F 1988 Human zygapophyseal joint inferior recess synovial folds; a light microscope examination. Anat Rec 220: 117-124. Copyright A R Liss, New York.)

A slightly oblique horizontally (axially) cut view of an intra-articular synovial fold (Fig. vii.9A).

High power light microscopy detail of a synovial fold (Fig. vii.9C–E).

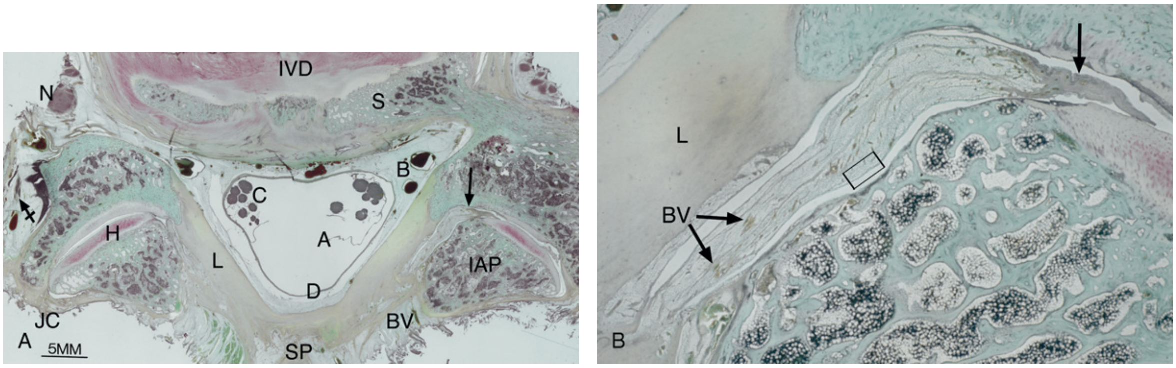


Figure vii.9A and B A 100-micron thick slightly oblique horizontal histopathology section of the lumbosacral zygapophysial joints at the level of the inferior joint recesses from a 54-year-old male. This shows a highly vascular intra-articular synovial fold (black arrow) with a fibrotic tip (see enlargement in B). A = arachnoid membrane; B = Batson's venous plexus; BV = blood vessel; C = cauda equina; D = dura mater; H = hyaline articular cartilage; IAP = inferior articular process of L5 vertebra; IVD = intervertebral disc; JC = posterolateral fibrous joint capsule; L = ligamentum flavum; N = spinal ganglion; R = right side; S = sacrum; SP = spinous process. A neurovascular bundle close to the zygapophysial joint is shown by the tailed arrow. High power light microscopy detail of a synovial fold is shown in Fig. vii.9C–E. (Ehrlich's haematoxylin stain with light green counter-stain.) (Modified and reproduced with permission from Giles L G F, Taylor J R 1982 Intra-articular synovial protrusion in the lower lumbar apophyseal joints. Bulletin of the Hospital for Joint Diseases Orthopaedic Institute 42 (2): 248–255.)

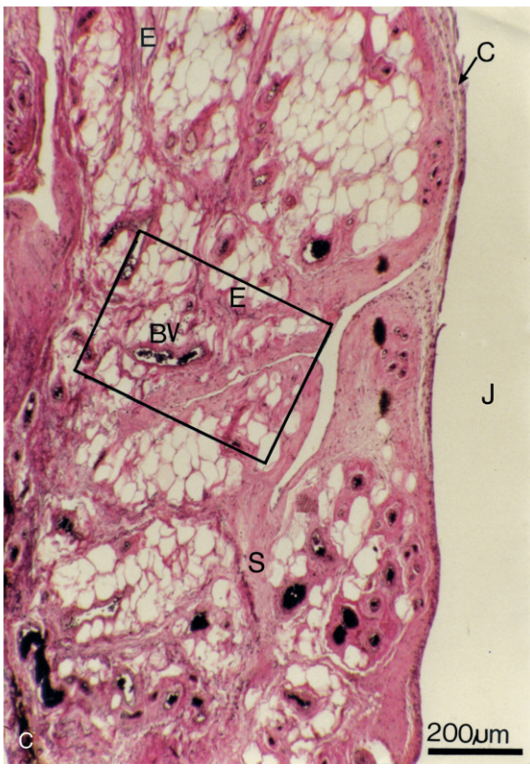


Figure vii.9C This 30-micron thick histology section shows part of a synovial fold from the lumbosacral zygapophysial joint of a 45-year-old female patient who underwent a partial facetectomy during surgery to remove a protruded intervertebral disc. Note the irregularly spaced synovial lining cells (C) in the synovial lining (intimal) layer. BV = blood vessels containing blood cells; J = joint cavity; S = interlocular fibrous septum in the subsynovial (subintimal) layer. There is a rich blood supply and the unilocular fat cells indicate that synovial folds consist of white adipose tissue in adults. The rectangle highlights an area where elastic fibres (E) run in various directions in the subsynovial tissue within interlocular fibrous septa (Fig. vii.9D). (Modified Schofield's silver impregnation and Verhoeff's haematoxylin counterstain). (Reproduced with permission from Giles L G F 1988 Human zygapophyseal joint inferior recess synovial folds; a light microscope examination. Anat Rec 220: 117-124. Copyright A.R. Liss, New York.)

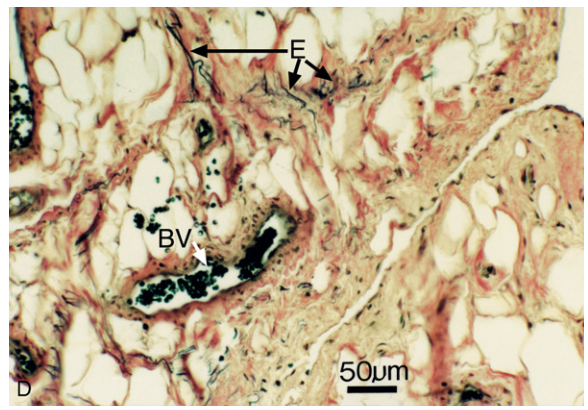


Figure vii.9D High-power magnification of the rectangle in Fig. vii.9C. Note the blood vessel (BV) and some of the numerous black stained elastic fibres (E) that are distributed throughout the synovial fold. Some of the blood vessels also show elastic fibres.

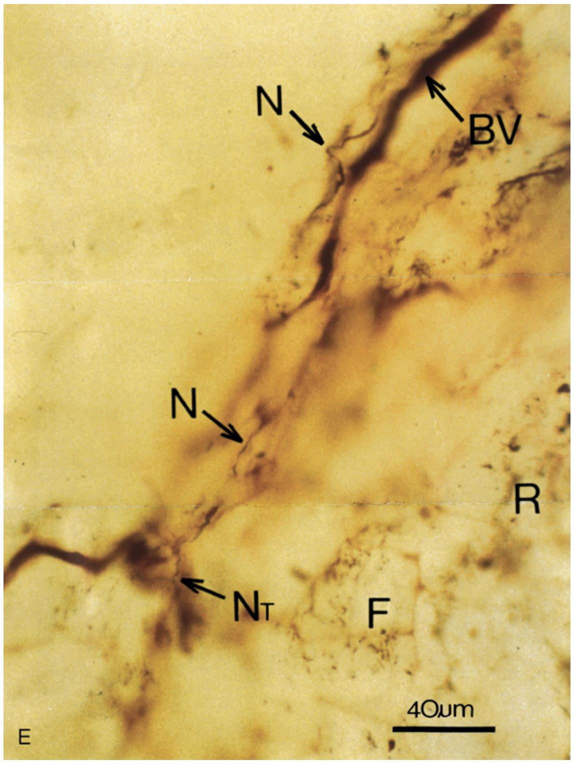


Figure vii.9E Montage of the L4–5 zygapophysial joint synovial fold from a 49-year-old male patient who underwent a partial facetectomy, showing the extremely small nerve fibre (N) that appears to terminate as a 'free ending' nerve (NT) in the synovial lining membrane. The average diameter of the nerve fibre between N and N is 1.1 µm. BV = blood vessel; F = fat cell; R = reticular fibres. (Modified Schofield's silver impregnation.) There are both paravascular and non-paravascular nerves in the synovial fold's lining membrane (Giles & Taylor 1987a, Grönblad et al 1991). The small diameter non-paravascular nerves are considered to have a putative function of nociception (Giles & Harvey 1987). (Reproduced with permission from Giles L G F 1988 Human zygapophyseal joint inferior recess synovial folds; a light microscope examination. Anat Rec 220: 117-124. Copyright A.R. Liss, New York.)

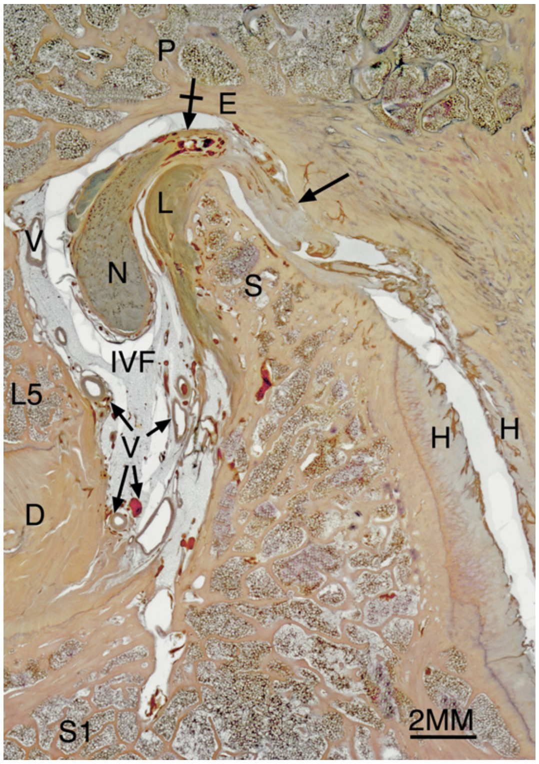


Figure vii.10 A parasagittal histopathology section of the left lumbosacral intervertebral canal foramen from an 82-year-old female showing how the neural complex (N) and the dense fibrous intra-articular synovial fold (arrow) have become attached to each other via a highly vascular connective tissue adhesion (tailed arrow). D = intervertebral disc; E = eburnation of the inferior aspect of the pedicle (P) of the L5 vertebra; H = hyaline articular cartilage (arthrotic); L = ligamentum flavum on the superior articular process of the sacrum (S). Note the numerous blood vessels (V) within the intervertebral canal (foramen) (IVF). (Ehrlich's haematoxylin and light green counterstain.) (Reproduced with permission from Giles L G F 1991 A review and description of some possible causes of low back pain of mechanical origin in homo sapiens. Proc Aust Soc Hum Biol 4: 193-212.)

Neural complex attached to a synovial fold (Fig. vii.10)
Clinical relevance

Pinching of the highly vascular connective tissue adhesion between the pedicle of L5 vertebra and the superior articular process of the sacrum can result in pain.

Pressure upon neural structures (Fig. vii.11)
Clinical relevance

Pressure upon neural structures will cause pain (Kobayashi et al 2005).

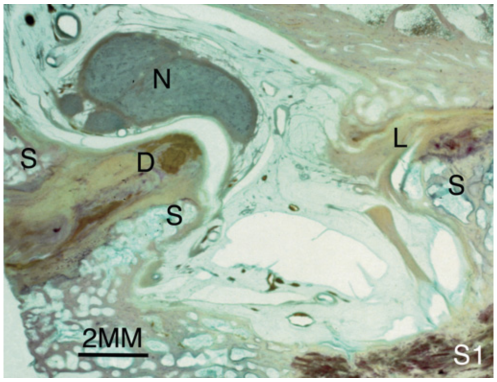


Figure vii.11 Parasagittal histopathology section across the lumbosacral intervertebral canal foramen in a 73-year-old cadaver. Note the large osteophytic spurs (S) on each side of the lumbosacral intervertebral disc protrusion (D) and how these structures have deformed the adjacent neural structures (N). The ligamentum flavum (L) is seen adjacent to an osteophytic spur (S) on the superior articular process of the first sacral segment (S1). This spur and the intervertebral disc protrusion, with adjacent bony spurs, considerably lessen the anteroposterior diameter of the central part of the intervertebral canal in this region. (Ehrlich's haematoxylin and light green counterstain.)

Transforaminal ligaments (Fig. vii.12)

Clinical relevance

Transforaminal ligaments may cause pain due to direct mechanical pressure on the neural complex (Amonoo-Kuofi et al 1988, Giles 1992a, Cramer et al 2002).

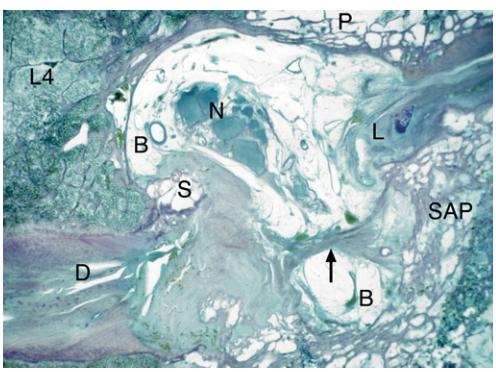


Figure vii.12 A 200-micron thick histopathology section cut in the parasagittal plain across the left L4–5 intervertebral canal of a 69-year-old male. The arrow shows a ligament traversing the lower part of the intervertebral canal, within the foramen and bisecting it. The depth of the ligament within the intervertebral canal is 4 mm. The transforaminal ligament was examined by light microscopy using thin sections stained with Richardson's stain and it was found to contain elastic fibres, myelinated nerves and blood vessels (Giles et al 1991). B = blood vessels; D = intervertebral disc; L = ligamentum flavum; L4 = fourth lumbar vertebral body; N = spinal nerve complex; P = pedicle of L4 vertebra; S = spur posterolaterally and inferiorly on the L4 vertebral body; SAP = sacral superior articular process. (Ehrlich's haematoxylin and light green counterstain.)

Blood vessels associated with intervertebral disc posterior protrusion or bulge (Figs. vii.13 and 14)

Clinical relevance

Pressure upon blood vessels due to intervertebral disc posterior protrusion or disc bulge may cause pain due to venous stasis and ischaemia (Giles 1973, Sunderland 1975, Hoyland et al 1989, Jayson 1997).

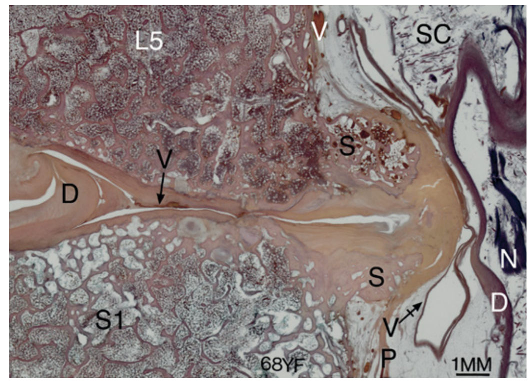


Figure vii.13 A 200-micron thick sagittal histopathology section from a 68-year old female cadaver showing a lumbosacral posterior midline intervertebral disc protrusion with large bilateral osteophytic spurs (S) projecting into the spinal canal (SC) and causing stenosis of the canal and traction and occlusion of the adjacent vascular structures (V tailed arrow). D = intervertebral disc; L5 = fifth lumbar vertebral body; N = neural structure within the dural tube; P = posterior longitudinal ligament; S1 = first sacral body; V (arrow) = small blood vessels within the degenerative intervertebral disc. (Ehrlich's haematoxylin and light green counterstain). (From Taylor J R, Giles L G F 1997 Lumbar intervertebral discs. In: Giles L G F and Singer K P (eds) Clinical anatomy and management of low back pain, Edinburgh, Butterworth-Heinemann, p 49-71.)

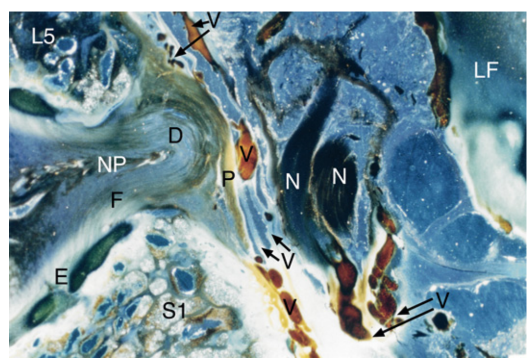


Figure vii.14 A 200-micron thick sagittal histopatholgy darkfield section from a middle-aged cadaver showing a lumbosacral posterior midline intervertebral disc (D) bulging into the spinal canal and elevating the posterior longitudinal ligament (P). The annular fibres (F) attached to the cartilaginous endplate (E) arch around the posteriorly migrated nucleus pulposus (NP). The bulging disc with the posterior longitudinal ligament press upon the adjacent blood vessels. L5 = fifth lumbar vertebral body; LF = ligamentum flavum; N = neural structures within the spinal canal; S1 = first sacral body; V = blood vessels posterior to the disc bulge and associated with the neural structures. (Ehrlich's haematoxylin and light green counterstain.)

Blood vessels associated with zygapophysial joint osteophytes (Fig. vii.15)

Clinical relevance

Deformation and tractioning of blood vessels may cause pain of vascular origin.

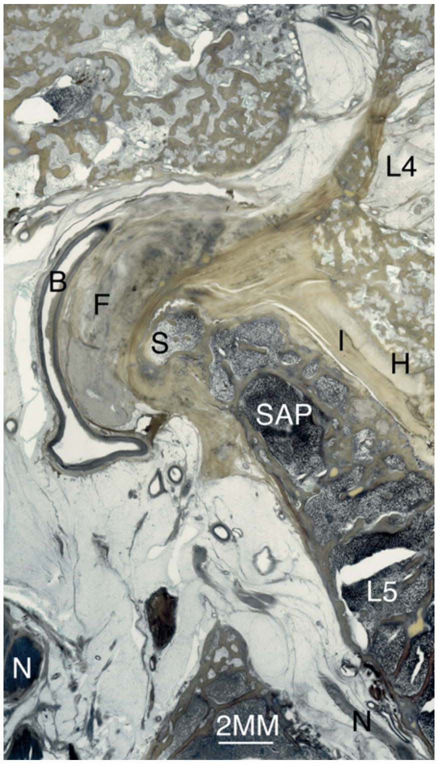


Figure vii.15 A parasagittal histopathology section of the left L4–5 intervertebral canal (foramen) from a 79-year-old male. Note how a blood vessel (B) can be deformed and tractioned by an osteophytic spur (S) projecting from the superior articular process (SAP) of the L5 vertebra and how the blood vessel conforms to the contour of the osteoarthrotic joint as it passes around the margin of the joint and its capsule. F = fibrous joint capsule-ligamentum flavum junction; H = hyaline articular cartilage (osteoarthrotic); I = fibrous interarticular synovial lined fold arising from the fibrous joint capsule-ligamentum flavum junction superiorly; L4 = part of the inferior articular process of the L4 vertebra; L5 = part of the superior articular process of the L5 vertebra; N = neural structures within the intervertebral canal foramen. (Reproduced with permission from Giles L G F 1991 A review and description of some possible causes of low back pain of mechanical origin in *homo sapiens*. Proc Aust Soc Hum Biol 4: 193-212.)

Trauma to blood vessels between moving bony parts (Fig. vii.16)

Clinical relevance

Pinching of blood vessels within synovial folds may cause traumatic synovitis; in addition haemarthrosis has been reported in zygapophysial facet joint injuries at surgery (Dr Ian Macnab, Personal communication 1983) and at autopsy (Taylor & Taylor 1996; Rauschning & Jónsson 1998). Haemarthrosis leads to cartilage damage, perhaps within two days (Jansen et al 2007).

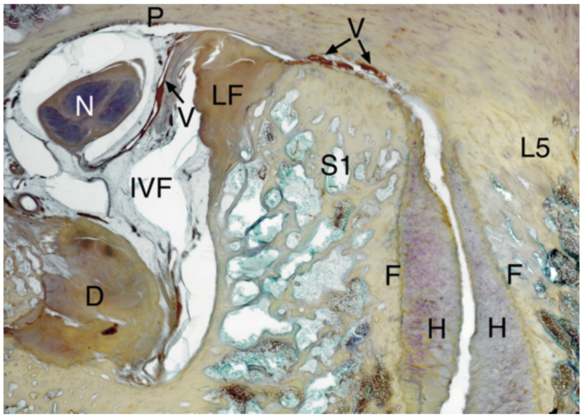


Figure vii.16 A parasagittal histopathology section at the lumbosacral level showing the intervertebral foramen (IVF) containing neural structures (N) within its upper region and an associated blood vessel (V). Note the blood in the small blood vessels (V) associated with synovial tissue in the upper pole of the zygapophysial joint that are vulnerable to being pinched between the adjacent L5 and S1 bony surfaces; such pinching could lead to bleeding within the zygapophysial joint. D = disc protruding posterolaterally into the intervertebral foramen, narrowing the foramen and deforming blood vessels; H = hyaline cartilage on the L5 and S1 facet (F) surfaces of the zygapophysial joint; LF = ligamentum flavum; P = pedicle of the L5 vertebra.

Osteoarthrosis (Fig. vii.17)

Clinical relevance

Osteoarthrosis is a common cause of spinal pain (Beers et al 2006). Beaman et al (1993) implicated osteoarthrotic lumbar facet joints with pain, having found Substance-P nerve fibres within the subchondral bone of degenerative facet joints.

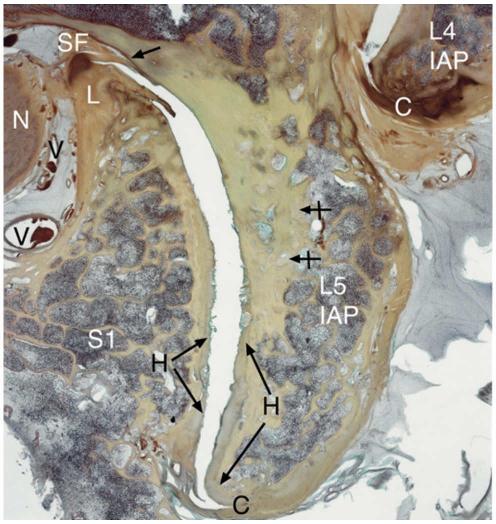


Figure vii.17 A parasagittal histopatholgy section at the lumbosacral level in a 93-year-old female. Note the osteoarthrotic zygapophysial joint in which only a small area of the opposing facets between the L5 inferior articular process and the S1 superior articular process still have hyaline articular cartilage (H). Also, there is subchondral eburnation in the L5 interior articular process (bisected arrows). A synovial fold (SF) with a fibrotic tip (arrow) projects into the upper pole of the zygapophysial joint. C = capsule at the inferior pole of the zygapophysial joint; L = ligamentum flavum; L4 IAP = inferior articular process of the L4 vertebral body; N = neural structure; V = blood vessels.

Vertebral body osteophytosis (Fig. vii.18)

Clinical relevance

Osteophytes projecting from the vertebral body can compromise adjacent neural structures causing pain.

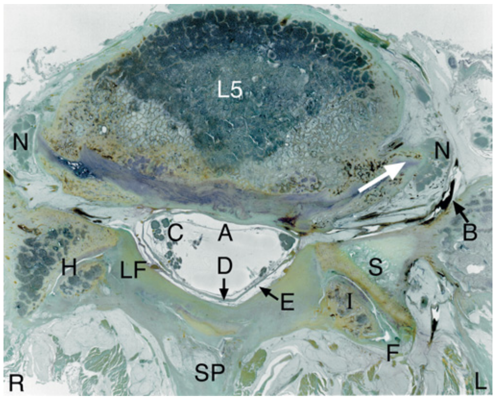


Figure vii.18 An axial histopathology section from the L5–S1 level of a 60-year-old postmortem specimen. On the right side (R), there is no osteophyte of any significance and the spinal nerve (N) passes freely beside the L5 vertebral body. However, on the left side (L), there is an osteophyte (arrow) abutting the adjacent spinal nerve that is being deformed by the osteophyte. A = arachnoid membrane; B = blood vessel; C = cauda equina nerve roots within the lumbar dural tube; D = dural membrane; E = epidural fat in the epidural space; F = fibrous capsule posteriorly for the zygapophysial joint; H = hyaline articular cartilage on the zygapophysial joint facet surfaces; I = inferior articular process of the L5 vertebra; LF = ligamentum flavum; L5 = body of the L5 vertebra; S = superior articular process of the sacrum; SP = spinous process.

Zygapophysial joint facet lipping (Fig. vii.19)

Clinical relevance

Such zygapophysial joints may well restrict motion between adjacent vertebral bodies resulting in abnormal biomechanics at such a level, resulting in pain.

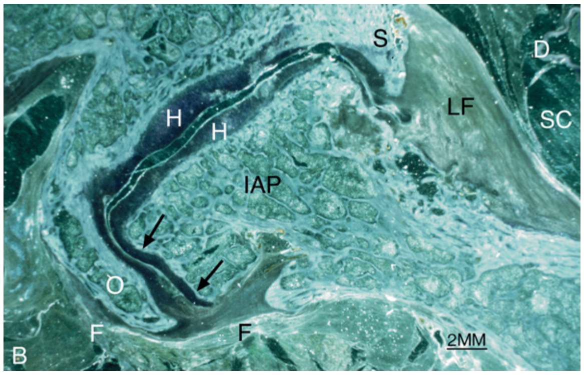


Figure vii.19 A 200-micron thick horizontal histopathology darkfield section through the right zygapophysial facet joint of a 65-year-old postmortem specimen. Note the hyaline articular cartilage (H) on the facet surfaces and how it has developed around the lateral margins of the joint forming 'bumper-fibrocartilage' (black arrows) as a result of the development of the large osteophytic spur (O) beneath the fibrous joint capsule (F). D = part of the dural tube; IAP = inferior articular process; LF = ligamentum flavum; S = spur adjacent to the ligamentum flavum and projecting from the superior articular process of the vertebra below. SC = spinal canal.

Adhesions within zygapophysial joint capsules and across joint surfaces (Fig. vii.20A and B)

Clinical relevance

Such adhesions may cause pain as the fibrous joint capsule is innervated by nociceptors. In addition, spinal manipulation may, in all likelihood, result in post-manipulation pain or discomfort due to breaking down of the adhesions.

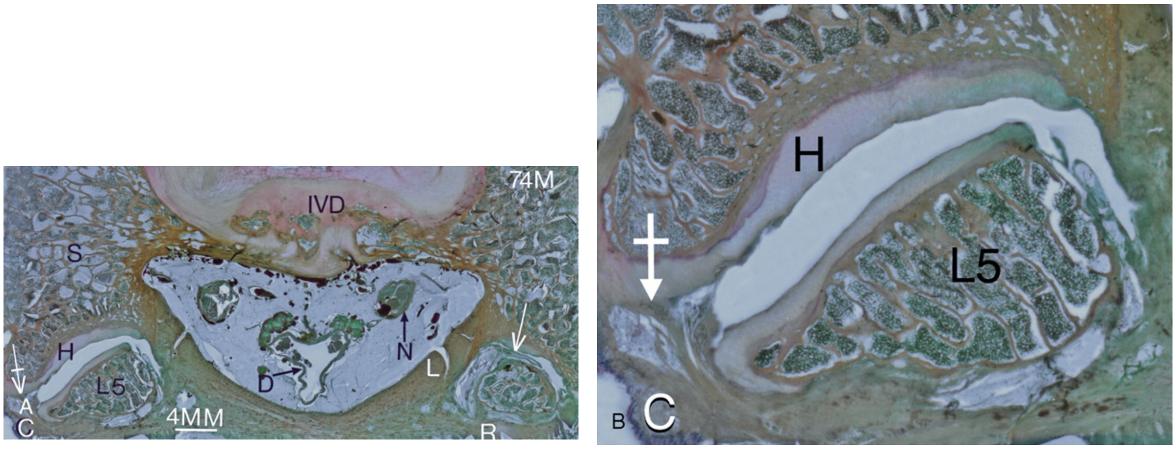


Figure vii.20A and B A 100-micron thick histopatholgy section cut in the horizontal plane from the lower one-third of the lumbosacral zygapophysial joints of a 74-year-old male cadaver. The right (R) zygapophysial joint shows a large highly vascular intra-articular synovial fold (arrow), with a fibrotic tip projecting between osteoarthrotic hyaline articular cartilage surfaces. The tip is probably fibrotic due to 'nipping' of the synovial fold between the joint surfaces during life. C = fibrous capsule, some fibres of which have become attached to the surface of the hyaline articular cartilage (H) on the sacral facet (tailed arrow) between the articulating surfaces; see enlargement in Fig. vii.20B. D = dural tube containing the cauda equina; IVD = intervertebral disc with a small midline bulge; L = ligamentum flavum with a vascular channel; L5 = inferior articular process of the fifth lumbar vertebra; N = nerve roots in the dural sleeve (see Fig. vii.21A and B for the development of nerve roots and their root sleeves); S = sacral ala. (Reproduced with permission from Giles L G F 1991 A review and description of some possible causes of low back pain of mechanical origin in *homo sapiens*. Proc Aust Soc Hum Biol 4: 193-212.)

Nerve roots (Fig. vii.21A and B)

Clinical relevance

As pressure upon nerve roots can cause radicular pain, the formation of these important structures within the normal spinal canal is illustrated in Fig. vii.21A and B.

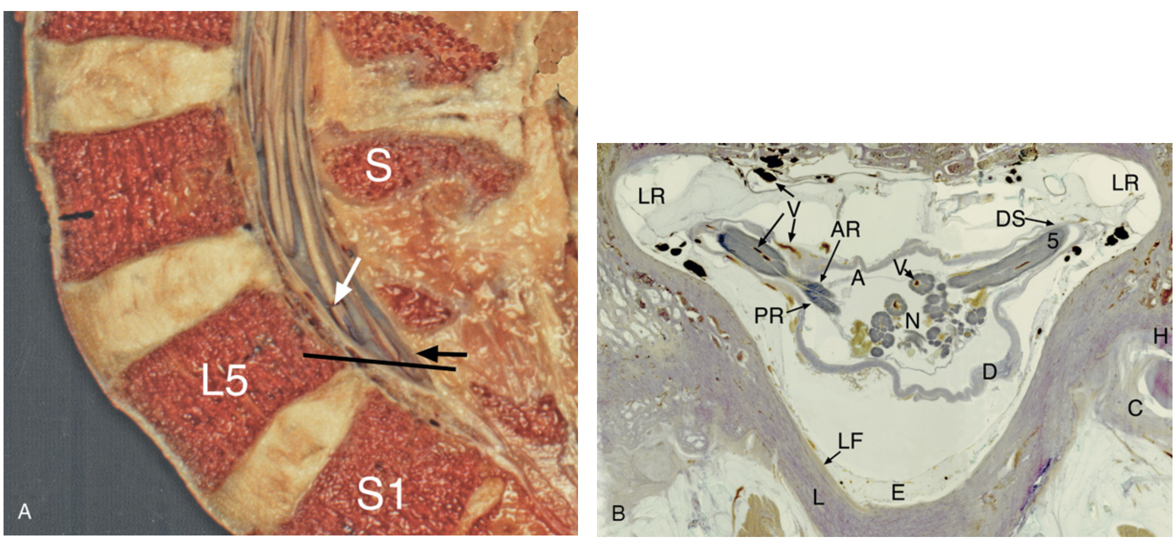


Figure vii.21 (A) Part of the lumbosacral spine showing a spine that has been bisected in the sagittal plane. Note how the nerve roots of the cauda equina (white arrow) descend with their small diameter blood vessels within the dural tube (black arrow) then pass out of the dural tube enveloped within root sleeves (Fig. vii.21B). The black line represents the approximate level of the histological section shown in (B). (B) A histological section cut approximately in the almost horizontal plane at the lumbosacral level shown by the back line in (A) from a 55-year-old male cadaver. Note that the dural tube contains the cauda equina nerve root trunks (N) with their small diameter blood vessels surrounded by epidural fat (E) within the spinal canal. A = arachnoid membrane; D = dural membrane. The anterior nerve root (AR) and the posterior nerve root (PR) trunks pass from the dural tube, within the dural sleeve (DS), to the intervertebral canal. C = fibrous joint capsule of the L5-S1 zygapophysial joint; H = hyaline articular cartilage on the sacral superior articular process; L = lamina; LF = ligamentum flavum; LR = lateral recess; V = blood vessels. (Ehrlich's haematoxylin and light green counterstain.)

Intervertebral disc protrusion with vertebral body osteophytes (Fig. vii.22A, B and C)

Clinical relevance

The osteophytes affect the paravertebral autonomic ganglia (Nathan 1962, 1968, 1987) and have been implicated in the vertebrogenic autonomic syndrome (Jinkins et al 1989, Jinkins 1997). In common practice this far-reaching perplexing, combined somatic autonomic neurogenic syndrome stems from spinal disease that includes varying degrees of (1) local somatic pain, (2) centripetally/centrifugally referred pain, (3) centripetally/centrifugally radiating pain, (4) local and referred sympathetic reflex dysfunction (diaphoresis, piloerection, vasomotor changes, somatic muscle spasm), (5) somatic reflex dysfunction, (6) somatic muscle weakness, (7) peripheral somatic dysethesias, and (8) generalized alterations in viscerosomatic tone (blood pressure, heart rate, respiratory rate, alertness) (Jinkins 1997).

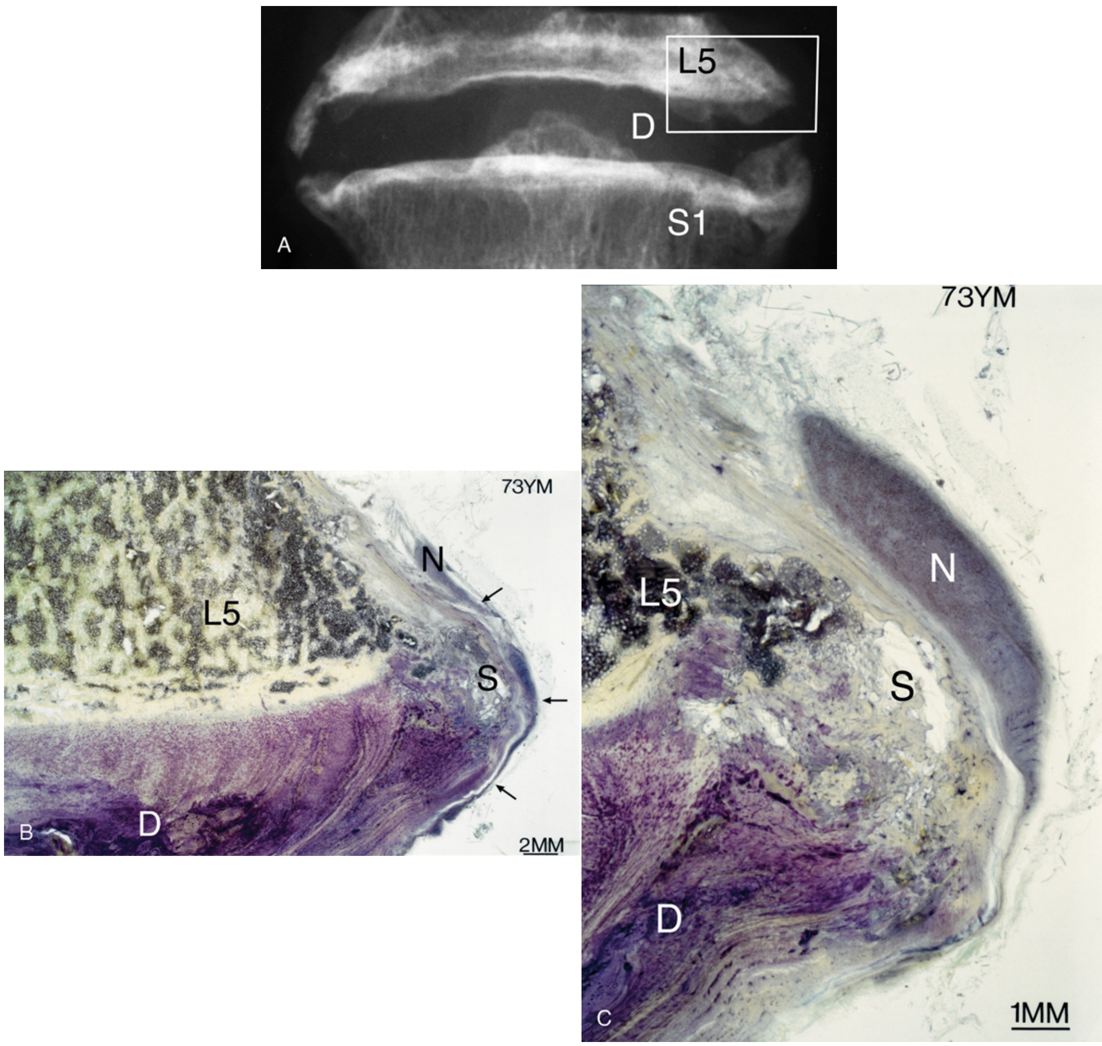


Figure vii.22A, B and C (A) Radiograph showing the L5–S1 intervertebral disc space (D) of a 73-year-old male. Arrows show osteophytes on the vertebral bodies adjacent to the intervertebral disc. (Modified from Giles L G F 1992b Pathoanatomic studies and clinical significance of lumbosacral zygapophyseal (facet) joints. J Manipulative Physiol Ther 15: 36-40.) Figure vii.22B represents a histopathology section from the approximate area shown in the rectangle in (A). (B) The paraspinal autonomic chain on the right side (arrows) is tractioned due to the large osteophyte (S) on the inferior anterolateral margin of the fifth lumbar (L5) vertebral body. D = intervertebral disc; N = lumbar paraspinal autonomic ganglion. (From Taylor J R, Giles L G F 1997 Lumbar intervertebral discs. In: Giles L G F and Singer K P (eds) Clinical anatomy and management of low back pain, Edinburgh, Butterworth-Heinemann: 49-71.) (C) A lumbar paraspinal autonomic ganglion (N), containing cell bodies, which appears to be tractioned by a large osteophyte (S) on the inferior antero lateral margin of the body of the L5 vertebra. D = intervertebral disc. (From Taylor J R, Giles L G F 1997 Lumbar intervertebral discs. In: Giles L G F and Singer K P (eds) Clinical anatomy and management of low back pain, Edinburgh, Butterworth-Heinemann, p 49-71.)

Fracture of the spine e.g. including the anterior (Fig. vii.23A and B) and posterior elements (Fig. vii.24) of the spine

Clinical relevance

Fractures of the spine can cause pain whether the fracture involves parts of the anterior elements e.g. vertebral body (Figs. vii.23A and B) or parts of the posterior elements e.g. pars interarticulares (Fig. vii.24).

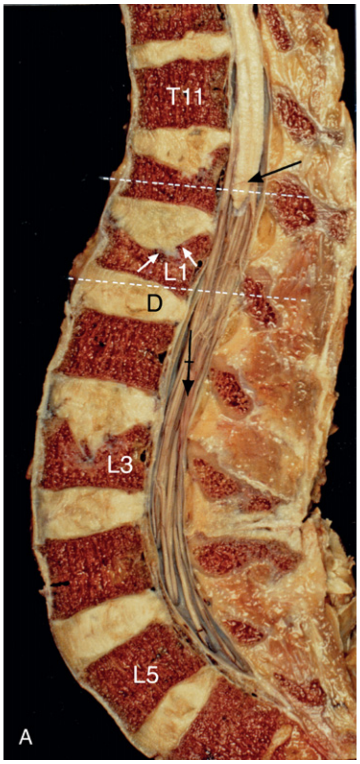


Figure vii.23A (A) A gross anatomical postmortem specimen cut in the sagittal plane from a 78-year-old male showing several vertebral body fractures of the superior endplates at T12, L1 and L3 where disc material can be seen extending into the fractured vertebral body. D = intervertebral disc; L1 = first lumbar vertebra and the white arrows show how the disc material extends inferiorly. The large black arrow indicates the lower part of the spinal cord (conus medularis) from which the cauda equina (nerve roots) extend. The nerve roots are highly vascular and the tailed arrow shows a blood vessel on one of the nerve roots. The rectangle within the broken lines represents the area from which the histological section shown in (B) was obtained.

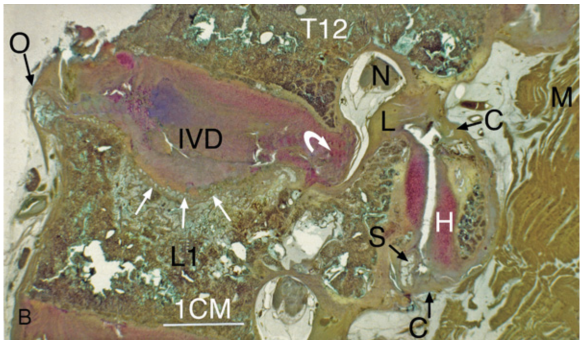


Figure vii.23B A 200-micron thick histopathology section cut in the sagittal plane through the L1 vertebral body compression fracture shown in (E). This shows an osteophyte (O) beginning to develop anteriorly. There is disruption of the intervertebral disc material (IVD) that extends inferiorly to the fractured vertebral body endplate (white arrows). There is some posterolateral bulging of disc material showing encroachment into the adjacent intervertebral canal (curved white arrow), but it is still contained within the annular fibres. In addition, the imbrication/subluxation of the opposing facet surfaces with their hyaline articular cartilage (H) is shown and this can result in tractioning of the joint capsule (C) and pinching of the synovial fold (S). H = hyaline articular cartilage on the inferior articular process of the T12 vertebral body; L = ligamentum flavum; L1 = first lumbar vertebra; M = muscle; N = neural structures within the intervertebral canal. (Ehrlich's haematoxylin and light green counterstain.)

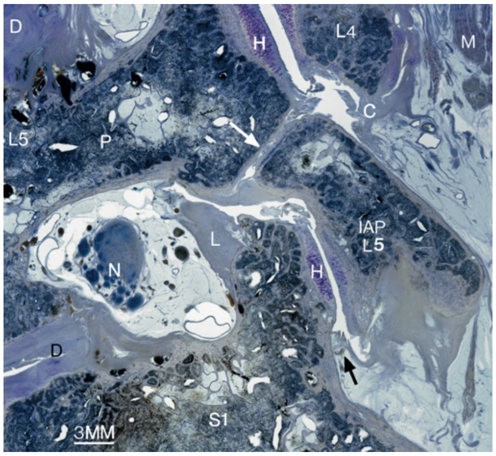


Figure vii.24 A 200-micron thick histological section cut in the parasagittal plane to include the pars interarticularis (isthmus) defect (white arrow) which has developed fibrocartilagenous type tissue on both bony surfaces. There is no true hyaline articular cartilage but there is some fibrous tissue crossing the pars defect. There is a distinct cortex on each side of the isthmus defect. C = fibrous joint capsule (disrupted); D = intervertebral disc; H = hyaline articular cartilage; IAP L5 = inferior articular process of the 5th lumbar vertebra (L5); L = ligamentum flavum (disrupted due to the pars defect); L4 = inferior articular process of the 4th lumbar vertebra; M = muscle; N = neural structures within the intervertebral canal; P = pedicle of L5; S1 = first sacral segment. Black arrow shows a synovial fold. (Ehrlich's haematoxylin and light green counterstain.) (Giles L G F 1997a Miscellaneous pathological and developmental (anomalous) conditions. In: Giles L G F and Singer K P (eds) Clinical anatomy and management of low back pain, Edinburgh, Butterworth-Heinemann, p 196-216.)

Sacroiliac joint dysfunction and osteoarthrosis (Fig. vii.25)

Clinical relevance

Sacroiliac joint dysfunction and osteoarthrosis may cause pain (Quon et al 1999).

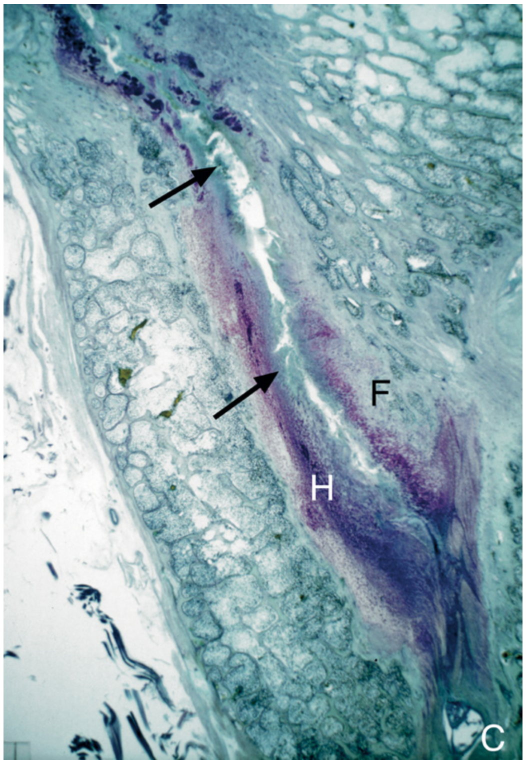


Figure vii.25 A 200-micron thick histopathology section cut in a slightly oblique plane through a sacroiliac joint from a 59-year-old female. C = fibrous articular capsule inferiorly; F = fibrocartilage on the iliac side of the joint; H = hyaline articular cartilage on the sacral articular surface, which shows osteoarthrotic changes (arrows) in this specimen. (Reproduced with permission from Giles L G F, Crawford C M 1997b Sacroiliac joint. In: Giles L G F, Singer K P (eds) Clinical anatomy and management of low back pain, Edinburgh, Butterworth-Heinemann, p 173-182.)

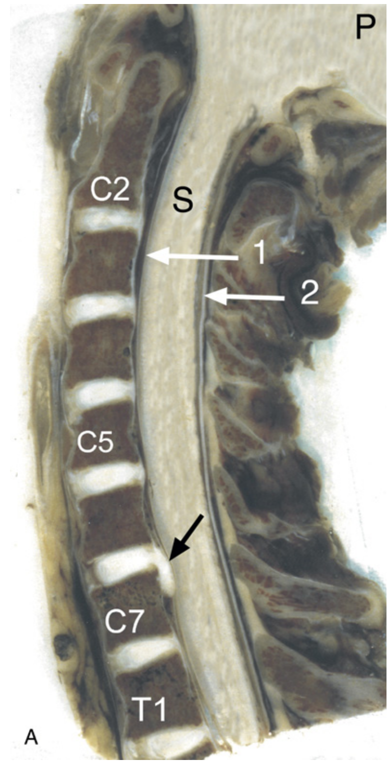
Cervical spine

Intervertebral disc posterior herniation (Figs vii.26A and B)

Clinical relevance

This may cause neck pain, with or without radicular symptoms due to:

- Pressure upon the pain sensitive anterior aspect of the dural tube (Summers et al 2005).
- Pressure upon the recurrent meningeal nerve between the protrusion and the dural tube.
- Pressure upon blood vessels (Giles 1973, Sunderland 1975, Hoyland et al 1989, Jayson 1997) between the herniation and the dural tube.
- Pressure upon a nerve root (Summers et al 2005).



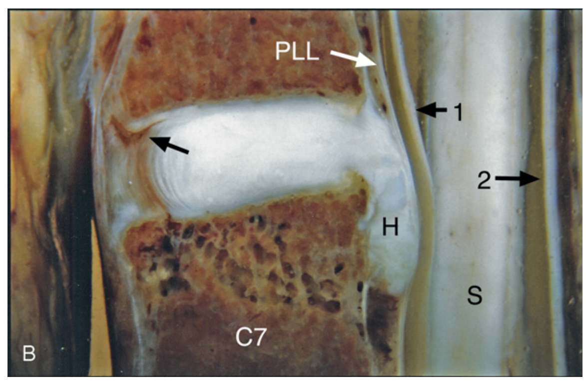


Figure vii.26A and B
(A) A sagittal section of the cervical spine from a 52-year-old woman thrown out of a vehicle that rolled over at speed. Note the spinal cord (S) within the spinal canal as well as the posterior intervertebral disc herniation at the C6–7 level (black arrow) that presses upon the pain sensitive anterior part of the dural tube and indents the anterior surface of the spinal cord. P = posterior; S = spinal cord; 1 (arrow) = dural tube (anteriorly); 2 (arrow) = dural tube (posteriorly). (Reproduced with permission from Professor J R Taylor (personal communication, 2006).)
(B) Enlargement of the C6–7 level large posterior intervertebral disc herniation (H) and a small anterior rim lesion (annular tear) (black arrow). PLL = posterior longitudinal ligament; 1 (arrow) = dural tube (anteriorly); 2 (arrow) = dural tube (posteriorly). (Reproduced with permission from Taylor J R, Taylor M M 1996 Cervical spine injuries: An autopsy study of 109 blunt injuries. Journal of Musculoskeletal Pain [The Haworth Medical Press, Inc.] Vol 4, No 4: 61-79.)

Intervertebral disc bulging and herniation, uncovertebral joint lipping, and zygapophysial joint facet osteoarthrosis (Fig. vii.27A and B)

Clinical relevance

These changes may cause neck pain, with or without radicular symptoms.

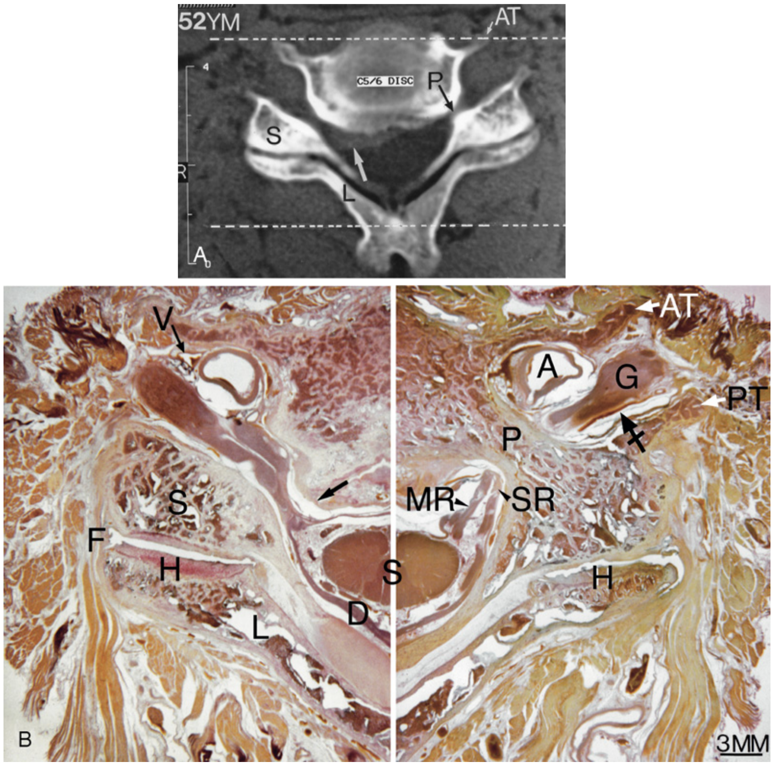


Figure vii.27A and B

(A) An example of an axial cervical spine CT scan from a 52-year-old man and a histopathology section montage, in a similar plane, from postmortem material (B). The histopathology section on the left is at the intervertebral foramen level, whereas the histopathology section on the right is at the pedicle level in order to correspond to the CT scan image. The axial CT scan (A) at the C5–6 level shows the approximate area (between the broken lines) of the histopathology sections in (B). The CT scan also shows some posterior spondylosis of the vertebral body with disc herniation on the right side (white arrow), both of which cause some narrowing of the spinal canal and the right intervertebral canal. The montage representing the two histopathology sections is from the mid-cervical spine of a middle-aged postmortem specimen. Note how osteophytosis of the posterolateral region of the uncovertebral joint (black arrow) can deform the nerve roots as shown in the histopathology section. A = vertebral artery within the transverse foramen; AT = anterior tubercle; F = fibrous capsule of the zygapophysial joint laterally; D = dural tube; G = spinal ganglion (highly vascular) and intermediate neural branch blood vessel (tailed arrow); H = hyaline articular cartilage on the facet of the inferior articular process seen in the histopathology section on the left side of the montage. The zygapophysial joint on the right shows osteoarthrotic changes; L = lamina; MR = motor root; P = pedicle; PT = posterior tubercle; S = superior articular process; SR = sensory root; V = thin-walled vein adjacent to the vertebral artery. Note the spinal cord (S) lying within the dural tube and how the motor (MR) and sensory (SR) roots pass into the intervertebral canal beneath the pedicle on the right side of the histopathology section. (Reproduced with permission from: Giles L G F 2000 Mechanisms of neurovascular compression within the spinal and intervertebral canals. J Manipulative Physiol Ther 23: 107–111.) See also colour plate section.

Synovial fold vascularity and zygapophysial joint osteoarthrosis (Fig. vii.28A and B)

Clinical relevance

Pinching of the highly vascular and innervated synovial folds could cause traumatic synovitis with haemarthrosis with the development of zygapophysial joint osteoarthrosis and pain. Synovial fold pinching with traumatic synovitis could lead to joint effusion with capsular distension which may (a) exert pressure on a nerve root (Jackson 1977), (b) cause capsular pain (Jackson 1966), or (c) cause nerve root pain by direct diffusion (Haldeman 1977).

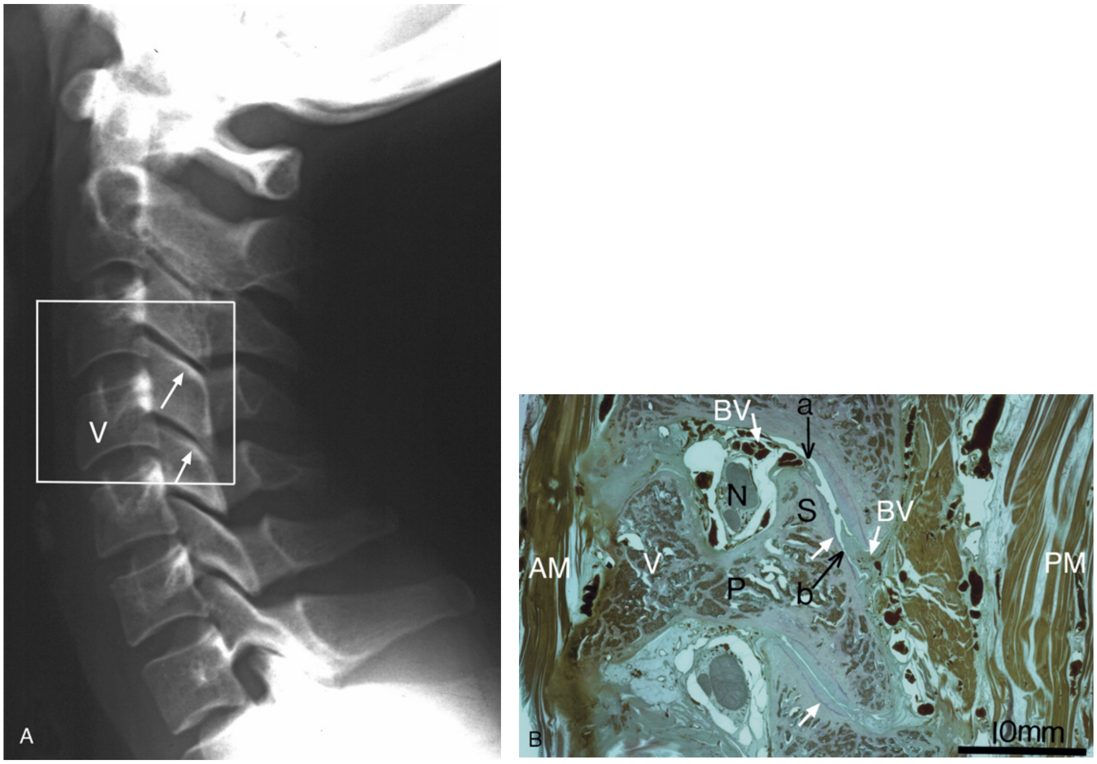


Figure vii.28A and B (A) A plain X-ray lateral view of a normal cervical spine. The square white box shows two zygapophysial 'facet' joints (white arrows), and (B) is a histopathology postmortem parasagittal section representing a similar area with two zygapophysial 'facet' joints (white arrows) and other related softtissue structures. The lower white arrow shows a zygapophysial joint with normal articular cartilage on each side of the joint's potential 'space'. The upper white arrow shows a joint with osteoarthrotic wear in the articular cartilage on the superior articular process (S). The black arrows (a) and (b) show highly vascular synovial folds projecting into the upper and lower parts of that zygapophysial joint; these structures that contain nociceptive nerves can be nipped between joint surfaces during injury, causing pain and bleeding into the joint. AM = anterior spinal muscles and PM = posterior spinal muscles, respectively; BV = blood vessels within the intervertebral foramen and in the synovial folds in the upper and lower poles of the joint; N = neural structures in the intervertebral foramen which are surrounded by fatty tissue and many blood vessels; P = pedicle that joins the vertebral body (V) to the posterior bony structures; S = superior articular process and I = inferior articular process, respectively, that form the zygapophysial joint. (From: Giles L G F 1986 Lumbo-sacral and cervical zygapophysial joint inclusions. Manual Medicine 2: 89-92.)

Intervertebral disc degeneration with associated osteophytosis (Fig. vii.29)

Clinical relevance

Intervertebral disc thinning with associated vertebral lipping may cause pain, not only due to the intervertebral disc degenerative changes but also due to their effect upon the facets of the zygapophysial joints at that level.

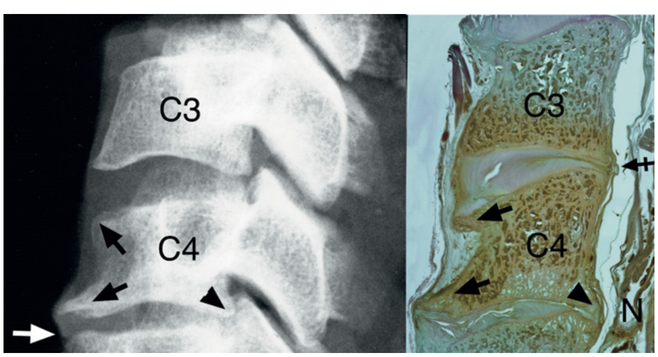


Figure vii.29
An X-ray of part of a cervical spine from post-mortem material with a corresponding histopathology section through the vertebral bodies from a 60-year-old female. Note that the X-ray shows: (i) A loss of disc space height at the C4–5 level (white arrow) with associated anterior osteophytes on the C4 vertebral body margins (black arrows) with a small osteophyte on the C4 body postero-inferiorly (black arrowhead). The corresponding degenerative disc and osteophytes are shown in the histopathology section; (ii) At the intervertebral disc one level higher (C3–4), a relatively normal intervertebral disc space height is present with an osteophyte (black arrow) at the anterosuperior margin of the C4 vertebral body. The corresponding histopathology section shows the relatively normal C3–4 level intervertebral disc, although this disc shows a small posterolateral bulge (tailed arrow), with the adjacent osteophyte on the C4 body anterosuperiorly. N = neural structures within the spinal canal.

Thoracic spine

Osteoarthrosis (Figs. vii.30 and 31)

Clinical relevance

Osteoarthrosis of the thoracic zygapophysial facet joints can cause thoracic spinal pain (Beers et al 2006). In addition, conditions such as osteophytes on the posterior margin of the vertebral body at the body and disc junction need only be small to compromise the dural tube, the recurrent meningeal nerves and blood vessels. The same principle applies to a posterior intervertebral disc bulge or protrusion

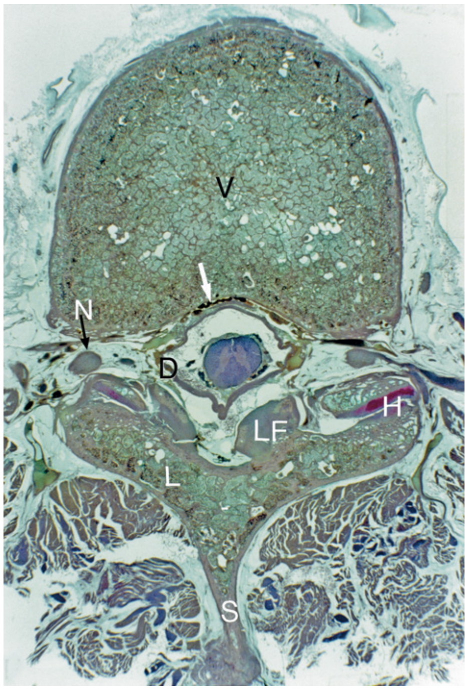


Figure vii.30 Superior to inferior horizontal view of a 200-micron thick postmortem histopathology section through the T10–11 level of the thoracic spine of a 40-year-old male. N = spinal nerve ganglion; LF = ligamentum flavum; L = lamina; D = dural tube containing the spinal cord; H = hyaline articular cartilage on the facet surfaces of the zygapophysial joints; the joint on the left side of the specimen shows osteoarthrotic changes; S = spinous process; V = vertebral body. The small space between the pain sensitive anterior part of the dural tube and the posterior part of the vertebral body, or intervertebral disc, has small blood vessels in it (white arrow). In addition, there is a recurrent meningeal nerve from each side (not visible at this magnification).

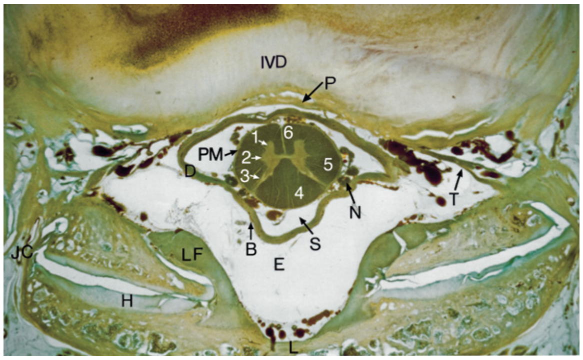


Figure vii.31 A 200-micron thick horizontal view histopathology section through the thoracic spine of a 40-year-old male cadaver. This shows the anatomy of the spinal canal and related structures at this level but without osteophytes. B = blood vessels within the dural membrane; D = dural tube; E = extra-dural (epidural) space; H = hyaline articular cartilage on the inferior articular process facet of the vertebra above; the facets on the left and right sides show osteoarthrotic changes on the superior articular processes (SAP); IVD = intervertebral disc; JC = zygapophysial (facet) joint capsule; L = lamina junction; LF = ligamentum flavum; N = dorsal (posterior) nerve root; P = posterior longitudinal ligament; PM = pia mater; S = subarachnoid space; T = transforaminal ligament crossing the intervertebral foramen; 1 = anterior grey column; 2 = lateral grey column; 3 = posterior grey column; 4 = posterior funiculus; 5 = lateral funiculus; 6 = anterior funiculus. (Ehrlich's haematoxylin and light green counter stain.)

Costovertebral joints (Fig. vii.32)

Clinical relevance

Innervation of the costovertebral joint synovial folds and the anterior capsule of the joint by requisite innervation for pain production (Erwin et al 2000) indicates that these joints are a likely source of thoracic spine pain.

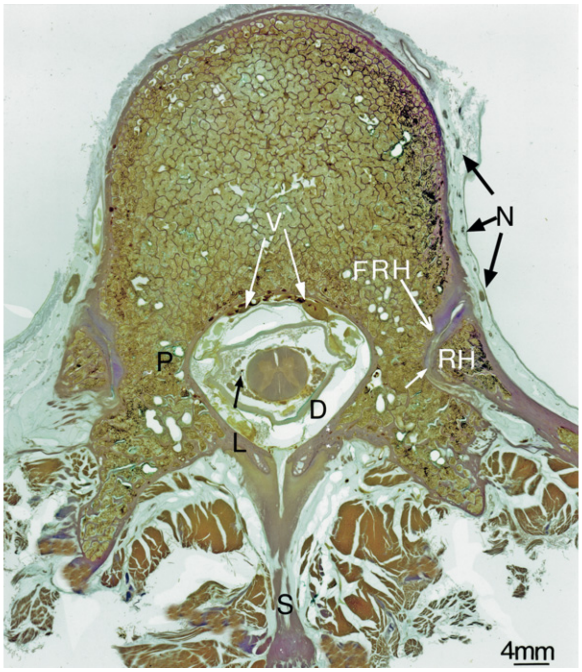


Figure vii.32 Superior to inferior view of a 200-micron thick histopathology section cut through the rib head level of a T11 vertebra from a 40-year-old male. Note the contents of the spinal canal where the spinal cord is protected within the dural tube (D) with an adequate amount of epidural fat. The black arrow shows a denticulate ligament, between the anterior and posterior nerve roots, which helps to protect the cord against shock and sudden displacement as it floats within the cerebrospinal fluid. FRH = facet (with hyaline articular cartilage) for the rib head; L = lamina; N = neural structures (autonomic nerves) adjacent to the vertebral body; P = pedicle; RH = rib head with hyaline articular cartilage; S = spinous process with muscles on its left and right sides; V = veins within the spinal canal. Note the synovial fold (short white arrow) projecting into the right costo-vertebral joint 'cavity' from its posterior margin. These synovial folds are innervated by axons expressing immunoreactivity to substance P and are a likely source of pain (Erwin et al 2000). Ehrlich's haematoxylin and light green counterstain. (Reproduced with permission from Giles L G F 1997b Introductory graphic anatomy of the lumbosacral spine. In: Giles L G F, Singer K P (eds) Clinical anatomy and management of low back pain. Oxford, Butterworth-Heinemann, p 40.)

TREATMENT

Before commencing treatment, remember that it is imperative to clearly communicate the clinician's findings from the history, examination, imaging and laboratory tests to the patient. This should be done in conjunction with the use of anatomical models as well as pictures showing gross anatomical and histopathology examples of the lumbar, cervical and thoracic spines, as appropriate, to ensure that the patient is able to understand how the findings are, in all likelihood, the cause of his or her symptoms.

Having carefully considered the above issues, including that of normal and abnormal anatomy before reaching a diagnosis, the clinician will be faced with deciding upon the most appropriate form of treatment, including referring the patient to another health discipline provider when necessary. If one adheres to the principle of '*do no harm*' and of initially taking the '*least invasive treatment approach*', patients will benefit.

There are several conservative treatment options for dealing with non-specific spinal pain syndromes of mechanical origin. For example, acupuncture, medication, spinal mobilization and or manipulation and an appropriate exercise programme, when no contraindications exist, as a conservative approach to treatment should always be considered as the first option, not only to protect patients but in order to minimize costs. However, the prescription of non-steroidal anti-inflammatory drugs requires a cautious approach because of uncertainty about safety and medico-legal concerns (Mikhail et al 2007). Taking into account the above, the vast majority of patients with degenerative disc disease can be treated with a host of conservative measures with generally good pain relief; surgical management is reserved for patients in whom these treatment options have failed (Steyn & Eksteen 2007).

In consultation with their treating clinician, patients should be given the opportunity to try whichever of these options they would prefer, as no one discipline has 'all the answers' for every patient. The ideal situation for diagnosing and treating acute and chronic spinal pain syndrome patients is to have a multidisciplinary team of individuals who specialize in different diagnostic and treatment modalities (Giles et al 2003, Giles & Muller 2003, Muller & Giles 2005) to act as a dedicated group in the best interests of patients.

At the beginning of the lumbar, cervical and thoracic spine sections, respectively, a list of some possible pathological and mechanical dysfunction causes of pain will be presented in order to supplement the abovementioned likely causes of non-specific spinal pain syndromes of mechanical origin.

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Further reading

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Whiplash injuries

In view of the number of motor vehicle accidents and the resulting possible serious injuries to the spine, soft tissues and viscera as a consequence of such accidents, a brief review of the literature relating to motor vehicle accident injuries follows, with emphasis on spinal injuries.

SOME MECHANISMS INVOLVED IN MOTOR VEHICLE ACCIDENTS

Much has been written over the years regarding the mechanisms involved in a whiplash-type injury, whether it occurs due to a collision from the rear, i.e. as a hyperextension/hyperflexion injury or due to a head on collision, i.e. as a hyperflexion/hyperextension injury (Kenna & Murtagh 1989). A simple diagram from White & Panjabi's (1990) textbook is shown (Fig. viii) to illustrate this mechanism for individuals wearing an appropriate seat belt.

Some comments regarding motor vehicles with headrests, seat belts and airbags:

Headrests

a. White & Panjabi (1990) showed that hyperextension can occur even with a headrest (as shown below); hyperflexion of the cervical spine occurs due to the restraining seat belt. White & Panjabi (1990) went on to state that the headrest limits the possible amount of extension in a collision but headrests should be at least as high as the level of the ears, which approximates the centre of gravity of the skull. Unfortunately, in many seat designs, headrests are below the centre of gravity, in which case they serve as a fulcrum and accentuate injury.
b. Olsson et al (1990) showed that a horizontal distance of more than 10 cm between the head and the head rest increases the risk of neck injuries in rear end impacts. More recently, Svensson & McIntosh (1997) found that, if the head restraint was positioned more than 5 cm behind the occiput, a rear end crash was more likely to produce neck injuries. An appropriately placed headrest may be of some assistance in minimizing soft-tissue hyperextension injuries but clearly it does not completely eliminate injuries because people do not normally drive with the back of the head resting against the headrest.

Seat belts

a. Galasko (1998) showed that there has been an annual increase in the number of patients attending an accident and emergency department (Hope Hospital, Salford, UK) with a whiplash-associated disorder (WAD) following a road traffic accident from year 1982/1983 (the year prior to the introduction of the compulsory wearing of seat belts in the United Kingdom) until 1991; there was virtually a three-fold increase in WAD presentations in the year after the compulsory introduction of seat belt usage. In addition, Galasko (1998) found that 99.64% of patients said that they had attended because of their symptoms and only 0.36% for insurance purposes.
b. The 'seat belt syndrome' described by Hayes et al (1991) is defined as consisting of skeletal, soft-tissue and visceral injuries associated with the use of two- and three-point restraints in patients involved in motor vehicle accidents; Hayes et al (1991) specifically include injuries such as 'fractures of the sternum'.

Airbags

Claytor et al (2004) examined the relationship between cervical spine injury and the type of occupant restraint systems for front-seat occupants involved in frontal motor vehicle collisions and found an increase in overall protection against cervical spine injury by combining airbag and seat belt restraint systems relative to seat belt alone.

GENERAL COMMENTS

For a condition that affects so many people in the Western World, the knowledge base is quite incomplete; we have relied on opinions and consensus instead of science (Bogduk 1998).

'More important than the exact mechanics of acceleration and deceleration collisions is the recognition of the injuries of the neck which result' (Jackson 1977). Furthermore,

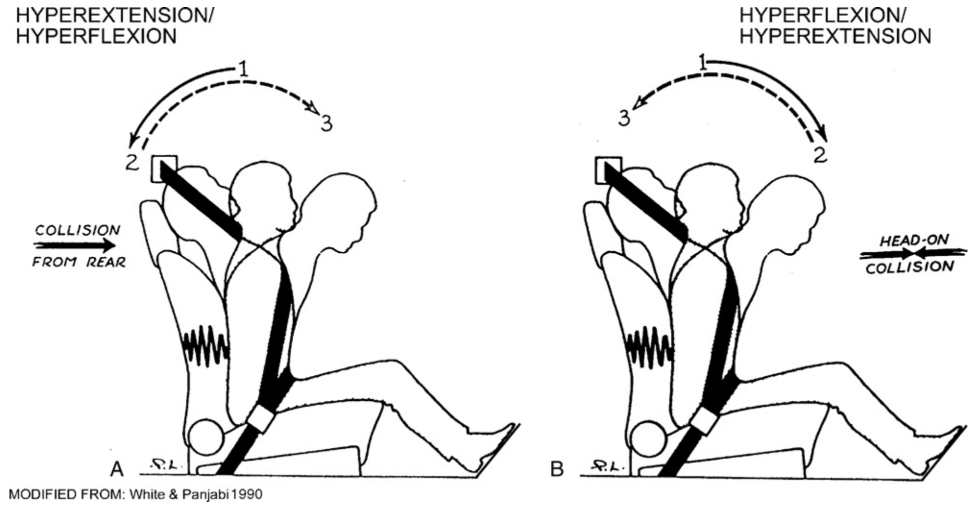

Figure viii Representation of the headrest, seat belt (including the shoulder strap), and seat back stiffness are three factors related to the extent of injury in a whiplash type accident; seat back stiffness is represented by the spring in the seat back – the stiffer the spring, the safer the seat (White & Panjabi 1990). (A) If the car is hit in the rear, the neck is thrown into hyperextension and then into hyperflexion, and (B) a head-on collision causes sudden hyperflexion of the neck, followed by a hyperextension recoil (Jackson 1977). (Modified and reproduced with permission from White AA, Panjabi MM 1990 Clinical biomechanics of the spine. Philadelphia, Lippincott Williams & Wilkins, p 233.)

Kenna & Murtagh (1989) stated: 'Patients with the whiplash syndrome present typically with varying degrees of pain, related loss of mobility of the cervical spine, headache and emotional disturbance in the form of anxiety and depression. Whiplash causes injury to soft tissue structures including muscle, nerve roots, the cervical sympathetic chain, ligaments, apophysial joints and their synovial capsules and intervertebral discs. Damage to the apophysial joints appears to be severe with possible micro-fractures (not detectable on plain X-ray) and long-term dysfunction'. It is known that injuries to the osseous or soft tissues of a joint predispose it to premature, painful, osteoarthritic change (Barnsley et al 1993). Furthermore, it has been known for many years that, 'of all the symptoms resulting from cervical sprain injuries, the most confusing are those attributable to the sympathetic nervous system' (Cailliet 1964).

Siegmund (2005) found that, during an automobile collision, the large variability in both occupant response and individual tissue injury is affected by a complex interaction of the vehicle, the seat and the occupant. This may explain why most investigators who have studied the natural history of whiplash injury patients have found differences in the severity and duration of symptoms. For example, long-term symptoms occur in 24–70% of patients, with 12–16% being severely impaired many years after the accident, resulting in difficulties with their work and daily activities (Radanov et al 1994, Borchgrevink et al 1996, Squires et al 1996, Bunketorp et al 2002, Krakenes & Kaale 2006). Galasko (1998) found that some 8.2% of patients remain disabled four years later and Greenough (1998) dispels the notion that chronic pain patients are malingering (Bogduk 1998), while Reid et al (1997) and Waddell et al (2002) confirmed that not all injuries recover even after a prolonged period of time and 5–10% of claimants go on to long- term incapacity. Furthermore, Freeman et al (1999) concluded that there is no epidemiological or scientific basis in the literature for the following statements: (1) whiplash injuries do not lead to chronic pain, (2) rear impact collisions that do not result in vehicle damage are unlikely to cause injury, and (3) whiplash trauma is biomechanically comparable with common movements of daily living.

One cannot unequivocally define precisely what were the soft-tissue injuries in many cases of whiplash-type injuries and it is necessary to be careful when referring to literature on this topic to ensure that the literature cited is based on scientific principles with appropriate methodology. Having said that, some brief comments follow.

Whiplash is a biomechanical event that occurs in a motor vehicle accident and some victims are injured while others are not; some develop symptoms while others do not (Bogduk 1998). Most symptoms are resolved but many persist and become chronic with neck pain and headache being the cardinal symptoms, although many patients also suffer from visual disturbances, disturbed balance, and altered cerebral function; however, conventional diagnostic techniques by and large fail to identify any responsible lesion or to pinpoint the source of pain (Bogduk 1998).

Acceleration of the head relative to the body results in the application of excessive torque and shear to the structures of the neck and thus in damage through both compression and distraction of tissues; clinical, animal, cadaver, and post-mortem studies have demonstrated that the cervical zygapophysial joints, intervertebral discs, muscles, and ligaments can be seriously injured through these forces without necessarily producing clinical or radiological signs (Barnsley et al 1993).

Flexion at the atlantoaxial joint will stress the alar ligament complex as the atlas attempts to rotate anteriorly to the axis; these structures appear to be susceptible to injury on the basis of postmortem studies, but demonstration of injuries in vivo is difficult (Barnsley et al 1993). However, Taylor & Twomey (2005) noted that recent studies have described strains and tears of the alar ligaments in whiplash (Volle 2000).

Clinical observations are limited to lesions that can be detected on clinical examination or at operation; ligamentous injuries cannot be diagnosed clinically (Barnsley et al 1993) but injuries do occur, even though they may not be demonstrable by contemporary imaging techniques (Bogduk 1998). For example, following injuries to the upper part of the cervical spine, adhesions may form between the vertebral artery, the first nerve root, and the bony arcuate foramen arch or canal through which they pass (Jackson 1977) and yet not be demonstrable, as is the case with injuries to many other bones and soft tissues. For example, the work of Hack et al (1995, 1997) describing the relationship between the deep suboccipital musculature (rectus capitis posterior minor muscle) and the posterior cervical dura via a connective tissue bridge, the fibres of which are oriented primarily perpendicular to the dura (Fig. ix), may explain why injury to this structure could result in tension headaches and yet the pathology, in all likelihood, would not be demonstrable.

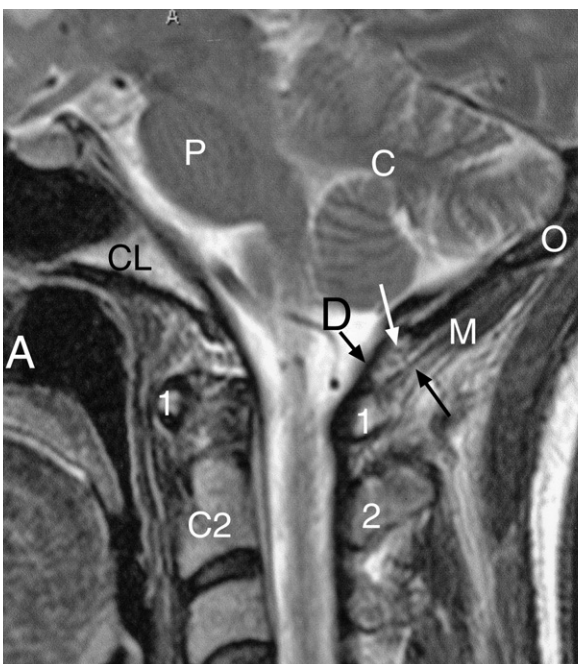

Figure ix Cervical spine MRI parasagittal T2-weighted image of a 54-year-old female patient. Note the connective tissue bridge (between the black and white arrows) connecting the rectus capitis posterior minor muscle (M) and the posterior cervical dura (D). C = cerebellum; CL = clivus; C2 = 2nd cervical vertebral body with its spinous process (2) posteriorly; O = occiput; P = pons; 1 = anterior and posterior tubercles respectively of the C1 vertebra (atlas).

No sound evidence sustains the belief that psychological factors or desire for monetary gain affect the outlook for patients with whiplash; in fact, the overwhelming majority of whiplash injuries result in organic lesions (Barnsley et al 1993). Furthermore, Centeno & Freeman (2005) performed a medical literature review and concluded that chronic symptoms following whiplash are explained most readily as an organic condition. Freeman & Croft (1998) dispel the erroneous conclusions of the Schrader et al (1996) study (Bogduk 1998) that concluded ‘chronic symptoms were not usually caused by the car accident’. In fact, Radanov et al (1998) showed that personality factors do not predict chronicity; what underlies chronicity is the lack of recognition, diagnosis and treatment of the persisting pain and other symptoms (Bogduk 1998). In a study looking at work-injured patients, Dersh et al (2007) concluded that psychiatric disturbance is not a risk factor for developing a chronic disabling occupational spinal disorder; psychiatric disorders are much more likely to develop after the onset of a work injury, indicating that such injuries and accompanying stressors are likely to be precipitants, rather than consequences, of psychopathology.

No matter how intangible or how bizarre they may seem, the symptoms of whiplash are real; these symptoms are all-too-easily dismissed as malingering if physicians rely simply on clinical examination and their own lack of knowledge of what can be done to diagnose them (Bogduk 1998).

Using simulated rear-impact accelerations on whole cervical spine specimens, Pearson et al (2004) showed that facet joint components are at risk for injury during whiplash due to facet joint compression and excessive capsular ligament strain. However, it is well known that plain radiographs essentially are non-contributory in whiplash type injuries unless there are significant bony or pre-vertebral space injuries. Soft tissues of the zygapophysial joints of the cervical spine are poorly seen but autopsy studies of the cervical spine have shown that radiology misses the majority of disc lesions, all facet joint haemarthroses and many facet fractures (Taylor & Taylor 1996). The inclusion of flexion and extension radiographs as part of the Davis (1945) series that includes the cervical spine neutral, lateral, flexion, and extension functional views are of diagnostic significance (Jackson 1977).

It should be noted that cervical spine zygapophysial joint facet capsules (Kallakuri et al 2004) and synovial folds (Inami et al 2001) have an extensive innervation so injury to neck ‘facet’ joints can cause pain as can injury to the lumbar spine zygapophysial joint facet capsules that have an extensive innervation (Giles & Taylor 1987, Grönblad et al 1991).

INTERVERTEBRAL DISC DEGENERATION

The problem of intervertebral disc degeneration has been approached from many sides, from orthopaedic surgery to molecular biology, and the scientific literature on the subject is particularly diverse; perhaps as a result of this, there is no consensus on what 'disc degeneration' actually is or how it should be distinguished from the physiological process of growth, ageing, healing and adaptive remodelling (Adams & Roughley 2006). While there is no standard definition of disc degeneration (Battie et al 2004), ageing and degeneration have been shown to be two separate processes (Rajasekaran et al 2004); the degenerative process of a lumbar disc is a cascading event which is often attributed to cumulative damage to the various spinal motion segment components (Natarajan et al 2004). Probably any abnormal loading conditions upon discs can produce tissue trauma and/or adaptive changes that may result in disc degeneration (Stokes & Iatridis 2004).

DISC DESICCATION

Desiccation relates to the secondary degenerative changes that occur with injury to intervertebral discs; it is supposed that overstress to the disc gives rise to microtears in the annulus, whereby nutritional pathways are destroyed and collagenous structures dissolve (Assheuer 2002) leading to desiccation. Numerous studies have shown that the decrease in signal intensity is related to loss of hydration and proteoglycan, typical of physiologic (age related) and pathological degeneration (Postacchini & Gualdi 1999).

There are approximately 90 fibres (Pope et al 1984) between the nucleus pulposus and the posterior margin of the lumbar intervertebral disc and these can tear due to trauma, resulting in a posterior disc bulge or protrusion that may press upon the pain-sensitive anterior part of the dural tube, or the adjacent nerve roots, causing pain (Summers et al 2005) while, at the same time, compressing blood vessels and the recurrent meningeal nerves between the bulge/protrusion and the dural tube – all these are likely methods of pain generation. If a disc suffers an annular tear causing nuclear material to leak out, that material can cause inflammation of the adjacent nerve roots, resulting in discogenic back pain and sciatica (Grönblad & Virri 1997).

Furthermore, because the dural nerves of the anterior part of the dural tube may extend up to eight spinal segments, with a great amount of overlap between adjacent nerves, compromise of these nerves may provide an anatomical basis for extra-segmentally referred dural pain (Groen et al 1988).

When a disc causes nerve root compression this may be a primary cause of decline in nerve function (due to pressure affecting the metabolism of neurotransmitters that flow inside the axons); this is important in understanding the mechanism of symptoms, such as dull pain in the leg(s) and paraesthesiae that occur in patients with nerve root compression (Kobayashi et al 2005).

The high-intensity zone is a frequent sign on MRI of patients with back pain that appears to be highly specific and strongly predictive of a symptomatic disc with severe annular disruption (Aprill & Bogduk 1992). This is supported by Peng et al (2006) who showed that the high-intensity zone in a posterior disc bulge or protrusion is a reliable marker of painful outer annular disruption and tears that may extend well into, or through, the outer third of the annulus fibrosus.

It should be noted that, once intervertebral discs are damaged, blood vessels grow into them with accompanying nociceptive nerves (Freemont et al 1997) which means that patients sustaining intervertebral disc injuries may well continue to suffer from spinal pain due to their injured discs.

CERVICAL SPINE CONTOUR

Figure x shows the expected normal smooth contour of the cervical spine on flexion and extension (Jackson 1977).

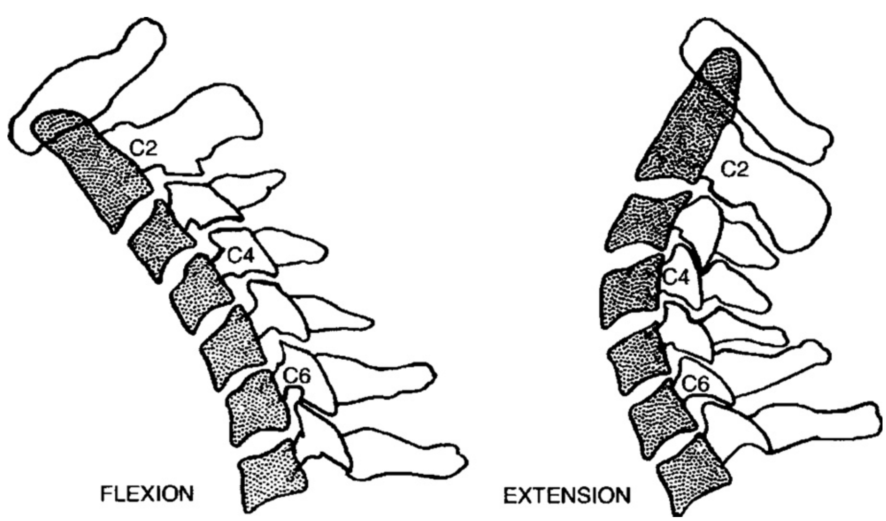

Figure x Normal contours on flexion and extension. (Modified and reproduced with permission from: Jackson R 1977 The cervical syndrome, 4th edn. Charles C Thomas, Springfield IL, p 12.)

However, in patients who have sustained a whiplash-type injury to the neck there is often a loss of the normal contour on forward bending and/or backward bending of the cervical spine indicating that, in all likelihood, soft tissue injuries have occurred to the neck. It is important to note that when there is loss of the normal cervical contour, with some straightening of the cervical spine, but no associated 'tucked-in' position of the mandible, the appearance of the cervical spine is not due to *voluntary* contraction of the deep lateral and pre-vertebral muscles of the neck. As Jackson (1977) noted, any voluntary straightening of the spine would be detected by a 'tucked-in' position of the mandible approximating or overlying the bodies of the upper cervical vertebrae; *involuntary* contraction of the muscles due to protective muscle spasm, in all likelihood, straightens the cervical spine to prevent painful movements of the joints of the neck. Furthermore, straightening of the spine enlarges the diameter of the intervertebral canals to give some relief of compressive forces on the nerve roots (Jackson 1977).

RANGES OF MOVEMENT

The measured range of overall cervical spine movement can be normal, even though injury to the neck has occurred. For example, the combined normal range of cervical spine flexion (50 degrees) and extension (60 degrees) is 110 degrees (Cocchiarella & Andersson 2000). However, Adams (1998) pointed out that the range of movement (in degrees) of cervical spine motion segments in vivo, as determined in studies on healthy individuals, is made up of a total of the movements at each spinal level, i.e. C1–C2 to C6–C7. Therefore, on clinical examination the neck may-appear to have a full range of motion in flexion (Jackson 1977) but the lateral view X-ray taken with the neck placed in maximum forward flexion may reveal that, what appeared to be a normal or average range of flexion for the entire cervical spine, was actually due to the motion taking place between the atlanto-axial, atlanto-occipital, and cervico-thoracic joints, with little or no motion occurring in the other joints. Therefore, if a spine is injured, resulting in less movement at certain levels but greater movement at other levels, the *overall* range of movement may be measured as falling within the range expected for healthy individuals.

ONLY 'DEGENERATED DISCS PROTRUDE'

There is no scientific evidence to support Dickson & Butt's (2004) contention, based on loading cadaver spines, that, in vivo, disc herniation only occurs in association with appreciable disc degeneration. On the contrary, acute trauma may produce disc herniation whether or not there is degeneration (Pope 2002); a single trauma may produce a tear in the inner lamella of the annulus fibrosus and the nucleus, under high pressure, can penetrate the fissure of the innermost annulus and, by rupturing the adjacent outer lamellae, gradually or suddenly reaches the external layers of the annulus, thus forming a contained herniation (Postacchini & Cinotti 1999); finally, a protrusion of the intervertebral disc beyond its natural borders can occur as an acute process in a healthy disc given sufficient force (Haldeman et al 2002). Thus, using cadaver spines to test loading of vertebral bodies and intervertebral discs, where the vertebrae do not have the normal haemodynamics of a living spine, cannot replicate biomechanical forces that would be experienced in life.

SOME SYMPTOMS FOLLOWING WHIPLASH-TYPE INJURIES

Headaches

When patients suffer from post-whiplash injury headaches, there may be the likelihood of a vascular (sympathetic autonomic nervous system involvement) component to the headaches and this may well be due to irritation of the recurrent meningeal nerves or to irritation of the sympathetic plexus on the vertebral and carotid arteries causing a sympathetic nervous system vaso-motor response affecting the arteries. The vertebral arteries ascend through the transverse foraminae of the cervical spine vertebrae then join to form the basilar artery; the basilar artery then provides the blood supply to various parts of the brain. Therefore, injury to the sympathetic chain on the vertebral and/or carotid arteries can lead to symptoms such as dizziness, blurred vision and memory disturbances (Jackson 1977).

Ringing in the ears and memory difficulties

Whiplash-type injuries can, in all probability, cause 'ringing in the ears' due to vascular insufficiency from vasospasm of the vertebral arteries, or from actual obstruction of the vertebral or basilar arterial supply, as well as 'memory difficulties' due to an actual stretching effect on, or trauma of, the brain stem (Jackson 1977).

Dizziness

Complaints of dizziness or vertigo-like symptoms are common following whiplash injuries (Oosterveld et al 1991), and several theories have been postulated to explain these features, including vertebral artery insufficiency, inner ear damage, injury to the cervical sympathetic chain and an impaired neck-righting reflex (Toglia 1976, Bogduk 1986). Dizziness and auditory disturbance with sensitivity to noise and feeling of disequilibrium have been described in many studies in spite of normal neurologic examination (Barnsley et al 1994, Evans 1992, Radanov et al 1994).

Arm pain

When arm pain is present, either through nerve root involvement or as somatic referred pain from the neck, the arm muscles may be inhibited directly, producing a sensation of heaviness (Barnsley et al 1993).

Late onset of symptoms

It is well known that symptoms do not necessarily appear at the time of the accident but can appear at a later time as has been documented in the literature. For example, Jackson (1977) wrote that symptoms may have occurred immediately following an injury or may have appeared a few hours, days, or even weeks after the injury; they may have appeared suddenly or they may have come on gradually.

With regard to ongoing symptoms, Teasell (1997) states: 'There has long been a misconception that all injuries should heal after six weeks. However, clinical experience and follow-up studies clearly demonstrate that not all patients necessarily get better and that there is a significant subset who continue to suffer with chronic symptoms'.

Low back pain in whiplash cases

With regard to the symptom of low back pain due to a motor vehicle accident, Teasell & Shapiro (1993) state: 'During motor vehicle accidents the lumbar and thoracolumbar spine may be suddenly forced into extension or flexed forwards as the torso moves in an arc over a fixed pelvis. In selected groups of whiplash patients the incidence of low back pain ranges from 25 to 60% (Hohl 1974, Wiley et al 1986, Hildingsson & Toolanen 1990, Braaf & Rosner 1995). Factors that have been reported to increase the risk of low back pain include side-on collisions, a soft seat back and a lap belt with no shoulder strap (McKenzie & Williams 1971, Croft & Foreman 1988). Low back pain usually resolves before the neck symptoms but, in some cases, may persist indefinitely and in some patients may become the prominent complaint (Teasell & Shapiro 1993).

IMAGING

Some imaging approaches to be considered as an adjunct to diagnosing spinal pain syndromes due to whiplash-type injuries are described below:

1. A plain X-ray Davis series (1945) i.e. a series of 7 plain X-ray views that includes flexion and extension functional views should be considered, unless the injury is so severe that it would be dangerous to perform functional (flexion and extension) views at that time.
2. Computed tomography (CT) that may be very useful for cervical spine bone injuries, although CT is considered to miss approximately 10–20% of intervertebral disc herniations.
3. Magnetic resonance imaging (MRI) is a fairly sensitive imaging procedure for injuries to the spine. However, MRI is known to fail to identify ligamentous injury (Shah & Marco 2007) and appropriate MRI techniques have to be used in order to derive maximum diagnostic information, otherwise injuries will be overlooked. For example, routine cervical spine MRI is not intended to detect, in detail, the ligamentous structures at the craniovertebral junction; a different MRI technique is required for this (Krakenes et al 2001). To investigate the craniovertebral junction (Krakenes et al 2001) and to assess the appearance of the alar ligaments, the transverse ligament, the tectorial membrane (Tubbs et al 2007) and the anterior and posterior atlanto-occipital membranes by MR imaging requires a high spatial resolution and good contrast between tissues. This can be obtained by using a proton-density-weighted (PDW) sequence with 2-mm-thick sections and the examination would need to be performed in three orthogonal planes, with the patient's head fixed in the neutral position and using a standard head coil. The axial sections should cover the foramen magnum to the base of the dens; coronal sections from the anterior arch of the atlas and halfway through the spinal canal and sagittal sections from one occipital condyle to the other. A paper titled 'Magnetic resonance imaging assessment of craniovertebral ligaments and membranes after whiplash trauma' (Krakenes & Kaale 2006) is a valuable reference. Without this specific MRI technique, structural changes in craniovertebral ligaments and membranes will, in all likelihood, be overlooked. Importantly, the study by Krakenes & Kaale (2006) found that injured soft tissue structures in the upper cervical spine play an important role in the understanding of chronic whiplash syndrome.

   From the above, it is not wise for radiologists reporting on routine cervical spine MR imaging to state 'the craniocervical junction appears normal', as the craniovertebral ligaments are not detailed on such images.

   Willen et al (1997) showed that, when lumbar CT or MR images were obtained of patients in the supine position, such images illustrated smaller disc bulge or protrusion than when the spine was under loading. With the advent of upright weight-bearing and functional MRI investigations, there are claims that functional or dynamic MRI can diagnose injuries to discs and to upper cervical spine ligaments in whiplash injuries, that are missed on static MRI (Schnarkowski et al 1993, Van Goethem et al 1996, Volle 2000, Bergholm & Johansson 2003, Schlamann et al 2007). Jinkins et al (2005) and Jinkins (2007, personal communication) showed the dramatic changes in spinal mechanical problems that are seen when comparison is made between routine recumbent MRI versus upright weight-bearing, dynamic-kinetic MRI of the spine.
4. Discography is a controversial examination that is regarded by some radiologists and spine surgeons as barbaric and non-efficacious (Wiley et al 1968, Clifford 1986, Shapiro 1986) and the procedure has the potential for producing discitis so it must be prescribed sparingly (Shelokov 1991). Although the reported complication rate of provocative lumbar discography is low, ranging from

0 to 2.5%, acute lumbar disc herniation has been precipitated by this procedure (Poynton et al 2005). The exact incidence of complications after cervical discography is uncertain, with discitis being the major serious concern (Gibson 1991).

Taking into account all the above comments regarding whiplash-type injuries, and the gross anatomy and histopathology specimens showing bony and soft-tissue structures of the spine in the Introduction, patients injured in motor vehicle accidents deserve to have a thorough diagnostic work-up that can lead to appropriate treatment and, when required, an informed medico-legal report being written for an attorney or a third party insurer. There is no place for a cursory work-up.

The following brief outline may be of assistance to clinicians as a basic general format for medico-legal reporting, even though different jurisdictions may dictate that a specific format is required for such reports.

Remember that no information should ever be released on a patient without the patient first signing an appropriate release form authorizing the clinician to write such a report.

YOUR NAME AND QUALIFICATIONS	ADDRESS Tel: Fax:

PRIVATE & CONFIDENTIAL

ADDRESSEE DATE

INDEPENDENT MEDICO-LEGAL REPORT

YOUR NAME

Qualifications: Please find attached an abridged copy of my current Curriculum Vitae.

I refer to your letter dated .... seeking my expert opinion in this matter.

The plaintiff:
Your reference:
Address:
DOB:
Occupation:
Date of accident:
Place of examination:
Date of examination:
ID:

MATERIAL FACTS

The documentation that has been provided to me for the purpose of providing this report is as follows:

- LIST REPORTS RECEIVED AND THEIR DATES

In addition, I took a history and examined the plaintiff today and viewed the imaging as described under Imaging in this report.

INFORMED CONSENT:

Prior to commencing the evaluation, I explained to your client that the interview was for legal purposes, that what he/she said to me will be put into a report which would be made available to the court, and that I could not guarantee that my opinion would be favourable to him/her. He/she indicated that he/she understood this and that he/she wished for me to continue.

REFERENCES

Any references used in the preparation of this report are shown as a footnote on the appropriate page.

PROFILE

AGE	
SCHOOL LEVEL COMPLETED	
OCCUPATION	
- AT TIME OF ACCIDENT	
- CURRENTLY	
MARITAL STATUS	
NUMBER AND AGE OF CHILDREN	
BUILD	
SMOKER	
ALCOHOL	
DOMINANT HAND	
HOBBIES BEFORE THE ACCIDENT	
HOBBIES SINCE THE ACCIDENT	

HISTORY OF ACCIDENT/INJURY

- *Symptoms following the accident*.

PAST HISTORY

PRESENT STATE

CURRENT TREATMENT

ACTIVITIES OF DAILY LIVING

- Personal care:
- Domestic activities:

CLINICAL EXAMINATION

PAIN ASSESSMENT

(1) Pain Diagram
(2) Visual Analogue Scale
(3) Low Back Oswestry Disability Questionnaire
(4) Neck Pain Disability Index

IMAGING

Please find attached a copy of the following reports for imaging:

DATE OF IMAGING – (approximately . . . .years . . . .months pre/post the accident on . . . . . .), AREA IMAGED, IMAGING FACILITY, REPORTING RADIOLOGIST'S NAME.

OPINION AND CONCLUSIONS

My following opinion is based on information obtained from the history, observation, examination and imaging.

DIAGNOSIS

- *Cervical:*
- *Thoracic:*
- *Lumbar:*

ATTRIBUTABILITY

I am of the opinion that the reported injuries are/are not consistent with having been caused by the accident in question.

Based on the history, examination and imaging it is my opinion that the cervical spine/thoracic spine/lumbar spine soft tissue injuries/symptoms are . . . .% attributable to the MVA/accident in question.

PROGNOSIS and STABILITY OF THE CONDITION

In my opinion, the prognosis must be guarded as his/her condition appears to be/not to be stable and stationary some . . . . post injury.

In my opinion, your client suffers a disability in that he/she is physically handicapped because he/she cannot perform his/her usual work. In addition, your client has some degree of impairment in that he/she now has an alteration of his/her previously asymptomatic spinal health status that interferes with his/her activities of daily living.

WORKABILITY – short- and long-term workability.

PRE-EXISTING CONDITION AND IMPAIRMENT APPORTIONMENT

As far as I am aware, there are/are no known pre-existing conditions.

ANY POSSIBLE INCONSISTENCIES IN THE EXAMINATION

There are/are no inconsistencies in the examination.

RECOMMENDATIONS

? Further diagnostic follow-up, e.g. MRI . . . . .

ASSISTANCE REQUIREMENTS – short and long term.

In my opinion, your client should see an occupational therapist regarding assistance requirements.

PERMANENT DISABILITY

Please see the above detailed discussion.

COMMENTS ON OTHER REPORTS

I have no comment in this regard apart from my comments relating to Imaging as stated above.
(Comment on other reports if requested to do so.)

OTHER COMMENTS AND INFORMATION RELEVANT

INVESTIGATION/EXPERIMENTATION

No experiments have been conducted for the preparation of this report.

FURTHER FACTS

There are no readily ascertainable additional facts or materials that would assist me in reaching a more reliable conclusion.

OTHER INFORMATION

If any additional information were to become available in the future it could change my opinion but, with the current information available, this opinion has been formulated today.

TO ANSWER YOUR SPECIFIC QUESTIONS

1.
2.
3.

The factual matters stated in this report are, as far as I know, true and correct. I have made all enquiries which I consider to be appropriate and the opinion stated in this report is genuinely held by me. I have referred to all matters which I consider significant in preparing this report.

All imaging that your client brought to the examination has been returned to your client.

Yours faithfully,

Encl. Imaging Reports
Abridged Curriculum Vitae

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Introduction

Before presenting the low back pain cases it is important to consider the following summary of some possible causes of low back pain with or without radiculopathy (Table iii).

PHYSICAL EXAMINATION

The physical examination should be orderly and systematic and should include the lumbar, abdominal and pelvic examinations as indicated by the patient's presenting complaint(s); in Table iv some general observations and orthopaedic and neurological tests are listed.

Figure xi represents the clinical features of the posterolateral lumbar intervertebral disc herniation and Fig. xii the motor innervation of the lower limb; central disc herniation may cause spinal pain alone without radiculopathy (Postacchini & Gumina 1999) or lower limb involvement.

Table iii Some possible causes of lumbar spine pain

Acute spinal pain
Febrile disorders
Injury

Chronic spinal pain

1. **Traumatic, mechanical or degenerative:**
   Low back strain; fatigue, obesity; pregnancy causing altered biomechanics
   Injuries of bone, joint, intervertebral disc or ligaments
   Degenerative or traumatic changes of the spine (osteoarthrosis; spondylosis)
   Lumbar spine instability syndromes e.g. spondylolisthesis
   Scoliosis: primary and secondary
   Spinal or intervertebral canal stenosis
   Sacroiliac joint strain
2. **Joint dysfunction:**
   Zygapophysial
   Intervertebral disc
3. **Metabolic:**
   Osteoporosis
   Osteomalacia
   Hyper- and hypo-parathyroidism
   Ochronosis
   Fluorosis
   Hypophosphataemic rickets
4. **Unknown causes:**
   Inflammatory arthropathies of the spine, such as ankylosing spondylitis and the spondylitis of Reiter's (Brodie's) disease, psoriasis, ulcerative colitis, Whipple's and Crohn's diseases
   Rarely polymyositis and polymyalgia rheumatica
   Paget's disease of bone
   Scheuermann's 'disease'
5. **Infective conditions of bone, joint and theca of the spine:**
   Osteomyelitis
   Tuberculosis
   Melioidosis
   Undulant fever (abortus and melitensis)
   Typhoid and paratyphoid fever and other *Salmonella* infections
   Syphilis
   Yaws
   Very rarely Weil's disease (leptospirosis icterohaemorrhagica)
   Spinal pachymeningitis
   Chronic meningitis
   Subarachnoid or spinal abscess
   Herpes zoster
   Poliomyelitis
   Tetanus

table continues

Table iii Some possible causes of lumbar spine pain—Cont'd

6. **Psychogenic:**
   Anxiety
   Depression
   Hysteria
   'Compensation neurosis'
   Malingering
7. **Neoplastic – benign or malignant, primary or secondary:**
   Osteoid osteoma
   Eosinophilic granuloma
   Metastatic carcinomatosis
   Bronchial carcinoma
   Oesophageal carcinoma
   Sarcoma
   Myeloma
   Primary and secondary tumours of spinal canal and nerve roots: ependymoma; neurofibroma; glioma; angioma; meningioma; lipoma; rarely cordoma
   Reticuloses, e.g. Hodgkin's disease
8. **Cardiac and vascular:**
   Subarachnoid or spinal haemorrhage
   Luetic or dissecting abdominal aorta aneurysm
   Enlarged aortic aneurysm
   Grossly enlarged left atrium in mitral valve disease
   Arteriovenous malformations
9. **Gynaecological:**
   Tuberculous disease
   Rarely prolapse or retroversion of uterus
   Dysmenorrhoea
   Chronic salpingitis
   Pelvic abscess or chronic cervicitis
   Tumours
10. **Gastrointestinal:**
    Pancreatitis
    Rarely appendicitis, or from new growth of intra-abdominal viscus (colon, stomach, pancreas), or from retroperitoneal structures
11. **Renal and genito-urinary:**
    Carcinoma of kidney
    Calculus
    Hydronephrosis
    Polycystic kidney
    Necrotizing papillitis
    Pyelitis and pyelonephritis
    Perinephric abscess
    Infection or new growth of prostate
12. **Blood disorders:**
    Sickle-cell crises
    Acute haemolytic states
13. **Drugs:**
    Corticosteroids
    Methysergide
    Compound analgesic tablets
14. **Normality:**
    (Non-disease, i.e. non-specific spinal pain of mechanical origin)

Modified from Hart F D 1985 Back, pain in. In: Hart FD (ed) French's index of differential diagnosis, 12th edn. Butterworth & Co. Ltd, Oxford, pp 72–73.

Table iv Some elements of the lumbar spine physical examination

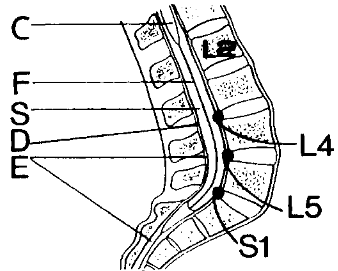
Erect posture examination	
Observe for	*Palpate for*
Fluidity of movement	Iliac crest levels
Body build	Anterior and posterior superior iliac spine levels
Skin markings – café-au-lait spots, lipomata & hairy patches often denote underlying neurologic or bone pathology	Any break in contour of spinous processes (spondylolisthesis)
	Muscle spasm
Posture	Myofascial trigger points
Deformities	Supraspinous and interspinous ligament tenderness
Pelvic obliquity	Adjacent muscle tenderness
Spine alignment	Sciatic nerve tenderness
Sacroiliac joint	Posterior aspect of coccyx
Examine for joint motion or joint pain	Relative motion between adjacent vertebrae (by motion palpation) in an attempt to find restricted movement

table continues

Table iv Some elements of the lumbar spine physical examination—Cont'd

Erect posture examination	
Test spinal column motion, with caution, for	*Observe gait*
Flexion	Walking on heels (tests foot and great toe dorsiflexion)
Extension	Walking on toes (tests calf muscles)
Side bending	Walking on toes (tests calf muscles)
Rotation	
Seated	
Neurological tests	*Sensory distribution around the anus (S2, S3, S4) with the patient flexed forwards*
Ankle jerk	Slump test
Knee jerk	Slump test plus straight leg raising
Plantar response (Babinski test*)	Thigh/calf circumference measurement bilaterally
Pinprick sensation of lower limbs	
Vibration sensation	
Supine	
Straight leg raising	Hip joint
Flex thigh on pelvis then extend knee with foot dorsiflexed (sciatic nerve stretch)	Fabere/Patrick test*
	Hip flexion, internal and external rotation
Hoover test*	Palpate abdomen
Kernig test (spinal cord stretch)*	Palpate for peripheral pulses and skin temperature
Tests to increase intrathecal pressure	Palpate for flattening of lumbar lordosis during straight leg raising
Milgram test*	
Naffziger test*	Measure leg lengths (anterior superior iliac spine to medial malleolus) for a very *approximate* clinical impression of leg lengths
Valsalva manoeuvre*	
Sacroiliac joint	Test sensation and motor power
Compression test	Listen for bruit (abdominal and inguinal)
Pelvic rock test	
Gaenslen's sign*	
Prone	
Palpate	
Sciatic nerve between ischial tuberosity and greater trochanter	Palpate trochanteric bursa
Ischial bursa	Spine extension
Cluneal nerves crossing the iliac crest for local tenderness	Femur extension test for hip extension
Prone or lateral decubitus position	
Femoral nerve stretch test*	

*See Definitions and abbreviations chapter.
Adapted from Hoppenfeld 1976, Mackenzie 1985, Keim & Kirkaldy-Willis 1987.



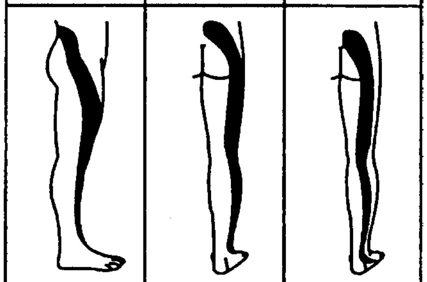
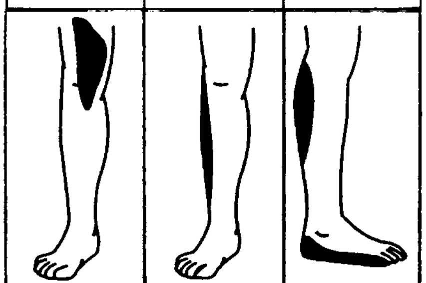
HERNIATION	L3-4	L4-5	L5-S1
NERVE ROOT	**L4**	**L5**	**S1**
PAIN			
	Low back ► hip ► antero-lateral thigh ► medial leg	Above S-I joint ► hip ► lateral thigh & leg ► dorsum of foot	Above S-I joint ► hip ► posterolateral thigh & leg ► heel
NUMBNESS			
	Anteromedial thigh & knee	Lateral leg & first 3 toes	Back of calf ► lateral heel & foot ► toe
ATROPHY	Quadriceps	Minor or non-specific	Gastrocnemius & soleus
MOTOR WEAKNESS	Extension of quadriceps	Dorsiflexion of great toe and foot	Plantar flexion of great toe and foot
SCREENING EXAM	Squat and rise	Heel walking	Walking on toes
REFLEXES	Knee jerk diminished	None reliable	Ankle jerk diminished

Figure xi Anatomy and clinical features of a posterolateral lumbar intervertebral disc herniation. C = conus medularis; D = dural tube; E = epidural space; F = filum terminale; S = subarachnoid space. (Modified from Wilkinson J L 1986 Neuroanatomy for medical students. John Wright and Sons, Bristol, p 46; Keim & Kirkaldy-Willis 1987; Bigos S, Bowyer O, Braen G et al 1994 Acute low back problems in adults. Practice guideline, Quick Reference Guide Number 14. US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, Rockville, MD, AHCPT Pub. No. 95-0643.)

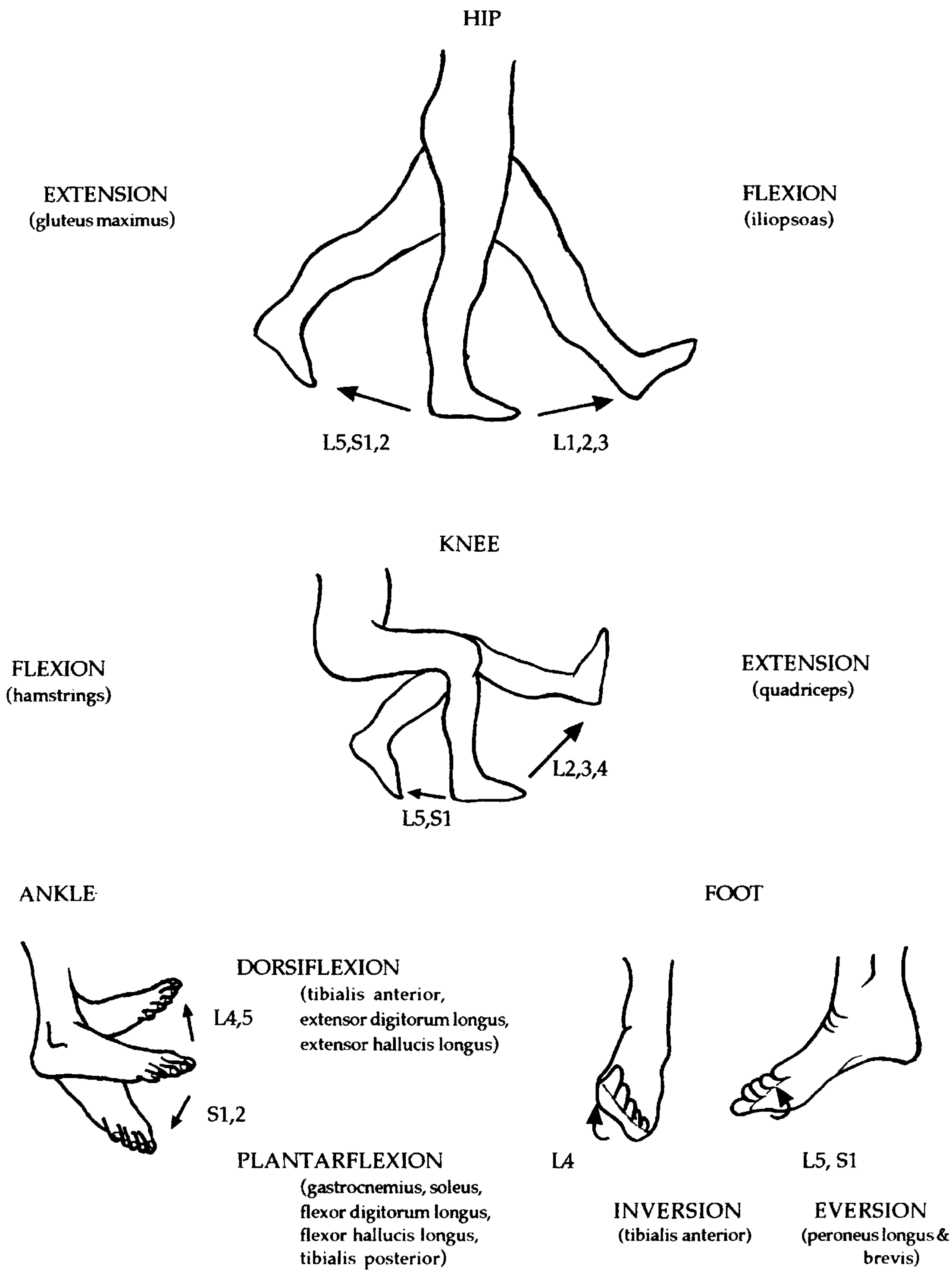


Figure xii Motor innervation of the lower limb. (Modified from: Hoppenfeld S 1977 Orthopaedic neurology. A diagnostic guide to neurologic levels. JB Lippincott, Philadelphia; Keim & Kirkaldy-Willis 1987; Moore K L & Dalley A F 2006 Clinically oriented anatomy, 5th edn. Williams & Wilkins, Baltimore.)

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Case 1

L5–S1 posterior central intervertebral disc protrusion not abutting adjacent nerve roots

COMMENT

When MRI shows a central disc protrusion not abutting the nerve roots, patients may only experience low back pain without radicular symptoms.

PROFILE

A fit 29-year-old female soldier who does not smoke cigarettes and only drinks alcohol socially.

PAST HISTORY

There is nothing contributory.

PRESENTING COMPLAINT(S) (Fig. 1.1)

She has experienced central low back pain intermittently for approximately 15 months. There are no symptoms in her lower limbs. Her low back may be stiff on awakening in the morning but there is no night pain. Bowel and bladder functions are normal. Non-steroidal anti-inflammatory drugs provide only temporary relief.

AETIOLOGY

The aetiology is unknown but she said she often jumps out of army vehicles while carrying a heavy backpack and this causes her to feel twinges of central low back pain.

EXAMINATION

In the erect posture there was no clinical evidence of leg length inequality, pelvic obliquity or scoliosis. Lumbar spine active ranges of movement were of full range and painless, apart from flexion and extension that caused some increase in low back pain at almost full range. The slump test slightly increased her low back pain. The addition of straight leg raising to the slump test did not cause any increase in low back pain. In the supine position, straight leg raising (SLR) was of full range and painless. Lasegue's test did not elicit any pain. Deep tendon reflexes in the lower extremities were normal as were power, pinprick sensation and vibration sensation. The Valsalva manoeuvre caused a slight increase in low back pain. Bilateral hip flexion caused an increase in low back pain. The abdomen was normal on examination.

IMAGING REVIEW

Plain X-ray films were reported as normal. However, they show a slight loss in the height of the L5–S1 intervertebral disc with a minor degree of retrolisthesis of L5 on S1.

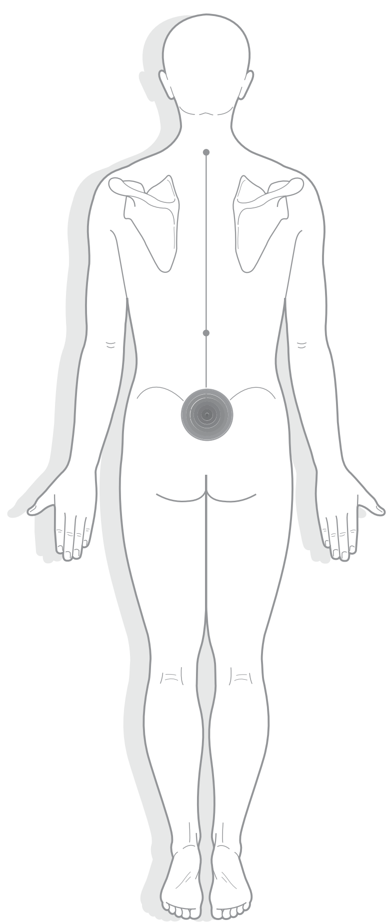

Figure 1.1

CLINICAL IMPRESSION

A possible L5–S1 central posterior intervertebral disc bulge or protrusion in view of a positive Valsalva manoeuvre with normal nerve root tension signs, normal sensation and power, and the slight loss in height of the L5–S1 intervertebral disc with a minor degree of retrolisthesis of L5 on S1.

WHAT ACTION SHOULD BE TAKEN?

A MRI lumbar spine was requested and the radiology report stated: 'L5–S1 central disc protrusion with reduced signal intensity and slight narrowing of this disc (Fig. 1.2A and B). Possible slight depression fracture of the posterior aspect of the S1 body with disc material protruding into this defect

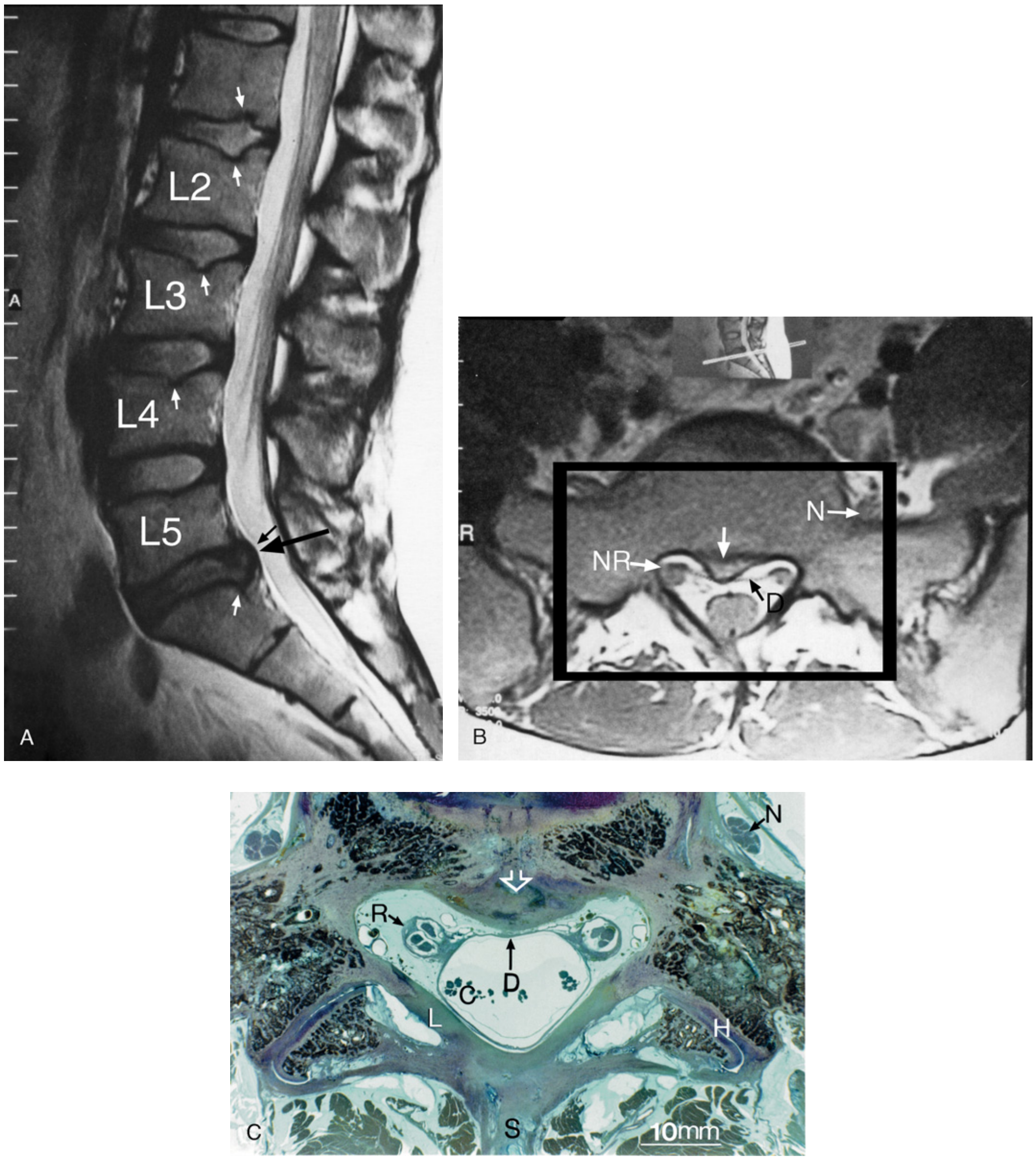

Figure 1.2 (A) Lumbar spine MRI sagittal T2-weighted image showing the: (i) L5–S1 central disc protrusion (black arrow) with reduced signal intensity and slight narrowing of this disc, (ii) possible slight depression fracture of the posterior aspect of the S1 body with disc material protruding into this defect as a Schmorl's node (white arrow), (iii) Schmorl's nodes involving some of the L1 to L4 endplates (small white arrows), and (iv) posterior disc protrusion elevating the posterior longitudinal ligament and effacing the pain sensitive anterior surface of the dural tube (small black arrow). (B) Lumbar spine MRI axial Intermediate Weighted Spin Echo image at the L5–S1 level showing the central disc protrusion (arrow) that does not abut the adjacent nerve roots (NR) but effaces the pain sensitive anterior surface of the dural tube (D). N = spinal nerve; R = right side of patient. The rectangle shows the approximate area shown in (C) where a central posterior disc protrusion does not abut the nerve roots but touches the dural tube in the spinal canal at the level of the L5–S1 zygapophysial joints. (C) A 200-micron thick histopathology section from a cadaver and cut through the level of the L5–S1 zygapophysial joints with a similar, central posterior disc protrusion; this is to orientate the reader to the various anatomical structures. The histological section is represented approximately by the area within the rectangle on (B). R = nerve root sleeve budding off from the dural tube (D) that contains small nerve roots arising from the cauda equina (C). H = hyaline articular cartilage on the zygapophysial joint facet surfaces. L = ligamentum flavum; N = spinal nerve. Open white arrow = intervertebral disc protrusion that is central and just indents the anterior surface of the dural tube. (Ehrlich's haematoxylin and light green counterstain.) See also colour plate section Fig. vii.2.

as a Schmorl's node. Schmorl's nodes are noted involving some of the L1 to L4 endplates'. Note that the posterior disc protrusion does not abut the nerve roots (Fig. 1.2B) but touches the pain sensitive anterior surface of the dural tube. This principle is illustrated in the histopathology section (Fig. 1.2C) where a posterior disc protrusion in a post-mortem specimen touches the pain sensitive anterior surface of the dural tube but does not abut the nerve roots; therefore, there is no sciatica but only central low back pain.

DIAGNOSIS

Musculoligamentous soft-tissue injuries of the lumbar spine.

L5–S1 central posterior intervertebral disc protrusion that touches the anterior surface of the dural tube but does not abut the nerve roots.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was told that there was a central posterior disc protrusion at the L5–S1 level not touching the adjacent nerve roots and that she should:

1. Change her daily activities so that she does not have to jump off the back of army vehicles. This should prevent sudden axial loading of the lumbosacral (and other lumbar) intervertebral discs in an attempt to reduce the risk of a larger protrusion occurring which may then compromise the adjacent nerve roots, causing sciatic type problems. A letter was written to the army medical doctor confirming the need for the patient to refrain from this activity.
2. Not lift heavy objects.
3. Undertake a more sedentary occupation that would be less likely to aggravate the existing Schmorl's nodes in the lumbar spine.
4. Avoid, if possible, non-steroidal anti-inflammatory drugs due to the possible associated adverse reactions.
5. Perform muscle strengthening exercises to strengthen the abdominal, buttock and lumbar muscles in order to provide good support for her lumbosacral spine (see Figs. 1.3 and 1.4), as well as an exercise to mobilize the lower lumbar nerve roots (see Fig. 1.5).

This treatment approach was satisfactory and, at follow-up one month later, she said that she could control her low back pain in view of the abovementioned precautions.

Note

According to Coventry et al (1945a), Fahey et al (1998) and Jensen et al (1994): 'A Schmorl's node refers to herniation of the disc material through the cartilaginous endplate into the adjacent vertebral body'. Most consider Schmorl's nodes to be asymptomatic. However, some believe that Schmorl's nodes may be associated with pain (Hamanishi et al 1994). In this regard, most believe that the Schmorl's node itself does not cause pain, but that symptoms may occur as a result of damage to associated structures (Coventry et al 1945b). The association between trauma and the subsequent development of a Schmorl's node may be particularly important in the medicolegal setting. Also, it has been reported that Schmorl's nodes may give rise to disc degeneration (Hilton et al 1976). Therefore, Schmorl's nodes may be an important predictor of degenerative spine disease in later life.

KEY POINT

Central posterior intervertebral disc protrusion that touches the pain-sensitive anterior surface of the dural tube, and compresses the intervening recurrent meningeal nerves and blood vessels, causes low back pain without radiculopathy.

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APPENDIX: BASIC LOW BACK EXERCISES

(Modified from: Burton C, Nida G 1977 Be good to your back. The Sister Kenny Institute Low Back Clinic. Rehabilitation Publication No. 738, Minneapolis, MN, with permission.)

If no adverse reaction:

Day 1: 5 repetitions of each exercise.
Day 2: 10 repetitions of each exercise.
Day 3 onwards: 20 repetitions of each exercise, then build up to 50–100 repetitions.

1. Begin in a relaxed position, lying on your back with knees bent and feet flat on the floor (Fig. 1.3).
2. Tighten the buttocks closely together.
3. Still holding the buttocks together, tighten and pull in the stomach.
4. Your low back should roll flat against the floor.
5. Hold for a slow count of 5, then relax.
6. Do *not* push with your feet.

1. Stand with your back touching a wall (Fig. 1.4).
2. Move your feet about 20–30 cm away from the wall, resting your back against the wall.
3. Squeeze your buttocks together and hold.
4. Now pull in your abdomen. The space between your back and the wall should have gone.
5. Holding your low back flat against the wall, gradually move your feet closer to the wall.
6. When you can stand straight with no curve, try walking around the room with your back in the same position, returning to the wall to double check your ability to maintain proper posture.

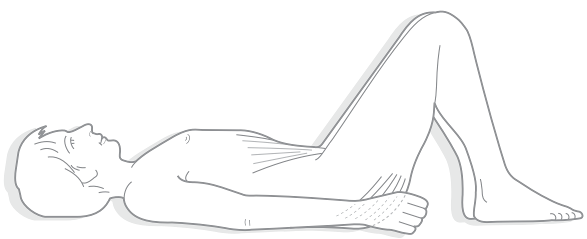

Figure 1.3

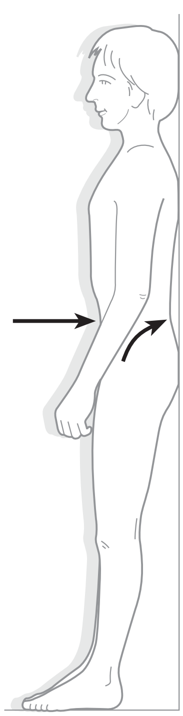

Figure 1.4

This exercise (Fig. 1.5) stretches and strengthens muscles of the back, abdomen, and legs and improves range of motion; it also promotes movement of nerve roots within the lower intervertebral foraminae.

1. Begin in a relaxed position, lying on your back with one knee bent.
2. Slowly raise the bent leg, straightening the leg as it moves upward and keeping the low back flat.
3. Raise the leg as far as possible without causing discomfort.
4. Slowly lower this leg, keeping the low back flat as the leg approaches the floor.
5. Relax a few seconds.
6. Repeat the motions, as outlined above, using the other leg.

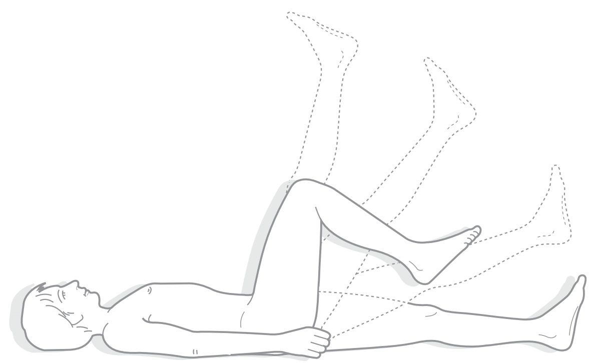

Figure 1.5

Be good to your back!

The human body has a remarkable ability to adapt to almost any form of activity if that activity is begun slowly and increased gradually over a period of time.

Recommended activities include:

1. Walking – on a daily basis, gradually increasing distance day-by-day.
2. Hiking – once walking is well established.
3. Swimming – it is with swimming that the paravertebral muscles can be gently exercised since the gravity-induced weight of the body is modified by the presence of water. Swimming is the closest that a low back pain patient can come to exercising in a gravity-free environment. Once again, frequent swimming, using gentle motion and progressively increasing effort and time is most desirable.

Case 2

L5–S1 posterior central intervertebral disc protrusion abutting adjacent nerve roots

COMMENT

Lumbosacral disc thinning and retrolisthesis of L5 on S1 is indicative of disc bulge or prolapse.

PROFILE

A 31-year-old male who worked in a manual capacity who is a non-smoker and only drinks alcohol socially.

PAST HISTORY

He had not experienced any unusual childhood illnesses or unusual adult illnesses. He said he had not had any significant falls and had never been unconscious. He had no surgical history. The first episode of low back pain that he ever experienced occurred about 10 years ago and was 'minor'; it lasted for approximately 7 days. That low back pain resulted from helping his father to lift a 'not too heavy' box from the back of a panel van. His father was a masseur, so gave him massage treatment over 2–3 days; he became asymptomatic within 7 days and went back to work without any low back pain. He said he subsequently passed a pre-employment medical examination before starting to work as a carpenter.

PRESENTING COMPLAINT(S) (Fig. 2.1)

He presented with constant low back pain that radiates to the left or right upper buttock region. At times, the pain radiates to the left buttock then to the thigh and calf posteriorly and he experiences a 'tingling' sensation in the left heel. Sometimes, but less frequently, he experiences pain radiating into the posterior aspect of the right thigh as far as the knee. After driving the car for approximately 1 hour the anterolateral part of his left thigh develops an 'aching pain'. There is no night pain. He awakens once or twice each night due to low back pain when he changes position. Coughing and sneezing do not aggravate his low back or leg pain although these actions did so when he injured his low back 10 months ago. Sometimes the pain radiates up the right side of the lumbar spine and he experiences an increase in low back pain on trying to walk up stairs. Arising from the seated position aggravates his low back pain; he finds it difficult to straighten his low back after bending forward.

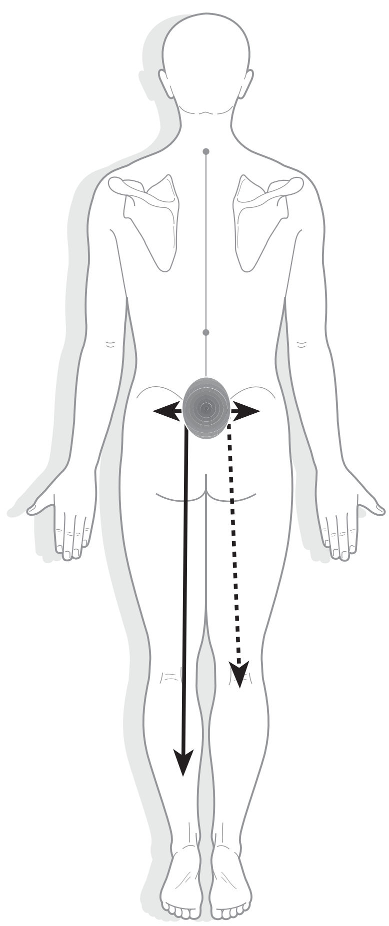

Figure 2.1

Non-steroidal anti-inflammatory drug medication and physiotherapy have not helped and no imaging had been performed. He had seen an orthopaedic specialist whose opinion was: '(1) False rotation gave equal pain as true rotation. (2) There were inappropriate signs and symptoms. (3) The best form of treatment would be for this man to resume his normal activities in the work force'.

AETIOLOGY

Ten months ago he lifted heavy and awkward shaped boxes that caused a gradual onset of low back pain at that time and, by next morning, his low back was 'extremely painful'.

EXAMINATION

In the erect posture, there was no obvious pelvic obliquity from a clinical point of view, nor was there any obvious scoliosis. Percussion of the spine elicited some low back pain (L4–S1 level). Deep palpation of the paraspinal muscles elicited pain at the L5–S1 level. Testing the sacroiliac joints did not cause any sacroiliac joint pain but he did feel pain on the left of the L5 vertebra. Toe walking power (S1) was normal as was heel walking power (L5), although the latter elicited some sciatica to extend into the left calf from the buttock.

The deep tendon reflexes in the upper and lower extremities were normal. Pinprick sensation of the lower extremities was normal apart from hypoaesthesia along the lateral side (S1) of each foot and below the heels (S1). Vibration sensation at the ankles was normal.

In the seated and slumped forward position, there was low back pain, and the addition of left straight leg raising (SLR) elicited an increase in low back and left leg pain posteriorly at a measured 45° elevation. Right SLR to 55° elicited similar low back pain.

Lumbar spine active ranges of movement were as shown below:

1. Flexion – with cautious movement his out-stretched fingers were able to go two-thirds of the way down his shins; further movement was then limited by increasing low back pain. He had difficulty in straightening his spine due to an increase in low back pain.
2. Extension was limited by approximately 10% due to low back pain.
3. Left rotation was limited by approximately 10% due to pain on the left side of the lumbosacral joint.
4. Right rotation was limited by approximately 10% due to pain on the right side of the lumbosacral joint.
5. Left lateral bending – his fingers were able to reach approximately 2 cm below his knee before limitation due to low back pain.
6. Right lateral bending – his fingers reached to approximately 10 cm below the right knee before limitation due to low back pain.

Power in the lower limbs was normal, as was the case with the foot pulses. On palpation the foot temperature appeared equal bilaterally. The circumference of the calf, 14 cm below the patella, was 40 cm bilaterally. The Milgram active bilateral SLR elicited low back pain at approximately 30° elevation of the legs from the examination table. Supine SLR was limited to a measured 60° (right) due to right leg pain posteriorly, and 65° (left) due to left thigh and calf pain posteriorly. Left SLR plus foot dorsiflexion elicited an increase in low back pain and left sciatica; right SLR plus foot dorsiflexion did not cause any additional pain. The Lasegue's sign for the left side elicited low back and left leg pain and the right side elicited slight right leg pain. Bilateral hip flexion elicited a significant increase in low back pain at approximately 110° elevation of the thighs from the examination table. The abdomen was normal on examination.

There were no physical signs of malingering. For example, there was no positive Hoover's sign. In addition, false rotation of the pelvis did not elicit any pain. Although left straight leg raising with foot dorsiflexion was painful, straight leg raising with plantar flexion of the foot was reported as not aggravating his low back pain or left leg sciatica.

IMAGING REVIEW

An imaging review was not possible, as no imaging had been performed.

CLINICAL IMPRESSION

A central L5–S1 disc prolapse.

WHAT ACTION SHOULD BE TAKEN?

Erect posture lumbar spine and pelvis plain film radiographs were performed (Fig. 2.2A). The radiologist noted that 'there is narrowing of the L5–S1 disc space' (Fig. 2.2B). In addition, note that there is a retrolisthesis of L5 on S1 due to the disc narrowing which is secondary to disc prolapse at L5–S1. A lumbar spine MRI scan (Fig. 2.2C and D) confirmed 'a central L5–S1 disc protrusion indenting the thecal sac anteriorly and abutting the proximal descending nerve roots. There is some narrowing of both L5 neural foraminae due to a combination of reduction in disc height and some bulging disc material extending into the inferior portion of both L5 neural foraminae'.

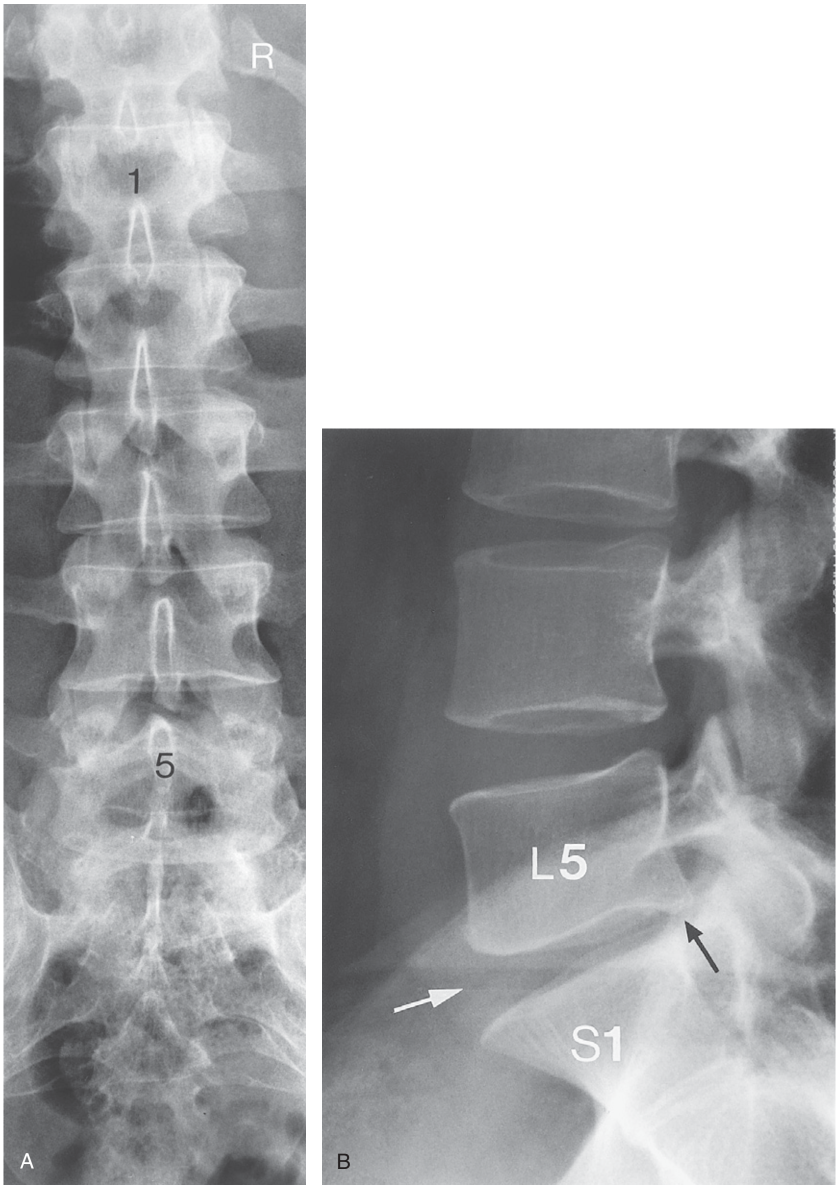

Figure 2.2 (A) Pelvis and lumbar spine anteroposterior (A-P) plain X-ray image (cropped and viewed from posterior to anterior). 1 = first lumbar vertebra, 5 = spinous process of fifth lumbar vertebra; R = right side of patient. (B) Lumbosacral spine lateral plain X-ray image. Note the L5–S1 disc height thinning (white arrow) as compared to the L4–5 disc space height above. Also, note the retrolisthesis (black arrow) of L5 on S1. L5 = fifth lumbar vertebra. S1 = first sacral segment.

(Continued)

DIAGNOSIS

L5–S1 posterior central intervertebral disc protrusion indenting the thecal sac and abutting the proximal descending nerve roots.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was grateful that his problem had been diagnosed and that he had not been arbitrarily dismissed as a malingerer. He decided to take a conservative treatment approach now that he fully understood his condition and he decided to perform light work only and said he would manage on analgesics as required. He would only contemplate surgery if his condition became intolerable.

This man managed to cope reasonably well on light duties and occasional analgesics in spite of his disability and impairment.

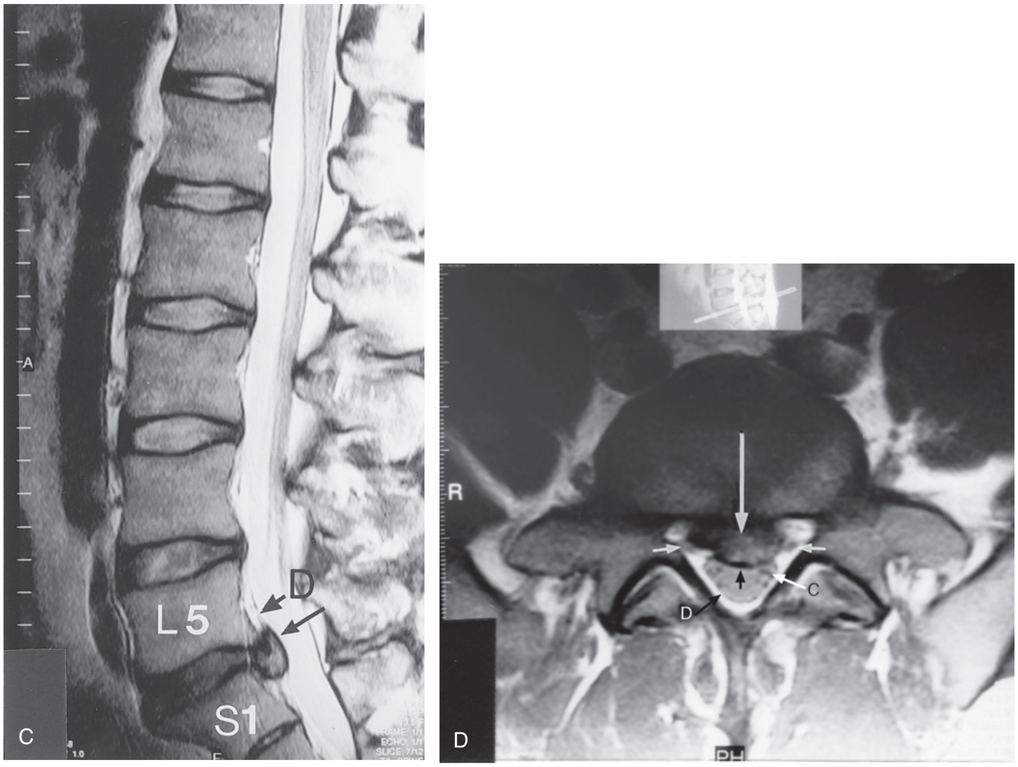

Figure 2.2 Cont'd (C) Lumbosacral spine MRI sagittal T2-weighted image showing the S1 = first sacral segment. The posterior disc protrusion at the L5–S1 level is shown by the black arrow; it can be seen compressing the anterior part of the dural tube (D). Note that the disc is becoming 'black' between L5 and S1 which indicates that it is undergoing dehydration (desiccation) as a result of injury. The L4–5 disc shows some early desiccation with essentially normal disc hydration at the levels above. (D) Lumbosacral MRI axial T2-weighted image at the L5–S1 level. The long arrow shows the degree of disc protrusion and the effect that it is having on the pain sensitive anterior surface of the dural tube (D) (small black arrow) and, to some extent, on the S1 nerve roots (small white arrows). C = cauda equina nerve roots; R = right side of patient.

KEY POINT

Retrolisthesis at L5–S1 is indicative of L5–S1 intervertebral disc prolapse (Giles et al 2006).

References

Giles L G F, Muller R, Winter G J 2006 Lumbosacral disc bulge or protrusion suggested by lateral lumbosacral plain X-ray film – Preliminary results. J Bone Joint Surgery 88-B: 450.

Further reading

Giles L G F 2000 Mechanisms of neurovascular compression within the spinal and intervertebral canals. Journal of Manipulative and Physiological Therapeutics 23: 107–111.

Postacchini F, Cinotti G 1999 Spinal fusion and disc prosthesis at primary surgery. In: Postacchini F (ed) Lumbar disc herniation. Springer-Verlag, New York, p 521–538.

Case 3

L5–S1 posterior left paracentral intervertebral disc protrusion

COMMENT

It is unwise not to promptly order at least erect posture pelvis and lumbar spine anteroposterior and lateral lumbosacral X-ray films for low back pain with radiculopathy; oblique views should also be considered. An appropriate and prompt diagnosis should be a clinician's goal – not a 'wait and see approach'.

PROFILE

A 33-year-old male of muscular build who is somewhat overweight.

PAST HISTORY

At approximately 18 years of age he developed some neck and upper thoracic spine pain due to lifting at work, and following X-rays it was concluded that he had aggravated an old neck injury. He had always been involved in sport such as touch football and indoor cricket up until injuring his low back 5 years ago.

Subsequently, i.e. approximately 4.5 years ago, he was shovelling sand and clay to cover an electrical cable when the bank of the trench collapsed. He was unable to prevent himself from falling across the trench; as he fell backwards across the trench his low back hit the opposite side of the trench. The trench was approximately 105 cm deep and 60 cm wide and he fell into it. He kept working for 2–3 hours, thinking that he had just 'strained' his low back. He sat and had lunch for approximately 20–30 minutes and, on getting up, he felt considerable low back pain.

PRESENTING COMPLAINT(S) (Fig. 3.1)

He presented with low back pain and pointed to the lumbosacral level, particularly the right side. In addition his low back pain can radiate to the left buttock, the posterior aspect of his left thigh and calf, then as far as his ankle. The left leg radicular pain began approximately 4.5 years ago, i.e. approximately 6 months after injuring his low back.

On the day of the accident, he went to his general medical practitioner who thought a 'muscle had been pulled'; the patient was told to take 2 days off from work and to use anti-inflammatory medication, then to return for a review. The pain 'subsided a lot' as a result of taking

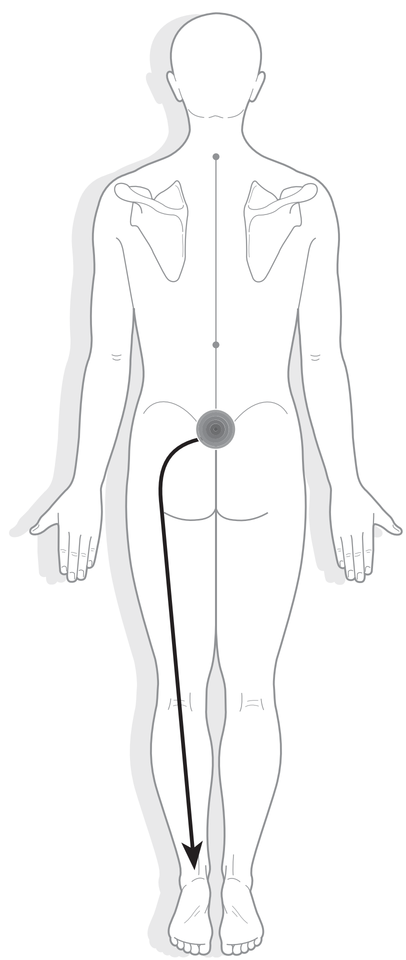

Figure 3.1

non-steroidal anti-inflammatory drugs, Panadeine Forte and Mersyndol.

He went back to work where there was some light lifting to be done but within a few minutes, the low back pain became 'intense'. Therefore, his general medical practitioner referred him to a physiotherapist for 2 weeks of treatment. He was given 20 minutes of traction daily which 'worked well' coupled with the anti-inflammatory medication. He believes he was off work for approximately 2 weeks. He returned to light duty work but had to terminate his employment approximately 2.5 years after the accident as there were no longer any light duty positions for him.

AETIOLOGY

He fell into a trench at work approximately 4.5 years ago.

EXAMINATION

In the erect posture his pelvis appeared to be approximately level without clinical evidence of pelvic obliquity. Percussion of the spine was painless and deep palpation of the paravertebral muscles did not elicit any pain. Lumbar spine active ranges of movement were as follows:

1. Flexion – limited by approximately 10% due to lumbosacral pain extending into the left leg posteriorly. He bent forward cautiously and slowly until the left leg pain (sciatica) occurred.
2. Extension – limited by approximately 10% due to low back pain.
3. Left lateral bending – full range but elicited pain on the right side of L5.
4. Right lateral bending – full range and painless.
5. Left and right rotation – normal range and painless.

The deep tendon reflexes in the legs were normal, as was the plantar response. Pinprick sensation of the legs and feet was normal. Left SLR in the seated and slumped forward position (slump test) caused left sciatic pain. No pain was felt on raising the right leg. Supine straight leg raising was limited to a measured 75° on the left due to pain in the left buttock and thigh posteriorly. Right SLR was unremarkable. Active SLR of both his legs, while lying in the supine position (Milgram test), caused considerable low back pain with some radiation into the lower abdomen. Bilateral hip flexion was of full range and painless but he felt low back pain on actively lowering his legs. In the supine position, flexion of the cervical spine did not cause any low back pain. Coughing caused low back pain. The pulses in his feet were normal and the temperature was equal in both the left and right feet. Power in the big toes, feet and legs was normal. The abdomen was normal on examination.

IMAGING REVIEW

No X-ray films or any other imaging films had been taken until approximately 4 years and 3 months following the injury when he was referred to an orthopaedic surgeon for continuing low back pain and left sciatica management; he was then sent for a supine lumbar spine plain film X-ray examination including anteroposterior, lateral and oblique views and told to continue with NSAIDs. The radiology report on these films read: 'There is some narrowing of the L4–5 and L5–S1 disc spaces; the remaining disc spaces appear normal. The facet joints and posterior elements appear normal'.

CLINICAL IMPRESSION

Central to left-sided disc protrusion at the L5–S1 level (or at the L4–5 level).

WHAT ACTION SHOULD BE TAKEN?

An erect posture pelvis and lumbar spine and a lateral lumbosacral spine X-ray view were performed (Fig. 3.2A and B). The former figure showed that the lumbar spine was antalgic above the L4 level with a 'lumbar curvature convex to the left'. As a diagnosis of L5–S1 disc protrusion was considered highly likely because of his history and symptoms, and the disc thinning with retrolisthesis of L5 on S1 noted on the lateral view (Fig. 3.2B), a CT scan was performed of the lower lumbar spine (L3–S1); this showed, at the L5–S1 level, 'a large left paracentral disc protrusion compressing the S1 nerve root' (Fig. 3.2C).

DIAGNOSIS

L5–S1 posterior left paracentral intervertebral disc protrusion with left S1 radiculopathy.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was very relieved to find that he had a genuine pathological condition causing his low back pain and left sciatica, having tried for approximately 4 years and 8 months to obtain a diagnosis. He decided to take a conservative approach to his problem by losing weight, exercising (i.e. going for long walks) and cutting down his anti-inflammatory medication. He said he did not want to consider a surgical approach and would let me know if the proposed conservative approach did not help him. He was given exercises

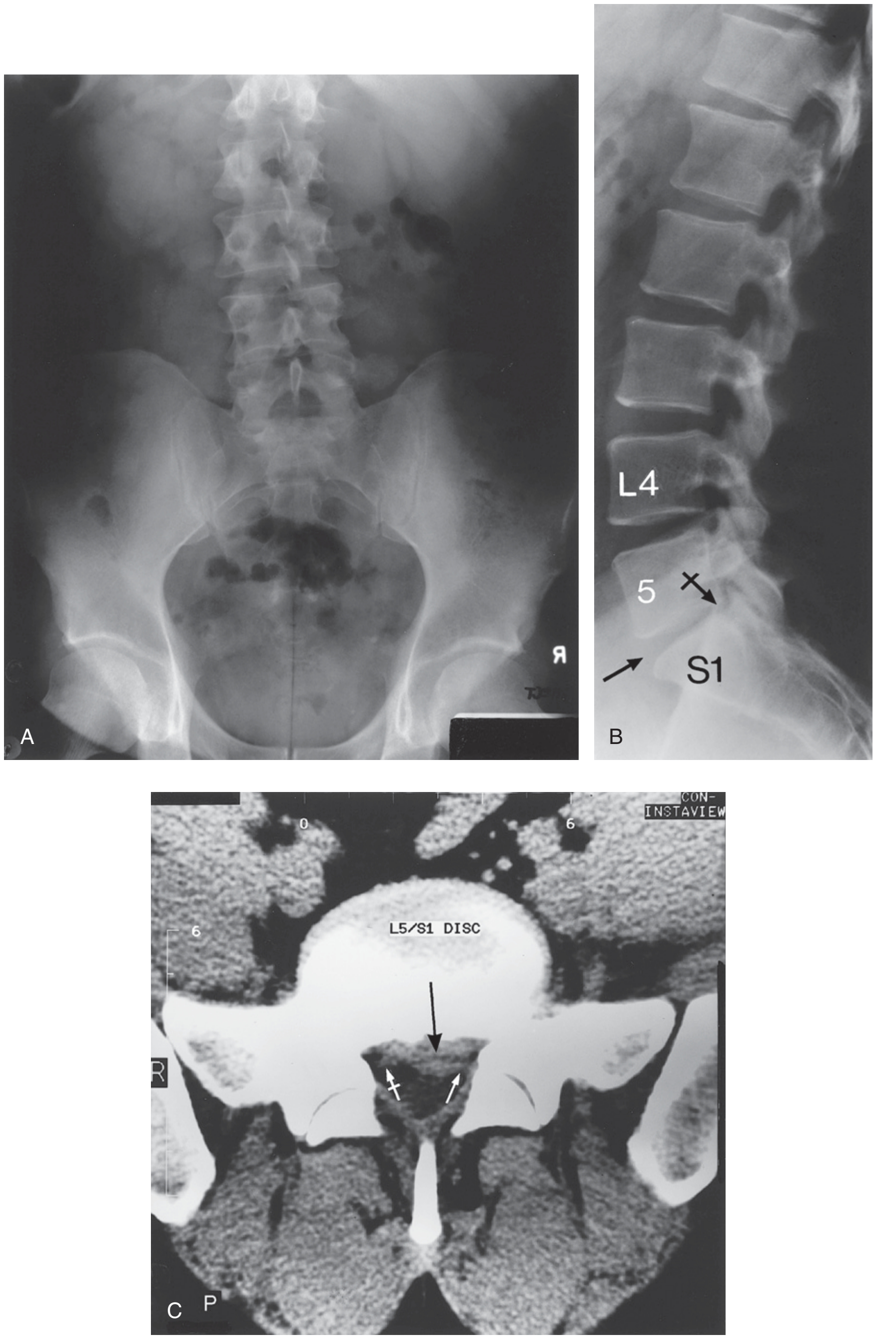

Figure 3.2 (A) Pelvis and lumbar spine anteroposterior plain X-ray image in the erect posture that shows some degree of antalgic scoliosis (lumbar curvature convex to the left). (B) Lumbar spine lateral plain X-ray image showing the fourth (L4) lumbar vertebra and the disc thinning at L5–S1 (black arrow), with retrolisthesis of L5 on S1 (tailed arrow). (C) Lumbar spine CT axial image at the L5–S1 level showing the large left paracentral disc protrusion (black arrow) compressing the left S1 nerve root (small white arrow). The small white-tailed arrow shows the right S1 nerve root which appears not to be compromised.

to perform to strengthen the abdominal, buttock and lumbar muscles (see Figs 1.3 and 1.4), as well as an exercise to mobilize the lower lumbar nerve roots (see Fig. 1.5) and he decided to find a light-duty occupation in which he was virtually self-employed so that he could accommodate his low back and left leg pain.

To date, some 9 years post-injury, he has managed to avoid surgery for his intervertebral disc prolapse by losing weight, going for long walks and performing exercises to strengthen the muscles mentioned above. The main reason for his cooperation was that a definitive diagnosis had been made and his condition clearly explained to him.

Box 3.1

	Protrusion location	Symptoms
1.	Central disc protrusion at L4–5 or L5–S1	Usually causes low back pain without leg symptoms if the nerve roots are not involved
2.	Central to one sided disc protrusion at L4–5 or L5–S1	Usually causes low back pain and leg symptoms on one side i.e. the side where there is root impingement
3.	Central protrusion at L4–5 or L5–S1 with bilateral nerve root involvement	Usually causes low back pain and bilateral leg symptoms
4.	Central to right sided protrusion at one level at L4–5, and central to left sided protrusion at L5–S1	Usually this causes bilateral leg symptoms if the left and right nerve roots, respectively, are involved. If only one root is involved, symptoms would be expected to occur on that side of involvement.

Note

The location of a disc protrusion and the side of radiculopathy symptoms depends on the location of the protrusion and its proximity to an adjacent nerve root as suggested in the summary in Box 3.1. It should be noted that sciatica can be caused by contralateral lumbar intervertebral disc protrusion, probably due to *traction* rather than direct compression (Sucu and Gelal 2006).

KEY POINT

When buttock and leg symptoms occur on the left and right sides in varying degrees, suspect a central intervertebral disc protrusion. If the symptoms are predominantly left sided, the disc protrusion will most likely be central to left sided, as in this case.

References

Sucu H K, Gelal F I 2006 Lumbar disk herniation with contralateral symptoms. *Euro* Spine J 15: 570–574.

Further reading

See Case 14.

Case 4

CT versus MRI for lumbar spine intervertebral disc protrusion

COMMENT

CT imaging is good for looking at bony structures but MR imaging is better for looking at soft tissues.

PROFILE

35-year-old male of average build who does not smoke and works as a self-employed delivery courier.

PAST HISTORY

At the age of 17 years he hurt his low back at work and had to take off two days from work; he then made a complete recovery.

Three months ago he lifted a 15 kg weight from above his shoulders and, as he turned round, he felt a sharp pain in his low back.

PRESENTING COMPLAINT(S) (Fig. 4.1)

Considerable low back pain that radiates to the back of both buttocks, thighs then to as far as just below the knees. He does not experience any night pain. Depending on his activities, he takes Voltaren and Panadeine Forte for pain.

AETIOLOGY

Lifting a heavy weight 3 months ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Deep palpation of the paraspinal muscles elicited pain in the lumbosacral muscles. Toe walking power (S1) and heel walking power (L5) were normal. Deep reflexes in the upper and lower extremities were normal. Vibration sensation at the ankles and elbows was normal. Pinprick sensation in the lower extremities was normal. The Valsalva manoeuvre (bearing down) caused a slight increase in low back pain.

When seated slumped forward he felt an increase in low back pain that was further aggravated by adding straight leg raising. Muscle power was normal. Supine straight leg raising was to a measured 30° (right) and 50° (left), with both aggravating his low back pain. The Lasegue's sign elicited low back pain. Straight leg raising with foot dorsiflexion elicited an increase in low back pain for the left and right sides. The Milgram active bilateral straight leg raise elicited some increase in low back pain.

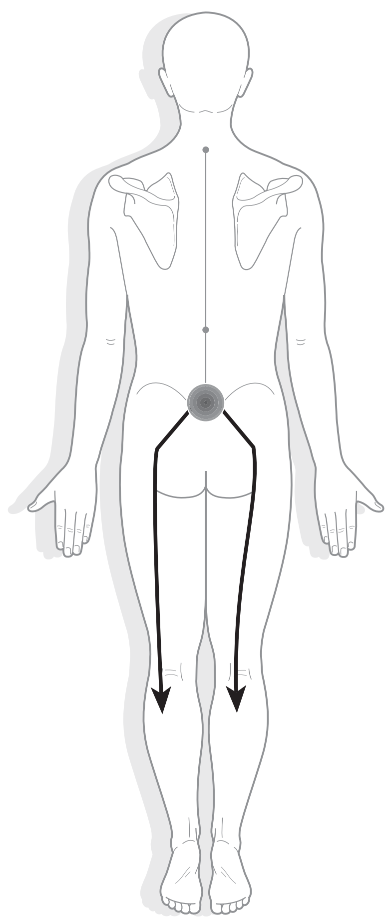

Figure 4.1

Active lumbar spine ranges of movement were as follows:

Flexion – was to his knees with aggravation of his low back pain.
Extension – was essentially of full range and only elicited slight low back pain.
Left and right lateral bending – his fingers reached his knees with slight low back pain.
Left and right rotation were of full range with only slight low back pain.

IMAGING REVIEW

Erect pelvis and lumbar spine plain X-ray (following his recent injury). The report stated: 'no significant bony or disc space abnormality is detected in the lumbar spine'. However, there is slight narrowing of the intervertebral disc height at L4–5 and L5–S1. This X-ray examination suggests that the spine is not one that has degenerative changes due to having been abused over the years as only two disc space heights are slightly narrowed (Fig. 4.2).

A CT scan of the lumbar spine was reported as showing: 'mild median disc protrusion at L4–5' level (Figs 4.3 and 4.4) with a 'small median disc protrusion at L5–S1' (Figs 4.3 and 4.5).

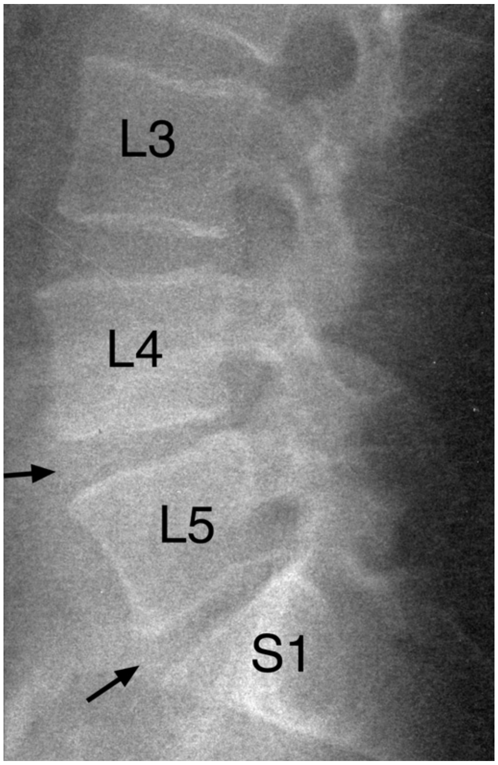

Figure 4.2 Lumbosacral spine lateral plain X-ray image. Note the slight disc space height narrowing at the L4–5 and L5–S1 levels (arrows) when compared with higher disc levels.

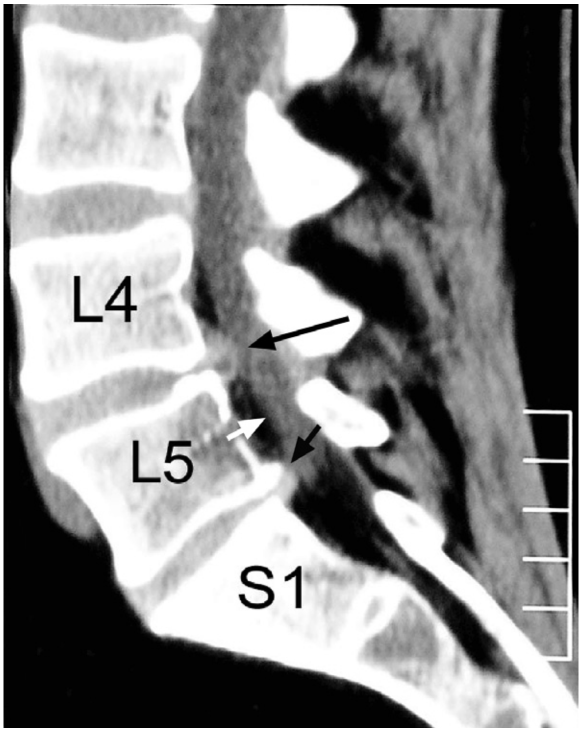

Figure 4.3 Lumbar spine CT sagittal image. Note: (i) the posterior disc protrusion at L4–5 level (long arrow), and (ii) the disc protrusion at L5–S1 (short arrow). Both the posterior disc protrusions indent the dural tube (white arrow) anteriorly.

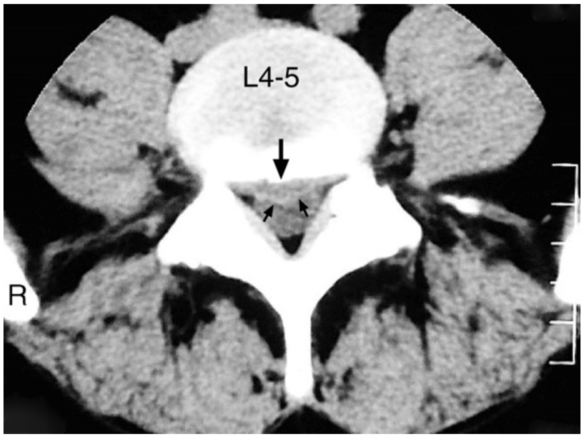

Figure 4.4 Lumbar spine CT axial image at the L4–5 level. Note: (i) the median disc protrusion (large black arrow) abutting the left and right nerve roots and indenting the dural tube anteriorly (small black arrows). R = right side of patient.

CLINICAL IMPRESSION

L4 and L5 posterior intervertebral disc protrusions abutting the adjacent nerve roots and the pain sensitive surface of the dural tube/thecal sac.

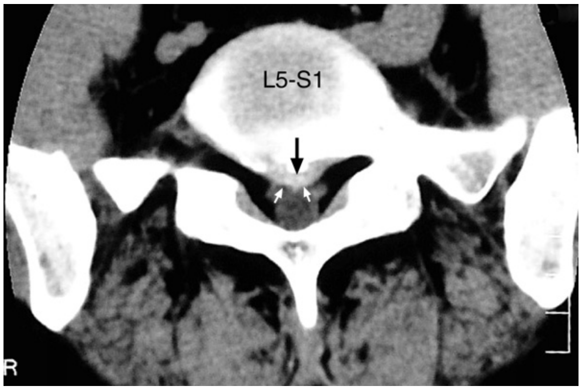

Figure 4.5 Lumbar spine CT axial image at the L5–S1 level. Note: (1) the median disc protrusion (black arrow) abutting the left, and particularly the right, nerve roots (small white arrows) and indenting the dural tube anteriorly. R = right side of patient.

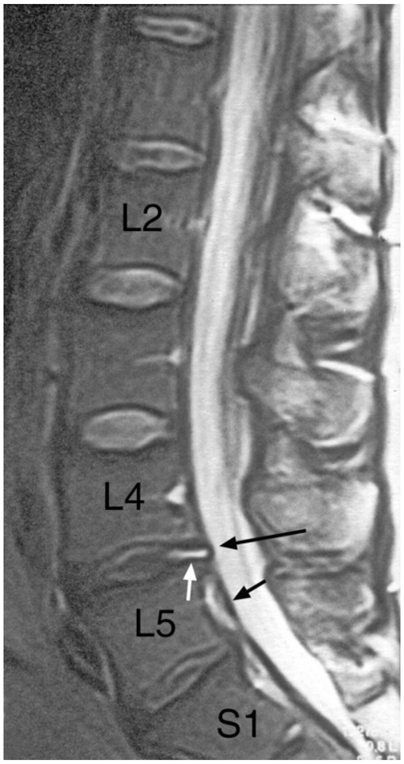

Figure 4.6 Lumbar spine MRI sagittal T2-weighted image. Note that: (i) At the L4–5 level there is a disc bulge (long black arrow), (ii) a tear in the posterior fibres of the intervertebral disc with a high intensity zone (white arrow), (iii) that the bulge impresses upon the pain sensitive anterior surface of the dural tube (small black arrow), and (iv) at the L5–S1 level there is a minor disc bulge with associated moderate desiccation.

WHAT ACTION SHOULD BE TAKEN?

In view of the above imaging, an MRI lumbar spine was requested to further evaluate the L4–5 and L5–S1 intervertebral disc protrusions (Fig. 4.6). The report stated: 'No intrinsic lesion of the conus is identified and a normal position is noted. There is a little loss of disc signal at L1-2 with loss of signal particularly noted at L4–5 and L5–S1. At the L3-4 level there is no significant disc bulge or focal disc protrusion. Normal appearance of the thecal sac and exiting foramina. At L4–5 level there is minor generalised disc bulge but no significant focal disc protrusion. No compromise of the exiting foramina'. In addition, it should be noted that at L4–5 there is a tear in the posterior fibres of the intervertebral disc with a high intensity zone and that the bulge impresses upon the pain sensitive anterior part of the thecal sac.

'At the L5–S1 level there is a minor disc bulge'.

DIAGNOSIS

- Chronic musculoligamentous soft tissue injury to the lower lumbosacral spine.
- L4–5 intervertebral disc bulge and tear with associated desiccation.
- L5–S1 intervertebral disc minor bulge with moderate desiccation.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. As he had tried chiropractic and physiotherapy treatment without relief he was advised not to lift heavy weights, to perform the exercises shown in Case 1 and to walk daily for exercise. He was told that, should his symptoms become worse due to him further injuring his L4–5 intervertebral disc, surgery may become necessary in the future, in an attempt to resolve his chronic low back pain syndrome. However, he was told that surgery has notoriously unpredictable outcomes. On review a couple of months later, he said he was able to control his symptoms by modifying his work loads, performing simple exercises, walking and occasionally taking paracetamol.

Note

A high signal intensity zone (HIZ) in the posterior aspect of a disc space (Fig. 4.6) represents nuclear material that has extended through a confluence of annular tears, leading to a radial fissure in the disc (Haldeman et al 2002).

KEY POINTS

1. This case clearly illustrates the advantage MRI has over CT when intervertebral disc structures are being investigated.
2. MRI has a great advantage, as it does not involve ionizing radiation.
3. When patients clearly understand their condition they are far more likely to comply with professional advice.

Reference

Haldeman S D, Kirkaldy-Willis W H, Bernard N Jr 2002 An atlas of back pain. Parthenon Publishing Group, London, p 27.

Case 5

L4 retrolisthesis with associated intervertebral disc protrusion

COMMENT

Retrolisthesis of L4 on L5 can be indicative of disc bulge or prolapse.

PROFILE

A fit 26-year-old male soldier who is a non-smoker and only drinks alcohol socially.

PAST HISTORY

Four-and-a-half years ago he was involved in a motorbike accident in which his motorbike skidded at a speed of approximately 65 kilometres per hour with the result that he was thrown clear of the bike. He immediately experienced low back pain that lessened in intensity but became chronic.

PRESENTING COMPLAINT(S) (Fig. 5.1)

Constant chronic low back pain of varying degree since an accident 4.5 years ago; the pain may spread across his low back. He points to the L4–5 level in particular and said this pain is aggravated by certain activities, sometimes trivial, such as bending forwards; such activities may cause a ‘shooting’ pain across his low back. He has no leg pain. His symptoms are aggravated by sitting for approximately 20 minutes, picking up grocery bags, washing dishes at the sink, coughing and bearing down. On arising of a morning he has low back stiffness and pain. Relieving factors are lying supine with a pillow under his knees, lying prone, or using a heat pack across his low back. Medication consists of Brufen or Panadol or Panadeine Forte, as required. In view of his low back injury he had had to change his occupation to light duty activities in the army.

AETIOLOGY

Motorbike accident 4.5 years ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic and lumbar spines elicited ‘tenderness’ at the L4–5 level. Deep palpation of the lumbar paraspinal muscles elicited pain in the L4–S1 region, particularly on the right. Sacroiliac joint strain testing elicited pain centrally at L4–5 but no sacroiliac joint

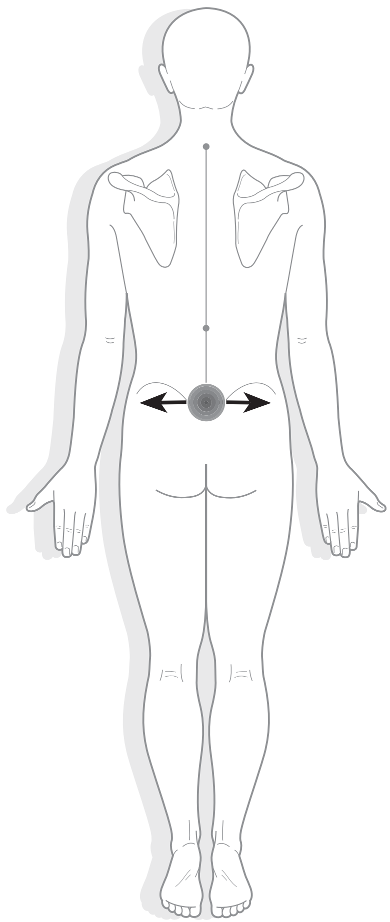

Figure 5.1

pain per se. Toe walking power (S1) and heel walking power (L5) were normal. The deep reflexes in the upper and lower extremities were normal apart from diminished knee jerks (L4) bilaterally. The plantar response was normal as was vibration sensation at the elbows and ankles. Pinprick sensation over the lower extremities appeared to be normal. Motor power in the lower extremities appeared to be normal. The foot pulses were normal and the temperature of the left and right feet appeared to be normal on comparative palpation.

The circumference of the calf (10 cm below the patella) was 36 cm bilaterally. The Valsalva manoeuvre (bearing down) caused an increase in pain across his low back. Supine straight leg raising caused an increase in low back pain at a measured 45° on the left and right sides. Straight leg raising with foot dorsiflexion for the left and right sides, respectively, caused an increase in low back pain but plantar flexion did not. Bilateral hip flexion did not aggravate his low back pain up to approximately 100° elevation of his thighs from the examination table but the addition of cervical spine flexion did aggravate his low back pain.

Lumbar spine active ranges of movement were as shown below:

1. Flexion – his fingers reached to approximately 5 cm above his knee and this caused an increase in low back pain although he said he could go further but with increasing pain.
2. Extension was limited by approximately 15% due to an increase in low back pain.
3. Left and right lateral bending – his fingers reached to approximately 5 cm above each knee, causing an increase in low back pain.
4. Left and right rotation were of full range and painless.

False rotation of the pelvis did not elicit any pain nor did downward pressure on his head.

IMAGING REVIEW

Lumbar spine plain film radiographs had not been performed before a time interval of 2 years 20 months had elapsed. The radiology report stated: 'The lumbar spine and sacroiliac joints are normal. No soft tissue abnormality'. However, note that there was a degree of retrolisthesis of the L4 vertebral body on the L5 vertebral body (Fig. 5.2) suggesting a posterior disc bulge/protrusion at this level.

CLINICAL IMPRESSION

Possible L4–5 intervertebral disc injury.

WHAT ACTION SHOULD BE TAKEN?

In view of the retrolisthesis of L4 on L5 a lumbar spine MRI was requested and this was reported as showing a 'prominent broad based central disc protrusion at L4–5 (Fig. 5.3A) that does not obviously cause neural compression' (Fig. 5.3B). Also, there is early atrophy of the multifidus muscles with fibro-fatty replacement of part of the muscles.

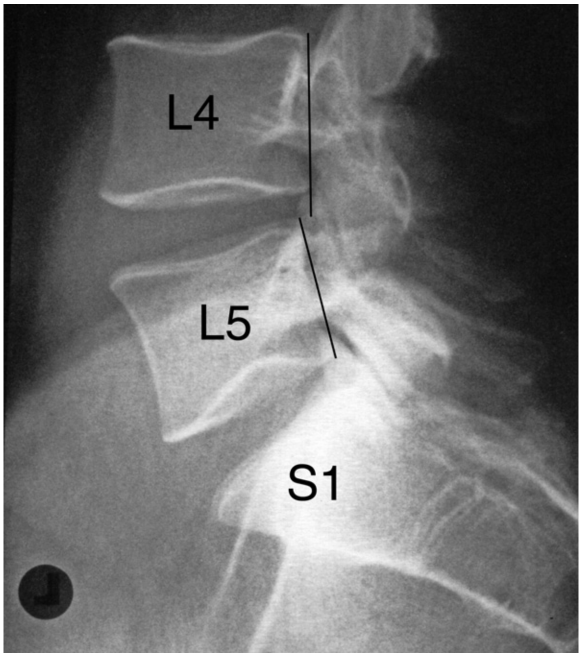

Figure 5.2 Lumbosacral spine lateral plain X-ray image that shows some retrolisthesis of the L4 vertebral body on the L5 vertebral body as indicated by the black lines drawn along the posterior margin of the L4 and L5 vertebral bodies, respectively. No vertebral body margin lipping is present. L4 = 4th lumbar vertebra; L5 = 5th lumbar vertebra; S1 = first sacral segment.

DIAGNOSIS

- Musculoligamentous soft tissue injuries of the lumbar spine.
- L4–5 posterior broad-based intervertebral disc protrusion.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. In particular he was shown the MR images illustrating the broad-based central disc protrusion at L4–5. He was advised (i) not to take part in any activities that would excessively load his lumbar spine, (ii) to undertake a daily exercise programme (see Case 1), (iii) to try to manage without any medication, and (iv) to keep fit and not gain weight, in order to minimize compressive forces across his L4–5 intervertebral disc. He was told that surgery was not necessary at this time and may not become necessary if he takes the abovementioned steps to protect his low back and does not further injure his low back.

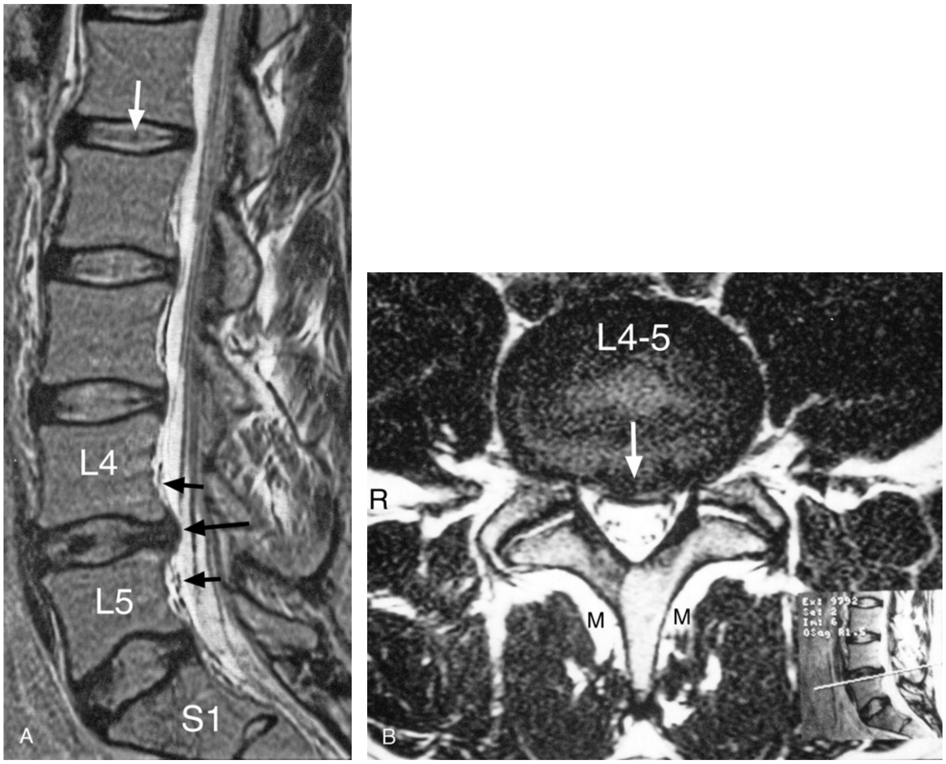

Figure 5.3 (A) Lumbar spine MRI sagittal T2-weighted image showing reduced signal intensity (blackening) of the L4–5 intervertebral disc, and to lesser extent of the L5–S1 disc, compared to the remaining discs. Also note the prominent disc protrusion (large black arrow) that is pressing upon the pain-sensitive anterior part of the dural tube (small arrows). Signal from the vertebral bodies is essentially normal. The intranuclear cleft (white arrow) is essentially normal in the four discs above the injured L4–5 disc level. (B) Lumbar spine MRI axial T2-weighted image through the L4–5 disc showing the prominent broad-based central disc protrusion (white arrow) compressing the pain sensitive anterior part of the dural tube. In addition, note the fibro-fatty replacement of part of the multifidus muscle (M).

He made satisfactory progress and was able to 'live with' his low back condition once he understood what the condition was and how it could be dealt with on a very conservative basis.

Note

A significant body of research shows that atrophy (degeneration) seen in the multifidus muscle in people with low back dysfunction is representative of a form of impaired motor control (Jemmett 2003) to the muscle and that patients with lumbar disc herniation show structural changes in the multifidus muscle at the involved level (Yoshihara et al 2003) due to nerve root impairment (Yoshihara et al 2001).

KEY POINT

Note the value of a correctly positioned lateral lumbosacral spine X-ray view in suggesting a level of intervertebral disc injury.

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Yoshihara K, Shirai Y, Nakayama Y et al 2001 Histochemical changes in the multifidus muscle in patients with lumbar intervertebral disc herniation. Spine 26: 622–626.

Yoshihara K, Nakayama Y, Fujii N et al 2003 Atrophy of the multifidus muscle in patients with lumbar disk herniation: histochemical and electromyographic study. Orthopedics 26: 493–495.

Further reading

Yoshihara K, Nakayama Y, Fujii N et al 2003 Atrophy of the multifidus muscle in patients with lumbar disc herniation: histochemical and electromyographic study. Orthopedics 26: 493–495.

Case 6

Carcinoma of the pancreas

COMMENT

Always listen to the patient!

PROFILE

A 57-year-old married female who has a sedentary occupation, smokes cigarettes but does not drink alcohol was referred by her medical practitioner for evaluation and treatment.

PAST HISTORY

There is nothing contributory.

PRESENTING COMPLAINT(S) (Fig. 6.1)

Constant mild to severe low back pain of 1 year duration for which she had seen several general medical practitioners. When the low back pain is severe, it sometimes radiates upwards to the mid-thoracic spine and her legs 'feel weak'. She required two analgesic injections in the week prior to consultation as the low back pain was so severe. There is no night pain other than the constant low back pain.

Physiotherapy treatment provided limited help and a non-steroidal anti-inflammatory drug was of no help, but caused constipation. She then used another NSAID as required; this eased the pain but caused gastric upsets, therefore she ceased that medication. She takes four to six Panadol tablets per day. She is post-menopausal and experiences some 'sweating' or 'cold spells' and, though there was some minor weight loss, a pelvic examination and pap smear test 9 months before this consultation were normal.

She had been referred to a psychologist because 'there are certainly many psychosocial issues exacerbating this lady's pain'. She said: 'I am told the low back pain is in my head – but it is not!'

AETIOLOGY

Unknown, but she periodically lifts approximately 35 kg weights at work.

EXAMINATION

On examination, deep palpation of the lumbar paraspinal muscles indicated that she was tender over the entire lumbar spine. Active lumbar spine flexion, extension and rotation aggravated her low back pain. Supine SLR was to 90° bilaterally and painless, indicating no nerve root impingement. Muscle power was normal. The deep tendon reflexes at the knees and ankles were normal, as was the case with pinprick sensation of the lower limbs. The abdomen appeared to be normal on examination.

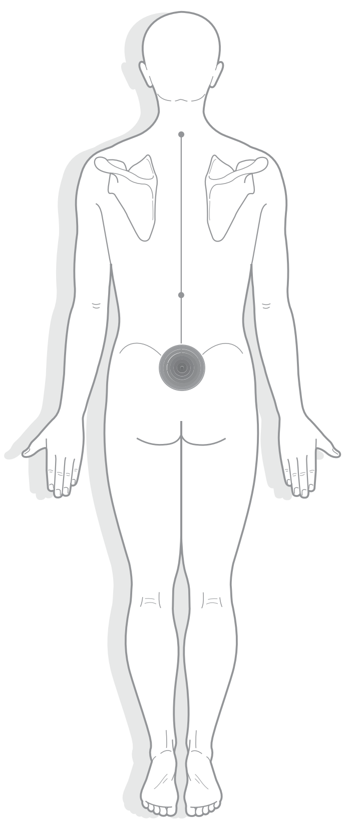

Figure 6.1

IMAGING REVIEW

Plain lumbosacral spine radiographs showed some thinning of the L3–4 disc space with some anterior lipping at the disc–vertebral body margins (Fig. 6.2A and B). The left and right oblique view films showed some osteoarthrotic changes of the L4–5 and L5–S1 zygapophysial joint facets (Fig. 6.2C and D). A lumbar spine CT scan performed from L3 to S1 levels did not show any spinal canal lesions. An ultrasound scan of the pelvis and abdomen showed three haemangiomas on the liver.

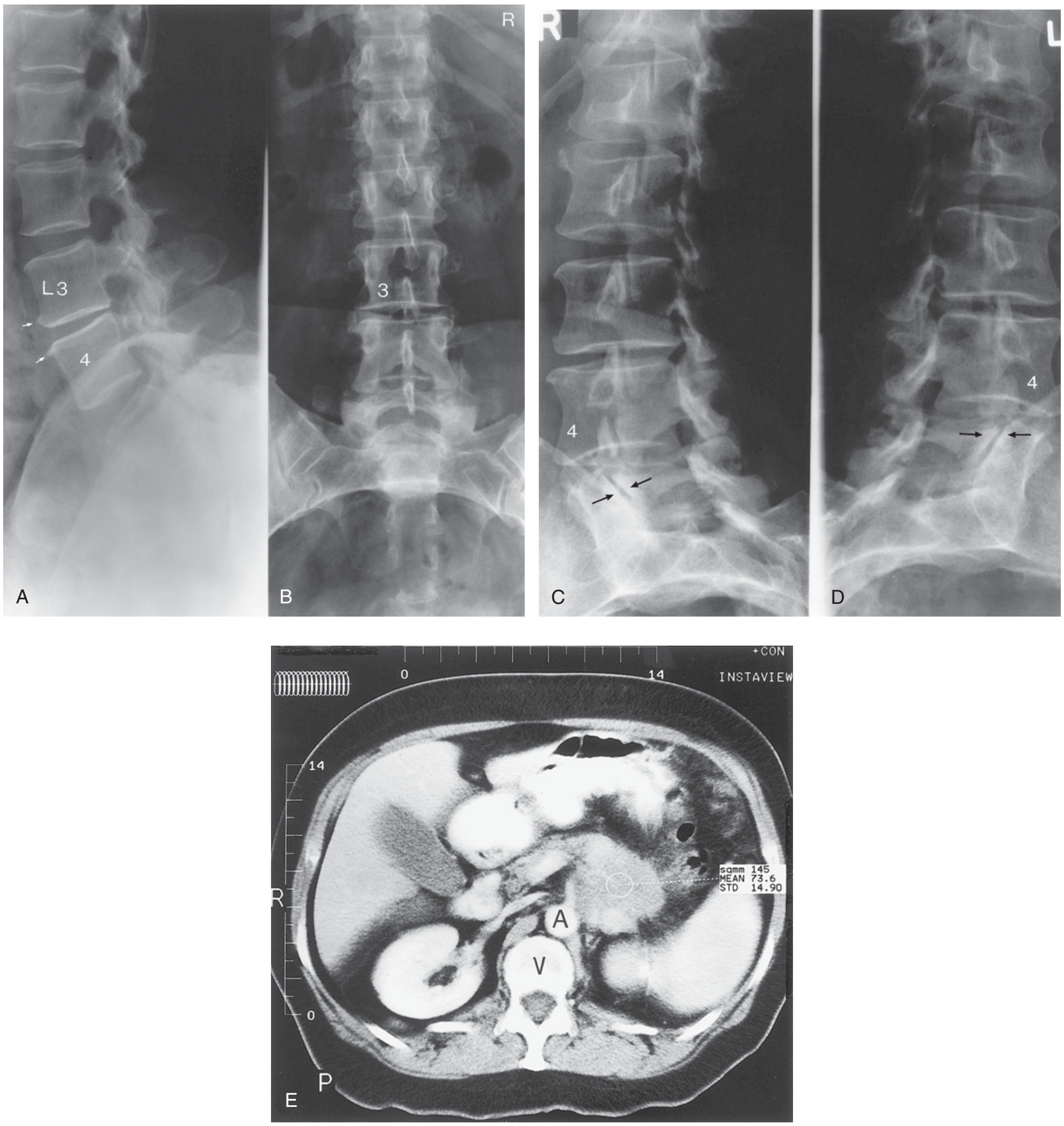

Figure 6.2 (A–D) The plain X-ray films were taken in the erect posture. The lateral lumbar spine image (A) shows disc thinning at the L3–4 level with anterior lipping (small white arrows) of the vertebral bodies adjacent to the intervertebral disc space. (C) and (D) represent the right and left oblique images and show zygapophysial joint facet early osteoarthrosis (black arrows) at the L5–S1 level with some imbrication (subluxation) of the opposing facet surfaces at the L3–4, L4–5 and L5–S1 levels. (R = right side of patient.) (E) CT axial image through the upper abdomen, with contrast, showing the pancreatic tumour (with a circle placed over part of it), the adjacent proximal aorta (A) and vertebral body (V). (R = right side of patient.)

CLINICAL IMPRESSION

Referred pain from a visceral organ as the deep tendon reflexes and pinprick sensation were normal in the lower limbs and supine SLR was to 90° bilaterally and painless and the CT lumbar spine scan did not show any spinal lesions. In addition, the ultrasound scan had shown what appeared to be haemangiomas on the liver. The unexplained minor weight loss was of concern while the 'sweating' and 'cold spells' suggested sympathetic nervous system involvement.

WHAT ACTION SHOULD BE TAKEN?

A bone scan was requested but the result was normal. Numerous laboratory tests were performed, as shown in Boxes 6.1 and 6.2.

In view of the slightly elevated C-reactive protein and ESR, a CT scan of the upper abdomen was performed with oral contrast and with and without intravenous contrast using a spiral technique. This showed a 5-cm pancreatic tail carcinoma encasing adjacent arteries and veins (Fig. 6.2E).

Box 6.1 Chemical pathology

		Units	Reference range
Sodium	140	mmol/l	(135–145)
Potassium	3.7	mmol/l	(3.2–4.5)
Chloride	107	mmol/l	(100–110)
Bicarbonate	24	mmol/l	(22–33)
Anion gap	9	mmol/l	(8–17)
Urea	4.7	mmol/l	(3.0–8.0)
Creatinine	0.07	mmol/l	(0.05–0.10)
AST (Aspartate aminotransferase)	19	U/l	(<35)
Protein (total)	70	g/l	(62–83)
Albumin	43	g/l	(33–47)
Globulin	27	g/l	
ALP (alkaline phosphatase)	80	U/l	(40–120)
Gamma GT	22	U/l	(<50)
ALT (alanine aminotransferase)	8	U/l	(<40)
Bilirubin (total)	14	μmol/l	(<20)
C-reactive protein	**14**	mg/l	(<6)
Tumour marker (carcinoembryonic antigen)	3.7	μg/l	(<5)

Box 6.2 Haematology

		Units	Reference range
Haemoglobin	135	g/l	(115–160)
White cell count	6.9	$\times 10^9$/l	(4.0–11.0)
Platelets	216	$\times 10^9$/l	(140–400)
Haematocrit	0.41		(0.39–0.52)
Red cell count	4.63	$\times 10^{12}$/l	(3.80–5.20)
MCV (mean corpuscular volume)	88.1	fL	(80.0–98.0)
Neutrophils	3.6	$\times 10^9$/l	(2.0–8.0)
Lymphocytes	2.6	$\times 10^9$/l	(1.0–4.0)
Monocytes	0.5	$\times 10^9$/l	(0.1–0.8)
Eosinophils	0.1	$\times 10^9$/l	(<0.2)
Basophils	0.0	$\times 10^9$/l	(<0.2)
ESR (erythrocyte sedimentation rate)	**23**	mm/hour	(<15)

DIAGNOSIS

Carcinoma of the pancreas.

TREATMENT AND RESULT

When the patient was told of her condition she sadly said 'so it is not in my head!'.

The patient underwent surgical removal of the main tumour and she received good symptomatic benefit from coeliac plexus blocks. This, in combination with MS Contin and paracetamol, resulted in her pain being 'very slight', although she became constipated and required lactulose for this problem.

It is interesting to note that the tumour marker, carcinoembryonic antigent, did not reach the positive range (5.1) until approximately 3 months after the diagnosis was made; she passed away 1 month after this test became positive.

KEY POINTS

1. The patient's low back pain was remote from the serious pathology.
2. The tumour marker test was normal until approximately 3 months after the diagnosis was made and did not reveal a serious tumour.
3. The combination of somewhat elevated CRP and ESR confirmed the presence of an inflammatory process but, of course, did not define the pathology involved.
4. Always take spinal pain patients seriously and never consider them to be malingerers unless you are sure of your facts.

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Case 7

Sacroiliac joint dysfunction

COMMENT

Clinicians treating low back pain syndromes should be appropriately trained in the diagnosis and management of such conditions.

PROFILE

A 25-year-old married housewife with three young children was referred by her general medical practitioner for evaluation and treatment.

PAST HISTORY

Her surgical history was that of an appendectomy 7 years ago and two Caesarean sections; otherwise she had been healthy.

Four years ago she fell onto her right buttock in particular and this caused her central to right-sided low back pain syndrome.

PRESENTING COMPLAINT(S) (Fig. 7.1)

Constant chronic low back pain following a fall 4 years ago, that is central to right sided, including the sacroiliac joint. This pain radiates into the right buttock, the right leg posteriorly and into the sole of the right foot. The pain prevents her from playing with her young children and undertaking normal home duties because of constant aggravation of the chronic low back pain syndrome. She said her appetite is reduced and that she has inexplicably lost 5 kg in weight during the last few weeks. Domestic relations are becoming strained because she feels her husband is 'sick and tired of my pain' and he asks 'why can't doctors fix it?'. At presentation she was tearful and anxious. She moved slowly and stiffly.

Extensive neurological investigations had apparently found no organic cause for the 'intolerable pain'. She was taking OxyContin (20 mg, 1–2 per day) and Endep (150 mg at night). She had tried various non-steroidal anti-inflammatory drugs, paracetamol, diazepam, and pethidine injections without relief.

She sleeps with a pillow between her knees in an attempt to get some relief from the right-sided low back pain. There is no night pain other than her constant right-sided low back pain. Coughing and sneezing cause an increase in the low back pain.

She had been investigated with a CT myelogram (Fig. 7.2A) and a lumbar MRI study. A steroid and anaesthetic injection into the right sacroiliac joint had not provided any benefit (Fig. 7.2B).

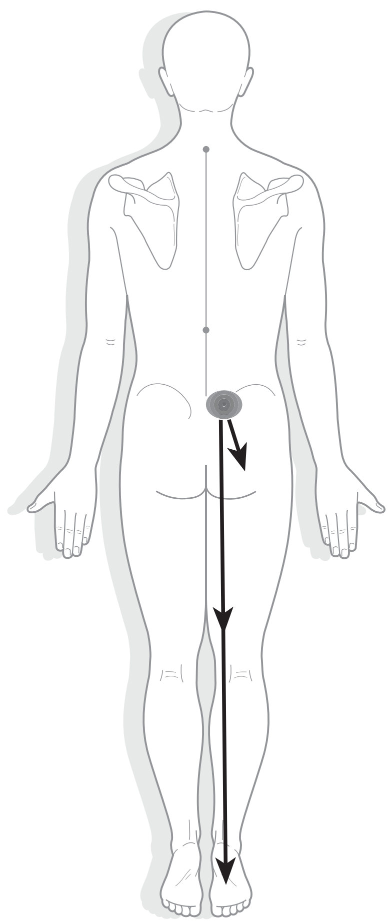

Figure 7.1

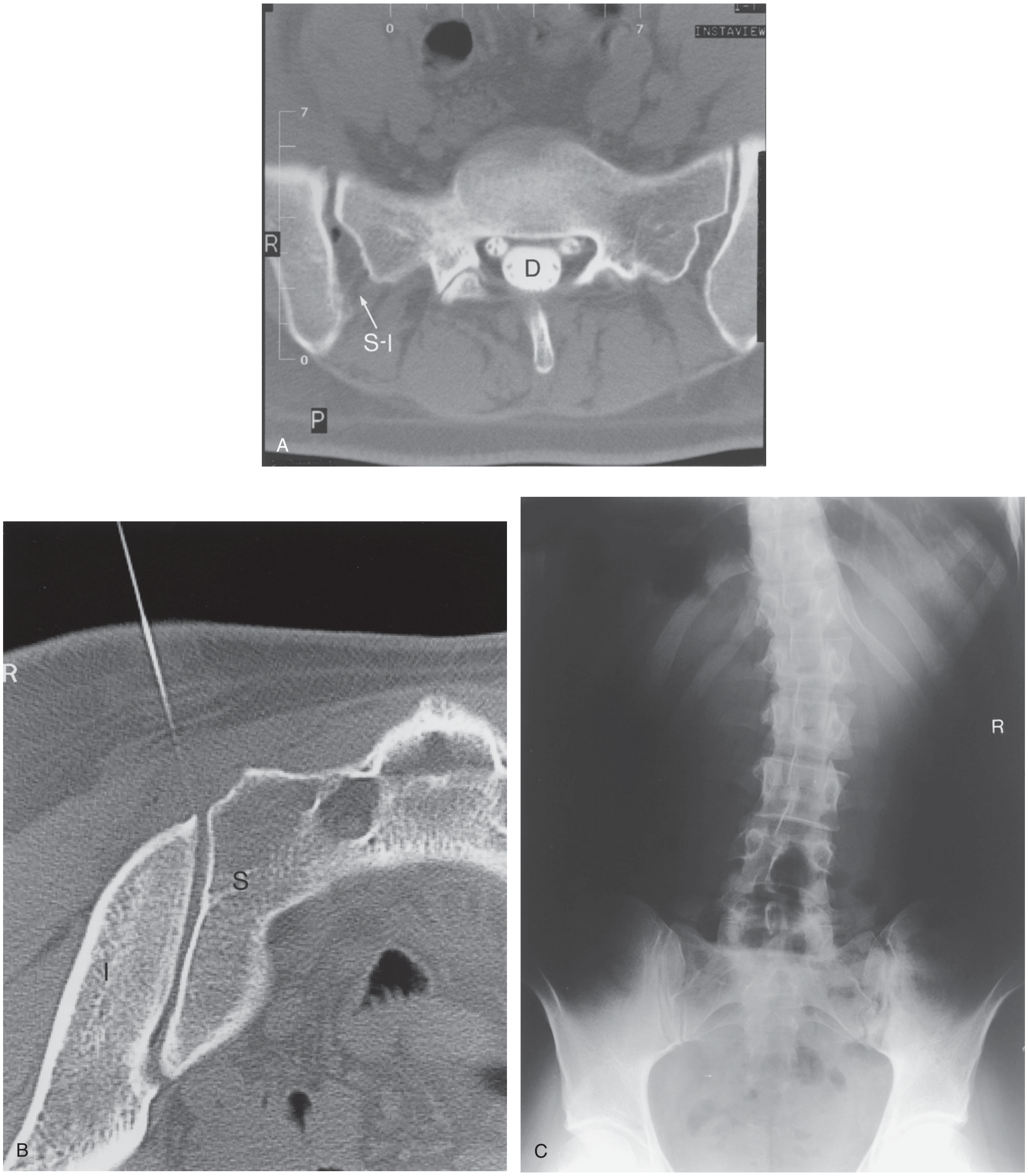

Figure 7.2 (A) CT myelogram axial image through the lower region of the lumbosacral zygapophysial joints and through the corresponding sacroiliac joint (SI) level which is above the synovial part of the sacroiliac joint. D = dural tube containing contrast material and some cauda equina nerve roots (grey dots) with adjacent nerve roots in the dural sleeve on the left and right sides, respectively. (B) CT image of the right sacroiliac joint showing needle placement for injection of the joint. I = ilium; S = sacrum. (C) Erect posture anteroposterior plain X-ray image (viewed from behind) showing the sacroiliac joints and the mild idiopathic scoliosis of the lumbar spine. R = patient's right side.

She was dismayed at having been told that the pain was 'psychological' and her husband was distressed by her pain and incapacity.

AETIOLOGY

A fall 4 years ago.

EXAMINATION

In the erect posture there was some clinical evidence of an idiopathic lumbar scoliosis, convex to the right side. Toe walking (S1) and heel walking (L5) power were normal. Erect posture straining of the left and right sacroiliac joints, respectively, elicited a significant increase in right sacroiliac joint pain. Active lumbar spine ranges of movement were all limited due to significant pain on the right side of the lumbosacral joint and over the right sacroiliac joint. The deep tendon reflexes at the knees and ankles were normal. Pinprick sensation of the lower extremities was normal, as was vibration sensation. Sitting in the slumped forward position aggravated the pain on the right of L5–S1 and over the right sacroiliac joint; the addition of right straight leg raising elicited an aggravation of this pain. Supine SLR on the right was limited to approximately 25° elevation due to similar pain. The Valsalva manoeuvre elicited a significant increase in her pain. The abdomen was normal on examination.

IMAGING REVIEW

Supine plain film radiographs showed a minor right convex thoracolumbar junction scoliosis. The CT lower lumbar myelogram and the lumbar spine MRI were normal.

CLINICAL IMPRESSION

Chronic right sacroiliac joint strain/subluxation in view of the history and normal neurology, in spite of the positive SLR on the right side.

WHAT ACTION SHOULD BE TAKEN?

An erect posture pelvis and lumbar spine radiograph (Fig. 7.2C) was taken to evaluate the degree of idiopathic scoliosis and to complement existing supine radiographs. Although the diagnosis of sacroiliac joint strain was made, because of the history of the right sacroiliac joint injection having not provided any relief and because her condition was so acute, it was considered prudent to perform a bone scan (reported as normal) and a full blood count as well as ESR and C-reactive protein tests; all the results were within normal limits.

DIAGNOSIS

Right sacroiliac joint strain causing dysfunction of the joint.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. It was considered safe to manipulate the sacroiliac joint, although she found it intolerably painful to be positioned for the manipulation. In spite of her pain, the sacroiliac joint moved easily and an audible 'release' was heard. The patient found the manipulation to be very painful but said she felt better on getting off the manipulating table. She was advised to speak to her referring family doctor about stopping all narcotics but advised to continue with the non-steroidal anti-inflammatory drug until her re-assessment. She was told not to lift the young children or perform any housework or shopping before returning for a re-assessment 3 days later.

At the following visit 3 days later it was gratifying to see the cheerful 'grin' on her face and she stated that she was very much better. A follow-up manipulation resulted in a slight audible 'release' and she was advised to return if her symptoms persisted. Both she and her husband were delighted at the result. The patient's husband asked why several specialists to whom she had been referred had suggested her symptoms were in her head and stated that this had almost wrecked their marriage.

The patient did stop taking OxyContin and Endep medication following the first sacroiliac joint manipulation.

This case is a good example of multidisciplinary co-operation leading to a satisfactory outcome for the patient.

The patient returned voluntarily 1 month later for a minor recurrence of right sacroiliac joint pain due to turning over in bed. Erect posture straining test for the sacroiliac joint caused pain over the right sacroiliac joint, so the joint was manipulated once more. She returned again approximately 2 months later with a further minor recurrence of symptoms in the right sacroiliac joint and one manipulation again provided relief. She was advised to return should symptoms recur, which she did on one occasion some 5 months later.

KEY POINTS

1. Sacroiliac joint stress tests can localize pain to a particular sacroiliac joint.
2. No improvement in sacroiliac joint pain following a steroid and anaesthetic injection into the joint does not mean that the joint is not the site of pain.

Further reading

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Case 8

Sacroiliac joint dysfunction and perineal pain

COMMENT

Clinical awareness of the association between sacroiliac joint dysfunction and possible perineal pain is essential.

PROFILE

A 60-year-old fit male of average weight who does not smoke cigarettes or drink alcohol and who works in a light duties occupation.

PAST HISTORY

Intermittent episodes of low back pain over the last 30 years.

PRESENTING COMPLAINT(S) (Fig. 8.1)

Perineal pain of insidious onset approximately two months ago. The pain is not aggravated by coughing, sneezing or bearing down. There is no night pain. A prostate gland examination had been performed and was found to be normal. The patient had been trialled on a non-steroidal anti-inflammatory medication that had only provided temporary relief from the perineal pain.

AETIOLOGY

There is no known aetiology.

EXAMINATION

In the erect posture there was no clinical evidence of leg length inequality or of postural scoliosis. Deep palpation of the paraspinal muscles did not elicit any local pain. Deep tendon reflexes in the lower extremities were normal as was the case with pinprick sensation. The plantar response was normal. Muscle power and tone were normal. Active lumbar spine ranges of movement were of full range and painless. Straight leg raising was limited to approximately 70° for the left and right sides due to hamstring tightness but not due to low back pain. Straining the sacroiliac joints elicited slight pain in the left sacroiliac joint.

IMAGING REVIEW

Erect posture radiographs of the pelvis and lumbar spine indicated equal leg lengths and iliac crest heights. There was slight sacral base obliquity (lower on the left) due to

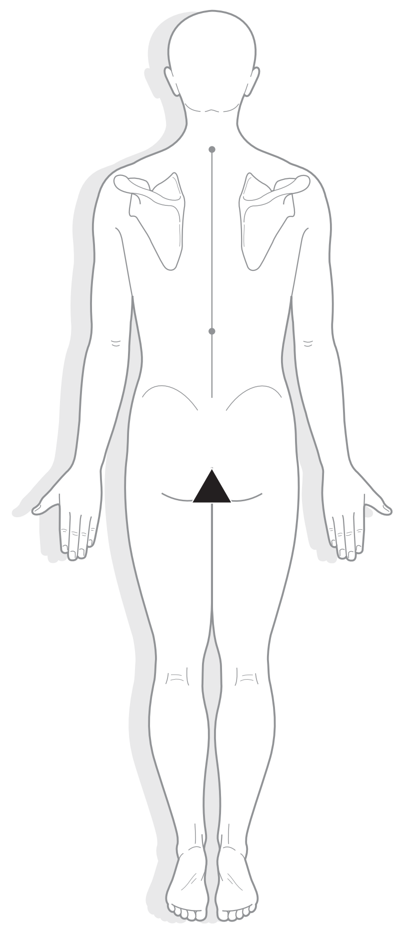

Figure 8.1

unequal development of the two halves of the sacrum (hypoplasia of the left side of the sacrum) with a slightly smaller left sacroiliac joint from superior to inferior.

CLINICAL IMPRESSION

Left sacroiliac joint dysfunction.

WHAT ACTION SHOULD BE TAKEN?

In view of the insidious onset of perineal pain, and as a precaution, he was referred to a urologist for further evaluation. The patient was referred by the urologist for micro-urinalysis, Prostate Specific Antigen (PSA), and an ultrasound of the urinary tract. The results indicated:

1. Micro-urinalysis was sterile and clear.
2. PSA – normal at 1.3 µg/L. Ref range = 0–4.5 µg/L.
3. Full blood count – normal.
4. Liver function test – normal.
5. Ultrasound – normal kidneys. Prostate volume of 40 cc. Normal bladder both ultrasonically and with respect to residual volume.

The urologist concurred that the perineal pain was, in all likelihood, referred from a left sacroiliac joint dysfunction.

DIAGNOSIS

Left sacroiliac joint dysfunction with referral of pain to the perineal region.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. A manipulation aimed at mobilizing the left sacroiliac joint was performed and the patient was seen the following day at which time his perineal pain had essentially gone. He was advised to walk as frequently as possible in order to maintain mobility of the sacroiliac joint before returning for re-evaluation two weeks later. At review, the patient's symptoms had resolved.

Note

Sacroiliac joint syndrome pain is felt over the sacral sulcus and in the region of the posterior iliac spine, often referring to the groin, buttocks, and posterior thigh and less often to the lower limb (Quon et al 1999).

The pudendal nerve is derived from the anterior rami of spinal nerves S2 to S4 and is the main nerve of the perineum and conveys the majority of sensory, sympathetic, and somatic motor fibres to the skin and muscles of the perineum (Moore & Dalley 2006). The medial divisions of the S1 to S4 nerves pass medially to the multifidus muscle while the lateral divisions pass between the interosseous and overlying posterior sacroiliac ligaments which also receive several fine filaments from the lateral divisions of the L5 and S1 to S3 nerves (Bradley 1974). The anterior sacroiliac ligament is innervated by ventral branches from the sacral plexus (S1–4) (Rickenbacher et al 1985).

In summary, all nerves adjacent to the sacroiliac joint supply small branches to the joint capsule (Giles & Crawford 1997) with the ventral branches coming largely from the sacral plexus; the inferior parts of the joint are supplied by a branch of the superior gluteal nerve (L4–S1 ventral rami); posteriorly, the joint receives branches from the dorsal rami of S1 and S2 (Rickenbacher et al 1985).

Thus, dysfunction of the sacroiliac joint may result in pain being referred to the perineum.

KEY POINT

Having ascertained that there is no overt pathology causing perineal pain, manipulation of a sacroiliac joint may be helpful when the clinical examination elicits pain in a sacroiliac joint.

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Case 9

Ewing's sarcoma

COMMENT

Prompt and accurate diagnosis, initially using plain X-ray films, is imperative, bearing in mind serious pathological conditions that may mimic 'benign' low back pain. To 'wait for 6 weeks before X-raying' can be a dangerous practice.

PROFILE

A 45-year-old non-smoker sedentary worker who only drinks alcohol socially.

PAST HISTORY

There was no history of trauma and he said he was very fit.

PRESENTING COMPLAINT(S) (Fig. 9.1)

He presented with left buttock/hip pain and some thigh pain that has been present for 3–4 weeks. There is no involvement of other joints. Active lumbar spine flexion causes an increase in his pain. The bowel and bladder function normally and there is no weight loss. There are no neurological symptoms and the pain does not radiate to the groin. There is no night pain.

AETIOLOGY

Unknown.

EXAMINATION

On examination he had a slight left-sided antalgic gait. He was able to touch his toes but with an increase in low back pain. The paraspinal muscles were not tender on deep palpation. Left hip joint ranges of movement were as follows – flexion to 50° with pain, abduction to 5° with pain, adduction to 20° without pain and internal rotation was limited by 50%.

He was tender to deep muscle palpation over the greater trochanter and the gluteus medius muscle. Neurologically, he was intact in the lower limbs. The abdomen was not tender and had no masses on palpation. There was a left leg length deficiency of approximately 0.5 cm on clinical estimation.

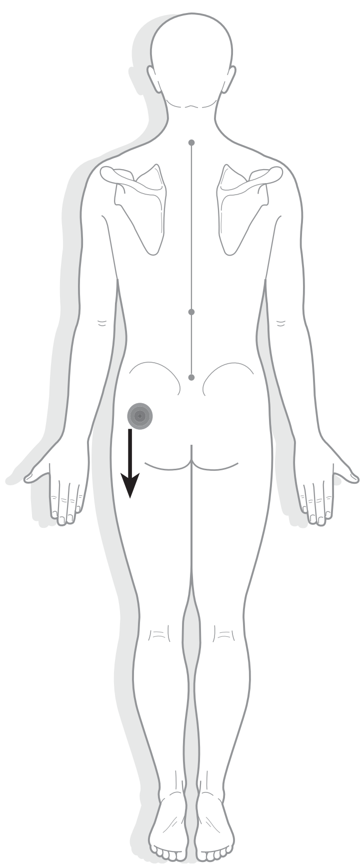

Figure 9.1

IMAGING

There was no previous imaging.

CLINICAL IMPRESSION

Suspicion of two possible conditions:

1. As there was absolutely no history of trauma and the pain was essentially localized to the left buttock/hip area with pain on deep palpation, a deep-seated bone pathology was suspected.
2. Possible L4–5 or L5–S1 disc changes as suggested by his slight left-sided antalgic gait.

WHAT ACTION SHOULD BE TAKEN?

- Pelvis and lumbar spine radiographs; these showed a lytic lesion in the left ilium (Fig. 9.2A).
- A pelvis CT scan; this showed 'considerable osseous erosion and a large extra-osseous mass as is typical of Ewing's sarcoma'.
- Plain chest X-ray films and CT chest films; these were found to be normal.
- A bone scan; this indicated that there was no evidence of metastatic disease.
- Biopsy; the patient was referred for a biopsy of the lytic lesion in the left iliac wing and a diagnosis of Ewing's sarcoma was made. Therefore, bone marrow aspirate and trephine was performed on the opposite iliac crest but this showed no evidence of infiltration with Ewing's sarcoma.

DIAGNOSIS

Ewing's sarcoma in the left ilium.

TREATMENT AND RESULTS

The patient was referred to a medical oncologist where he was given ongoing courses of chemotherapy to maximal tumour response. A repeat CT scan of the affected area was performed at 6-weekly intervals following courses of chemotherapy until maximum tumour response was attained. He required eight courses of chemotherapy administered at 3-weekly intervals and this treatment was well tolerated. He then underwent surgery (Fig. 9.2B) approximately 6 months following diagnosis, radiotherapy and then adjuvant chemotherapy.

Four months following resection of the original tumour he underwent bone graft surgery to the area of original treatment. This was followed by chemotherapy for a further 6 months (making a total of 70 weeks of chemotherapy).

The patient had a very positive outlook on life and is now 11 years post-surgery and, although he has experienced bouts of considerable low back pain since the time of the operation, he manages to walk reasonably well, in spite of developing pseudoarthroses at the left hip joint.

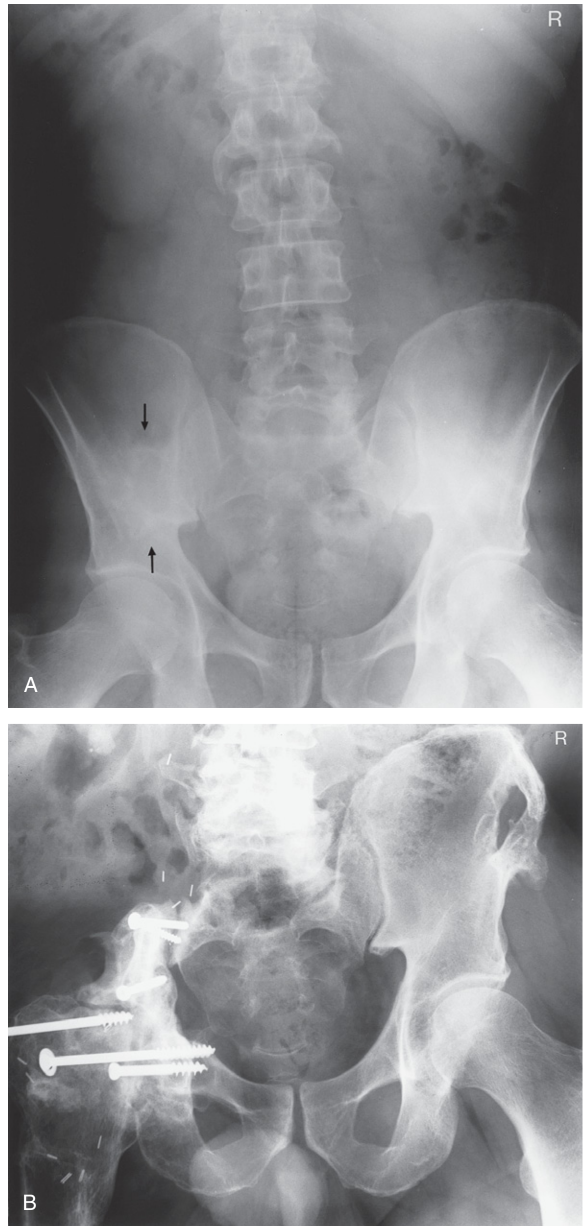

Figure 9.2 (A) Pelvis and lumbar spine anteroposterior plain X-ray image showing a lytic and sclerotic lesion in the left ilium (Ewing's sarcoma) (arrows). R = right side of patient. (B) Pelvis anteroposterior plain X-ray image showing previous resection of the ilium and arthrodeses of the left hip. Degenerative changes are noted at the lumbosacral level. No focal bony erosive change is demonstrated.

KEY POINTS

1. Be wary of patients who present with pain and absolutely no history of trauma.
2. Bogduk's 1999 'Modified criteria for the use of plain films in low back pain' that are based on the paper by Deyo and Diehl (1986) are shown to be questionable by this case.

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Case 10

Abdominal aorta aneurysm

COMMENT

This is another example to show how important it is to look at the imaging films and not to rely only upon a radiological report.

PROFILE

A 76-year-old retired male manual worker of average build.

PAST HISTORY

He underwent surgery for the removal of a cyst on the right knee 8 years ago. He has been on medication for hypertension for 2 years.

Forty-two years ago he slipped and fell, landing on his right buttock.

PRESENTING COMPLAINT(S) (Fig. 10.1)

Chronic low back pain intermittently since a work-related injury 42 years ago. The chronic low back pain does not trouble him unduly, although he has noted ‘sharp stabs of pain in the low back’ during the last month or two. There is no night pain.

AETIOLOGY

He slipped and fell 42 years ago.

EXAMINATION

The examination was unremarkable apart from minor low back pain on right rotation of the lumbar spine. Supine SLR was to 90° on the left and right sides without any increase in low back pain. The deep tendon reflexes in the lower extremities were normal as was the case with muscle tone, power, pinprick sensation, and vibration sensation.

IMAGING REVIEW

No recent imaging was available.

CLINICAL IMPRESSION

Although he was neurologically intact in the lower limbs and he had a 42-year history of chronic intermittent low back pain, it was necessary to be sure of the pathology causing his low back pain so as to exclude pathology other than lumbar spine ‘wear and tear’.

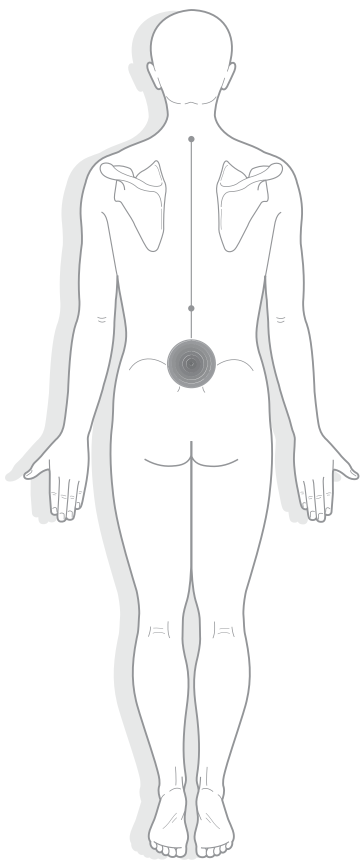

Figure 10.1

WHAT ACTION SHOULD BE TAKEN?

Laboratory tests were performed as a precaution in view of his age, with the following results: Serum prostate specific antigen was 3.5 ng/ml (range = 0.0–13.1). A full blood count was normal as was the ESR at 4 mm/1 hour (range 2–20).

Lumbar spine plain film radiographs were ordered in the erect posture (Fig. 10.2A and B) and the radiology report stated: 'Advanced degenerative change is noted throughout the lumbar spine with disc narrowing and gas in the disc space at all five lumbar discs. There is an

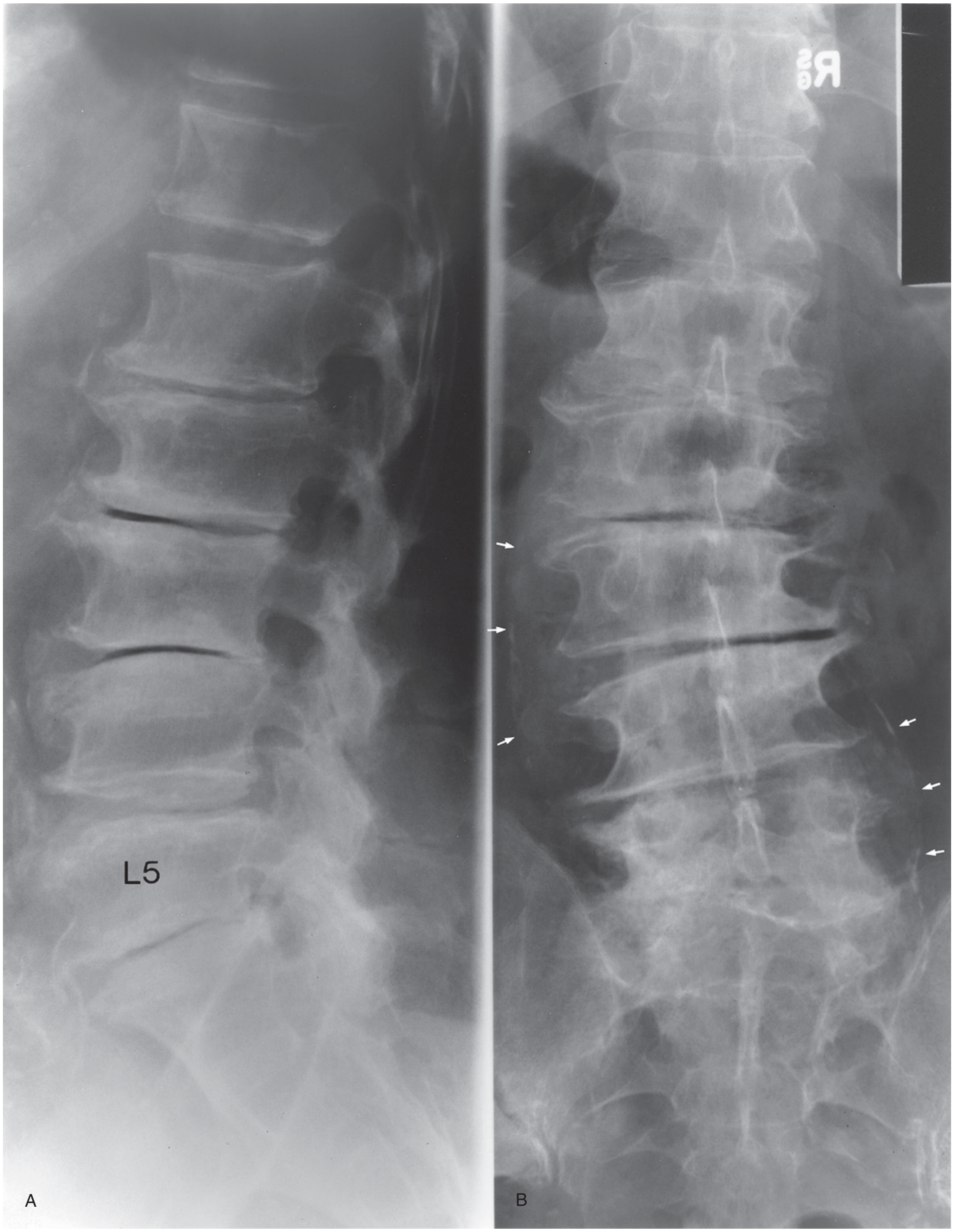

Figure 10.2 (A and B) Lumbosacral spine lateral and anteroposterior plain X-ray images showing the advanced discogenic spondylosis with partial calcification in the abdominal aorta aneurysm (small arrows). L5 = fifth lumbar vertebra.

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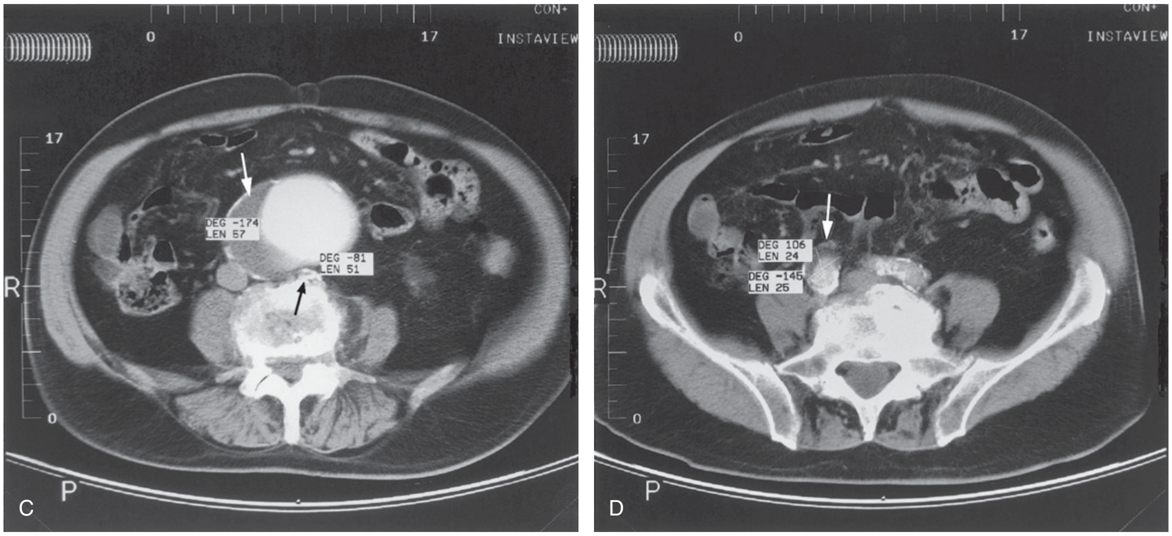

Figure 10.2 Cont'd (C and D) CT Axial image with contrast showing the large abdominal aortic aneurysm (8 × 6 cm) with the right lateral eccentric thrombus (white arrow). Note how the lumbar spondylosis (small black arrow) is indenting the aortic aneurysm posteriorly which could make the wall of the aneurysm vulnerable to bleeding. (D) Axial CT scan showing that the aneurysm involves the origin of the right common iliac artery which has a maximal diameter of 2.4 × 2.5 cm (arrow).

associated lumbar scoliosis convex to the left and centred at L3 with a mild rotational component. There is severe degenerative change in the zygapophysial joints at all levels, particularly from L3 to S1'.

A vitally important finding was missed on the plain X-ray report. Note the large aneurysm in the abdominal aorta at the L3 to S1 level (small arrows). Therefore a CT abdomen scan was ordered using the technique of post contrast axial scans performed from the diaphragm to the symphysis. This confirmed the extent of the large abdominal aortic aneurysm: There is an ectatic fusiform abdominal aortic aneurysm with a maximal diameter of 8 × 6 cm. It has right lateral eccentric thrombus. It arises well below the origins of the renal arteries. There is diffuse calcification and atheroma within the abdominal aorta. The aneurysm involves the origin of the right common iliac artery that has a maximal diameter of 2.4 × 2.5 cm. Both kidneys perfuse normally. (Fig. 10.2C and D).

DIAGNOSIS

Abdominal aorta aneurysm.

TREATMENT AND RESULTS

The imaging was explained to the patient who was immediately referred to a vascular surgeon and underwent successful surgery to repair the aneurysm. This gave him great relief from most of his low back pain.

Some months following surgery the patient required intermittent relief for minor residual low back pain symptoms for which he was given needle acupuncture treatment that resulted in great relief. Occasionally, he returns for a course of 6–10 needle acupuncture treatments to relieve his chronic intermittent low back pain.

KEY POINTS

1. Be wary of patients presenting with a long history of intermittent low back pain who may subsequently have developed life-threatening pathology that apparently mimics the 'longstanding low back pain'.
2. Always look at the imaging films and not only at the report.

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Case 11

Small aortic aneurysm

COMMENT

When necessary, 3-D CT reconstruction can be useful for looking at the relationship between adjacent anatomical structures in greater detail.

PROFILE

A 64-year-old male manual worker who smokes approximately 40 cigarettes daily and drinks six beers a day.

PAST HISTORY

Nothing contributory. There is a family history of ischaemic heart disease.

PRESENTING COMPLAINT(S) (Fig. 11.1)

Constant low back pain, varying in intensity from a mild ache to severe pain, with considerable radiation into the right hip, groin and down the back of the right leg as sciatica during the last 6 months. Coughing and sneezing makes the pain worse. He can get some relief from the low back pain syndrome by lying on his right side. Non-steroidal anti-inflammatory drugs have not been helpful so he was referred by his general medical practitioner for an opinion and treatment; his prostate-specific antigen (PSA) level was normal.

AETIOLOGY

There was no obvious cause for the low back pain syndrome that becomes worse as the day progresses.

EXAMINATION

Lumbar spine movements were of full range and painless, except for extension of the lumbar spine which elicited some lumbosacral pain. External rotation of the right hip elicited slight pain in the right groin. Supine SLR was of a good range for his age and painless. Neurologically he was intact in the lower limbs. Muscle tone and power, as well as pinprick sensation and reflexes, were normal. His peripheral pulses were all intact. A small aortic aneurysm was palpable on abdominal examination.

IMAGING REVIEW

A previous lower lumbar CT scan suggested minor L4–5 spinal canal stenosis.

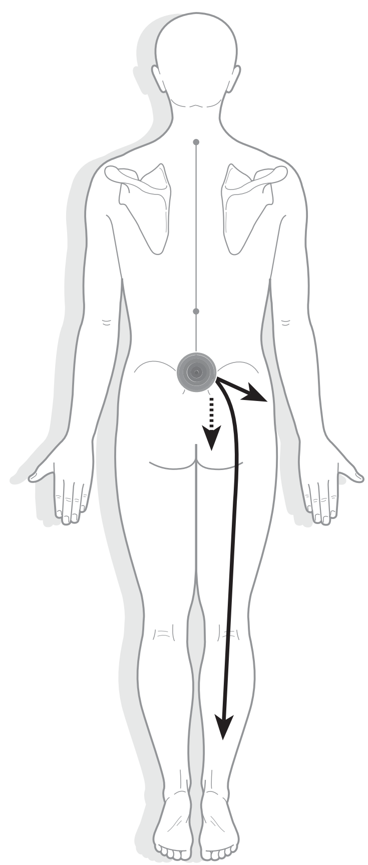

Figure 11.1

CLINICAL IMPRESSION

A degree of abdominal aortic aneurysm and probable L4–S1 degenerative stenosis changes.

WHAT ACTION SHOULD BE TAKEN?

Plain film radiographs were taken of the pelvis and lumbar spine in the erect posture and these showed minor osteoarthrosis in the lower lumbosacral zygapophysial joints with minor lipping of the vertebral bodies anteriorly; in addition, patchy atheromatous plaques were noted in the abdominal aorta (Fig. 11.2A).

Therefore, he was sent for an ultrasound examination of the abdominal aorta that showed: 'Evidence of calcification with atheroma within the abdominal aorta. The anteroposterior diameter of the abdominal aorta just distal to the superior mesenteric artery is 2.2 cm. The renal origins are identified and the abdominal aorta at this level measures 2.2 × 1.8 cm. Below the renal arteries there is a focal aneurysmal dilatation of the abdominal aorta with an anteroposterior diameter of 2.6 cm and a transverse diameter of 3 cm. There is calcification of the iliac arteries with no evidence of aneurysmal dilatation'.

A CT abdomen and pelvis was then performed to further evaluate the aneurysm using a technique of pre-contrast scans followed by contrast-enhanced dynamic helical scans of the abdominal aorta and iliac arteries with 3-D reconstruction of the abdominal aorta. The findings were: 'Extensive calcification of the abdominal aorta and iliac arteries. There is no evidence of intraluminal thrombus (Fig. 11.2B). The CT confirms the presence of focal aneurysmal dilatation below the renal arteries (Fig. 11.2C). The maximum transverse diameter is 3.2 cm. There is no evidence of aneurysmal extension into the iliac arteries, normal spleen and both kidneys with no mass in the pancreas and no retroperitoneal lymphadenopathy'.

The reconstruction view (Fig. 11.2D) clearly shows the spatial relationship between the kidneys, various blood vessels and the aneurysmal dilatation below the renal arteries.

A CT lumbar spine was ordered to examine the spinal canal prior to consideration of treatment options. This showed that: 'At the L4–5 disc level, there is significant canal stenosis due to a combination of a very diffuse disc bulge, hypertrophy of the ligamenta flava and hypertrophic degenerative change in the zygapophysial joints. At the L5–S1 disc level there is also a prominent generalized disc bulge and hypertrophy of the ligamenta flava causing a minor degree of relative canal stenosis. There is advanced degenerative change in the zygapophysial joints occurring bilaterally'.

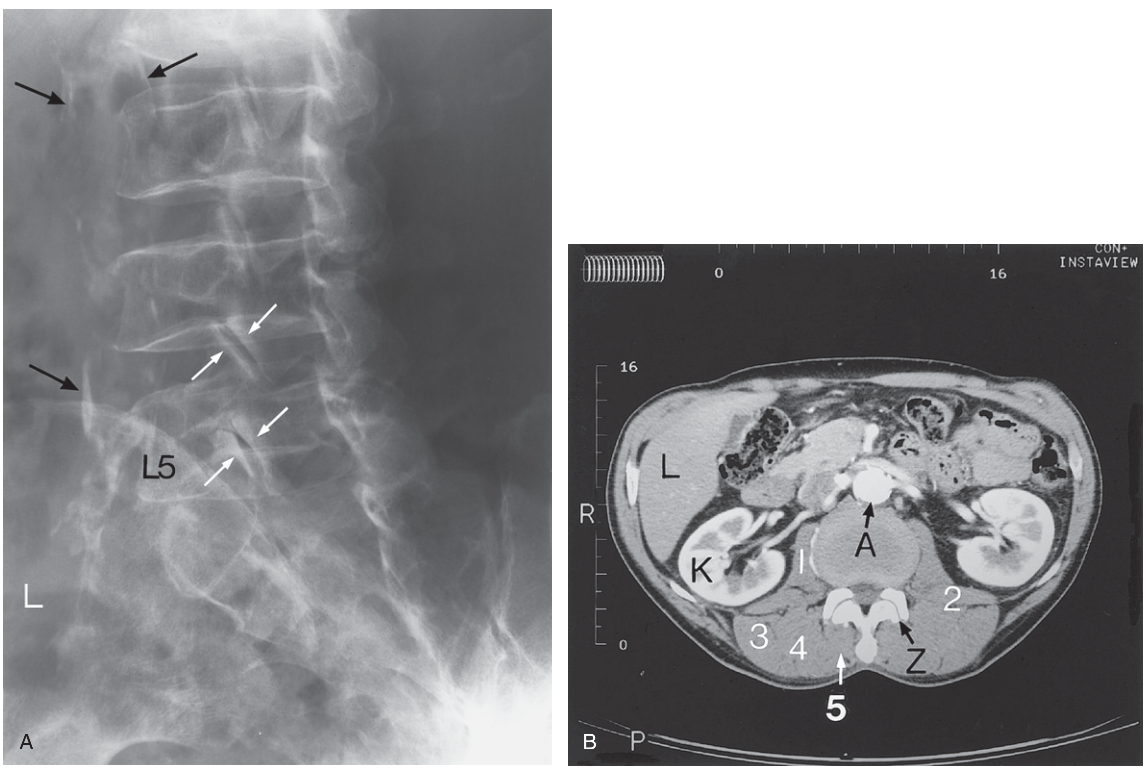

Figure 11.2 (A) Lumbar spine oblique plain X-ray image. L = left side of the patient. Note the osteoarthrotic changes in the zygapophysial joints as indicated by subchondral sclerosis (white arrows). The patchy atheromatous plaques in the abdominal aorta are indicated with black arrows. L5 = fifth lumbar vertebra. (B) CT axial image showing the aorta (A), right kidney (K) and blood vessels leaving it, and the liver (L). Z = left zygapophysial (facet) joint; 1 = psoas major muscle; 2 = quadratus lumborum muscle; 3 = iliocostalis lumborum muscle; 4 = longissimus thoracis muscle; 5 = multifidus muscle.

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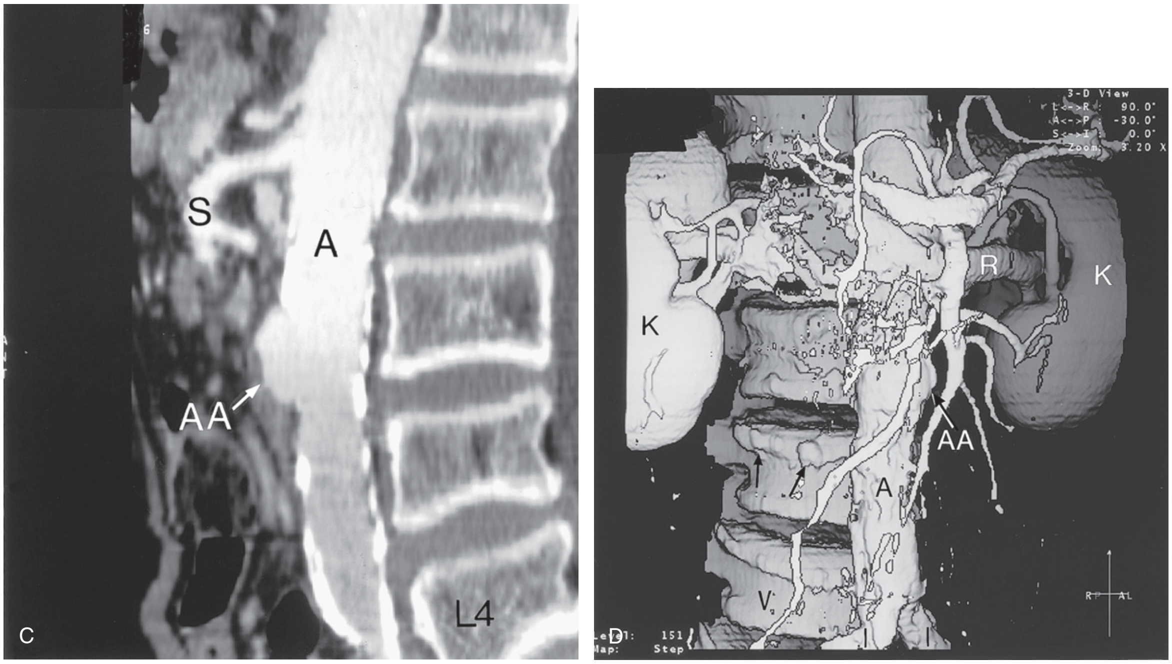

Figure 11.2 Cont'd (C) CT 3-D reconstruction image showing the infra-renal small abdominal aortic aneurysm (AA), abdominal aorta (A) and superior mesenteric artery (S). L4 = fourth lumbar vertebral body. (D) CT 3-D reconstruction image showing the kidneys (K), renal artery (R), abdominal aorta (A), small aneurysm of the abdominal aorta (AA) and common iliac arteries (I). V = vertebral body of the fourth lumbar vertebra. Black arrows indicate osteoarthrotic lipping along the superior margin of the L3 vertebral body; lipping can also be seen at some of the other vertebral body levels adjacent to the intervertebral disc spaces.

The conclusion was: 'Canal stenosis and hypertrophic degenerative change in the zygapophysial joints leads to foraminal stenosis, which is most marked at the L5–S1 level but the most significant overall finding is the canal stenosis at the L4–5 level'.

DIAGNOSIS

L4–5 and L5–S1 spinal canal and foraminal stenosis with right sided radiculopathy.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. In view of the low back pain with considerable right sciatica he was advised to stop smoking, lose weight and stop lifting heavy containers during his work. He was given a trial of needle acupuncture while waiting to see a vascular surgeon for an opinion but this treatment was not helpful. As he had intact peripheral pulses and only a small aortic aneurysm that was palpable on abdominal examination, it was decided by the vascular surgeon that no surgery was necessary at this time. However, should the aneurysm eventually reach 5 cm in size (as found on follow-up ultrasound or CT scans), elective repair would be indicated.

The patient elected to stop working as he was of approximately retirement age and he agreed to 6-monthly follow-up abdominal ultrasound scans to determine whether the aneurysm progressed.

On review 6 months after he stopped working, he said his pain had improved considerably since ceasing work, although he required occasional antidepressant medication and analgesics to help to control his chronic low back pain syndrome symptoms. A follow-up ultrasound scan showed no progression of the small aortic aneurysm at that time.

KEY POINTS

1. A small palpable aortic aneurysm should be investigated by ultrasound and further studies as indicated.
2. Further sophisticated imaging such as 3-D CT reconstruction may be useful to augment an ultrasound examination.

Further reading

Greenhalgh R, Powell J 2007 Screening for abdominal aortic aneurysm. BMJ 335: 732–733.
Also see Case 10.

Case 12

Minor internal disc disruption

COMMENT

When an MRI study appears to be normal, in spite of a patient having a history and physical findings suggesting a disc problem, it is important to have a clinical suspicion of internal disc disruption.

PROFILE

A 20-year-old male with muscular athletic build who is a non-smoker and a non-drinker who works in the armed forces.

PAST HISTORY

He had always been very fit; however, 6 months ago, while lifting a heavy weight with three work colleagues, 6 months before consultation, he felt a central 'moderate' low back pain. He thought he had 'pulled a muscle' in the low back, so performed some exercises that he normally performed to keep fit; these caused a marked increase in the low back pain and radiation down the lateral aspect of his left thigh and down the back of the left leg, with paraesthesiae in the left foot. He was hospitalized and treated with bed rest, analgesia, muscle relaxants and physiotherapy but with little, if any, improvement.

PRESENTING COMPLAINT (Fig. 12.1)

Constant moderate to severe low back pain centrally at the L4 to S1 level with some pain radiating to the left thigh posterolaterally and down the back of the left leg with paraesthesiae in the left foot. Coughing aggravates the low back pain and sneezing aggravates the paraesthesiae in the left foot. Walking causes an increase in low back and left thigh pain as do long standing and sitting. The pain is sometimes felt as a 'sharp throbbing low back pain'.

The degree of pain causes him difficulties when trying to sleep as he feels that there are 'severe muscle spasms' in the left leg.

Nerve conduction studies had been performed and reported as being normal. He was particularly upset as the clinicians whom he had consulted (medical practitioners, chiropractor and physiotherapist) thought he was malingering and, in fact, one comment recorded stated '? supratentorial component'.

He tried non-steroidal anti-inflammatory drugs, needle acupuncture, chiropractic manipulation and physiotherapy treatment without success and was currently depressed by his genuine symptoms that have been trivialized.

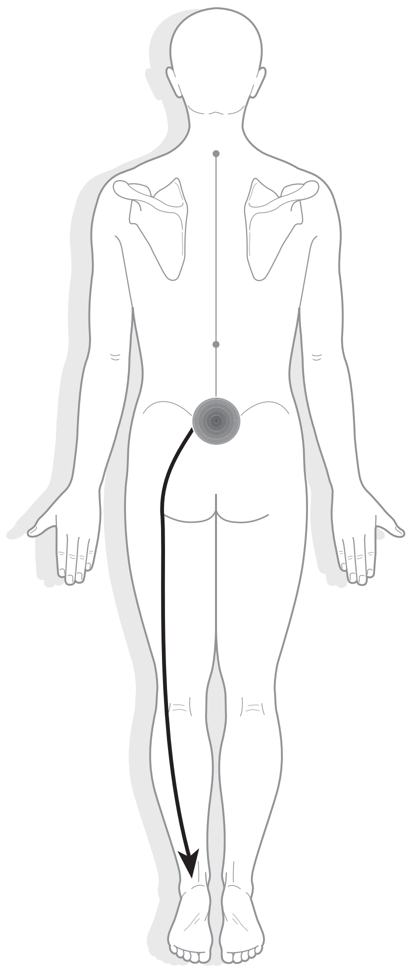

Figure 12.1

AETIOLOGY

Lifting a heavy weight 6 months ago.

EXAMINATION

Percussion of the spine elicited L4–S1 pain, as did deep palpation of the paraspinal muscles in the same region. Lumbar spine active ranges of movement were as follows:

1. Flexion – limited by approximately 60% due to L4–S1 pain with a 'flat back' on forward bending.
2. Extension – limited by approximately 15% due to L4–S1 pain.
3. Left rotation – limited by approximately 10% due to L4–S1 pain.
4. Right rotation – painless and of full range.

Sitting slumped forward caused an increase in low back pain. Seated straight leg raising was limited due to low back pain to a measured 30° (left) and 45° (right). Supine SLR was limited due to low back pain to a measured 30° (left) and 45° (right). Pinprick sensation was diminished in the lateral aspect of the left calf (L5) and foot (S1). The foot pulses were normal and the temperature of the feet and legs appeared equal and normal bilaterally on palpation. Axial loading on the shoulders caused an increase in low back pain, whereas gentle axial loading of the skull did not cause any low back pain. Power in the big toes, feet and legs was normal. Sacroiliac joint stress tests were normal. Active bilateral SLR was very limited due to L5–S1 pain.

CLINICAL IMPRESSION

L5–S1 intervertebral disc bulge/protrusion at the lumbosacral disc level; definitely not psychosomatic!

IMAGING REVIEW

A review of his plain film radiographs (Fig. 12.2A and B) showed normal imaging, as did the oblique views.

WHAT ACTION SHOULD BE TAKEN?

An MRI of the lumbosacral spine. This was essentially normal, apart from some T10–11 and T12–L1 Schmorl's nodes (Fig. 12.2C and D). The axial image at L5–S1 shown in Fig. 12.2E shows that there is no disc protrusion and the nerve roots, spinal ganglion and spinal nerves are normal. Because this young man was going to lose his employment as he was considered a malingerer, steps were taken to try to prove that he had internal disc disruption or an annular tear.

Erect posture left (Fig. 12.2F) and right (Fig. 12.2G) lateral bending radiographs were taken. It can be seen that the disc spaces above the L5 vertebra wedge from being narrow on the concave side to wide on the convex side on lateral bending. However, there is apparently no movement between the

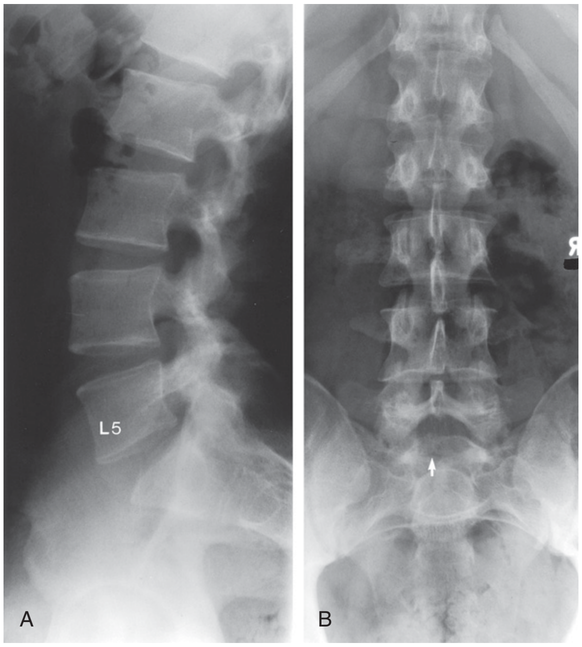

Figure 12.2 (A) A normal lumbosacral spine lateral plain X-ray image. L5 = fifth lumbar vertebra. (B) A normal lumbosacral spine anteroposterior plain X-ray image apart from a very minor spina bifida occulta at the S1 level (arrow).

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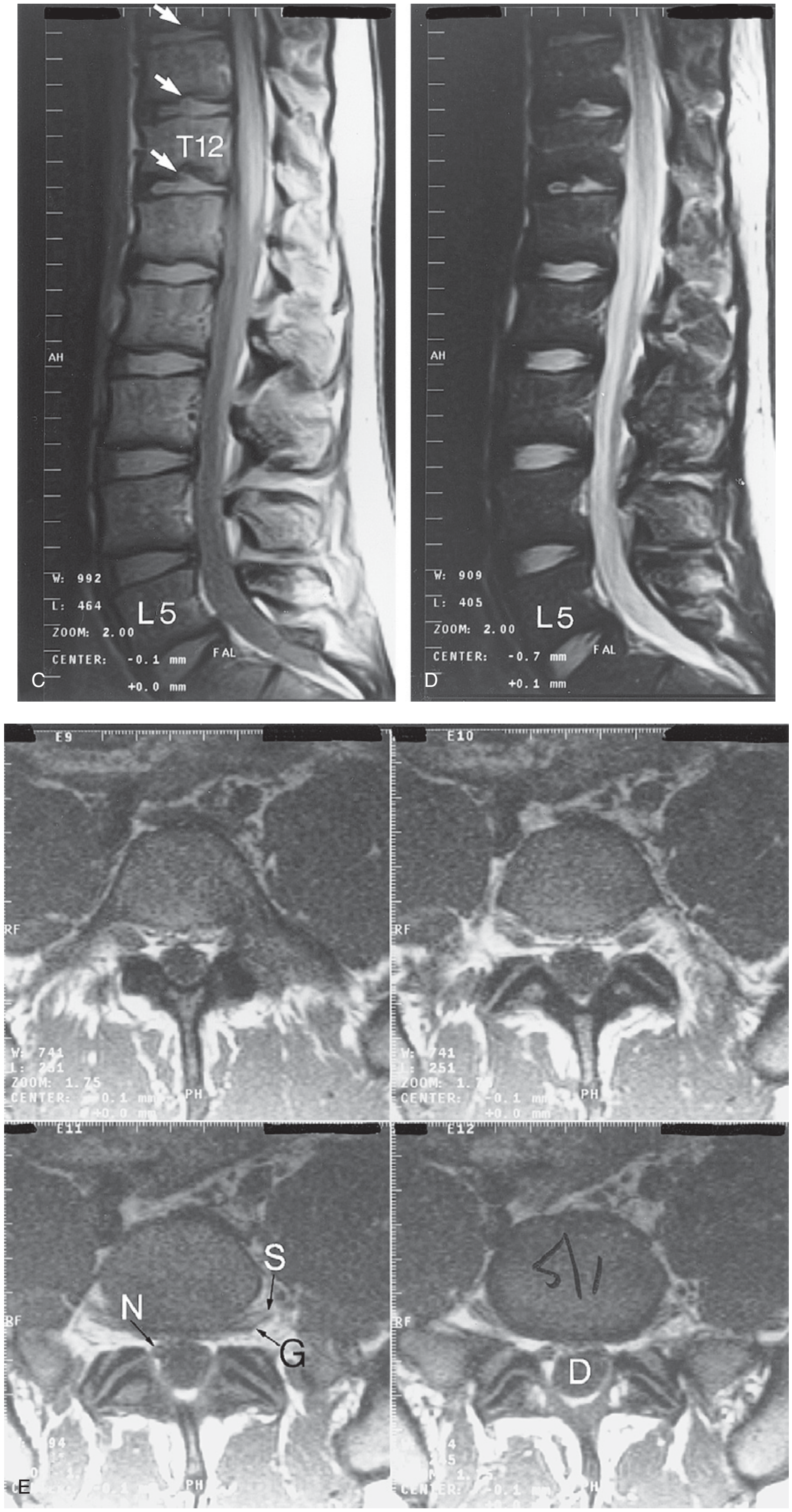

Figure 12.2 Cont'd (C) Lumbosacral spine MRI sagittal T1-weighted image. Note the Schmorl's nodes (vertebral body end plate fractures due to vertical rupture of nucleus pulposus material) at the T10, T11 and T12 levels (white arrows). (D) Lumbosacral spine MRI sagittal T2-weighted image. (E) Normal MRI axial images of the L5–S1 level. Note the nerve root (N), spinal ganglion (G) and spinal nerve (S). D = Dural tube; RF = right side of patient.

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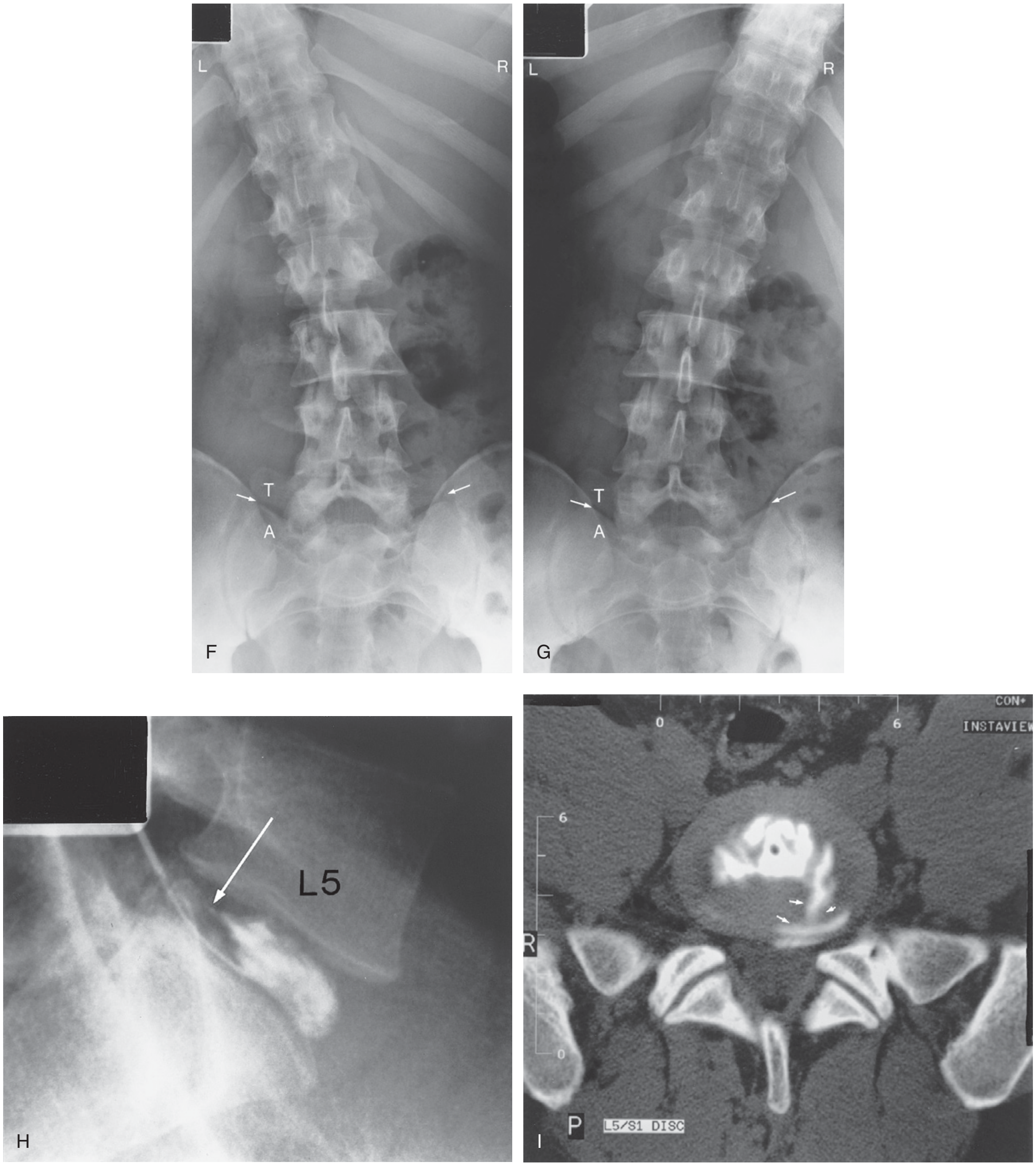

Figure 12.2 Cont'd (F) Lumbosacral spine erect posture left lateral bending anteroposterior image. Note that the disc spaces above the L5 vertebra wedge from being narrow on the left to wide on the right. However, there is apparently no movement between the transverse processes (T) of L5 and the sacral ala (A) (white arrows). (G) Lumbosacral spine erect posture right lateral bending anteroposterior image. Note that the disc spaces above the L5 vertebra wedge from being narrow on the right to wide on the left. However, there is apparently no movement between the transverse processes (T) of L5 and the sacral ala (A) (white arrows). (H) Lateral image discogram of the L5–S1 disc which shows tracking of the contrast medium posteriorly (arrow). (I) Lumbar spine CT axial image of the L5–S1 disc containing contrast material following the discogram which clearly shows a full thickness left posterolateral tear in the annular fibres (arrows) with some internal disc disruption centrally.

transverse processes of L5 and the sacral ala on comparing the left and right sides for both left and right lateral bending. Because further evidence was required to confirm that he did have internal disc disruption and a probable tear of the annular fibres, it was necessary to perform a discogram, a procedure which, according to Clifford (1986), Shapiro (1986) and Wiley et al (1968), is a barbaric and a non-efficacious procedure that can have significant complications (Yochum & Rowe 1996). However, a discogram had to be ordered to override the suggestions that he was a malingerer and the lateral view of the L5–S1 disc is shown in Fig. 12.2H. This figure clearly shows tracking of the contrast medium posteriorly.

A CT scan of the L5–S1 disc containing contrast material (following the discogram) clearly shows a full thickness left posterolateral tear in the annular fibres with some internal disc disruption (Fig. 12.2I).

Referral to an orthopaedic surgeon for a second opinion confirmed that 'the posterior annular tear would cause his low back pain syndrome'.

DIAGNOSIS

L5–S1 internal disc disruption with a full thickness left posterolateral annular tear causing adjacent nerve root inflammation with radiculopathy.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was greatly relieved to finally have a diagnosis made and to be told that he had an organic cause for his pain that clearly was not in his head. He agreed to have an epidural block injection, which gave some relief, and he was advised to perform only light duties and to keep fit and ensure that his muscles remained strong and supportive for his low back. Apart from walking and swimming, he was advised to perform an exercise programme (see Case 1).

The patient continues to experience minor intermittent low back pain with some pain and paraesthesiae extending into his left lower limb, some 4 years after his injury. He is reluctant to have any invasive procedure and only performs light duties.

Note

It is important to visualize what internal disc disruption looks like at the gross anatomical level (Fig. 12.3A) and what it can look like in the early histopathological stage (Fig. 12.3B).

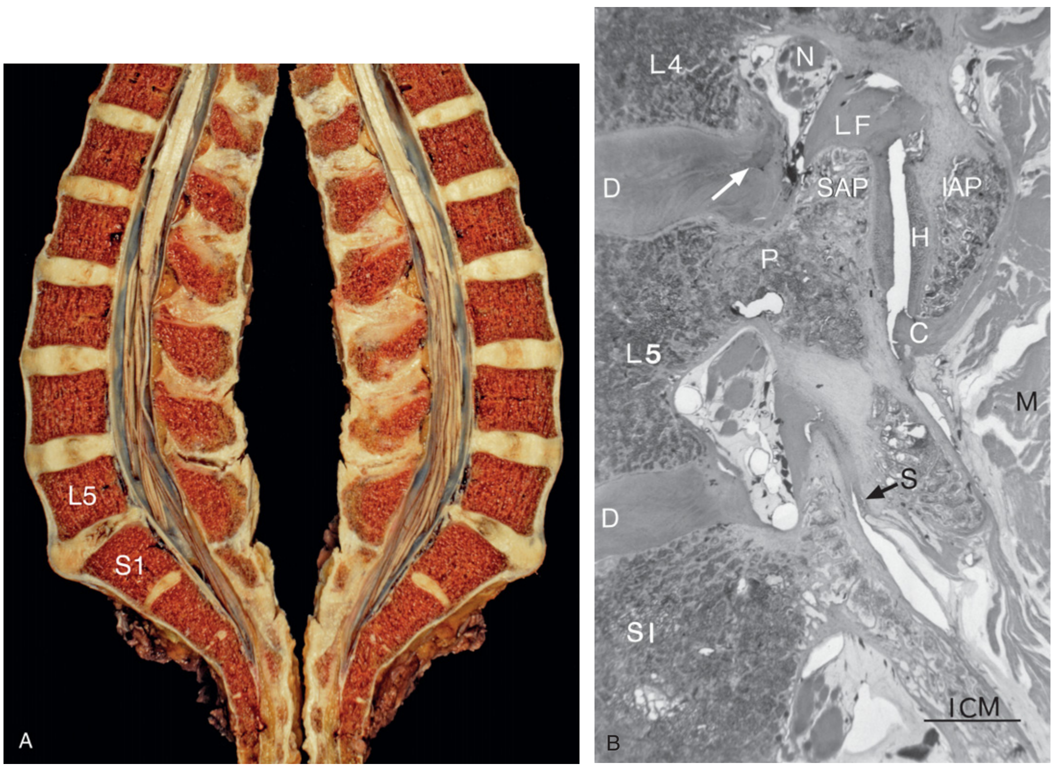

Figure 12.3 (A) Anatomical specimen of a sagittally sectioned spine. Note the approximation of the elongated fifth lumbar spinous process and the adjacent sacral spinous tubercle, in spite of the normal lumbar lordosis in this 70-year-old female. The L5 intervertebral disc shows degenerative changes which include (i) anterior 'bulging' of the disc, and (ii) posterior herniation of the disrupted nucleus pulposus which has elevated the posterior longitudinal ligament above and below this disc level. (Reproduced with permission from Giles L G F Miscellaneous pathological and developmental (anomalous) conditions. In: Giles L G F, Singer K P (eds) 1997 Clinical anatomy and management of low back pain. Butterworth-Heinemann, Oxford, p 196–216.) See also colour plate section Fig. vii.4B. (B) A 200-micron thick histopathology parasagittal section showing a tear within the L4–5 intervertebral disc with retrograde movement of nuclear material in this disc (white arrow). C = capsule (inferiorly) of the L4–5 zygapophysial joint; D = intervertebral disc; H = hyaline articular cartilage on the facet of the inferior articular process of the L4 vertebra; IAP = inferior articular process of the L4 vertebra; L4 = part of the fourth lumbar vertebral body; L5 = part of the fifth lumbar vertebral body; LF = ligamentum flavum; M = muscle; N = neural structures within the nerve root sheath located in the pear-shaped intervertebral canal; P = pedicle of the L5 vertebra; S = synovial fold projecting into the inferior recess of the zygapophysial joint at the L5-S1 level; SAP = superior articular process of the L5 vertebra. (Ehrlich's haematoxylin and light green counterstain.) See also colour plate section Fig. vii.4B.

It should be noted that *internal disc disruption and annular tears are not part of the normal ageing process*. Discs can be perfectly normal even in the 70-year age group as shown by the specimen in Fig. 12.3A where the discs are normal except for at the L5–S1 level where there is internal disc disruption with anterior and posterior bulging of disc material. Clearly this is a phenomenon related to injury and it is not related to the ageing process, as only one disc in the 70-year-old spine showed internal disc disruption and those above are perfectly normal with a normal nucleus pulposus and annulus fibrosus.

It should be noted that the incidence of vascular penetration during contrast confirmed fluoroscopically guided lumbosacral transforaminal epidural injections is 8.9%; the incidence of a vascular injection alone was 4.2% for a total vascular injection incidence of 13.1% (Smuck et al 2007).

KEY POINTS

1. A 'normal' MRI study does not mean the disc is normal – there may be internal disc disruption and/or an annular tear allowing disc material to inflame the adjacent neural structures.
2. Osti and Fraser (1992) in their study of lumbar discs showed that 'discography is more accurate than MRI for the detection of annular pathology'.

Furthermore, the study by Schellhas et al (1996) on cervical spine discs showed that 'significant cervical disc annular tears often escape magnetic resonance imaging detection'.

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Case 13

Major internal disc disruption

COMMENT

Low back pain can arise from a variety of low back structures.

PROFILE

A 49-year-old housewife who does not smoke or drink alcohol.

PAST HISTORY

Three years ago she had a cervical spine discectomy and fusion for left arm pain but she still experiences neck pain.

PRESENTING COMPLAINT(S) (Fig. 13.1)

Three years of low back pain radiating into both thighs anteriorly with a sensation like 'electric shocks' due to straightening up from bending forwards. These attacks initially took place approximately once a year and would last for 2–3 weeks. Her symptoms are now constant and chronic, with a deep burning pain over the lumbar area that radiates into both thighs and groins. Over recent months, she has developed right sciatica extending to halfway down her right calf and a 'cold and burning' sensation in the feet.

Her symptoms are exacerbated by bending, standing, sitting and housework but they are not aggravated by coughing or sneezing.

She had tried physiotherapy, swimming (including aqua-aerobics), acupuncture and non-steroidal anti-inflammatory drugs; the latter caused indigestion, oesophageal reflux and oesophagitis, for which she takes antacids.

She is often awakened by low back pain at night but not at a particular time, so she takes an analgesic to try to relieve the pain.

AETIOLOGY

Her low back pain syndrome began after straightening up from being bent forwards, at which time she also felt the 'electric shock' sensation across her lower back and it extended into the thighs anteriorly.

EXAMINATION

She was neurologically intact in the lower limbs. Foot pulses were normal. She was tender on deep palpation of

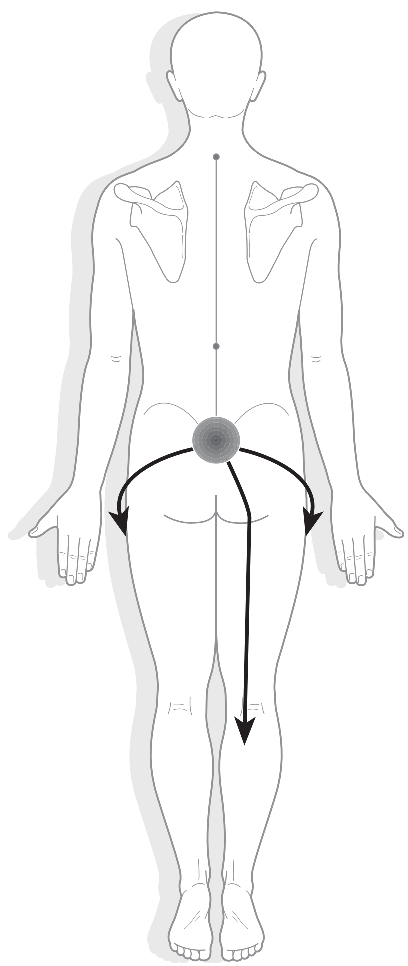


Figure 13.1

the paravertebral muscles of the lower lumbar spine. SLR was to 80° bilaterally and painless. Active ranges of lumbar spine movement were full with pain only on flexion.

IMAGING REVIEW

Plain lumbosacral spine films dating back 2 years were reported as showing 'slight degenerative changes present at the L4–5 and L5–S1 disc levels'. A CT scan through the lower three disc spaces found 'no disc herniation at any of the visualized levels, no spinal canal stenosis or paravertebral soft tissue mass'.

CLINICAL IMPRESSION

The symptoms suggested mechanical involvement of soft tissue structures in the lower lumbosacral spine as there was no abnormal neurology and SLR was painless, suggesting no large disc bulge posteriorly; internal disc disruption was considered likely. The 'cold and burning' sensation in the feet was considered to be due to autonomic involvement either via the paraspinal sympathetic nerve chain or the recurrent meningeal nerves (see Fig. ii, General Introduction).

WHAT ACTION SHOULD BE TAKEN?

In view of her presenting complaint the following laboratory tests were performed as a precaution: full blood count, urea and electrolytes, liver function tests and serum calcium; all were within the normal range except for a very slightly elevated ESR (Box 13.1).

A lumbar MRI study ordered concurrently showed: Mild circumferential disc bulges at L4–5 and L5–S1 (Fig. 13.2A and B) and desiccation of the L4–5 and L5–S1 discs (Fig. 13.2B). Facet and ligamentum flavum hypertrophy at these levels causes mild lateral recess stenosis bilaterally. Paravertebral soft tissues are within normal limits. An incidental finding of minor haemangiomas at L1 and L3 was noted.

Box 13.1

		Units	Reference range
ESR (erythrocyte sedimentation rate)	12	mm in 1 hour	(3–9)
Rheumatoid factor protein	<20	IU/ml	(<30)
Anti-nuclear antibody	Negative		

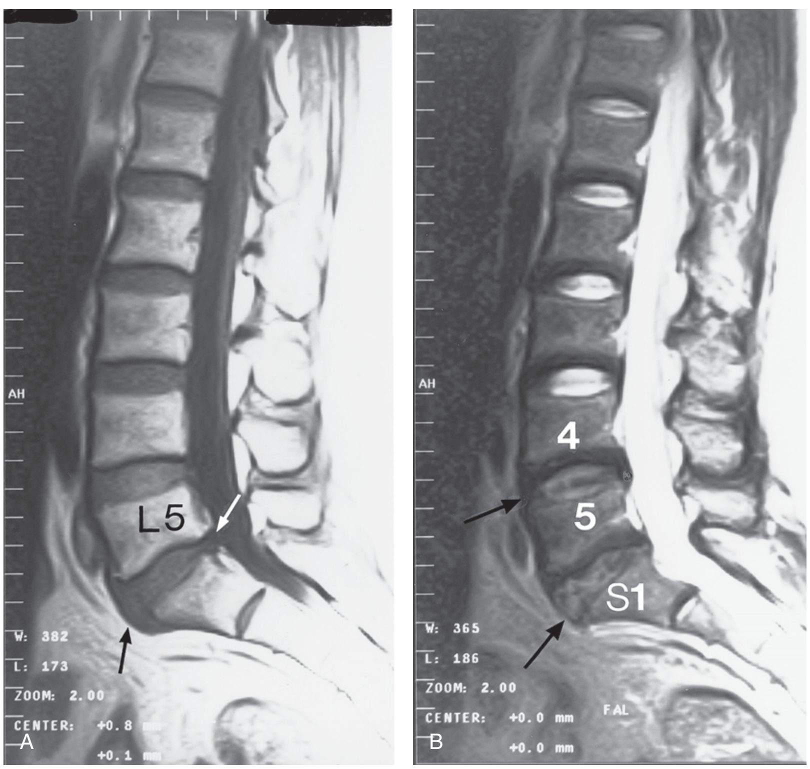
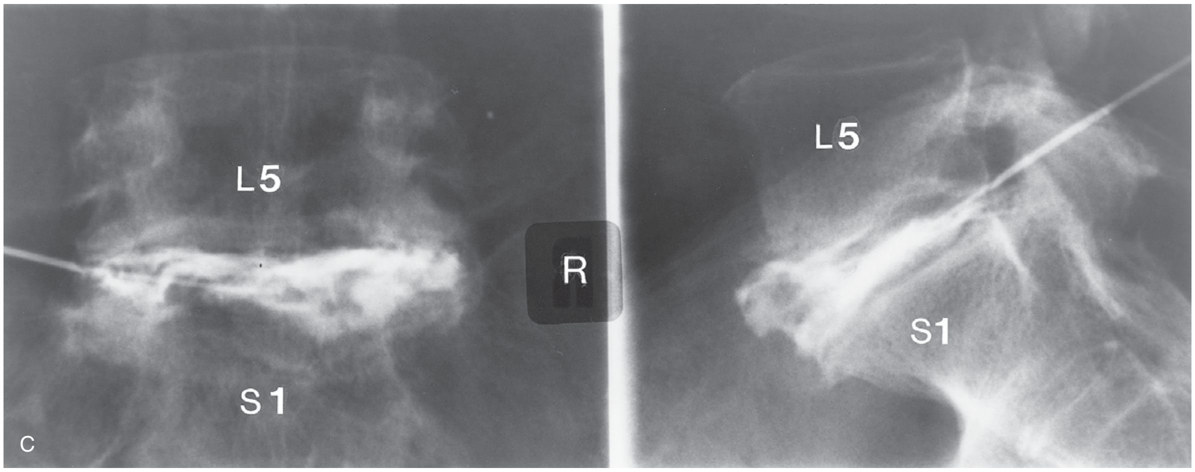


Figure 13.2 (A) Lumbosacral spine MRI sagittal T1-weighted image showing the large anterior (black arrow) and small posterior (white arrow) disc bulges at the L5–S1 level. (B) Lumbosacral spine MRI sagittal T2-weighted image showing considerable desiccation of the L4–5 and L5–S1 discs (arrows) which have become quite dark in colour compared to the discs above, which have a normal grey colour with a normal black line through them, i.e. the intranuclear cleft.

(Continued)



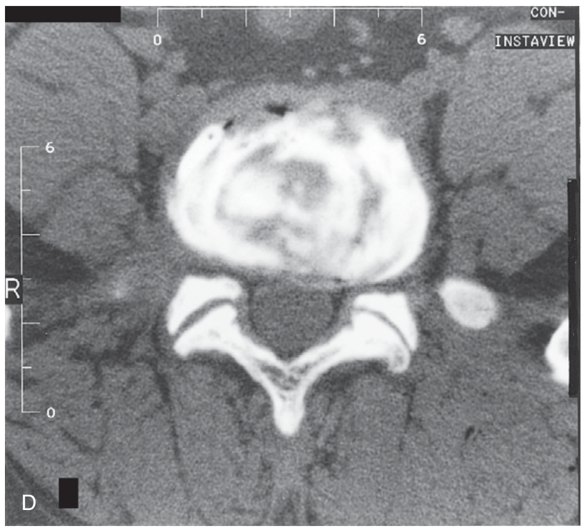


Figure 13.2 Cont'd (C) Anteroposterior and lateral discograms of the lumbosacral disc (L5–S1) showing the internal disc disruption and extravasation of contrast material during injection. (D) L5–S1 intervertebral disc CT axial image view showing significant internal disc disruption with loss of the normal annular fibres and extravasation from the disc across the endplates and into the paravertebral soft tissues.

DIAGNOSIS

L5–S1 internal disc disruption with mild circumferential disc bulges at L4–5 and L5–S1.

TREATMENT AND RESULTS

In view of her symptoms of an 'electric shock' type of pain going to the upper thighs anteriorly and the 'cold and burning' in the feet with a constant deep 'burning' low back pain, it was considered that she may have pain due to lower lumbar spine autonomic nervous system involvement, as well as pain from the changes within the L5–S1 disc. This disc had fairly large anterior bulging (Fig. 13.2A), probably affecting the autonomic chain, as well as some minor posterior bulging probably interfering with the recurrent meningeal nerves posteriorly. Therefore, a discogram (Fig. 13.2C) was ordered with a request that a local anaesthetic be injected into the disc if there were significant internal disc disruption. The discogram confirmed advanced internal disc disruption when 4 ml of contrast were injected into the disc under low pressure. This aggravated her chronic pain syndrome shortly after injection, the delay suggesting that the pain was coming from tissue structures adjacent to the disc that may have been irritated by the extravasated contrast anteriorly and laterally. The CT scan that followed (Fig. 13.2D) demonstrated total internal disc disruption at L5–S1 with contrast tracking freely across the endplates and extravasating anteriorly and to the right. The contrast coursed around the disc to the true posterior position beneath the posterior longitudinal ligament (see also Fig. 13.3).

Following the discogram, 1 ml of local anaesthetic was injected into the disc. This abolished all her pain for 3 weeks, at which time she kept a pre-arranged appointment with a surgeon who said that the 'anaesthetic could not have helped the pain' and suggested that she should have some traction, although she was virtually pain free.

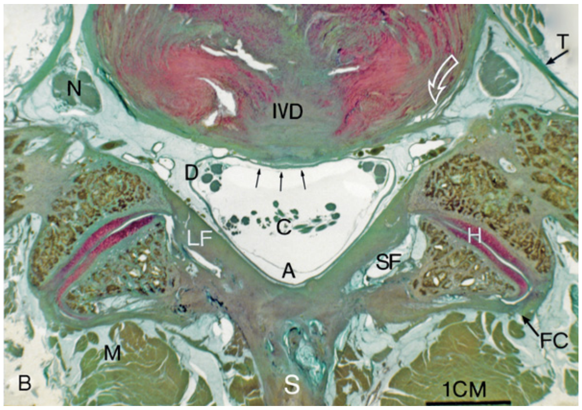


Figure 13.3 A 200-micron thick horizontal histopathology section from a 51-year-old female postmortem specimen. Note the internal disc disruption within the intervertebral disc (IVD) with some radial tears seen as white spaces and there is some disruption of the outer annulus fibrosus fibres (curved white arrow). There is some central posterior bulging of the disc which effaces (small black arrows) the anterior part of the dural tube (D) which contains the cauda equina (C) that is surrounded by cerebrospinal fluid and the arachnoid membrane (A). The neural structures (N) passing through the lateral part of the intervertebral foramen are in close proximity to transforaminal ligaments (T). S = remains of spinous process. SF = synovial fold adjacent to the ligamentum flavum (LF). H = hyaline articular cartilage surfaces on the facets of the zygapophysial joints. FC = fibrous joint capsule posterolaterally. M = multifidus muscle. (Ehrlich's haematoxylin and light green counterstain.) See also colour plate section Fig. vii.3B.

Therefore, she had traction and her pain syndrome returned. However, she did not want to undergo the discogram procedure again 'with that big needle', so she decided that she would prefer to put up with her chronic low back pain syndrome now that she knew what was causing her problem and she opted to use one Indocid suppository at night as required.

Note

Internal disc disruption will, in all likelihood, throw abnormal biomechanical stresses upon the zygapophysial joints that are considered to carry 16-40% of the incumbent body weight (Hakim & King 1976, Hutton & Adams 1980, Yang & King 1984). These abnormal biomechanical stresses most likely will lead to accelerated osteoarthrotic changes at levels where internal disc disruption has occurred.

KEY POINTS

1. Low back pain associated with apparently bizarre symptoms such as 'cold and burning' in the feet should suggest possible involvement of the paraspinal sympathetic chain.
2. Paraspinal sympathetic chain involvement most likely suggests irritation due to an anterolateral disc herniation or a claw spondylophyte affecting the paraspinal sympathetic chain.
3. Freemont et al (1997) showed nerve fibres that express substance-P deep within diseased intervertebral discs. In chronic low back pain patients the nerves extended into the inner third of the annulus fibrosus and into the nucleus pulposus in 46% and 10% of samples, respectively. Therefore, it is reasonable to suggest that the pain cycle from such a disc may be broken by an intradiscal injection of anaesthetic and steroid or by thermal annular heating and coagulation.

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Case 14

L4 discectomy

COMMENT

Spinal surgery has its limitations even when all conservative treatments have failed.

PROFILE

A somewhat overweight 34-year-old male of solid build who performed manual work and smokes approximately 20 cigarettes a day but does not drink alcohol.

PAST HISTORY

Four months before consultation he had lifted a 40 kg tin of paint; while doing so, he had slipped and twisted his low back, causing an immediate 'sharp pain in the low back'. He continued to work on light duties for approximately 6–8 weeks with low back pain, then there was no longer any light work to be performed so he was dismissed.

PRESENTING COMPLAINT(S) (Fig. 14.1)

Unbearable low back pain, due to lifting a heavy weight 4 months ago, that radiates posteriorly to the left leg as far as the ankle; he describes the low back pain as a 'sharp pain in the low back'. The pain may awaken him in the early hours of the morning but is not a deep-seated 'night' pain.

He had tried non-steroidal anti-inflammatory drugs and analgesics from his general medical practitioner. He had also seen an anaesthetist for an epidural block. A discogram had been performed for the two lower discs but this procedure had, unfortunately, caused an increase in his left-sided sciatica. He was taking 120 mg MS Contin daily and he was on antidepressants because of his chronic low back pain syndrome. He found that physiotherapy treatment, manipulation and acupuncture did not help; traction had caused a 'burning' sensation at the lower lumbar spine level at 30 kg of traction.

He was concerned that he could not completely empty his urinary bladder, but bowel function was normal. Coughing did not exacerbate his symptoms but sneezing did, as did bearing down; the latter caused a significant increase in low back pain. His general medical practitioner referred him for a further opinion.

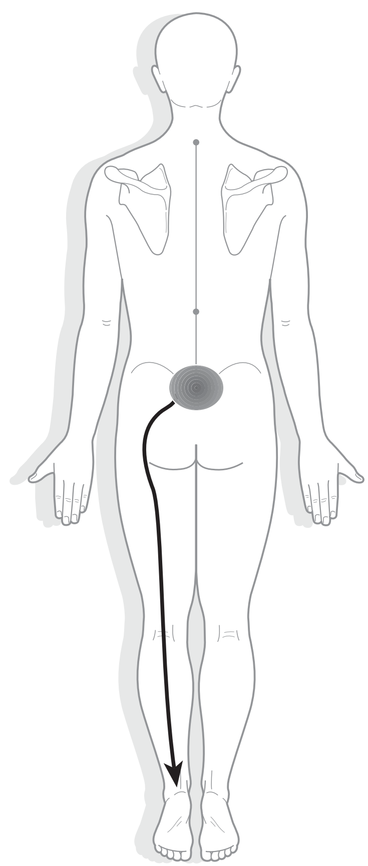

Figure 14.1

AETIOLOGY

Slipped while lifting a heavy weight 4 months ago.

EXAMINATION

He did not have a temperature and felt well apart from his presenting complaint. Lumbar spine active ranges of movement were as follows:

1. Flexion limited by 80% due to pain at the L4–5 level.
2. Extension limited by 90% due to pain at the L4–5 level.
3. Left and right lateral bending limited by 50% due to L4–5 pain.
4. Left and right rotation with the pelvis fixed were painless.

Toe walking power (S1) was normal but he felt there was an increase in his low back pain. Heel walking power (L5) was normal but elicited an increase in his low back pain. The ankle and knee jerks were normal. Bilateral hip flexion elicited a significant increase in low back pain. The foot pulses were normal. Pinprick sensation over the lower extremities was normal apart from an area of hypoaesthesia of approximately 2 cm in diameter on the anterolateral aspect of his left thigh. There was hypoaesthesia in the left little toe (S1 dermatome) and in the big toe (L5 dermatome). Supine SLR was limited by low back pain to a measured 15° on the left and to 20° on the right. Hoover's sign was normal, i.e. there was no attempt to malinger. The circumference of the thighs, 15 cm above the knee, was equal and within normal limits, as was the circumference of the calves. The Naffziger test elicited an increase in low back pain. The Milgram test (actively lifting both straight legs together) elicited an increase in low back pain. In the supine position, cervical spine flexion elicited an increase in low back pain.

IMAGING REVIEW

Plain film radiographs were within normal limits and a myelogram (Fig. 14.2A), followed by a CT, had shown, at the L4–5 level, 'mild wasting of the thecal sac with a broad-based anterior impression on the thecal sac due to generalized annular bulging, without significantly compromising the spinal canal or exiting L4 nerve roots'. A recent bone scan showed a normal examination.

CLINICAL IMPRESSION

A left posterolateral L4–5 intervertebral disc bulge/protrusion.

WHAT ACTION SHOULD BE TAKEN?

In view of existing imaging and his clinical presentation, an MRI was performed of his lumbar spine to further evaluate his lower lumbar discs. The MRI showed that 'the left L4–5 intervertebral foramen appeared narrowed by disc material' (Fig. 14.2B). As he was depressed by his chronic low back pain syndrome and his invalidity, he was referred for a surgical opinion that led to a discogram being performed at L3–4 to L5–S1 levels (Fig. 14.2C). This showed posterior leakage of contrast and reproduced his symptoms. A CT scan following the discogram showed a full thickness tear of the posterior disc fibres with leakage of contrast material at the L4–5 level (Fig. 14.2D).

DIAGNOSIS

L4-5 internal disc disruption with a broad-based annular bulge encroaching upon the adjacent nerve on the left side causing radiculopathy.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised to lose weight and to have a trial of acupuncture treatment but he did not make much progress in either attempt. Therefore, in view of the CT, discogram and MRI results, the patient elected to have a discectomy at the L4–5 disc level, in spite of advice regarding the unpredictability of spinal surgery, as he found his symptoms unbearable.

Following surgery he had 'good and bad days' and, although he walked for 2.5 km daily, he experienced severe pins and needles in the soles of both feet which he felt were 'cold' since surgery. Unfortunately, in the long term, surgery was not successful and he has been left with a chronic mixed mechanical and neuropathic low back pain that occasionally radiates to both legs despite the L4–5 discectomy.

Note

Neuropathic pain is a complex, chronic pain state that usually is accompanied by tissue injury; the nerve fibres themselves may be damaged, dysfunctional or injured, and send incorrect signals to other pain centres. The consequence of nerve fibre injury includes a change in nerve function both at the site of injury and areas around the injury (Grayson 2007).

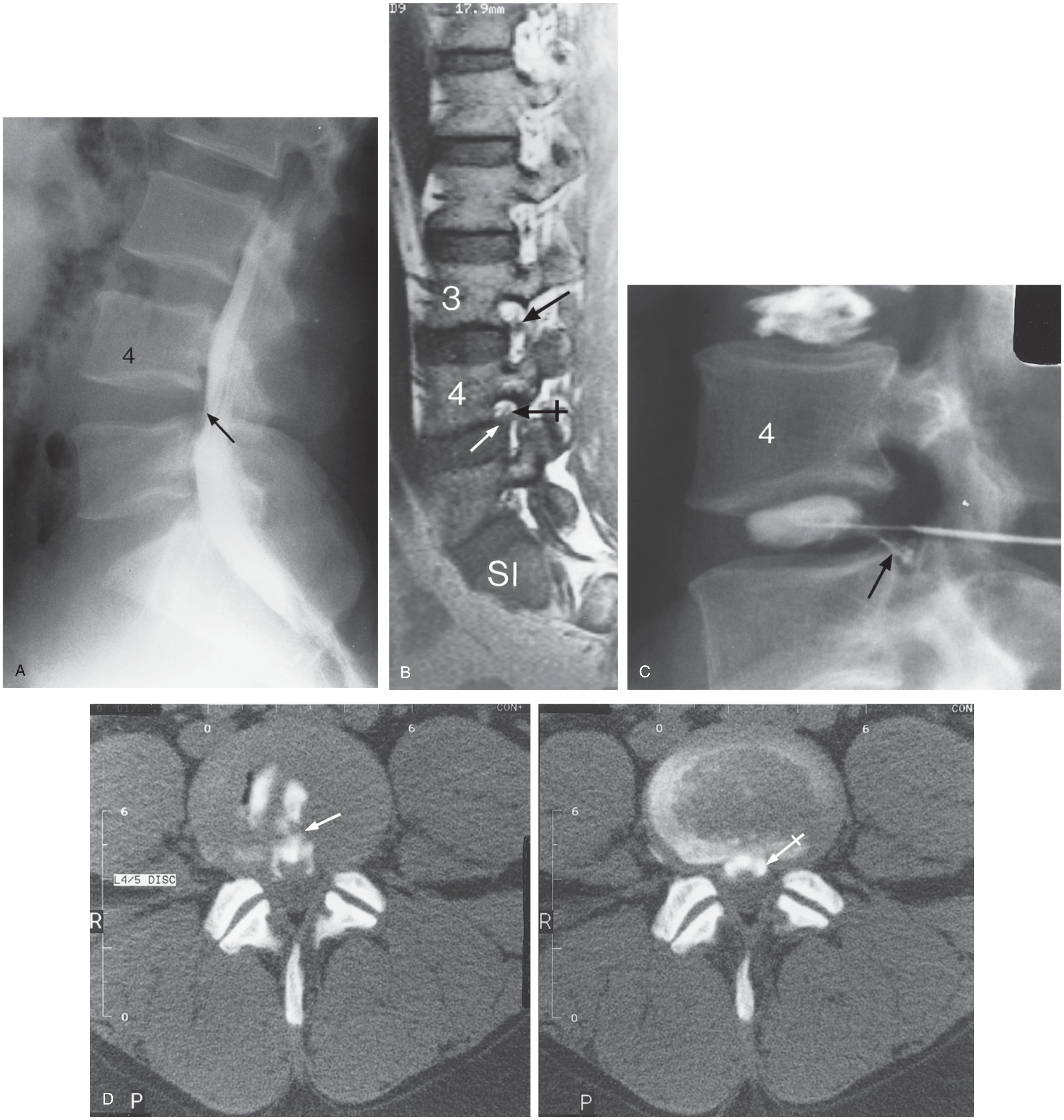

Figure 14.2 (A) Lateral lumbar spine myelogram contrast image showing some indentation of the thecal sac at the L4–5 level (arrow). (B) Lumbar spine MRI parasagittal T1-weighted image showing a normal intervertebral foramen at the L3–4 level with normal neural structures in the foramen (black arrow). Note the encroachment of disc material into the left L4–5 foramen (white arrow) with some pressure upon the nerve root at this level (tailed black arrow). Note how the disc material extends into the lower part of the intervertebral foramen narrowing its anteroposterior dimension considerably when compared with the L3–4 foramen. The nerve root at the L5–S1 disc level is normal, and lies within a normal intervertebral foramen. S1 = first sacral segment. (C) Lateral lumbar spine image of the discogram procedure at the L3–4 and L4–5 disc levels; at the L4–5 level, leakage of contrast material posteriorly is noted (arrow). (D) CT axial post-discogram images of the contrast in the L4–5 disc. This shows some internal disc disruption (white arrow) with posterior leakage of contrast material (tailed arrow) with a full thickness tear of the posterior disc fibres and reproduction of his pain.

Neuropathic pain is different to complex regional pain syndrome (CRPS) (reflex sympathetic dystrophy syndrome) for which there is no cure; it is a chronic pain condition in which high levels of nerve impulses are sent to an affected site due to dysfunction in the central or peripheral nervous systems (Grayson 2007).

KEY POINTS

1. The results of spinal surgery are unpredictable, even when the patient sees a good surgeon.
2. The long-term results of standard lumbar discectomy are not very satisfying (Loupasis et al 1999), supporting the notion that disc surgery involves a high rate of failure (Postacchini 2001).

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Case 15

Lumbosacral metastasis

COMMENT

There are three important lessons to be learned from this case, viz: (i) beware of spinal night pain, (ii) always carefully examine imaging films and never just read the report, and (iii) remember that laboratory tests can be essentially normal in spite of serious pathological processes.

PROFILE

A 40-year-old female scientist of average build who is a non-smoker and a non-drinker.

PAST HISTORY

She had always been very fit.

PRESENTING COMPLAINT(S) (Fig. 15.1)

Constant chronic central low back pain with a 'burning and aching' quality following a fall approximately 1 year prior to consultation. She had seen a chiropractor for low back manipulation and her general medical practitioner for non-steroidal anti-inflammatory drugs but without any relief of pain. The pain is of variable intensity and she was beginning to develop pain in the right buttock and hip joint region extending to the lateral aspect of the thigh. On awakening in the morning, her back pain was minimal, but as the day progressed her pain increased, even though she did not perform heavy work as an office worker.

The low back pain is aggravated by any sudden movements or twisting and with sneezing and coughing. The low back pain increases with menstruation. She takes two analgesics on going to bed in order not to be awakened by low back pain during the night. She had had a cortisone injection into the right hip joint for bursitis but without any relief.

AETIOLOGY

This was considered to be due to having fallen approximately 1 year ago, and the significance of night pain previously had been overlooked.

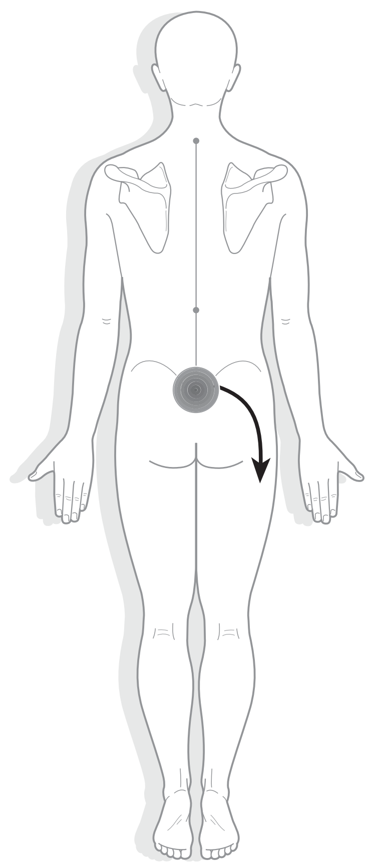

Figure 15.1

EXAMINATION

The deep tendon reflexes in the lower extremities were normal as was muscle power. Pinprick sensation and vibration sensation were normal. There was no evidence of leg length inequality. Sitting in the slumped forward position did not aggravate the low back pain; the addition of SLR did not elicit any increase in low back pain. Active supine SLR was of full range and painless. Hip joint tests were normal apart from slight discomfort on performing the right Fabere test.

Lumbar spine active ranges of movement were as follows:

1. Flexion was full and painless.
2. Extension was limited by approximately 10% due to slight low back 'discomfort'.
3. Right lateral bending was limited by approximately 10% but painless, while left lateral bending caused some low back 'discomfort' with approximately 10% limitation of movement.

The abdomen appeared to be normal.

CLINICAL IMPRESSION

Lumbosacral significant pathology in view of the night pain and because the deep tendon reflexes, SLR, vibration sensation and pinprick sensation were normal.

IMAGING REVIEW

Pelvis and lumbar spine radiographs performed 12 months prior to consultation were reported as normal apart from 'minimal narrowing of the L5–S1 intervertebral disc'. However, upon review, the oblique view indicated some minor concavity of the right anterolateral part of the L5 body, even though the lateral view (Fig. 15.2A) and other views appeared to be normal.

WHAT ACTION SHOULD BE TAKEN?

In view of the night pain that required two Nurofen tablets upon going to bed, in order to minimize the pain, the following laboratory (Boxes 15.1 and 15.2) and imaging tests were performed.

Repeat plain radiographic anteroposterior and lateral views were taken of the lumbosacral spine; the report stated 'no significant bony abnormality shown'. However, this was clearly incorrect, as there was a minor concave appearance of the anterior aspect of the L5 vertebral body (see Fig. 15.2B). Therefore, a CT scan and a bone scan were ordered.

The CT soft tissue axial scan at L5–S1 (Fig. 15.2C) and the bone window scan (Fig. 15.2D) both showed some loss of the right anterolateral margin of the L5 body with an adjacent soft tissue mass.

The bone scan views (Fig. 15.2E and F) showed 'very minimally increased uptake in the superior aspect of the anterior part of the L5 vertebral body, suggesting a minor degree of non-specific osteoblastic activity in the anterior aspect of the L5 body'. A focus of increased uptake was also seen in the region of the T10 vertebral body. Therefore, a lumbar MRI study was ordered. The MRI (Fig. 15.2G and H) showed: Pre-vertebral subligamentous mass eroding the anterior aspect of the L5 vertebral body with maintenance of the adjacent disc spaces, suggesting that an

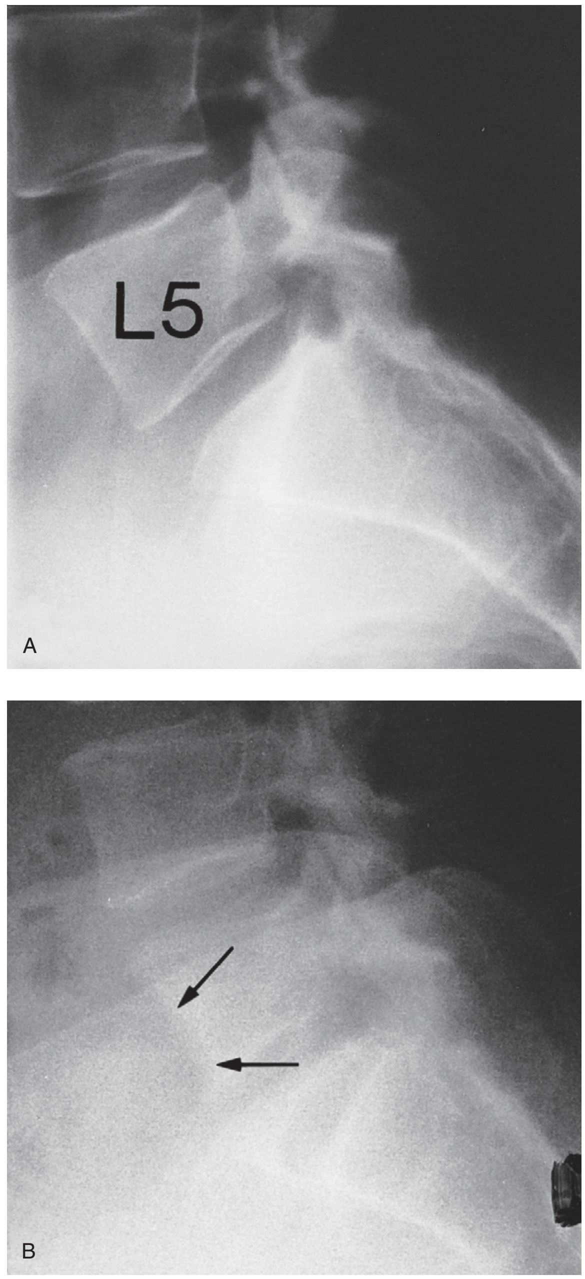

Figure 15.2 (A) Lateral plain X-ray image of the L4–S2 level. Note the 'minimal narrowing of the L5–S1 intervertebral disc'. (B) Lumbosacral spine lateral plain X-ray image of the region taken 12 months after the X-ray shown in (A). Note the minor concave appearance of the anterior aspect of the L5 vertebral body (arrows). *(Continued)*

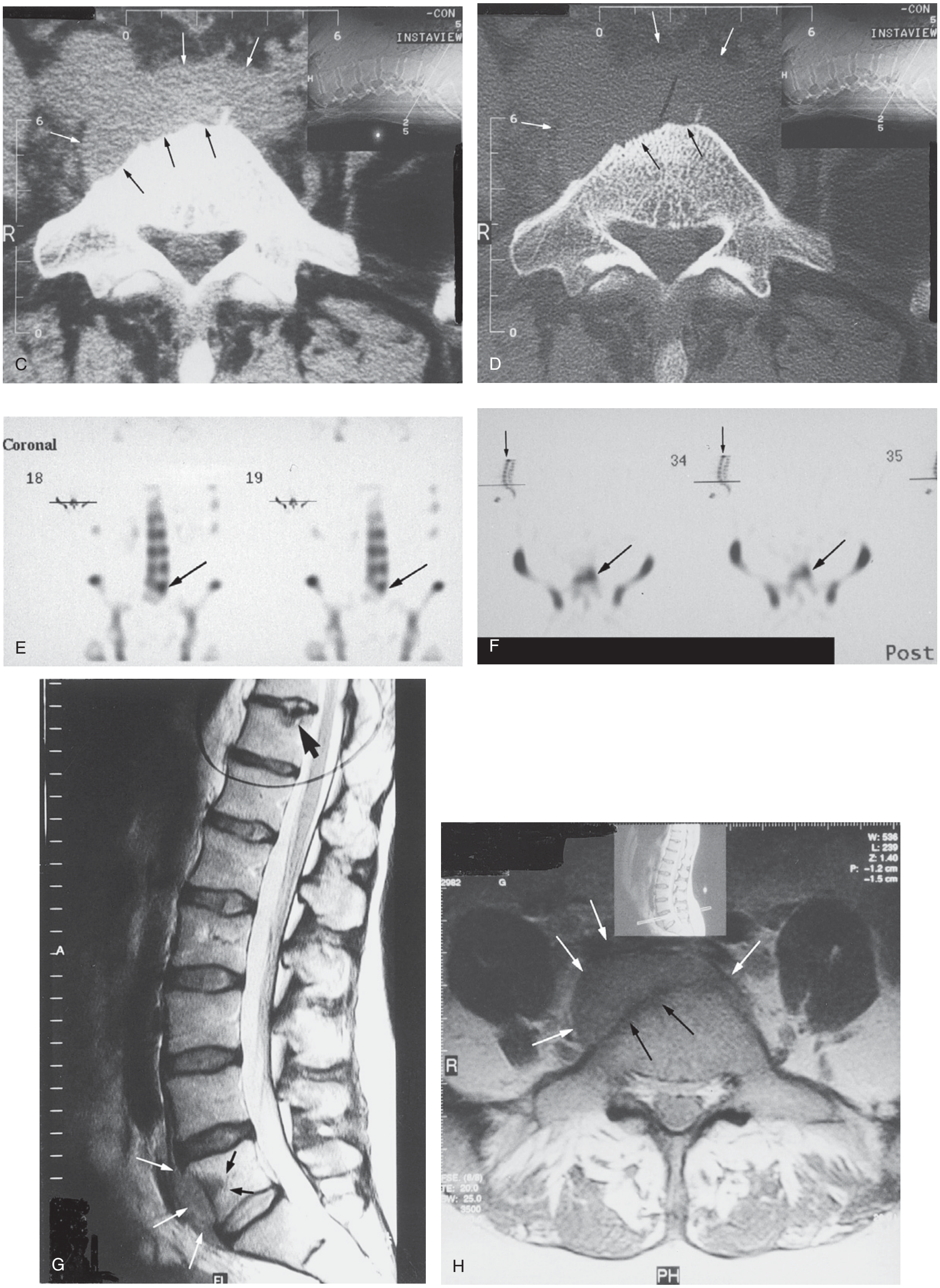

Figure 15.2 Cont'd (C) A CT axial soft tissue image through the upper one-third of the L5 vertebral body. Note the pre-vertebral soft tissue density, with similar density to muscle tissue, anterior to the vertebral body margin (white arrows) and the bony erosion along the anterior cortical margin of the L5 body (black arrows). R = right side of patient. (D) CT axial bone window image showing the features mentioned in (C). R = right side of patient. (E) Bone scan coronal images showing increased uptake in the L5 vertebral body (arrow). (F) Bone scan horizontal image through the L5 body showing the increased uptake in the L5 vertebral body (arrows) and what appears to be a hotspot in the T11 body, although this is at the very edge of the SPECT scanning and therefore could be due to possible 'edge effect' (short black arrows). (G) Lumbosacral spine MRI sagittal T2-weighted image showing 'significant soft tissue mass in the subligamentous location' anterior to the L5 vertebral body (white arrows) with some post-contrast enhancement within the L5 vertebral body consistent with inflammatory response related to this erosive process (black arrows). Note the Schmorl's node in the upper endplate of the T11 vertebra. (H) MRI axial T1-weighted image through the upper one-third of the L5 vertebral body, showing the subligamentous soft tissue mass (white arrows) with erosion of the anterior aspect of the right half of the vertebral body (black arrows). R = right side of patient.

Box 15.1 Chemical pathology

Specimen type		Units	Reference range
ALP (alkaline phosphatase)	69	U/l	(30–115)
C-reactive protein	5.8	mg/l	(0–10)

Box 15.2 Haematology

		Units	Reference range
Haemoglobin	120	g/l	(115–165)
White cell count	7.6	$\times 10^9$/l	(3.5–12.0)
Platelets	329	$\times 10^9$/l	(150–400)
Haematocrit	0.37		(0.35–0.47)
Red cell count	4.3	$\times 10^{12}$/l	(3.9–5.6)
MCV (mean corpuscular volume)	86	fl	(81–97)
MCH (mean corpuscular haemoglobin)	28	pg	(27–34)
MCHC (mean corpuscular haemoglobin concentration)	322	g/l	(310–360)
Neutrophils	4.16	$\times 10^9$/l	(2.5–8.0)
Lymphocytes	2.83	$\times 10^9$/l	(1.2–4.0)
Monocytes	0.54	$\times 10^9$/l	(0.0–0.8)
Eosinophils	0.07	$\times 10^9$/l	(0.0–0.6)
Basophils	0.01	$\times 10^9$/l	(0.0–0.3)
ESR (erythrocyte sedimentation rate)	18	mm/hour	(2–20)
Ovarian tumour markers were very slightly increased			

inflammatory process is less likely than a neoplastic process with metastatic deposit or subligamentous lymphoma; consideration should be given to a granulomatous type infection. An acute Schmorl's node is demonstrated involving the upper endplate of T11 (Fig. 15.2G) accounting for the hot spot seen on the bone scan.

Upon consultation with the radiologist about the T11 'Schmorl's node', he felt confident that this is a Schmorl's node and not a secondary deposit.

In view of these findings, a CT-guided fine needle aspiration was performed for microscopic cytology examination and Ziehl–Neelsen's stain for acid-fast bacilli. This showed 'inflammatory cells and inflammatory debris. Some apoptotic cells with epithelial appearance very suspicious of neoplasia rather than infective or just purely inflammatory condition. No acid-fast bacilli seen; no growth and cultures. Follow-up suggested'.

A chest X-ray study and a CT thorax were ordered to look for possible metastases; these showed 'clear lungs and a normal heart and mediastinum with no significant axillary, mediastinal or hilar adenopathy; superior aspect of liver and spleen unremarkable'. A CT abdomen and pelvis found 'the liver, spleen, pancreas and gall bladder to be normal' and confirmed the 'ill-defined soft tissue anterior to the L5 body'.

The patient was advised to have a gynaecological examination by a gynaecologist and a hysteroscopy showed no abnormality.

She was referred to an orthopaedic surgeon for review and she was sent for a nuclear medicine whole body scan using cells labelled with technetium and single proton emission computerized tomography (SPECT) that 'suggested infiltration of the L5 body is more likely than inflammatory pathology; the liver is enlarged'.

A follow-up needle aspiration biopsy found tumour cells of undifferentiated epithelial origin, so a diagnosis was made of a secondary tumour from an unknown primary location.

DIAGNOSIS

Ovarian carcinoma with metastatic disease.

TREATMENT

The patient was referred to an oncologist for ongoing management.

Note

On comparing retrospectively the use of MR imaging, laparotomy reassessment, and serum CA-125 values in predicting the presence of residual tumour in women who have been treated for ovarian cancer, Low et al (2005) found that gadolinium enhanced spoiled gradient-echo MR imaging depicts residual tumour with an accuracy, positive predictive value, and negative predictive value that are comparable to those of laparotomy and superior to those of serum CA-125 values alone.

KEY POINTS

1. Beware of night pain!
2. Always carefully examine imaging in spite of a 'normal' report.
3. Routine laboratory tests can be essentially normal in spite of serious pathology.

Reference

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Case 16

Lytic L5 Grade 1 spondylolisthesis of long standing

COMMENT

Spinal surgery can be helpful in carefully selected cases.

PROFILE

A 44-year-old slim male manual worker with reasonably good muscle tone who smokes approximately 50 cigarettes per week and drinks beer.

PAST HISTORY

He said he had fallen through a ceiling 3 years ago but did not injure his low back although he had previously experienced intermittent low back pain.

PRESENTING COMPLAINT(S) (Fig. 16.1)

Severe low back pain that radiates to the right buttock and intermittently to the ankle since lifting heavy boxes at work 1 month ago.

Walking up steps aggravated the low back pain, as did coughing. On awakening in the morning he experienced low back pain. Heat was of no help. Cold aggravated the low back pain so he wore a low back support belt during the winter months to keep his lower back warm.

He had been treated with rest, non-steroidal anti-inflammatory drugs and analgesics which had given him some relief and this was followed by physiotherapy treatment. He returned to work approximately 2 weeks later but found that his back was too painful for working so he stopped.

AETIOLOGY

Sudden onset of severe low back pain which came on while lifting heavy boxes 1 month ago.

EXAMINATION

On deep palpation of the paraspinal muscles, there was tenderness throughout the lumbosacral spine and in the right buttock centrally. Power in the lower extremities was normal and SLR was to 90° bilaterally before he

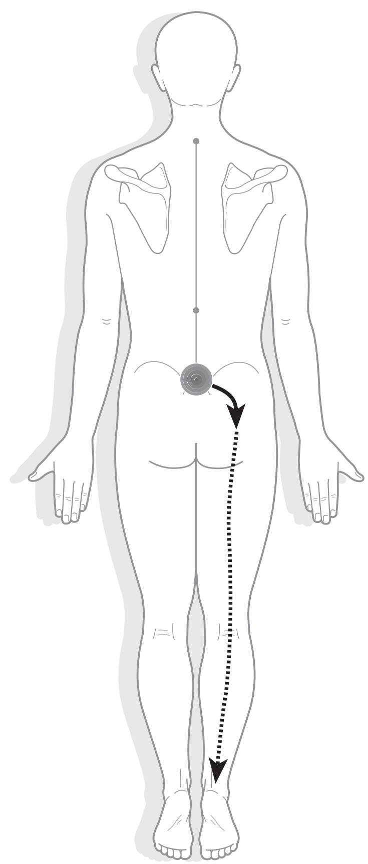

Figure 16.1

experienced low back pain on right SLR. The right knee and ankle deep tendon reflexes were reduced to 1 plus (2 plus being normal).

IMAGING REVIEW

A plain film radiograph showed a Grade 1 spondylolisthesis of L5 on S1 with bilateral pars defects (Fig. 16.2A). A bony spicule projecting into the L5–S1 intervertebral foramen was present on the right side. The L5–S1 intervertebral disc was very thin and there were anterior osteophytes adjacent to it on the L5 and S1 bodies.

CLINICAL IMPRESSION

Grade 1 spondylolisthesis of L5 on S1 with right L5 nerve root entrapment and probable L5–S1 disc bulge/ protrusion.

WHAT ACTION SHOULD BE TAKEN?

A CT scan was ordered and this showed a large central and right-sided disc herniation at the lumbosacral level.

The patient was referred to an orthopaedic surgeon for an opinion regarding possible stabilization of the spondylolisthesis and removal of the bony spicule projecting into the right L5–S1 intervertebral foramen and a discectomy at this level.

DIAGNOSIS

Grade 1 spondylolisthesis with right sided L5-S1 posterior intervertebral disc herniation and bony spicule causing right L5 nerve radiculopathy.

TREATMENT AND RESULTS

A posterolateral spinal fusion was performed from L4 to the sacrum (Fig. 16.2B) with decompression of the nerve roots on the right side, and removal of the central and right-sided disc herniation. The post fusion result shown in Fig. 16.2B shows the pedicle screw fixation from L4 to S1 with a patent intervertebral foramen following removal of the bony spicule.

He reported that he had no more leg pain and also had excellent relief of back pain.

Following surgery, this man periodically suffered from low back pain and responded well to needle acupuncture treatment on a symptomatic basis.

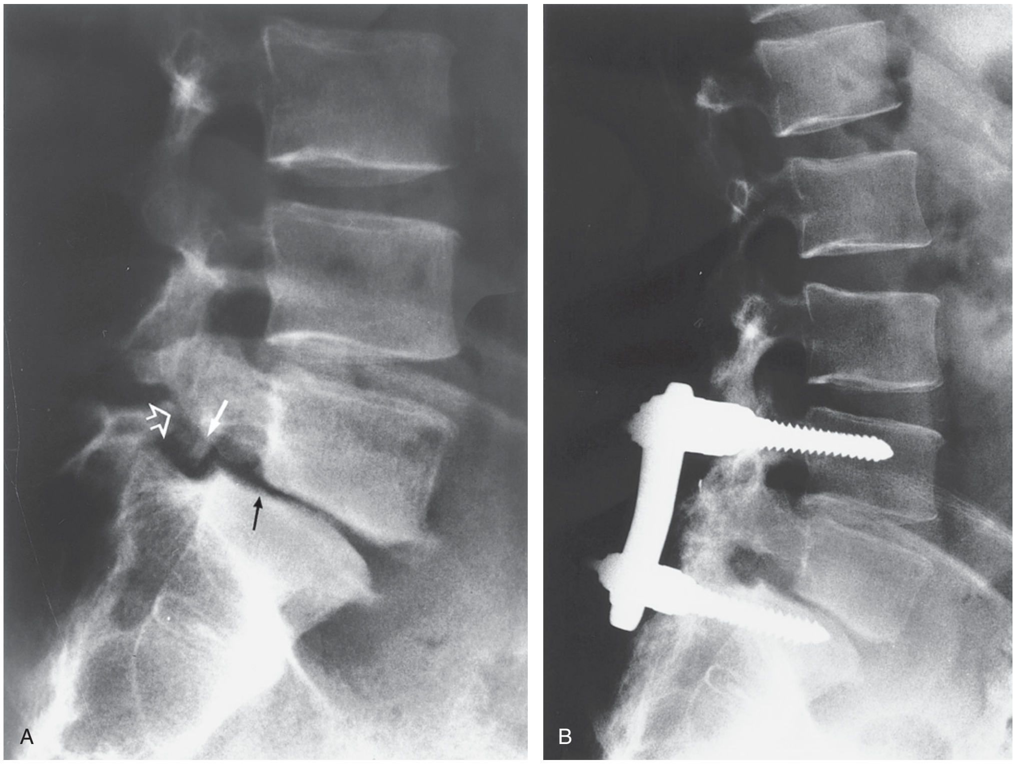

Figure 16.2 (A) Lumbosacral spine lateral plain X-ray image showing the grade 1 spondylolisthesis of L5 on S1 with advanced thinning of the intervertebral disc at this level and osteophytic lipping at the anterior margins of the L5 and S1 bodies. The open arrow shows the fractured pars interarticulares. The white arrow indicates the bone spicule projecting into the right L5–S1 intervertebral foramen. The black arrow shows the degree of spondylolisthesis, i.e. the L5 body has moved approximately one quarter of the distance along the sacral body, hence the grade 1 classification according to Meyerding's (1932) classification (Yochum & Rowe 1996, Giles 1997). (B) A post-surgical lumbosacral spine lateral plain X-ray image showing the pedicle screw fixation from L4 to S1 and the patent L5–S1 intervertebral foramen following removal of the bony spicule.

KEY POINT

In carefully selected cases, spinal surgery can be successful.

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Case 17

Lytic L5 Grade 1 spondylolisthesis in a young man

COMMENT

Be wary of radiology reports referring to a so-called 'pseudodisc' appearance, as a real disc bulge or protrusion may well be present.

PROFILE

A 21-year-old male sedentary worker who does not smoke cigarettes and only drinks alcohol socially.

PAST HISTORY

He has no recollection of sustaining a low back injury but is very involved in sporting activities such as rugby.

PRESENTING COMPLAINT(S) (Fig. 17.1)

Intermittent low back pain, for some months, with some referral of pain into the right buttock, right knee and right calf (L5) laterally with some tingling in the right little toe (S1). Bowel and bladder function were normal and there was no night pain. He had experienced a recurrence of his symptoms a couple of weeks before presentation and, therefore, had sought medical and physiotherapy treatment based on a diagnosis of musculoligamentous injury.

AETIOLOGY

The aetiology is unknown but he is a keen rugby player apart from participating in other sporting activities.

EXAMINATION

In the erect posture there was no evidence of leg length inequality, pelvic obliquity or scoliosis. Straining the sacroiliac joints did not elicit any sacroiliac joint pain but elicited some increase in lumbosacral pain.

Lumbar spine active ranges of movement were as follows:

1. Flexion was of full range and painless.
2. Extension caused a slight increase in low back pain at full range.
3. Left and right lateral bending were of full range and painless.
4. Left and right rotation were of full range and painless.

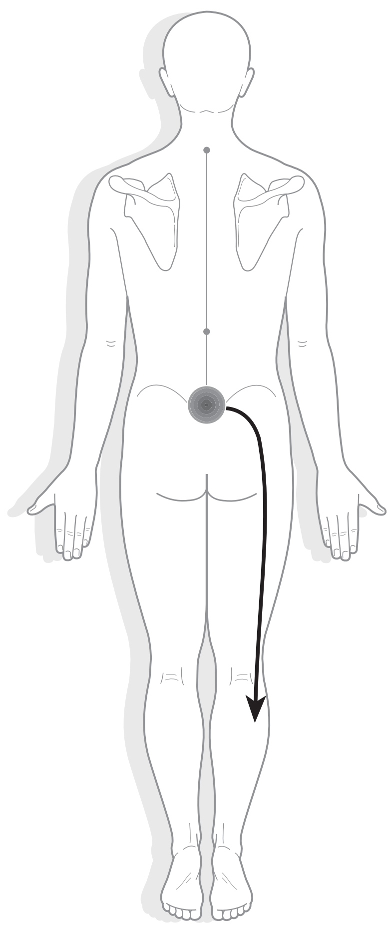

Figure 17.1

On forward bending it was noted that there appeared to be a slight 'step' at the L4–5 level, suggestive of a spondylolisthesis of L5 on S1.

Deep palpation of the paraspinal muscles at the L5 level elicited local tenderness. Percussion at the L5 level elicited tenderness. The deep tendon reflexes in the lower extremities were normal as was the case with the plantar response. Pinprick sensation indicated hypoaesthesia in the right L5 dermatome of the lower limb. Light touch also suggested a minor loss of sensation in the same area of the right calf and on the lateral aspect of the right foot. The Valsalva manoeuvre did not elicit any increase in symptoms. Straight leg raising was slightly limited on the right side due to pain in the right calf. Straight leg raising with the addition of cervical spine flexion did not cause any significant symptoms.

IMAGING REVIEW

No imaging had previously been performed.

CLINICAL IMPRESSION

Probable spondylolisthesis of L5 on S1 with associated pars interarticularis defects.

WHAT ACTION SHOULD BE TAKEN?

Plain film anteroposterior, lateral, and oblique radiographs were ordered with a specific request for an evaluation of the L5–S1 pars interarticulares and L5–S1 disc space height.

The lumbar spine plain X-ray report stated: 'Bilateral pars defects at L5 with a Grade I spondylolisthesis of L5 on S1. The intervertebral disc spaces and vertebral body heights are maintained'.

In view of the X-ray report and the symptoms of tingling in the right little toe, a lumbar CT scan was requested with reverse gantry angle scans through the pars interarticulares of L5 to better demonstrate the L5 pars defects as well as any nerve root compromise. The CT scan showed no disc or zygapophysial joint pathology at the L3–4 and L4–5 levels. However, the lateral scout view showed the Grade I spondylolisthesis of L5 on S1 (Fig. 17.2A). This figure also shows the routine angle of CT slices. Although the routine axial view at L5–S1 was not conclusive (Fig. 17.2B), 'the reverse gantry view (Fig. 17.3A and B) clearly demonstrated the bilateral L5 pars defects with sclerotic margins reflecting longstanding pathology. A pseudodisc appearance secondary to the spondylolisthesis is noted. There is no evidence of central or lateral stenosis'.

In view of the right leg symptoms, and the radiologist's comment that there was a 'pseudodisc' appearance at the

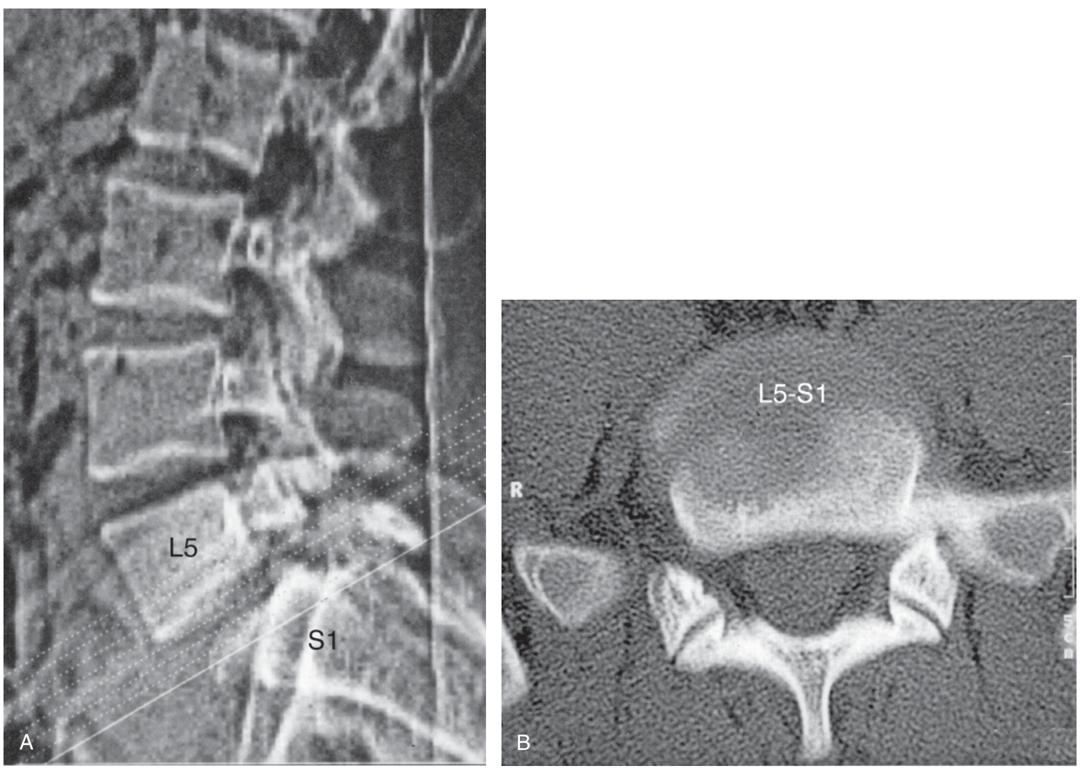

Figure 17.2 (A) Lumbar spine CT lateral scout image showing the angle of routine CT slices (broken lines) and the Grade I spondylolisthesis of L5 on S1. (B) Lumbar spine CT routine axial image at L5–S1 level; this was not conclusive for L5 bilateral pars defects. R = right side of patient.

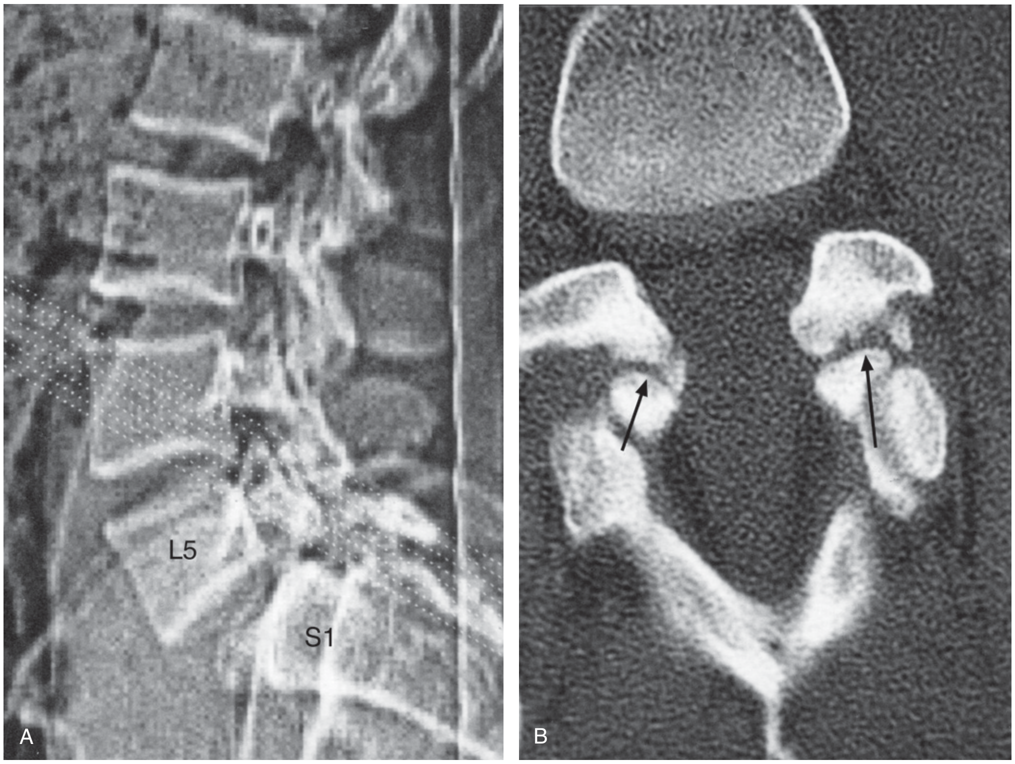

Figure 17.3 (A) Lumbar spine CT lateral scout image showing the reverse gantry angle CT scans (broken lines) through the pars interarticulares of L5. (B) Lumbar spine CT reverse gantry angle image at L5–S1 clearly demonstrating the bilateral L5 pars defects (arrows) with sclerotic margins reflecting longstanding pathology. A 'pseudodisc' appearance secondary to the spondylolisthesis was reported (white arrows).

L5–S1 intervertebral disc level, an MRI lumbar spine was performed. The report's Conclusion stated: 'L5 pars interarticularis defects with Grade I spondylolisthesis of L5 on S1 with bilateral foraminal narrowing and impingement of the right L5 root in the narrowed foramen. At the L5–S1 level there is a broad-based posterior annular bulging and a faint annular tear'.

DIAGNOSIS

L5–S1 bilateral pars interarticularis defects with a Grade 1 spondylolisthesis of L5 on S1 with impingement of the right L5 root in the narrowed intervertebral canal by the broad-based L5–S1 disc posterior annular bulge.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was told the following:

1. The bilateral pars interarticularis defects are, in all likelihood, due to traumatic fracture of the left and right pars interarticulares.
2. The recent onset of pain extending into the right buttock, calf and knee with tingling in the right little toe is most likely due to the broad-based posterior annular bulging with minor compression of the right L5 root in the foramen, i.e. soft tissue changes secondary to the Grade I spondylolisthesis of L5 on S1.

He was advised not to play contact sport but rather to get exercise by swimming and walking, as well as performing exercises to maintain muscle tone (see Case 1). He was also advised not to lift heavy items, for example at home or when travelling.

He was advised to have chiropractic mobilization of his spine if his symptoms did not settle with the above treatment regime. However, at four years post consultation he has not required mobilization in view of modifying his sporting activities and performing the prescribed exercises.

Note

1. It has been stated by some radiologists and other clinicians that CT imaging of the lumbar spine may show 'pseudodisc' appearance but it should be noted that this so called 'pseudodisc', in all likelihood, represents a real broad based posterior bulging of the annulus. This is exemplified in this case where the CT scan was reported as showing a 'pseudodisc' but the MRI report clearly confirmed that there was indeed a broad-based posterior annular bulging at L5–S1.

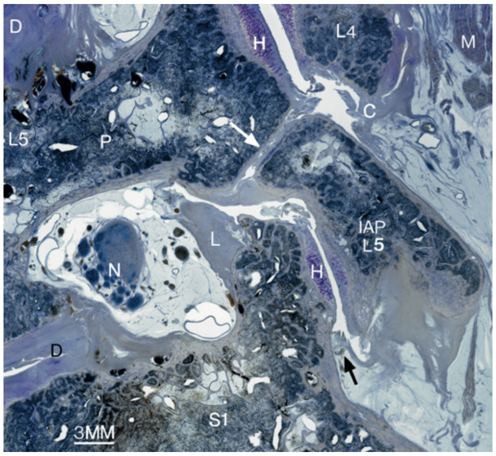

Figure 17.4 A 200-micron thick histopathology section cut in the parasagittal plane to include the pars interarticularis (isthmus) defect (white arrow) which has developed fibrocartilagenous type tissue on both bony surfaces. There is no true hyaline articular cartilage but there is some fibrous tissue crossing the pars defect. There is a distinct cortex on each side of the isthmus defect. C = fibrous joint capsule (disrupted); D = intervertebral disc; H = hyaline articular cartilage; IAP L5 = inferior articular process of the 5th lumbar vertebra (L5); L = ligamentum flavum (disrupted due to the pars defect); L4 = inferior articular process of the 4th lumbar vertebra; M = muscle; N = neural structures within the intervertebral canal; P = pedicle of L5; S1 = first sacral segment. Black arrow shows a synovial fold. (Ehrlich's haematoxylin and light green counterstain.) (Reproduced with permission from Giles L G F 1997 Miscellaneous pathological and developmental (anomalous) conditions. In: Giles L G F and Singer K P (eds) Clinical anatomy and management of low back pain. Edinburgh, Butterworth-Heinemann, p 196–216.) See also colour plate section Fig. vii.24.

2. A histopathology section through a postmortem specimen illustrating spondylolysis shows how the pars interarticularis (isthmus) defect develops fibrocartilagenous tissue on both bony surfaces of the defect (Fig. 17.4). The pars interarticularis defect may gradually increase due to forward displacement of the vertebral body on the sacrum (Adams & Hamblen 2001), especially during adolescence, and it sometimes reaches a severe degree. There may be minor irritation of one of the issuing nerves, with consequent sciatica (Adams & Hamblen 2001).
3. It has been shown in the literature that spondylolisthesis with bilateral pars defects is traumatic in origin and that it is not an anomaly or a developmental finding. For example, Rosenberg et al (1981), who examined radiographs of patients that had never walked, found that no spondylolisthesis was detected, compared to a 5.8% incidence in the general ambulatory population. Therefore, they concluded that 'spondylolisthesis represents a fatigue fracture resulting from activities associated with ambulation'.

Had this young man not been involved in rugby, the previously asymptomatic spondylolisthesis at the L5–S1 level may well have remained asymptomatic, as the clinical features of spondylolisthesis are inconstant but, when symptoms do occur, they take the form of chronic backache with or without sciatica (Adams & Hamblen 2001). Displacement may gradually increase due to forward displacement of the vertebral body on the sacrum, especially during adolescence, and it sometimes reaches a severe degree.

KEY POINTS

1. Be aware of the advantage of a reverse gantry angle CT examination versus a routine angle CT examination for pars interarticulares defects.
2. Be aware of the description on CT of a 'pseudodisc' as it may well be misleading with respect to real disc pathology.
3. To show a stress fracture in the pars interarticulares, the best MR imaging technique is a short-time inversion recovery (STIR) technique.

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Case 18

Lumbosacral intervertebral disc protrusion

COMMENT

Never label a patient as a malingerer unless you are absolutely certain of your facts.

PROFILE

A 25-year-old, tall, overweight female sedentary worker who does not smoke and hardly ever drinks alcohol.

PAST HISTORY

Her past history was unremarkable.

PRESENTING COMPLAINT(S) (Fig. 18.1)

She presented, upon referral from her general medical practitioner, with considerable low back pain and right greater than left leg pain due to bending and twisting in a cramped space. The leg pains extend down the posterolateral aspects of each leg to the heels. Apparently, it felt as though there was a 'permanent band of pain' across her low back as far as her hips, worse on the right side.

She had undergone an epidural block injection that caused numbness on the lateral aspect of her right thigh, just above the knee joint, and she had become unwell due to a severe post-epidural infection that required two antibiotics, via a drip, to resolve the infection. Approximately 1 month before consultation, she had been admitted to hospital for 2 weeks due to an acute recurrence of her low back and leg pains; pethidine had been administered 4-hourly at one period because of her severe low back pain syndrome.

She had difficulty in getting to sleep because of the low back pain, so was taking two 5 mg Valium tablets before going to bed. This resulted in her having to take laxatives even though she was eating fruit and wholemeal bread. Coughing and sneezing caused an increase in low back pain. She stood with her right knee slightly flexed due to pain radiating to the right heel from her low back.

She had tried acupuncture but without long-lasting relief.

She now wore a rigid back support that does not allow her to bend or lift as her general medical practitioner thought this might help and, in fact, it does if she wears it at all times. The only time she takes it off is when she goes to bed and when she showers. A transcutaneous

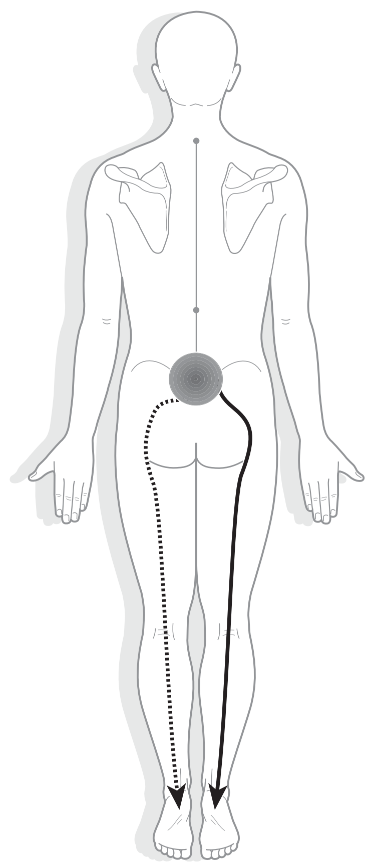

Figure 18.1

electrical nerve stimulator (TENS) and physiotherapy were of no help. Bladder and bowel functions were normal apart from constipation brought about by taking Valium. She tries not to use medication as she does not feel well on it and, when she recently took an analgesic, she developed an allergic reaction and the skin began to peel on the palms of her hands.

She had seen a number of neurosurgeons and orthopaedic surgeons, none of whom considered that surgery was a good option and she had, in fact, been labelled as a malingerer by the orthopaedic and neurosurgeons that she had consulted; this greatly distressed her and her family.

AETIOLOGY

The patient had first felt low back pain when working in a cramped space in which she had to bend and twist her spine. She said that, because she is tall, bending and twisting in the cramped space had placed a lot of strain on her low back. The work place was cramped because two people worked at the same time in a confined space. Apparently the pain began early during the course of her employment and it rapidly increased during several months of repetitive bending and twisting.

EXAMINATION

She stood up cautiously from the chair and removed the rigid back support. When she lay down on the examination table she was equally guarded about her movements and found it too painful to allow her right leg to lie flat on the table, so she kept it slightly flexed at the knee. SLR was limited by low back pain to 25° elevation (right) and to 30° (left). The slump test elicited considerably increased low back pain at the lumbosacral area; the addition of SLR was greatly limited bilaterally due to increased low back pain. She could not perform active bilateral SLR as this elicited a great deal of low back pain. Forward bending of the cervical spine, in the supine position, elicited an increase in low back pain. Bilateral hip flexion was painful as soon as movement was felt at the lumbosacral region. The posterior tibial and dorsalis pedis pulses were normal. The left knee jerk (L2,3,4) was difficult to elicit, with only a slight response. The right knee jerk was normal, as was the case with the ankle jerks (S1,2) bilaterally. Pinprick sensation of the legs was normal, apart from an area of decreased sensation on the lateral aspect of the right thigh (L2,3) just above the knee joint, and her left fifth toe (S1). Vibration sensation at her ankles was normal. Her spine was very tender to palpation in the L4–S1 region.

Active lumbar spine ranges of movement were as follows:

1. Flexion – limited by 80% due to low back pain and her fingers could not reach to her knees.
2. Extension – limited by approximately 85% due to considerable low back pain.
3. Left and right rotation – limited by approximately 85% due to considerable low back pain.

There was no loss of power in her legs. The circumference of the calves, 11 cm below the knee cap, was 39.5 cm (left) and 40 cm (right); the thigh circumference was equal on both sides. Axial compression of the spine, due to pressing down on her shoulders, reproduced the low back pain. The abdomen was normal on examination.

IMAGING REVIEW

An MRI scan showed a moderately large central disc protrusion at the L5–S1 level that may be confined by the posterior longitudinal ligament which is displaced posteriorly (Fig. 18.2). The moderately severe degree of compression on the anterior aspect of the thecal sac is clearly visible. A much smaller posterior disc bulge at the L4–5 level was noted. The L5–S1 disc showed considerable desiccation as compared to the mildly desiccated L4–5 disc.

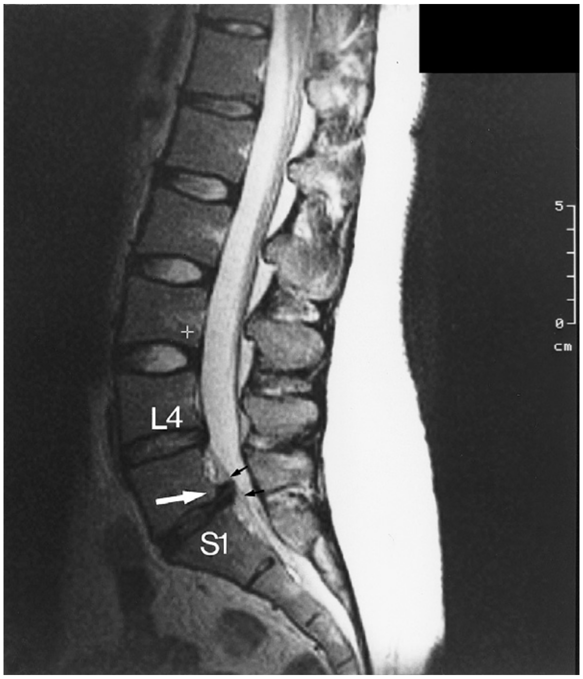

Figure 18.2 Lumbar spine MRI sagittal T2-weighted image showing the moderately large central disc protrusion at the L5–S1 level (white arrow) that may be confined by the posterior longitudinal ligament which is displaced posteriorly. The moderately severe degree of compression on the anterior aspect of the dural tube/thecal sac is clearly visible (black arrows). Note that the L5–S1 intervertebral disc shows more advanced desiccation than does the L4–5 disc.

CLINICAL IMPRESSION

L5–S1 central disc protrusion.

WHAT ACTION SHOULD BE TAKEN?

She was sent to another surgeon who said surgery would not solve her problem. Over a period of approximately 10 months she developed a 2-cm loss of right thigh circumference, so she was sent interstate for a neurosurgical opinion.

DIAGNOSIS

L5–S1 posterior intervertebral disc protrusion.

TREATMENT AND RESULTS

The surgeon to whom she was referred realized the significance of the L5–S1 posterior intervertebral disc protrusion and the associated clinical signs and symptoms and performed an L5–S1 discectomy.

The patient told me that the low back surgery was very helpful and that her pain had decreased dramatically. She occasionally felt slight pain in the right leg in the sciatic distribution and sometimes one foot felt 'colder' than the other. However, she stated that she is coping very well psychologically. In addition, she had considerably decreased her medication and had returned to work in a new light duties position. She still had some 'weakness' of her right leg intermittently, so she goes for frequent walks to exercise her low back and to strengthen her legs.

KEY POINTS

1. Over the last 30 years it has been my experience that patients only very rarely malinger.
2. A careful history, physical examination and appropriate imaging will differentiate between genuine patients and malingerers.
3. When the history, physical examination and appropriate imaging correlate well with the patient's symptoms, it is negligent to label a patient as a malingerer.
4. Because a patient has a work-related injury it should not be automatically assumed that the patient is a malingerer.

Further reading

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Case 19

Adolescent tethered cord syndrome

COMMENT

Always listen carefully to the patient, even when a complex and apparently bizarre symptom pattern is being described.

PROFILE

A 15-year-old schoolgirl who plays hockey and gets good grades for her school subjects.

PAST HISTORY

Her past history is non-contributory and she has no history of accidents or any serious infections. There is no history of bowel or bladder incontinence. Her general medical practitioner apparently did 'blood tests' approximately 2 months ago and said that there was nothing abnormal. An ultrasound scan of her abdomen performed in view of her unusual symptoms was normal. Her general medical practitioner considered that she had growing pains.

PRESENTING COMPLAINT(S) (Fig. 19.1)

Chronic (approximately 3 years) increasing thoracolumbar junction pain, of unknown aetiology, that spreads down to the lower lumbar spine especially on the right side. She also experiences pain extending bilaterally into the buttocks and posterior to the hip joints, with some intermittent bilateral pain 'around the lower rib cage' laterally and anteriorly. The thoracolumbar pain begins in the morning and becomes progressively worse during the afternoon until it becomes 'really bad' by approximately 7.30 p.m. Bearing down causes an increase in pain between the shoulder blades and at the thoracolumbar region with some increase in low back pain. Coughing aggravates the thoracolumbar junction pain. She feels generally 'tired' because of the pain which makes her feel 'weary'. She has taken Nurofen and Panadol without any relief. She is able to play hockey and this does not aggravate her symptoms while she is playing.

She thinks there is a night pain component (approximately 2.00 a.m.) but is not certain.

A secondary, and far less painful complaint, is some cervico-shoulder pain bilaterally, especially on the left side. There is no associated radiation and there are no arm symptoms. The minor neck pain has been present for approximately 6 months.

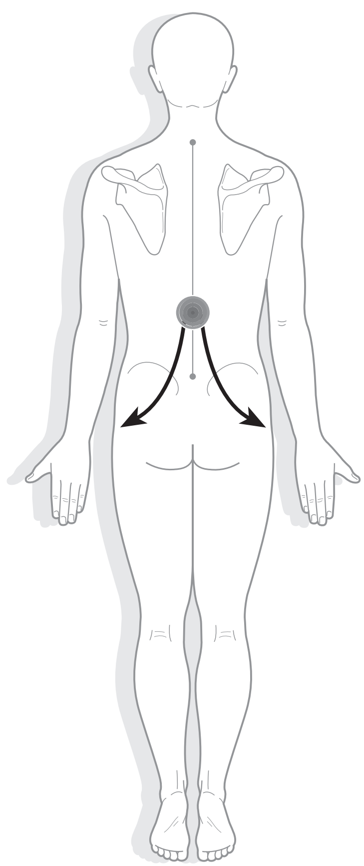

Figure 19.1

AETIOLOGY

Unknown. Gradual onset of progressive symptoms during the last 3 years.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or scoliosis. There was an increased lumbar lordosis. On the right side of the T10 region there was a faint skin blemish measuring 4.5 × 1.8 cm that does not blanch on pressure. She also had unusual horizontal striae across the thoracolumbar region. Toe and heel walking power were normal. There was no obvious muscle weakness in the lower extremities, although she appeared to have minor instability of the ankle joints with slight inversion of the right ankle. There was no osseous or soft tissue anomaly involving the feet. The deep tendon reflexes in the upper and lower extremities were normal. The plantar response was normal and pinprick sensation over the legs and the posterior part of the torso was normal. As she stated that she did not have any difficulty with sphincter control, pinprick sensation was not performed in the perianal area. Vibration sensation at the ankles was normal.

When seated in the slumped forward position, this aggravated the thoracolumbar pain; the addition of SLR elicited a significant increase in this pain. Supine SLR elicited low back pain at approximately 50° elevation of the left and right legs, respectively. Bilateral hip flexion aggravated her low back pain. Bilateral hip flexion with the addition of cervical spine flexion elicited an increase in the thoracolumbar junction pain. Milgram's bilateral active SLR elicited some low back pain but nothing significant. The Naffziger jugular compression test elicited some increase in thoracolumbar junction pain. Hip joint mobility tests were normal. There was no paravertebral muscle spasm. Deep palpation of the paraspinal muscles between T10 and L5 elicited local tenderness. Pressure on the lumbosacral area with the patient prone caused some vague lumbar discomfort.

Active ranges of lumbar spine movement were as follows:

1. Flexion elicited an increase in thoracolumbar pain when her fingers reached halfway down the tibia.
2. Extension elicited an increase in pain at approximately the L3–4 level.
3. Left and right lateral bending elicited some pain to extend from L2 to L5.
4. Left and right rotation elicited pain in the approximately L2 to L4 region.

On close questioning, she had noted the following changes in her symptoms:

- She can walk less distance due to feeling 'tired', but walking does not cause numbness in the legs.
- Flexing the spine aggravates the thoracolumbar pain and extension causes some midlumbar spine pain.
- The thoracolumbar pain extends to the lumbosacral area, the sacrum and then to both inguinal areas and hips, with some knee pain.
- She cannot lie on her back at night due to lumbar spine pain, so she lies on her left side in a fetal position.
- Two of the three 'B' postures (Yamada et al 1996), i.e. sitting in the Buddha pose and Bending slightly over the sink aggravated the lumbar spine pain; holding a small Baby or dog did not aggravate the lumbar spine pain.
- Sitting in a slouched position caused an increase in lumbar spine pain.
- Being driven in a car over an uneven surface, or for a long distance, caused an increase in thoracolumbar pain.
- After standing for 30 minutes, for example while cooking, the thoracolumbar pain becomes aggravated and, after an hour, extends into the hips.
- That she had an exaggerated lumbar lordosis.

IMAGING REVIEW

Plain X-ray films taken 10 months previously were reported as normal. However, spina bifida occulta at the first sacral level was noted (Fig. 19.2A). The lateral lumbosacral view (Fig. 19.2B) did not show any abnormality.

CLINICAL IMPRESSION

1. A high suspicion of a functional tethered cord syndrome in view of the history, unusual symptoms and signs and the spina bifida occulta at S1.
2. A differential diagnosis of a neurofibroma at the T10 level in view of the faint skin blemish on the right side of the spine at this level.
3. Possible mechanical thoracolumbar joint pain.

Tests for nerve root tension did not provide a typical response for a space-occupying lesion, so such a lesion was considered unlikely.

WHAT ACTION SHOULD BE TAKEN?

Because of the clinical suspicion of a functional tethered cord syndrome an MR lumbar spine and sacrum was ordered. This was reported as follows: 'The vertebral bodies and disc spaces appear intact with no desiccation or narrowing. No focal disc herniation. The exit foramina and spinal canal appear normal in size. No paraspinal soft tissue abnormality. The conus is normal in position'. Because the filum appeared to approximate the posterior spinal structures within the spinal cord, the radiologist was asked to re-examine the imaging to check for a tethered cord but again reported that there was no evidence of tethered cord. Laboratory tests were ordered and the results are shown in Boxes 19.1 and 19.2.

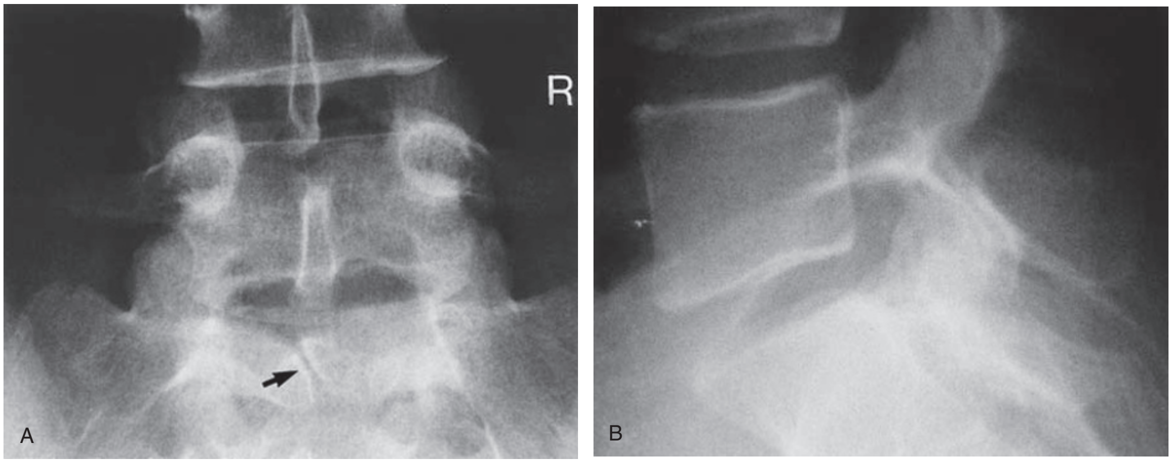

Figure 19.2 (A) Lumbosacral anteroposterior plain X-ray image. Note the spina bifida occulta at the S1 level (black arrow). R = right side of patient. (B) This lateral lumbosacral plain X-ray image did not show any abnormality.

Box 19.1 Chemical pathology

Status	Fasting	Units	Reference range
Sodium	139	mmol/l	(135–145)
Potassium	**4.6**	mmol/l	(3.2–4.5)
Chloride	106	mmol/l	(100–110)
Bicarbonate	28	mmol/l	(22–33)
Anion gap	5	mmol/l	(4–13)
Osmolality (calculated)	277	mmol/kg	(270–290)
Glucose	5.5	mmol/l	(4–13)
Urea	3.7	mmol/l	(3.0–8.0)
Creatinine	0.06	mmol/l	(0.05–0.10)
Urea/creatinine	62		(40–100)
Protein (total)	75	g/l	(62–83)
Albumin	46	g/l	(33–47)
Globulin	29	g/l	(25–45)
Bilirubin (total)	8	µmol/l	(<20)
Alkaline phosphatase	63	U/l	(40–250)
Gamma-glutamyl transferase	13	U/l	(<50)
Alanine transaminase	**46**	U/l	(<40)
Aspartate transaminase	27	U/l	(<35)

Box 19.2 Haematology

		Units	Reference range
Haemoglobin	135	g/l	(120–160)
White cell count	6.1	$\times 10^9$/l	(4.0–11.0)
Platelets	336	$\times 10^9$/l	(140–400)
Haematocrit	0.39		(0.39–0.46)
Red cell count	4.52	$\times 10^{12}$/l	(4.10–5.10)
MCV (mean corpuscular volume)	87	fl	(78–100)
Neutrophils	3.13	$\times 10^9$/l	(2.0–8.0)
Lymphocytes	2.28	$\times 10^9$/l	(2.0–8.0)
Monocytes	0.53	$\times 10^9$/l	(0.10–1.00)
Eosinophils	0.15	$\times 10^9$/l	(<0.60)
Basophils	0.01	$\times 10^9$/l	(<0.20)
ESR (erythrocyte sedimentation rate)	**12**	mm/hr	(<10)
Rheum. factor (neph)	<20	IU/ml	(<20)

As the results were essentially normal, apart from a very slight increased ESR and alanine transaminase, an opinion was sought from a specialist physician who was asked to consider this case in view of the spinal symptoms, the striae across the lumbar region and the skin blemish on the right side of the thoracolumbar region. The specialist physician performed the following laboratory tests and repeated several of the previous tests which were found to be normal (Boxes 19.3 and 19.4).

In addition, a bone scan was performed to look for any osseous lesion such as an osteoid osteoma. However, the bone scan was reported as normal with only mild increased

Box 19.3 Serum-specific protein chemistry

		Units	Reference range
Immunoglobulin G (total IgG)	9.87	g/l	(5.76–15.36)
Immunoglobulin A (total IgA)	**0.80**	g/l	(1.24–4.16)
Immunoglobulin M (total IgM)	1.34	g/l	(0.48–3.10)

Box 19.4 Serum autoantibodies

		Units	Reference range
Anti-nuclear antibody titre	Negative <1:40		
Anti-DNA (Farr assay)	<3	IU/ml	(<5)
Anti-ENA (ELISA)	Negative		
HIA B27	Peripheral blood negative		

ELISA, enzyme-linked immunosorbent assay

uptake in the anterior ends of the ribs, the femoral growth plates and the sacroiliac joints, with these changes being related to growth plate activity in view of the patient's age. The specialist physician suggested that the patient very likely had a spondyloarthritis as there was approximately 4 cm restriction in the lumbar spine range of flexion, tenderness over the sacroiliac joints and in the region of the left hip joint, because her problem had developed over quite a time and appeared to be getting worse, and because her father suffers from psoriasis with a lot of aches and pains. The skin lesion on the right side of the spine was considered to be a plaque of morphea (a localized form of scleroderma); the striae were considered to be related to morphea having left characteristic scars. She was referred to a dermatologist regarding the skin changes and he concurred with the diagnosis of morphea.

DIAGNOSIS

Adolescent tethered cord syndrome. A differential diagnosis of a possible mechanical thoracolumbar spinal pain syndrome was considered but thought to be unlikely.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Having explained to the patient and her mother that adolescent tethered cord syndrome was the most likely diagnosis, with a slight possibility of a mechanical thoracolumbar spinal pain syndrome, it was decided to try three manipulations of the lumbar spine extending from the thoracolumbar junction to the sacrum. The patient and her mother agreed with this approach. Therefore, three lumbar spine manipulations were performed to determine whether there was a mechanical component to her thoracolumbar pain but this was of no help. Naprosyn (750 mg) at night was prescribed to see if she would get pain relief through the night, with 500 mg Naprosyn in the morning. This provided some slight temporary relief, suggesting that she may have a rheumatological component.

However, the relief was short lived so the Naprosyn dose was increased to 2000 mg daily. Approximately 3 weeks later, she was started on Salazopyrin (1000 mg morning and night) but her symptoms gradually worsened.

A new lumbar spine MRI in the supine and prone positions was performed at a different facility with a specific request for careful examination for a 'functional tethered cord'. However, the report stated: '*There are no features of tethered cord or other dysraphic disorder*'. The full report follows.

'The conus medullaris terminates normally at T12/L1 disc. The conus is normal in configuration and signal intensity with no focal lesion demonstrated. Cauda equina are normal. In the prone position the nerve roots are seen to separate freely with no evidence of arachnoiditis. There is no thickening of the filum terminale and there is no evidence of filum lipoma. There is no spinal stenosis. The intervertebral discs appear normal throughout. Marrow signal intensity of the vertebral elements is normal. No epidural or intradural mass lesion is identified. The paravertebral soft tissues appear normal'.

A T2-weighted sagittal MRI view of the lumbosacral spine is shown in Fig. 19.3.

The patient determinedly kept going to school but, on returning home, would immediately lie down on the couch; she began to miss days at school and became

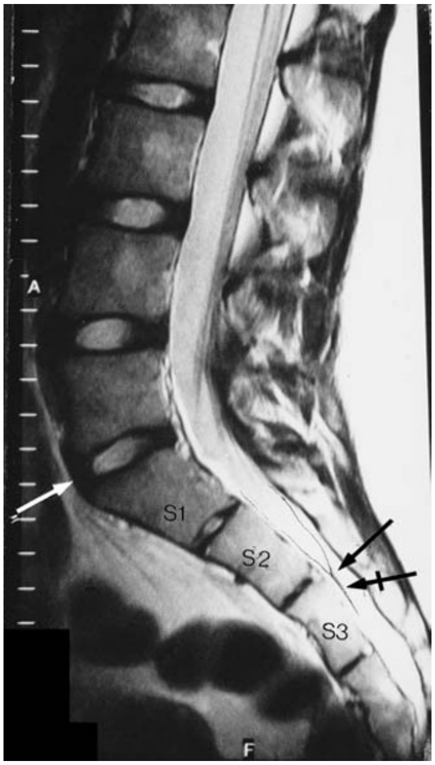

Figure 19.3 Lumbosacral MRI sagittal T2-weighted image showing the thecal sac extending to the lower S2/upper S3 level (black arrow) with a suggestion of thickening of the filum at this level (tailed arrow). The L5–S1 intervertebral disc level is indicated by the white arrow.

depressed about her increasing thoracolumbar spine and buttock/hip joint pains and spent a lot of time in bed, lying prone in an attempt to lessen the lumbar spine pain.

Because of the persisting clinical impression of tethered cord syndrome, the patient was advised to obtain second opinions from another specialist physician, a neurologist and a neurosurgeon. The specialist physician could not provide a diagnosis for her symptoms and, because of clashing appointments, the patient did not see the neurologist but saw the neurosurgeon. The neurosurgeon suggested a further lumbar MRI and concluded that she did not have a tethered cord syndrome and strongly recommended against any surgical intervention but did state that he was not experienced enough to comment on the presence of tethered cord in the absence of MRI abnormality.

The patient's MRI films were then sent to an authority on tethered cord who discussed the images with a radiologist and the conclusion was that, although there was no fat density found in the filum tip, the filum was displaced posteriorly to touch the posterior arachnoid membrane and the thecal sac extended to the lower S2 level (considered to be lower than usual) with a suggestion of thickening of the filum at this level. The patient was, therefore, advised to see another neurosurgeon, following a second bone scan that had been ordered by the specialist physician in view of the increasing symptoms, and which was, once again, reported as being within normal limits for her age. Meanwhile the patient stopped all medication to see if her symptoms would increase but they did not, so the specialist physician agreed that she did not have a rheumatological condition.

The second neurosurgeon decided that the symptoms could relate to a functional tethered cord syndrome and, because the patient was now beginning to get 'numbness in the legs and feet', he agreed to section the filum. An L5 laminectomy was performed to release the tethered spinal cord. At surgery, the filum was identified approaching the dorsal dura, and when the filum was divided, it spontaneously retracted cephalad.

The patient began cautiously to mobilize the next day, went home 4 days later and the staples were removed 6 days following surgery. She rapidly became asymptomatic and has made a complete physical and psychological recovery. Her mother says it is a pleasure to have her 'old' happy daughter back again.

Note

1. The following signs should alert a clinician to the possibility of adolescent tethered cord syndrome. (i) No paravertebral muscle spasm, (ii) No lumbosacral spine tenderness, (iii) Exaggerated lumbar lordosis, (iv) Scoliosis, (v) Deformity of foot or leg, (vi) Weakness of (a) extensor hallucis longus, (b) peroneus longus, (c) posterior tibialis, (d) anterior tibialis, or (e) gastrocnemius, (vii) Flabby or atrophic muscles in the lower extremities, (viii) Ankle joint instability on toe or heel walking, (ix) Pinprick sensation diminished (a) on the dorsum of the foot, (b) perianal area and groin, and (c) patchy sensation in the lower limbs, (x) Diminished or lost anal wink reflex, (xi) Diminished sphincter tone reflex on voluntary contraction during digital insertion, (xii) Hypoactive deep tendon reflexes, (xiii) Normal straight leg raising test, (xiv) Post-void residual urine.
2. For a detailed understanding of tethered cord syndrome with its various presentations see the textbook edited by Yamada (1996).
3. For a diagnostic work-up, see Yamada et al (2004).

KEY POINT

In cases of unexplained lumbar, buttock and leg symptoms, clinical awareness of adolescent and adult tethered cord syndrome due to spinal cord dysfunction is important.

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Case 20

Adult tethered cord syndrome

COMMENT

Patients with unexplained urinary incontinence should be fully investigated, including an MRI study of the lumbosacral spine when indicated.

PROFILE

A 49-year-old female administrator who does not smoke and only drinks alcohol socially.

PAST HISTORY

Urinary difficulties for many years which have gradually become worse over the last 2 years. She has undergone surgery for a bladder neck suspension procedure but without a change in incontinence.

PRESENTING COMPLAINT(S) (Fig. 20.1)

A 13-year history of low back pain that began while she was pregnant. The pain radiates bilaterally to the buttocks with leg pain extending particularly to the right thigh and right calf medially, with some aching in the right calf laterally, on prolonged standing. There is minor numbness intermittently in both feet with prolonged sitting. There is no groin pain but occasional genitorectal pain, especially premenstrually. She reported urinary urgency and occasionally was incontinent of urine, with very occasional fecal incontinence.

She has tried various abdominal muscle strengthening exercises, traction, gym work-outs, walking programmes, massage, acupuncture and non-steroidal anti-inflammatory medication. She currently uses paracetamol and occasional Panadeine Forte.

AETIOLOGY

Pregnancy was thought to have been the precipitating factor.

EXAMINATION

She has a normal gait, an increased lumbosacral lordosis with a dimple centrally over the lumbosacral region and dropped arches with pronation of both feet. The left knee jerk was depressed (one plus instead of two plus) and sensation was

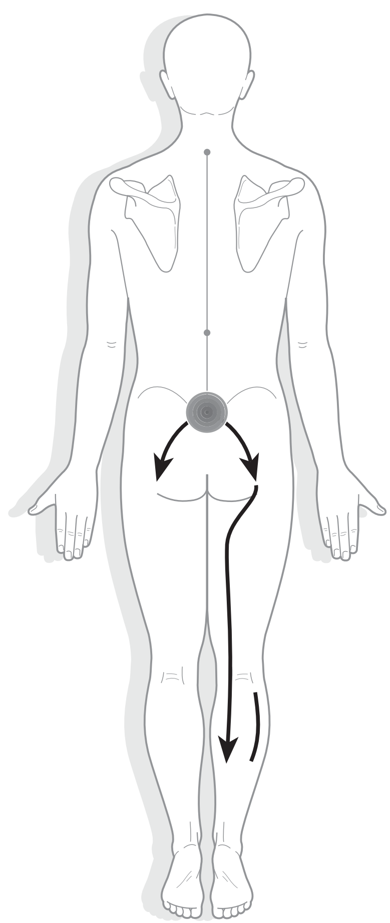

Figure 20.1

slightly impaired to light touch and pinprick on the dorsum of both feet in the L5–S1 dermatomes.

She could sit in the Buddha position (cross-legged) but felt leg discomfort. Bending slightly at the waist increased the low back 'discomfort'. When carrying a light weight (2–3 kg) she had to hold it close to her abdomen, otherwise she experienced low back discomfort.

Urological examination, in spite of bladder neck suspension surgery, found the urodynamics basically showed evidence of detrusor instability and genuine stress incontinence with low urethral pressures; bladder compliance was quite good with a capacity of about 400 ml. An ultrasound scan showed a post-void urinary residual of 105 ml.

IMAGING REVIEW

Plain X-ray films showed a spina bifida occulta of L5 and lumbarization of the right transverse process of the transitional presacral segment; bilateral pars interarticularis defects at L5 were associated with a Grade 1 spondylolisthesis of L5 on the transitional segment below. Lumbar spine stress views showed no movement of the spondylolisthesis.

CLINICAL IMPRESSION

Possible adult tethered cord syndrome.

WHAT ACTION SHOULD BE TAKEN?

In view of the unexplained incontinence and spinal anomalies, a clinical suspicion of tethered cord syndrome led to a lumbar MRI study, even though neurological findings were normal apart from only subtle abnormalities noted above.

The lumbar MRI showed a fatty mass (lipoma) in the posterior part of the spinal canal from L3 to the lumbarization level, with the conus unusually low at L4 level (Fig. 20.2A and B).

DIAGNOSIS

Adult tethered cord syndrome associated with a lipoma in the lower spinal canal.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Untethering of the spinal cord in order to prevent further neurological deterioration in the future was considered. However, an attempt to remove the lipoma was considered to be too risky in this particular case.

A conservative approach was taken: (1) a continued exercise programme to strengthen the lumbar, buttock and abdominal areas; (2) hormone replacement therapy by her general medical practitioner to help with the incontinence; (3) the patient was advised not to abuse her lumbosacral spine; she was told not to participate in any activities that could injure her low back and the tethered

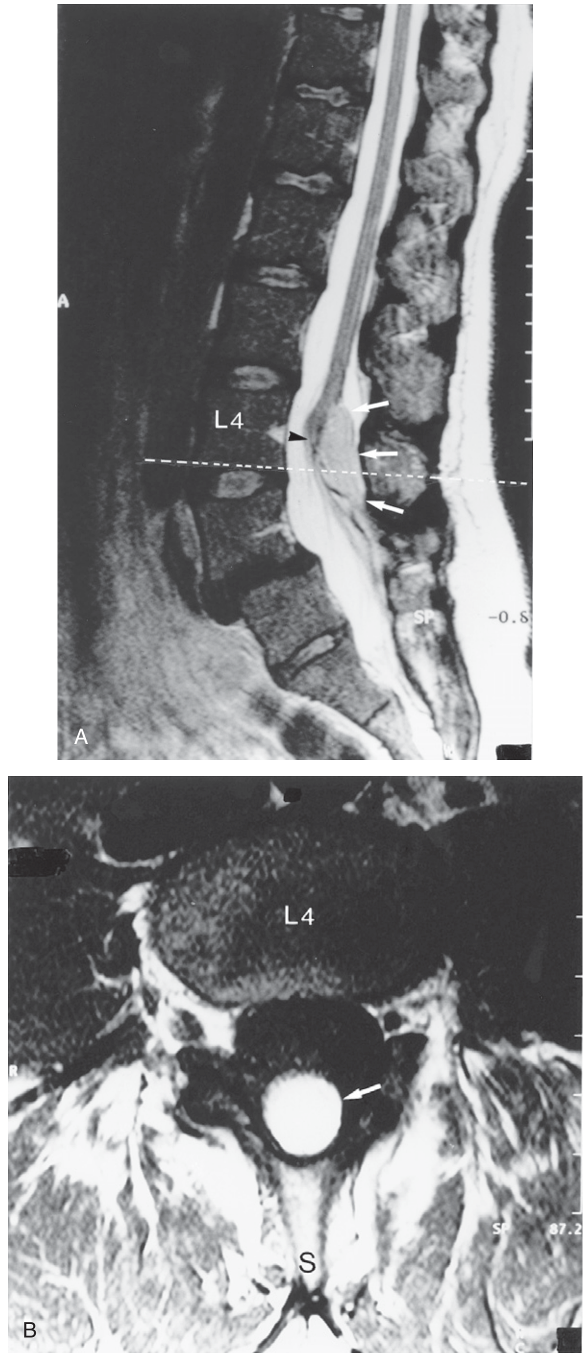

Figure 20.2 (A) MRI sagittal T2-weighted image showing the lipoma (white arrows) in the posterior part of the canal and the conus medullaris at the L4 level (arrowhead). The broken line shows the level at which the axial view was taken for (B). (B) MRI axial T2-weighted image through the area shown by the white broken line in (A). The arrow shows the lipoma. S = spinous process; L4 = 4th vertebral body endplate; R = right side of patient.

cord, as injury may well lead to a surgical emergency which would require an attempt to untether the cord.

The above conservative approach led to a considerable improvement in the symptoms, so surgery was not performed.

Note

Clinical awareness of the association between tethered cord syndrome and bladder dysfunction is essential. Bladder dysfunction is associated with tethered cord syndrome in 40–72% of adult cases (Yamada & Lonser 2000) and is the exclusive complaint in 4% (French 1990) and may represent the earliest sign of tethered cord syndrome (Hadley & Holevas 1996).

KEY POINT

Bladder dysfunction is associated with tethered cord syndrome in 40–72% of adult cases and may represent the earliest sign of tethered cord syndrome.

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Also see Case 19.

Case 21

Lumbar neuroma

COMMENT

Beware of chronic recurring symptoms that mimic spinal mechanical dysfunction but may actually have a pathological basis.

PROFILE

A 57-year-old male who performed manual work.

PAST HISTORY

He fell from a height of approximately 8 m at the age of 26 years and this caused low back pain with left sacroiliac joint pain. He recovered with anti-inflammatory medication then aggravated his low back pain 12 years later while lifting heavy objects; again he recovered after using non-steroidal anti-inflammatory drugs.

PRESENTING COMPLAINT(S) (Fig. 21.1)

Severe pain in the left buttock with some low back pain that radiated to the lateral aspect of the left lower limb and medial aspect of the left foot since twisting his low back 7 days ago. On this occasion anti-inflammatory and analgesic medication did not help his pain so he was referred by his general medical practitioner for a second opinion.

AETIOLOGY

One week ago he twisted his low back and caused the above presenting symptoms.

EXAMINATION

On examination there was loss of pinprick sensation on the lateral aspect of the left calf (L5) and the medial aspect of the left foot (S1). Supine SLR was limited by low back pain at 45° elevation (left) and by hamstring tightness at 90° (right). The left knee jerk (L4) was absent. All lumbar spine movements were painful. Coughing and bearing down did not aggravate his symptoms. Hip joint movements were of normal range and painless.

IMAGING REVIEW

Recent lumbar spine and pelvis radiographs were reported as showing only ‘minor degenerative changes’ (Fig. 21.2A and B).

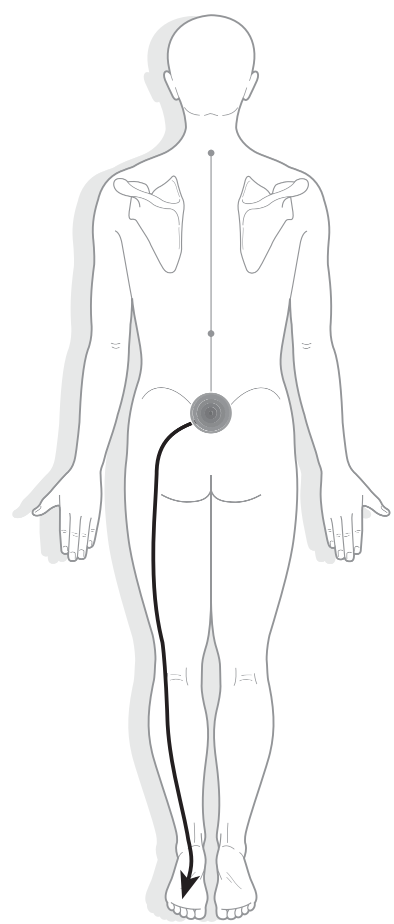

Figure 21.1

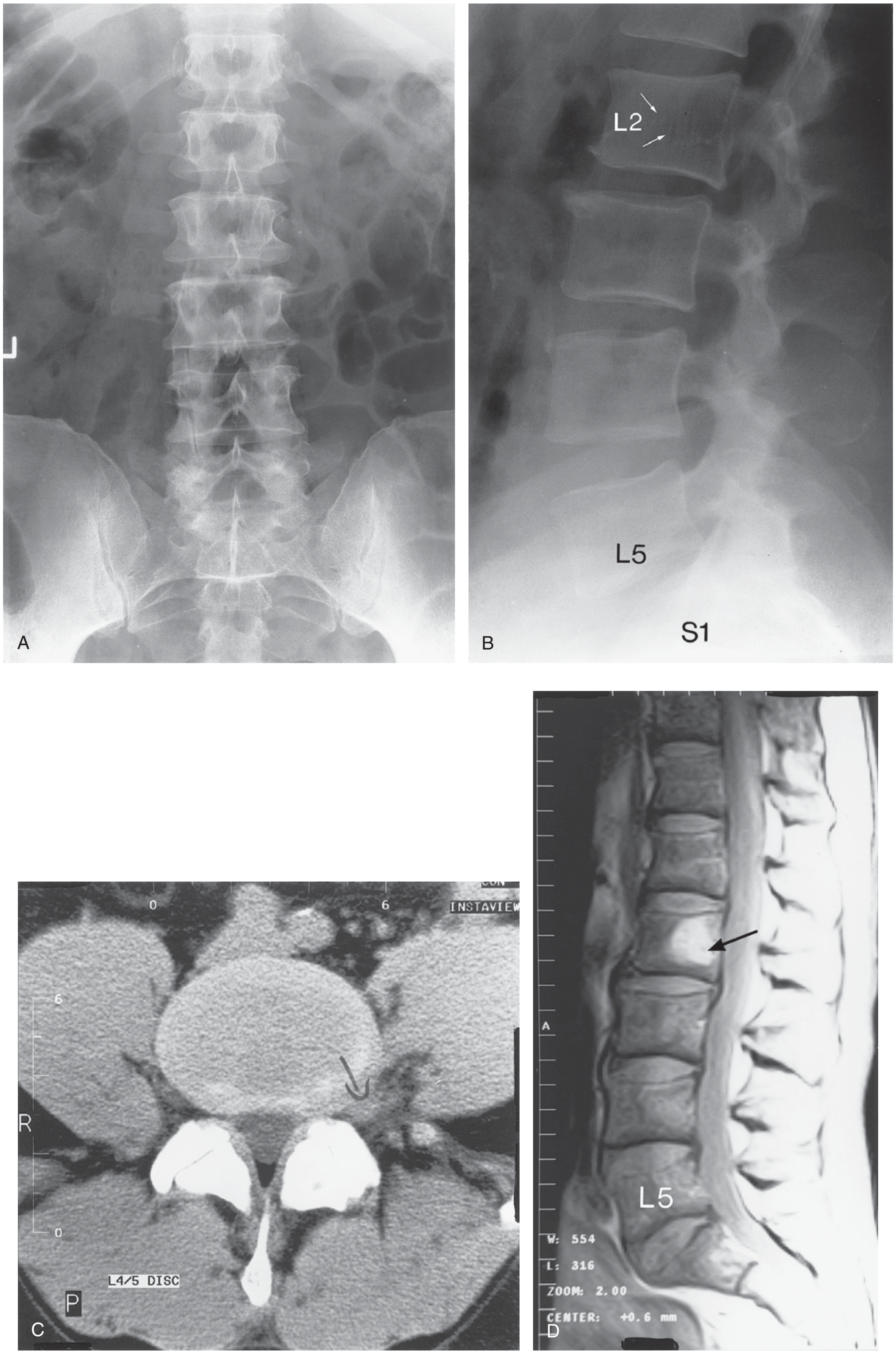

Figure 21.2 (A) Lumbosacral spine anteroposterior plane X-ray image showing minor lipping of most vertebral bodies and tropism of some zygapophysial joints. L = left side of patient. (B) Lumbar spine lateral plane X-ray image showing some thinning of the L2–3 intervertebral disc with lipping anteriorly on the adjacent bony margins and some retrolisthesis of L2 on L3 vertebra. Note the vertical 'corduroy' trabecular pattern in the L2 body (arrows) indicating a haemangioma. L5 = fifth vertebral body; S1 = first sacral segment. (C) Lumbar spine CT axial image at the L4–5 disc level showing a possible left far lateral L4–5 disc protrusion or a tumour of neural origin (arrow). P = posterior side of patient; R = right side of patient. (D) Lumbosacral spine MRI sagittal T1-weighted image showing the haemangioma (arrow) involving the L2 vertebral body with some posterior bulging of the L2–3 intervertebral disc.

(Continued)

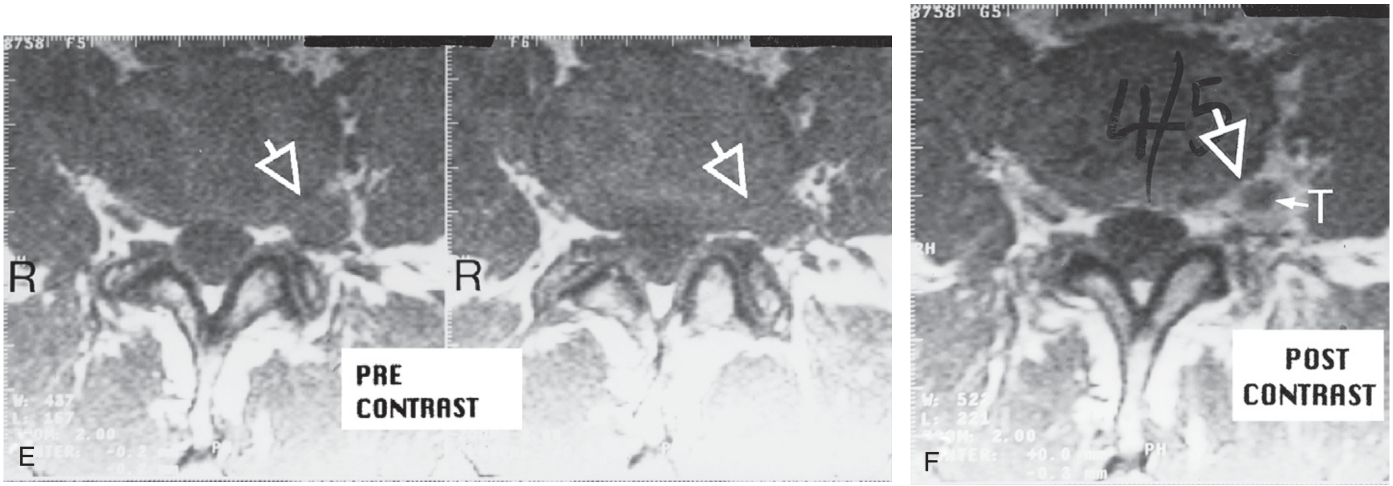

Figure 21.2 Cont'd (E) Axial T1-weighted pre-contrast MRI scan at the L4–5 disc area. The arrow shows apparent continuity between disc material and the far lateral 'lesion'. (F) Lumbar spine MRI axial T1-weighted post-contrast scan at the L4–5 disc area. The arrow clearly shows there is no continuity between the tumour (T) and the disc material.

CLINICAL IMPRESSION

A possible left sided L4–5 or L5–S1 intervertebral disc prolapse.

WHAT ACTION SHOULD BE TAKEN?

As the plain radiographs did not correlate with the degree of low back and leg symptoms chemical pathology tests and haematology tests were performed as a precaution (Boxes 21.1 and 21.2) as well as a lumbar (L3–S1 levels) CT scan.

The chemical pathology and haematology results were considered to be essentially normal and not related to his low back pain.

The CT report concluded: 'Possible left far lateral L4–5 disc protrusion. Differential diagnosis is a tumour of neural origin. CT myelography or MRI suggested for further evaluation. Disc degeneration L3–4, L4–5 and L5–S1 levels. L4–5 level mild canal stenosis' (Fig. 21.2C).

In order to differentiate between a possible left far lateral L4–5 disc protrusion and a tumour of neural origin, an MRI with and without contrast was performed; the sagittal T1-weighted image (Fig. 21.2D) was non-contributory but showed a lesion of increased signal intensity involving the L2 body posteriorly (arrow) which reduces in signal intensity on T2 images, the appearance favouring a haemangioma or an area of fatty deposition. The axial views through the L4–5 disc area showed the lesion under investigation but the pre-contrast study (Fig. 21.2E) was not able to differentiate between a far lateral disc lesion and a neural tumour. However, the post-contrast view (Fig. 21.2F) demonstrated that there is no continuity between the lesion and the disc, therefore indicating a neural tumour, i.e. a neurofibroma or a Schwannoma.

Box 21.1 Chemical pathology

		Units	Reference range
Sodium	138	mmol/l	(133–146)
Potassium	4.3	mmol/l	(3.3–4.5)
Chloride	106	mmol/l	(97–110)
Bicarbonate	28	mmol/l	(20–32)
Anion gap	**4**	mmol/l	(8–17)
Urea	3.9	mmol/l	(3.0–6.0)
Creatinine	0.11	mmol/l	(0.04–0.12)
AST (aspartate aminotransferase)	20	U/l	(10–40)
Protein (total)	67	g/l	(60–80)
Albumin	45	g/l	(35–50)
Globulin	22	g/l	
ALP (alkaline phosphatase)	52	U/l	(40–120)
Gamma-glutamyltransferase Serum	22	U/l	(<60)
ALT (alanine aminotransferase)	11	U/l	(5–60)
Bilirubin (total)	15	μmol/l	(<17)
Calcium	2.23	mmol/l	(2.10–2.60)
Calcium (corrected)	2.13	mmol/l	(2.10–2.60)
Phosphate	**0.77**	mmol/l	(0.80–1.40)

A histopathology section cut in the horizontal plane is shown in Fig. 21.3. This indicates how there should be fatty tissue surrounding neural structures which should not normally touch disc material as the neural structures pass through the intervertebral foramen.

Box 21.2 Haematology

		Units	Reference range
Haemoglobin	151	g/l	(135–180)
White cell count	10.8	$\times 10^9$/l	(4.0–11.0)
Platelets	341	$\times 10^9$/l	(140–400)
Haematocrit	0.44		(0.39–0.52)
Red cell count	5.11	$\times 10^{12}$/l	(4.50–6.00)
MCV (mean corpuscular volume)	86.1	fl	(80.0–98.0)
Neutrophils	**8.2**	$\times 10^9$/l	(2.0–8.0)
Lymphocytes	1.6	$\times 10^9$/l	(1.0–4.0)
Monocytes	**0.9**	$\times 10^9$/l	(0.1–0.8)
Eosinophils	0.1	$\times 10^9$/l	($<$ 0.2)
Basophils	0.0	$\times 10^9$/l	($<$ 0.2)
ESR	10	mm/hour	($<$ 15)

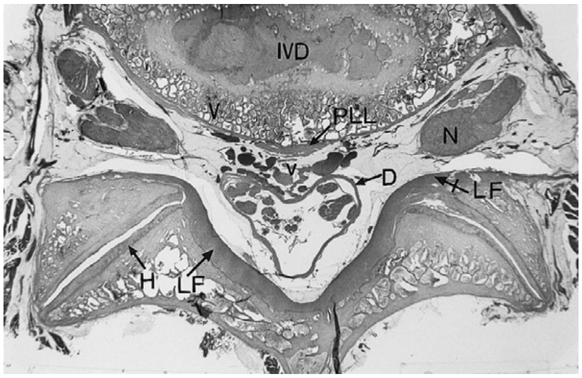

Figure 21.3 A 200-micron thick horizontal histopathological section through the lower lumbar spine of a 46-year-old male that serves to illustrate the principle involved in the above case. Note the intervertebral disc material (IVD), the posterior of the vertebral body (V) and the nerve root ganglion (N) within the intervertebral foramen. The space between the neural structures (N) and the posterior part of the vertebral body and disc and, for that matter, the anteromedial portion of the ligamentum flavum (LF tailed arrow) can be seen. Therefore, with contrast medium in the nerve root sleeve (sheath) it should be possible to see a space between the neural structures and the adjacent structures, due to contrast material being introduced, if the structures under investigation are adjacent to each other and not apart from each other. The dural tube (D) is shown containing some of the cauda equina nerve roots. Small blood vessels (v) are seen between the posterior longitudinal ligament (PLL) and the dural tube. The hyaline articular cartilage (H) on the articular facet surfaces can be seen. (Ehrlich's haematoxylin and light green counterstain).

DIAGNOSIS

Left L4-5 spinal nerve neurofibroma or a Schwannoma.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was told that surgery would most likely not help because of the benign neural tumour. A non-steroidal anti-inflammatory drug was prescribed. He was asked to return should his condition not improve. The administration of non-steroidal anti-inflammatory drugs gave him adequate symptomatic relief.

KEY POINT

Chronic recurring symptoms of low back pain and sciatica may be related to pathology other than nerve root pressure.

Further reading

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Case 22

Sacral Tarlov cyst

COMMENT

Listen carefully to patients and ask yourself whether extensive imaging that has previously been performed is appropriate for identifying the patients' level of spinal involvement.

PROFILE

A 37-year-old slim female housekeeper who occasionally smokes a cigarette and occasionally drinks alcohol.

PAST HISTORY

Six years ago she experienced a minor low back strain at work but returned to work within a few days and her low back pain resolved without treatment.

Approximately 4 weeks ago she was lifting wet clothes out of a washing machine and felt some 'discomfort' at the lumbosacral level of her spine. She continued working, although the discomfort gradually increased. Next morning she awoke with lumbosacral level pain that did not prevent her from returning to work.

PRESENTING COMPLAINT(S) (Fig. 22.1)

Low-grade lumbosacral pain since lifting approximately 4 weeks ago that prevents her from sitting or standing for long periods of time. The low back pain radiates down her left lower limb posterolaterally and she has noticed that she has some 'numbness' in the heels bilaterally. Her symptoms are aggravated by coughing, sneezing and bearing down. She has tried non-steroidal anti-inflammatory medication and physiotherapy treatment (traction and massage) but neither form of treatment has been helpful. She experienced urinary bladder incontinence on two or three occasions some months ago and one episode of bowel incontinence.

AETIOLOGY

Lifting wet clothes out of a washing machine 4 weeks ago.

EXAMINATION

In the erect posture, there was no clinical evidence of leg length inequality or of pelvic obliquity or scoliosis. There was some spasm of the lumbosacral level muscles and active ranges of movement were considerably restricted in all planes. Straight leg raising was limited to a measured 15° bilaterally due to lumbosacral pain aggravation. The left achilles reflex (S1) was slightly diminished at one plus. The deep tendon reflex at the knees was normal bilaterally,

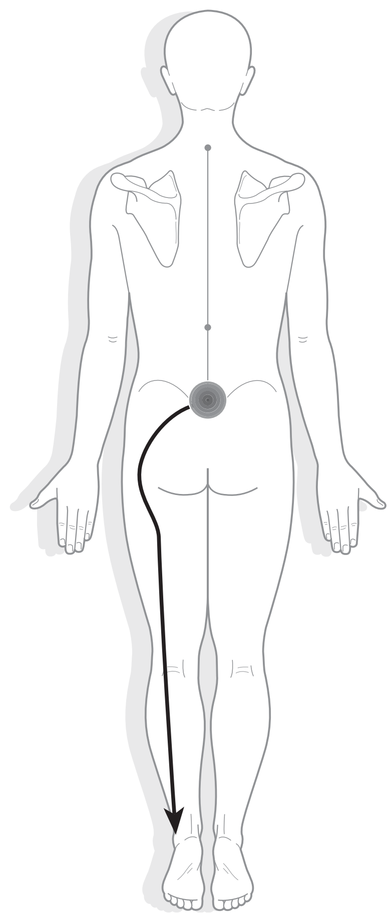

Figure 22.1

as was muscle tone and strength. Pinprick sensation indicated hypoaesthesia over both heels (S1).

IMAGING REVIEW

A lumbar spine plain X-ray film report stated: 'The disc spaces are normal. There is minimal spur formation on the anterior body margins of L3 and L4. The intervertebral foramina appear normal. There is a spina bifida occulta at L5–S1'.

A CT lumbar spine (L3 to S1 levels) stated: 'L3–4 level – normal findings. L4–5 level – a mild diffuse annular disc bulge is noted with mild facet joint hypertrophic changes but without narrowing of the intervertebral foramen and no neural compression. L5–S1 level – no disc protrusion, no narrowing of the intervertebral foramen and no neural compression'.

A CT abdomen (full length) report stated: 'Enhanced 10 mm thick slices were performed through the pelvis following oral and IV contrast'. 'Comment: Normal study'.

A Bone scan + SPECT report stated: 'Blood flow and blood pool images of the lumbar spine and pelvis were normal. Delayed static planar images were acquired together with SPECT of the lumbar spine and pelvis. No significant focus of abnormal uptake was seen. Bone scan of the lumbar spine and pelvis is within normal limits'.

CLINICAL IMPRESSION

Possible unidentified space occupying lesion within the sacrum as coughing, sneezing and bearing down aggravate her lumbosacral level pain and in view of her history of bowel and urinary bladder symptoms.

WHAT ACTION SHOULD BE TAKEN?

As there was no explanation for the patient's symptoms and signs, further investigations were performed including chemical pathology and haematology tests (Boxes 22.1 and 22.2).

Box 22.1 Chemical pathology

Specimen type	Blood	Units	Reference range
Sodium	141	mmol/l	(135–145)
Potassium	3.7	mmol/l	(3.2–4.5)
Chloride	107	mmol/l	(100–110)
Bicarbonate	26	mmol/l	(22–33)
Anion gap	8	mmol/l	(4–13)
Urea	3.4	mmol/l	(3.0–8.0)
Creatinine	0.05	mmol/l	(0.05–0.10)
Urea/creat	63		(40–100)
Albumin	44	g/L	(33–47)
Calcium	2.36	mmol/l	(2.15–2.60)
Calcium (corrected for albumin)	2.28	mmol/l	(2.15–2.60)
Phosphate	1.08	mmol/l	(0.70–1.40)
C-reactive protein	<5.0	mg/l	(<5.0)

Box 22.2 Haematology

Specimen type	Blood	Units	Reference range
Haemaglobin	130	g/l	(115–160)
White cell count	6.1	$\times 10^9$/l	(4.0–11.0)
Platelets	317	$\times 10^9$/l	(140–400)
Haematocrit	0.38	$\times$	(0.33–0.47)
Red cell count	4.34	10^{12}/l	(3.80–5.20)
MCV (mean corpuscular volume)	86	fL	(80–98)
Neutrophils	3.26	$\times 10^9$/l	(2.00–8.00)
Lymphocytes	2.35	$\times 10^9$/l	(1.00–4.00)
Monocytes	0.35	$\times 10^9$/l	(0.10–1.00)
Eosinophils	0.12	$\times 10^9$/l	(<0.60)
Basophils	0.02	$\times 10^9$/l	(<0.20)
ESR	**16**	mm/hour	(<12)

A lower lumbar and sacral MRI was requested with gadolinium to investigate the lower lumbar and sacrococcygeal spines. The MRI report stated: 'The appearances suggest a prominent Tarlov cyst at the S1-2 level'. The sagittal image (Fig. 22.2) shows the 'Tarlov cyst'. The axial view (Fig. 22.3) shows a 'dumbbell type cystic lesion with no enhancement or CSF signal intensity extending into the left half of the spinal canal and through the left S1–2 neural foramen'.

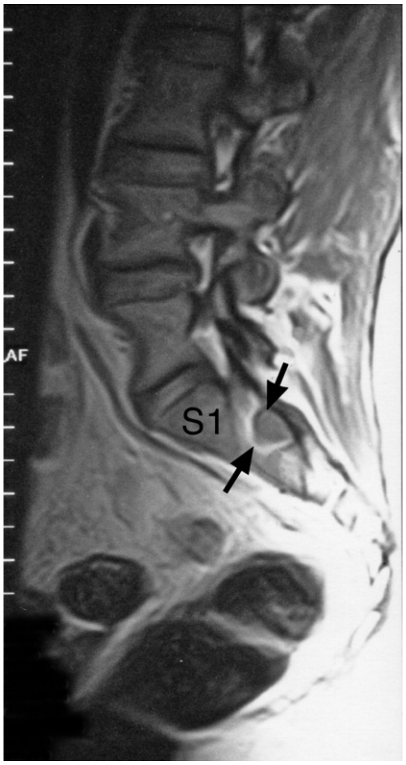

Figure 22.2 Lumbosacral MRI parasagittal image showing a Tarlov cyst (black arrows) at the S1-2 level.

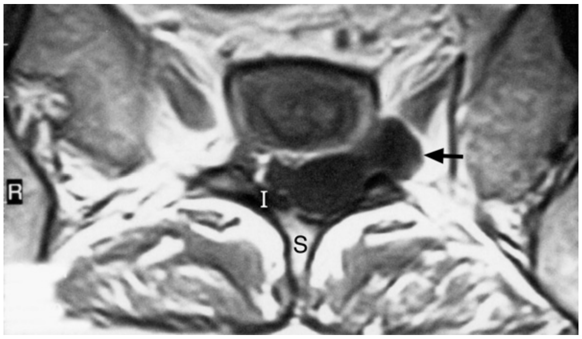

Figure 22.3 S1–2 level MRI axial image showing a dumbbell type cystic lesion with no enhancement or CSF signal intensity extending into the left half of the spinal canal and through the left S1-2 neural foramen (arrow). I = intermediate crest of the sacrum; S = spinous tubercle of the median crest.

As the axial view MRI (Fig. 22.3) suggested almost complete erosion of the posterior cortex of the intermediate crest of the sacrum on the left side, a CT scan was requested from L5 to the lower end of the S2 segment. The report stated for L5–S1 level: 'The L5 and S1 nerve roots appear normal at the disc level with minor degenerative change involving the facet joints'. For S1-2 level (Fig. 22.4): 'There is enlargement of the S1 neural foramen that corresponds to the MRI finding of dumbbell type cystic lesion involving the left half of the spinal canal and extending through the left S1 neural foramen. The appearances are compatible with a perineural/Tarlov cyst'.

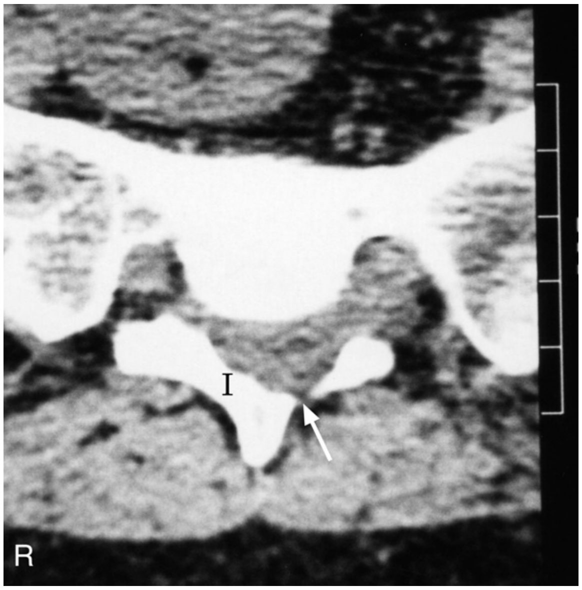

Figure 22.4 S1–2 level CT axial image. Note the dumbbell shaped cystic lesion within the left side of the sacral canal that extends through the left S1-2 neural foramen. producing some scalloping of the sacral foramen. The cystic lesion is expansile and has eroded the intermediate crest (I) of the sacrum on the left side (arrow) suggesting a possibly longstanding perineural, Tarlov or spinal arachnoid cyst.

DIAGNOSIS

S1–2 level Tarlov cyst.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was referred to a neurosurgeon for an opinion. The neurosurgeon said the expansile lesion was of no consequence. She was referred for a second neurosurgical opinion and was told by that neurosurgeon that the lesion was an 'incidental finding' and ordered an EMG that was reported as having provided 'normal findings'. She was referred to a third neurosurgeon who stated that the lesion was not causing her symptoms.

Unfortunately, this lady was not able to find a solution for her ongoing low back and left lower limb symptoms. She continued to experience bilateral numbness in her heels and increasing weakness in both legs. She experienced two episodes of urinary incontinence during the few weeks while she consulted the neurosurgeons.

Note

While no consensus exists regarding definitive treatment of symptomatic Tarlov cysts, it is clear from the literature that such Tarlov cysts are by no means always an incidental finding (Birch et al 2003). They may, indeed, contribute to, or be responsible for, the patient's symptoms. Birch et al's (2003) classification system of Types I to III addresses this issue and offers guidance on patient management. Type I cysts are small and are unrelated to a patient's symptoms but Type III cysts that are large can cause both erosion of bone and compression of the lower sacral nerve roots. Furthermore, Tarlov cysts, when present in the sacral neural canal and foramina have been found to cause a variety of symptoms including radicular pain, paraesthesias, and urinary or bowel dysfunction (Acosta et al 2003).

KEY POINTS

When a patient appears to have had a thorough imaging evaluation and no significant pathology is found, consider the possibility that there is a pathology that simply has not been found. I have seen a number of cases where patients complaining of lumbosacral pain have not been investigated with imaging *below* that level and yet imaging of the sacrum has shown space-occupying lesions.

References

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Case 23

Zygapophysial joint synovial cyst

COMMENT

More than one cause of low back pain with referred leg pain may be present requiring appropriate surgical intervention.

PROFILE

A 68-year-old retired builder who stopped smoking some 40 years ago and only drinks alcohol moderately. He has generalized osteoarthritic pains for which he takes an NSAID.

PAST HISTORY

He had prostatism diagnosed some years ago. Recently he was found to have hypertension (200/90).

PRESENTING COMPLAINT(S) (Fig. 23.1)

Mild low back pain, of unknown aetiology, for approximately 5 years with periodic left or right lower limb radicular pain of 3 months duration. Two years ago he developed ‘shooting’ pains in the lateral aspect of the right leg but this completely resolved until 2 months ago when there was a recurrence of some pain in the right lower leg, particularly on getting up in the morning or on sitting for long periods of time.

Coughing and sneezing did not aggravate his low back pain and he said he could walk for long distances without any paraesthesiae or weakness; he did not have any pain in the feet. Bowel and bladder function were normal.

AETIOLOGY

He could not recall any specific injury.

EXAMINATION

He had a good range of active lumbar spine movements. Supine SLR was limited to 80° on the right due to an

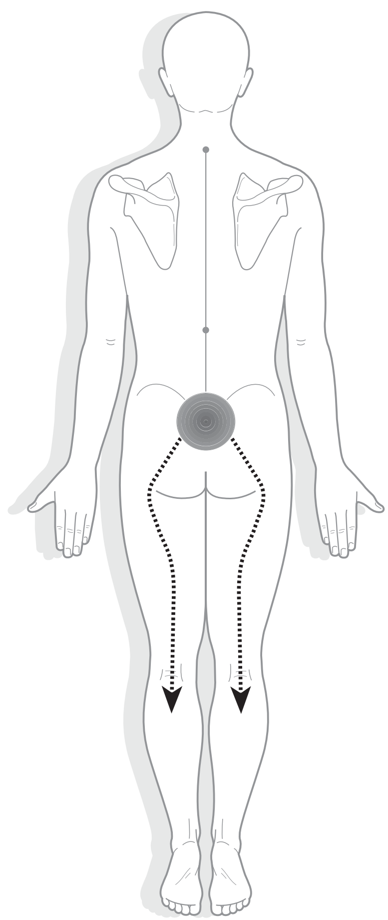

Figure 23.1

increase in low back pain. There were no sensory or reflex changes and power was normal.

IMAGING REVIEW

Plain X-ray films showed a grade 1 spondylolisthesis of L4 on L5 due to osteoarthrotic degenerative changes of the L4–5 zygapophysial joint facets, i.e. degenerative spondylolisthesis. Disc thinning was present at both the L4–5 and L5–S1 levels with associated osteoarthrotic involvement of the zygapophysial facet joints. There were no pars interarticularis defects.

CLINICAL IMPRESSION

Low back pain and alternating left and right leg pains probably due to the degenerative grade 1 spondylolisthesis of L4 on L5.

WHAT ACTION SHOULD BE TAKEN?

In view of the patient's history and age, chemical pathology and haematology tests were performed (Boxes 23.1 and 23.2). Although there were minor variations from normal, these were considered to be unrelated to his presenting symptoms and signs and he was referred to his general medical practitioner for further consideration of the results.

As a precaution in view of the alternating left and right leg symptoms associated with his low back pain, a CT lumbar spine (L3–S1 levels) was performed; this confirmed the grade 1 spondylolisthesis at L4–5 and extensive degenerative changes in the 'facet' joints at this level which are responsible for the 'degenerative spondylolisthesis'. There was no evidence of pars interarticularis defects. The spinal canal was reasonably spacious but was encroached upon to some extent at the upper border of L5 by a combination of the spondylolisthesis and pronounced ligamentum flavum thickening. This thickening had a nodular appearance on the left at the L4–5 joint level where it indents the theca (Fig. 23.2A). The extensive degenerative change in the facet joints shows osteoarthrotic changes and remodelling of the bone on the left and right sides of the spine. In order to give the reader an appreciation of this type of osteoarthrotic degenerative zygapophysial joint change, a histopathology section is shown in Fig. 23.2B.

An MRI scan of the lumbar spine was performed and showed a round area of soft tissue projecting into the spinal canal from the left L4–5 facet joint – this had the appearance of a synovial cyst and caused some spinal canal stenosis due to impingement of the thecal sac (Fig. 23.2C).

Box 23.1 Chemical pathology

		Units	Reference range
Sodium	139	mmol/l	(135–148)
Potassium	4.5	mmol/l	(3.2–5.0)
Chloride	109	mmol/l	(95–109)
Carbon dioxide	27	mmol/l	(23–32)
Urea	7.3	mmol/l	(2.5–7.5)
Creatinine	0.09	mmol/l	(0.04–0.12)
Prostate-specific antigen	0.2	ng/ml	(0.0–4.0)

Box 23.2 Haematology

		Units	Reference range
Haemoglobin	136	g/l	(125–175)
White cell count	9.6	$\times 10^9$/l	(4.0–11.0)
Platelets	243	$\times 10^9$/l	(150–400)
Haematocrit	**0.39**		(0.40–0.54)
Red cell count	**4.12**	$\times 10^{12}$/l	(4.20–6.50)
RBC distance width	**11**		(12–14)
MCV (mean corpuscular volume)	95	fl	(75–95)
MCH (mean corpuscular haemoglobin)	**33.1**	pg	(27.0–32.0)
MCHC (mean corpuscular haemoglobin concentration)	349	g/l	(310–350)
Neutrophils	5.0	$\times 10^9$/l	(2.0–7.5)
Lymphocytes	3.5	$\times 10^9$/l	(1.0–4.0)
Monocytes	**0.8**	$\times 10^9$/l	(0.0–0.8)
Eosinophils	0.2	$\times 10^9$/l	(0.0–0.4)
Basophils	0.1	$\times 10^9$/l	(0.0–0.1)

DIAGNOSIS

- Synovial cyst of the left zygapophysial joint at L4–5 level compressing the dural tube and the left nerve root;
- Bilateral osteoarthrosis at L4–5 and L5–S1.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was referred for a neurosurgical opinion regarding (i) his leg symptoms and (ii) the abnormal tissue noted adjacent to and affecting the L4–5 facet joint and lamina on the left. It was decided by the surgeon that the right-sided nerve root entrapment at L4–5, due to a combination

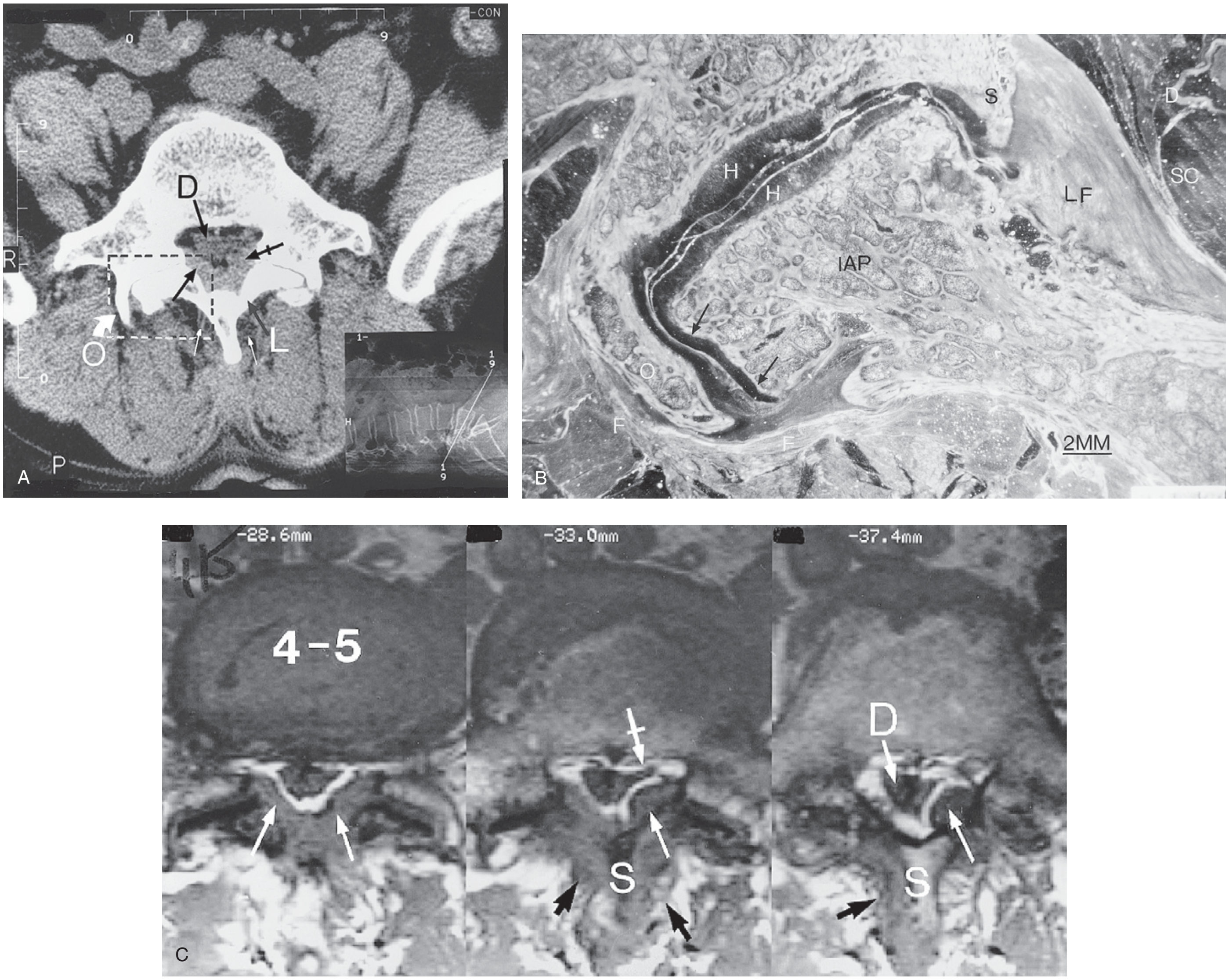

Figure 23.2 (A) CT axial image through the L4–5 zygapophysial joint 'facets'. Note the hypertrophy of the ligamentum flavum bilaterally (small arrow) and the nodular thickening of the ligamentum flavum on the left side opposite the L4–5 zygapophysial joint (tailed arrow). Note how the left side of the dural tube (D) is being indented by the nodular synovial cyst. L = lamina; O = osteophyte on the right superior articular process of the L5 vertebra. The rectangle shown between the broken lines represents an area shown histologically in (B) from postmortem material. Note in (A) the quite marked irregularity of the spinous process with thickening of the soft tissues adjacent to the laminae and the spinous process (small white arrows). P = posterior side of the patient's lumbar region. (B) A 200-micron thick horizontal histopathology darkfield section through the right zygapophysial facet joint of a 65-year-old postmortem specimen. Note the hyaline articular cartilage (H) on the facet surfaces and how it has developed around the lateral margins of the joint forming 'bumper-fibrocartilage' (black arrows) as a result of the development of the large osteophytic spur (O) beneath the fibrous joint capsule (F). D = part of the dural tube; IAP = inferior articular process; LF = ligamentum flavum; S = spur adjacent to the ligamentum flavum and projecting from the superior articular process of the vertebra below. SC = spinal canal. See also colour plate section Fig. vii.19. (C) MRI axial T1-weighted images through the L4–5 zygapophysial joints at approximately the same level as shown in the CT scan (A). Note the thickening of the ligamentum flavum bilaterally (white arrows) and the synovial cyst on the left side that compresses the dural tube (D), and to some extent the left nerve root at this level (small tailed arrow). Note the abnormal tissue (small black arrows), tracking along the right lamina and the spinous process (S) suggesting an inflammatory or degenerative process. In addition, there is a degree of trefoil stenosis of the central canal and the lateral recesses (left Figure in (C)).

of thickened ligamentum flavum and zygapophysial joint arthropathy, indicated that surgery should be undertaken to decompress the right L5 nerve root in the lateral recess via an L4–5 fenestration; this was done, as well as removing a moderate-sized disc protrusion at that level. However, the left-sided synovial cyst was not removed as the surgeon considered that the synovial cyst was a completely incidental finding and did not warrant exposing the left side of the spine during surgery; he felt it was 'completely unnecessary' to do this.

The surgery produced good relief from the patient's right-sided sciatica. However, the patient presented again with symptoms of spinal stenosis, so a laminectomy and decompression was performed including the lower portion of L3 to L5, the spinouses and laminae being removed due to the 'significant central canal stenosis, particularly at L4–5' where there was a 'large synovial cyst' as originally noted.

The patient made an uneventful postoperative recovery.

Note

This raises the interesting question of whether, when two lesions are present at the same level of the spine, i.e. L4–5 in this case, both lesions should be removed at the time of initial surgery to minimize the risk to a patient having to undergo follow-up surgery, while also lessening the cost of surgery, hospitalization, etc.

KEY POINT

When more than one spinal pathology is present it would appear to be wise to address both pathologies at the same time in order to maximally help the patient and to save the costs associated with re-admission to hospital and a second surgical procedure at the same level.

Further reading

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Case 24

L5–S1 posterior left paracentral intervertebral disc protrusion with a probable epidural haematoma

COMMENT

It is important for clinicians to be aware of the condition of epidural haematoma as a potential source of spinal pain syndromes.

PROFILE

42-year-old male manual worker of average build who does not smoke.

PAST HISTORY

Two and a half years before consultation he sustained a low back injury when he lifted a 60-kg weight off a shelf then twisted to his right side to miss a bench. The resulting severe pain was central at approximately the L4–S1 level without radiation. He sat down then had to lie down due to his low back pain. The morning after the injury, he awoke with a very painful low back so he took 1 week off from work during which time he consulted his medical practitioner who prescribed Nurofen. He returned to work after the week off and worked in pain; he kept working but required a couple of days off periodically and he avoided any heavy work. Later he was referred for physiotherapy treatment but only received temporary relief.

He was referred to a medical specialist 1.5 years after his accident at which time a lumbosacral X-ray examination was arranged that was reported as being normal.

PRESENTING COMPLAINT(S) (Fig. 24.1)

Chronic constant low back pain, since heavy lifting 2.5 years ago, that is central at the L4–S1 level and is of variable intensity. There is no radiation of pain from the lumbosacral area but he has noticed some 'tingling' extending from the low back to his left buttock then to behind the left thigh as far as his knee. He said he can walk for 30 minutes and does so in order to keep fit. There is no night pain per se. He said that he had not experienced any bowel or bladder problems. The pain is aggravated by sitting or standing for more than an hour, rising from the

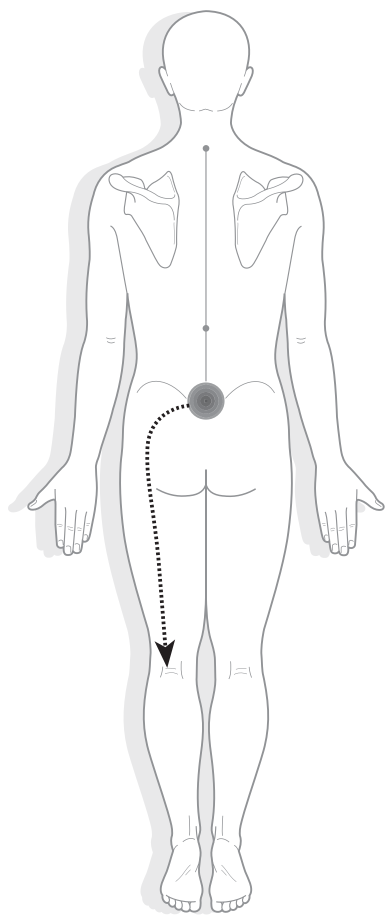

Figure 24.1

seated position, coughing, bearing down, work activities and being in bed overnight. On arising in the morning, his low back is stiff and painful. The pain is temporarily relieved by heat, lying down and using Voltaren medication.

He is very frustrated at not being able to recover and work without low back pain and 'tingling' in his left leg as these symptoms concern him regarding his future which he feels is very uncertain, particularly in view of his inability to lift objects at work.

AETIOLOGY

Lifting a heavy weight from a shelf and twisting 2.5 years ago.

EXAMINATION

In the erect posture there was no clinical evidence of leg length inequality or of scoliosis. Percussion of the thoracic and lumbar spines did not elicit any pain. Deep palpation of the thoracic and lumbar paraspinal muscles elicited pain bilaterally at the lumbosacral level, with some palpable muscle spasm on the right. Erect posture testing of the sacroiliac joints did not cause any sacroiliac joint pain but elicited low back pain. Toe walking power (S1) and heel walking power (L5) were normal. Deep tendon reflexes in the upper and lower extremities were normal. The plantar response was normal. Vibration sensation at the elbows and ankles was normal. Pinprick sensation in the lower extremities was normal. Sitting in the slumped forward position elicited an increase in low back pain; the addition of straight leg raising on the left and right sides, respectively, aggravated his low back pain. The foot pulses were normal. The circumference of the calf (10 cm below the patella) was 40.5 cm (left) and 39.5 cm (right), i.e. within normal limits. The Valsalva manoeuvre caused a significant increase in his low back pain, suggesting a space occupying lesion within the spinal canal, e.g. a disc bulge or protrusion. Supine straight leg raising was to a measured 60° bilaterally, both being limited by hamstring tightness. Left and right straight leg raising with foot plantar flexion were painless, whereas straight leg raising with left foot dorsiflexion elicited an increase in low back pain. Lasegue's sign was painless. The Milgram active bilateral straight leg raise elicited low back pain. Bilateral hip flexion did not elicit any increase in low back pain. Cervical spine flexion with bilateral hip flexion did not elicit any increased low back pain.

Active lumbar spine ranges of movement are shown in Box 24.1.

Box 24.1 Active lumbar spine ranges of movement

	Approx. range	Patient's comments
Flexion	Fingers reached to approx.10 cm above his knees	Elicited low back pain
Extension	Full	Elicited low back pain
Lt lateral bending	Fingers reached to approx. 5 cm above the knee	Elicited low back pain
Rt lateral bending	Fingers reached to approx. 5 cm above the knee	Elicited low back pain
Lt rotation	Approx. full	Elicited slight low back pain
Rt rotation	Approx. full	Painless

IMAGING REVIEW

Lumbosacral spine plain X-ray images reported as being normal were viewed. However, in spite of the 'normal' radiology report, the L5–S1 intervertebral disc space height had narrowed when compared with the L4–5 intervertebral disc space height (Fig. 24.2).

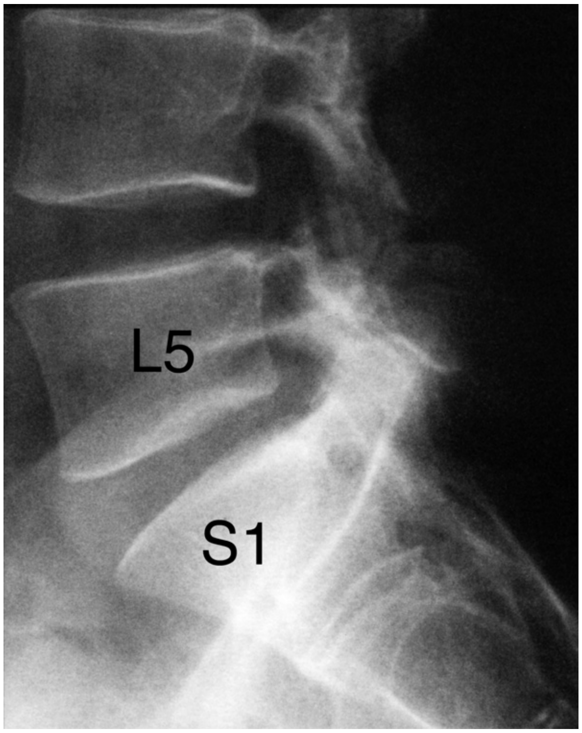

Figure 24.2 Lumbosacral spine lateral plain X-ray image. Note that the L5–S1 intervertebral disc height is narrower than is the L4–5 intervertebral disc height.

CLINICAL IMPRESSION

Possible left paracentral L5–S1 intervertebral disc posterior bulge or protrusion.

WHAT ACTION SHOULD BE TAKEN?

An MRI lumbar spine examination was requested and was reported as follows. L4–5 level: 'There is early disc desiccation with a mild annular bulge.' (Fig. 24.3). L5–S1 level: 'There is disc narrowing and desiccation. There is a large posterior left paracentral sequestrated disc fragment, extending superiorly behind the L5 body that measures approximately 1.9 cm high × 1.2 cm in diameter (Figs 24.3 and 24.4). There is an associated central to left paracentral disc protrusion with effacement and compression of the left S1 nerve root'.

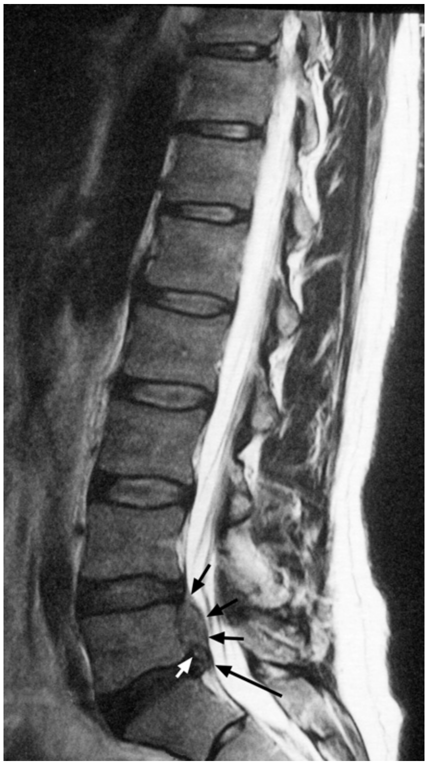

Figure 24.3 Lumbar spine MRI parasagittal T2-weighted image. Note what was reported as a large sequestrated disc fragment extending superiorly behind the L5 body (short black arrows), above the large protrusion (long black arrow) at the L5–S1 level, that has a tear/high intensity zone in it (white arrow). (See Fig. 24.4 for a series of axial views.) However, the density of the supposed sequestrated disc fragment is quite different to that of the large protrusion posterior to the L5–S1 intervertebral disc, so a differential diagnosis of epidural haematoma should be considered for the material extending superiorly behind the L5 body.

A series of axial images through the L5–S1 intervertebral disc posterior protrusion and the supposed sequestration is shown in Figure 24.4.

The L5–S1 posterior intervertebral disc protrusion (Figure 24.4D) presses upon the pain sensitive anterior surface of the dural tube and the forming left S1 nerve root.

A year and nine months later a further MRI examination was performed and reported as follows. L5–S1 level: 'Comparison has been made with the previous study performed. During this time interval there has been a remarkable improvement with the previously noted sequestrated disc fragment at L5–S1 level no longer apparent (Fig. 24.5). Only a posterior disc protrusion with a small annular tear/fissure is noted at this level, contacting the thecal sac and the descending S1 nerve roots (Fig. 24.6). Mild disc space narrowing and desiccation are also noted at the L5–S1 level'.

This confirms the suspicion that, rather than a large sequestrated disc fragment extending superiorly behind the L5 body, the lesion was most likely an epidural haematoma that subsequently resolved.

The L5–S1 posterior disc protrusion still contacts the anterior pain sensitive part of the dural tube/thecal sac and is close to the forming left S1 nerve root (Figs 24.5 and 24.6). The atrophy of the multifidus muscles is again noted.

DIAGNOSIS

Chronic musculoligamentous soft tissue injuries of the lumbar spine including:

- L5–S1 significant posterior disc protrusion with associated epidural haematoma (later re-absorbed)
- L5–S1 annular tear in the disc protrusion
- L4–5 disc annular bulge.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised to undertake the following measures:

1. Undertake a suitable exercise programme to strengthen the muscles supporting his low back (See Case 1).
2. Gradually increase his walking ability.
3. Minimize his Voltaren medication.
4. Perform only light duties in his self-employment capacity.
5. Should conservative treatment not provide adequate relief he was advised that he may eventually require decompressive surgery at the L5–S1 level.

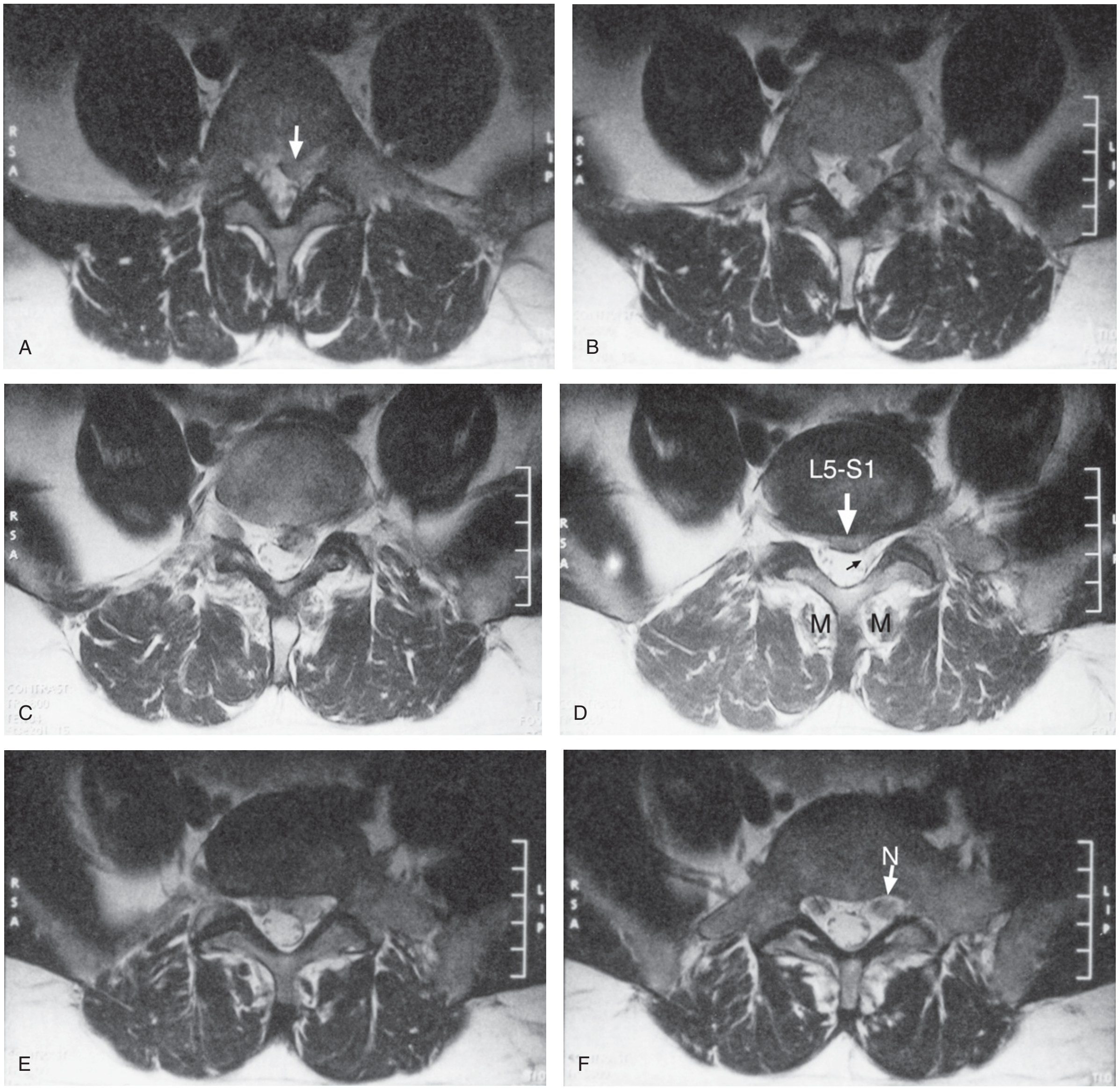

Figure 24.4 A series of MRI axial T2-weighted images showing the top of the probable epidural haematoma (arrow) in A and lower images showing it in B and C. The L5–S1 central to left paracentral posterior intervertebral disc protrusion (white arrow) that presses upon the dural tube anteriorly is close to the forming left S1 nerve root (small black arrow in D). Note the normal spinal canal seen in F with normal nerve roots (N). Early atrophy of the multifidus muscles (M) is seen.

Note

Compare this case and its imaging with Case 25 which shows a posterior intervertebral disc protrusion (of uniform density) without an epidural haematoma.

> **KEY POINT**
>
> Carefully view all images so you may decide whether the reports accurately reflect the pathology present.

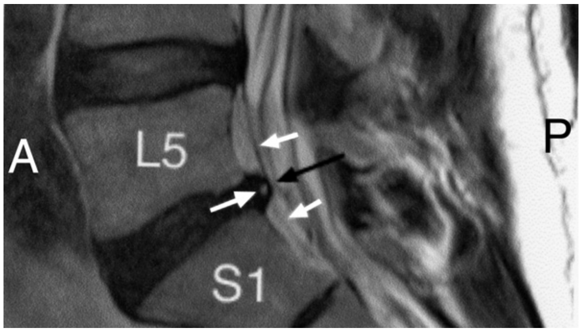

Figure 24.5 Lumbar MRI parasagittal T2-weighted image. Note: (i) there is early desiccation of the L4–5 and L5–S1 intervertebral discs, (ii) there still is a significant posterior disc protrusion at the L5–S1 level (large black arrow) that presses upon the pain sensitive anterior part of the dural tube (small white arrows) and, (iii) there still is a high intensity zone representing an annular tear/fissure in the disc protrusion (white arrow). (Compare Fig. 24.3 which is almost in the same parasagittal plane.) The highly probable epidural haematoma has resolved.

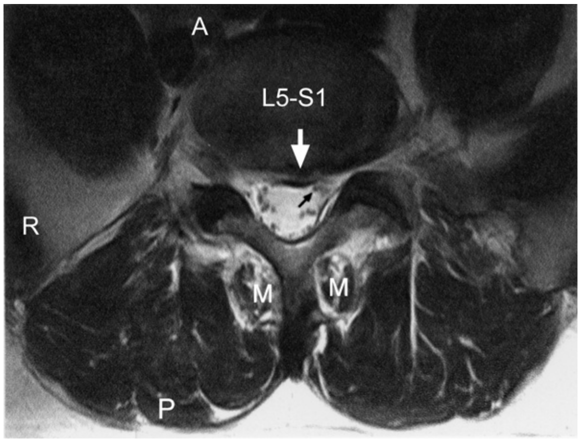

Figure 24.6 L5–S1 MRI axial T2-weighted image. Note: (i) the persisting L5–S1 posterior central to left paracentral disc protrusion (white arrow) presses upon the thecal sac and is close to the forming left S1 nerve root (small black arrow) and, (ii) there is early atrophy of the multifidus muscles (M). (Compare Fig. 24.4D which is almost in the same axial plane.)

Further reading

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Case 25

Re-absorption of intervertebral disc material

COMMENT

Prolapsed disc material may be re-absorbed with cautious conservative care.

PROFILE

A 49-year-old male who is a non-smoker and only drinks alcohol socially and who is involved in manual work was referred by his general medical practitioner for evaluation and treatment.

PAST HISTORY

The referring doctor's letter stated that the patient had experienced intermittent low back problems for approximately 10 years but that there was no other past medical history of note, which the patient confirmed.

PRESENTING COMPLAINT(S) (Fig. 25.1)

Periodic low back pain that has occurred since he sneezed 10 years ago while bending forwards and lifting; this painful episode caused him to spend 8 days in bed. Two weeks ago he developed severe acute low back pain and, for the first time, right sciatica with paraesthesiae and a 'cold' feeling in the right foot.

He was trying non-steroidal anti-inflammatory medication and Panadeine Forte without much success. He had undergone several physiotherapy traction sessions which gave some temporary relief.

AETIOLOGY

Nothing specific for this episode; the symptoms had begun during his work 2 weeks ago; he thought he may have twisted his back getting in and out of the car.

EXAMINATION

Because of the acute low back pain and right sciatica with paraesthesiae in the right foot, he sat on the chair on his left buttock with his right leg extended, in order to minimize the low back pain and sciatica. The right ankle jerk

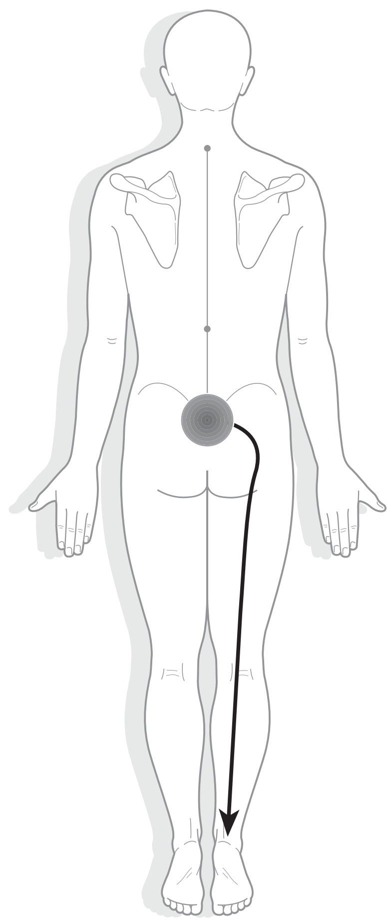

Figure 25.1

(S1) was absent but other deep tendon reflexes in the lower extremities were normal. Supine SLR was normal on the left but right SLR to 20° elicited an increase in the low back pain and radiation into the right buttock. Active lumbar spine movements were considerably restricted due to low back pain and an increase in pain radiating into the right buttock.

IMAGING REVIEW

Plain lumbosacral spine and sacroiliac joint radiographs taken 6, 4 and 1 year(s) previously were reviewed; these showed a complete left-sided lumbarization of S1 and a partial right-sided lumbarization. Minimal lipping changes were present at the anterior margins of most lumbar vertebral bodies, although the disc space heights were intact at the time of these investigations.

CLINICAL IMPRESSION

An acute right-sided disc protrusion at the L4–5 or L5–S1 level.

WHAT ACTION SHOULD BE TAKEN?

A CT scan was ordered of the lower lumbosacral spine (L3 to S1). The radiology report stated that, at the L4–5 disc level, there was a 'disc prolapse impinging upon the right L5 nerve root with effacement of the epidural fat. The differential diagnosis includes a right paracentral focal disc protrusion/oedema of the adjacent L5 or S1 nerve root, or a small neurofibroma'. A further possible differential diagnosis of epidural haematoma could be considered (as the clinical findings in spontaneous epidural haematoma are identical to those in acute disc herniation (Gundry & Heithoff 1993)). However, the posterior to right sided intervertebral disc protrusion in this case is of uniform density, suggesting that it is not an epidural haematoma.

DIAGNOSIS

L4–5 posterior to right sided disc protrusion with right L5 radiculopathy.

TREATMENT AND RESULTS

The patient was told that he had a disc prolapse at the L4–5 level that was pressing on the adjacent nerve root (Fig. 25.2A). He was advised to continue with his NSAID medication and, in order to resolve his acute low back pain and sciatica, that it would be prudent to give him a few trial treatments using a chiropractic flexion-distraction technique modified from Cox (1999), i.e. 4 × 15 seconds of traction. He was told that the prolapse may become worse, requiring urgent surgical intervention. As he was already in so much pain, surgery was a likely possibility in any case. He signed a release form and treatment was begun immediately and a letter was written to his referring medical practitioner asking for the patient to be given 3–4 weeks off work. The patient was told to rest in bed before being seen 3 days later. He experienced some temporary soreness immediately after the flexion-distraction treatment but that had soon settled, so he decided to go to a wedding reception that evening where he sat for approximately 2 hours without undue pain. He then rested in bed. When he returned 3 days later he reported that he was 'much better' overall and that the pain had virtually gone from the right lower limb.

He was then seen again 2 days later for further flexion-distraction treatment and he said he was much better still. He had two further treatments and, because he was so much better, a CT scan was performed 2 weeks after the first CT scan through the L4–5 disc with a request to the radiographer to position the patient as closely as possible to the previous CT scan examination position. The disc prolapse was still evident but was perhaps a little smaller (Fig. 25.2B). Following a total of seven flexion-distraction treatments during a 2-week period he was so much better that he wanted to go back to work, in spite of still having a valid off-work certificate; his general medical practitioner was asked to give him a 'Return to Work' certificate for light duties for a 3-month period at which time he would be reviewed again before he returned to normal duties. As a differential diagnosis had included the conditions mentioned above, an MRI scan was performed approximately 3 months after the initial CT scan as he still had some minor residual low back pain and some intermittent paraesthesiae in the right leg (see Fig. 25.2C and D). This confirmed the L4–5 right sided disc protrusion.

Approximately 8.5 months following the original MRI scan (Fig. 25.2C and D) a further MRI scan was performed (Fig. 25.2E). This showed no disc protrusion. Ten months after the initial CT scan was performed the patient was asked whether he would mind having a further CT scan performed (just through his L4–5 disc) to see what the spinal canal looked like now that he was virtually asymptomatic and was performing normal duties. He replied that he was interested to know and that he would be pleased to have this done, so a CT scan was performed (see Fig. 25.2F).

The final MRI and CT scans (Fig. 25.2E and F), which again were taken in almost exactly the same position as the prior scan positions, showed that there was complete re-absorption of the disc protrusion.

The patient was asymptomatic for 1 year and 8 months until he fell onto his lower back and caused some pain in the upper lumbar spine but there was no recurrence of low back pain or right leg symptoms.

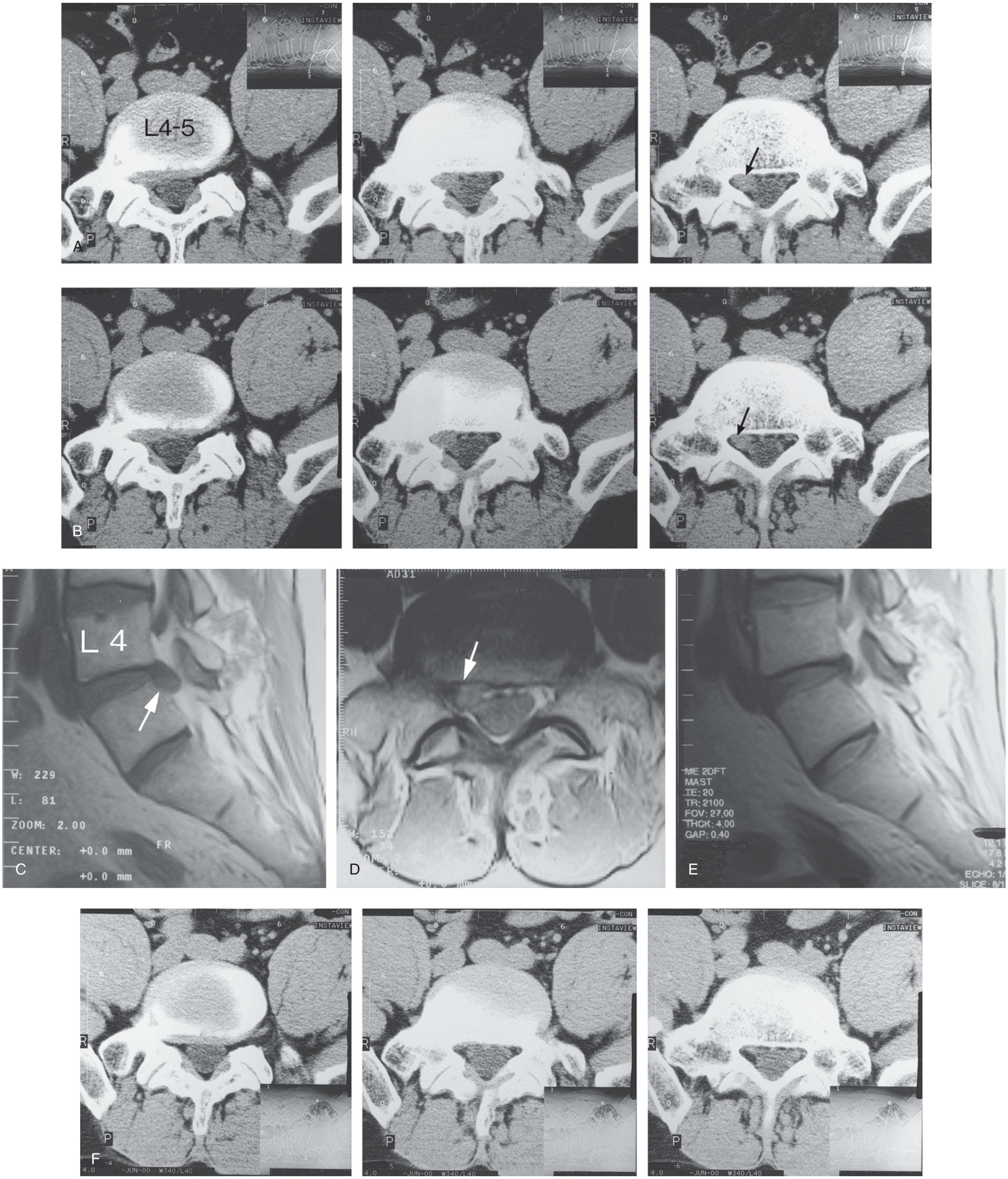

Figure 25.2 (A) Initial L4–5 disc CT axial image. Note that the radiology report stated: 'A fairly large right sided soft disc prolapse at L4–5 that is impinging upon the right L5 root as it buds off from the theca (arrow). No other significant feature is apparent'. (B) First follow-up L4–5 disc CT axial image 2 weeks after the initial CT scan. Note that the disc prolapse (arrow) remains essentially the same. (C) MRI parasagittal T1-weighted image (3 weeks after the CT scan in B). Note the L4–5 disc protrusion is still present. This parasagittal view shows the disc protrusion in another dimension and that it migrated inferiorly (arrow). (D) An MRI axial T1-weighted image (3 weeks after the CT scan in B) showing the right lateral protrusion of the L4–5 disc (arrow) impinging upon the right L5 nerve root. (E) MRI parasagittal T1-weighted image, taken approximately 8 months after the original MRI shown in (D), shows that the right-sided soft disc prolapse at L4–5 has been absorbed. (F) L4–5 disc level CT axial image taken 10 months after the initial CT scan in (A) to confirm that re-absorption, as compared with the original CT scan, was maintained. Note that the right sided disc protrusion at L4–5 remains re-absorbed.

Note

Many papers have been written stating that prolapsed disc material has been re-absorbed. However, this can only be stated with certainty when initial and follow-up imaging is performed in a manner that produces follow-up imaging slices at the same anatomical level and in the same plane, or very close to the initial anatomical position and imaging plane. The bony and soft tissue structures in the CT and MRI slices show that this requirement was met in this case. Therefore, the absorption of prolapsed disc material in this case is a real phenomenon.

This case, and the writings of Dr James Cox (1999), raise the question of whether surgery should be undertaken without prior appropriate multidisciplinary conservative management having first been tried in an attempt to help patients with acute low back pain and sciatica.

KEY POINTS

1. Prolapsed disc material may be re-absorbed with conservative treatment.
2. Surgery should not be considered as an initial option unless there are symptoms and signs of cauda equina syndrome.
3. Because the protrusion was right sided and impinged upon the right L5 nerve root the patient experienced typical low back pain and right sciatica.

References

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Ito T, Takano Y, Yuasa N 2001 Types of lumbar herniated disc and clinical course. Spine 26: 648–651.

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Saal J A, Saal J S, Herzog R J 1990 The natural history of lumbar intervertebral disc extrusions treated nonoperatively. Spine 15: 683–686.

Case 26

L5–S1 intervertebral disc protrusion

COMMENT

Flexion–distraction treatment in cases with no contraindication can be very effective.

PROFILE

A 31-year-old housewife of average build who smokes approximately 25 cigarettes per day and drinks alcohol occasionally.

PAST HISTORY

Ten years ago she first experienced low back pain after sustaining a low back injury while she was working as a disability support carer. The low back pain gradually settled. Approximately 1 year later she experienced an acute flare-up of low back pain with muscle spasm after sneezing. Panadeine and Panadeine Forte had been prescribed then a non-steroidal anti-inflammatory (Vioxx 25 mg per day) and Valium (5 mg per day) but her condition became chronic.

PRESENTING COMPLAINT(S) (Fig. 26.1)

She presented with a 2 month recurrence of semi-acute low back pain with radiation into the left buttock and left thigh posteriorly with some occasional right-sided buttock pain. She has great trouble coping with lifting her 18-month old daughter, especially when trying to put her into the car baby seat. She moves slowly and cautiously in a somewhat flexed lumbar spine posture. In view of her ongoing symptoms, she had required a recent pethidine injection for the low back pain that provided relief but only on a temporary basis.

AETIOLOGY

Possibly lifting her daughter into the car with acute aggravation of her chronic low back pain.

EXAMINATION

In the erect posture, there was no clinical evidence of leg length inequality or of pelvic obliquity. There was a degree of lumbosacral muscle spasm with tenderness

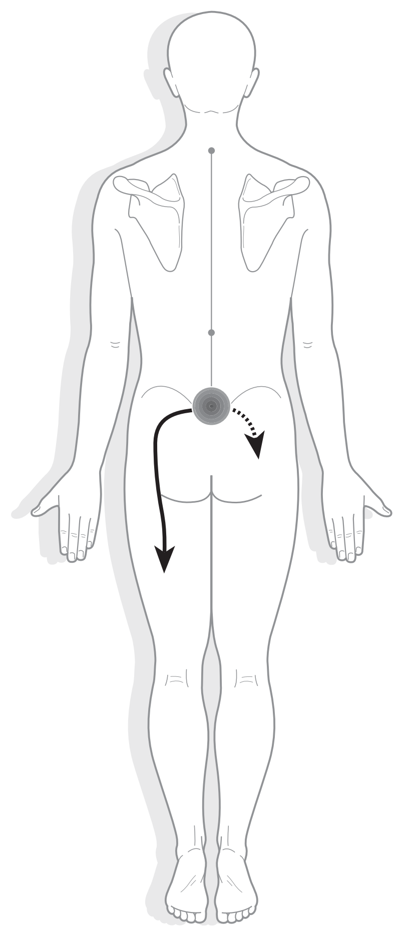

Figure 26.1

on deep palpation of the paraspinal muscles at the lumbosacral level. All lumbar spine active ranges of movement were very limited due to low back pain and slight pain radiating into the left buttock. The slump test was normal and the addition of straight leg raising did not elicit any significant increase in low back pain. Supine straight leg raising was to approximately 70° elevation bilaterally without eliciting any increase in her low back symptoms. Deep tendon reflexes in the lower extremities were normal as were the foot pulses. Pinprick sensation did not elicit any abnormality. She presented as a cheerful, stoic lady who was used to coping with low back pain.

IMAGING REVIEW

A plain X-ray examination of the pelvis and lumbar spine performed eight years ago had been reported as follows: 'Degenerative changes at the L5–S1 disc space with minor loss of disc space height. No neural arch defect or spondylolisthesis. Sacroiliac joints appear normal. There is some loss of the normal lumbar lordosis, a finding contributing to her current muscle spasm'.

A CT scan performed 3 years ago had been reported as showing central posterior disc bulges at L4–5 and L5–S1 levels.

CLINICAL IMPRESSION

Central disc protrusion at L5–S1.

WHAT ACTION SHOULD BE TAKEN?

In view of the lumbosacral plain film and CT findings, an MRI lumbar spine was requested to clarify the current clinical situation. The report stated: 'The upper lumbar spine appears intact. There is mild disc desiccation of the three lower lumbar discs. L3–4 level: There is a moderate sized central disc bulge contacting and impressing the dural tube (Fig. 26.2). L4–5 level: There is a shallow central disc bulge contacting and impressing the dural tube. L5–S1 level: There is early narrowing and a large central to left sided disc protrusion (Figs 26.2 and 26.3) that compresses the S1 nerve roots, predominantly on the left'.

DIAGNOSIS

- Musculoligamentous soft tissue injuries of the lumbar spine.
- Large focal central to left sided intervertebral disc protrusion at the L5–S1 level causing low back pain with left, and occasional right, sided radiculopathy.

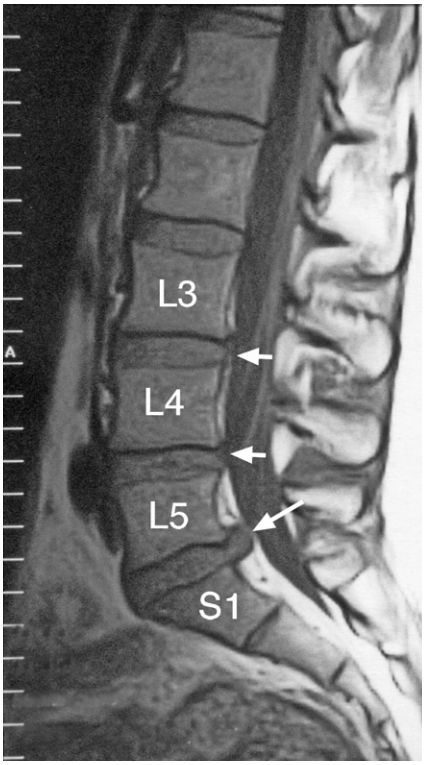

Figure 26.2 Lumbosacral spine MRI sagittal T1-weighted image showing the (i) L3–4 disc level moderate sized central disc bulge contacting and impressing the dural tube (short white arrow), (ii) L4–5 level shallow central disc bulge contacting and impressing the dural tube (short white arrow), and (iii) L5–S1 early narrowing and a large focal disc protrusion (large white arrow).

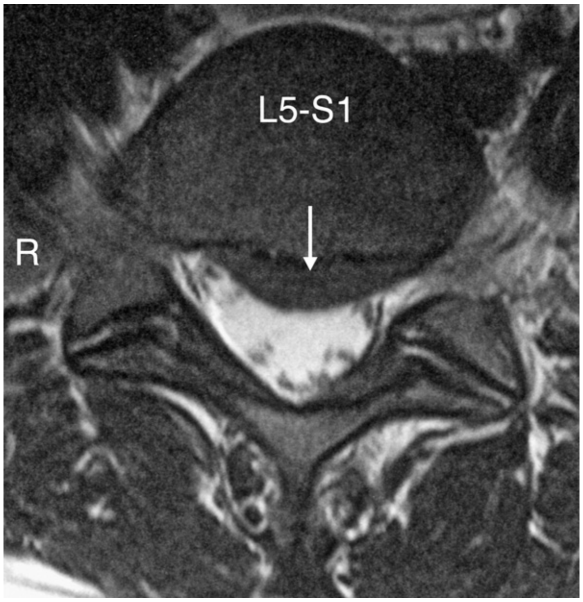

Figure 26.3 L5–S1 MRI axial (slightly oblique) T2-weighted image. Note that the large central to left sided disc protrusion (arrow) compresses the S1 nerve roots, predominantly on the left.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Treatment options were discussed with her with a view to attempting to minimize her symptoms and to lessen her intake of medication. She opted for needle acupuncture treatment to be followed by flexion-distraction chiropractic treatment and, if required, an orthopaedic or neurosurgical opinion. She was advised to walk as much as she could for exercise.

Traditional needle acupuncture treatment was performed on five occasions with initial improvement but then, due to lifting her baby, she caused a considerable deterioration of her symptoms of low back pain with pain radiating into the left buttock and into the left leg posteriorly.

She was offered a trial of three chiropractic flexion-distraction treatments (Cox 1999) (lasting 20 seconds with contact being made at the L4 level), as she was now experiencing significant difficulty trying to look after her 18-month-old daughter. When seen 2 days later she said that she had felt 'very sore' during the night following flexion-distraction treatment but felt 'really good' the following day and again on the second day when she underwent similar flexion-distraction treatment. When she returned 1 week later for further flexion-distraction treatment she said she had experienced her 'best week in years' with no pain at all. One week later she said she was still doing 'really well', so she underwent one further flexion-distraction treatment and was asked to return 2 weeks later for review. At that time she said her improvement had been maintained and that she was very pleased that she could now lift her daughter without aggravating her low back or left leg pains. She was discharged from care and advised to continue walking as much as possible for exercise and to return for flexion-distraction treatment as required. She has required occasional flexion-distraction treatment but feels that her condition has been well controlled and that she now has a good quality of life and essentially takes no medication, apart from an occasional paracetamol tablet.

KEY POINT

Flexion-distraction treatment used by chiropractors who have a flexion-distraction manipulation table can be very effective.

Reference

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Case 27

Leg length inequality

COMMENT

Radiographic erect posture examination for leg length inequality is essential when leg length inequality is suspected.

PROFILE

A 37-year-old male sedentary worker of muscular build who does not smoke and only drinks alcohol occasionally.

PAST HISTORY

No recollection of unusual illnesses or of trauma of any kind and has always been fit and healthy.

PRESENTING COMPLAINT(S) (Fig. 27.1)

Low back pain of insidious onset of several years duration. The pain does not awaken him at night and he feels better of a morning. There is no radiation to the lower extremities.

Coughing and sneezing do not aggravate his symptoms. Non-steroidal anti-inflammatory drugs have not been helpful nor have analgesics. He had seen three medical practitioners regarding his chronic low back pain and presented with three sets of good quality lumbosacral spine radiographs taken in the anteroposterior, lateral and oblique views while in the recumbent position.

AETIOLOGY

Unknown.

EXAMINATION

The deep tendon reflexes in the lower extremities were normal as was the case with pinprick sensation and vibration sensation at the ankles. Power and tone were normal

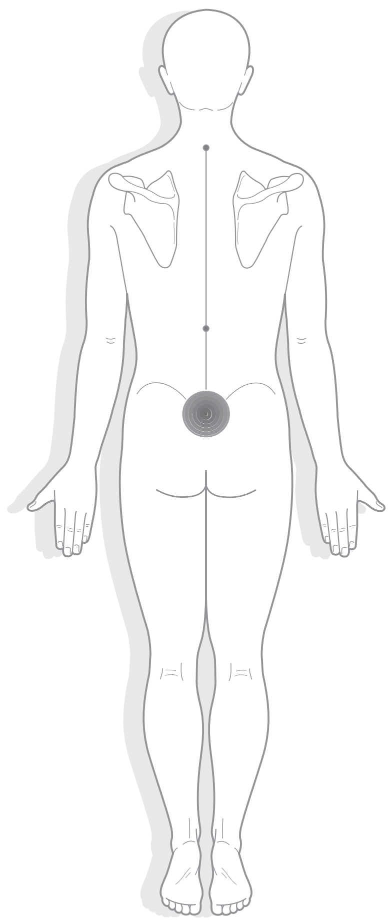

Figure 27.1

and the plantar response was normal. Deep palpation of the paraspinal muscles elicited pain at the lumbosacral level. The circumference of the thighs and calves was equal on both sides. Bearing down did not increase his low back pain. SLR did not aggravate his low back pain. Erect posture examination indicated pelvic obliquity, with the posterior superior iliac spine lower on the right side, with a postural scoliosis. The abdomen was normal on examination.

IMAGING REVIEW

All the previous imaging had been performed in the recumbent position and, although the imaging was of good quality (see Fig. 27.2A), there was no indication of his underlying pelvic obliquity.

CLINICAL IMPRESSION

Low back pain due to leg length inequality, pelvic obliquity and postural scoliosis.

WHAT ACTION SHOULD BE TAKEN?

As clinical measurements for leg length inequality are inaccurate and it is doubtful if many would trust a measured difference of less than three-quarters of an inch (19 mm) (Gofton 1971), and Giles & Taylor (1981) showed that erect posture radiography had a mean error of 1.12 mm (± 0.92 mm), an erect posture pelvis and lumbar spine anteroposterior radiograph was taken with him standing on a horizontal steel platform with the film in a horizontal bucky and touching the inside bottom of the X-ray cassette. This showed a significant leg length inequality of 26 mm on the right side (Fig. 27.2B). The anteroposterior pelvis and lumbar spine radiograph was repeated with the patient standing on a right foot-raise of 26 mm that eliminated the pelvic obliquity and the postural scoliosis (Fig. 27.2C).

DIAGNOSIS

Chronic low back pain associated with leg length inequality, pelvic obliquity and postural scoliosis.

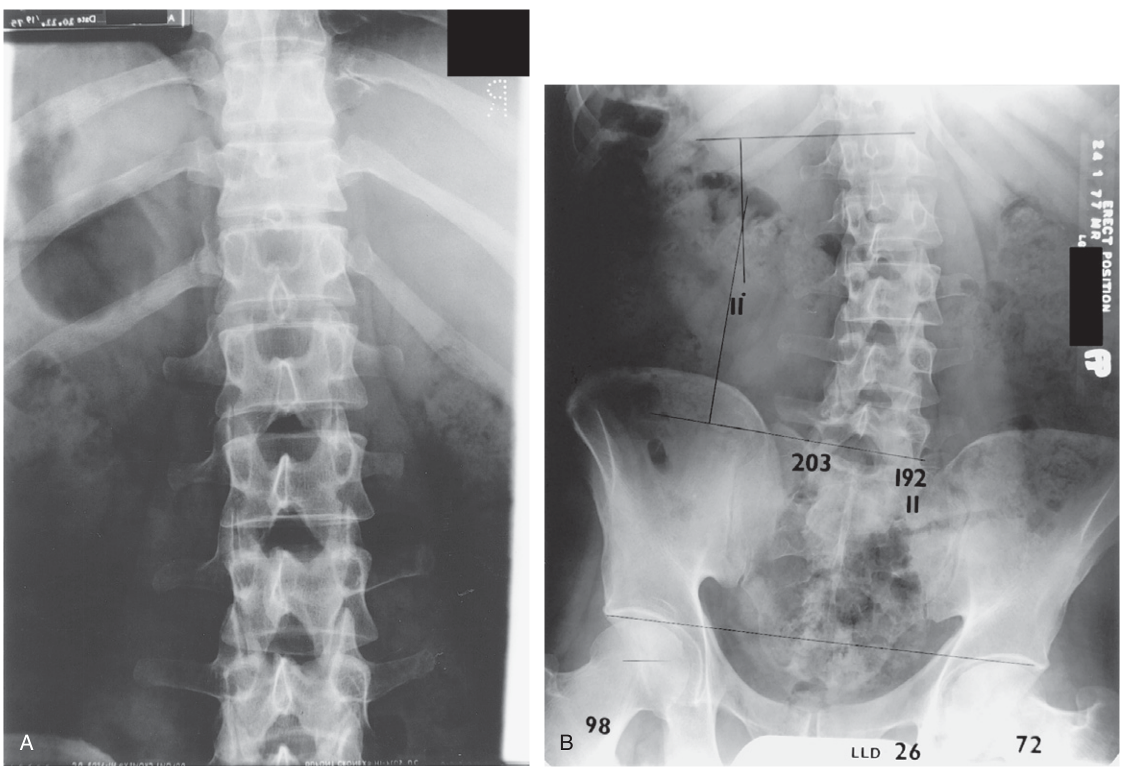

Figure 27.2 (A) A recumbent lumbosacral spine anteroposterior image shows that the lumbar spine is almost straight and gives no indication of the pelvic obliquity and postural scoliosis seen in (B). (B) An erect posture pelvis and lumbar spine anteroposterior image. Note the right leg length discrepancy of 26 mm, the sacral obliquity of 11 mm when measured across the left and right superior sacral notches, and the superimposed lumbar postural scoliosis of 11° measured using the Cobb (1948) method. In addition, the following can be noted: (i) the L3–4 facets on the concave side of the postural scoliosis are apparently more closely apposed than on the convex side; (ii) the L4–5 facets appear to be more closely apposed on the convex side with less apposition of the facets on the concave side; and (iii) the concavity in the inferior surface of the L4 vertebral body is asymmetrical – the concavity is nearer to the convex side of the postural scoliosis.

(Continued)

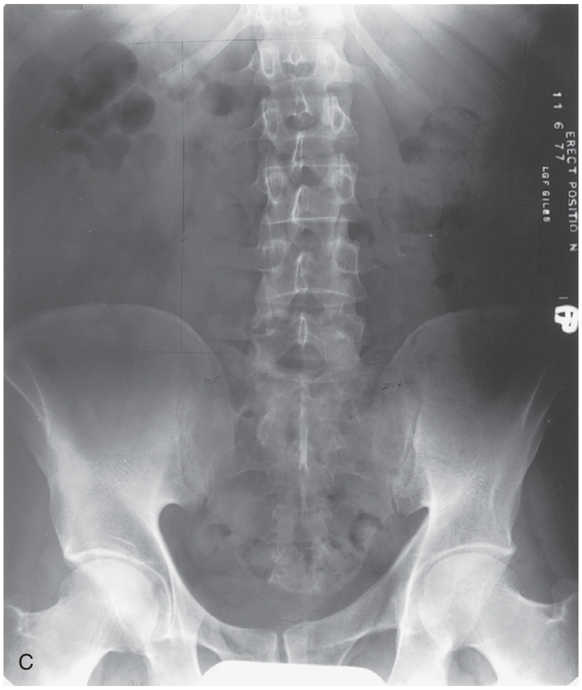

Figure 27.2 Cont'd (C) An erect posture pelvis and lumbar spine anteroposterior image taken with the patient standing on a right foot-raise of 26 mm; this raise virtually eliminated the pelvic obliquity and the postural scoliosis.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised to always wear a right shoe raise of 26 mm and was given a lumbosacral and right sacroiliac joint manipulation with him lying on his left side. He was asked to return in 1 week following building up of his shoes, sandals and slippers.

At the time of his follow-up consultation he said his low back pain had virtually gone. He was delighted that this simple approach should provide him with such dramatic relief; he was advised always to wear built up footwear. He was not seen for 2 years at which time he returned and stated that he was experiencing some recurrence of his low back pain. He was asked whether he was still wearing built up shoes, to which he replied: '*No, because my back was feeling so good!*' He was reminded that he would always have to wear built up footwear under the right foot and to wear one thicker thong (equivalent to approximately 26 mm) on his right foot when he showered. He promised to remember to have all new shoes built up and he was not seen again. A relative stated that his back pain had completely disappeared since wearing the built up shoes.

Note

As a precaution, it should be noted that there may be a leg length inequality but virtually no pelvic obliquity on comparing the left and right superior sacral notches on an erect posture anteroposterior X-ray film. Therefore, this should always be taken into account when measuring for leg length inequality (see Fig. 27.3).

When considering what structures may contribute to low back pain in cases of leg length inequality and pelvic obliquity, apart from the difference in EMG muscle activity between the left and right sides of the spine that is known to occur with patients with a 1-cm or more leg length discrepancy (Vink & Kamphuisen 1989), there are many other possible sources of low back pain. For example, the sacroiliac joints and the lower lumbar spine zygapophysial joints could be sources of pain. A postmortem histological section is shown in Fig. 27.4 which shows some soft tissue structures that may be affected in cases of pelvic obliquity and postural scoliosis. For example,

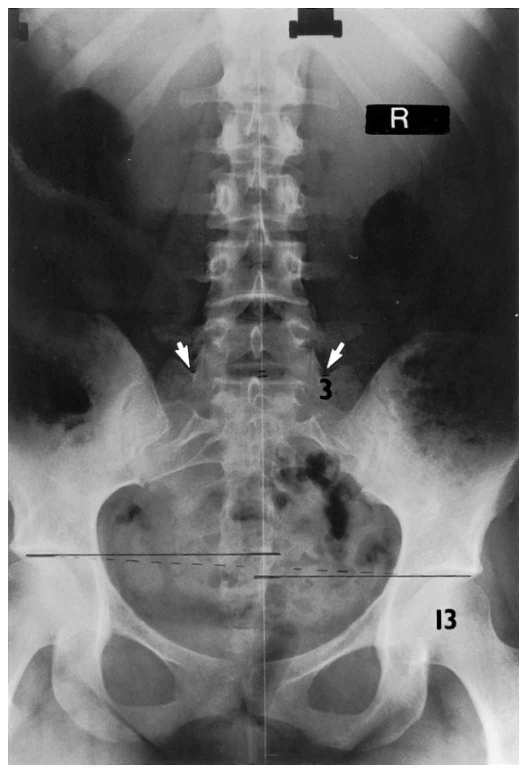

Figure 27.3 An erect posture pelvis and lumbar spine anteroposterior image of a 20-year-old female showing a right leg length discrepancy of 13 mm on drawing a horizontal line from the left femur head to the vertical plumb line and a similar line from the right femur head. The broken line shows the inclination between the left and right femur heads. Note that there is no postural scoliosis as the left and right superior sacral notches (white arrows) are almost at the same height (within 3 mm) as measured to the plumb line. As there is no significant sacral base obliquity, this patient would not require a shoe-raise. (Modified from Giles 1984).

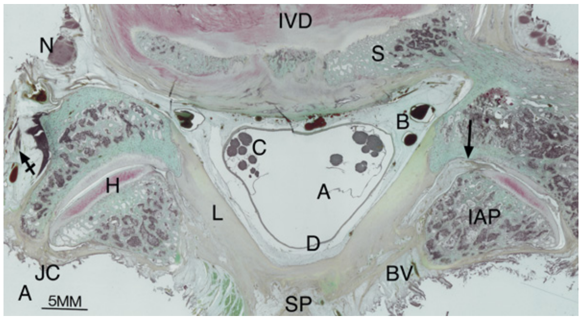

Figure 27.4 A 100-micron thick slightly oblique horizontal histopathology section of the lumbosacral zygapophysial joints at the level of the inferior joint recesses from a 54-year-old male. A = arachnoid membrane; B = Batson's venous plexus; BV = blood vessel; C = cauda equina; D = dura mater; H = hyaline articular cartilage; IAP = inferior articular process of L5 vertebra; IVD = intervertebral disc; JC = posterolateral fibrous joint capsule; L = ligamentum flavum; N = spinal ganglion; R = right side; S = sacrum; SP = spinous process. The intra-articular synovial fold is shown by the black arrow. A neurovascular bundle close to the zygapophysial joint is shown by the tailed arrow. (Ehrlich's haematoxylin stain with light green counterstain). (Reproduced with permission from Giles L G F, Taylor J R 1982 Intra-articular synovial protrusion in the lower lumbar apophyseal joints. Bulletin of the Hospital for Joint Diseases Orthopaedic Institute 42(2): 248–255.) See also colour plate section Fig. vii.9A.

the fibrous joint capsule may be subjected to different stresses between the left and right sides of the spine and the intra-articular synovial fold may become more vulnerable to pinching. In addition it is possible that the outermost layers of the annulus fibrosus are subjected to abnormal stresses leading to disc injuries.

Patients who have leg length discrepancy due to disorders in the lower extremities are at a greater risk of developing disabling spinal disorders due to exaggerated degenerative change; therefore, treatment for leg length discrepancy may be helpful in preventing degenerative spinal changes (Kakushima et al 2003).

KEY POINTS

1. The relationship between low back pain and leg length inequality of greater than 9 mm has been noted by many authors over the years. These authors have come from many different backgrounds as indicated by the further reading list.
2. Erect posture estimation of leg length inequality using the method described by Giles and Taylor (1981) is very useful for accurately determining leg length inequality.

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Case 28

Scanogram versus erect posture X-ray examination for evaluating leg length inequality

COMMENT

When evaluating leg length it is the *overall* length of the lower limb that needs to be evaluated – not just the length of the long bones in a lower limb.

PROFILE

A 42-year-old average weight female teacher who does not smoke cigarettes or drink alcohol.

PAST HISTORY

There is no past history of relevance.

PRESENTING COMPLAINT(S) (Fig. 28.1)

Approximately 6 months prior to consultation she experienced an onset of low back pain. There was no history of a specific precipitating factor but she had experienced an increase in low back pain and right leg pain down the entire length of that leg. There was no night pain and she did not feel unwell apart from her now chronic low back pain with right sided sciatica. She felt that there had been some improvement in her low back and right leg symptoms following 2 months of physiotherapy treatment but this improvement was not long lasting. She then saw her general medical practitioner who prescribed Celebrex but she said this caused gastrointestinal symptoms after 1 month, particularly regarding nausea. She had seen another medical practitioner who provided her with lower lumbar paraspinal injections of corticosteroid with good temporary relief from low back pain. She then saw a podiatrist who arranged a CT scanogram to evaluate leg lengths and a plain X-ray examination of her feet as she appeared somewhat flat-footed. Based on the scanogram, she was told that she had equal leg lengths and she was advised to wear foot orthotics for the 'flattening of the plantar arch bilaterally, more prominent on the left side'.

AETIOLOGY

There was no obvious aetiology for the low back pain and left sided sciatica.

EXAMINATION

In the erect posture there was considerable clinical evidence of pelvic obliquity (down on the left side) with a

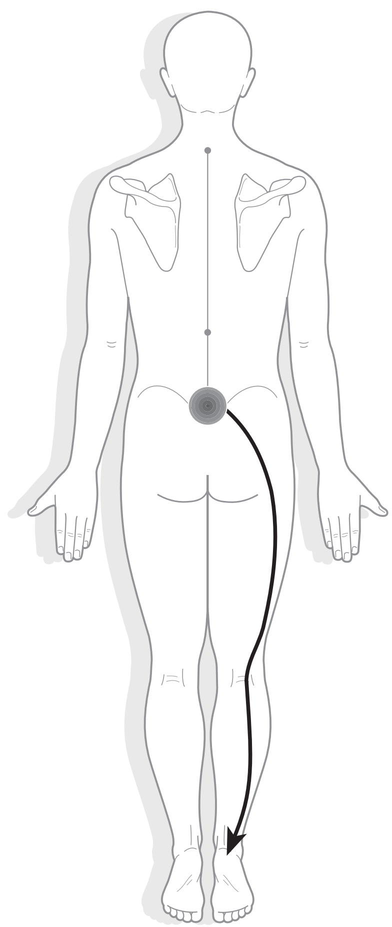

Figure 28.1

degree of left convex postural scoliosis. Deep palpation of the paraspinal muscles elicited local tenderness at the L4–S1 level.

Active lumbar spine ranges of movement in the erect posture were as follows:

1. Flexion – limited to approximately 50% due to an increase in low back pain and referral of pain to the right leg. There was minor pain referral into the left leg.
2. Extension – very limited due to an increase in low back pain.
3. Left and right lateral bending – limited by approximately 50% due to an increase in low back pain.
4. Left and right rotation – limited by approximately 50% due to slight low back pain.

There was a degree of pronation of her left foot. Seated straight leg raising was limited due to low back pain. Supine straight leg raising was limited to a measured 60° on the right due to an increase in low back pain and to 75° on the left. Reflexes in the lower extremities appeared to be normal, as was the case with pinprick sensation. The foot pulses were normal as was muscle power. The abdomen was normal on examination.

Figure 28.2 CT scanogram image. The radiology report stated: 'The right femur measures 472 mm and the left femur measures 469 mm. The right tibia measures 374 mm and the left tibia 374 mm. The overall right leg length is 849 mm and the overall left leg length is 849 mm'.

IMAGING REVIEW

A CT scan showed: 'An L4–5 large central and right sided disc protrusion compromising the region of the origin of the right L5 nerve root sleeve and extending down into the right L5 nerve root foramen'.

A CT scanogram for leg lengths was reported as follows: 'The overall right leg length is 849 mm and the overall left leg length is 849 mm' (Fig. 28.2).

CLINICAL IMPRESSION

Left leg length inequality with pelvic obliquity and left convex scoliosis with a large central and right sided disc protrusion at the L4–5 level.

WHAT ACTION SHOULD BE TAKEN?

The patient was referred for an erect posture pelvis and lumbar spine radiograph (Fig. 28.3) with a request that the examination should be repeated with the patient standing on a 2 cm left foot raise (Fig. 28.4). The radiologist reported a left leg length inequality of 2 cm (Fig. 28.3) with pelvic obliquity and a left convex postural scoliosis and disc thinning at the L4–5 level. In addition, there was early osteoarthrotic change of the L4–5 facets.

In view of the above, a lumbar spine MRI was requested. The radiology report stated: 'There is some loss of disc signal and disc height involving the L3–4 but particularly the L4–5 disc. At L3–4 there is a mild broad based

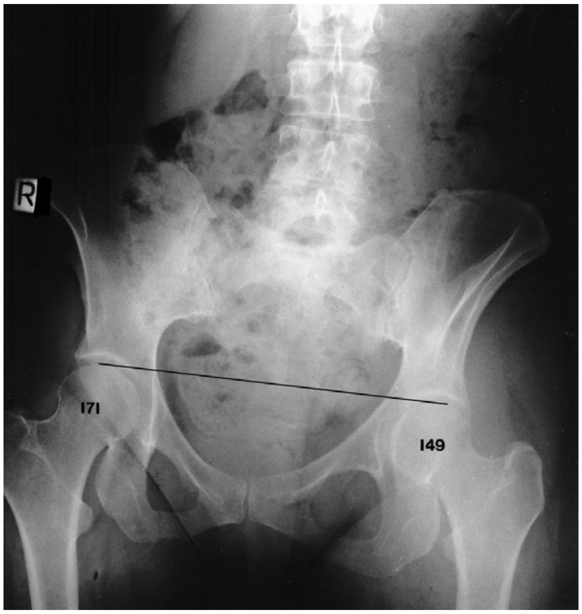

Figure 28.3 Pelvis and lumbar spine erect posture anteroposterior image. Note the left leg length discrepancy of 22 mm, the pelvic obliquity and the left convex postural scoliosis. R = right side of patient.

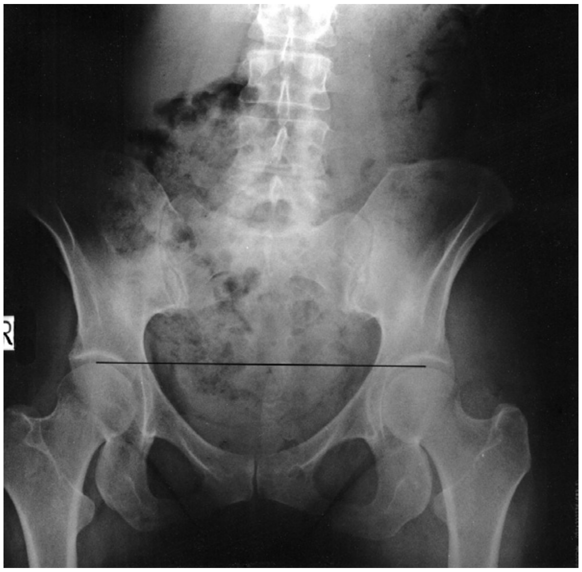

Figure 28.4 Pelvis and lumbar spine erect posture anteroposterior image taken with the patient standing on a right foot raise of 20 mm. Note that this has virtually eliminated the leg length inequality and the left convex postural scoliosis.

disc protrusion while at L4–5 level there is a large central and right paracentral anterior epidural mass that appears continuous with the disc and is likely to represent a large focal disc protrusion. There are likely compressive effects upon the right L5 nerve root. This disc is causing marked reduction in the canal space. There is also a little hypertrophy noted of the posterior facet joints. At the L5–S1 level there is no significant disc bulge and compromise to the canal or exiting nerve roots'.

DIAGNOSIS

Left leg length discrepancy of 22 mm with pelvic obliquity and postural scoliosis as well as:

- L3–4 level mild broad based disc protrusion.
- L4–5 level large central and right paracentral disc protrusion.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. In addition, I explained to the patient that, because the anteroposterior radiographs were weight-bearing radiographs, they provided a more accurate overall lower limb length measurement than the scanogram. However, she initially would not accept this explanation as her podiatrist had told her that the scanogram was a 'completely accurate' method for measuring leg length differences. I explained that the scanogram did not take into account the sphyrion height, i.e. the height of the medial malleolus of the tibia from the sole of the heel (Giles 1989; *Dorland's Illustrated Medical Dictionary* 1994). I further explained to her that the difference in measurements between the erect posture radiographs and the scanogram most likely represented the pronation of her left foot as well as soft tissue compression. However, she was still sceptical, so I spoke to the radiologist who had reported on the scanogram and on the erect posture radiographs. He agreed that there was a real leg length inequality with shortening on the left side and that the scanogram does not take into account issues of pronation and soft tissue compression of the joints in the lower limbs. I explained this to the patient and she eventually accepted that there was an actual leg length discrepancy between the lengths of her lower limbs.

The patient was told that it would be advisable for her to wear a left shoe raise in an attempt to lessen the mechanical strains on her low back region, apart from lessening the risk of her developing osteoarthrotic changes in her right hip and knee joints, i.e. 'long leg arthropathy' (Dixon & Campbell-Smith 1969).

An initial raise of 10 mm was suggested, with this being built up to 20 mm if it did not aggravate her low back and left leg symptoms in view of the L4–5 level intervertebral disc protrusion. She was also advised to see an orthopaedic surgeon regarding the L4–5 intervertebral disc changes demonstrated on the MRI scan. She was referred back to her podiatrist with a recommendation that the patient should see an orthopaedic surgeon.

There was no further follow-up regarding this patient.

Note

This case illustrates the importance of realizing that CT scanograms should not be relied upon as being accurate for evaluating overall leg length as they do not take into account compression of soft tissues within the lower limbs or pronation of a foot. To determine whether there is radiological evidence of leg length inequality in patients with clinical evidence of pelvic obliquity in the erect posture, carefully standardized erect posture radiography (Giles & Taylor 1981) should be considered. Clinical measurement is notoriously inaccurate and it is doubtful if many would trust a clinical measurement difference of less than 19 mm (Gofton 1971), as mentioned in Case 27.

KEY POINT

Be wary of making a clinical diagnosis of overall leg length using scanography.

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Case 29

Seropositive inflammatory arthropathy

COMMENT

It is important for the treating clinician to actually view the imaging films and never to rely only upon the imaging report.

PROFILE

A 44-year-old male sedentary worker who kept fit by going to the gymnasium and running regularly.

PAST HISTORY

His history included psoriasis and he recalled that his maternal grandmother had rheumatoid arthritis and that his mother had some form of mild arthritis.

PRESENTING COMPLAINT(S) (Fig. 29.1)

Low back pain of about 5 years duration that he said was worse in the cold weather and if he slept on a soft mattress. Sometimes he had severe low back pain, although he is usually free of pain. Occasionally, running caused slight low back pain but he preferred to keep fit, so kept on running. Lifting reasonably heavy items did not cause an increase in low back pain. There was no night pain. During the 2 weeks prior to his consultation the pain had been noticeable on a daily basis but a diagnosis had not been made.

AETIOLOGY

Unknown.

EXAMINATION

In the erect posture there was no evidence of leg length inequality or pelvic obliquity. Reflexes, tone, power, and sensation were normal. Lumbar spine ranges of movement appeared to be normal and there was no evidence of nerve root tension on SLR. The abdomen was normal on examination.

IMAGING REVIEW

An imaging review was not possible, as no imaging had been performed.

CLINICAL IMPRESSION

The history suggested a probable rheumatological condition.

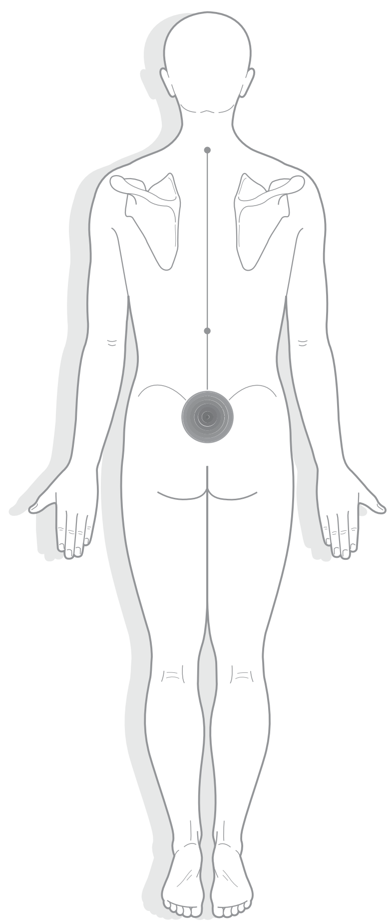

Figure 29.1

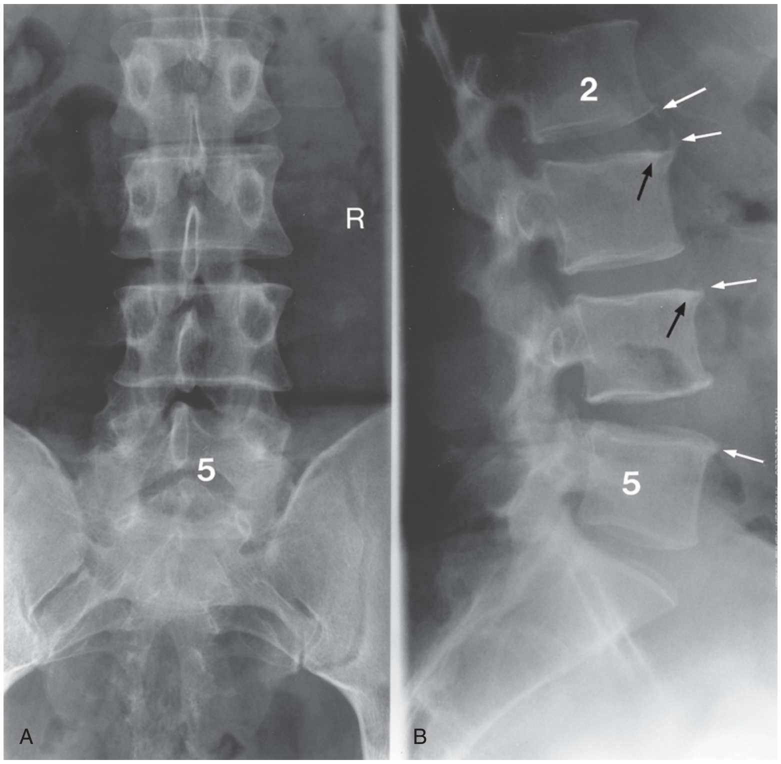

Figure 29.2 (A) Pelvis and lumbar spine erect anteroposterior plain X-ray image (cropped). Note the minimal lumbar scoliosis and the apparently normal sacroiliac joints. 5 = fifth lumbar vertebra; R = right side of patient. (B) Lumbosacral lateral plain X-ray image. Note the early thin bridging osteophytes, particularly at the anterior margins of the L2–3 intervertebral disc space (white arrows), with adjacent sclerotic changes in the intervertebral body endplates anteriorly (black arrows). 2 and 5 represent the second and fifth lumbar vertebrae, respectively.

WHAT ACTION SHOULD BE TAKEN?

Plain lumbar and pelvis X-ray films and appropriate laboratory tests. The plain film X-ray examination performed (Fig. 29.2) was reported as showing 'degenerative marginal osteophytes at all lumbar levels, especially at L2–3. The sacroiliac joints appear normal. There is a minimal lumbar scoliosis'.

However, the thin bridging osteophytes, particularly at the anterior margins of the L2–3 intervertebral disc, with adjacent sclerotic changes anteriorly adjacent to the intervertebral disc space, suggested an arthritic process. Therefore, some haematology and serology laboratory tests were performed; the full blood count, ESR (3 mm/hr; range 2–15), and C-reactive protein (1 mg/l; range <6) were normal. Anti-nuclear serum antibodies were negative as was the HLA B27. However, the rheumatoid factor was 274 IU/ml (normal range <30). Therefore, a diagnosis of seropositive inflammatory arthropathy was made.

DIAGNOSIS

Seropositive inflammatory arthropathy.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. The laboratory finding of a high rheumatoid factor was explained to him. He was prescribed a non-steroidal anti-inflammatory drug to be used only as necessary; this proved to be very helpful and he was referred to a rheumatologist for further management. He made excellent progress, and he was able to keep up his gymnasium activities and running so as to keep fit.

KEY POINTS

1. Always look at the imaging films.
2. Laboratory tests have their limitations, as shown in this case with respect to the ESR and the CRP as indicators of inflammation; these are not infallible markers of inflammation. However, based on a thorough history, clinical examination and imaging findings a clue as to which tests should be performed is possible.

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Case 30

Lumbar vertebral body compression fracture

COMMENT

Some patients appear to have a higher threshold for pain than do other patients, so be wary of treating without a detailed history, physical examination and appropriate imaging.

PROFILE

A tall (approximately 195 cm; 6 feet 5 inches) 54-year-old fit male who is a manual worker in the building industry.

PAST HISTORY

Episodes of low back pain when lifting heavy objects or on bending down to tie his shoe laces.

Eighteen months ago he was involved in a motor vehicle accident, in which he was a passenger in a car that caught the edge of the bitumen strip causing it to skid severely before the car hit a bank; it became airborne before landing heavily on its wheels. The patient hit his head on the roof of the car in spite of wearing a seat belt.

PRESENTING COMPLAINT(S) (Fig. 30.1)

Constant thoracolumbar junction pain that radiates bilaterally to his low back and which began 5 days after the motor vehicle accident 18 months ago. He awakens with increased pain in the morning and has to get up early as he cannot lie in bed due to this pain.

Coughing does not normally cause an increase in pain but taking a deep breath may aggravate the thoracolumbar pain which then radiates around his chest.

Lifting concrete building blocks, weighing approximately 20 kg each, causes a significant increase in his thoracolumbar pains. Before his motor vehicle injury, he used to lay 300–500 building blocks each day but this activity has been significantly curtailed as a result of the injury.

Non-steroidal anti-inflammatory drugs caused ‘stomach pains’ so had to be stopped.

AETIOLOGY

Motor vehicle accident 18 months ago.

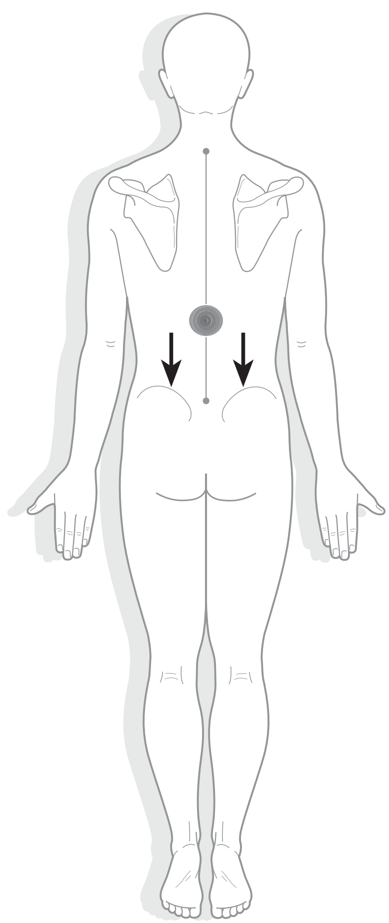


Figure 30.1

EXAMINATION

The knee jerks were asymmetrical in their response (++ on the left and + on the right). The ankle jerks were normal as were the pulses in both feet. Pinprick sensation of the thighs and calves was normal. Vibration sensation of the lower extremities was normal. The circumference of the thighs, 15 cm above the knee, was 47 cm (left) and 48 cm (right). The circumference of the calves, 13 cm below the knee, was 39 cm (left) and 38 cm (right). Power in the legs was normal. Left SLR elicited slight lumbosacral pain at approximately 90° elevation. Bilateral hip flexion elicited slight lumbosacral pain at approximately 120° elevation of the thighs from the examination table. The Naffziger test (compression of the jugular veins accompanied by coughing) elicited some pain in the mid-thoracic spine. Percussion of the spine elicited pain from the thoracolumbar junction to the low back.

Active ranges of lumbar spine movement were as follows:

1. Flexion – the patient was able to touch the floor with his finger tips without any spinal pain.
2. Extension – limited by approximately 40% due to thoracolumbar pain.
3. Left and right rotation – slightly limited due to thoracolumbar 'stiffness'.

IMAGING REVIEW

No imaging was available.

CLINICAL IMPRESSION

Thoracolumbar injury to bone and or soft tissues.

WHAT ACTION SHOULD BE TAKEN?

Plain film thoracolumbar spine X-ray films were ordered (Fig. 30.2A) and showed a fracture of the L1 vertebral body. As more detail was deemed necessary to better visualize the L1 fracture, CT films were ordered, including a 3-D reconstruction (Fig. 30.2B to D). The CT scan report stated: 'Some degeneration of the T12–L1 disc with gas seen in this disc space but there is no disc protrusion. There is no significant compromise of the spinal canal. The L1–2 disc is unremarkable and there is no disc protrusion at this level and no canal stenosis. The anterior wedge fracture of the L1 vertebral body is associated with only a very minimal bowing of the posterior vertebral body margin'.

DIAGNOSIS

L1 vertebral body compression fracture with associated soft tissue injuries.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised to (i) change his employment so that he no longer has to lift heavy building blocks, (ii) perform only light duties, (iii) perform back muscle strengthening exercises (prone thoracic extension exercises), and (iv) to trial a course of needle acupuncture treatment to help with pain control as non-steroidal anti-inflammatory drugs caused gastric pain.

The above advice resulted in a satisfactory outcome for pain management.

Note

R. Maigne (1974, 1978) and J.-Y. Maigne (2000) pointed out that the cutaneous dorsal rami (cluneal nerve) of the T10–L1 or L2 root pass to the left and right iliac crests, explaining why pain may be felt in the low back area due to an injury at the thoracolumbar junction. The cluneal nerves pass over the iliac crest approximately 8 cm from the posterior iliac spine (Kostuik 1997).

In order to more clearly understand what may happen to the spine when a vertebral body compression fracture is sustained, a gross anatomical specimen is shown in the sagittal plane (Fig. 30.2E). This figure shows several vertebral fractures at the superior endplates of T12, L1 and L3; disc material can be seen extending into the fractured vertebral bodies. In this example there is no burst fracture with retropulsion of bone into the spinal canal. A histopathology section showing an endplate fracture in shown in Fig. 30.2F.

KEY POINTS

1. Some patients with serious spinal injuries may have a high pain threshold that appears to trivialize a considerable injury.
2. When a vertebral body compression fracture is present it is important to determine whether only an endplate has fractured, as in this case, or whether there is an associated burst fracture component with bony material encroaching upon the spinal canal (see Case 87).

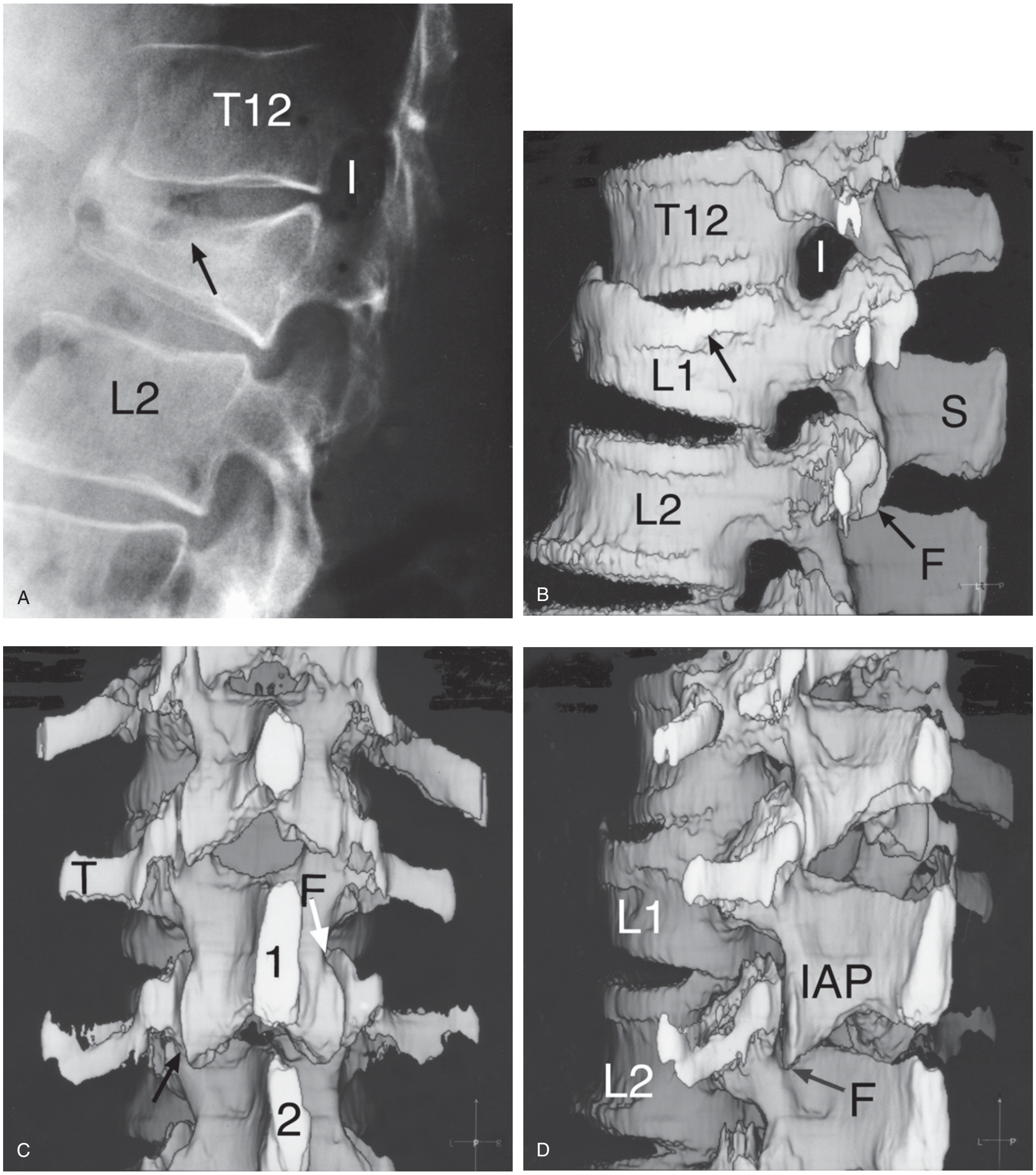
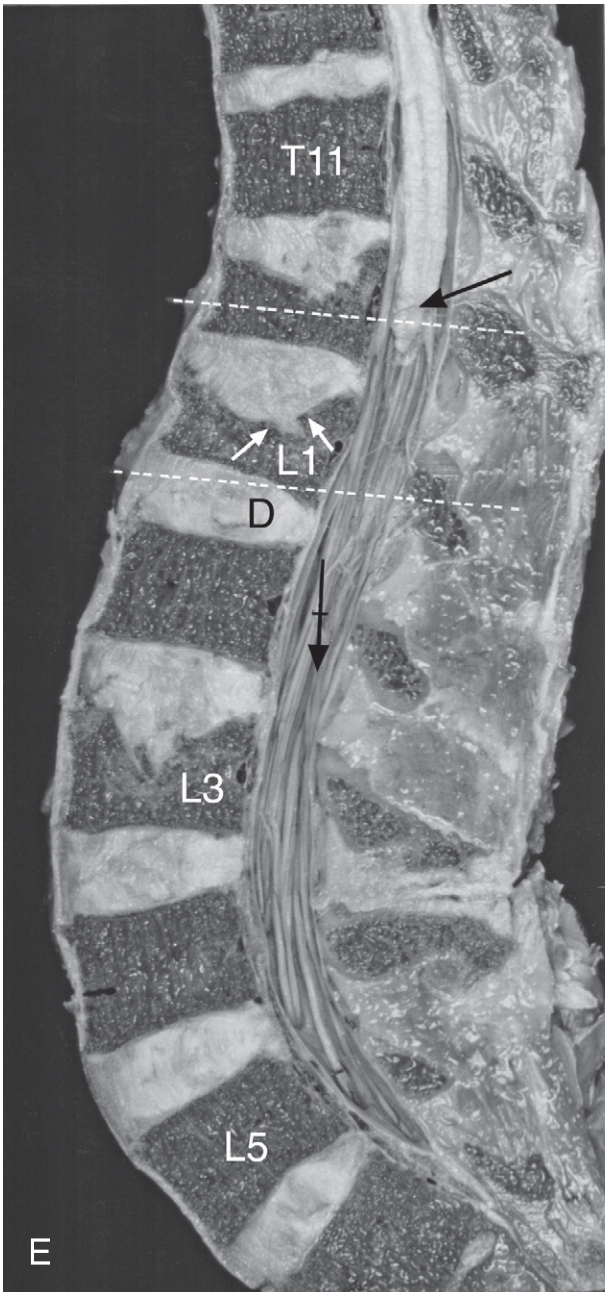


Figure 30.2 (A) A lateral thoracolumbar spine plain X-ray image showing compression of the superior endplate of the body of the L1 vertebra (arrow), consistent with a fracture. I = intervertebral foramen at the T12–L1 level; L2 = second lumbar vertebra. (B) A CT lateral 3-dimensional reconstruction image. This reconstruction image better shows the T12–L1 disc space with the compression of the superior endplate of the L1 vertebral body (arrow). There is some extension of the L1 vertebra on the L2 vertebra with subluxation/imbrication of the opposing facet surfaces (F) at L1–2. This subluxation is seen to be occurring bilaterally in the reconstruction (C) and is also clearly demonstrated in the oblique projection (D). I = intervertebral foramen; S = spinous process. Note how the intervertebral foramen at the L1–2 level (B) is distorted, when compared to the level above, because of the L1–2 subluxation of the facet surfaces due to the L1 vertebra tipping backwards ('extension') on the vertebra below. (C) CT postero-anterior 3-dimensional reconstruction image. T = transverse process; 1 = spinous process of the first lumbar vertebra; 2 = spinous process of the second lumbar vertebra; F = facet surface and subluxation. (D) CT oblique 3-dimensional reconstruction image. Note how the inferior articular process of the L1 vertebra (IAP) approximates the pars interarticularis area of the L2 vertebra below. F = facet surface and subluxation.

(Continued)



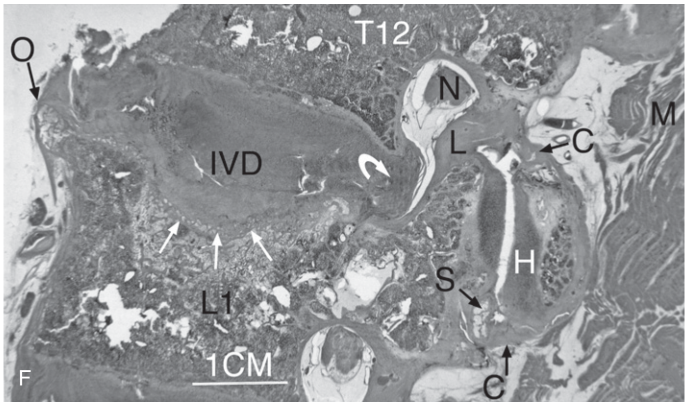


Figure 30.2 Cont'd (E) A gross anatomical postmortem specimen cut in the sagittal plane from a 78-year-old male showing several vertebral body fractures of the superior endplates, i.e. at T12, L1 and L3 where disc material can be seen extending into the fractured vertebral body (white arrows). D = intervertebral disc; L1 = first lumbar vertebra and the white arrows show how the disc material extends inferiorly. Large black arrow indicates the lower part of the spinal cord (conus medularis) from which the cauda equina (nerve roots) extend. The nerve roots are highly vascular and the tailed arrow shows a blood vessel on one of the nerve roots. The rectangle within the broken lines represents the area from which the histological section shown in (F) was obtained. See also colour plate section Fig. vii.23A. (F) A 200-micron thick histopathology section cut in the sagittal plane through the L1 vertebral body compression fracture shown in (E). This shows an osteophyte (O) beginning to develop anteriorly. There is disruption of the intervertebral disc material (IVD) that extends inferiorly to the fractured vertebral body endplate (white arrows). There is some posterolateral bulging of disc material showing encroachment into the adjacent intervertebral foramen (curved white arrow). In this specimen, the intervertebral disc is bulging posteriorly (curved white arrow) into the lower part of the intervertebral foramen but it is still contained within the annular fibres. In addition, the subluxation of the opposing facet surfaces with their hyaline articular cartilage (H) is shown and this can result in tractioning of the joint capsule (C) and pinching of the synovial fold (S). H = hyaline articular cartilage on the inferior articular process of the T12 vertebral body; L = ligamentum flavum; L1 = first lumbar vertebra; M = muscle; N = neural structures within the intervertebral foramen. (Ehrlich's haematoxylin and light green counterstain.) See also colour plate section Fig. vii.23B.

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Case 31

Lumbar intervertebral disc symptoms aggravated by spinal manipulation

COMMENT

Some patients request treatment by spinal manipulation stating that previous manipulation was helpful when, in fact, they did not have spinal manipulation elsewhere.

PROFILE

A 34-year-old average weight female clerical worker. She smokes approximately 20 cigarettes per day but does not drink alcohol.

PAST HISTORY

There is no history of injury apart from an incident approximately 10 years ago when she experienced severe low back pain following bending forward and twisting to pick up a light item at work. She went to her general medical practitioner who suggested bed rest for 10 days and anti-inflammatory medication. She recovered but experienced intermittent episodes of low back pain over the years since that time.

PRESENTING COMPLAINT(S) (Fig. 31.1)

Chronic low back pain of varying intensity since her original low back pain 10 years ago which recently became much worse with a degree of associated right leg sciatica due to bending forwards 2 weeks ago. She found that lying supine temporarily relieved the low back and right leg symptoms. Coughing aggravates the low back pain as do rising from a chair and performing manual activities. Anti-inflammatory medication and rest have not been helpful now that she has right leg symptoms.

AETIOLOGY

Bending forwards 2 weeks ago caused her current episode.

EXAMINATION

There was no clinical evidence of leg length inequality or pelvic obliquity or scoliosis. The right Achilles reflex (S1, 2) was absent and the left and right patella reflexes were depressed at plus one. The circumferences of the

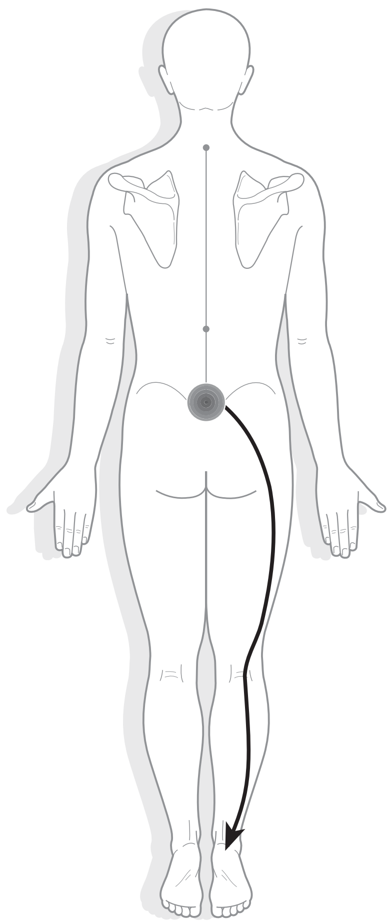

Figure 31.1

left and right thighs were equal as were the circumferences of the left and right calves. Right supine straight leg raising was limited to a measured 30° due to low back pain, while left straight leg raising was to 90° and painless. Power in the lower extremities was normal as were the foot pulses. The slump test aggravated her low back pain. Bilateral hip flexion did not elicit any symptoms, nor did the Valsalva manoeuvre involving bearing down. Ely's heel to buttock test was normal. The abdomen was normal on examination.

Lumbar spine active ranges of movement were:

1. Flexion – fingers reached to her knees and elicited low back pain.
2. Extension – full with slight low back pain.
3. Left lateral bending – fingers reached to her knees and it was painless.
4. Right lateral bending – full range but elicited slight low back pain.
5. Left and right rotation – full range with only sight low back pain.

IMAGING REVIEW

Recent plain film radiographs of the lumbar spine and pelvis were essentially normal apart from possible minor loss of disc space height at the L5–S1 level.

CLINICAL IMPRESSION

A tentative diagnosis of possible right-sided L5–S1 disc bulge.

WHAT ACTION SHOULD BE TAKEN

A CT lumbar spine of the L3–S1 disc levels was requested and reported as being normal apart from a small right paracentral L5–S1 disc bulge slightly deforming the thecal sac anteriorly and slightly compromising the proximal descending right S1 nerve root.

DIAGNOSIS

- Musculoligamentous soft tissue injuries of the lumbar spine.
- L5–S1 posterior right paracentral disc bulge slightly compressing the right S1 nerve root.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Once the CT imaging was explained to the patient, and conservative treatment options of acupuncture or spinal mobilization were suggested, she opted for a trial of three gentle mobilizations of the lumbar spine as non-steroidal anti-inflammatory drugs and analgesics had not helped her. Following one mobilization, she said that she experienced considerable relief, so no further treatment was undertaken at that time and she was advised to return should her residual symptoms trouble her.

She was seen 6 months later for a recurrence of low back pain with some radiation to the left calf as a result of lifting a light item. On clinical examination the findings were similar to the original examination findings. apart from left straight leg raising that now elicited low back pain at approximately 60° elevation of the leg; left lateral bending was now recorded as being painful in addition to flexion and extension active ranges of movement. Once again, gentle mobilization of the lumbosacral area provided her with significant relief.

Nine months later she presented with acute left leg pain extending from the lumbosacral area to the back of the left leg and into her big toe. She said she had been well since her last visit until she had lifted a reasonably heavy object at home. This had aggravated her low back pain and caused left sided sciatica. She had consulted a 'closer' chiropractor who had taken a history and performed a physical examination. He did not have access to her previous imaging. However, as he understood from her that '*manipulation*' had previously been beneficial (whereas the patient had previously received only *mobilization*), he attempted to help her by performing low back manipulation on 3 consecutive days. Unfortunately, this aggravated her low back pain and left sciatica, causing nausea.

NEW PRESENTING COMPLAINT

Coughing caused a significant increase in low back pain and left sciatica extending to the mid-calf. Bearing down at the toilet produced similar symptoms as did sitting. Lumbar spine active ranges of movement were as follows:

1. Flexion – her fingers reached to halfway down her thighs, being limited due to low back pain and left sciatica.
2. Extension – was limited by approximately 50% due to low back pain and left sciatica.
3. Left lateral bending – her fingers reached to approximately 5 cm above her knee.
4. Right lateral bending – her fingers reached to approximately 5 cm above her knee.
5. Right rotation – was of full range and painless, apart from some transient low back pain on returning to the neutral position.
6. Left rotation – was of full range but elicited low back pain and left leg pain.

The slump test aggravated her low back pain and left sciatica; an attempt to add left straight leg raising elicited severe low back pain and left sciatica at only approximately 15° elevation of the left leg. Deep reflexes in the right lower limb were normal but in the left lower limb

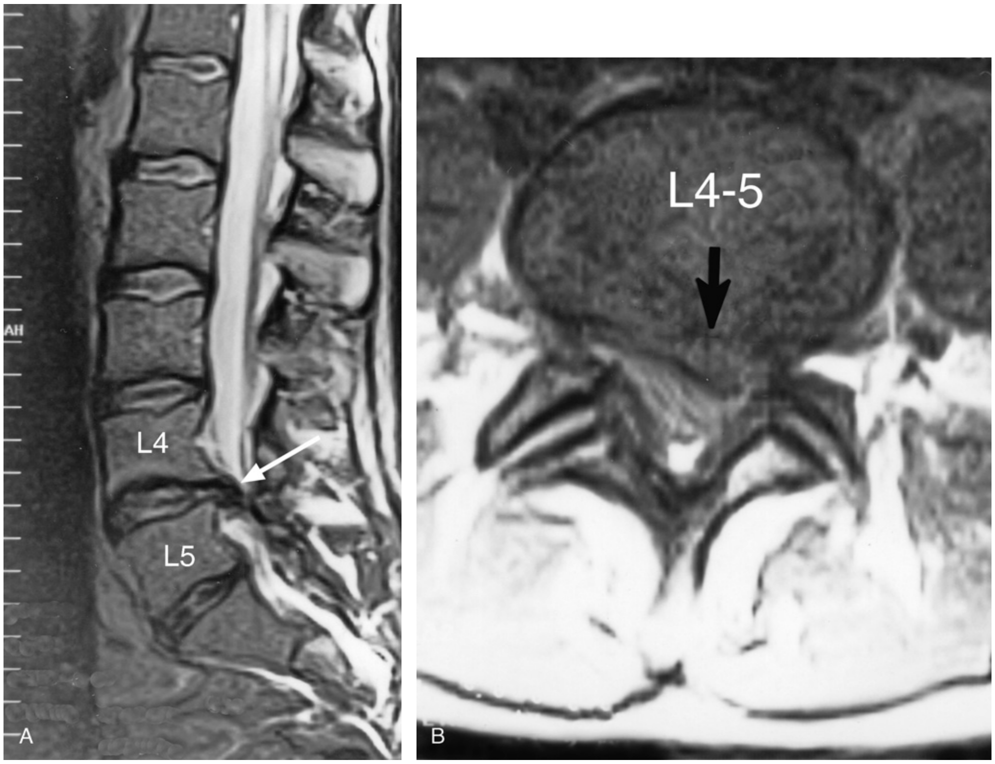

Figure 31.2 (A) Lumbar spine MRI slightly left parasagittal T2-weighted image. Note the moderate sized L4–5 intervertebral disc protrusion posteriorly (arrow). At L5–S1 level there is only a minimal posterior intervertebral disc bulge posteriorly. (B) MRI axial T2-weighted image at the L4–5 level. Note the L4–5 level moderate sized central to left paracentral disc protrusion impinging upon the left L5 nerve root and intervertebral foramen.

the Achilles reflex was reduced to plus one. Supine right straight leg raising was slightly restricted due to hamstring tightness; left straight leg raising was limited to 10° due to acute low back pain and left sciatica. Power in the left big toe was 1/5. Pinprick sensation of the lower extremities was normal except along the lateral side of the left calf (L5) where hypoaesthesia was noted.

WHAT ACTIONS SHOULD BE TAKEN?

A lumbar spine MRI was requested and reported: 'A moderate sized central to left paracentral L4–5 disc protrusion, with a small fragment of disc extending inferiorly into the left lateral recess behind the upper body of L5 (Fig. 31.2A and B). It is likely to impinge the left L5 nerve root at this level. There is a minimal bulge of the L5–S1 intervertebral disc'.

DIAGNOSIS

L4–5 posterior central to left paracentral disc protrusion impinging upon the left L5 nerve root.

TREATMENT AND RESULTS

She was advised not to lift even light items from below waist level and to walk as much as possible in order to exercise her low back. In addition, she was referred to a

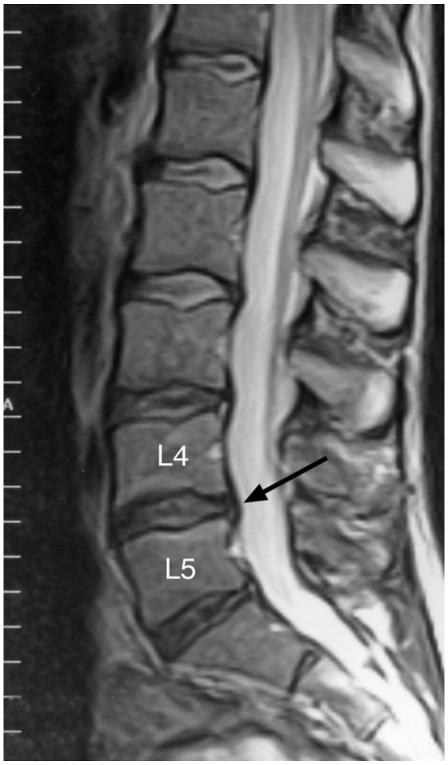

Figure 31.3 Lumbar spine MRI slightly left parasagittal T2-weighted image. Note the L4–5 level residual posterior annular bulge (arrow).

neurosurgeon in view of her acute low back pain and left sciatica. A left sided L4–5 microdiscectomy was performed for the L4–5 intervertebral disc protrusion. However, she experienced a recurrence of left sided sciatica a couple of months later. Therefore, a repeat MRI was requested and it showed a 'residual L4–5 level annular bulge (Fig. 31.3), more prominent to the left of the midline with effacement of the epidural fat but without any enhancing fibrotic tissue'.

She had an epidural steroid injection that reduced her symptoms to intermittent episodes of left sided sciatica that were less severe than before the microsurgery. She continued to have reduced left straight leg raising (to 30°) and intermittent paraesthesia in the left L5 distribution. In order to minimize her symptoms, she cut down the number of hours that she worked and this gave considerable relief and enabled her to cope with her residual intermittent symptoms.

KEY POINT

Be wary of patients who present without imaging and who inadvertently confuse previously successful spinal 'mobilization' with spinal 'manipulation'.

Case 32

Cauda equina syndrome

COMMENT

Cauda equina syndrome is a condition that demands prompt surgical intervention.

PROFILE

A 48-year-old male sedentary worker of solid build who normally performs light duties. He does not smoke and only drinks alcohol occasionally.

PAST HISTORY

He had previously experienced minor intermittent low back pain, without radiation, for approximately 6 years for which he had periodically undergone successful chiropractic manipulation and physiotherapy, heat and exercises. There was no other past medical history of significance.

PRESENTING COMPLAINT(S) (Fig. 32.1)

He presented with central low back pain of approximately 6 weeks duration that radiated to the left buttock, left calf and ankle. His symptoms were worse in the morning but, on getting up and moving around, they lessened somewhat. The symptoms had become acute 2 days before, when he had sneezed.

AETIOLOGY

The original cause was due to bending forwards to pick up a relatively light object.

EXAMINATION

In the erect posture, there was no pelvic obliquity and straining the sacroiliac joints did not elicit any sacroiliac joint pain but elicited some pain at the lumbosacral

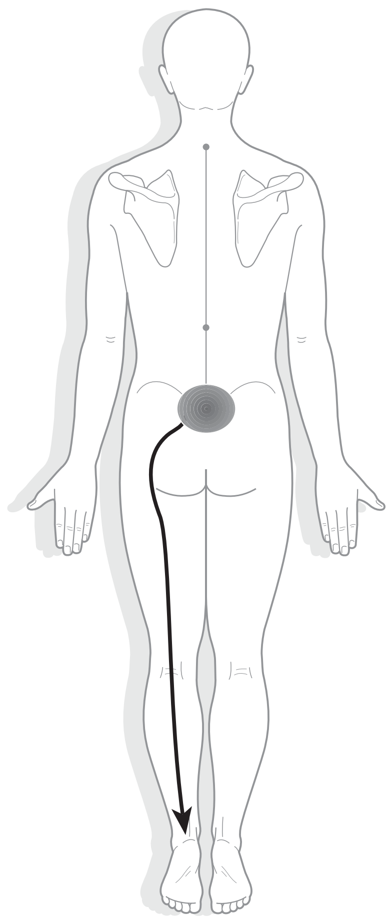

Figure 32.1

area. Active lumbar spine ranges of movement were as follows:

1. Flexion – very limited due to an increase in low back pain.
2. Extension – full range and painless.
3. Left and right lateral bending – both movements elicited an increase in pain on the left of the L5–S1 level.
4. Right rotation elicited a significant increase in low back pain but left rotation caused only a slight 'pulling' sensation on the left of the lumbosacral level.

The deep tendon reflexes in the lower extremities were normal as was the plantar response. Pinprick sensation elicited hypoaesthesia on the lateral aspect of the left calf (L5). Sitting slumped forward elicited an increase in low back pain; addition of left SLR to a measured 15° elicited a significant increase in low back pain that radiated to the left thigh posteriorly. Supine SLR was limited to 20° (left) and 75° (right) due to low back pain. Bilateral hip flexion elicited slight low back pain. Vibration sensation at the ankles was normal.

IMAGING REVIEW

No previous imaging was available.

CLINICAL IMPRESSION

L4 or L5 disc protrusion.

WHAT ACTION SHOULD BE TAKEN?

He was referred for plain film radiographs and the report stated 'no obvious significant disc space narrowing'. However, he was referred for a CT scan of the L3, L4 and L5 discs and was given a non-steroidal anti-inflammatory drug. The CT scan showed a central to left sided soft disc prolapse at L4–5 impinging upon the anterior part of the thecal sac.

DIAGNOSIS

Cauda equina syndrome due to a broad-based L4–5 disc protrusion more prominent on the left, causing moderately severe canal and lateral recess stenosis.

TREATMENT AND RESULTS

He underwent a trial of acupuncture treatment and was advised to walk but not to lift anything or twist his spine. He was also advised to sit down to dry his feet after showering and to rest periodically if the symptoms became aggravated.

Ten days later he noticed a significant improvement. However, prolonged sitting aggravated the left sciatica. On returning to work his symptoms were aggravated and he experienced a recurrence of sciatica extending to the left ankle. Flexion-distraction chiropractic treatment (3×20 second distraction) was tried and this provided some relief. The combination of anti-inflammatory medication, acupuncture and flexion-distraction treatment enabled him to perform some normal activities such as being able to personally remove his socks. Lumbar spine ranges of movement began to increase and he said he was sleeping well. Approximately 2 months after treatment was commenced he was carrying some items when he suddenly twisted his back and caused a severe exacerbation of low back pain with left buttock pain, sciatica and paraesthesiae in the left foot; he also had left leg cramps, so he returned for a further consultation. Examination showed that he now walked with difficulty, had a loss of pinprick sensation in the S1 dermatome of the left foot and a decrease in pinprick sensation in the anal region as well as a decrease of anal sphincter tone.

He was immediately referred for an emergency surgical opinion with a diagnosis of cauda equina syndrome because this condition demands prompt surgical intervention (Henriques et al 2001).

An MRI investigation (Fig. 32.2) was not performed until 7 hours after presentation with cauda equina syndrome and this showed 'L4–5 canal stenosis due to a broad-based disc protrusion, more prominent on the left, causing a moderately severe canal and lateral recess stenosis at the level of the disc with a developmentally small canal'.

He then noted that he could not move the toes on his left foot, although he could move the entire foot, and he felt numbness from the low back to his toes. He was catheterized as he now had urinary incontinence.

Approximately 23 hours following the diagnosis of cauda equina syndrome he underwent a laminectomy for the removal of a single large sequestration of nucleus pulposus at the L4–5 level.

Unfortunately, he had a very difficult few months with weakness of the left foot and having to use self-catheterization; he was left with impaired left lower limb power and sensory changes. Although bowel and bladder function are now normal some months post surgery, he has been left with left lower limb weakness, atrophy of some muscles and some neurological dysfunction.

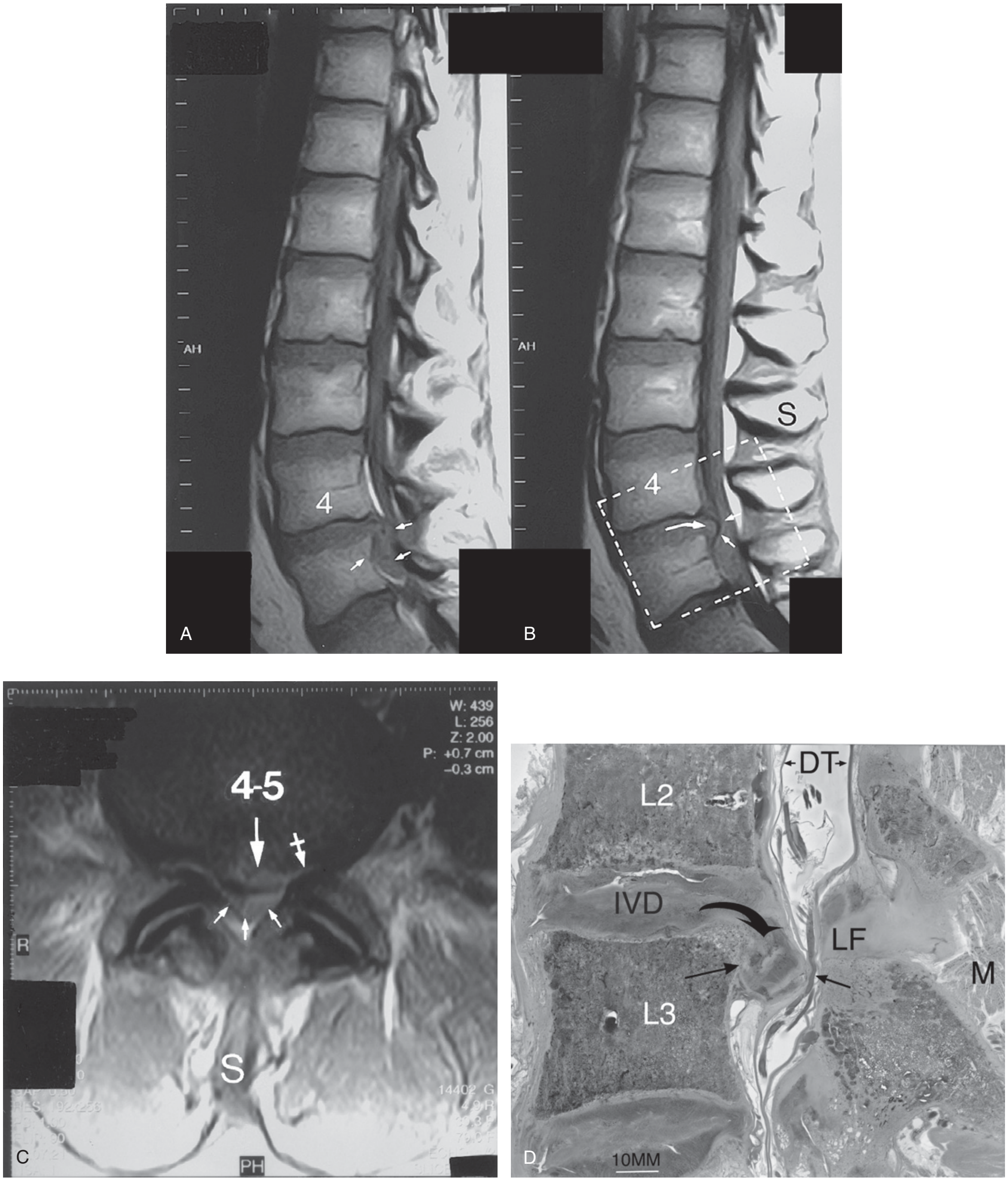

Figure 32.2 (A) MRI sagittal T1-weighted image showing inferior extension of the large L4–5 disc protrusion (arrows) causing moderately severe canal stenosis. (B) MRI sagittal T1-weighted image showing the large disc protrusion at the L4–5 level (arrow) that compresses the cauda equina posteriorly (small white arrows). S = spinous process. The rectangle shows the orientation for the histopathology section described in (D). (C) MRI axial T1-weighted image showing the large L4–5 level broad based central to left-sided disc protrusion (large white arrow) producing moderately severe spinal canal and left lateral recess stenosis (tailed arrow). The small arrows show the considerable compression of the dural tube containing the cauda equina nerve roots. S = spinous process; R = right side of patient. A large central to lateral left sided disc protrusion can be represented as shown in the postmortem histological preparation (D). (D) A 200-micron thick histopathology parasagittal section from a 62-year-old male cadaveric spine. The large open arrow shows protruded disc material compressing the dural tube (DT) and a nerve root (small white arrow) between the protrusion and the ligamentum flavum (LF) posteriorly. As the section is 200 microns thick (1/5th of a millimetre thick) only one nerve root is seen being compressed. However, in a broad-based disc protrusion a number of nerve roots would be compressed, thus causing the cauda equina syndrome. IVD = intervertebral disc. M = muscles. (Ehrlich's haematoxylin and light green counterstain.) For orientation purposes, this parasagittal plane histological section can be approximately equated to the rectangle in (B). See also colour plate section Fig. vii.1B.

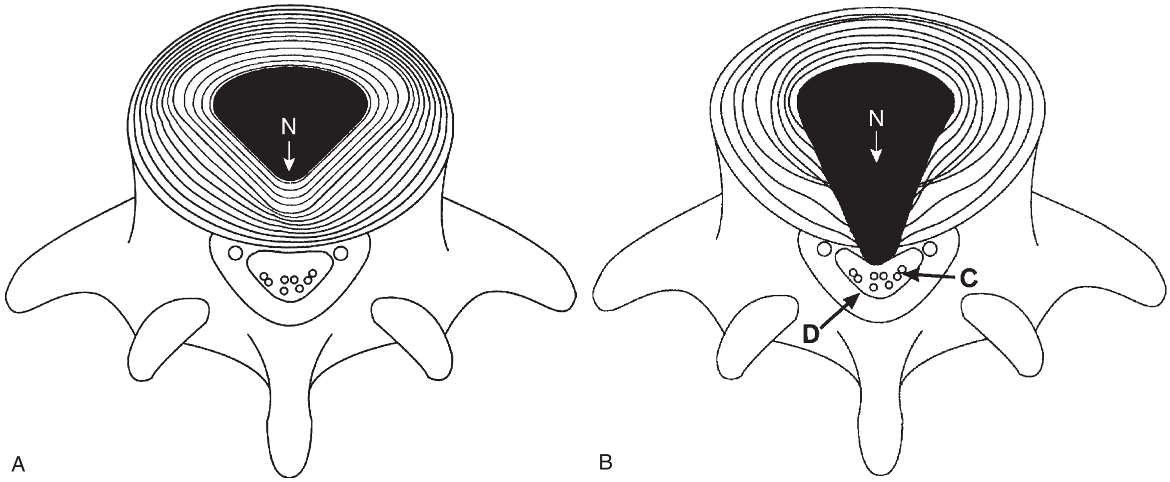

Figure 32.3 A schematic diagram showing: (A) The nucleus pulposus (N) is bulging into the approximately 90 layers of annular fibres located at the posterior of the disc; (B) the nucleus pulposus (N) has now ruptured through the annular fibres and is pressing upon the dural tube (D) containing the cauda equina (C) nerve roots which become compressed.

KEY POINTS

1. A patient with cauda equina syndrome requires very urgent surgical intervention to decompress the cauda equina otherwise serious complications will occur.
2. Figure 32.3A schematically represents how the nucleus pulposus may initially cause posterior bulging of the intervertebral disc then proceed to nuclear prolapse (Fig. 32.3B).

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Case 33

Perineural fibrosis

COMMENT

When spinal surgery is unsuccessful, thoroughly reinvestigate the patient.

PROFILE

A 45-year-old muscular male manual worker who does not smoke and only drinks alcohol socially.

PAST HISTORY

A 12-year history of low back pain which began while lifting heavy objects at work. Following this injury, left sided sciatica had become so severe that he had to undergo a microdiscectomy at the L5–S1 level 2 years prior to this consultation; surgery temporarily relieved the low back pain and sciatica enabling him to walk better for several months.

PRESENTING COMPLAINT(S) (Fig. 33.1)

Constant left sided low back pain and sciatica, due to lifting heavy objects, which recurred after the microdiscectomy. He is unable to sit or stand for any length of time, lift more than approximately 5 kg weights, carry out any gardening (apart from simple hosing) and drive his car without low back pain and left sciatica extending to his calf. He has chronic sleep disturbance due to severe nocturnal back pain that does not occur at any particular time. The left leg feels 'numb and weak' and he has a 'sharp, deep and stabbing pain' across the low back. Coughing and sneezing aggravate the low back pain.

Physiotherapy has not been helpful and he has stopped taking non-steroidal anti-inflammatory drugs and analgesics as they were not helpful.

He has not been able to work since the discectomy operation 2 years before this consultation.

AETIOLOGY

Carrying heavy objects at work.

EXAMINATION

SLR was limited by low back pain to approximately 65° (right) and 20° (left). There was hypoaesthesia over the posterolateral aspect of the left leg and over the dorsum

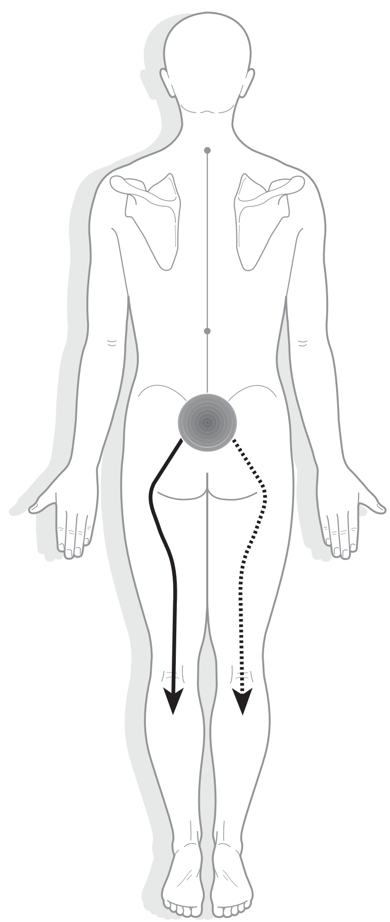

Figure 33.1

of the left foot. The left ankle jerk was absent and the plantar responses were normal. All lumbar spine active ranges of movement were very limited and painful and there was bilateral tenderness on deep palpation over the paraspinal muscles in the lower lumbosacral spine at approximately the L5–S1 level. There was L5 root motor weakness. The abdomen was normal on examination.

CLINICAL IMPRESSION

L5–S1 left-sided recurrent disc or perineural fibrosis.

IMAGING REVIEW

No post-surgical imaging had been performed to re-evaluate his condition.

WHAT ACTION SHOULD BE TAKEN?

A lumbar MRI was performed to see what his lumbosacral spine looked like following surgery. The sagittal views showed disc space narrowing at the L5–S1 level (Fig. 33.2A). In addition there was a generalized annular bulge of the L5–S1 disc. The axial views (Fig. 33.2B) showed a soft-tissue structure in the left posterolateral aspect of the spinal canal adjacent to the left S1 nerve root which showed no evidence of enhancement with intravenous contrast. This was considered to be post-operative inflammatory tissue and the lack of enhancement was considered to be due to some problem with the contrast injection. Soft tissue was seen anterior to and investing the left nerve root and was considered to be disc material (see Fig. 33.3).

DIAGNOSIS

Post-surgical perineural fibrosis on the left side at the L5–S1 level.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was referred for a further surgical opinion and subsequently underwent a laminectomy at

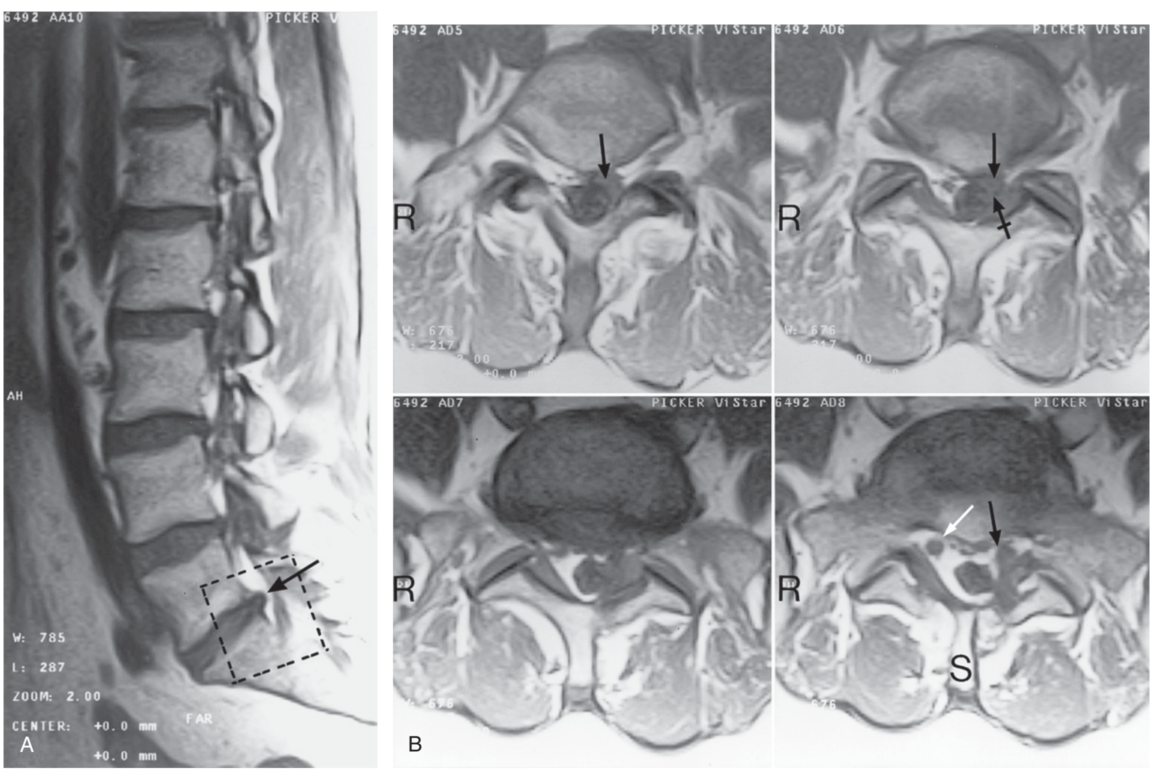

Figure 33.2 (A) MRI parasagittal left-sided T1-weighted image of the lumbosacral spine showing the generalized annular bulge of the L5–S1 disc (arrow). The area within the broken lines is represented histologically in Fig. 33.3. (B) MRI axial T1-weighted images (AD5 to AD8) of the L5–S1 disc. These show the soft tissue in the left lateral aspect of the spinal canal (top left axial image AD5 see arrow) around the left S1 nerve root; this has the configuration of postoperative inflammatory tissue. This soft tissue is also seen anterior to and investing the left nerve root in the top right axial image (tailed arrow). The bottom right axial image shows a normal S1 nerve root on the right side (white arrow), and the swollen S1 nerve root on the left side (black arrow) surrounded by perineural fibrosis. R = right side of patient; S = spinous process.

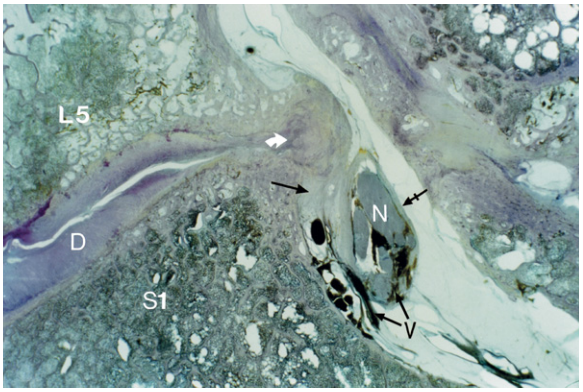

Figure 33.3 A 200-micron thick histopathology section, cut in the parasagittal plane through the lumbosacral intervertebral disc of a 59-year-old cadaver, showing a disc protrusion (small curved white arrow) with perineural adhesions (arrow) between the protrusion and the adjacent dural sleeve (tailed arrow) containing neural structures (N). D = intervertebral disc; L5 = fifth lumbar vertebral body; S1 = first sacral segment. Note the extensive vascularity (V) posterior to the sacrum and within and around the neural structures. This histological section represents a similar area to the area shown within the broken lines on Fig. 33.2A. (Ehrlich's haematoxylin and light green counterstain.) See also colour plate section Fig. vii.6.

the L5–S1 level. Further inferior L5 left lamina and part of the superior lamina of S1 were removed to display the theca and ligamentum flavum. No free disc fragments were found but there was a 'grossly swollen' left S1 nerve root that was fully mobilized and the lateral recess was decompressed.

Unfortunately, the patient continued to suffer from disabling low back pain and left sided sciatica, so an L5 rhizolysis was performed but without success so, 18 months later, a spinal cord stimulator was inserted with variable results.

KEY POINTS

1. Post-surgical complaints need to be fully investigated to look for recurrent disc material and perineural fibrosis affecting the adjacent nerve root.
2. According to Loupasis et al (1999), the long-term results of standard lumbar discectomy are not very satisfying; microdiscectomy results can be disappointing too.

Reference

Loupasis G A, Stamos D, Katonis P G et al 1999 Seven-to-20-year outcome of lumbar discectomy. Spine 15: 2313–2317.

Further reading

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Fan Y F, Chong V F, Tan S K 1995 Failed back surgery syndrome: differentiating epidural fibrosis and recurrent disc prolapse with Gd-DTPA enhanced MRI. Singapore Medical Journal 36: 153–156.

Spencer D L 1999 The anatomical basis of sciatica secondary to herniated lumbar disc: A review. Neurol Res 21 (Suppl 1): S33–S36.

Vishteh A G, Dickman C A 2001 Anterior lumbar microdiscectomy and interbody fusion for the treatment of recurrent disc herniation. Neurosurgery 48: 334–337.

Case 34

Intervertebral disc dysfunction

COMMENT

Erect posture functional imaging of the lumbar spine can be very useful.

PROFILE

A 39-year-old married male who does not drink alcohol or smoke cigarettes and who worked as a patient carer.

PAST HISTORY

Suffers from high blood pressure and oesophageal ulcers for which he takes medication.

Eighteen months ago he was helping a large disabled man from a car into a wheelchair; the disabled man suddenly slipped and this strained the carer's low back, resulting in immediate and acute low back pain as he took the weight of the patient. While sitting in the car after the incident, he momentarily felt a 'sharp jab like an electric shock' in the low back. The pain radiated from the lower back to both testicles.

PRESENTING COMPLAINT(S) (Fig. 34.1)

Low back pain at the L4–5 level, which sometimes radiates to his right buttock and into the posterior aspect of his right thigh.

The low back pain improves with lying down but he has to be cautious as he moves about because he can suddenly get 'twinges of pain'. If he sits on the floor to play with his children, he has to gradually get up and slowly straighten his low back as it becomes very stiff. Sometimes he can bend forwards without pain but bending backwards is always painful.

The only medication that he takes is an analgesic as he is wary of taking medication.

He had to resign from work as he was unable to manage with the physical demands of his work due to his chronic low back pain. He had been told by orthopaedic consultants that there was nothing wrong with his back and that he should return to his carer work and one orthopaedic surgeon stated that the patient should be placed under the surveillance of a Field Officer. Fortunately, one orthopaedic surgeon agreed that he could have internal disc disruption, as did a specialist in rehabilitation medicine.

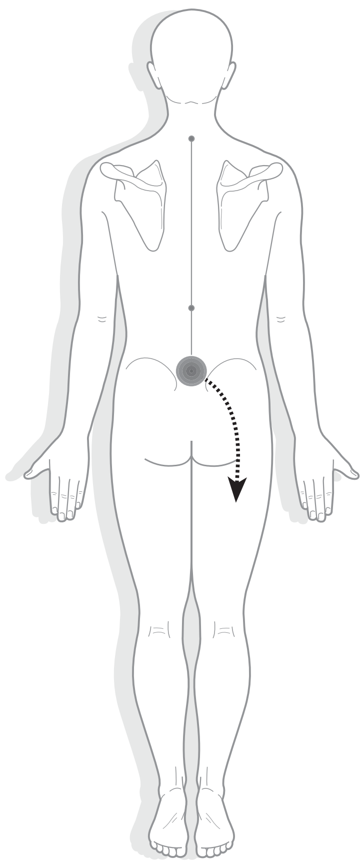

Figure 34.1

Unfortunately, anti-inflammatory medication, physiotherapy treatment, facet block injections, medical acupuncture and a paravertebral muscle injection of anaesthetic and cortisone did not provide long-lasting relief. He had had manipulation on two occasions but this apparently aggravated the low back pain. He had tried swimming but it did not help, so he was currently performing hydrotherapy exercises with some benefit.

AETIOLOGY

Helping a disabled man who slipped while being helped from a car into a wheelchair.

EXAMINATION

The knee and ankle jerk deep tendon reflexes were normal, as was the case with pinprick sensation of the lower extremities. His thigh circumference was 50 cm bilaterally, 18 cm above the patella. In the erect posture, there was no evidence of pelvic obliquity on palpation of the posterior superior iliac spines.

Active lumbar spine ranges of movement were as follows:

1. Flexion – he was able to bend forwards to within 2 cm of the floor but he was very cautious as he flexed his spine due to pain at the L4–5 level and also as he unrolled, i.e. extended his spine to the normal erect posture.
2. Extension was limited by approximately 70% due to pain at the L4–5 level.
3. Left and right lateral bending were painless.
4. Right rotation was painless.
5. Left rotation was limited by about 15% due to L4–5 pain.

Palpation over the L4 and L5 spinous processes and the interspinous ligament elicited localized pain. Deep palpation of the lumbar paraspinal muscles elicited pain bilaterally from L4 to S1. He was able to toe and heel walk without difficulty. He had normal power in the lower extremities. Upon getting onto the examination table he did so cautiously. In the supine position, SLR was limited to approximately 75° (right) and 80° (left) due to 'tightness' of the hamstring muscles. Bilateral hip flexion elicited considerable low back pain at approximately 80° elevation of the thighs from the examination table. Cervical spine flexion elicited only very slight low back pain and compression of the jugular veins while he coughed (Naffziger test) did not reproduce the low back pain. The pulses in the feet were normal. Hoover's sign for malingering was normal, i.e. it did not indicate any malingering. He was hardly able to raise both his legs simultaneously (Milgram's test) due to an increase in low back pain. In the seated position, the slump test did not aggravate his low back pain. The abdomen was normal on examination.

IMAGING REVIEW

An MRI scan taken approximately 12 months prior to his present consultation reported the following: 'There is no evidence of disc herniation or nerve root compression. Normal marrow signals are observed. The conus ends at L1 and is of normal size and configuration. No evidence of spinal stenosis'.

CLINICAL IMPRESSION

Soft tissue injury at L4–5 level – probably internal disc disruption.

WHAT ACTION SHOULD BE TAKEN?

Because the patient was considered to be entirely genuine in his low back pain complaint, in spite of an apparently normal MRI examination, lumbosacral erect posture anteroposterior and left and right lateral bending functional radiographs were taken. A plumb line was used as a reference point for measuring the angle between the inferior endplate of the L4 vertebra and the plumb line in order to determine whether he had L4–5 joint dysfunction. In the neutral erect posture anteroposterior view there was minor wedging of the L4–5 intervertebral disc that was narrower on the left and wider on the right (Fig. 34.2A).

Although the superior endplate of the L5 vertebral body remained essentially at an angle of 90° to the vertical plumb line in the erect posture neutral position, it can be seen that on left and right lateral bending there is a significant difference between the angle made by the inferior endplate of L4 and the plumb line, as shown by the broken line drawn along the inferior endplate of the L4 vertebral body (Fig. 34.2A, B and C).

In left lateral bending (Fig. 34.2B) the intervertebral joints at the L4–5 level allow bending in conformity with the normal lateral bending posture, i.e. the disc spaces become narrower on the left and wider on the right. However, on right lateral bending, the L4–5 joint refuses to allow normal movement between the vertebral bodies L4 and L5, preventing normal wedging of the disc space. This finding indicates that he has a spinal functional problem at L4–5.

Because this man had a claim for injuries at work, the question arose of whether he should have a discogram performed to confirm that he had internal disc disruption. However, the risks were explained to him and he did not wish to take the risk. He went to court to settle his injury claim based on the non-invasive functional plain X-ray films; the court found in his favour in view of the abnormal functioning of the L4–5 intervertebral disc, as shown by the L4 vertebral body movement with respect to the plumb line.

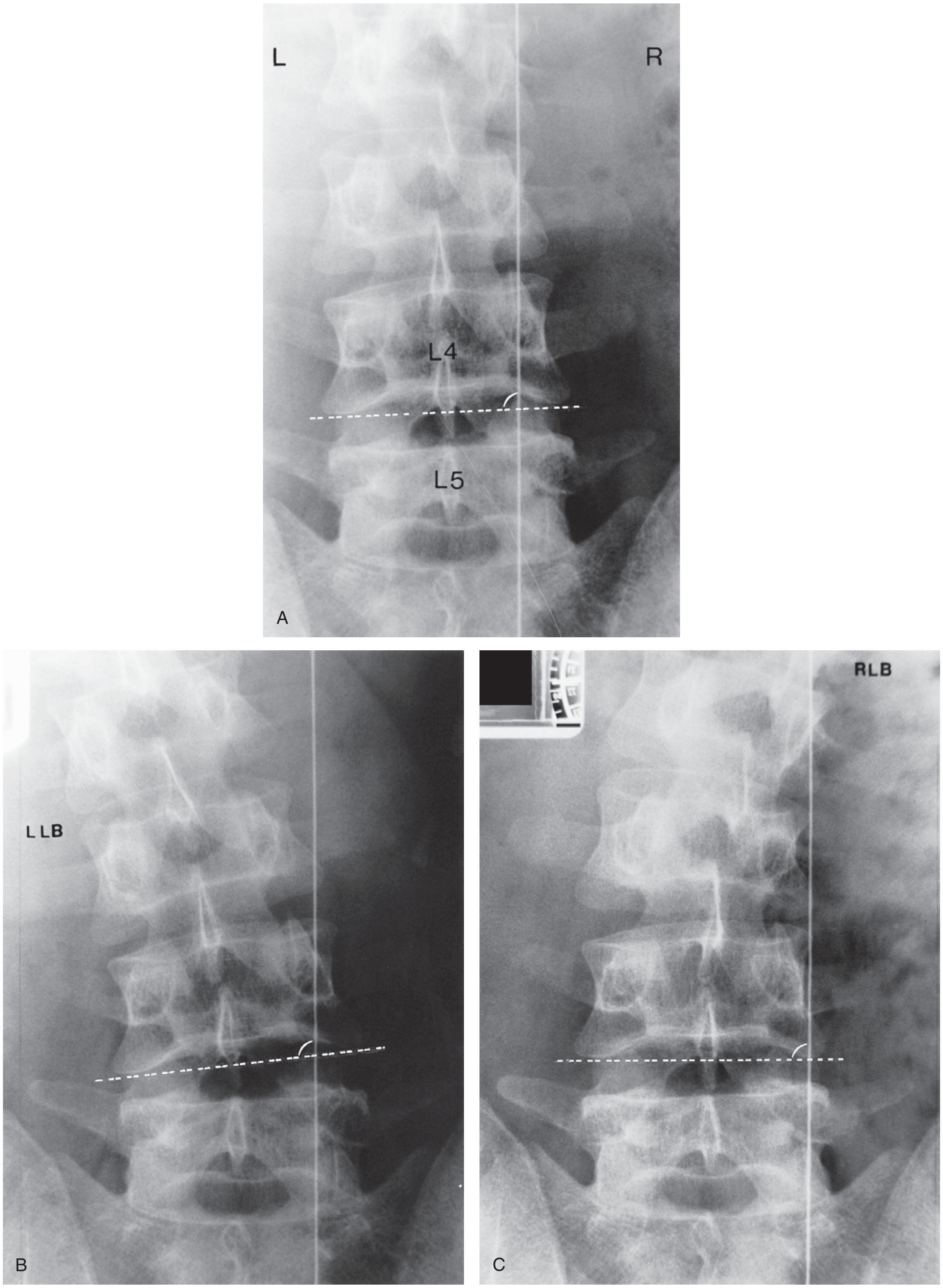

Figure 34.2 (A) Erect posture anteroposterior plain X-ray of the lumbosacral spine (L2–S1). Note the minor wedging of the L4–5 intervertebral disc which is slightly narrower on the left (L) as shown between the broken line across the inferior endplate of L4 and the superior endplate of L5. The angle between the broken line showing the inferior endplate of L4 and the vertical plumb line measures 93°. R = right side of patient. (B) Erect posture left lateral bending (LLB) plain X-ray of the lumbosacral spine showing normal wedging of the L4–5 intervertebral disc (which is narrower on the left) and the discs above, i.e. all disc spaces are narrower on the left side and wider on the right. The angle between the L4 inferior endplate line and the plumb line measures 97°. (C) Erect posture right lateral bending (RLB) plain X-ray of the lumbosacral spine showing the L4–5 intervertebral disc which refuses to wedge normally, i.e. to become narrower on the right and wider on the left; the discs above L4 do wedge normally, i.e. become narrower on the right side and wider on the left. The angle between the endplate line and the plumb line measured 91°, i.e. only approximately 2° less than in the neutral anteroposterior view shown in (A).

DIAGNOSIS

L4–5 intervertebral disc dysfunction/internal disc disruption.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Facet block injections, acupuncture and paraspinal muscle blocks had not given him lasting relief, and anti-inflammatory medication gave him gastric problems. As surgery was not indicated, he was advised not to perform any heavy manual work but to continue with water aerobics and then to swim as frequently as possible. He was also advised to perform back exercises (see exercises presented in Case 1).

In addition, he was advised to undergo retraining so that he could perform light duties only.

The main issues that helped him were that he was taken seriously and that an explanation was provided as to why he had chronic low back pain. In addition, he was grateful that an exercise programme was established for him so that he could control his pain levels, which he did satisfactorily over a period of some months.

KEY POINT

The value of plain film anteroposterior erect posture radiography of the lumbar, including functional left and right lateral bending views, should not be underestimated.

Further reading

Brightbill T C, Pile N, Eichelberger R P, Whitman M 1994 Normal magnetic resonance imaging and abnormal discography in lumbar disc disruption. Spine 19: 1075–1077.

Ohnmeiss D D, Vanharanta H, Ekholm J 1997 Degree of disc disruption and lower extremity pain. Spine 22: 1600–1605.

Schwarzer A C, Aprill C N, Derby R et al 1995 The prevalence and clinical features of internal disc disruption in patients with chronic low back pain. Spine 20: 1878–1883.

Case 35

Discitis and osteomyelitis

COMMENT

This case highlights complications that may occur in intravenous drug users.

PROFILE

An 18-year-old thin, tall unemployed male.

PAST HISTORY

A history of intravenous drug use. No history of trauma.

PRESENTING COMPLAINT(S) (Fig. 35.1)

A recent feeling of being generally unwell and feverish with various spinal aches and pains and hip joint pains. He was immediately hospitalized and seen by a specialist physician.

AETIOLOGY

Intravenous drug addiction.

CLINICAL IMPRESSION

Possibly septicaemia/osteomyelitis.

EXAMINATION AND ACTION TAKEN

A comprehensive diagnostic work-up resulted in a diagnosis of bacterial endocarditis and spinal osteomyelitis secondary to septicaemia. He was found to have positive cultures for the following organisms: methycillin-sensitive *Staphylococcus aureus*, *Staphylococcus epidermidis* and *Bacillus cereus*. His ESR was 130 mm/hour (normal 1–10) and his blood pressure was 90/60.

An initial transthoracic echocardiogram showed some tricuspid regurgitation but no vegetations. Transoesophageal echocardiogram showed vegetations consistent with endocarditis of the tricuspid valve. An initial bone scan showed probable abnormalities in his clavicle, mid-lumbar vertebral region and in the sacroiliac joints.

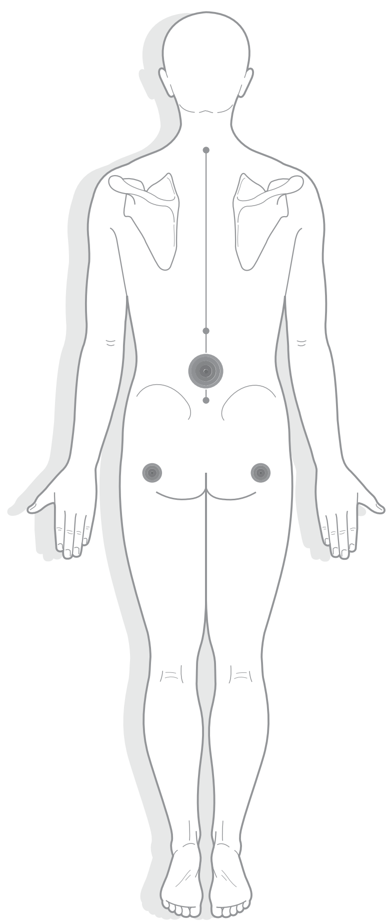

Figure 35.1

He was hospitalized for 8 weeks while undergoing intravenous antibiotic drug therapy. Following 8 weeks of antibiotic treatment a three phase bone scan with SPECT and an echocardiogram were repeated. The echocardiogram showed lesser residual vegetations on the tricuspid valve and the bone scan showed: 'Increased blood pool activity in the left sacroiliac joint and a slight increase in blood pool activity in the right side of the mid-lumbar spine, approximately in the region of the L3–4 body consistent with hyperaemia. SPECT tomographic images have confirmed increased tracer uptake in the left sacroiliac joint and at the L3–4 level of the lumbar spine on the right. Relatively increased tracer uptake in both shoulders is symmetrical and almost certainly represents activity in incompletely fused epiphyses. Increased tracer uptake is noted in the medial aspect of the left clavicle. Tracer distribution elsewhere in the skeleton is otherwise unremarkable. Comments: appearance on bone scan suggests a sacroiliitis, some focus in the medial end of the left clavicle and a further focus of probable infection in the L3–4 region of the lumbar spine (Fig. 35.2A)'.

He was referred for a CT scan that showed lytic lesions in the L3 and L4 lumbar vertebrae where he still had pain. Therefore, although all organisms were reported sensitive to the antibiotic used, this obviously did not eliminate the infection, perhaps because the antibiotic has brief peaks of high serum concentration and then washed out very quickly and the sporing nature of the *Bacillus cereus* allows some organisms to resist the peaks. Antibiotic treatment was changed and he was referred for an orthopaedic opinion regarding his L3–4 discitis secondary to infection.

An MRI scan was performed (Fig. 35.2B) and found: 'There is discitis of the L3–4 disc with destruction of the endplates and associated marrow oedema. There is a small degree of enhancement of this disc posteriorly. No epidural extension seen. The other discs appear normal'.

DIAGNOSIS

L3–4 discitis, spinal osteomyelitis and bacterial endocarditis secondary to septicaemia.

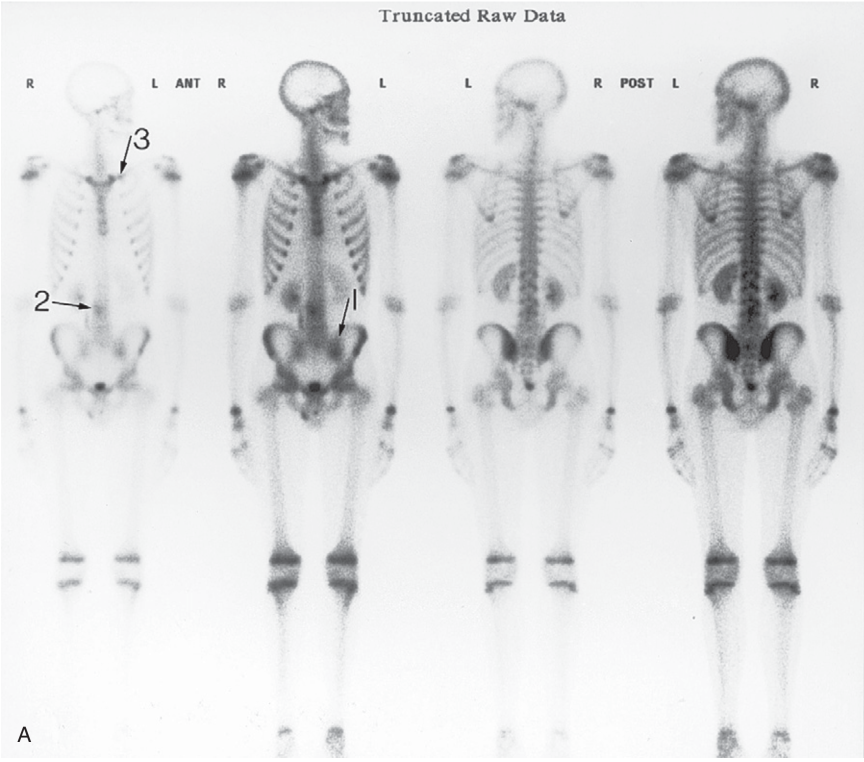

Figure 35.2 (A) The three phase bone scan with SPECT truncated raw data image shows 'activity in the left sacroiliac joint (arrow 1) and a slight increase in activity in the right side of the mid lumbar spine in the region of L3/L4 (arrow 2). Relatively increased tracer uptake in both shoulders is symmetrical and almost certainly represents activity in incompletely fused epiphyses. Increased tracer uptake is noted in the medial aspect of the left clavicle' (arrow 3). The 'hot spot' (arrow 2) at the L3–4 level is where surgery was later performed.

(Continued)

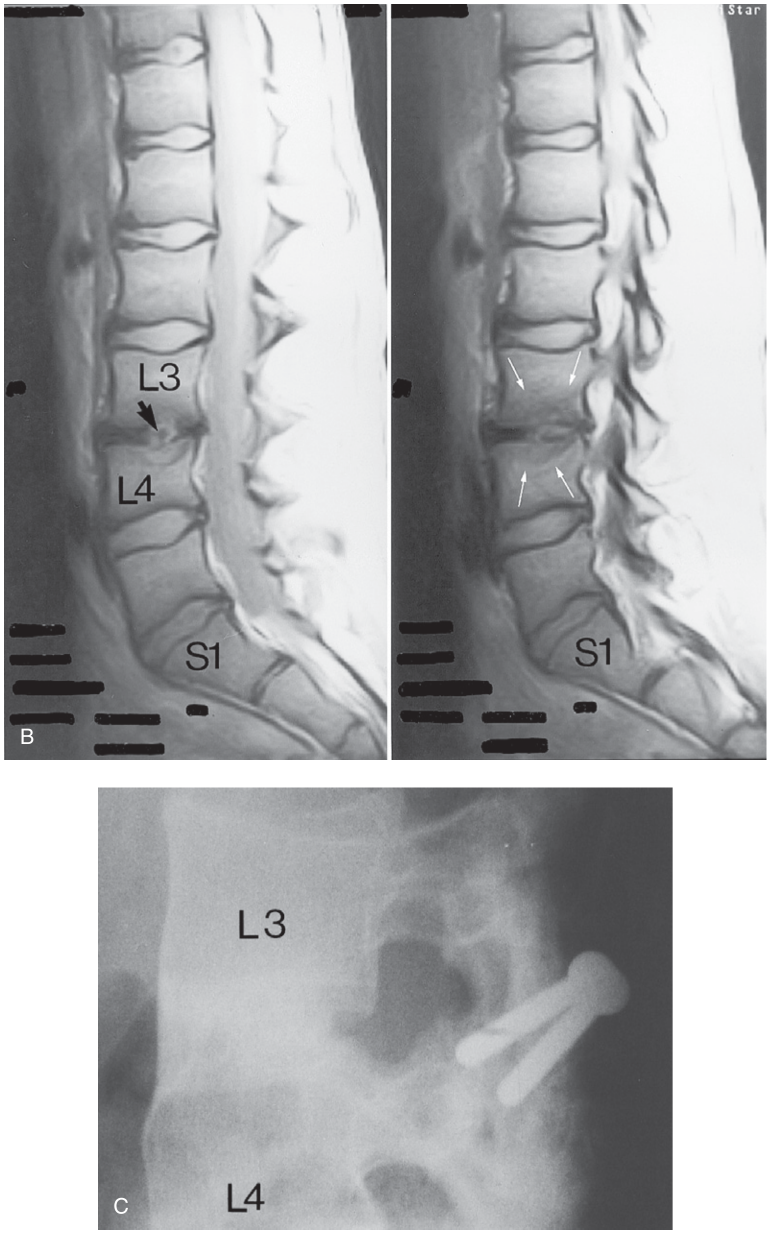

Figure 35.2 Cont'd (B) Lumbar spine MRI parasagittal STIR image showing discitis of the L3–4 disc with destruction of the adjacent vertebral body endplates and associated marrow oedema (arrows). There is a small degree of enhancement of this disc posteriorly (black arrow). The remaining discs appear normal. L3 = third lumbar vertebral body; L4 = fourth lumbar vertebral body; S1 = first sacral segment. (C) A lateral plain X-ray image of the L3 and L4 bodies showing an anterior interbody fusion with posterior fixation (using two screws).

TREATMENT AND RESULTS

With the intention of eradicating the infective discitis, surgery was then performed to remove the L3–4 disc and adjacent endplates, followed by fusion of the levels involved by simple fixation (Fig. 35.2C).

Following the above treatment that involved prompt referral for care by several specialists, this young man recovered in due course. His blood pressure was 110/65, he was afebrile at 36.5°C, there was no evidence of an enlarged pulsatile liver and no swelling of his ankles. Heart sounds indicated a soft pan systolic murmur at the left sternal edge consistent with tricuspid regurgitation. ECG showed normal sinus rhythm and a chest X-ray did not reveal any abnormalities. He was left with a mild to moderate tricuspid regurgitation and he has periodic re-evaluations.

KEY POINT

Intravenous drug users with spinal and other pains may present with life-threatening infections that require immediate referral for specialized medical care.

Further reading

Fortun J, Navas E, Martinez-Beltran J et al 2001 Short-course therapy for right-sided endocarditis due to *Staphylococcus aureus* in drug abusers: cloxacillin versus glycopeptides in combination with gentamicin. Clinical Infectious Diseases 33: 120–125.

Hopkinson N, Stevenson J, Benjamin S 2001 A case ascertainment study of septic discitis: clinical, microbiological and radiological features. Quarterly Journal of Medicine 94: 465–470.

Lopez-Majano V, Miskew D B 1980 Sacro-iliac joint disease in drug abusers: the role of bone scintigraphy. European Journal of Nuclear Medicine 5: 459–463.

Lopez-Majano V, Miskew D, Sansi P 1981 Bone scintigraphy in drug addiction. European Journal of Nuclear Medicine 6: 17–21.

Case 36

L4 and L5 posterior intervertebral disc protrusions

COMMENT

More than one level of posterior intervertebral disc protrusion may occur.

PROFILE

A slim 48-year-old athletic woman with a 20-year history of low back pain who normally runs a total of approximately 15 kilometres per week, swims or cycles daily. She does not smoke cigarettes or drink alcohol.

PAST HISTORY

There is no past history of relevance except for a trampolining fall some 20 years ago that resulted in intermittent low back pain.

PRESENTING COMPLAINT(S) (Fig. 36.1)

One month of worsening and intolerable low back pain with acute pain extending into the left buttock and hip that extends into the posterolateral aspect of the left leg as far as the sole of the foot with various paraesthesiae since recent prolonged sitting. She had 'learned to live with her low back pain' but the left sided sciatica has become intolerable. Coughing and bearing down increase her symptoms.

Non-steroidal anti-inflammatory medication and analgesics have not been of much help. The sciatic pain is partly relieved with standing, it is aggravated by sitting, and some relief is obtained from lying supine.

AETIOLOGY

A long aeroplane journey with associated lifting of suitcases 2 months previously had caused her acute symptoms.

EXAMINATION

Supine straight leg raising was to 90° (right) and 85° (left), the latter causing an increase in low back pain.

There were no abnormal sensory signs. The left ankle jerk was absent but the physical examination was otherwise unremarkable.

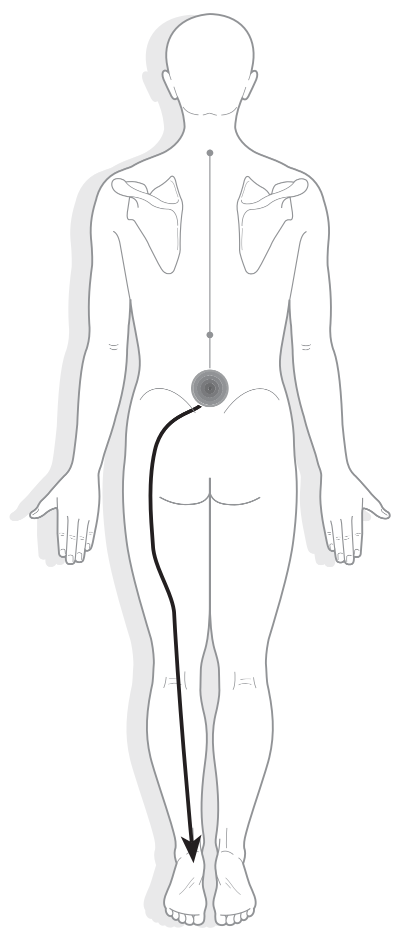

Figure 36.1

IMAGING REVIEW

No imaging had been performed.

CLINICAL IMPRESSION

A possible L4–5 or L5–S1 intervertebral disc protrusion, most likely left sided.

WHAT ACTION SHOULD BE TAKEN?

A lumbar spine plain X-ray examination was requested, including an erect posture pelvis and lumbar spine view. The examination showed narrowing of the L4–5 and L5–S1 intervertebral disc space height with some mild endplate sclerosis and small anterior osteophyte formation at both of these levels. The remaining lumbar disc spaces were normal.

In view of the plain X-ray examination findings and the absent left ankle jerk, a lumbar spine MRI was requested. This showed (i) the posterior right paracentral intervertebral disc protrusions at L4–5 and L5–S1 levels (Fig. 36.2A and B), and (ii) a loss of signal in the L3–4, L4–5 and L5–S1 intervertebral discs in keeping with desiccation at these levels (Fig. 36.2B). A prominent right posterolateral disc protrusion was present at the L4–5 level, and at the L5–S1 level there was a large central and left sided disc protrusion compressing the thecal sac and impinging upon the left S1 nerve root.

DIAGNOSIS

- Musculoligamentous soft tissue injuries of the lumbar spine.
- L5–S1 large central and left sided intervertebral disc protrusion causing left sided radiculopathy.
- L4–5 prominent right posterolateral intervertebral disc protrusion.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was referred for a neurosurgical opinion in view of her acute and intolerable left sided sciatica. She underwent a left sided L5–S1 microdiscectomy that showed the large sequestrated disc material.

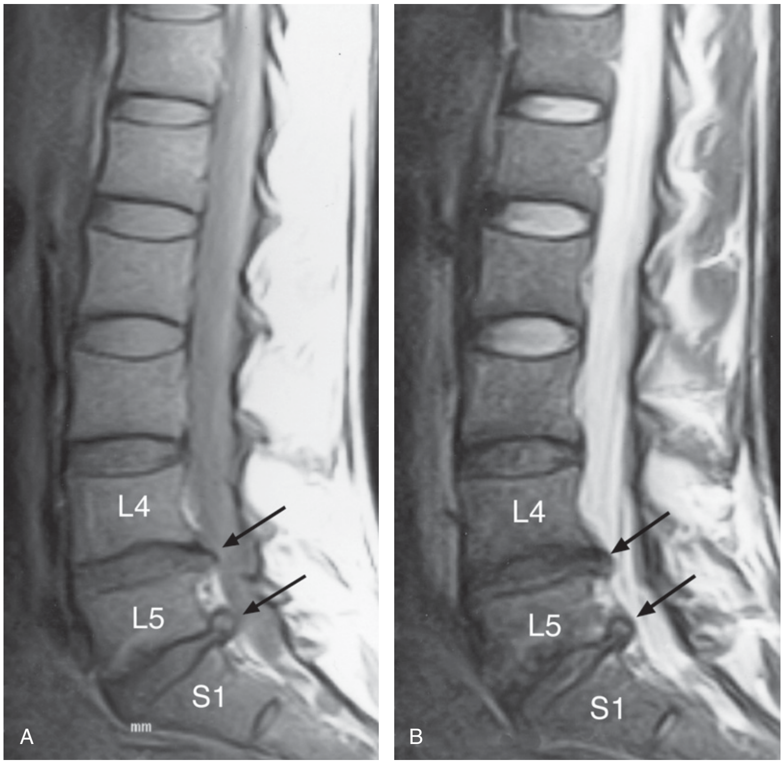

Figure 36.2 (A and B) Lumbar spine MRI parasagittal T1-weighted and T2-weighted images showing the disc protrusions at the L4–5 and especially the L5–S1 levels (black arrows). Note how the disc protrusions indent the anterior pain sensitive surface of the dural tube. The desiccation of the L3-4, L4–5 and L5–S1 intervertebral discs is seen in the T2-weighted view (B).

She had an unremarkable postoperative course and was discharged relatively pain-free on the second postoperative day. She was advised to exercise gently, beginning with walking, then swimming as soon as she felt that she was able to do so. She was advised to give up running.

The above surgery and advice on mobilizing but no longer running resulted in a satisfactory outcome with respect to pain management for her sciatica. She continued to experience intermittent episodes of low back pain but managed to control these with the conservative approach as recommended above.

KEY POINT

A careful diagnostic work-up is necessary to arrive at an appropriate diagnosis.

Case 37

Failed low back surgery

COMMENT

A radiology facility should provide the imaging requsted unless the radiologist contacts the referring clinician to state that there is a contraindication to the requested procedure.

PROFILE

A 36-year-old female secretary of average build who does not smoke cigarettes or drink alcohol.

PAST HISTORY

She experienced intermittent attacks of low back pain during the last 13 years, with increasing severity during the last 6 years following an exercise programme that she undertook in an attempt to help her chronic low back problem. Unfortunately, the exercise programme resulted in sciatica in the left leg. She and her general medical practitioner are concerned as no explanation has been provided by specialists regarding her continuing low back pain and left sciatica, in spite of numerous plain film radiographic investigations and a lumbosacral myelogram.

PRESENTING COMPLAINT(S) (Fig. 37.1)

Unremitting severe low back pain radiating to the posterolateral aspect of the left leg as far as the ankle. Occasionally, she experiences minor radiation into the right thigh posteriorly. Twisting movements such as vacuuming can cause an acute exacerbation of left sciatica. Only when she has an acute attack do coughing and bearing down cause an increase in low back pain and left sciatica. She has tried several non-steroidal anti-inflammatory drugs, analgesics and Valium with no significant relief of her symptoms; she developed gastritis and constipation while taking these medications. She has tried using a Transcutaneous Electrical Nerve Stimulator (TENS) machine, needle acupuncture, spinal manipulation, and massage without any 'real' help.

AETIOLOGY

Unknown for the present severe low back pain syndrome.

EXAMINATION

She did not appear to have any clinical evidence of leg length inequality but, because of her acute symptoms, it

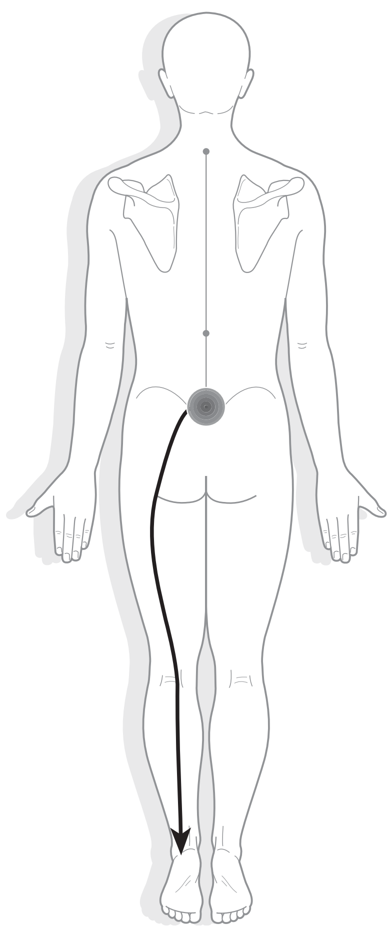

Figure 37.1

was not possible to definitively evaluate any possible pelvic obliquity. There was muscle spasm involving the lumbosacral region and she was antalgic to the right side. All active lumbar spine ranges of movement in the seated position were severely limited by increasing low back pain.

Deep tendon reflexes in the lower extremities were normal. The plantar response was normal. Muscle power and tone were normal. Pin prick and light touch sensations over the left lower limb felt 'patchy' to the patient. Left straight leg raising was limited to a measured 20° due to an increase in low back pain and left sciatica. Right straight leg raising was limited to 40° due to some aggravation of her low back pain. Left straight leg raising coupled with foot dorsiflexion caused an increase in low back pain and left sciatica. Right straight leg raising coupled with foot dorsiflexion did not significantly alter her pain. The Valsalva manoeuvre (bearing down) did not significantly aggravate her low back pain or left sciatica, suggesting that no space-occupying lesion was present.

IMAGING REVIEW

Numerous plain film radiographic examinations had been undertaken but none of the reports mentioned the following:

1. The pre-sacral anomaly on the right side at S1–2, i.e. lumbarization of S1 (Fig. 37.2).
2. The left sided osteophytes bridging across the S1–S2 vestigial intervertebral disc space.

The lumbosacral myelogram had been performed and was reported as '*normal*'.

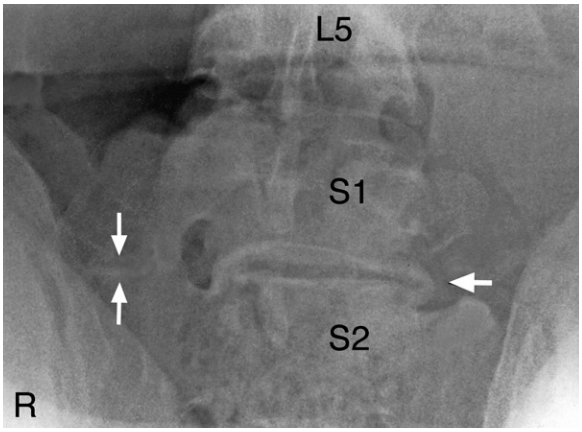

Figure 37.2 Lumbosacral erect posture anteroposterior plain X-ray image. Note (i) the right sided lumbarization of S1, (ii) the partial fusion of the right transverse process of S1 with the sacral ala (white arrows), and (iii) the left sided osteophytes (white arrow) bridging across the S1–2 vestigial intervertebral disc space.

CLINICAL IMPRESSION

Possible spinal nerve compromise due to the left sided osteophytes at the S1–2 level pressing upon the adjacent spinal nerve.

WHAT ACTION SHOULD BE TAKEN?

A lumbar MRI was requested, including coronal views, to better visualize the course of the nerve roots, as well as the spinal nerves passing to the left of the osteophytic spur at the S1–2 level. However, the radiology facility decided it was not necessary to include coronal views!

The MRI report stated: 'There is a transitional lumbosacral vertebra termed S1 for this examination with a vestigial disc at S1–2. At the L5–S1 intervertebral disc level there is a loss of signal in keeping with moderate disc desiccation with a minimal disc bulge (Fig. 37.3). No canal stenosis or nerve root impingement is seen'. However, no mention was made of the osteophytes on the left side at the S1–2 vestigial disc level (Figs 37.4 and 37.5).

Figure 37.3 Lumbar spine MRI sagittal T2-weighted image showing 'the L5–S1 intervertebral disc level loss of signal in keeping with moderate disc desiccation with a minimal disc bulge'.

Figure 37.4 Lumbar spine MRI left parasagittal T1-weighted image including the left side of the lumbosacral spine at the S1–2 level. Note the nerve root (N) in the S1–2 foramen before it passes the osteophytes (black arrow) on the lateral side of the S1–2 level. P = psoas muscle.

Axial images at the S1–2 vestigial disc level show the nerve roots in the lateral recess (Fig. 37.5A and B) and progression of the roots caudad to become spinal nerves, with the left nerve abutting the osteophytes on the left side at the S1–2 level (Fig. 37.5C, D and E).

DIAGNOSIS

Left S1–2 level osteophytes compromising the adjacent spinal nerve.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was advised to see a spinal surgeon for a further opinion regarding the left sided S1–2 osteophytes abutting the adjacent spinal nerve.

As a result, an anterior spinal fusion was performed at the S1–S2 level. Unfortunately, the patient's low back pain and left leg symptoms persisted, so she continued to take analgesic tablets and underwent a course of needle acupuncture treatment; these measures provided only short-term relief.

Approximately 1 year after surgery, a request was made for a follow-up lumbar spine CT scan to include the vestigial disc at the S1–2 level for a re-evaluation in view of her continuing symptoms. The CT scan report stated: 'A spinal fusion is shown at the S1–2 level with two screws in the disc space. The bony spinal canal is adequate throughout with no evidence of disc prolapse or any other encroachment. Nerve roots exit normally without any impingement. The right transverse process of S1 is lumbarized and partly fused with the right sacral ala. Unfortunately, the osteophytes projecting from the right side of the S1–S2 vestigial disc had not been removed.

The proximity of a laterally projecting osteophyte to the adjacent neural structures is shown in a histopathology section from postmortem material in Figure 37.6.

Note

The MRI of the lumbar spine showed that the spinal nerve in question passes close to the osteophytes on the left side of the vestigial disc space just beyond the root sleeve. Therefore, one would not expect a myelogram examination to be abnormal, as contrast medium cannot pass beyond the root sleeve.

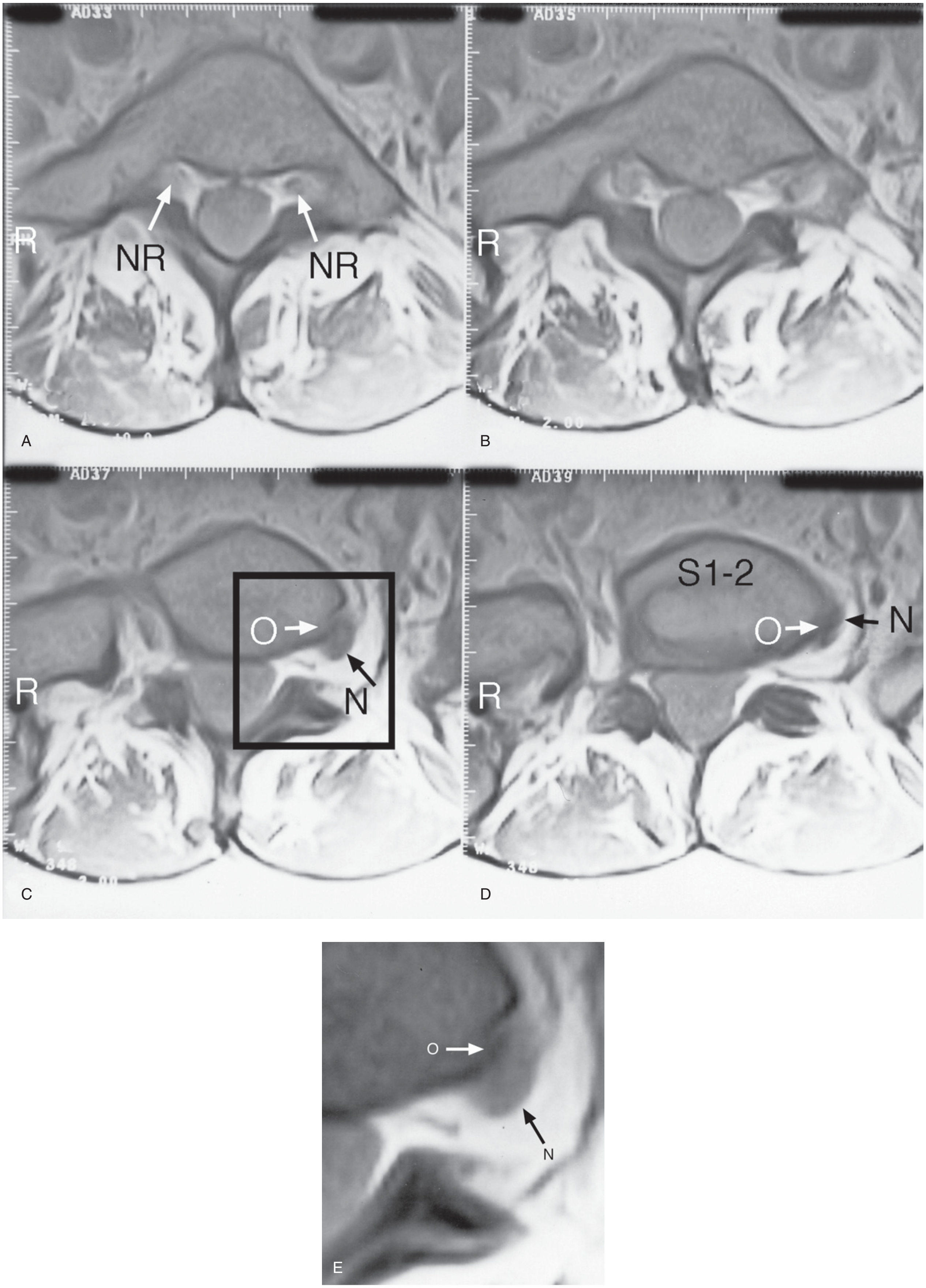

Figure 37.5 (A to E) MRI axial T1-weighted images at the S1–2 vestigial disc level. Note the nerve roots (NR) in the lateral recesses in A and the progression of the roots caudad to become spinal nerves (N), with the left nerve abutting the osteophytes (O) on the left side (C, D and E). The square in C is shown enlarged in E. Compare the MRI osteophyte/spinal nerve relationship with the osteophyte/spinal nerve relationship in the histopathology section (Fig. 37.6).

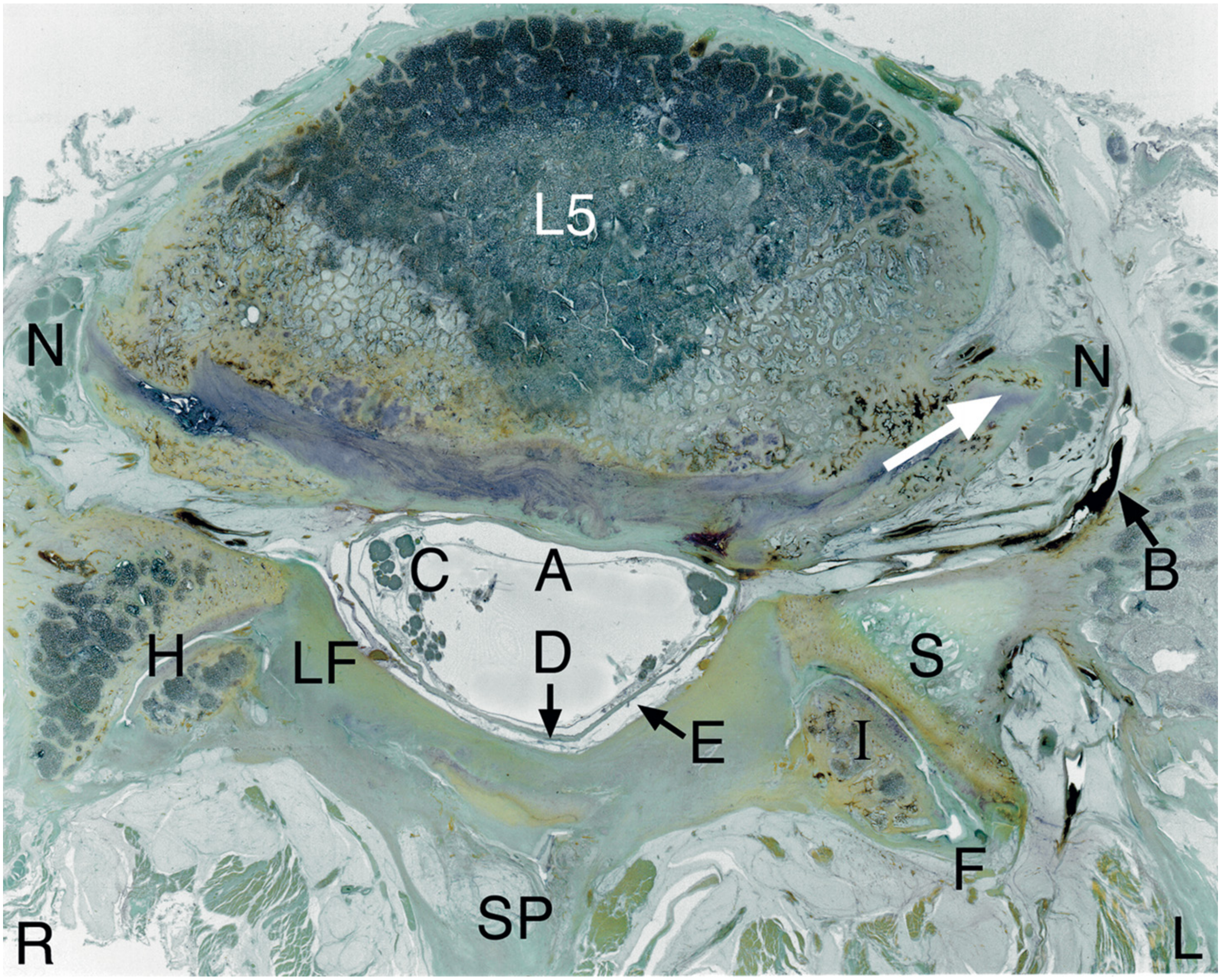

Figure 37.6 An axial histopathology section from the L5–S1 level of a 60-year-old postmortem specimen. On the right side (R), there is no osteophyte of any significance and the spinal nerve (N) passes freely beside the L5 vertebral body. However, on the left side (L), there is an osteophyte (arrow) abutting the adjacent spinal nerve that is being deformed by the osteophyte. A = arachnoid membrane; B = blood vessel; C = cauda equina nerve roots within the lumbar dural tube; D = dural membrane; E = epidural fat in the epidural space; F = fibrous capsule posteriorly for the zygapophysial joint; H = hyaline articular cartilage on the zygapophysial joint facet surfaces; I = inferior articular process of the L5 vertebra; LF = ligamentum flavum; L5 = body of the L5 vertebra; S = superior articular process of the sacrum; SP = spinous process.

KEY POINT

When a clinician requests a particular series of MR imaging planes based on the history and clinical examination, one expects the radiology facility to comply with that request based upon an informed clinical decision. Perhaps the outcome for this case would have been successful had the surgeon had such additional information.

Further reading

Chang H S, Nakagawa H 2004 Altered function of lumbar nerve roots in patients with transitional lumbosacral vertebrae. Spine 29: 1632–1635.

Lee C H, Park C M, Kim K A et al 2007 Identification and prediction of transitional vertebrae on imaging studies: Anatomical significance of paraspinal structures. Clin Anat 20: 905–914.

Matsumoto M, Chiba K, Nojiri K et al 2002 Extraforaminal entrapment of the fifth lumbar spinal nerve by osteophytes of the lumbosacral spine: Anatomic study and a report of four cases. Spine 27: E169–E173.

Case 38

Incorrect laminectomy level for lumbar intervertebral disc bulge

COMMENT

When a patient's symptoms persist following surgery, perform appropriate imaging to look at the surgical level.

PROFILE

A 42-year-old male manual worker of average build who does not smoke cigarettes but drinks a few beers socially.

PAST HISTORY

He had undergone an 'L4–5 laminectomy' 9 years ago for low back pain and right leg pain following jumping out of a moving vehicle.

PRESENTING COMPLAINT(S) (Fig. 38.1)

Constant lower lumbar pain with referral of pain posteriorly to both buttocks and thighs due to his above injury. Sometimes he experiences pain to halfway down the left calf with 'pins and needles' in the last two toes of the left foot (S1). The severity of pain varies with his level of activity. For example, if he drives for a long journey, i.e. approximately 5 hours, he has to stop the car and walk around for 10 minutes in an attempt to lessen his lower lumbar pain and lower limb symptoms. In order to change gear in his manual car, he has to lift his left foot and lower leg by pulling up on his trouser leg to place his foot on the clutch. The pain in his low back awakens him at night on turning over in bed but it does not awaken him at the same time each night. He said that the L4–5 laminectomy had resulted in a disappointing maximum 30% relief for his low back pain syndrome. He takes Di-gesic and Celebrex (200 mg per day) for his symptoms with some temporary lessening of symptoms but he wants to know why he still suffers considerable symptoms following surgery 9 years ago.

He had post-surgery letters from three orthopaedic surgeons. The first stated: 'The patient has a degenerative lumbo-sacral spine. One would generally expect that the widespread nature of the problem would mean that an injury or insult to the back has not been the only cause of his continuing problems and it is interesting to note that most of the degenerative changes occur at the L3–4 level whereas the laminectomy was apparently at the L4–5 level'. The second letter stated: 'The patient presents a very bizarre picture and part of this may be over-presentation with inconsistencies. He would never have seen out his

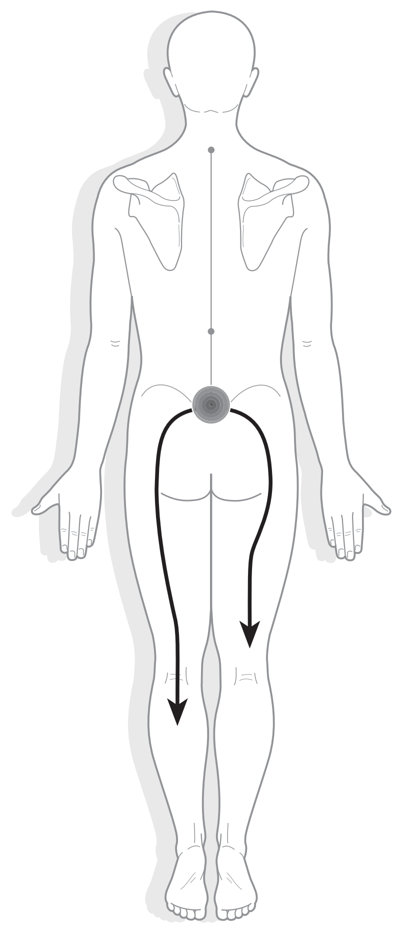

Figure 38.1

working life and most probably would have had to retire at the age of approximately 55 years due to his pre-existing degeneration'. The third letter stated: 'I performed a laminectomy on the [date] removing the L4–5 disc'.

AETIOLOGY

When a vehicle that he was driving ran out of control, due to brake failure, he jumped out of the cabin and landed on his low back and right side and immediately felt low back pain with some right leg pain.

EXAMINATION

In the erect posture, there was no evidence of pelvic obliquity or scoliosis. Percussion of the lumbar spine was painless. Deep palpation of paraspinal muscles elicited pain bilaterally at L3–4 and at L5–S1. Erect posture straining of the sacroiliac joints did not elicit any sacroiliac joint pain but elicited an increase in central low back pain. The deep tendon reflexes at the knees and ankles were normal. The plantar response was normal. Pinprick sensation of the lower extremities appeared to be normal. Vibration sensation was normal at the ankles. Slumping forwards while seated elicited an increase in low back pain at approximately the L4 level; addition of straight leg raising elicited an increase in lower lumbar pain at a measured 40° (left) and 60° (right). Motor power in the lower limbs was normal as was the case with the foot pulses and the temperature of the left and right feet on palpation appeared to be normal and equal. The Milgram active bilateral straight leg raise elicited an increase in low back pain. Straight leg raising was limited by lower lumbar pain to a measured 40° (left) and 55° (right). Lasegue's sign was positive for low back pain for the left and right legs, respectively. Straight leg raising with left and right foot dorsiflexion, respectively, elicited an increase in low back pain. Straight leg raising with left and right foot plantar flexion, respectively, was painless. Movement of the left and right hip joints was normal.

Erect posture lumbar spine active ranges of movement were as follows:

1. Flexion was limited by low back pain when his fingers reached to his knees.
2. Extension was limited by approximately 25% due to increasing low back pain.
3. Left and right rotation were limited by approximately 40% due to pain at the L4–5 level.
4. Left and right lateral bending were limited by low back pain when his fingers reached his knees.

IMAGING REVIEW

Prior to surgery a CT lumbar spine scan from the upper body of L3 to the upper sacrum had been performed with the gantry vertical then with the gantry angled. The report stated: 'The L4–5 disc is bulging and this is more marked on the right side. There could well be compression of the right L4 nerve root due to this. There is no evidence of a herniation of the remaining lumbar discs scanned'.

CLINICAL IMPRESSION

Possible recurrent L4–5 intervertebral disc or perineural fibrosis.

WHAT ACTION SHOULD BE TAKEN?

Current erect posture pelvis and lumbar spine plain film radiographs were requested and were reported as showing: 'L3–4 intervertebral disc space gross narrowing with minor narrowing of the L4–5 interspace (Fig. 38.2). No osteophytic reaction is noted on the vertebral body surfaces. Neural arches are intact and the lumbar apophyseal joints and sacro-iliac joints appear normal. No evidence of any previous laminectomy'. However, Figure 38.3 clearly shows that a right L3–4 hemilaminectomy has been performed.

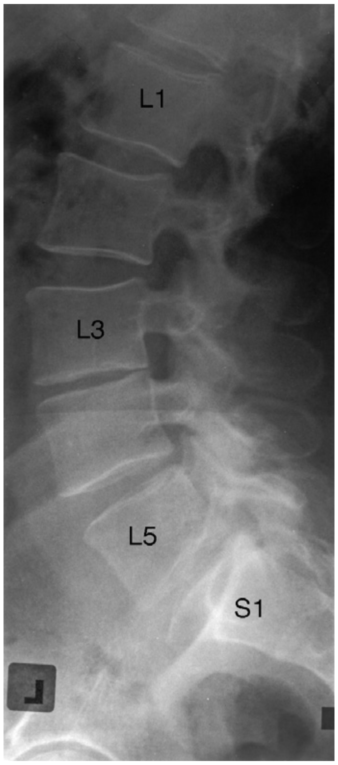

Figure 38.2 Lumbosacral spine erect posture lateral plain X-ray image. Note (i) the L3–4 intervertebral disc space height is grossly narrowed due to surgery at this level, (ii) the minor narrowing of the intervertebral disc space height at L4–5 level (due to posterior disc bulging seen on the pre-surgery CT scan), and (iii) there is essentially no osteophytic lipping on any vertebral bodies to suggest that the patient has a 'degenerative lumbo-sacral spine'.

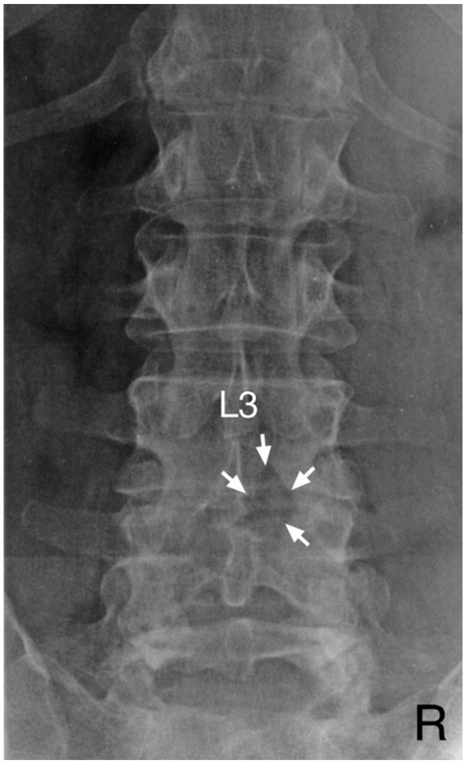

Figure 38.3 Lumbosacral spine erect posture anteroposterior plain X-ray image. Note the right sided L3–4 hemilaminectomy (arrows). L3 = L3 vertebral body; R = right side of patient.

Therefore, a repeat, i.e. post-surgery CT lumbosacral spine scan was requested through the three lower lumbar discs. The report stated: 'At the L3–4 level, a right L3 laminectomy defect is noted. There is loss of normal fat density in the right lateral recess, consistent with post-operative scarring versus prolapsed disc material. There may be slight posterior displacement of the adjacent right nerve root at this level. L4–5 level shows a mild concentric disc bulge (Fig. 38.4), without significant displacement of the thecal sac or compromise at the nerve roots in the neural foramina. At L5–S1, the appearances are normal'. On review, there appeared to be a degree of contact between the L4–5 mild concentric posterior disc bulge and the left and right nerve roots (Fig. 38.4).

DIAGNOSIS

L4–5 posterior concentric disc bulge abutting the pain sensitive anterior surface of the dural tube and the adjacent left and right nerve roots.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under

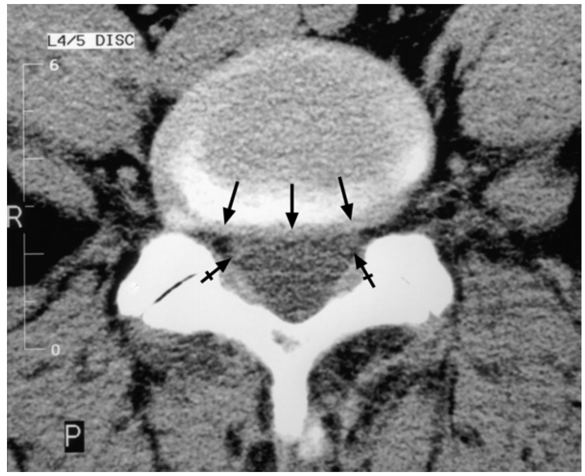

Figure 38.4 CT axial image through the L4–5 intervertebral disc level. Note the 'mild concentric disc bulge' (black arrows) that, in fact, abuts, to some degree, the pain sensitive anterior surface of the dural tube and the left and right nerve roots (black tailed arrows).

Treatment, to ensure that the likely cause of symptoms was understood. In particular, he was told that the gross loss of disc space height at the L3–4 intervertebral disc level with the right sided laminectomy at this level clearly showed that this was the level of laminectomy surgery. The finding of the persisting L4–5 mild concentric disc bulge with disc material abutting the pain sensitive anterior surface of the dural tube and the left and right nerve roots was explained to him and that this, in all likelihood, causes his low back pain syndrome.

The abovementioned orthopaedic reports that the patient produced at the time of presentation are of considerable interest in view of the subsequent plain film and lumbosacral spine CT imaging.

The patient said that, now that he understood why he still had symptoms, he would prefer to depend upon anti-inflammatory medication and performing a prescribed exercise routine (see Case 1) rather than consulting another orthopaedic surgeon.

KEY POINTS

1. It has been well documented that surgery can be performed at the wrong spinal level.
2. When the history and symptoms suggest that a patient has not dramatically improved following surgery, it is worth looking at the spine with appropriate current imaging.

Case 39

Chronic low back pain with an incidental finding

COMMENT

Always listen carefully to the patient and think laterally.

PROFILE

A 59-year-old housewife who does not smoke or drink alcohol.

PAST HISTORY

She had a mastectomy performed 10 years ago due to breast cancer. A pelvic metastatic lesion had been excised 5 years ago, followed by radiotherapy and medication.

PRESENTING COMPLAINT(S) (Fig. 39.1)

Chronic low back pain exacerbated by home duties. She mentioned, as an aside, that she had noticed a small swelling in front of her left ear approximately 2 months ago. She also mentioned having a dry mouth and said she had told her dentist about this as he was performing left lower jaw root canal treatments. The dentist apparently said she should not worry about the swelling or dry mouth but that a 'wait and see' approach should be taken. On close questioning, she said she had been experiencing night sweats and that she had lost some weight; she thought the weight loss was due to her eating less food because of her dry mouth and her dental problems.

AETIOLOGY

1. Chronic low back pain – but probably related to L5–S1 intervertebral disc degenerative changes shown on a previous X-ray examination.
2. Swelling anterior to the left ear at approximately the level of the cavity of the ear, i.e. in the upper region of the parotid gland – unknown.
3. Sensation of a dry mouth – unknown.
4. Night sweats – unknown.

EXAMINATION

The swelling over her left parotid gland measured approximately 1.5 cm × 1.00 cm and, on palpation, appeared to be somewhat solid and circumscribed. It was painless except when pressure was applied to it. The examination was terminated in view of this finding.

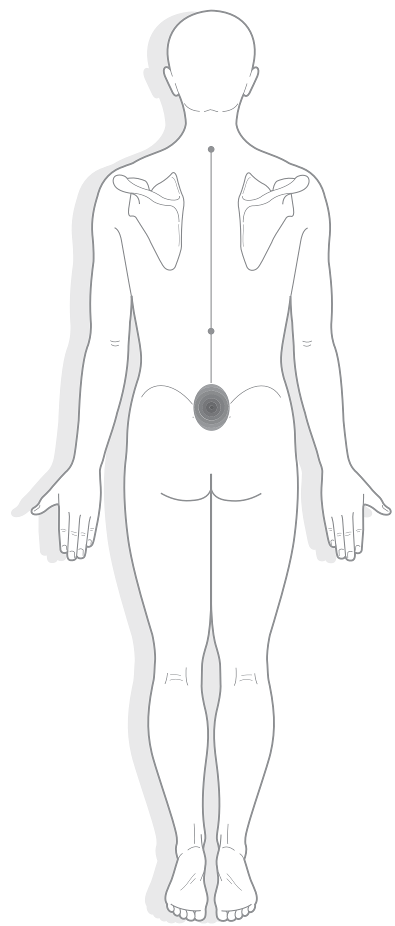

Figure 39.1

CLINICAL IMPRESSION

Possible malignant tumour in the left parotid gland.

Possible lymphoma in view of the night sweats and weight loss.

WHAT ACTION SHOULD BE TAKEN?

Because of the swelling she was advised to (i) see her general medical practitioner promptly for a CT scan of her face and neck, as well as a biopsy of the swelling, and (ii) to have a Positron Emission Tomography PET scan, that uses radioactive glucose injected into a vein, performed. Her general medical practitioner suggested she should wait and see what transpired in view of the current dental treatment. Approximately 1 month later, her dentist noticed that the swelling had increased so the patient was referred to a maxillo-facial surgeon, i.e. 3 months after the lump was first noticed. The maxillo-facial surgeon, upon examining the patient, ordered a contrast CT scan of the maxilla, mandible and parotid glands as well as a biopsy. The CT scan showed a 2.5 cm diameter ovoid mass on the surface of the left parotid gland (Fig. 39.2) with a medial lobule of this mass indenting the anterior aspect of the superficial lobe but with no deep lobe involvement. In addition, there were multiple normal sized and enlarged hypervascular lymph nodes within the left neck and in the pre-laryngeal region and the superior aspect of the neck on the right side (Fig. 39.2) and deep to the sternomastoid muscle with extension to the adjacent posterior triangle. Extensive foci are present in the left posterolateral aspect of the neck in the tissue plane between the trapezius and levator scapulae muscles as well as in the trapezius muscle itself. Some of the lymph nodes involved in the neck and upper thoracic muscles are shown in Figures 39.2 to 39.6.

The biopsy indicated the possibility of malignant infiltration. The maxillo-facial surgeon referred her to an oncologist who admitted her to hospital and arranged for a facial plastic and reconstructive surgeon to see her. The surgeon requested a CT scan of the chest, abdomen and pelvis and these were reported as being normal; he then removed the left parotid gland tissue containing the malignancy, as well as some lymph nodes in the neck on that side. The surgeon sent the resected tissue for histopathological evaluation and referred the patient back to her oncologist. The pathology report suggested a non-Hodgkin's type of lymphoma.

Laboratory tests were performed to determine exactly what type of non-Hodgkin's lymphoma it was, i.e. a complete blood count with differential, a peripheral blood smear test and blood chemistry (including lactate dehydrogenase (LDH)) tests were performed as well as liver and kidney function tests. In addition, a bone marrow biopsy did not indicate any lymphoma in the bone marrow.

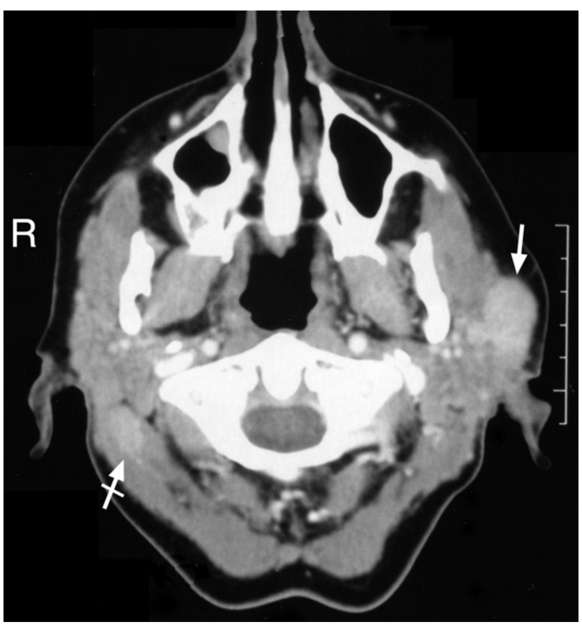

Figure 39.2 CT axial image showing (i) a mass on the left parotid gland (arrow), and (ii) involvement of some lymph nodes in the right upper neck (tailed arrow).

Figure 39.3 CT axial image at the C7–T1 level showing involvement of lymph nodes on the left side at this level (three arrows).

Figure 39.4 CT axial image at the T1–2 level showing involvement of lymph nodes on the left side at this level (two arrows).

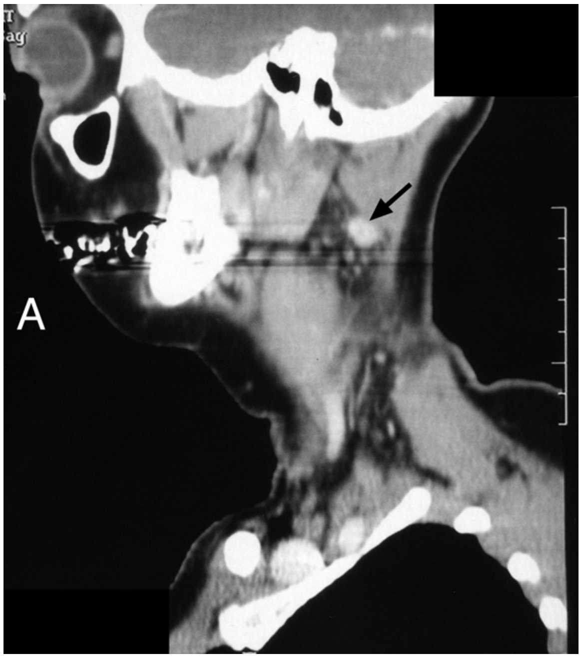

Figure 39.5 CT parasagittal image showing lymph node involvement in the upper neck on the right side (arrow).

Meanwhile, the oncologist arranged for a PET scan procedure. Following injection of 140 Mbq of F-18 FDG (fluorodeoxyglucose), emission tomographic imaging was performed from the top of the brain to the mid-thighs. Measured attenuation correction was performed. Blood sugar level was 5.6 mmol/L. Correlation was made with prior CTs of the neck, thorax, abdomen and pelvis. The report concluded: 'There is FDG avid disease above and below the diaphragm (Fig. 39.7). FDG avid lymph nodes are seen in the right neck. Persistent FDG activity in the left neck may relate to the recent surgery however residual left sided lymph node disease cannot be excluded. FDG avid left inguinal and external iliac lymph nodes are present. There is intense FDG uptake related to the thyroid gland more intense on the left. I am uncertain whether this uptake is in the thyroid gland or within adjacent lymph nodes. Intense FDG avid disease is seen in the left lower posterolateral neck extending into the upper back related to the trapezius and levator scapulae muscles. FDG avid disease is seen in the right anterior chest wall and within other muscles of the abdominal wall and back. There are in addition FDG avid lesions in the right upper arm and in the anterior left thigh'.

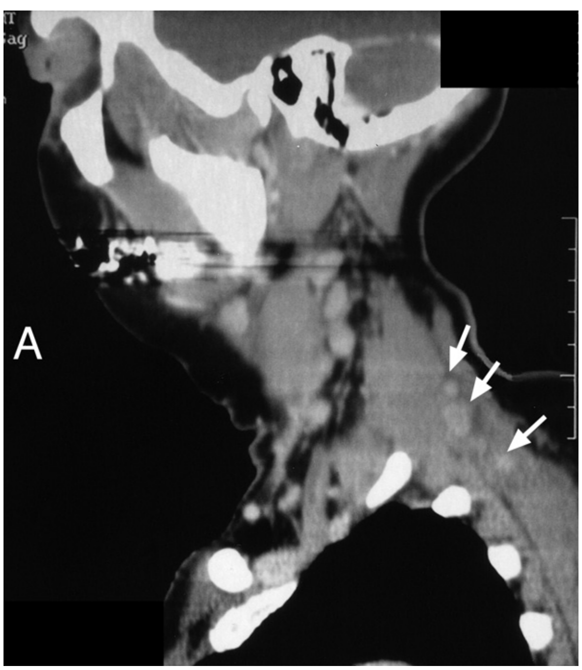

Figure 39.6 CT parasagittal image showing lymph node involvement at the cervico-thoracic region on the left side (arrows).

DIAGNOSIS

Anaplastic lymphoma kinase negative (Stage IIIE), also known as large cell anaplastic lymphoma.

TREATMENT AND RESULTS

Before chemotherapy commenced: (i) a peripherally inserted central catheter (PICC) line was inserted into the basilic vein in her right forearm with its tip resting in the distal superior vena cava to facilitate chemotherapy doses and blood tests, (ii) a gated cardiac blood pool scan – 99mTc RBC was performed for baseline left ventricular function; this was found to be normal, and (iii) her weight and height were recorded to determine chemotherapy doses using the Hyper-CVAD chemotherapy drug protocol over a 6 month period in an attempt to achieve remission or a cure. One year later the condition returned.

Note

Night sweats are a common outpatient complaint with tuberculosis and lymphoma being diseases in which night sweats are a dominant symptom; in general terms, causes include malignancy, infections, endocrine (including peri-menopausal women), rheumatologic, drug and other complaints (Mold et al 2002, Viera et al 2003). Individuals who have a malignant condition frequently have other symptoms, such as unexplained weight loss, that are indicators of the likelihood of an underlying malignant condition.

A PET scan can help to pinpoint the source of cancer as many malignant cells are highly metabolic and, therefore, will synthesize radioactive glucose injected into the patient prior to the examination. Areas of high glucose uptake are seen in the PET scan imagery. CT and MRI cannot detect active malignant tumours.

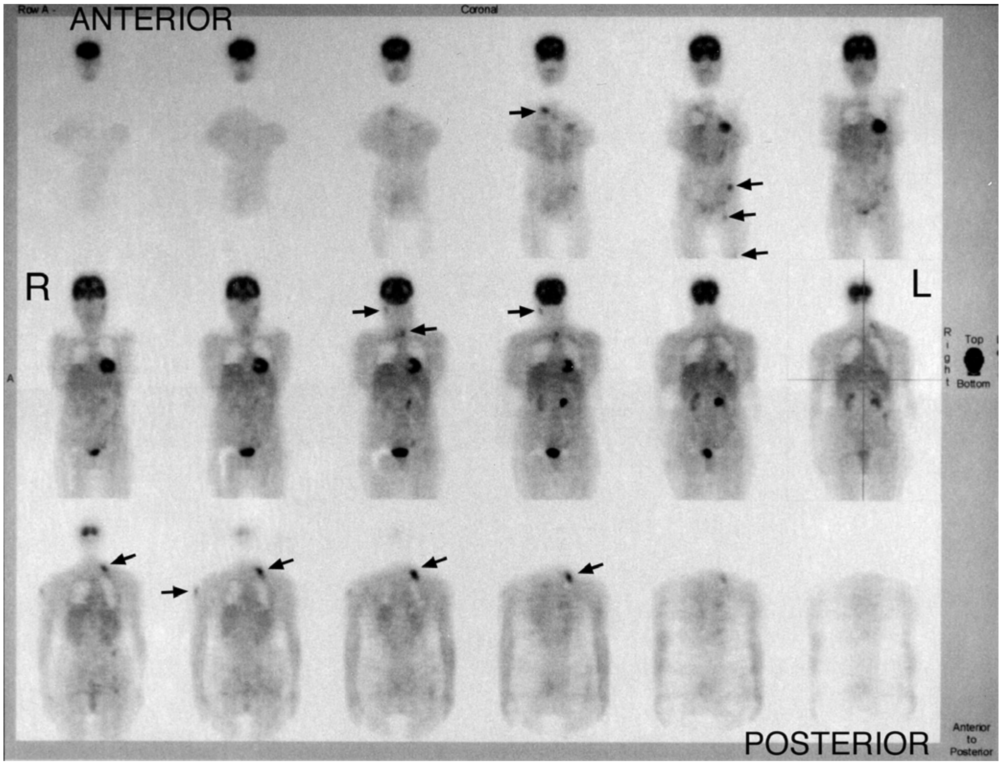

Figure 39.7 Coronal plane images from the pre-treatment F-18 FDG PET scan. The arrows indicate FDG activity above and below the diaphragm. R = right side of patient, L = left side of patient. A follow-up PET scan after 7 months of treatment was normal but a PET scan at 12 months showed a Grade I recurrence.

KEY POINTS

1. This case clearly illustrates that one has to listen carefully to the patient.
2. Remember that patients may present with more than one condition.

References

Mold J W, Mathew M K, Belgore S, DeHaven M 2002 Prevalence of night sweats in primary care patients: an OKPRN and TAFP-Net collaborative study. J Fam Pract 51: 452–456.

Viera A J, Bond M M, Yates S W 2003 Diagnosing night sweats. Am Fam Physician 67: 1019–1024.

Further reading

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Ghanem N A, Packe G, Lohrmann C et al 2007 MRI and ^{18}FDG-PET in the assessment of bone marrow infiltration of the spine in cancer. Eur Spine J 16: 1907–1912.

Sørensen H T, Mellemkjaer L, Skriver M V et al 2005 Fever of unknown origin and cancer: a population-based study. Lancet Oncol 6: 851–855.

Case 40

Multiple level intervertebral disc degeneration

COMMENT

This case illustrates the value of a complementary medicine approach and how it would be beneficial for patients if clinicians from different backgrounds worked together.

PROFILE

A 31-year-old slim professional woman who does not smoke cigarettes and only rarely drinks alcohol.

PAST HISTORY

Nothing contributory.

PRESENTING COMPLAINT(S) (Fig. 40.1)

Chronic lumbar spine back pain increasing in intensity during the last 2 years. During the last 2 weeks she has experienced acute, i.e. 'intense' lumbosacral pain following a sneezing episode that resulted in her having to spend 2 days in bed. She has been left with a 'stabbing' pain in the left and right legs posterolaterally as far as the upper calves and a sense of 'weakness' and 'numbness' in the left leg. She can no longer stand for more than a few minutes without aggravating her symptoms. She has tried physiotherapy and a TENS machine without relief. A non-steroidal anti-inflammatory drug (Brufen) was not helpful so she now takes the non-steroidal anti-inflammatory drug Fenac.

AETIOLOGY

There is no history of trauma prior to the development of lumbar spine pain 2 years ago. Sneezing 2 weeks ago caused the acute low back and lower limb symptoms.

EXAMINATION

In the erect posture, there was no clinical evidence of pelvic obliquity, leg length inequality or scoliosis. There was palpable bilateral paraspinal lumbar muscle spasm involving particularly the lower lumbar spine. Deep palpation of the paraspinal muscles elicited pain bilaterally at the T11-L1 levels and from the L4-S1 levels. Active thoracic spine ranges of movement were essentially normal apart from slight thoracolumbar junction pain. Active lumbar spine ranges of movement were as follows:

1. Flexion – limited by 50% due to increasing low back pain.
2. Extension – limited by approximately 100% due to low back pain.

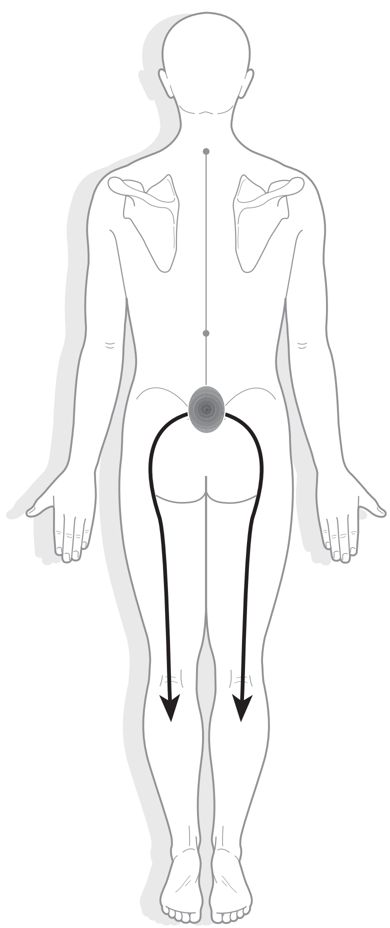

Figure 40.1

3. Left and right lateral bending, respectively, were very limited due to low back pain.
4. Left and right rotation were very limited due to low back pain.

Supine straight leg raising was limited to a measured 60° bilaterally due to increasing low back pain. The right Achilles deep tendon reflex (S1) was very weak (i.e. 0 to +). Pinprick sensation indicated hypoaesthesia in the left and right L5 and S1 dermatomes. Power was diminished (4/5) for right foot eversion (L5, S1) and for right big toe dorsiflexion (L5). The right femoral nerve stretch test elicited pain on the right side of the lower lumbar spine. The Valsalva manoeuvre aggravated her low back pain.

IMAGING REVIEW

Plain X-ray films showed 'a loss of disc space height at the T12–L1, L3–4 and L4–5 levels'.

CLINICAL IMPRESSION

Possible intervertebral disc bulge/protrusion at the T12–L1 and lower lumbar spine levels.

WHAT ACTION SHOULD BE TAKEN?

In view of the original insidious onset of low back pain beginning 2 years previously, laboratory tests were considered but not undertaken prior to obtaining results from a thoracic and lumbar spine MRI that was requested. The MRI report stated for the Thoracic spine: 'Narrowing and loss of signal of the T11–12 and T12–L1 intervertebral discs (Fig. 40.2A) in keeping with disc degeneration and small focal protrusions that indent the thecal sac (Fig. 40.2B) but cause no significant canal stenosis or cord compression.

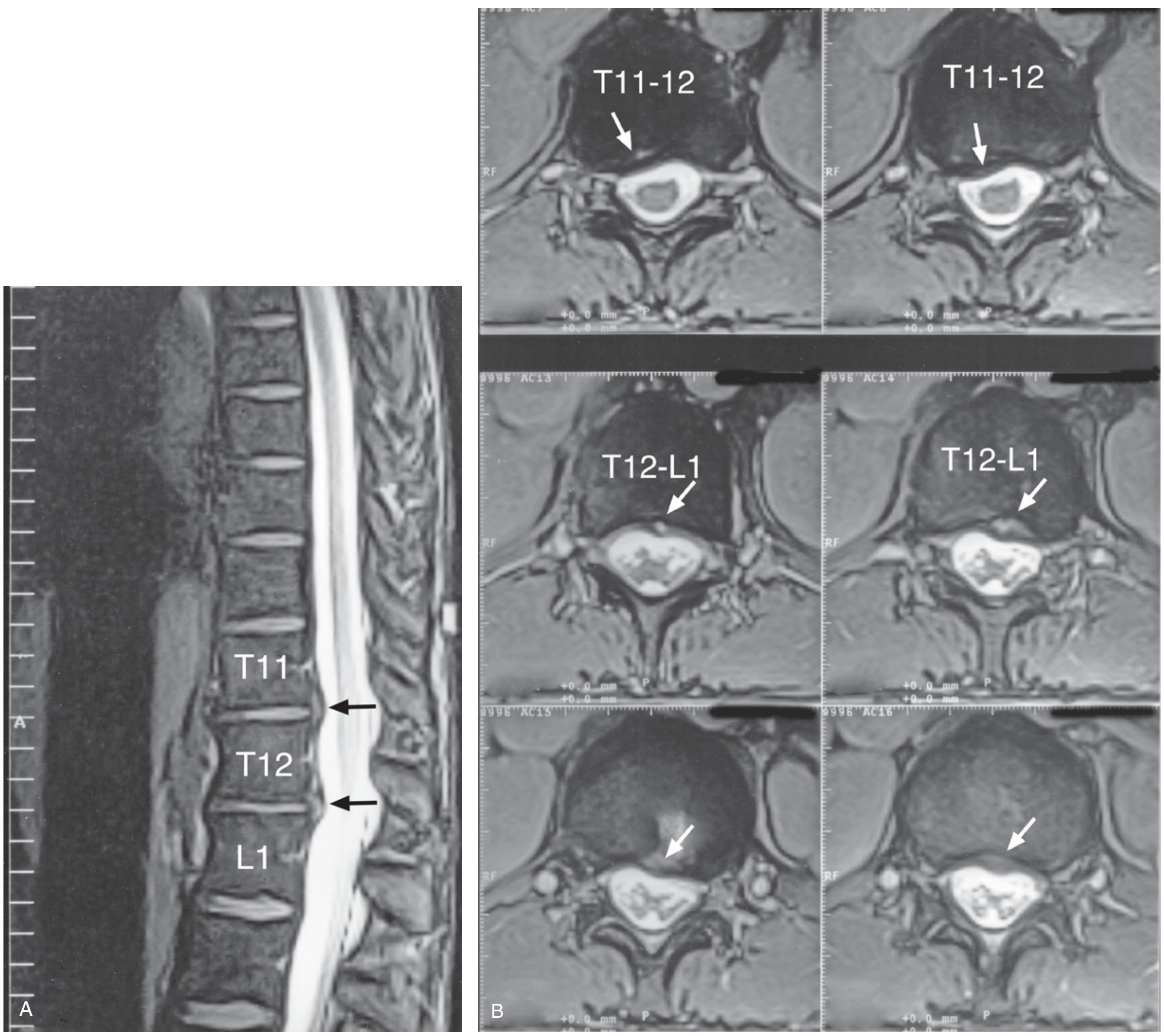

Figure 40.2 (A) MRI sagittal T2-weighted image showing narrowing and loss of signal of the T11–12 and T12–L1 intervertebral discs that have posterior disc protrusions (arrows) that indent the thecal sac. Axial views are shown in Fig 40.2B. (B) MRI axial T2-weighted images at the T11–12 and T12–L1 levels. Note (i) the central to right sided disc protrusions at T11–12 (arrows), and (ii) the central to left sided disc protrusion at T12–L1 (arrows).

For the Lumbar spine: 'Narrowing and loss of signal of the L3–4 and L4–5 intervertebral discs (Fig. 40.3) in keeping with disc degeneration. At L3–4 and L4–5 level, a combination of a broad based disc protrusion and facet joint arthropathy produce a mild canal stenosis as well as stenoses of both lateral recesses'.
'No intrinsic cord lesion. The remainder of the intervertebral foramina appear clear.'

DIAGNOSIS

T11–12, T12–L1, L3–4 and L4–5 intervertebral disc posterior protrusions with some degree of canal stenosis at all levels with some indentation of the dural tube. At L3–4 and L4–5 levels mild stenosis of both lateral recesses was reported.

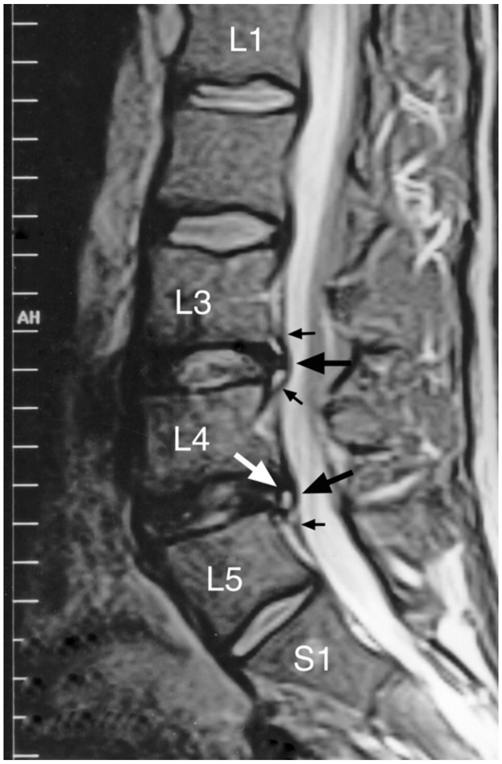

Figure 40.3 Lumbar spine MRI sagittal T2-weighted image showing posterior disc protrusions at the L3–4 and L4–5 levels (black arrows). At the L3–4 and L4–5 levels the posterior intervertebral disc protrusions elevate the posterior longitudinal ligament (small black arrows). The L4–5 intervertebral disc has a posterior tear with a high intensity zone (white arrow).

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. In view of the lower thoracic and lower lumbar disc protrusions, needle acupuncture using the 'near and far' technique was suggested and provided twice per week for 3 weeks, then on an as required basis for pain control. A Pilates exercise programme was commenced under physiotherapy supervision then continued without supervision at home on a continuing basis. She was advised to minimize her intake of Fenac medication so as to prevent any possible adverse reactions. She responded well to treatment and her lumbar spine and leg symptoms gradually diminished and she became asymptomatic over a period of approximately 5 weeks.

Approximately 8 weeks after commencing conservative treatment she experienced an acute recurrence of her low back and leg symptoms following lifting and, as a precaution, a neurosurgical opinion was sought. Unfortunately, she was given an unflattering description of the complementary medicine approach to date in spite of her significant progress prior to her acute recurrence. However, the neurosurgeon told her that neurosurgery would not be the answer for her but would not advise her on how to deal with her lumbar spine and leg symptoms. She decided to continue with acupuncture and Pilates exercises as previously suggested which, again, resulted in great success. Some years later, she was still doing well and had learnt to be careful in her daily professional activities so as not to create a recurrence of her symptoms.

KEY POINTS

1. The advantage of complementary medicine should not arbitrarily be dismissed by those who are not trained in it – particularly when safe alternative treatment is not available.
2. The important issue should always be to help patients.
3. Also please see Case 89.

Further reading

Haake M, Muller H-H, Schade-Brittinger C et al 2007 German acupuncture trials (GERAC) for chronic low back pain. Randomised, multicenter, blinded, parallel-group trial with three groups. Arch Intern Med 167: 1892–1898.

Case 41

Lumbar osteoporotic fractures

COMMENT

A conservative approach to treatment was helpful for this elderly lady.

PROFILE

A 94-year-old fit female of slim build who does not smoke cigarettes or drink alcohol.

PAST HISTORY

Approximately 40 years ago she was a passenger in a motor vehicle accident in which the vehicle rolled over and she sustained two fractures of the pelvis. She made a very good recovery and only occasionally suffered mild right sided sciatica extending into the right buttock and thigh posteriorly, and sometimes as far as the calf.

PRESENTING COMPLAINT(S) (Fig. 41.1)

Centrally located lumbar spine pain that extends from the thoracolumbar junction to S1 level with occasional aches in the paraspinal muscles.

AETIOLOGY

While walking in a poorly lit room she tripped on a carpet and fell head over heels; she was not aware of any immediate pain as she was somewhat shocked by the fall. A couple of weeks later she experienced increasing lumbar spine pain for which she took an analgesic but the pain persisted.

EXAMINATION

On examination she had no clinical evidence of leg length inequality, pelvic obliquity or scoliosis. All active ranges of lumbar spine movement were limited by approximately 50% due to lumbar spine pain. Bilateral paraspinal muscle spasm was present and percussion of the spinous processes elicited pain at several lumbar spinal levels. The deep reflexes in the lower limbs were normal as were pinprick sensation, power, muscle tone and the Babinski response. Straight leg raising was essentially painless and of normal range for this age. On getting up from the supine or seated position, twinges of intense pain in the lumbar

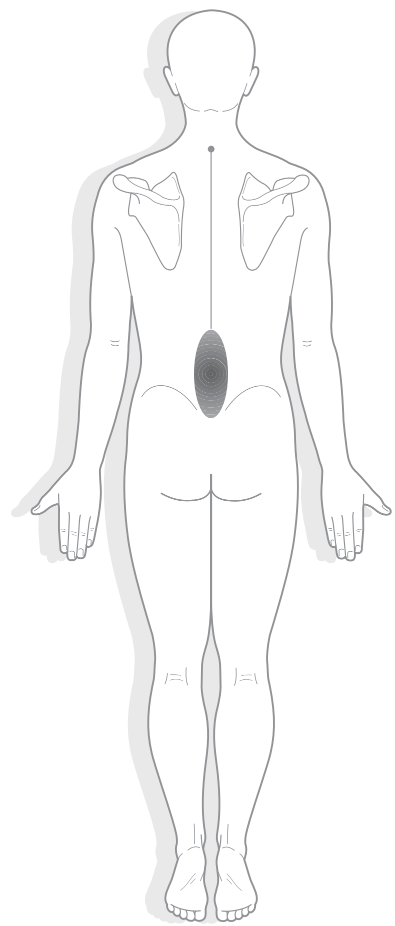

Figure 41.1

spine were reported. Bowel and bladder function were reported as being normal.

IMAGING REVIEW

A plain X-ray examination of the thoracolumbar spine and pelvis reported: 'Age-related osteoporosis with biconcave endplate indentation at most levels. Mild anterior wedging of the L1 vertebral body resulting in approximately 25% loss of height. This may indicate a recent fracture. No other evidence of a vertebral fracture. No paraspinal haematoma. No spondylolisthesis. Lumbosacral junction shows advanced zygapophysial joint degenerative changes. No pelvic or proximal femoral fracture. Moderate narrowing of the hip joint spaces'.

CLINICAL IMPRESSION

The radiology appeared to underestimate the patient's spinal condition when compared to the physical examination.

WHAT ACTION SHOULD BE TAKEN?

As the plain radiographs were not of optimal quality, an MRI lumbar spine examination was requested. As her condition had become more painful, she had to be transported by ambulance for the MRI investigation. The MRI showed the following: 'There is some degree of desiccation of all the lumbar and the 12th thoracic intervertebral discs (Fig. 41.2A). Depression of the L1 superior endplate with lesser depressions of the superior endplate at the L2 and L5 levels (Fig. 41.2A) with altered marrow signal intensity consistent with acute osteoporotic compression fracture. A haemangioma is noted in the T12 vertebral body (with flow voids noted within it, no pedicle abnormality or loss of vertebral body height, and no pathological marrow infiltrates) (Fig. 41.2B). L4–5 spinal canal stenosis as a result of bilateral ligamentum flavum and zygapophysial joint facet hypertrophy and circumferential disc protrusion (Fig. 41.3A). The nerve roots at the L4–5 level are spared. L5-S1 bilateral ligamentum flavum and zygapophysial joint facet hypertrophy (Fig. 41.3B) resulting in posterolateral dural tube effacement and central spinal canal stenosis due to posterior disc protrusion resulting in the ventral thecal sac effacement. There is a degree of lateral recess narrowing due to the hypertrophied ligamentum flavum and bilateral facet hypertrophy. There is mild osteoarthrosis of the zygapophysial joint facets. There is a normal cord, conus and cauda equina'.

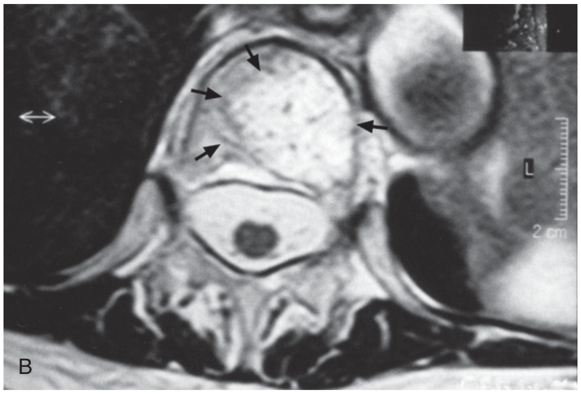

Figure 41.2 (A) Lumbar MRI sagittal T2-weighted image showing the depression of the L1 superior endplate with lesser depressions of the superior endplate at the L2 and L5 levels with altered marrow signal intensity consistent with acute osteoporotic compression fracture. There is some degree of desiccation of all the lumbar and the 12th thoracic intervertebral discs. There is a normal cord, conus and cauda equina. The white arrows indicate the large benign haemangioma in the T12 vertebral body. (B) MRI axial T2-weighted/TSE* image at the T12 level showing the large benign haemangioma (arrows) in the vertebral body with no pedicle abnormality and no pathological marrow infiltrates.

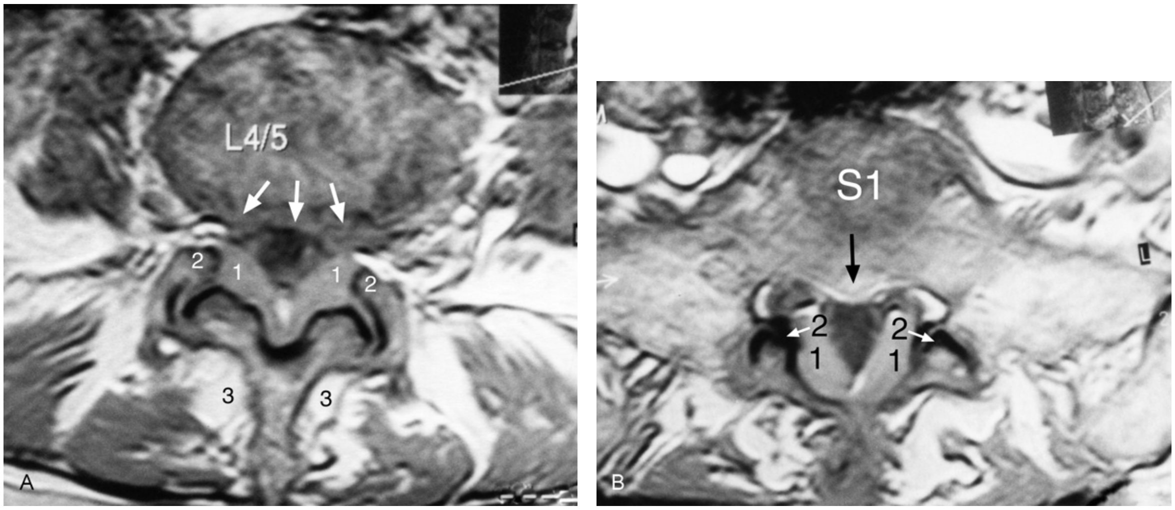

Figure 41.3 (A) MRI axial T1-weighted image at the L4–5 level showing the spinal stenosis as a result of bilateral ligamentum flavum hypertrophy (1), zygapophysial joint facet hypertrophy (2), and circumferential disc protrusion (arrows). There is mild osteoarthrosis of the zygapophysial joint facets. Note the fibro-fatty replacement of part of the multifidus muscle (3). (B) MRI axial T1-weighted image showing the L5-S1 level bilateral ligamentum flavum hypertrophy (1) and zygapophysial joint facet hypertrophy (2) resulting in posterolateral dural tube effacement. In addition, there is central canal stenosis due to posterior disc protrusion (arrow) resulting in the ventral thecal sac effacement. There is a degree of lateral recess narrowing due to the hypertrophied ligamentum flavum and bilateral facet hypertrophy. S1 = first sacral segment.

DIAGNOSIS

- L1, L2 and L5 vertebral body compression fractures with associated osteoporosis.
- Pre-existing asymptomatic spinal degenerative changes.
- Incidental finding of a large haemangioma in the T12 vertebral body.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. After the imaging was carefully explained to this intelligent lady she was told that she had the option of trialling a conservative treatment approach or being sent to an orthopaedic surgeon for an opinion regarding possible lumbar vertebroplasty. The possible risks of vertebroplasty were explained to her and she decided that she would prefer to take the conservative treatment approach. This involved the following steps.

She was advised to wear a supportive corset to protect and immobilize the lumbar spine as much as possible; the corset extended from the lower thoracic spine to approximately the S1 level. She had to wear the corset day and night apart from when she was showered of a morning by a carer. As she experienced considerable pain on moving, especially of a morning, the following treatment regimen was adopted:

5.30 a.m. 1 or 2 paracetamol 500 mg tablets and 1 Norflex Co tablet (an analgesic and muscle relaxant containing 35 mg orphenadrine citrate and 450 mg paracetamol). These were taken with yoghurt approximately 1 hour before arising of a morning.

She used a walker to go to the recreation room to have breakfast. One Calcium Sandoz Forte (Novartis) 1000 mg tablet was taken with breakfast.

Evening meal. One Coxflam 15 mg tablet (a selective COX2 inhibitor drug – each tablet containing 15 mg metoxicam) was taken at this time.

Bedtime. ¼ Lexotan tablet (3 mg bromazepam) to help her to sleep more easily and, last thing on going to bed, she took one Senokot tablet as a laxative.

If she experienced unacceptable pain during the day, she was given one additional 500 mg paracetamol tablet.

All medication was taken with food so as not to cause gastric symptoms.

Additional activities to minimize bouts of pain that were precipitated by changing position: A slip sheet was placed on her bed so that when she got up in the morning one carer was able to swivel the end of the sheet at her feet while another carer swivelled the sheet at her head, which meant her torso was kept straight. As her feet were lowered over the edge of the bed, the carer at her head raised her torso so that she was able to stand with the minimum of pain. The reverse of this was applied when she returned to her bed at night. The slip sheet enabled the carers to position her comfortably in the middle of the bed. In order for her to semi-recline in her bed, an electrically operated bed was used and the head and torso portion was raised to an angle where she felt comfortable. A carer slept in her room so that, should she need to go to the toilet at night, she was always taken care of and never left alone.

In order to shower she would sit on an appropriate shower chair and a carer would help her so that she did not twist or bend.

During the day, she used her walking frame to go from her bedroom to the recreation room where she would cautiously turn round and sit on an electrically operated chair that would lower her then elevate her feet so that she semi-reclined during the day. She was encouraged to perform lower limb exercises so as to maintain muscle tone and bulk, and to walk around the house several times a day.

Monitoring was undertaken to specifically look for any symptoms of adverse reactions to the medication.

Two months later she felt much improved but, unfortunately, bent over to pick up a light item from the floor and caused a severe exacerbation of pain, in the lower thoracic spine. In view of the high location of this pain and the large haemangioma demonstrated previously in the T12 vertebral body, she was taken by ambulance for a follow-up MRI thoracic and lumbar spine. This showed a 'hypo-intense band extending through the inferior aspect of the T11 vertebral body with adjacent hyper-intensity on the T2-weighted and stir images with a mild loss of T11 vertebral body height suggesting an acute fracture at this level'. In view of this finding, a corset that extended several segments higher into the thoracic spine was made for her and she was told that, under no circumstances, should she attempt to bend her spine. Medication continued as above, including taking the Calcium tablet, for a further 2 month period at which time the anti-inflammatory medication (Coxflam) dose was reduced to 7.5 mg each morning. Three months later the Coxflam medication was withdrawn as she felt much less pain so she only took one paracetamol tablet and one Norflex Co tablet each morning approximately 1 hour before arising. She continued with the Calcium tablet at breakfast on a daily basis. She had very little thoracic or lumbar pain approximately 1 year following her original fall, as long as she wore her corset and took one paracetamol tablet (500 mg) and one Norflex Co tablet of a morning. She still took one quarter of a Lexotan tablet and one Senokot tablet of an evening.

She stopped wearing her corset approximately 1.5 years following her fall. She became weaker as a result of her long period of incapacity and she used the walking frame for some 2 years after her fall. She then had to be assisted by her carer when getting up and sitting down and on being showered and when dressing. Fortunately, she was blessed with a good mind and, although she sat much of the time, she was able to take part in daily activities and social gatherings, mainly at home. When going out for a drive, she found it easiest to sit on a car seat covered with plastic so that her legs could be lifted and put into the car while her pelvis and torso swivelled easily on the plastic, in contrast to sitting on the material upholstered seating of her car. A wheelchair was used to transport her over uneven terrain so that she did not fall.

Approximately 16 months after commencing Norflex Co she stopped taking it as there was a suspicion of a slight gastric bleed, so she continued only with 500 mg paracetamol at 5.30 a.m. and 1/4 Lexotan and 1 Senokot tablet on going to bed.

She remained mentally alert. Understandably, she became frail and required a full time carer to make sure she did not fall. She still enjoyed being taken out in her car quite frequently and continued to do so until she quietly passed away in her ninety-eighth year.

Note

Vertebroplasty has risks such as combined extraforaminal and intradiscal cement leakage and intractable neurological complications can occur (Chen et al 2007).

KEY POINTS

- This conservative treatment approach was used in view of the patient's advanced years and as she did not want to risk undergoing vertebroplasty with its possible complications.
- Although haemangiomas are benign and are usually asymptomatic, when these highly vascular lesions become very large there is the potential for pathological fracture (Haldeman et al 2002) with considerable complications.

References

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Haldeman S D, Kirkaldy-Willis W H, Bernard N Jr 2002 An atlas of back pain. Parthenon Publishing Group, London, p 65.

Further reading

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Kim D H, Vaccaro A R 2006 Osteoporotic compression fractures of the spine; current options and considerations for treatment. Spine Journal 6: 479–487.

Lee W S, Sung K H, Jeong H T 2006 Risk factors of developing new symptomatic vertebral compression fractures after percutaneous vertebroplasty in osteoporotic patients. Eur Spine J 15: 1777–1783.

Ploeg W T, Veldhuizen A G, The B et al 2006 Percutaneous vertebroplasty as a treatment for osteoporotic vertebral compression fractures: A systematic review. Eur Spine J 15: 1749–1758.

Prince R L, Devine A, Dhaliwal S S 2006 Effects of calcium supplementation on clinical fracture and bone structure. Results of a 5-year, double-blind, placebo-controlled trial in elderly women. Arch Intern Med 166: 869–875.

Wu Z-X, Wei L, Hu Y-Y et al 2007 Staged-injection procedure to prevent cement leakage during vertebroplasty. An *in vitro* study. Spine 32: 2437–2442.

Case 42

L3–4 far lateral intervertebral disc protrusion

COMMENT

Look carefully for any possible sources of pain even when there is a known existing medical condition affecting the spine.

PROFILE

A 46-year-old housewife of average build who does not smoke cigarettes or drink alcohol.

PAST HISTORY

She developed rheumatoid arthritis approximately 7 years ago that affected her neck, low back and hands; her mild symptoms have been very well controlled with medication.

Two years ago she was involved in a motor vehicle accident at which time she was jolted upwards, her knee hitting the dashboard of the car, with resulting lumbar spine pain.

PRESENTING COMPLAINT(S) (Fig. 42.1)

Constant lower lumbar spine pain localized to approximately the L4 level due to the motor vehicle accident 2 years ago. She experiences a 'buning' pain in the left leg in the L3/L4 dermatomal distribution. The left leg symptoms awaken her at night but there is no night pain per se. Prolonged standing aggravates her lower lumbar pain and the left sided leg symptoms. Coughing and bearing down do not cause an increase in her symptoms. At night she puts a pillow behind her knees when lying supine, or between her knees when lying on her side, to lessen her lower lumbar spine and left leg symptoms. On awakening in the morning, her lower lumbar spine feels 'stiff'. She has not experienced any bowel or bladder problems.

She also complained of minor intermittent bilateral cervical spine pain that was thoroughly investigated but not dealt with here because the purpose of this case is to highlight her main symptoms of constant lower lumbar pain and left leg symptoms.

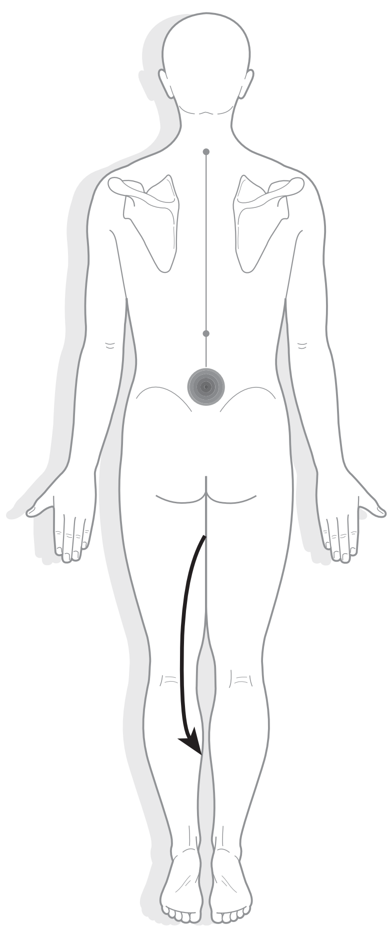

Figure 42.1

AETIOLOGY

Motor vehicle accident 2 years ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic and lumbar spines was painless. Sacroiliac joint strain testing did not elicit any sacroiliac joint pain. Motor power in the lower extremities appeared to be normal. The deep tendon reflexes in the upper and lower extremities were normal as was the plantar response. Vibration sensation at the ankles and elbows was normal. Pinprick sensation appeared to be normal over the lower extremities. The Valsalva manoeuvre did not elicit any pain. When seated and slumped forward this elicited lower lumbar spine pain; the addition of left straight leg raising aggravated the burning pain in the left leg. Supine straight leg raising was limited to a measured 45° (left) and 52° (right) due to hamstring tightness. The femoral nerve traction test was positive on the left side, i.e. resulted in pain radiating into the anterior thigh. The Fabere sign for hip joint function was normal. The foot pulses were normal and the temperature of both feet appeared to be normal on palpation. Active lumbar spine ranges of movement were as follows:

1. Flexion – her fingers reached to her knees but this aggravated her lower lumbar spine pain.
2. Extension was of full range and painless.
3. Left and right lateral bending – her fingers reached to her knees but this aggravated her lower lumbar spine pain.
4. Left and right rotation were of full range and painless.

IMAGING REVIEW

Plain film lumbar spine radiographs appeared to be normal apart from possible early thinning of the L3–4 intervertebral disc.

CLINICAL IMPRESSION

A possible intervertebral disc lesion at the L3–4 level.

WHAT ACTION SHOULD BE TAKEN?

She was referred for a CT lumbar spine and pelvis and the report stated: ‘At L3–4 level there is a far lateral left sided disc protrusion beyond the exit foramen (Fig. 42.2) that distorts the path of the left L3 nerve. At L4–5 and L5–S1 levels there is no disc herniation, canal or foraminal stenosis. The sacroiliac joints and hip joints appear normal. No bony pelvic abnormality is seen. No abnormal soft tissue masses are detected and there is no free fluid’.

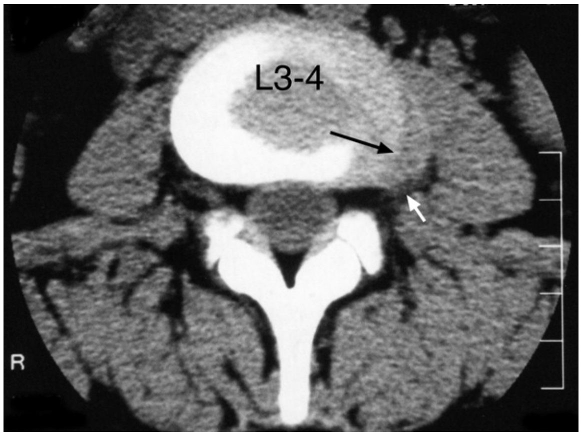

Figure 42.2 CT axial image at the L3–4 level showing the far lateral left sided disc protrusion (long black arrow) beyond the exit foramen; the protrusion distorts the path of the L3 nerve (short white arrow).

As there was no lipping of the vertebral body margins at the L3–4 level, no calcification in the protruded disc material and no subchondral sclerosis in the L3–4 facet joints, it was concluded that this large far lateral left sided disc protrusion occurred as a result of the motor vehicle accident that caused her symptoms.

DIAGNOSIS

Musculoligamentous soft tissue injuries of the lumbar spine with a far lateral left sided disc protrusion distorting the path of the L3 nerve.

TREATMENT AND RESULTS

The patient’s condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She said that, because she now understood the cause of her lower lumbar spine pain and left leg symptoms, she would live with her symptoms unless they became worse. Also, the anti-inflammatory medication that she was taking for her rheumatoid arthritis was of some help. She would not entertain having an orthopaedic opinion at this time and did not want manual therapy treatment, acupuncture or a perineural injection. She chose the option of protecting her low back by (a) losing some weight, (b) not performing any activities that would aggravate her symptoms, and (c) performing muscle strengthening exercises as shown in Case 1. This conservative approach resulted in satisfactory pain control for her and she was advised to see an orthopaedic or neurosurgeon if necessary.

KEY POINTS

1. In order to separate mid-to-upper lumbar radiculopathy from lower lumbar radiculopathy, it is important to perform the femoral nerve traction test for the L2, L3 and L4 nerve roots.
2. As the far lateral intervertebral disc protrusion was outside the spinal canal, the Valsalva manoeuvre was painless.

Case 43

Lumbar perineural 'block' injection

COMMENT

It is important to clinically differentiate between mid-to-upper lumbar radiculopathy and lower lumbar radiculopathy.

PROFILE

An 81-year-old fit male of average build who does not smoke cigarettes or drink alcohol.

PAST HISTORY

Chronic intermittent mild low back pain for many years.

PRESENTING COMPLAINT(S) (Fig. 43.1)

Low back pain radiating to his right hip then to the ankle during the last 4 weeks. Bowel and bladder function are normal. Coughing and sneezing do not cause any significant increase in symptoms. There was no night pain per se. He had been told that there was thinning of the L4–5 and L5–S1 intervertebral disc heights. He had been given an epidural block injection at the lower lumbar spine level and this had provided him with some temporary relief. However, he wanted to know whether more long-lasting relief may be possible.

AETIOLOGY

He was unaware of any specific incident that set off his low back and right leg symptoms but, on close questioning, he recalled tripping on a staircase approximately 8 weeks ago.

EXAMINATION

On examination there was no clinical evidence of leg length inequality or pelvic obliquity. Deep palpation of the paraspinal muscles in the lower lumbar spine elicited some tenderness, particularly on the right side. Right straight leg raising was limited to 30° due to an increase in low back pain but left straight leg raising did not elicit any symptoms. The right femoral nerve traction test elicited an increase in low back pain. The right knee deep tendon reflex was decreased at plus one, and pinprick sensation in the lower limbs was normal apart from hypoaesthesia in the right L4 dermatome. There was slight limitation of all lumbar spine active ranges of movement

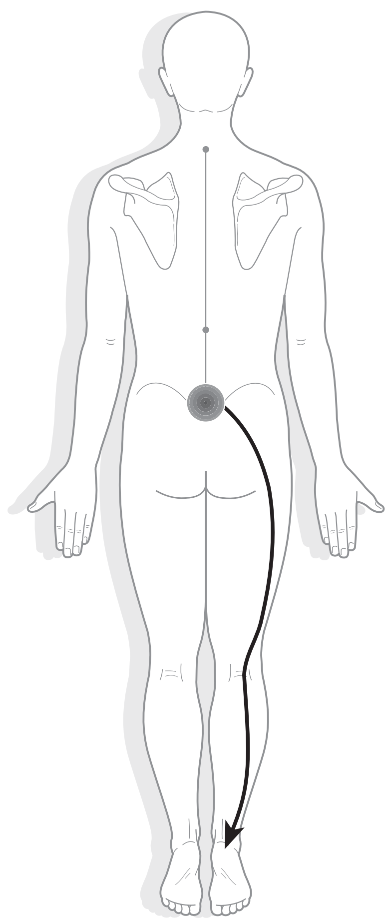

Figure 43.1

due to low back pain. The Valsalva manoeuvre only resulted in minor lumbosacral level pain.

IMAGING REVIEW

Plain film radiographs of his pelvis, sacrococcygeal spine and lumbosacral spine showed marked narrowing of the L5–S1 disc space height with moderate narrowing of the L4–5 disc space height. There was some lipping of all anterior vertebral body margins with moderate osteoarthritic changes in the zygapophysial facet joints. There was mild degenerative change at the L4–5 and L5–S1 facet joints. The sacroiliac joints were within normal limits. There were mild degenerative changes at both hips with minimal subchondral cyst formation and marginal spurring but no significant loss of joint space.

CLINICAL IMPRESSION

Probable right sided L4–5 or L5–S1 intervertebral disc protrusion.

WHAT ACTION SHOULD BE TAKEN?

A lumbar spine MRI was requested and the report stated: 'There is degenerative disc disease at all lumbar levels with desiccation (Fig. 43.2). The conus terminates at T12–L1 and there is no abnormal signal within the distal spinal cord. At L5–S1 there is marked loss of disc height and a moderate posterior disc protrusion without neural compression (Fig. 43.2 and Fig. 43.4). At L4–5 there is a right foraminal and far lateral disc extrusion (Fig. 43.3) producing compression of the exiting right L4 nerve. There is no lateral recess or left neuroforaminal stenosis. Moderate zygapophysial facet joint degenerative changes are present bilaterally at L4–5 and to a lesser extent at L5–S1.

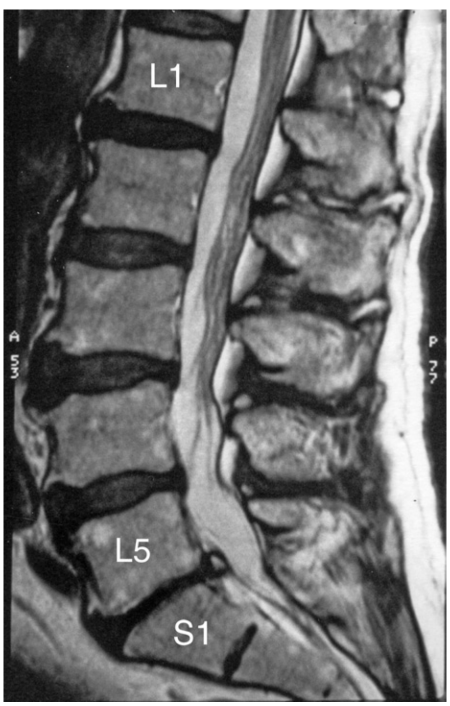

Figure 43.2 Lumbar spine MRI sagittal T2-weighted image showing (i) degenerative disc disease with desiccation at all lumbar levels and (ii) at L5–S1 a marked loss of disc height and a moderate posterior disc protrusion.

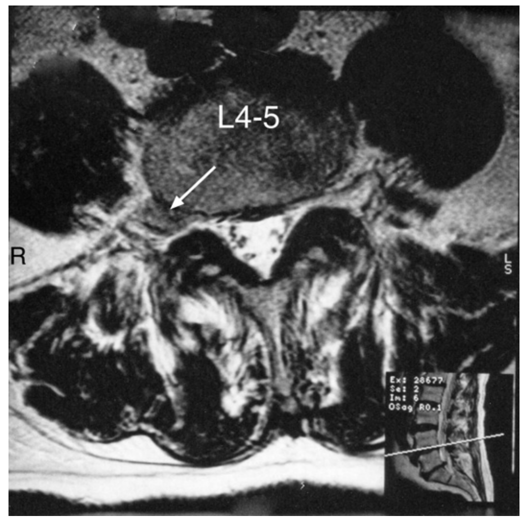

Figure 43.3 MRI axial T2-weighted image at the L4–5 level showing a right foraminal and far lateral disc extrusion (arrow) producing compression of the exiting right L4 nerve. Moderate zygapophysial facet joint degenerative change is present bilaterally.

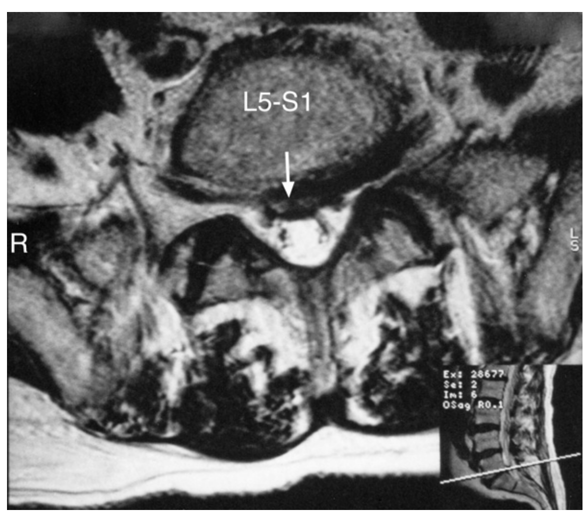

Figure 43.4 MRI axial T2-weighted image at the L5–S1 level showing a moderate posterior disc protrusion (arrow) without neural compression. Minor zygapophysial facet joint degenerative change is present bilaterally.

DIAGNOSIS

- A right foraminal and far lateral disc extrusion compressing the L4 nerve most likely causing his right lower limb symptoms.
- A moderate posterior intervertebral disc protrusion touching the anterior pain sensitive surface of the dural tube.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. As he had obtained some relief from a previous epidural block injection, a right L4 nerve block under CT guidance was suggested, to which he agreed. This was arranged to be performed by an interventionist radiologist. The patient underwent CT guided placement of a 22 gauge spinal needle with the tip at the right L4 perineural region (Fig. 43.5) before he was given an injection of 1 ml Celestone and 2 ml 0.5% Bupivacaine.

He tolerated the procedure very well without evidence of any acute complication. He received considerable relief from his low back pain and right leg symptoms and, as he now understood that he had a significant L4–5 right foraminal and far lateral disc extrusion producing compression of the exiting right L4 nerve, he agreed to be careful with his back. He was advised not to perform any activity that could overload his lumbar spine, such as lifting, or put his lumbar spine into extreme ranges of movement, such as bending forward too far. He was also advised to stop playing golf but to continue walking for exercise and to continue riding his stationary exercise bicycle as long as he is careful as to how he gets on and off the bicycle.

Figure 43.5 CT axial image at the L4–5 level showing the CT guided placement of a 22 gauge spinal needle with the tip at the right L4 perineural region.

At 15 months follow-up since the L4 nerve block procedure, he had been careful to protect his low back and, as a result, had maintained a very satisfactory level of pain control, not requiring a further nerve block injection. He was able to work cautiously in his garden but he never undertakes heavy work or any activity that could twist his low back. He has maintained his daily exercise programme by walking and riding his stationary bicycle.

KEY POINTS

1. In order to separate mid-to-upper lumbar radiculopathy from lower lumbar radiculopathy, it is important to perform the femoral nerve traction test for the L2, L3 and L4 nerve roots.
2. As the far lateral intervertebral disc protrusion was outside the spinal canal, the Valsalva manoeuvre was painless.

Further reading

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Case 44

Intervertebral disc protrusion into the intervertebral canal

COMMENT

Believe your patient until it is proven that the complaint is not genuine! Rely upon your own diagnostic acumen and not that of others.

PROFILE

A 53-year-old male invalid pensioner who was depressed because of his 4-year history of undiagnosed low back pain after being hit by a car. He does not smoke and only drinks one glass of wine per day.

He only came for a consultation because his wife was concerned about his suicidal ideation as he had been told that there was 'nothing wrong' with him following several specialists' consultations. His wife made the appointment for him and accompanied him to the consultation.

PAST HISTORY

A degree of asthma but otherwise unremarkable.

PRESENTING COMPLAINT(S) (Fig. 44.1)

Approximately L3–4 level pain extending into the left buttock with radiation to the lateral aspect of the left thigh and some left testicular pain. He was also experiencing urinary difficulties, with occasional hesitancy and poor stream, but there was no burning/dysuria or urethral discharge. He had also been constipated as he was taking non-steroidal anti-inflammatory drugs and analgesics.

The symptoms are worsened on sneezing. Sitting causes a severe increase in his symptoms, so he spends much of his time walking or standing when he is not resting on his bed.

AETIOLOGY

He was knocked over by a car and suffered severe low back and left buttock and posterolateral thigh pain for 1.5 years before his symptoms settled to a 'tolerable' level. He then inadvertently twisted his low back causing an acute exacerbation of his low back pain syndrome; following chiropractic treatment, his symptoms settled over a 1-month period. He then had four further acute episodes, the most recent being due to bending over to pick up a

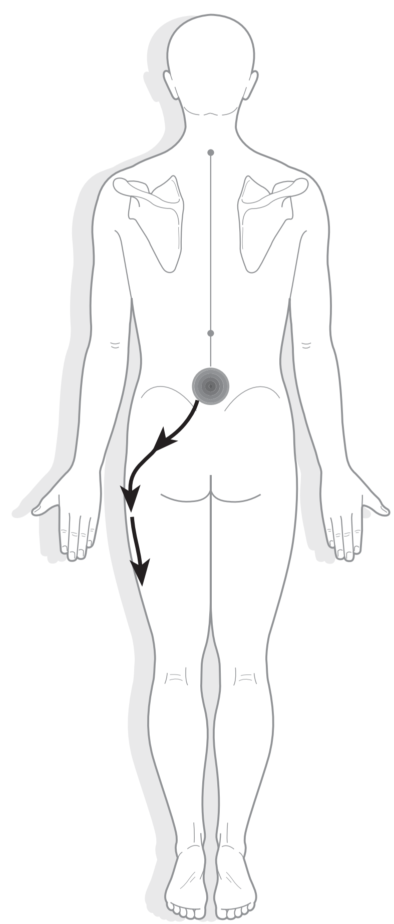

Figure 44.1

light weight at home, approximately 8 weeks ago; this caused such acute low back and left buttock and thigh pain that he had to stop working.

EXAMINATION

His blood pressure was 170/100 in the supine position with a pulse rate of 68 per minute. His chest was clear without any evidence of a wheeze. His abdomen was normal on auscultation and on palpation. On deep palpation of the paraspinal muscles he was tender over approximately the L3–L4 level of the lumbar spine. Supine SLR was limited by low back pain to 40° (left) and 60° (right). The left knee jerk was decreased. Muscle tone in both lower limbs was normal but there was a decrease in power in the left lower limb; this appeared to be due to his low back pain syndrome. The plantar response was normal as was sensation in the legs. The range of active lumbar spine movements was markedly reduced in all directions due to lower lumbar pain. The prostate was smooth but somewhat enlarged. There was normal sacral sensation and anal tone.

IMAGING REVIEW

A series of low back X-rays showed some mild disc narrowing from L2 to L5 with small marginal osteophytes and mild zygapophysial joint osteoarthrosis.

CLINICAL IMPRESSION

Possibly L3–4 or L4–5 disc protrusion.

WHAT ACTION SHOULD BE TAKEN?

In view of the history, laboratory tests were performed for chemical pathology and haematology (Boxes 44.1 and 44.2).

An MRI lumbar spine was requested concurrently and this showed a spinal canal of normal dimension with a mild disc bulge present at L4–5 and quite a large L3–4 left-sided focal disc protrusion outside the spinal canal in a far lateral position, i.e. partly extra-foraminally (see Fig. 44.2A and B). This protrusion displaced the left L3 nerve root as shown in the axial T1-weighted view (Fig. 44.2B). See also Figure 44.3.

Box 44.1 Chemical pathology

		Units	Reference range
Sodium	141	mmol/l	(135–148)
Potassium	4.8	mmol/l	(3.5–5.0)
Chloride	109	mmol/l	(95–109)
Carbon dioxide	31	mmol/l	(23–32)
Urea	5.9	mmol/l	(2.5–7.5)
Creatinine	0.10	mmol/l	(0.04–0.12)
Prostatic specific antigen	1.8	ng/ml	(0.0–4.0)

Box 44.2 Haematology

		Units	Reference range
Haemoglobin	175	g/l	(130–180)
Red cell count	5.69	$\times 10^{12}$/l	(4.50–6.50)
Red blood cell distance width	12		(12–14)
Haematocrit	0.52		(0.40–0.54)
MCV (mean corpuscular volume)	92	fl	(75–95)
MCH (mean corpuscular haemoglobin)	30.7	pg	(27.0–32.0)
MCHC (mean corpuscular haemoglobin concentration)	333	g/l	(310–350)
Platelet count	250	$\times 10^9$/l	(150–400)
White cell count	9.1	$\times 10^9$/l	(4.0–11.0)
Neutrophils	5.5	$\times 10^9$/l	(2.0–7.5)
Lymphocytes	2.8	$\times 10^9$/l	(1.0–4.0)
Monocytes	0.5	$\times 10^9$/l	(0.0–0.8)
Eosinophils	0.4	$\times 10^9$/l	(0.0–0.4)
Basophils	0.0	$\times 10^9$/l	(0.0–0.1)
Bands	0.0	$\times 10^9$/l	
Metamyelocytes	0.0	$\times 10^9$/l	
Myelocytes	0.0	$\times 10^9$/l	
ESR	6	mm/hour	(2–10)

DIAGNOSIS

L3–4 left sided focal posterior intervertebral disc protrusion compressing the left L3 nerve root.

TREATMENT AND RESULTS

When the patient came back to hear the results of his blood test and the MRI scan he was shown the large disc protrusion at the L3–4 level. He broke down and cried, then rushed out to the waiting room and called his wife in, stating 'they have found out what is wrong with me – look a large disc protrusion! It's not in my head!'. He and his wife were delighted and he was referred for a surgical opinion and this led to decompression of the left L3 nerve root with an extended fenestration and partial facetectomy to access the lateral disc protrusion and the intervertebral foramen; a single large sequestrated fragment of nucleus pulposus compressing the nerve was removed, freeing it. His postoperative course was

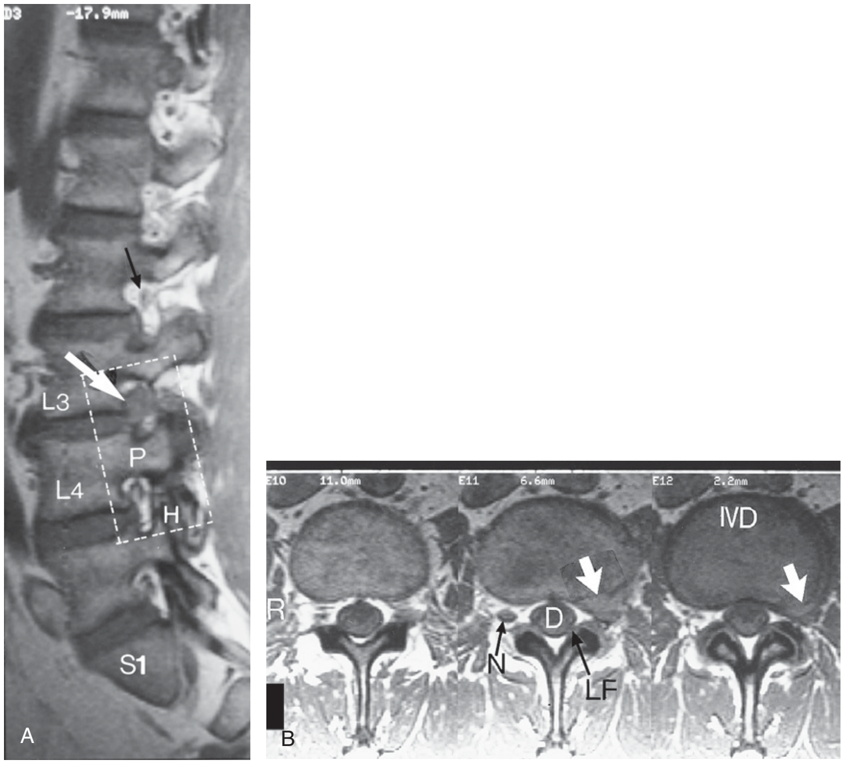

Figure 44.2 (A) Lumbosacral spine MRI parasagittal left sided T1-weighted image showing a large L3–4 disc protrusion (white arrow) projecting into the intervertebral foramen and displacing the left L3 nerve root. Note the intervertebral foramina above and below the L3–4 level that show the normal position of the nerve within the foramen (small black arrow). L3 = third lumbar vertebral body; L4 = fourth lumbar vertebral body; S1 = first sacral segment; P = pedicle of the fourth lumbar vertebra. H = hyaline articular cartilage of the zygapophysial (facet) joint between the inferior articular process of the L4 vertebra and the superior articular process of the L5 vertebra. The area shown within the rectangle is represented in a histopathology section in Fig. 44.3. (B) A series of three consecutive MRI axial T1-weighted images at the L3–4 disc level. The white arrows show the far lateral partly extra-foraminal disc protrusion at the L3–4 level that compresses the nerve in the middle and right-hand scans. The right nerve root ganglion (N) is clearly shown in the middle scan but the left neural structures are severely compressed. The dural tube (D) is shown within the spinal canal adjacent to the ligamenta flava (LF). IVD = intervertebral disc; R = right side of the patient.

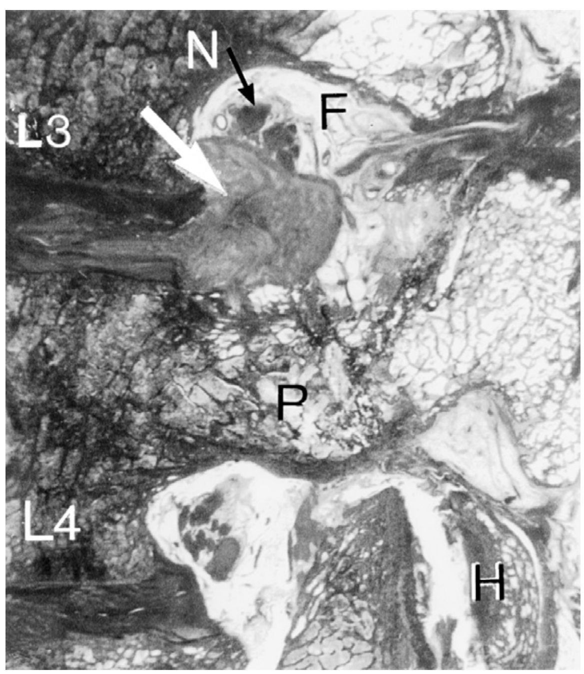

Figure 44.3 A 200-micron thick parasagittal histopathology section from a 69-year-old male postmortem specimen approximately corresponding to the rectangle in Fig. 44.2A. Note the disc protrusion (arrow) projecting into the intervertebral foramen (F) and how the neural structures (N) are being compromised. L3 = part of the third lumbar vertebral body; L4 = part of the fourth lumbar vertebral body; P = pedicle of the fourth lumbar vertebra; H = hyaline articular cartilage on the inferior articular process of the L4 vertebra. (Ehrlich's haematoxylin and light green counterstain.)

unremarkable and he was discharged on the seventh postoperative day.

At 1 month post-surgery he was doing well and he experienced good relief from his sciatica, although he still experienced some low back discomfort by the end of the day. He was advised to gradually start swimming to strengthen his back muscles and he continues to do well some 5 years postoperatively. He was extremely grateful that his condition had, at last, been taken seriously and that a proper diagnostic work-up had led to a diagnosis and subsequent treatment.

KEY POINTS

1. Never disbelieve your patient unless you have good evidence to do so.
2. A detailed history and careful physical examination, supplemented if necessary by MRI, can differentiate a herniated disc from other possible causes of similar symptoms.
3. Rely upon your own diagnostic acumen and not on that of others.

Further reading

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Case 45

Sacral fracture

COMMENT

When a patient re-presents, be careful to perform a thorough work-up leading to a current diagnosis.

PROFILE

A 67-year-old retired professional female of average build who does not smoke cigarettes or drink alcohol.

PAST HISTORY

There is a past history of intermittent low back pain during the last 20 years. This had responded well to occasional chiropractic spinal manipulation in the past. There is a history of hypertension and hypercholesterolaemia. A hysterectomy had been performed years ago.

PRESENTING COMPLAINT(S) (Fig. 45.1)

Since falling off a chair a few weeks ago, she has experienced chronic central lumbosacral pain, essentially without radiation, although occasionally there is some minor referral of pain into the right upper thigh posteriorly. Coughing and bearing down do not cause any significant increase in lumbosacral pain. Prescribed analgesics, chiropractic spinal manipulation and physiotherapy treatment had not been helpful for the current lumbosacral level pain.

AETIOLOGY

She fell off a chair when she bent to one side to pick up a light item.

EXAMINATION

In the erect posture there was no clinical evidence of leg length inequality or of pelvic obliquity. A minor left convex lumbar scoliosis was present. Active lumbar spine ranges of movement were as follows:

1. Flexion – her fingers reached to the knees, eliciting some increase in lumbosacral pain.
2. Extension – limited by approximately 25% due to an increase in lumbosacral pain.
3. Left and right lateral bending did not elicit any increase in lumbosacral pain.
4. Left and right rotation were limited by approximately 15% but were painless.

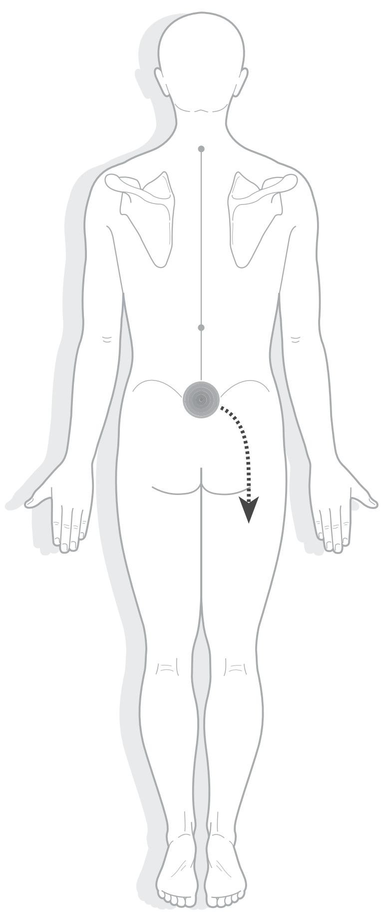

Figure 45.1

The deep tendon reflexes in the lower extremities were normal, as was pinprick sensation. Straight leg raising was limited bilaterally to 65° elevation due to hamstring tightness. There was some tenderness at the lumbosacral region on palpation and over the sacrum. Abdominal palpation elicited slight left iliac fossa discomfort but did not appear to be significant.

IMAGING REVIEW

Plain X-ray films of the lumbar spine and pelvis taken 20 years ago revealed some thinning of the L5–S1 intervertebral disc space height with slight retrolisthesis of L5 on S1. Plain X-ray films taken 10 years ago were reported as showing: 'A mild lateral curve, convex to the left, present in the lumbar spine. Mild osteoporosis. No pars defects are noted. The sacroiliac joints are within normal limits. Reasonably well maintained intervertebral disc spaces. The slight narrowing at L5–S1 may simply be developmental in origin'. However, the thinning of the L5–S1 intervertebral disc space height (Fig. 45.2) with retrolisthesis strongly suggested a posterior disc bulge or protrusion at that level. Mild osteoarthrotic changes involving the L5–S1 zygapophysial facet joints were noted.

CLINICAL IMPRESSION

Possible L5–S1 intervertebral disc midline bulge or protrusion. In view of the recent fall from a chair, a fracture in the lower lumbar spine or sacrum was considered a possibility. In order to be thorough, the possibility of an ovarian tumour was also considered in view of her now treatment resistant pain in case she had more than one condition.

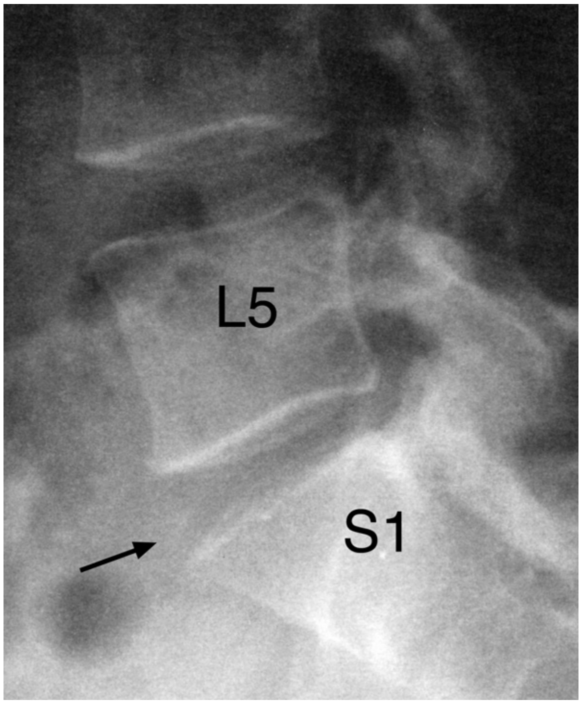

Figure 45.2 Lumbosacral spine lateral plain X-ray image. Note the thinning of the L5–S1 intervertebral disc space height (arrow) with retrolisthesis of L5 on S1 strongly suggesting a posterior disc bulge or protrusion.

WHAT ACTION SHOULD BE TAKEN?

The following tests were requested:

1. Pathology:
   a. Ovarian tumour marker – CA 125 antigen (serum) – 12 (range < 35 U/ml).
   b. Full blood examination (Box 45.1).
2. Imaging:
   a. Pelvic ultrasound. This was reported as follows: 'A hysterectomy has been performed. No free fluid or pelvic mass is detected. The urinary bladder has a normal configuration. 20 cc remains in it after voiding. The right kidney measures 92 mm and the left measures 97 mm – these are normal in size, shape and position and show no focal abnormality. No pathological lesion is seen in the right or left iliac fossa'.
   b. MRI lumbar spine. This was reported as follows: 'Desiccation of the lumbar discs, increasing caudally (Fig. 45.3). There is mild bulging of the annulus at each level from L2 to S1. There is a posterior mid-line annular tear at L3-4, L4–5 and L5–S1 with shallow focal posterior annular bulging at L4–5 (Fig. 45.4) and at L5–S1 (Fig. 45.5). There is no significant encroachment on the spinal canal or foramina throughout. There is no evidence of extrinsic impingement on the cauda equina

Box 45.1 Full blood examination

		Results	Normal range	Units
Haemoglobin		148	(115–155)	g/l
RCC		4.70	(3.80–5.30)	$\times 10^{12}$/l
PCV		0.440	(0.340–0.450)	
MCHC		339	(320–360)	g/l
MCV		92	(82–99)	fL
MCH		31.40	(27.00–34.00)	pg
RDW		12	(<16)	%
ESR		22	(1–30)	mm/h
Platelets		206	(140–400)	$\times 10^9$/l
White cell count		**11.9**	(0.4–11.0)	$\times 10^9$/l
Neutrophils	84%	**10.0**	(1.8–7.5)	$\times 10^9$/l
Lymphocytes	9%	1.1	(1.0–4.0)	$\times 10^9$/l
Monocytes	6%	0.7	(0.1–1.2)	$\times 10^9$/l
Eosinophils	1%	0.1	(0.0–0.6)	$\times 10^9$/l

FILM: Red cell and platelet morphology is unremarkable. Several neutrophils show a slight 'left shift' (indicating a slight infection is in progress) or nerve roots. Marrow signal is normal. There is no significant facet arthropathy. No lesion is seen in the lower spinal cord or in the paraspinal soft tissues'. However, on reviewing the MRI images (Fig. 45.3) there is a line through the upper one-third portion of the third sacral segment, so a Dynamic Localized Bone Scan with SPECT was requested.

c. The bone scan was reported as follows: 'The initial blood pool images of the lumbosacral spine and pelvis show no abnormal vascularity. Delayed planar and tomographic images show a mild lumbar scoliosis convex to the left. There is mildly increased uptake within the left L2–3 facet at the apex of the scoliosis (Fig. 45.6). No focal increased uptake is noted within the lumbar facet joints. There is mild curvilinear uptake across the mid-sacrum at the level of the inferior sacro-iliac joints' (Fig. 45.6) and uptake is also seen at this level on the lateral view (Fig. 45.7). 'Conclusion: (1) There is mild focal active facet arthropathy on the left at L2–3. (2) The curvilinear activity extending across the mid-sacrum at the level of the inferior sacro-iliac joints is an unusual appearance. This may be due to degenerative change or possibly a previous insufficiency fracture'.

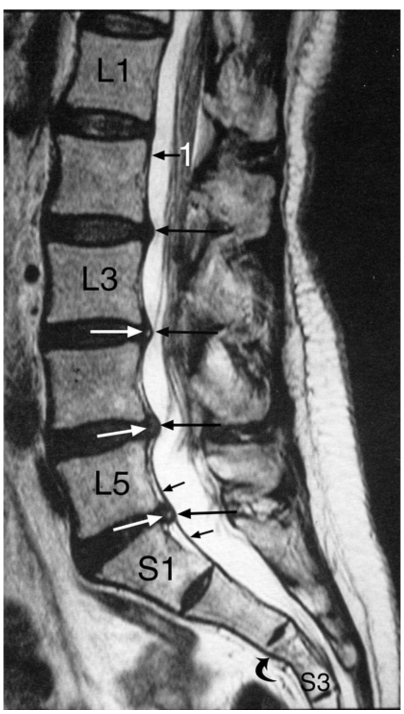

Figure 45.3 Lumbar spine MRI sagittal T2-weighted image showing desiccation of the lumbar discs, increasing caudally. 1 = posterior longitudinal ligament. There is mild bulging of the annulus at each level from L2 to S1 (long black arrows). There is a posterior mid-line annular tear at L3–4, L4–5 and L5–S1 seen as a high intensity zone (white arrows). The anterior surface of the dural tube is indicated by short black arrows. Note the line through the upper one-third portion of the third sacral segment (S3) (curved black arrow).

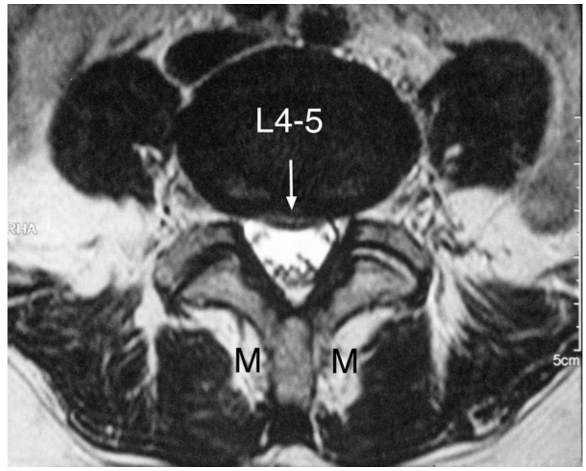

Figure 45.4 MRI axial T2-weighted image at the L4–5 level showing the shallow focal posterior annular bulge (arrow). M = multifidus muscle with fibro-fatty replacement of part of the muscle.

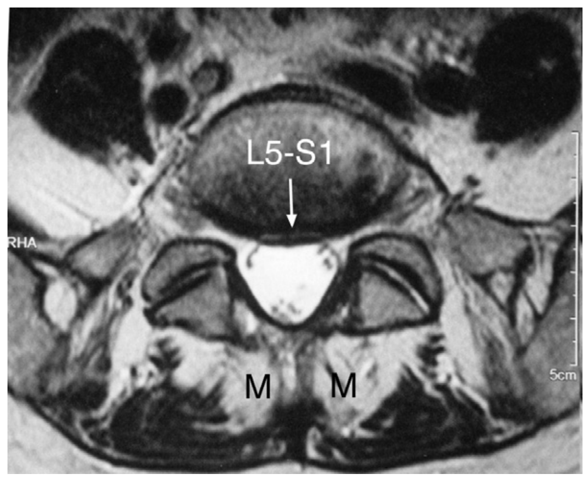

Figure 45.5 MRI axial T2-weighted image at the L5–S1 level showing the shallow focal posterior annular bulge (arrow). M = multifidus muscle with fibro-fatty replacement of part of the muscle.

DIAGNOSIS

1. Insufficiency fracture of the third sacral segment.
2. Chronic degenerative intervertebral disc changes of desiccation with mild bulging posteriorly of the last four intervertebral discs.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms

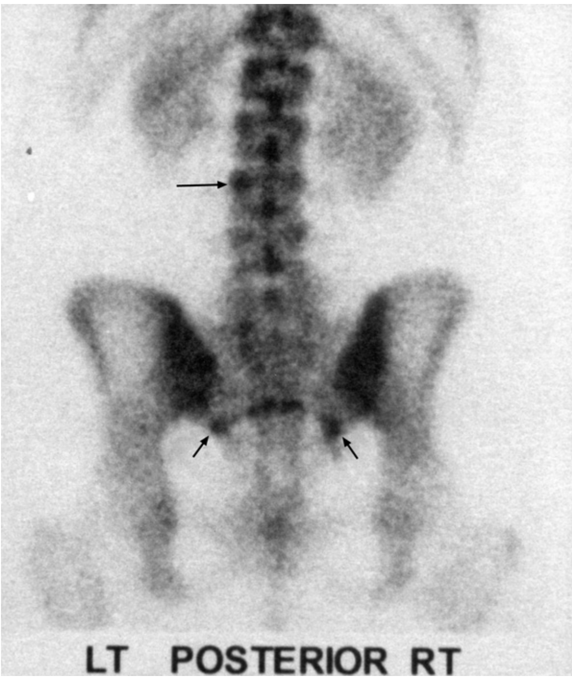

Figure 45.6 Dynamic Localized Bone Scan with SPECT image. Note the mildly increased uptake within the left L3 facet (long arrow) and the mild curvilinear uptake extending across the mid-sacrum (short arrows) at the level of the inferior sacroiliac joints.

was understood. Treatment with non-steroidal anti-inflammatory and analgesic medication on an 'as required basis' with Pilates exercises (within tolerance levels so as not to aggravate her condition), coupled with walking, provided relief from her symptoms over a period of 4 weeks. She has to be careful with her lumbosacral so as not to aggravate it as she experiences periodic episodes of mild lumbosacral pain. However, she is able to lead a normal active life as long as she performs activities within her limits of tolerance.

Note

In cases where a sacral fracture or a tumour of a sacral nerve is suspected of being the cause of radicular symptoms, an MRI of the sacrum, including coronal slices through the sacrum, should be considered in order to view the anatomy of sacral nerves in greater detail. In order to illustrate this point, an example of an axial slice (Fig. 45.8) and a coronal slice (Fig. 45.9) through the sacrum of a male from such a study are shown. In the axial image (Fig. 45.8), the extent of the visualized nerves is limited to each image level, whereas with an appropriate coronal image much more neural anatomy can be seen (Fig. 45.9).

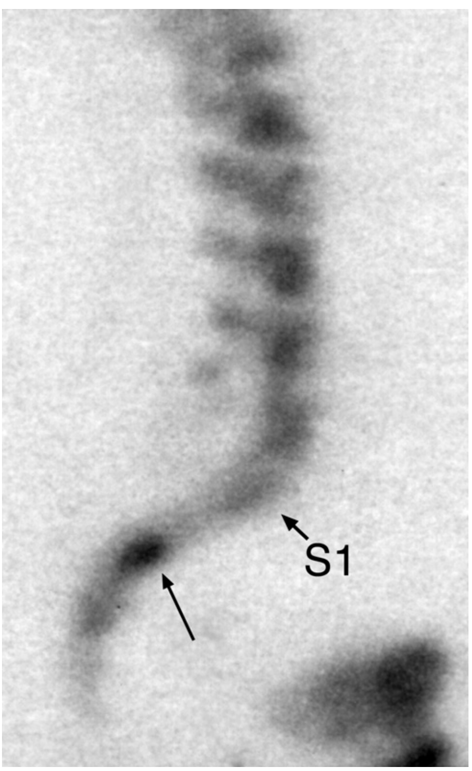

Figure 45.7 Lateral lumbosacral spine Dynamic Localized Bone Scan with SPECT image. Note the mildly increased uptake extending across the mid-sacrum at the level of the inferior sacroiliac joints (arrow). S1 = first sacral segment.

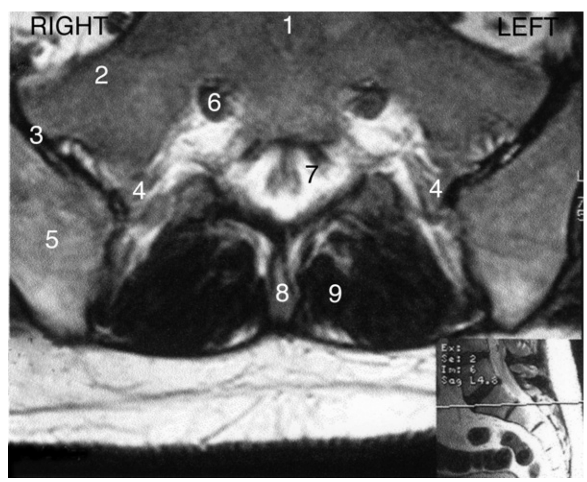

Figure 45.8 MRI axial image through the first sacral segment (see insert for orientation) illustrating the sacral nerves at this level. 1 = body of first sacral vertebra; 2 = ala of sacrum; 3 = sacroiliac joint; 4 = posterior sacroiliac ligament; 5 = ilium; 6 = first sacral nerve within the first sacral foramen; 7 = sacral canal; 8 = median crest of sacrum; 9 = multifidus muscle.

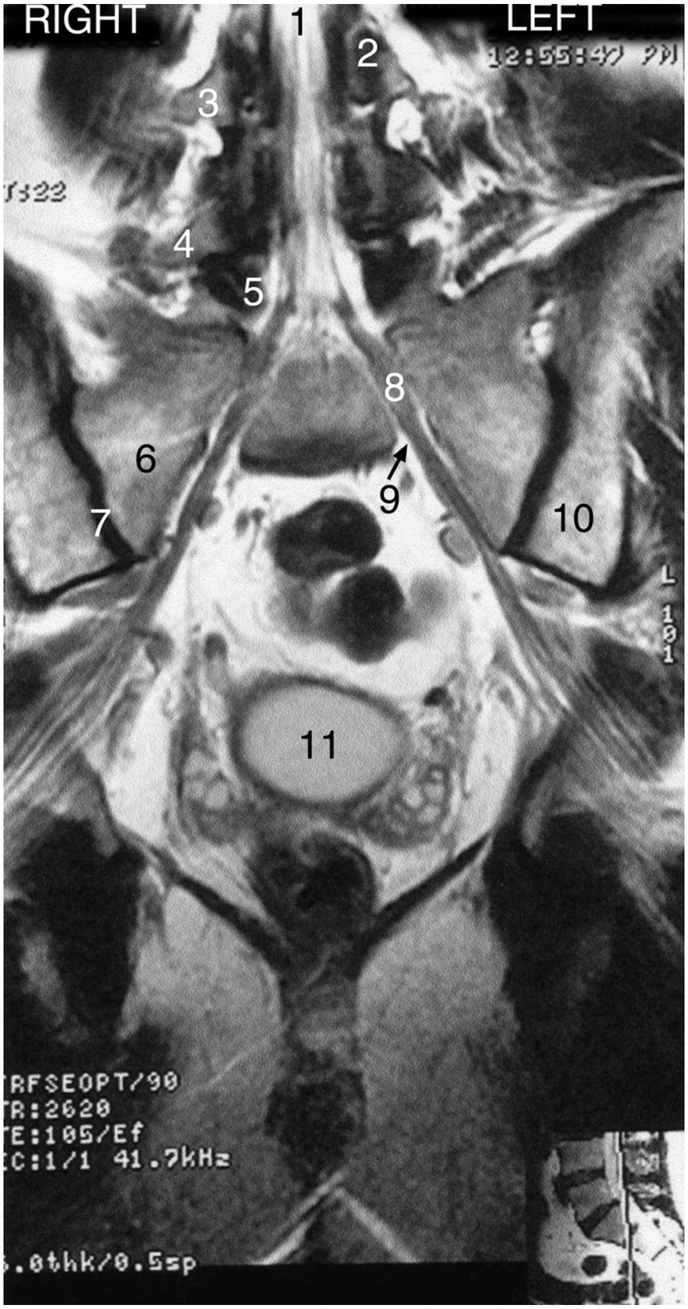

Figure 45.9 MRI coronal image essentially through the first sacral segment (see insert for orientation) to illustrate the first sacral nerves passing through the first anterior (pelvic) sacral foramina into the pelvis. 1 = spinal canal containing the cauda equina; 2 = pedicle of L4 vertebra; 3 = transverse process of L4 vertebra; 4 = transverse process of L5 vertebra; 5 = zygapophysial joint; 6 = ala of sacrum; 7 = sacroiliac joint; 8 = first sacral nerve; 9 = first anterior (pelvic) sacral foramen containing the first sacral nerve; 10 = ilium; 11 = urinary bladder.

KEY POINTS

1. This case illustrates the point that clinicians must obtain updated information on re-presenting patients' symptoms as a current evaluation may lead to a new diagnosis.
2. An insufficiency fracture of the sacrum may be suspected clinically from the presence of marked local tenderness, and later, the appearance of an ecchymosis. In the absence of complications no special treatment is required. In the rare instances in which fragments are markedly displaced there is a risk of injury to the cauda equina (Adams 1968).

Reference

Adams J C 1968 Outline of fractures, 5th edn. E&S Livingstone, Edinburgh.

Further reading

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Case 46

Low back pain and left sided sciatica

COMMENT

Patients with a history of malignancy should have a diagnostic work-up to specifically eliminate any possible 'red flag' conditions.

PROFILE

A 42-year-old housewife of average weight who does not smoke cigarettes or drink alcohol.

PAST HISTORY

Two years ago she underwent surgery to remove a left ovarian serous cyst and adenocarcinoma. Her gynaecologist and oncologist had arranged for her to have tumour marker blood tests, i.e. CA 125 (for ovarian cancer) and CASA (Cancer Associated Serum Antigen) a few months previously. The combination of these two markers can improve the detection of patients with suspected cancer (Sehouli et al 2003).

PRESENTING COMPLAINT(S) (Fig. 46.1)

She was referred by her general medical practitioner for investigation of her low back pain syndrome of unknown aetiology. The low back pain radiates down the left leg posteriorly to the lower calf and began approximately 5 years ago. Her symptoms have become worse during the last 2 months, especially during menstruation and when she performs manual activities such as vacuuming.

Coughing or sneezing do not increase her pain. She has normal bowel and bladder function and there is no night pain. Her low back is somewhat stiff in the morning.

AETIOLOGY

The aetiology is unknown.

EXAMINATION

There was no clinical evidence of leg length inequality, pelvic obliquity or scoliosis. Deep palpation of the lumbar paraspinal muscles elicited tenderness at the L5–S1 level and towards the left sacroiliac joint. Sacroiliac joint stress testing, deep tendon reflexes, pinprick sensation and power in the upper and lower extremities were normal. Lumbar spine active ranges of

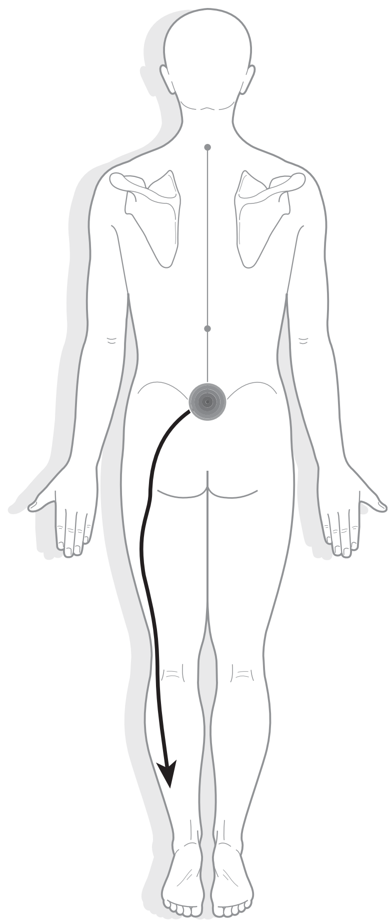

Figure 46.1

movement were normal, apart from flexion that caused an increase in her low back and left leg pains when her fingers reached her knees. Supine straight leg raising was to 90° bilaterally before she felt an increase in low back pain. Bearing down caused an increase in low back pain. The abdomen was normal on examination.

IMAGING REVIEW

Plain X-ray films of the pelvis and lumbar spine were reported as being normal.

CLINICAL IMPRESSION

A possible left sided L4–5 or L5–S1 intervertebral disc bulge or protrusion.

A differential diagnosis of a metastatic lesion.

WHAT ACTION SHOULD BE TAKEN?

The following tests were performed as a precaution in view of her history of malignancy and to obtain current results:

1. Laboratory tests:
   a. Full blood count – reported as normal.
   b. ESR – reported as normal.
   c. Tumour markers CA125 and CASA – reported as normal.
2. An abdominal ultrasound scan – reported as 'normal'.
3. A whole-body bone scan. The report stated:
   a. 'Discrete focus of increased tracer uptake in the distal left femoral shaft (Fig. 46.2) suspicious of a metastatic deposit. Correlation with plain films recommended'.
   b. 'Probable fracture of the right 9th rib. Plain film correlation is recommended'.
   c. 'No abnormality is demonstrated in the lumbosacral spine to account for the low back pain'.

In view of the bone scan results, plain X-ray films of the ribs and left femur were taken. The report stated:

a. 'No abnormality is seen in the right 9th rib'.
b. The left femur (Fig. 46.3) shows a 'fairly well defined irregular area of sclerotic bone change with a very faint surrounding halo of decreased attenuation without any endosteal scalloping or periosteal new bone formation or bone destruction. The relatively benign appearance is consistent with a chondroma. However, it is not possible to exclude a well differentiated chondrosarcoma'.

A second radiological opinion was obtained and the diagnosis was that of a benign chondroma.

A lumbar spine MRI was then requested to further investigate the low back and left leg pains; the report stated: 'There is generalized loss of signal from the lumbar discs in keeping with disc degeneration. There is a prominent posterior left central L4–5 disc protrusion (Figs 46.4

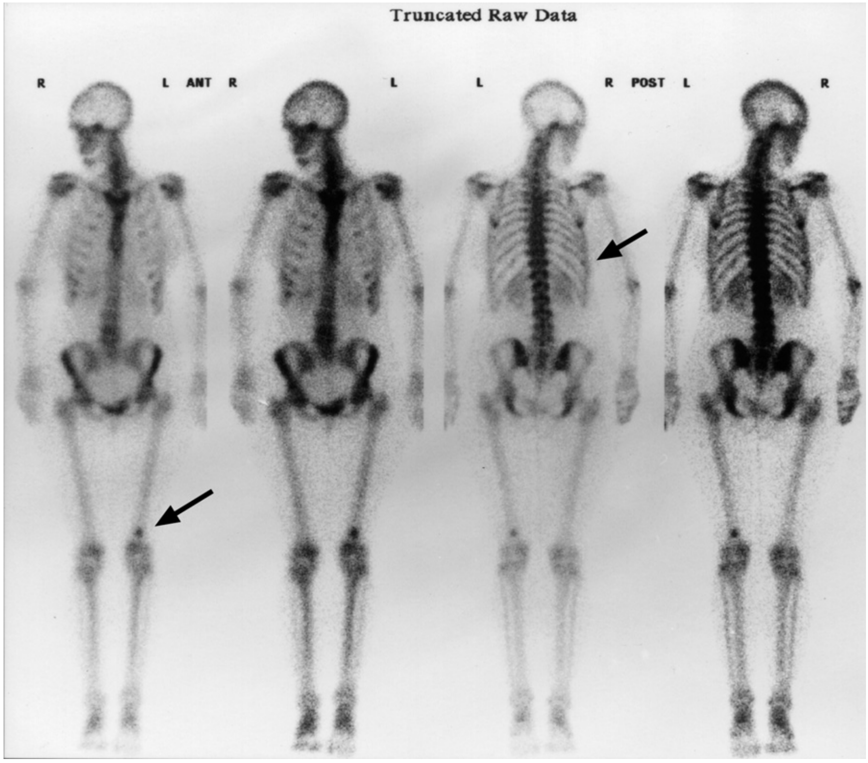

Figure 46.2 Whole-body bone scan. Note the discrete focus of increased tracer uptake in the distal left femoral shaft suspicious of a metastatic deposit (arrow) and the probable fracture of the right 9th rib (arrow).

Figure 46.3 Part of left femur plain X-ray image. Note the well defined irregular area of sclerotic bone change with a very faint surrounding halo of decreased attenuation without any endosteal scalloping or periosteal new bone consistent with a chondroma.

Figure 46.4 Lumbar MRI left paracentral T1-weighted image showing the L4–5 disc protrusion (arrows).

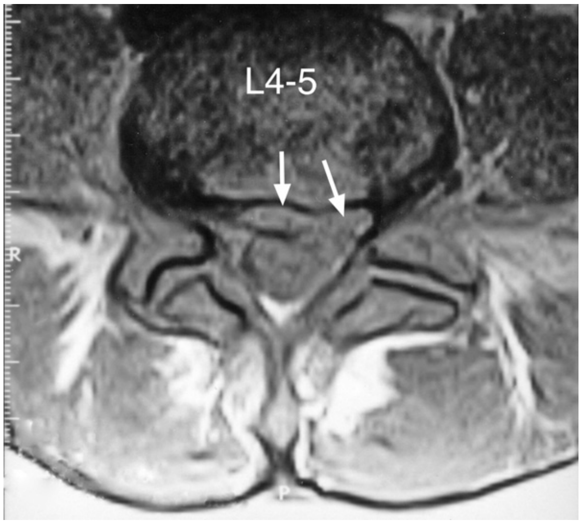

Figure 46.5 MRI axial T1-weighted image showing the L4–5 prominent central to left sided disc protrusion (arrows).

and 46.5). Disc material extends into the lateral recess at this level and is likely to impinge upon the left L5 nerve root from this region (Fig. 46.5). The canal is adequate at all levels. The intervertebral foramina appear adequate'.

DIAGNOSIS

Prominent posterior left central L4–5 intervertebral disc protrusion impinging, in all likelihood, upon the left L5 nerve root.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was advised to try six gentle chiropractic spinal mobilizations for her low back and left leg pains and this provided good symptomatic relief. Treatment was then to be provided on an 'as needed basis'.

As a precaution, the bone scan, laboratory tests and left femur plain X-ray films were repeated at 6 and 12 month intervals, respectively, by her general medical practitioner, but no change was reported in any of these investigations.

The patient was satisfied with the treatment approach of returning on an as required basis for her low back pain syndrome and she was advised to continue to see her general medical practitioner who made plans for her to undergo periodic monitoring by laboratory tests over the next 5 years, 'just as a precaution', in view of her history of left ovarian adenocarcinoma.

KEY POINTS

1. Patients with a history of malignancy who present with spinal pain syndromes should be thoroughly investigated in order to identify whether spinal symptoms are due to malignancy.
2. Do not assume that a patient's symptoms are due to mechanical dysfunction of spinal joints.

References

Sehouli J, Akdogan Z, Heinze T et al 2003 Preoperative determination of CASA (Cancer Associated Serum Antigen) and CA-125 for the discrimination between benign and malignant pelvic tumor mass: a prospective study. Anticancer Res 23: 1115–1118.

Further reading

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Case 47

Low back and unilateral buttock pain

COMMENT

Clinicians who adopt a strategy of first eliminating all serious, i.e. 'red flag' conditions are less likely to miss life threatening pathology.

PROFILE

A 59-year-old female of average build who does not smoke cigarettes or drink alcohol and who is a professional in the health care system.

PAST HISTORY

A thyroidectomy at approximately 22 years of age. A 30-year history of intermittent low back pain due to lumbosacral intervertebral disc degeneration. Breast carcinoma 10 years ago that required radical mastectomy and continuing tamoxifen treatment for hormone-dependent breast carcinoma.

PRESENTING COMPLAINT(S) (Fig. 47.1)

Intermittent low back pain with right buttock and hip pain of unknown aetiology. The buttock/hip pain is associated with night pain that requires analgesic medication. There is no pain radiation to the lower limbs. Her walking distance is becoming restricted. She recently developed severe headaches, so tamoxifen medication was ceased and substituted with Femara medication (used primarily for treating certain kinds of breast cancer in post-menopausal women by reducing the total amount of oestrogen produced in the body, thus helping to starve cancer cells by depriving them of oestrogen) before a plain X-ray examination of her pelvis and right hip was requested by her general medical practitioner. This X-ray was reported as follows: 'Hip joints are symmetrically normal. The remainder of the bone, joint and soft tissue detail in relation to the pelvis, proximal femora and specifically the region of the right hip was normal'. Therefore, her medical practitioner advised her to walk as much as possible to strengthen the muscles in her low back and hip regions. She noted that her walking distance became more limited due to her right buttock/hip pain, so her general medical practitioner requested a whole-body bone scan that was reported as follows: 'The whole body bone image shows increased uptake in the right hip, right neck of femur and the medial

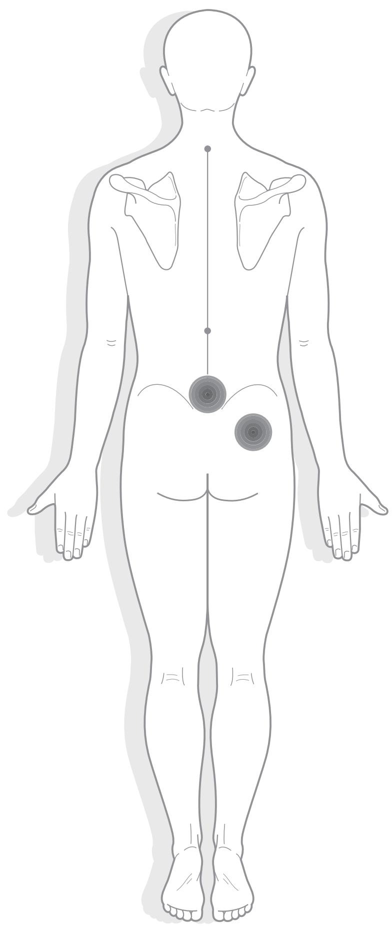

Figure 47.1

and lateral aspects of the right upper femoral shaft abutting the trochanter. There is a very mild scoliosis of the mid-thoracic region without focal features. There are no focal abnormalities to indicate the presence of metastatic deposits'. The nuclear medicine physician concluded that the bone scan changes in the right hip were 'most consistent with arthritis', that 'reflex hyperaemia could be responsible for the mild increased uptake in the right femoral shaft' and that 'the appearance there is not that of metastatic disease'.

AETIOLOGY

Unknown.

EXAMINATION

In the erect posture there was some clinical evidence of left leg length inequality, pelvic obliquity or scoliosis. Deep tendon reflexes in the lower extremities were normal as was the case with pinprick sensation and vibration sensation. Lumbar spine ranges of movement were essentially full with a complaint of low back pain on flexion and extension. Supine straight leg raising was unremarkable apart from some discomfort in the region of the right buttock/hip joint. The Valsalva manoeuvre was painless.

IMAGING REVIEW

On review, the pelvis anteroposterior view showed a large osteolytic lesion in the right ilium adjacent to the right hip joint (Fig. 47.2). The whole body bone scan showed a significant increase in uptake in the right ilium adjacent to the hip joint (Fig. 47.3).

CLINICAL IMPRESSION

Metastatic bone disease in the right acetabulum/ilium region.

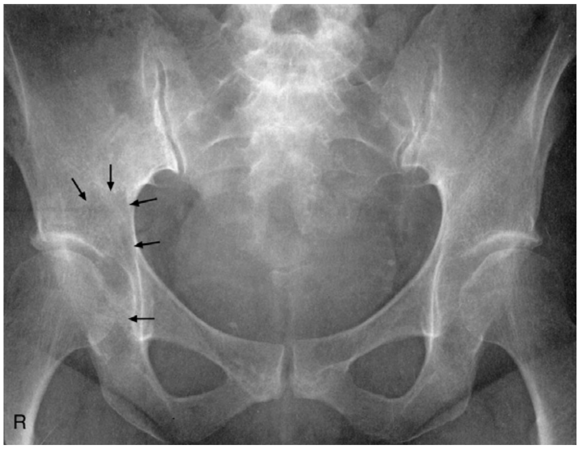

Figure 47.2 Anteroposterior supine image of the pelvis that was reported as normal. However, note the large metastatic lesion in the right acetabulum/ilium region (arrows).

Figure 47.3 Whole body bone scan that was reported as showing 'no focal abnormalities to indicate the presence of metastatic deposits'. However, note the significant increase in uptake in the right acetabulum/ilium region (arrows).

WHAT ACTION SHOULD BE TAKEN?

A CT scan of the right hip was requested (Fig. 47.4 A,B and C) and this was reported as showing 'a lytic, slightly expansile bone metastasis occupying the postero-superior

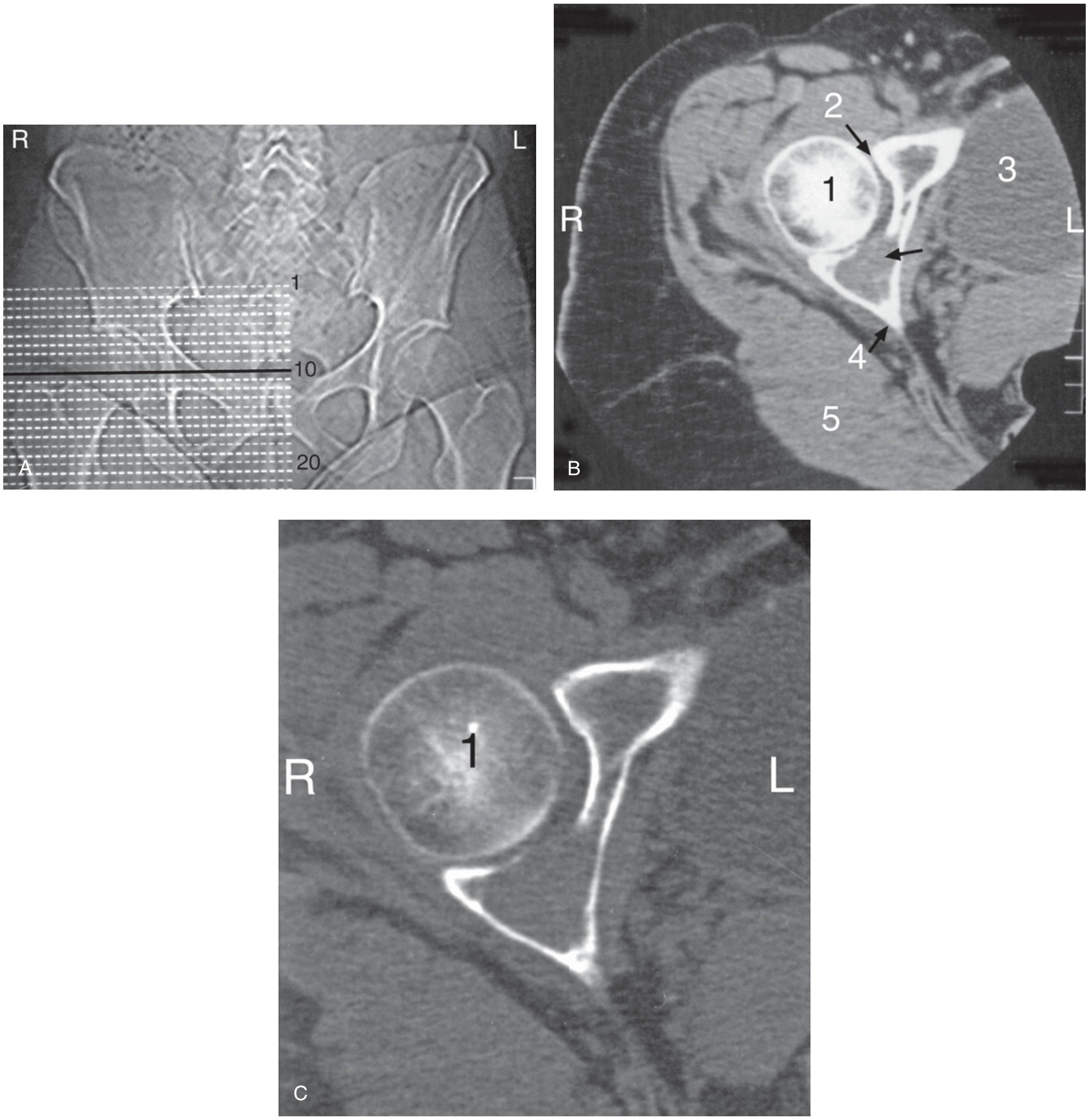

Figure 47.4 CT scout view (A) and axial images of the right hip (B = soft tissue window and C = bone window) at the level of the solid line (10) on the scout film. The axial images show part of the bony metastasis (arrow) producing a lytic defect with articular surface destruction in the right posterior acetabulum. 1 = head of femur; 2 = acetabulum; 3 = urinary bladder; 4 = ischial spine; 5 = gluteus maximus muscle; R = right side of patient.

aspect of the acetabulum. A portion of the articular surface of the acetabulum has been destroyed postero-superiorly. There is also thinning of the medial cortex of the right acetabulum/ilium. The metastasis measures approximately 4.5 cm in cranio-caudal length, 3.0 cm maximum transverse diameter and 3.0 cm anteroposterior diameter'.

DIAGNOSIS

Metastatic bone disease in the right acetabulum/ilium.

TREATMENT AND RESULTS

She was referred to an orthopaedic surgeon who treated the lesion surgically with replacement of the metastatic lesion with 'cement' (Fig. 47.5) and a total right hip joint replacement prosthesis. She then underwent radiotherapy treatment of the right hip/ilium region (10 treatments during a 2 week period).

Six months following surgery a follow-up right hip X-ray was performed (Fig. 47.6) to view progress since surgery and was reported as follows: 'Position and alignment following right total hip replacement has been

Figure 47.5 Plain X-ray image of the right hip post-surgery for right total hip replacement and excision of metastasis affecting the right acetabulum. The X-ray report stated: 'Position and alignment following right total hip replacement has been demonstrated; the hip appears enlocated on this single view that shows no adverse features'.

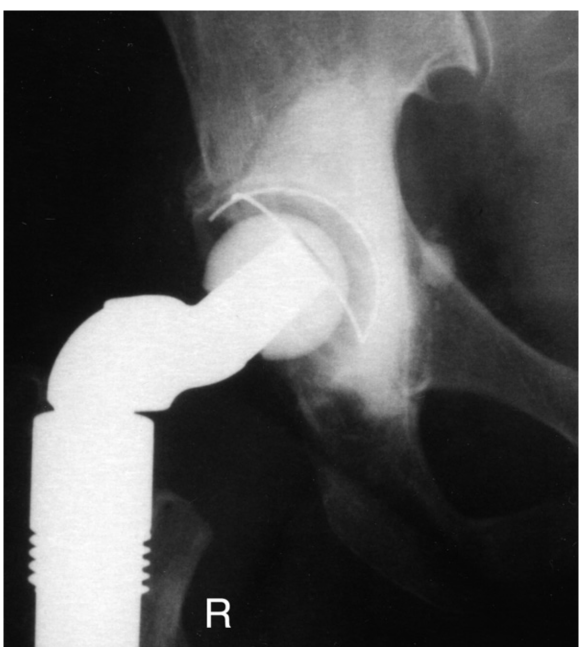

Figure 47.6 Plain X-ray image of the right hip at 6 months follow-up for right hip replacement and excision of metastasis which shows: 'The components of the right total hip prosthesis remain in satisfactory position. Cement within the acetabular fossa is unchanged and no localized bone disease can be seen around the margins of the cement'.

demonstrated; the hip appears enlocated on this single view. No adverse feature is seen'.

A follow-up CT thorax and abdomen 6 months following surgery showed: 'No evidence of pulmonary metastases; the supra-renal glands were normal as were the kidneys, spleen, pancreas and gall bladder with associated bile ducts; no lymph node enlargement or ascites identified. No structural abnormalities were detected in the pelvis'.

A follow-up whole-body bone scan 6 months following surgery showed: 'Right total hip prosthesis with reaction around the stem of the prosthesis probably related to recent surgery and persisting abnormalities in the acetabulum of the right hip consistent with the effects of surgery and possibly remnant disease but no new metastases are identified'.

A follow-up CT right hip (Fig. 47.7) and a Dynamic Localized Bone Scan with SPECT 99mTc HDP were performed 5 years following surgery because of increased pain in the right hip.

1. The CT right hip showed: 'There is some lucency at the cement-bone interface at the cortex of the right ilium (Fig. 47.7) without evidence of significant osteolysis. No definite abnormality of bone architecture to suggest metastatic involvement'.

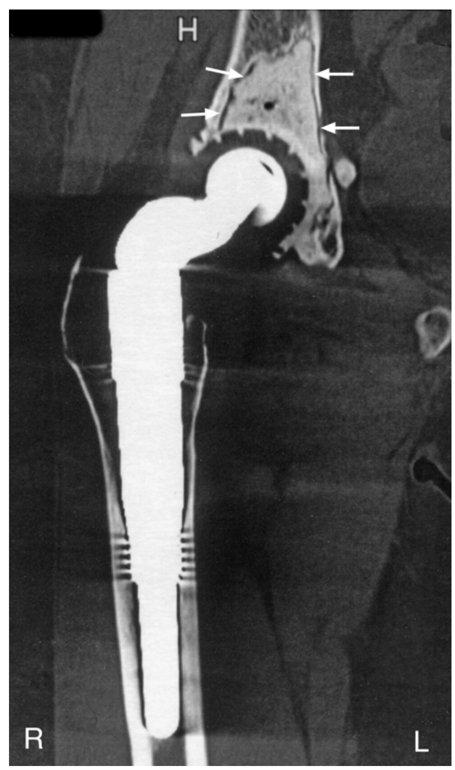

Figure 47.7 CT right hip performed 5 years following right total hip replacement and excision of metastasis. (A) Coronal image. Note the lucency at the cement-bone interface at the cortex of the right ilium (arrows) without evidence of significant osteolysis.

2. The bone scan showed: 'There is no high-grade bone turnover about the patient's right hip prosthesis to suggest new problems of metastatic disease, pathologic or traumatic fracture or significant osteolysis. Low-grade activity about the acetabular component with some focal uptake inferior to its anterior and posterior margins may be related to minor loosening or remodelling as a result of minor trauma. Low-grade diffuse activity about the femoral prosthesis is slightly greater than expected 5 years post arthroplasty but is not high grade. No definite evidence for metastasis is seen on a background of degenerative change'.

At review 5 years following her right hip surgery she still experienced some low back pain and increased right hip pain. As she had been left with a limp due to a 17 mm longer right leg (as measured by scanography) causing pelvic obliquity, she was advised to have an erect posture pelvis and lumbar spine anteroposterior X-ray examination performed to evaluate overall leg length inequality because of the possible inaccuracy associated with scanography (see Case 28).

The low back pain and hip pain are controlled to some extent by taking two paracetamol tablets (500 mg each) on arising every morning to enable her to get through the day with less pain and by using a left shoe raise.

Note

To determine bone loss on plain X-ray images requires a loss of approximately 40% of bone structure before it can be visualized. On the other hand, to determine bone loss on MRI requires a loss of approximately 2–3% of bone structure before it can be visualized. Therefore, MRI is far more sensitive.

KEY POINTS

1. This example highlights the importance of performing appropriate imaging and the necessity for treating clinicians to carefully view all imaging, even when radiology reports are available.
2. Not to personally view all imaging of one's patient is a recipe for disaster, particularly when mechanical intervention, such as manipulation, is considered for relief of low back pain in view of reportedly 'normal' plain X-ray and whole body bone scan reports.

Further reading

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Kuhl C K, Schrading S, Bieling H B et al 2007 MRI for diagnosis of pure ductal carcinoma in situ: a prospective observational study. Lancet 370: 485–492.

Shehadi J A, Sciubba D M, Suk I et al 2007 Surgical treatment strategies and outcome in patients with breast cancer metastatic to the spine: a review of 87 patients. Eur Spine J 16: 1179–1192.

Case 48

L4–5 and L5–S1 degenerative intervertebral disc replacement with Charité artificial discs

COMMENT

In carefully selected cases, Charité disc prostheses can be very helpful.

PROFILE

A 46-year-old female secretary of average build who does not smoke cigarettes and only drinks alcohol socially.

PAST HISTORY

Approximately 6 years ago she had bent forward to lift a heavy object followed by turning her torso to one side, with the result that she experienced intense low back pain radiating to both buttocks then to the thighs posteriorly. Her medical practitioner had advised her to rest and take a narcotic analgesic for several days before seeing him again; at that time she was advised to rest periodically and to gradually mobilize. This approach was of benefit to her but she had to take part in some activities, such as bending forward, that aggravated her low back and leg symptoms, so she had physiotherapy treatment. This gave some relief due to gentle mobilization and massage. However, her condition continued with various degrees of intensity, becoming almost constant during the last 3 months before consultation. Non-steroidal anti-inflammatory medication had caused gastritis so she stopped taking it and relied on narcotic analgesia, although this only 'dulled' the pain. She is aware of low back and left leg symptoms being aggravated when she coughs, sneezes or bears down. A CT lumbar spine and an MRI lumbar spine had been performed as noted under Imaging.

PRESENTING COMPLAINT(S) (Fig. 48.1)

Almost constant low back pain that radiates to the left buttock then to the posterolateral side of her left leg as far as the fourth and fifth toes of her foot. Sometimes there is associated 'numbness' on the lateral aspect of her left calf.

AETIOLOGY

Lifting a heavy object, followed by twisting, 6 years ago.

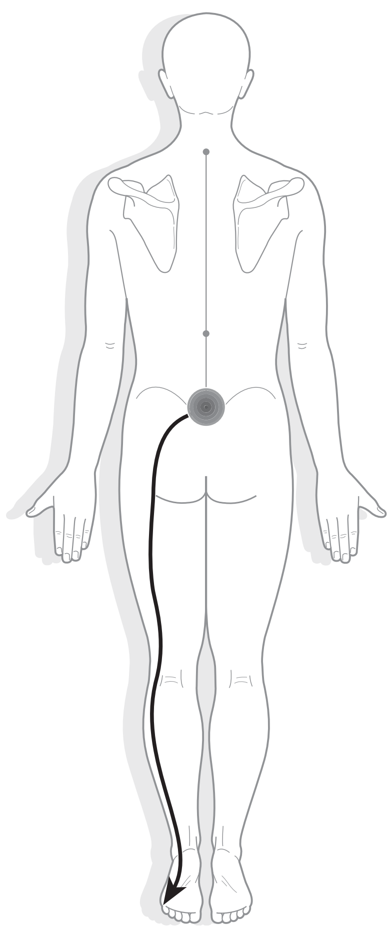

Figure 48.1

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity, leg length inequality or scoliosis. Percussion of the lumbar spine was painless. Deep palpation of the paraspinal muscles elicited pain at the L4–S1 levels.

The deep tendon reflexes at the knees (L4) were brisk and equal but the left Achilles reflex (S1) was absent. The plantar response was normal. The temperature of the feet was equal on comparison by palpation. The foot pulses were normal. Vibration sensation at the left and right ankles was normal. Power was normal in the legs and feet, apart from some left sided weakness (4/5) on foot dorsiflexion (L4 and L5), foot eversion (4/5) (S1), and extension of the big toe (3/5) (L5). Toe walking power (S1) was normal but heel walking power (L5) was diminished on the left side. The circumference of the calves, 8 cm below the patella, was 27 cm bilaterally. In the seated position, slumping forwards caused some low back pain and left lower limb sciatica; addition of left straight leg raising exacerbated the low back pain and left sciatica. Supine straight leg raising was limited to a measured 25° (left) due to low back pain and sciatica and to 45° (right) due to low back pain. Bilateral hip flexion elicited an increase in low back pain.

Lumbar spine active ranges of movement were as follows:

1. Flexion – limited by approximately 80% due to low back pain and left sciatica.
2. Extension – limited by approximately 50% due to low back pain.
3. Lateral bending – full range and painless bilaterally.
4. Rotation – full range and painless bilaterally.

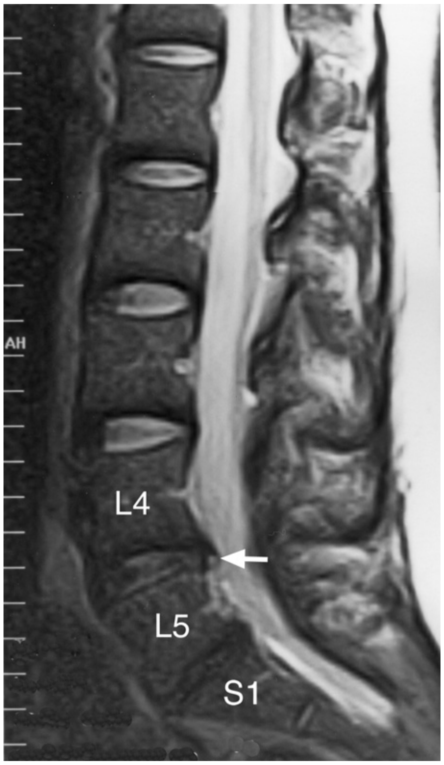

Figure 48.2 Lumbar spine MRI sagittal T2-weighted image showing loss of signal at the L4–5 and L5–S1 intervertebral disc levels in keeping with disc desiccation. There are minimal bulges of these discs with adequate spinal canal dimensions. Note the high intensity zone in the posterior bulge of the L4–5 intervertebral disc (arrow).

IMAGING REVIEW

A CT lumbar spine performed 1.5 years following her injury was reported as follows: 'L3–4 level there is a normal disc with minimal zygapophysial joint degenerative change'. The L4–5 disc level shows a 'large generalised disc bulge with a smaller central protrusion pressing upon the thecal sac causing moderate narrowing of the spinal canal'. The L5–S1 disc level shows 'evidence of disc degeneration with gas within the disc space'.

The MRI lumbar spine performed 1 month following her CT examination was reported as follows: 'Loss of signal is noted at the L4–5 and L5–S1 intervertebral disc levels in keeping with disc desiccation (Fig. 48.2), There are minimal bulges of these discs with adequate spinal canal dimensions. In particular there is no compressive lesion of the left L5 nerve root'. However, no mention was made of (1) the high intensity zone in the L4–5 intervertebral disc bulge (Fig. 48.2), and (2) the intervertebral disc material seen in the L5–S1 level left lateral recess (Fig. 48.3) compromising the adjacent nerve root.

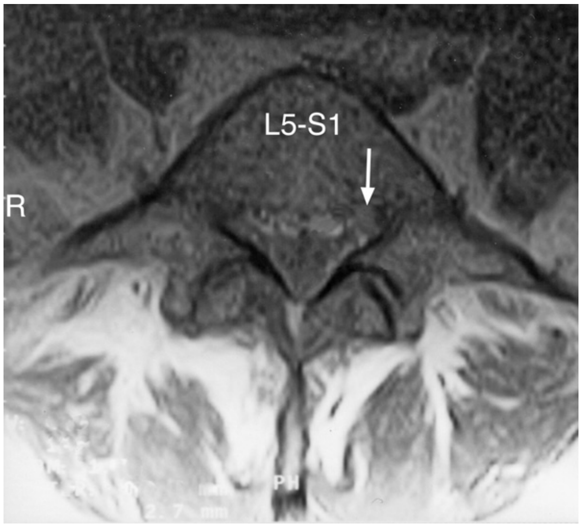

Figure 48.3 MRI axial T1-weighted image at the L5–S1 level. Note the disc material in the left lateral recess (arrow) compromising the adjacent left nerve root.

CLINICAL IMPRESSION

Central to left sided intervertebral disc protrusion at L5–S1, with a L4–5 intervertebral disc tear.

WHAT ACTION SHOULD BE TAKEN?

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. A neurosurgical referral was then made at which time the patient was advised that a further MRI should be undertaken in 1 year because of her persisting symptoms. That MRI reported 'some desiccation at the L4–5 and L5–S1 intervertebral disc levels with a mild posterior central disc bulge at L4–5 and an L5–S1 mild left parasagittal posterior disc bulge causing slight anteroposterior narrowing of the spinal canal' (Fig. 48.4).

DIAGNOSIS

- L5–S1 posterior paracentral intervertebral disc bulge compromising the left L5–S1 nerve root.
- L4–5 posterior intervertebral disc bulge.

Figure 48.4 Lumbar spine MRI central to left parasagittal T1-weighted image showing a mild central posterior intervertebral disc bulge at L4–5 and a left sided posterior paracentral intervertebral disc bulge (arrow) at the L5–S1 level.

TREATMENT AND RESULTS

The neurosurgeon told the patient that surgery was not recommended.

Approximately 4 years later, due to her ongoing and worsening symptoms, an orthopaedic spine surgeon was consulted and, based on the history, ongoing symptoms of chronic low back pain with left buttock and leg symptoms and a continuing lack of improvement with medication, manual therapy and acupuncture, as well as an updated lumbar MRI scan, a Charité artificial disc arthroplasty was performed at both the L4–5 and L5–S1 levels (Fig. 48.5).

Following recovery from surgery, the patient noticed a dramatic improvement in her low back, left buttock and left leg symptoms. Over a period of some months she continued to improve, became asymptomatic, had a full and painless range of active lumbar spine movements, and full and painless straight leg raising bilaterally.

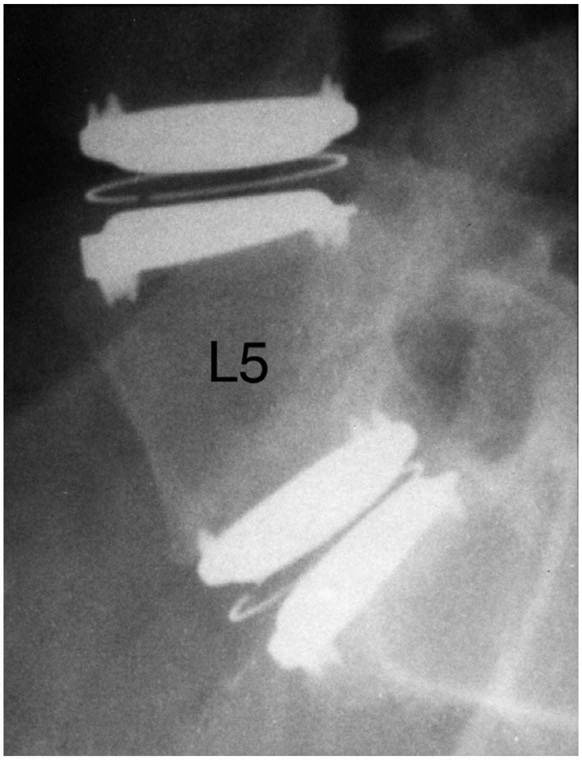

Figure 48.5 Lateral lumbosacral spine image showing the Charité artificial disc prostheses at both the L4–5 and L5–S1 levels.

KEY POINT

Carefully selected patients can respond very well to appropriate surgery involving the use of Charité artificial disc prostheses.

Further reading

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Serhan H A, Dooris A P, Parsons M L, Ares P J, Gabriel S M 2006 *In Vitro* wear assessment of the Charité artificial disc according to ASTM recommendations. Spine 31: 1900–1910.

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Introduction

Before presenting the cervical spine cases it is important to consider the following summary of some possible causes of cervical spine pain with or without radiculopathy (Table v).

Table vi summarizes some possible causes of pain referred into the arm from the neck and thorax. In addition, pain arising in the brachial plexus and peripheral nerves is included, as also are lesions at the shoulder, elbow, wrist and hand, which specifically or characteristically affect the upper limb (Keat 1996).

PHYSICAL EXAMINATION

The physical examination should be orderly and systematic and should include the following cervical spine examinations, as indicated by the patient's presenting complaint (s) (Table vii).

It is essential to remember that the signs and symptoms caused by a herniated cervical disc depend on the location of the herniation:

Table v Some possible causes of cervical spine pain

Acute spinal pain
Febrile disorders
Injury

Chronic spinal pain

1. **Traumatic, mechanical or degenerative:**
   Cervical spine strain; 'whiplash'
   Injuries of bone, joint, intervertebral disc or ligaments; clay shoveller's avulsion fracture of C6 or C7 spinous process
   Degenerative or traumatic changes of the spine (osteoarthrosis; spondylosis)
   Cervical spine instability syndromes, e.g. spondylolisthesis
   Scoliosis: primary and secondary
   Spinal or intervertebral canal stenosis
   Cervical rib
   Upper brachial plexus avulsion
2. **Joint dysfunction:**
   Zygapophysial
   Intervertebral disc
3. **Metabolic:**
   Osteoporosis
   Osteomalacia
   Hyper- and hypo-parathyroidism
   Ochronosis
   Fluorosis
4. **Unknown causes:**
   Inflammatory arthropathies of the spine, such as ankylosing spondylitis and the spondylitis of Reiter's (Brodie's) disease, psoriasis, rheumatoid arthritis, polymyalgia rheumatica
5. **Infective conditions of bone, joint and theca of the spine:**
   Osteomyelitis
   Tuberculosis
   Melioidosis
   Undulant fever (abortus and melitensis)
   Typhoid and paratyphoid fever and other *Salmonella* infections
   Syphilis
   Yaws
   Very rarely, Weil's disease (leptospirosis icterohaemorrhagica)
   Spinal pachymeningitis
   Chronic meningitis
   Subarachnoid or spinal abscess
   Herpes zoster
   Cervical adenitis
   Poliomyelitis
   Tetanus
   Febrile states – meningism
   Malaria
6. **Psychogenic:**
   Anxiety
   Depression
   Hysteria
   'Compensation neurosis'
   Malingering

table continues

Table v Some possible causes of cervical spine pain—Cont'd

7. **Neoplastic – benign or malignant, primary or secondary:**
   Osteoid osteoma
   Eosinophilic granuloma
   Metastatic carcinomatosis
   Bronchial carcinoma
   Oesophageal carcinoma
   Sarcoma
   Myeloma
   Primary and secondary tumours of spinal canal and nerve roots: ependymoma; neurofibroma; glioma; angioma; meningioma; lipoma; rarely cordoma
   Reticuloses, e.g. Hodgkin's disease
8. **Cardiac and vascular:**
   Subarachnoid or spinal haemorrhage
   Aneurysm
   Vertebral artery tortuosity causing bony erosion
   Visceral referral – heart, oesophagus and lung carcinoma
   Arteriovenous malformations
9. **Blood disorders:**
   Acute haemolytic states

Modified from Hart (1985) and Patten (1996).

Table vi Some possible causes of arm pain specific to the neck

Lesions in the neck	**Lesions of the thorax and thoracic spine**
Disc prolapse	Cardiac ischaemia
Spondylosis	Syphilitic aortitis
Syringomyelia	Thoracic disc
Fracture dislocations	Oesophagitis
Post-herpetic neuralgia	**Lesions at the shoulder**
Radiculitis – paralytic/viral (neurologic amyotrophy)	Periarthritis/capsulitis
Spinal abscess	Subacromial bursitis
Tuberculous	Calcific tendonitis
Brucella	Bicipital tendonitis
Pyogenic	Shoulder–hand syndrome
Epidural abscess	**Lesions at the elbow**
Pachymeningitis cervicalis	Epicondylitis
Tumours	Olecranon bursitis
Spinal cord	**Lesions of the forearm, wrist and hand**
Meninges	Carpal tunnel syndrome
Nerve roots	Tenosynovitis
Vertebrae – primary, secondary	Ulnar neuritis
Metastatic carcinoma in deep cervical nodes	Trigger finger
Lesions of the brachial plexus	Algodystrophy (reflex sympathetic dystrophy, RSD)
Cervical rib	Hypertrophic osteoarthropathy
Malignant infiltration, e.g. Pancoast tumour	Pachydermoperiostitis
Costoclavicular compression	Repetitive strain injury (RSI)
Subclavian aneurysm	**Lesions which may arise from skin disease are excluded.**
Scalenus anterior syndrome	

Modified from Keat (1996) and Patten (1996).

- Figure xiii shows the clinical features of a *posterolateral* cervical disc herniation that may cause nerve root impingement.
- *Midline* herniation may cause symptoms in the arm, upper thoracic region and/or in the legs.

The motor innervation of the upper limb is shown in Figure xiv.

RADIOLOGY

As mentioned under the whiplash injuries section, when reporting on cervical spine MR imaging radiologists should not state that 'no abnormality of the craniovertebral junction is seen', unless specific craniovertebral junction imaging has been performed; the routine cervical spine MRI is not intended to detect, in detail, the ligamentous

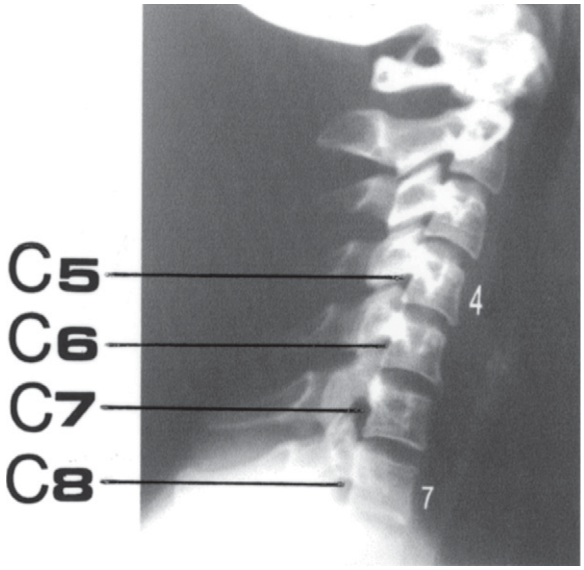
Table vii Some elements of the cervical spine physical examination

Erect posture examination	
Observe for: Fluidity of movement Body build Skin markings – café-au-lait spots Posture	*Observe gait:* Steady or unsteady
Seated	
Test cervical spine motion, with caution and bearing in mind possible injury to the vertebral arteries, for: Flexion Extension Side bending Rotation Cervical rotation plus extension	*Palpate for:* Muscle spasm Myofascial trigger points Supraspinous and interspinous ligament tenderness Adjacent muscle tenderness
Bony palpation: Anterior aspect Hyoid bone Thyroid cartilage First cricoid ring Carotid tubercle Posterior aspect Occiput Inion Superior nuchal line Mastoid processes Spinous processes of the cervical vertebrae Facet joints	*Neurological tests:* Biceps reflex Triceps reflex Brachioradialis reflex Muscle power testing Pinprick sensation upper extremities Vibration sensation at elbows *Test major peripheral nerves:* Wrist extension Thumb extension Thumb abduction Thumb pinch Apposition of thumb and index finger Deltoid
Soft tissue palpation: Lymph glands in the neck and axillary areas *Special tests:* Naffziger test* Valsalva manoeuvre* Compression test* Swallowing test* Adson's test*	*Test for upper motor neuron lesion:* Hoffmann's sign Plantar response (Babinski test) *Measure* Arm/forearm circumference bilaterally

*See Definitions and abbreviations chapter.
Adapted from Hoppenfield 1976, Mackenzie 1985, Keim & Kirkaldy-Willis 1987.

structures at the craniovertebral junction; a different MRI technique is required for this.

To investigate the craniovertebral junction and to assess the appearance of the alar ligaments, the transverse ligament, the tectorial membrane and the anterior and posterior atlanto-occipital membranes by MR imaging requires a high spatial resolution and good contrast between tissues (Krakenes et al 2001). This can be obtained by using proton-density-weighted (PDW) sequence with 2-mm-thick sections and the examination would need to be performed in three orthogonal planes with the patient's head fixed in neutral position using a standard head coil (Krakenes et al 2001). The axial sections should cover the foramen magnum to the base of the dens; coronal sections from the anterior arch of the atlas and halfway through the spinal canal, and sagittal sections from one occipital condyle to the other (Krakenes et al 2001). Also see Krakenes & Kaale's (2006) paper titled 'Magnetic resonance imaging assessment of craniovertebral ligaments and membranes after whiplash trauma'.



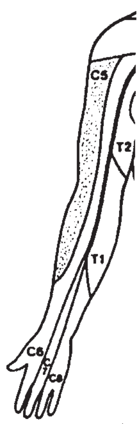
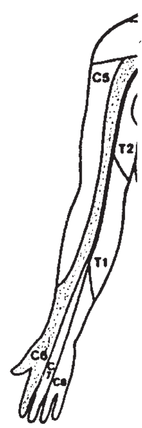
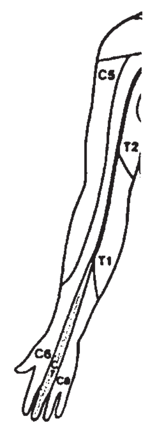
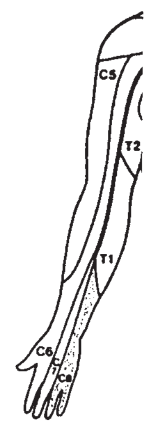
HERNIATION	C4-5	C5-6	C6-7	C7-T1
NERVE ROOT	C5	C6	C7	C8
SENSORY SUPPLY				
PAIN	Lateral arm, medial scapula	Lateral forearm ▶ thumb and index finger	Triceps, front and back of mid-forearm ▶ middle finger; Medial border of scapula	Medial forearm ▶ ring and little finger
MOTOR WEAKNESS	Deltoid Supraspinatus Infraspinatus Rhomboids	Wrist extensors (C6) Biceps (C5,6) Forearm pronators & supinators	Triceps Wrist extensors & flexors Pectoralis major Latissimus dorsi	Finger flexors Hand intrinsic muscles
SCREENING EXAM	Shoulder abduction Shoulder internal & external rotation Elbow flexion	Elbow flexion Wrist extension Forearm pronation & supination	Finger extension Elbow extension Shoulder adduction Wrist flexion	Finger flexion Thumb & forefinger pinching
REFLEXES	Biceps	Brachioradialis	Triceps	-

Figure xiii The clinical features of a posterolateral cervical disc herniation that may cause nerve root impingement. C5–C8 shows nerve root level. Adapted from Hoppenfeld (1976, 1977), Patten (1996) and Wilkinson (1986).

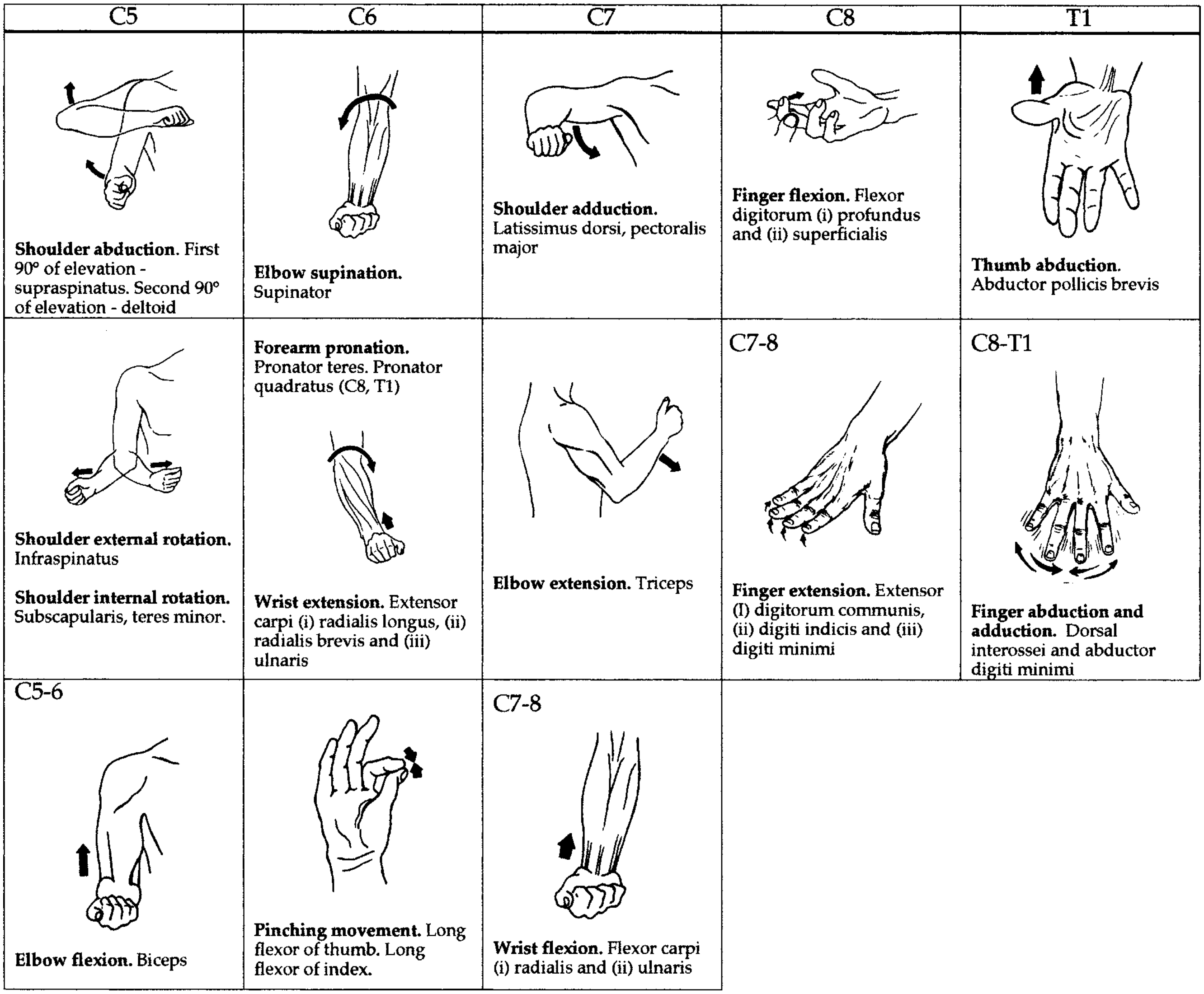


Figure xiv Motor innervation of the upper limb. Modified from Patten (1996) and Hoppenfeld (1976, 1977).

Note

When contemplating treatment of the cervical spine, it is prudent to auscultate the major arteries of the neck for bruits, and the heart for murmurs that may occur due to bacterial endocarditis vegetations on the valves, particularly in patients with a history of rheumatic fever, as dislodged vegetations in left-sided bacterial endocarditis may cause an infarct of the brain (Netter 1969).

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Case 49

Post motor vehicle accident CT versus MRI investigations

COMMENT

It is unreasonable not to investigate a patient's cervical spine symptoms with appropriate imaging until 2 years and 11 months following a serious motor vehicle accident, especially when symptoms of ongoing neck pain and headaches are present.

PROFILE

A 30-year-old full-time secretary who does not smoke and only drinks alcohol occasionally.

PAST HISTORY

Approximately 3 years ago, she was the front seat belted passenger in a vehicle involved in a two car collision at approximately 65 km per hour when a car passed through an intersection and failed to give way, causing the vehicle in which she was a passenger to hit the left side of the other vehicle, i.e. a T 'bone' type accident. During the impact, her seat moved forward, so her knees hit the dashboard. She was taken by ambulance to a hospital Accident and Emergency Department where she was reviewed.

Although she was 'aching all over', her low back was the most painful area and she also had neck pain and a 'buzzing' sensation in her head.

Medication: overall, she had been advised to take Panadiene tablets, Panadol and occasional Nurofen tablets but without significant relief.

PRESENTING COMPLAINT(S) (Fig. 49.1)

1. Constant bilateral neck pain of varying intensity. Almost daily, the neck pain radiates bilaterally to the occiput then over the head to the temporal areas causing 'pounding in the temples'. Initially, she had more right sided neck pain but currently the neck pain can extend into the left and right cervico-shoulder regions. On a good day she has a reasonable amount of neck movement, whereas on some days it is very restricted and painful. She is not aware of any upper extremity symptoms. Although she has neck pain that disturbs her sleep, there is no night pain per se.

   Sneezing, looking up or down, hanging up the washing, driving a car, using an inappropriate pillow and after being in bed overnight aggravate her symptoms. On arising in the morning, her neck is stiff and painful. Heat packs, massage and Panadeine (4–6 tablets per day; a couple of tablets are also taken before going to bed when the neck pain is really bad) temporarily

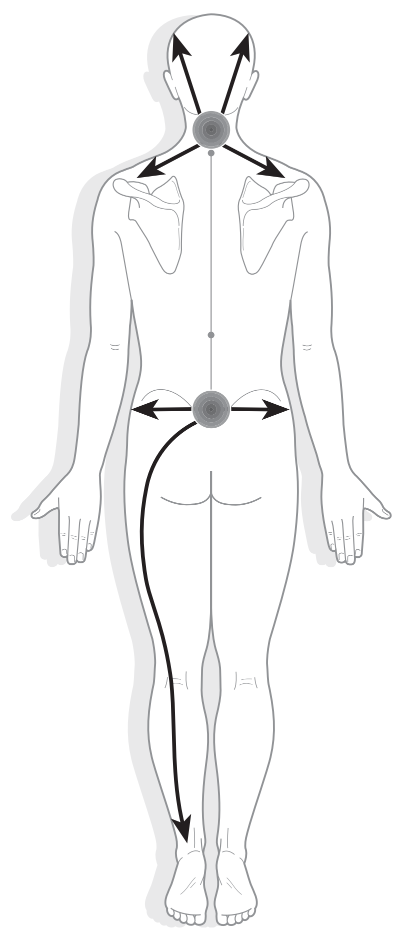

Figure 49.1

relieve her symptoms. She had tried physiotherapy treatment but it had aggravated her neck pain.

2. Low back pain felt across the low back and extending to both iliac crests. Pain radiates from the low back to the left buttock, to the thigh postero-laterally, to the calf and then to the left foot where the sole feels 'numb'. This sciatic distribution of pain also has a component of general 'numbness'. Lying down does not provide a great deal of relief. Sneezing, changing from sitting to standing posture and bending forwards, for example to change nappies, aggravate her low back pain symptoms. After being in bed over night, she experiences low back pain and stiffness until she gets up and moves about. However, daily activities cause worsening symptoms by the end of the day, i.e. the pain becomes 'progressively worse'. Heat, exercises and generally moving about temporarily relieve her symptoms.

AETIOLOGY

Motor vehicle accident approximately 3 years ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Deep palpation of the paraspinal muscles in (i) the neck elicited bilateral pain and 'numbness' from C1 to C7, with greatest pain bilaterally at the C4–6 level where there was localized muscle spasm, and in (ii) the lumbar spine elicited lumbosacral pain. Sacroiliac joint strain testing elicited some pain in the left and right sacroiliac joints, suggesting that these joints may have been injured during the accident; she also experienced some low back pain during the sacroiliac joint strain test. Toe walking power (S1) and heel walking power (L5) were normal. Deep reflexes in the upper and lower extremities were normal, apart from diminished right triceps (C7) and right knee jerk (L4) reflexes. Vibration sensation at the elbows and ankles was normal. Pinprick sensation over the upper posterior torso elicited slight hypoaesthesia in the left C4 dermatome and, in the upper extremities, there was hypoaesthesia on the lateral side of the right arm (C5), the right forearm anterolaterally (C6) and the right forearm ulna surface (T2). There appeared to be hypoaesthesia of the left middle finger (C7), the lateral aspect of the left thigh (L2), the lateral aspect of the left calf (L5) and the lateral aspect of the left foot (S1). The brachial plexus stretch test suggested the presence of pain on tractioning the left and right brachial plexuses. When seated slumped forward, this aggravated her low back pain and the addition of straight leg raising on the left and right sides elicited a 'pulling' sensation at the lumbosacral level. Motor power in the upper and lower extremities appeared to be normal, apart from some weakness (4/5) of left little finger adduction (C8/T1) and left toe extension (L5). The pulses in the feet were normal and the temperature of both feet appeared to be normal on palpation.

The circumference of the arms, forearms and calves was measured and found to be normal on comparing the left and right sides.

The blood pressure in the right arm was 120/70 in the seated position. Supine straight leg raising was to a measured 65° (left), limited by low back pain, and to a painless 70° (right). The addition of left foot dorsiflexion to straight leg raising elicited an increase in low back pain but not when testing the right side. The plantar responses were normal. Lasegue's sign elicited low back pain when testing the left side but the right side was normal. Bilateral hip flexion elicited an increase in low back pain. Bilateral hip flexion, with the addition of cervical spine flexion, elicited an increase in low back pain and mid-cervical spine pain. The Valsalva manoeuvre elicited an increase in cervical and lower lumbar spine pain. The Milgram active bilateral straight leg raise elicited low back pain. The Fabere sign was normal, i.e. there was no hip joint pain.

Cervical spine active ranges of movement were measured using a Cervical Range of Motion instrument (CROM product of Performance Attainment Associates, St Paul, MN, USA) (see Box 49.1).

Other cervical spine and cervico-shoulder region tests to check for pain of cervical spine origin are shown in Box 49.2.

Active lumbar spine ranges of movement were measured and are shown in Box 49.3.

Active thoracic spine ranges of movement were essentially normal.

Box 49.1 Cervical spine active ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	38°	Elicited pain at approximately the C3–6 level
Extension	60°	48°	Elicited pain in the sub-occipital and lower cervical spine areas
Lt lateral bending	45°	36°	Elicited a "stretching" sensation on the right side of her neck but no pain
Rt lateral bending	45°	28°	Elicited a "pulling" sensation on the left of her mid-cervical spine but no pain
Lt rotation	80°	40°	Elicited pain on the left and right sides of her mid-to-lower cervical spine
Rt rotation	80°	38°	Elicited a "pulling" sensation down the left side of her cervical spine but no pain

Box 49.2 Cervical spine and cervico-shoulder region tests

	Patient's comments
Cervical spine traction	Painless and gave slight relief
Cervical spine compression	Elicited an increased feeling of "tightness" in the neck
Downward shoulder pressure	Painless
Trapezius trigger point pressure	Elicited pain on the left with increased pain on the right

Box 49.3 Active lumbar spine ranges of movement

	Approximate range	Patient's comments
Flexion	Fingers reached to halfway down her thigh.	Elicited low back pain
Extension	Full.	Elicited low back pain
Lt lateral bending	Fingers reached to 8 cm above the knee.	Elicited low back pain
Rt lateral bending	Fingers reached to 8 cm above the knee.	Elicited low back pain
Lt rotation	Full.	Painless
Rt rotation	Limited by 20%.	Elicited low back pain

IMAGING REVIEW

Available imaging was reviewed.

1. A lumbar spine X-ray 9 months following the motor vehicle accident showed that the sacroiliac joints appeared normal from a radiological point of view. There was some thinning of the L5–S1 disc space height (Fig. 49.2) and a minor degree of retrolisthesis of L5 on S1 suggesting that there may be some posterior disc bulge at this level. There was no lipping of the lumbar vertebral bodies or any osteoarthrotic wear and tear. The radiologist correctly pointed out that 'plain films cannot rule out the possibility of disc prolapse' in the lumbar spine.
2. A CT cervical spine examination performed 2 years 11 months following the motor vehicle accident was reported as showing 'mild posterior bulging at the C3–4 and C5–6 disc levels' (Fig. 49.3) as well as 'a small focal disc protrusion at the C4–5 disc level posteriorly in the midline (Fig. 49.4) causing minor indentation on the cervical spinal cord'.

CLINICAL IMPRESSION

As the Valsalva manoeuvre was positive, causing cervical and lumbar spine pain, disc bulging or protrusion was suspected at both levels.

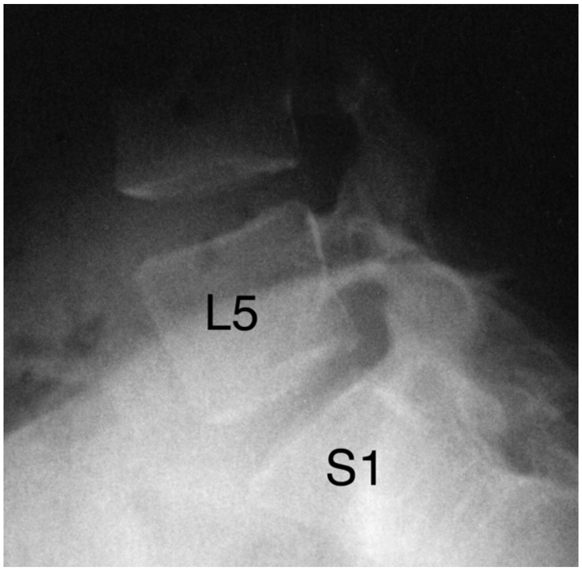

Figure 49.2 Lower lumbar spine lateral image. Note that there is some thinning of the L5–S1 disc space height, as compared to the disc above, with mild retrolisthesis of L5 on S1 suggesting that there may be some posterior disc bulge or prolapse at this level.

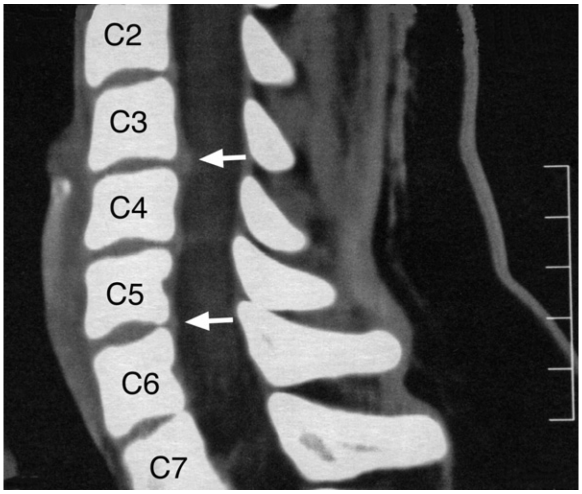

Figure 49.3 Cervical spine CT sagittal image. Note the 'mild posterior bulging at the C3–4 and C5–6 disc levels' (arrows).

WHAT ACTION SHOULD BE TAKEN?

As it was now 3 years and 2 months post-injury, an MRI of the cervical and lumbar spines was requested. However, she could not complete the examination as she became claustrophobic so only sagittal views of the cervical spine were obtained.

The partial cervical spine examination was reported as showing 'disc pathology at C4–5 and C5–6 with some bulging of both discs and early osteophyte formation' (Fig. 49.5). On reviewing the images it appeared that there was also posterior disc bulging at the C3–4 and C6–7 levels (Fig. 49.6). Considerable desiccation of the C3–4, C4–5 and C5–6 intervertebral discs, and to a lesser degree at C6–7

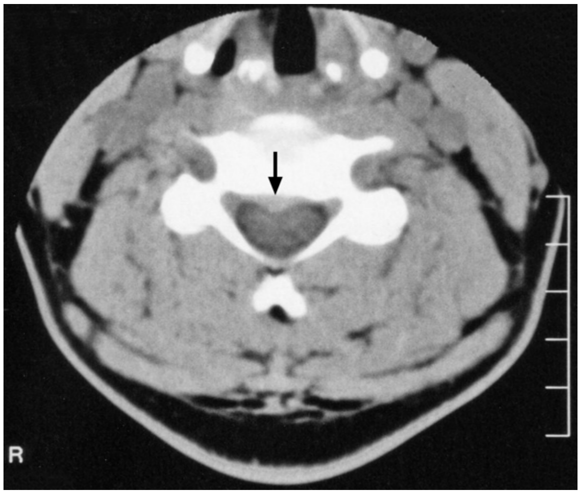

Figure 49.4 Cervical spine CT axial image. Note that the report stated: 'There is a small focal disc protrusion at the C4/5 level posteriorly in the midline causing minor indentation on the cervical spinal cord'.

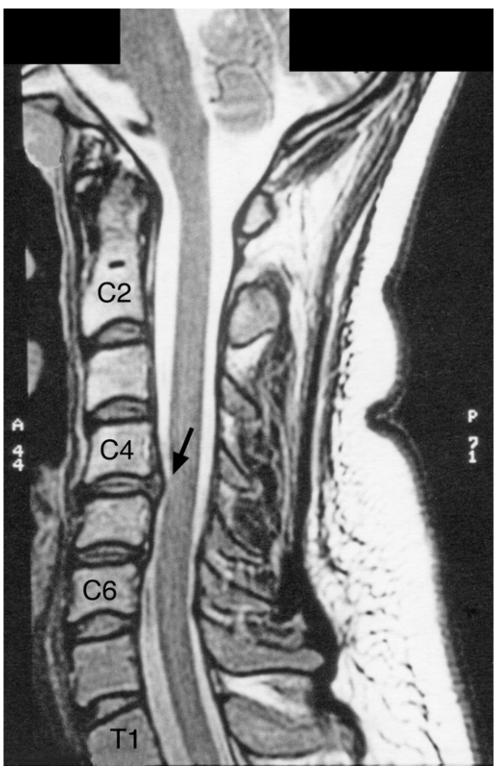

Figure 49.5 Cervical spine MRI sagittal T2-weighted image. In this sagittal section note the 'disc pathology at C4–5 and C5–6 with some bulging of both discs and early osteophyte formation'. At the C4–5 level the posterior disc bulge/protrusion is in close proximity to the anterior surface of the spinal cord itself (arrow), let alone the considerable pressure exerted upon the pain sensitive anterior part of the thecal sac/dural tube at this level. Note the associated loss of normal cervical spine contour with, in fact, a kyphosis.

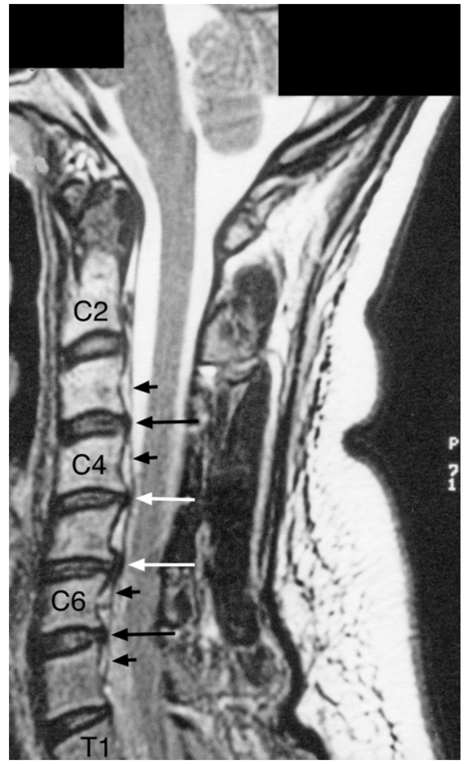

Figure 49.6 Cervical spine MRI sagittal T2-weighted image. Note the 'disc pathology at C4–5 and C5–6 with some bulging of both discs (white arrows) and early osteophyte formation. It appears that there is also disc bulging at the C3–4 and C6–7 levels (large black arrows). Also, the posterior disc bulges/protrusions at C3–4, C4–5, C5–6 and C6–7 press upon the pain sensitive anterior part of the dural tube (small black arrows). It is important to note that there is desiccation of the C3–4, C4–5 and C5–6 intervertebral discs, and to a lesser degree at the C6–7 disc level.

disc level, was noted (Fig. 49.6). In addition, it can be seen how the posterior disc bulges/protrusions at C3–4, C4–5, C5–6 and C6–7 press upon the pain sensitive anterior part of the dural tube (Fig. 49.6). The associated loss of normal cervical spine contour is considerable (Figs 49.5 and 49.6).

A repeat MRI was organized and was performed 4 months later with the patient sedated. This showed compression of the spinal cord at C4–5 (Fig. 49.7) where its diameter is measured at 4 mm on the MRI scan, whereas the diameter of the cord is 5 mm at the C3–4 and C5–6 levels. In addition, an axial view showed a posterior central to slightly right sided disc protrusion (Fig. 49.8) compressing the spinal cord anteriorly.

The lumbar spine MRI was reported as follows: 'There is mild retrolisthesis of L5 on S1 (Fig. 49.9). Degenerative change (desiccation) with loss of signal intensity on T2 sequence, loss of disc volume and diffusely bulging discs is seen at all levels, predominantly at L4–5 and L5–S1. A tiny area of high signal intensity on T2 sequence is seen in the posterior aspect of the L5–S1 disc that could represent an annular tear. There is mass effect on the anterior epidural space contiguous to the discs L4–5 and L5–S1. No definite

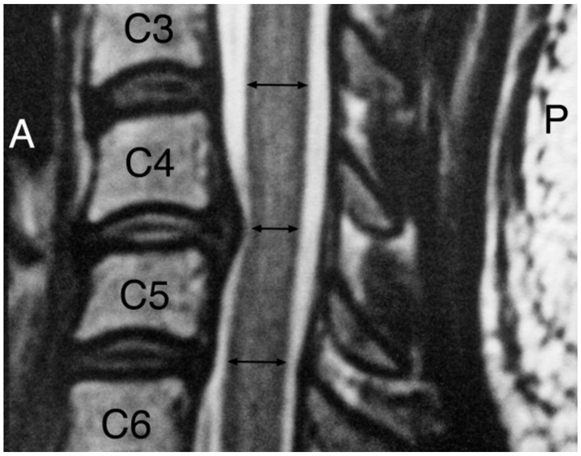

Figure 49.7 Cervical spine MRI sagittal T2-weighted image. Note that the posterior disc herniation at C4–5 compresses the anterior surface of the spinal cord where its diameter is measured at 4 mm (shorter arrow) on the MRI scan, whereas the diameter of the cord is 5 mm at the C3–4 and C5–6 levels (longer arrows).

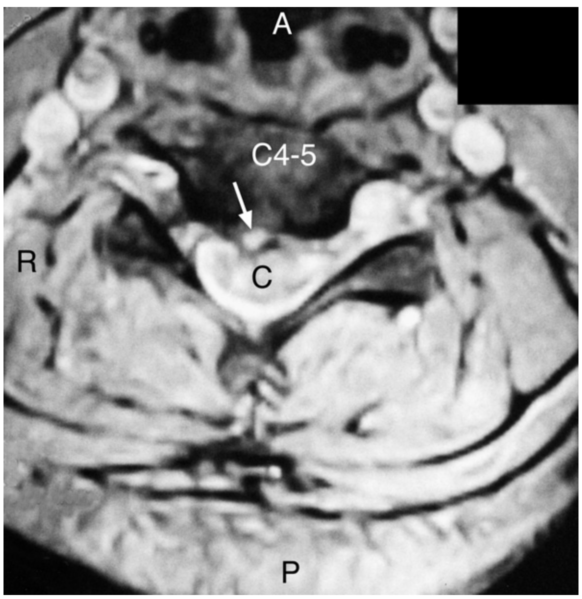

Figure 49.8 Cervical spine MRI axial T2* Gradient Echo image through the C4–5 disc level. Note the posterior central to slightly right sided disc protrusion (arrow) compressing the spinal cord (C) anteriorly.

evidence of compressive neuropathy. All neural exit foramina are clear. Degenerative changes are seen bilaterally in the facet joints L4–5 and L5–S1'.

In addition, it appears there is a posterior central to left sided disc bulge (Fig. 49.10) possibly compromising the adjacent left nerve root which may well explain the symptom of pain radiating to the left buttock, thigh posterolaterally, calf and left foot, with some numbness of the sole of the left foot. In addition, the tear may suggest the possibility of nuclear material leaking out from the disc to irritate the adjacent nerve root.

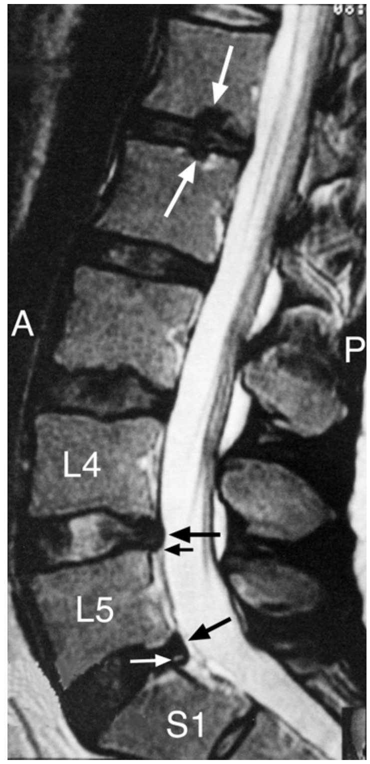

Figure 49.9 Lumbar spine MRI sagittal T2-weighted image. Note the posterior bulging discs at the L4–5 and L5–S1 levels (arrows) that touch, and at L4–5, elevate the posterior longitudinal ligament and compress the pain sensitive anterior surface of the dural tube (small black arrow). In addition, there is a tear (small white arrow) in the L5–S1 disc posteriorly. Schmorl's nodes are present in the endplates on each side of the L1–2 intervertebral disc (long white arrows).

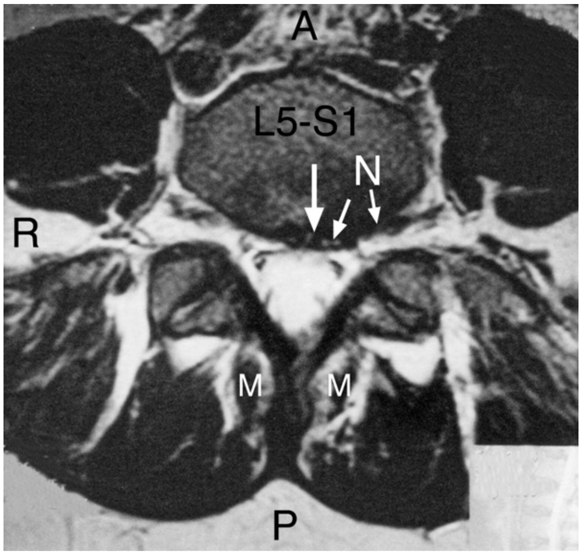

Figure 49.10 Lumbar spine MRI axial T2-weighted image through the L5–S1 disc level. Note the proximity between the L5–S1 central to left sided disc bulge (large white arrow) with a disc tear (small white arrow) to the nerve root (N) in the adjacent nerve root canal. In addition, there is early atrophy (degenerative change) of the multifidus muscles (M).

Furthermore, at the L5–S1 level (Fig. 49.10), and to a much lesser extent at the L4–5 level, there is early atrophy (degenerative change) of the multifidus muscles.

DIAGNOSIS

Cervical spine

- Musculoligamentous soft tissue injuries.
- Various cervical spine posterior disc bulge/protrusion lesions.
- C4–5 anterior disc bulge.
- Spinal canal stenosis at the C4–5 level.
- Loss of normal cervical spine contour.
- Headaches of cervicogenic origin.

Lumbar spine

- Musculoligamentous soft tissue injuries.
- L5–S1 internal disc disruption with loss of disc height, posterior disc bulge and disc tear.
- L4–5 posterior disc bulge.
- Left sciatic pain with 'numbness' extending as far as the sole of the left foot, i.e. radiculopathy.
- Strain of the left and right sacroiliac joints.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. After all imaging findings were explained to her, she was advised that it may be helpful for her to try acupuncture treatment, or gentle cervical and lumbar spine traction. She was advised that this was the only physiotherapeutic/manual therapy type of treatment that she should try at this time.

She was also advised that cervical spine surgery may be necessary in the future in an attempt to decompress the spinal cord at the C4–5 level where there is pressure upon it. She was told that there is a problem in that there are several adjacent disc lesions, perhaps meaning that surgery may not be an option apart from decompressing the spinal cord at the C4–5 level.

Note

Some cervical spine gross anatomy and histopathology sections are provided below to show the complexity of cervical spine anatomy (Figs. 49.11. to 49.13).

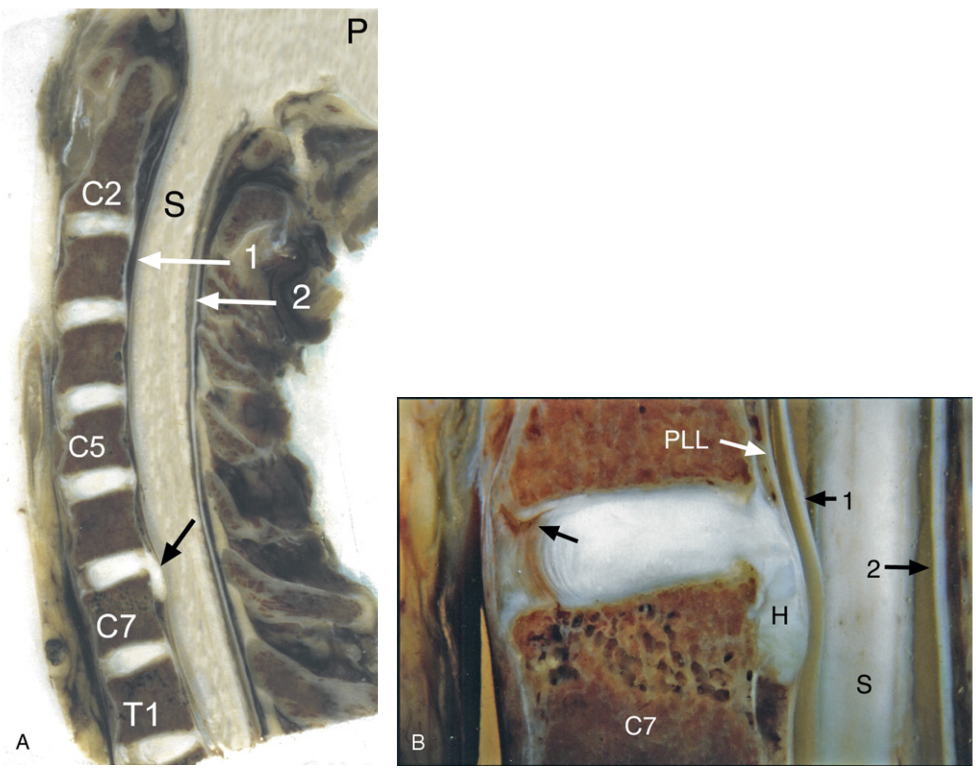

Fig 49.11 (A) A sagittal section of the cervical spine from a 52-year-old woman thrown out of a vehicle that rolled over at speed. Note the spinal cord (S) within the spinal canal as well as the posterior intervertebral disc herniation at the C6–7 level (black arrow) that presses upon the pain sensitive anterior part of the dural tube and indents the anterior surface of the spinal cord. P = posterior; S = spinal cord; 1 (arrow) = dural tube (anteriorly); 2 (arrow) = dural tube (posteriorly). Reproduced with permission from Professor J R Taylor (personal communication 2006). (B) Enlargement of the C6–7 level large posterior intervertebral disc herniation (H) and a small anterior rim lesion (annular tear) (black arrow). PLL = posterior longitudinal ligament; 1 (arrow) = dural tube (anteriorly); 2 (arrow) = dural tube (posteriorly). (Reproduced with permission from Taylor J R, Taylor MM 1996 Cervical spine injuries: An autopsy study of 109 blunt injuries. Journal of Musculoskeletal Pain [The Haworth Medical Press, Inc.] vol 4, no. 4: 61-79.) See also colour plate section Fig. vii.26 A and B.

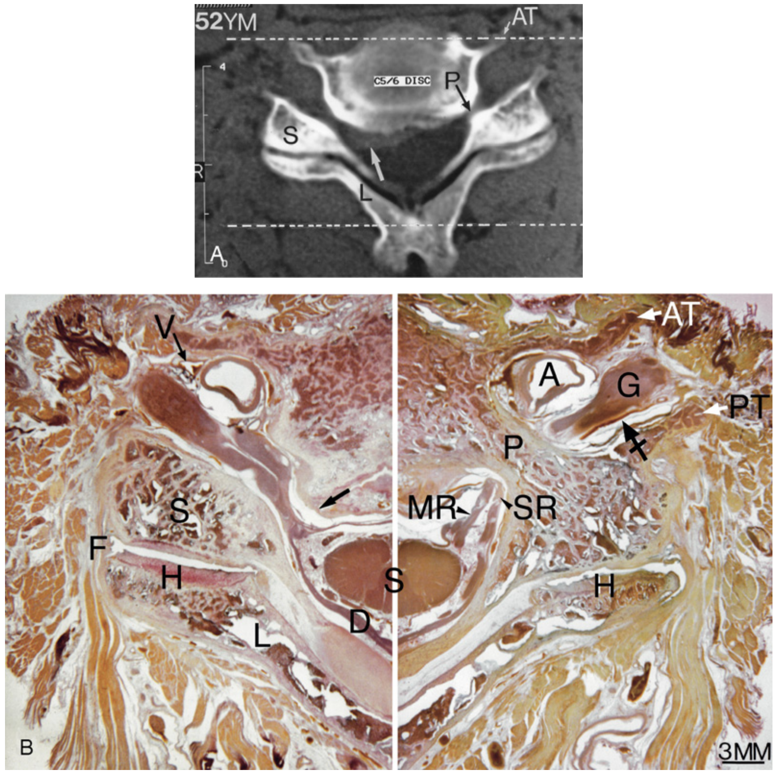

Figure 49.12 (A) An example of an axial cervical spine CT scan from a 52-year-old man and a histopathology section montage, in a similar plane, from postmortem material (B). The histopathology section on the left is at the intervertebral foramen level, whereas the histopathology section on the right is at the pedicle level in order to correspond to the CT scan image. The axial CT scan (A) at the C5–6 level shows the approximate area (between the broken lines) of the histopathology sections in (B). The CT scan also shows some posterior spondylosis of the vertebral body with disc herniation on the right side (white arrow), both of which cause some narrowing of the spinal canal and the right intervertebral canal. The montage representing the two histopathology sections is from the mid-cervical spine of a middle-aged postmortem specimen. Note how osteophytosis of the posterolateral region of the uncovertebral joint (black arrow) can deform the nerve roots as shown in the histopathology section. A = vertebral artery within the transverse foramen; AT = anterior tubercle; F = fibrous capsule of the zygapophysial joint laterally; D = dural tube; G = spinal ganglion (highly vascular) and intermediate neural branch blood vessel (tailed arrow); H = hyaline articular cartilage on the facet of the inferior articular process seen in the histopathology section on the left side of the montage. The zygapophysial joint on the right shows osteoarthrotic changes; L = lamina; MR = motor root; P = pedicle; PT = posterior tubercle; S = superior articular process; SR = sensory root; V = thin-walled vein adjacent to the vertebral artery. Note the spinal cord (S) lying within the dural tube and how the motor (MR) and sensory (SR) roots pass into the intervertebral canal beneath the pedicle on the right side of the histopathology section. (Reproduced with permission from: Giles L G F 2000 Mechanisms of neurovascular compression within the spinal and intervertebral canals. Journal of Manipulative and Physiological Therapeutics 23: 107–111.) See also colour plate section Fig. vii.27A and B.

KEY POINT

When intervertebral disc posterior bulges or protrusions press upon the pain sensitive anterior surface of the dural tube, compressing the intervening recurrent meningeal nerves and blood vessels, this can cause neck pain without radiculopathy.

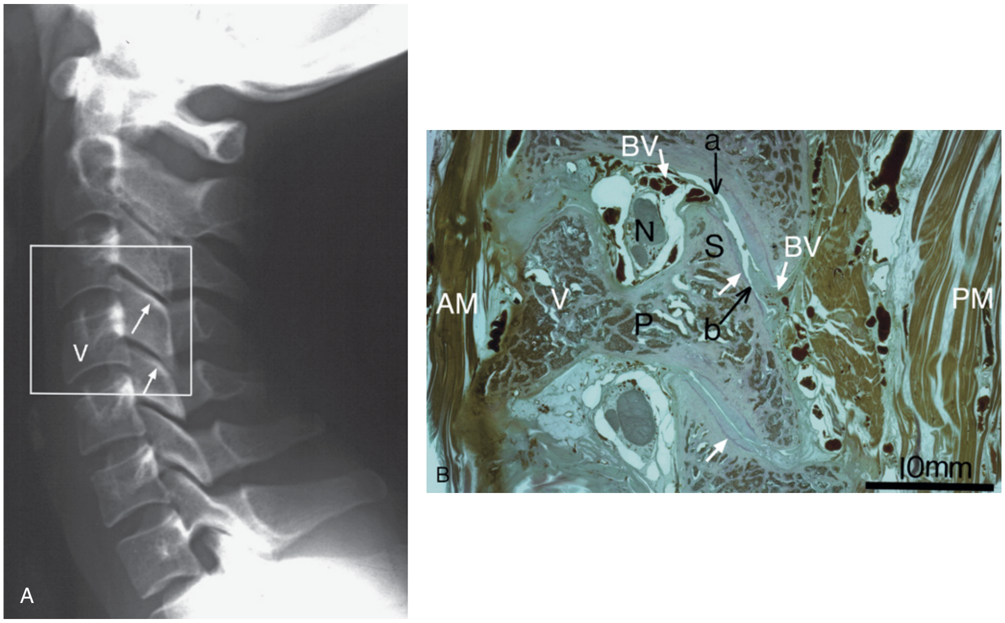

Fig. 49.13 (A) A plain X-ray lateral view of a normal cervical spine. The square white box shows two zygapophysial 'facet' joints (white arrows), and (B) is a histopathology postmortem parasagittal section representing a similar area with two zygapophysial 'facet' joints (white arrows) and other related soft tissue structures. The lower white arrow shows a zygapophysial joint with normal articular cartilage on each side of the joint's potential 'space'. The upper white arrow shows a joint with osteoarthrotic wear in the articular cartilage on the superior articular process (S). The black arrows (a) and (b) show highly vascular synovial folds projecting into the upper and lower parts of that zygapophysial joint; these structures that contain nociceptive nerves can be nipped between joint surfaces during injury, causing pain and bleeding into the joint. AM = anterior spinal muscles and PM = posterior spinal muscles, respectively; BV = blood vessels within the intervertebral foramen and in the synovial folds in the upper and lower poles of the joint; N = neural structures in the intervertebral foramen which are surrounded by fatty tissue and many blood vessels; P = pedicle that joins the vertebral body (V) to the posterior bony structures; S = superior articular process and I = inferior articular process, respectively, that form the zygapophysial joint. From: Giles L G F 1986 Lumbo-sacral and cervical zygapophyseal joint inclusions. Manual Medicine 2: 89-92. See also colour plate section Fig vii.28B.

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Giles L G F 1986 Lumbo-sacral and cervical zygapophyseal joint inclusions. Manual Medicine 2: 89–92.

Taylor J R, Taylor M M, 1996 Cervical spine injuries: An autopsy study of 109 blunt injuries. Journal of Musculoskeletal pain 4(4): 61–79.

Case 50

C5–6 posterolateral intervertebral disc protrusion

COMMENT

Subtle clinical signs and subtle plain X-ray findings are important when considering whether manipulation would be safe.

PROFILE

A 53-year-old fit female who does not smoke, only drinks alcohol socially and is self employed in a light duty profession.

PAST HISTORY

There is no relevant medical history apart from intermittent neck pain over the last 15 years for which she had non-manipulative chiropractic and physiotherapy treatment whenever she experienced episodes of such pain. A plain X-ray examination 15 years ago did not indicate any significant cervical spine joint problem.

PRESENTING COMPLAINT(S) (Fig. 50.1)

Five months ago she experienced what she considered to be a recurrence of her intermittent neck pain syndrome, so attended for a further opinion. She described right sided lower cervical spine pain at approximately the C5–7 level with intermittent slight pain extending into the right cervico-shoulder region. The pain sometimes extends to the right side of the upper thoracic region. There were no symptoms in the upper or lower extremities. On awakening in the morning there is some aching and stiffness on the right side of the lower cervical spine. Non-manipulative chiropractic and physiotherapy treatment had not resolved her symptoms for this episode.

Seven days before consulting me, plain X-ray films had been taken of the cervical spine.

AETIOLOGY

Unknown.

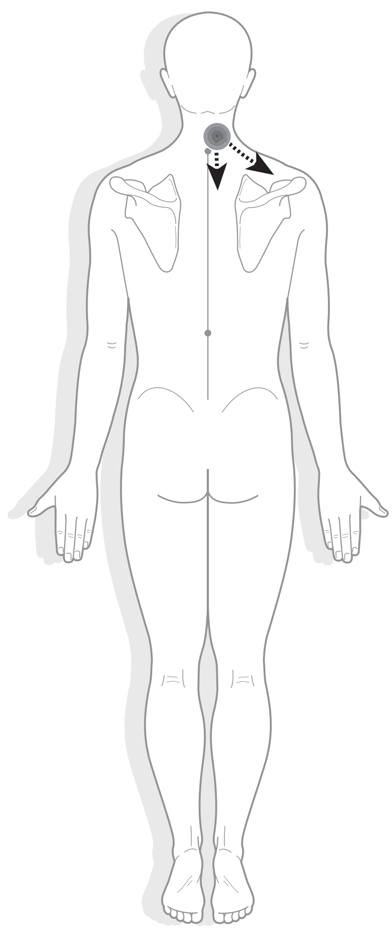

Figure 50.1

EXAMINATION

There was no clinical evidence of scoliosis. Deep palpation of the paraspinal muscles elicited slight pain on the right side at the C5–6 level. Cervical spine ranges of movement were only slightly restricted and painless, except for some discomfort in the lower cervical spine on cervical spine extension. Gentle downward pressure on the head did not aggravate her symptoms. Cervical spine traction 'felt good'. The deep reflexes in the upper extremities were normal. Pinprick sensation elicited very slight hypoaesthesia near the tip of the right thumb (C6). Vibration sensation at the elbows was normal. The Valsalva manoeuvre was reported as not causing any sharp pain but perhaps slight discomfort in the lower cervical spine.

IMAGING REVIEW

Her original cervical spine X-ray images were reported as being normal, so these were not considered for the present consultation.

The recent cervical spine X-ray films were viewed as well as the radiology report stating: 'Within the limited range between flexion (Fig. 50.2) and extension (Fig. 50.3A), no significant instability is identified. The pre-vertebral soft tissues are within normal limits. The vertebral body height and alignment is satisfactory. There is mild disc space narrowing at the C5–6 level. At other levels the disc spaces are reasonably well maintained. No significant facetal arthropathy is identified and the exit foramina are widely patent bilaterally (Fig. 50.4)'.

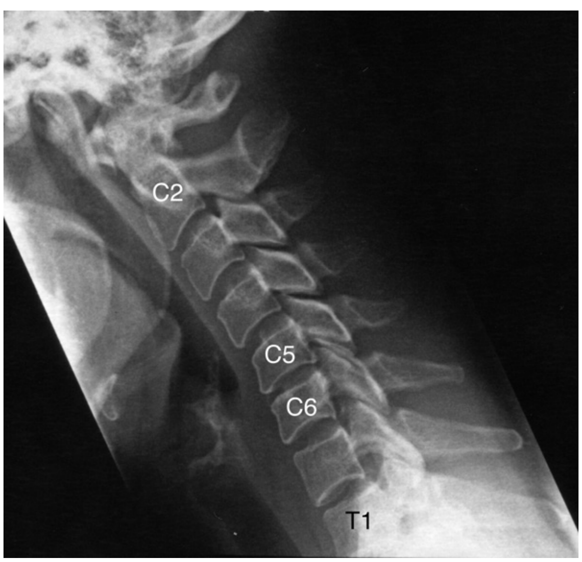

Figure 50.2 Cervical spine flexion image. Note the reported limited range of cervical spine flexion with the mild disc space narrowing at the C5–6 level. In addition, note the angulation at the C5 level.

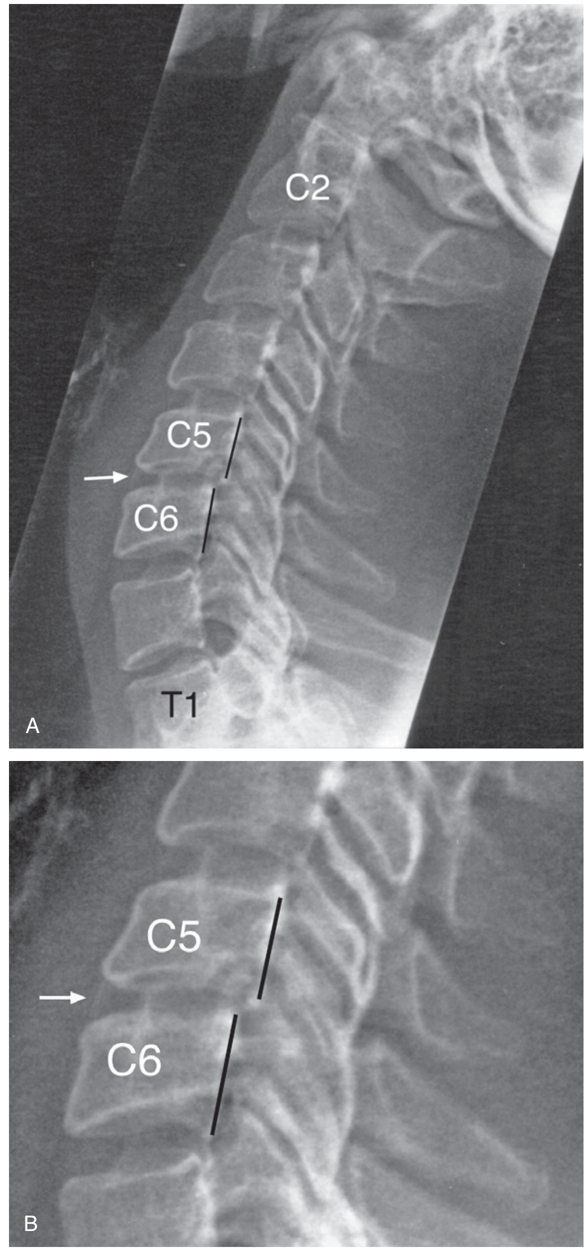

Figure 50.3 Cervical spine extension image (A). Note the reported limited range of cervical spine extension. In addition, note (i) there is slight retrolisthesis of C5 on C6 as shown by the lines drawn along the back of the C5 and C6 vertebral bodies, and (ii) a thin curved line of calcification in the anterior region of the C5–6 intervertebral disc (white arrow); this is best seen on the enlarged insert (B).

However, no mention was made of the thin line of calcification in the anterior region of the C5–6 intervertebral disc (Fig. 50.3). Furthermore, no mention was made of the angulation of the cervical spine at the C5 level on cervical spine flexion (Fig. 50.2) or of the slight retrolisthesis of C5 on C6 on cervical spine extension (Fig. 50.3A and B).

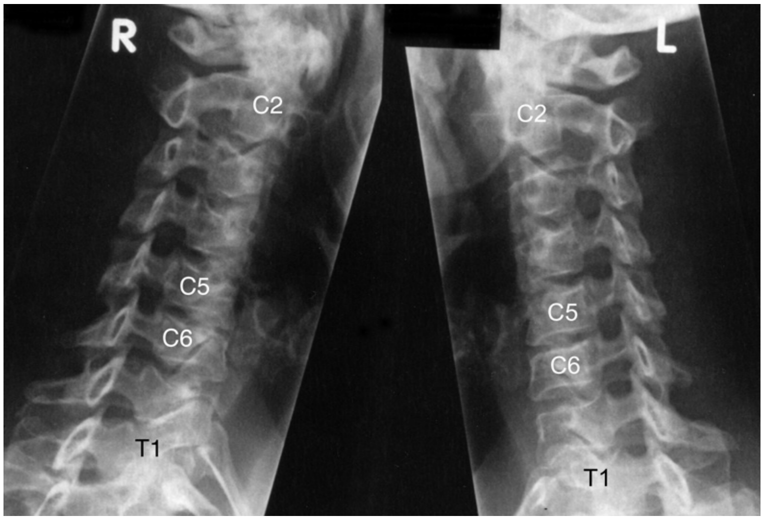

Figure 50.4 Cervical spine oblique images of the right and left intervertebral foramina, respectively, showing that the exit foramina are widely patent bilaterally.

CLINICAL IMPRESSION

Right sided C5–6 intervertebral disc bulge/protrusion.

WHAT ACTION SHOULD BE TAKEN?

A cervical spine MRI was requested to further evaluate the C5–6 intervertebral disc reported on the plain X-ray films as showing ‘mild disc space narrowing’. The MRI report stated: ‘At C5–6 there is moderate cervical spine degeneration (Fig. 50.5). At this level there is a prominent right posterolateral disc protrusion (Fig. 50.6A to D), extending up to 5 mm posterior to the usual disc margin in the lateral canal and foraminal entrance, posteriorly displacing and rotating the cervical cord and compressing its anterior surface and impinging upon the exiting right C6 root. At C4–5 there is a very small posterior central disc protrusion extending no more than 2 mm posterior to the disc margin without neural impingement. No intrinsic abnormality of the cervical or upper thoracic cord or abnormality of the craniocervical junction is shown. There is no evidence of bony canal or foraminal stenosis. No facet arthropathy or other bone or paravertebral soft tissue lesion.’

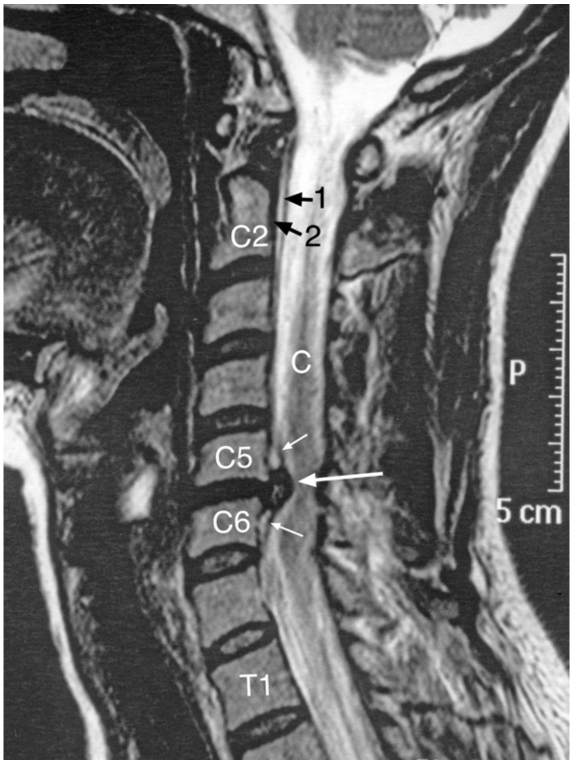

Figure 50.5 Cervical spine MRI right parasagittal T2-weighted image. 1 = anterior surface of the dural tube; 2 = posterior longitudinal ligament. Note the relatively large right paracentral posterolateral C5–6 disc protrusion (large white arrow) that compresses (i) the pain sensitive anterior part of the dural tube (small white arrows), and (ii) the spinal cord (C). P = posterior.

DIAGNOSIS

C5–6 level prominent right posterolateral disc protrusion displacing and rotating the cord, compressing its anterior surface, and impinging upon the right C6 roots.

TREATMENT AND RESULTS

The patient’s condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was advised to avoid all activities that may aggravate her intervertebral disc protrusion such as running, working above her head, and extending her neck to look up. In addition, she was advised to (i) have

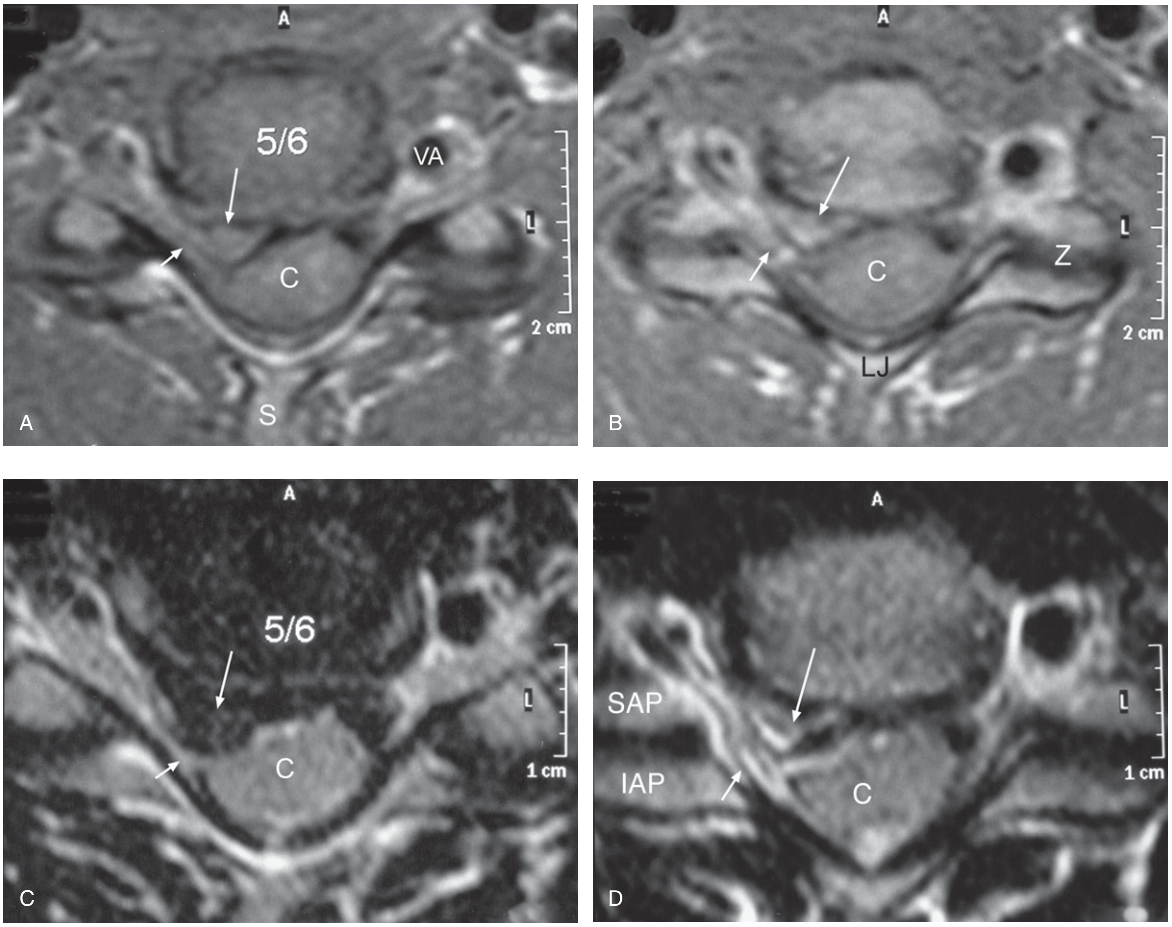

Figure 50.6 Cervical spine MRI axial T1-weighted images at the C5–6 level (A and B), and T2-weighted images at this level (C and D). Note the prominent right posterolateral disc protrusion (large arrow), extending up to 5 mm posterior to the usual disc margin in the lateral canal and foraminal entrance, posteriorly displacing and rotating the cervical cord (C) and compressing its anterior surface and impinging upon the exiting right C6 root (small arrow). IAP = inferior articular process; L = left side of patient; LJ = lamina junction; S = spinous process; SAP = superior articular process; VA = left vertebral artery within the left transverse foramen that is normal in cross-section while the right artery is hypoplastic; Z = zygapophysial facet joint. (See Fig. 49.12A and B.)

a follow-up MRI in 6 weeks following intermittent gentle manual traction of the cervical spine in the supine posture, and (ii) to see a spine surgeon for an assessment in view of the large right sided posterolateral C5–6 disc protrusion with spinal cord rotation and right C6 neural compression.

She obtained two opinions, one from a neurosurgeon (who suggested leaving the disc protrusion as it is and waiting to see what further developments may occur) and one from an orthopaedic surgeon (who concurred that a follow-up MRI cervical spine may be helpful to reassess her progress).

The gentle manual cervical spine traction in the supine position performed daily for 3 months, with 6 repetitions of traction, provided quite good relief without the need of taking medication.

The follow-up MRI showed essentially no change but the orthopaedic surgeon noted that the patient's symptoms had 'settled extremely well' with the conservative treatment.

KEY POINTS

1. This case clearly demonstrates the importance of functional plain X-ray films and the importance of MRI in assessing patients, as this patient has a prominent intervertebral disc protrusion with spinal cord rotation and right C6 neural compression with only minimal symptoms.
2. The clinical diagnosis was suspected in view of the patient's mild symptoms extending from the right side of approximately the C5–7 level to the right cervico-shoulder region and sometimes extending into the right side of the upper thoracic region, as well as the subtle sign of very slight hypoaesthesia to pinprick sensation near the tip of the right thumb (C6). However, the magnitude of the C5–6 protrusion displacing and rotating the cord with impingement upon the right C6 roots, was unexpected. This case shows how important it is to *thoroughly evaluate patients* before performing spinal manipulation.
3. The importance of (i) taking a thorough history, (ii) performing an appropriate thorough physical examination, and (iii) imaging the painful region of the spine, by initially using plain X-ray images, including functional views, is demonstrated in this case. Some patients appear to have a high pain threshold and, unless the clinician understands that significant cervical spine disc protrusion may be present with minimal symptoms, this could lead to a serious adverse event if cervical spine mobilization or manipulation were to be performed without first determining what pathology is present.

Further reading

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Case 51

Post motor vehicle accident intervertebral disc injuries

COMMENT

In addition to routine cervical spine radiographs, functional cervical spine radiographs are essential following neck trauma unless there is a risk to the vertebral/carotid arteries and/or cord with neck flexion and extension.

PROFILE

A 44-year-old female clinician who does not smoke and only drinks alcohol socially.

PAST HISTORY

Approximately 4 weeks ago she had been driving her car, with her seat belt in place, and had almost stopped behind a line of cars when her vehicle was hit by a large vehicle from behind. Her car was then pushed into a vehicle in front of hers. She was looking straight ahead during the impact. She immediately felt neck pain and 'numbness' in her left arm; the numbness persisted for a few days. Initially, she experienced a severe headache.

PRESENTING COMPLAINT(S) (Fig. 51.1)

A 'deep and intense pain' bilaterally in the cervical spine and extending to the T5 level with radiation of pain to the left arm, forearm, thumb, index and middle finger (C6 and 7). Her neck is very painful on arising from bed and she finds that heat from the hot shower helps temporarily. She experiences significant bilateral occipital headaches which she says are 'different' to very occasional headaches from which she had suffered previously. She had no history of migraine.

She had had two analgesic injections for the pain and depended on analgesic medication to help with her neck and arm pain.

AETIOLOGY

Motor vehicle accident approximately 4 weeks ago.

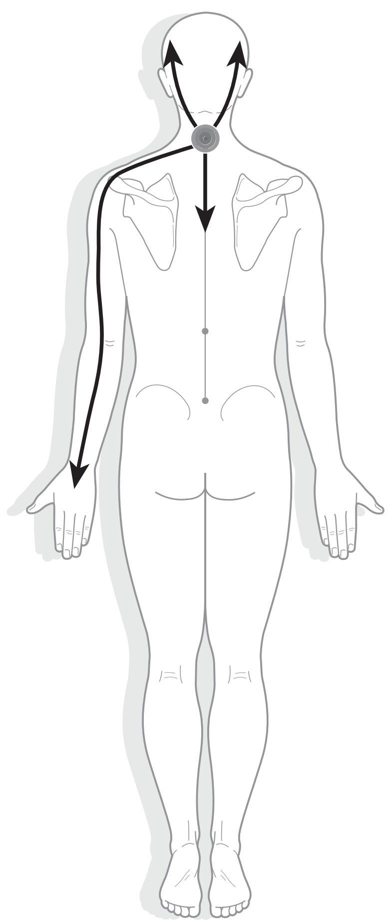

Figure 51.1

EXAMINATION

The deep tendon reflexes in the upper extremities were normal but the left patella reflex was diminished to one plus, as compared to the right which was two plus, i.e. normal. The ankle reflexes were normal. Pinprick sensation elicited hypoaesthesia in the left thumb (C6) and in the left middle finger (C7). Light touch in the left hand was diminished. Vibration sensation in the legs and arms was normal. Power appeared to be approximately equal and normal for the upper and lower extremities. Eye movements and visual fields appeared to be normal, as was the case with the consensual light and accommodation reflexes. The optic discs were normal. She was able to perform the finger to nose test normally. In the seated position, the blood pressure in the arms was 129/87 (right) and 129/85 (left). The Valsalva manoeuvre was normal, as were foot plantar responses.

Deep palpation of the paravertebral muscles of the cervical spine elicited tenderness bilaterally as far as C7; the thoracic spine was tender to T4.

Cervical spine active ranges of movement were measured using a CROM instrument (see Box 51.1).

Both sternocleidomastoideus muscles were very tender to palpation and when she tried to perform various neck movements. Percussion of the thoracic and lumbar spines was painless.

IMAGING REVIEW

A cervical spine X-ray study, including cervical spine flexion and extension functional views reported a small bony fragment adjacent to the inferior endplate of the C5 body anteriorly and described this as being 'consistent with a limbus vertebra, i.e. a normal variant'.

However, further findings were noted on reviewing the films themselves (Fig. 51.2A and B). Note how the 'limbus' bone moves between the extension and flexion views, raising the possibility that this structure is, in fact, calcification in the anterior fibres of this disc, or in the anterior longitudinal ligament, due to injury having caused bleeding followed by calcification. The cervical spine extension view (Fig. 51.2A) shows some widening of the C5–6 intervertebral disc space anteriorly that is greater than at the C4–5 and C6–7 levels, suggesting that the anterior fibres of the intervertebral disc have been torn as well as some fibres of the anterior longitudinal ligament. On cervical spine flexion (Fig. 51.2B) the C5–6 intervertebral disc space is narrower anteriorly. In addition, there is some widening of the C6–5 intervertebral disc space posteriorly. These findings suggest that there has been some tearing of the posterior fibres of the intervertebral disc at C5–6 and perhaps some tearing of the posterior longitudinal ligament. Furthermore, the flexion view (Fig. 51.2B) shows that there is limited movement below the C4 vertebral level.

Box 51.1 Cervical spine active ranges of movement

	Normal range	Measured range	Comments
Flexion	50°	37°	Caused centrally located neck pain
Extension	60°	48°	Limited due to 'neck stiffness'
Left & right lateral bending	45°	23°	Caused pain on the contralateral side at the C5 to T1 levels
Left rotation	80°	64°	Caused a 'pulling' sensation on the right of C5
Right rotation	80°	64°	Caused pain in the left cervico-shoulder region

CLINICAL IMPRESSION

Soft tissue injuries to various parts of the cervical spine, but most likely to the C5–6 and C6–7 intervertebral discs, with possible tearing of the anterior fibres at C5–6 and the posterior fibres at C6–7; i.e. a cervical spine 'whiplash'-type injury.

WHAT ACTION SHOULD BE TAKEN?

In view of the plain X-ray film findings a cervical spine MRI was requested. The MRI report noted a 'small disc protrusion at the C6–7 disc posteriorly' (Fig. 51.3A). However, no mention was made of the anterior disc bulge at C5–6. The small disc protrusion posteriorly at C6–7 was confirmed on the axial views (Fig. 51.3B).

Approximately 1 year later a further MRI was performed which showed similar findings to the previous MRI but with clearer visualization of the posterior annular tearing on the axial scans.

A C4–C7 cervical spine discogram, followed by a CT examination, was performed. This showed the following.

C5–6 level: there is a full thickness left posterolateral tear in the annular fibres with contrast extravasating adjacent to the posterior margin of the left uncovertebral joint; C6–7 level: there is a full thickness posterior tear in the annular fibres with extravasation of contrast material beneath the posterior longitudinal ligament.

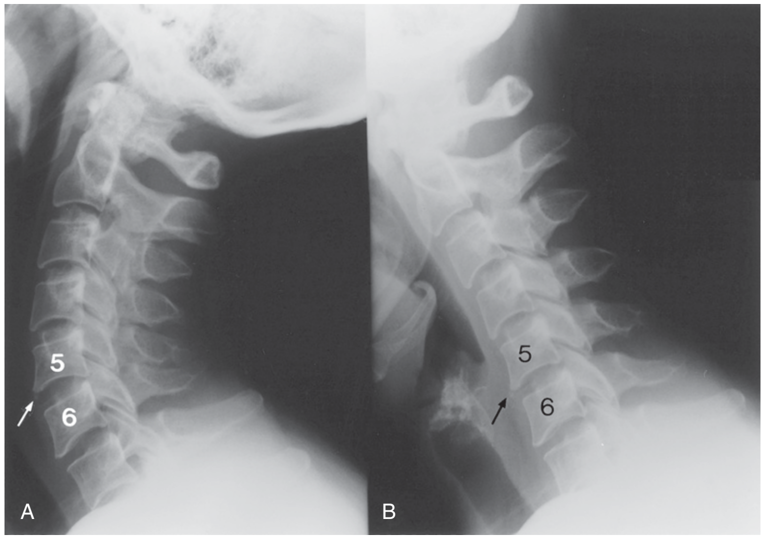

Figure 51.2 (A) Cervical spine extension plain X-ray image which shows a small calcific density adjacent to the inferior endplate of C5 anteriorly (white arrow). Also, the disc height is increased anteriorly at C5–6 compared to the discs at C4–5 and C6–7 levels, suggesting weakness of the anterior annular fibres and perhaps an injury to the anterior longitudinal ligament at C5–6. The disc height posteriorly at C6–7 is greater than the disc height anteriorly, raising the possibility of injury posteriorly to the intervertebral disc and the posterior longitudinal ligament at this level. In addition, note the slight retrolisthesis of the C3 body on the C4 body. (B) Cervical spine flexion image showing the calcific density described above (black arrow). Note how this structure changes its position on extension and flexion. Also, note that there is an angulation at the C4–5 level with the vertebral bodies below this level aligned in an almost straight line, particularly from C5 to C7, indicating limited movement below the C4 level.

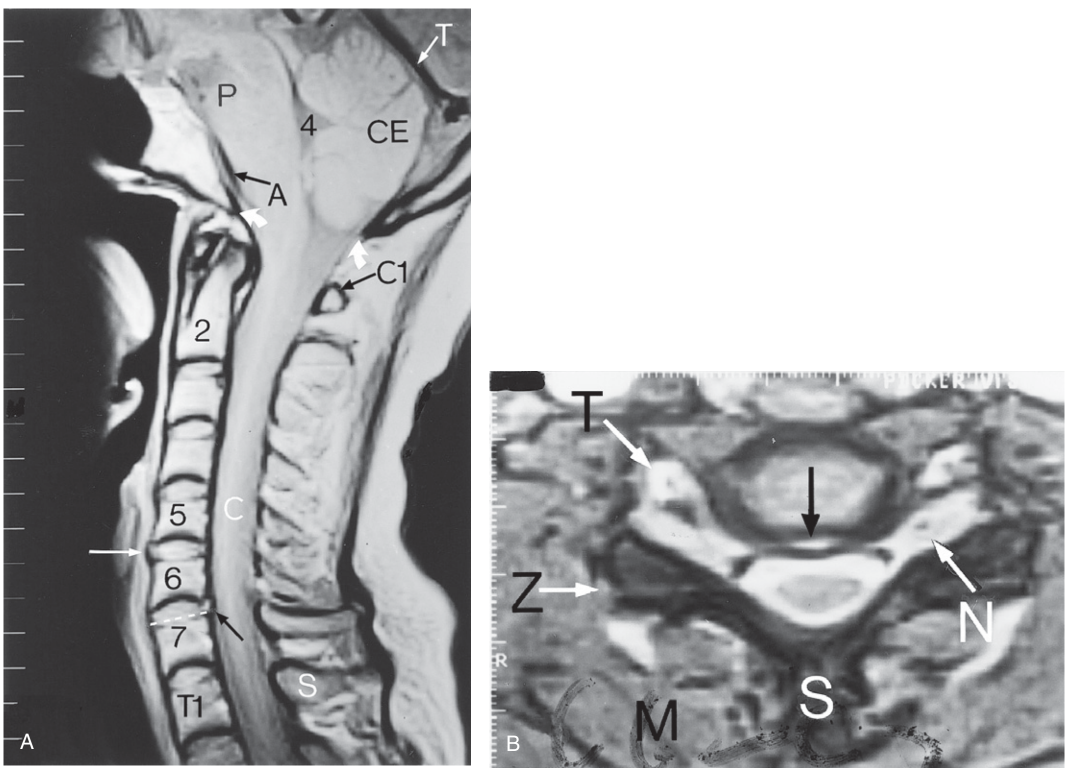

Figure 51.3 (A) Cervical spine MRI sagittal T1-weighted image showing the small posterior disc protrusion at the C6–7 disc (black arrow) and the anterior disc bulge at C5–6 (white arrow). 2 = body of the second cervical vertebra; 5, 6 and 7 show the bodies of the fifth, sixth and seventh cervical vertebrae; T1 = body of first thoracic vertebra; C = spinal cord; S = spinous process of T1 vertebra; C1 = posterior tubercle of the first cervical vertebra; CE = cerebellum; 4 = fourth ventricle; P = pons; T = tentorium cerebelli; A = basilar artery; curved white arrows show the anterior (clivus) and the posterior (or occiput) margins of the foramen magnum. The approximate level for the axial scan shown in (B) is represented by the white broken line that gives the plane of the axial section. (B) MRI axial T2-weighted image at the C6–7 level (see A). M = muscles adjacent to the spinous process (S); T = transverse foramen (containing the vertebral artery, vertebral vein and the sympathetic plexus on the vertebral artery); Z = zygapophysial joint with superior and inferior articular facets opposite each other; N = nerve root ganglion in the left intervertebral foramen; black arrow shows the disc protrusion posterior to the C6–7 intervertebral disc.

DIAGNOSIS

- Cervical spine soft tissue injuries, including C5–6 and C6–7 disc injuries.
- Cervicogenic headaches.
- Left C6–7 radiculopathy.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. In view of persisting deep and intense pain in the cervical spine and the associated referred pain, the patient was referred to a neurosurgeon who performed a C6–7 anterior cervical discectomy and fusion (Cloward procedure) using a right iliac crest graft and metallic plate fixation.

Unfortunately, surgery did not resolve the left upper limb symptoms. However, the bilateral occipital headaches resolved.

Note

In order to fully appreciate the soft tissue anatomy surrounding the cervical spine, see Figures 49.12 and 49.13.

KEY POINTS

The value of cervical spine flexion and extension functional views has been well documented in the literature and this case indicates how important it is to look at lateral view functional plain film radiographs to determine:

a. the overall contour of the cervical spine;
b. the disc space height for any possible disc thinning, suggesting injury;
c. disc space height anteriorly and posteriorly which may suggest tearing of the anterior and/or posterior fibres of the intervertebral disc and the associated ligaments;
d. whether any segmental instability is present.

Further reading

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Case 52

Post motor vehicle accident soft tissue injuries

COMMENT

Cervical spine flexion and extension functional views are essential to properly evaluate the cervical spine following a neck injury, as long as the patient feels comfortable in these positions and does not feel 'dizzy' or nauseous during the examination.

PROFILE

A 29-year-old married female of cheerful disposition who is somewhat overweight. She does not smoke or drink alcohol. She is very concerned about having lost her job, which she really enjoyed, due to her motor vehicle accident.

PAST HISTORY

The patient had always been healthy and her only surgical history was that of an operation on her left eardrum about 6 years ago.

Sixteen months ago she was the seat-belted driver of her truck waiting at a red traffic light and, just as the light turned green, her truck was suddenly hit from the rear, with considerable impact, by a motorcar which pushed her truck several metres across the intersection. Her head 'jerked backwards and forwards' but she does not recall it hitting anything in the truck, although it may have hit a bar behind the headrest. She felt severe neck pain a few minutes after the accident so was taken to the hospital where she was examined and allowed to go home.

PRESENTING COMPLAINT(S) (Fig. 52.1)

She was referred by her general medical practitioner regarding her chronic neck and upper thoracic spine pain due to a motor vehicle accident 16 months ago. Her main complaint was the neck pain that radiates to both arms variably and sometimes has a 'burning' sensation. She also experiences some intermittent pain radiating around her rib cage at approximately the breast level.

She found that physiotherapy treatment made her neck and upper thoracic spine feel worse, so she decided

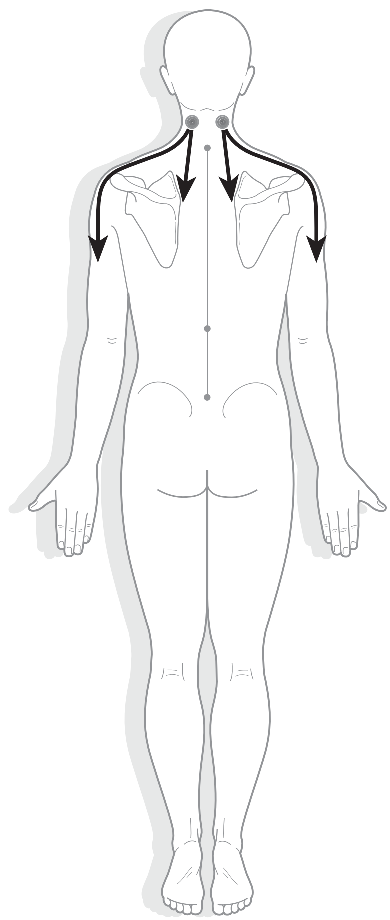

Figure 52.1

to stop this form of treatment. She felt that one of the mobilizing manoeuvres had caused some 'numbness in the hands'.

She was referred to a neurologist where an MRI study was ordered. She has tried many different types of medication without lasting relief. The neurologist advised her to go back to work but this caused an increase in her neck and arm pains so she had to take Endone.

She saw many specialists including a psychiatrist and a clinical psychologist as she was severely depressed and tearful about her condition.

In an attempt to help her stop the increasing medication, she had been advised to try a transcutaneous electrical nerve stimulator (TENS) machine for her chronic neck and arm pains; this was of considerable help, although she still could not wear a shoulder bag or a necklace as these items aggravate her neck pain. She feels a 'squishey' sensation at times on moving her neck.

She has trouble falling asleep and is awakened by the pain during the night, but at no particular time; she has to get up and walk around before trying to go to sleep again.

She uses a 'really hot' shower on her cervicothoracic region and this gives her some temporary relief. However, she cannot understand why the water feels hot on her arms and hands but not when it is directed onto her cervicothoracic region.

AETIOLOGY

A motor vehicle accident 16 months ago.

EXAMINATION

The biceps (C5,6) and radial (C5,6,7) deep tendon reflexes were normal, as was the right triceps (C7) reflex. However, it was not possible to elicit the left triceps reflex. The knee jerk (L2,3,4) and ankle jerk (S1,2) deep tendon reflexes were normal.

Resisted tests for shoulder movement:

1. Abduction (C5–6) appeared normal bilaterally.
2. Shoulder shrug (spinal accessory and C3, 4 ± C5 nerves) appeared bilaterally equal but possibly diminished in strength and caused cervical spine pain bilaterally.
3. Tensioning the left and right brachial plexus nerves (C5–T1) elicited ipsilateral brachial plexus pain.

Adson's manoeuvre elicited contralateral cervical spine pain but no obliteration of the radial pulse.

Eye movements were normal, as were the optic fundi and discs. Auscultation of the heart and lungs was normal and the blood pressure in the right arm (seated position) was 118/82. The circumference of the forearms, 8 cm below the elbow joint, was 28.3 cm (left) and 28.8 cm (right). Pinprick sensation to the upper and lower extremities was normal, except for a subjective decrease in sensation in the C6 dermatome bilaterally between the thumb and index finger. Deep palpation of the paraspinal muscles of the neck elicited bilateral pain, particularly at the C1, 2 and C4–6 levels. There was also minor tenderness at approximately the T4, T12 and lumbosacral levels.

Cervical spine active ranges of movement were measured using a CROM instrument (see Box 52.1).

Left and right SLR were limited at 70° elevation due to minor low back pain.

IMAGING REVIEW

The cervical spine plain X-ray films had been reported as 'normal' but did not include functional views.

CLINICAL IMPRESSION

Whiplash-type syndrome due to soft tissue injuries particularly at the C1–2 and C4–6 levels.

WHAT ACTION SHOULD BE TAKEN?

Cervical spine flexion and extension views to augment the previous imaging; the report stated: 'Normal examination'. When the patient returned with the X-ray films she was in tears and said that 'nothing was found on the X-ray films'. She was shown the flexion and extension views and a long discussion ensued regarding particularly the extension view (Fig. 52.2A). The patient was told:

There is some backward displacement (retrolisthesis) of C3 vertebra on C4 and of the C4 vertebra on C5. The zygapophysial facet joint planes are parallel except at the C3–4

Box 52.1 Cervical spine active ranges of movement

	Normal range	Measured range	Comments
Flexion	50°	**40°**	Elicited pain to extend from C1 to C7 with a bilateral 'pulling' sensation in the back of the neck.
Extension	60°	**54°**	Elicited pain in the right cervico-shoulder region.
Left & right lateral bending	45°	**40.5°**	Elicited cervico-shoulder pain on the contralateral side.
Left & right rotation	80°	**72°**	Elicited pain in the ipsilateral cervico-shoulder region.

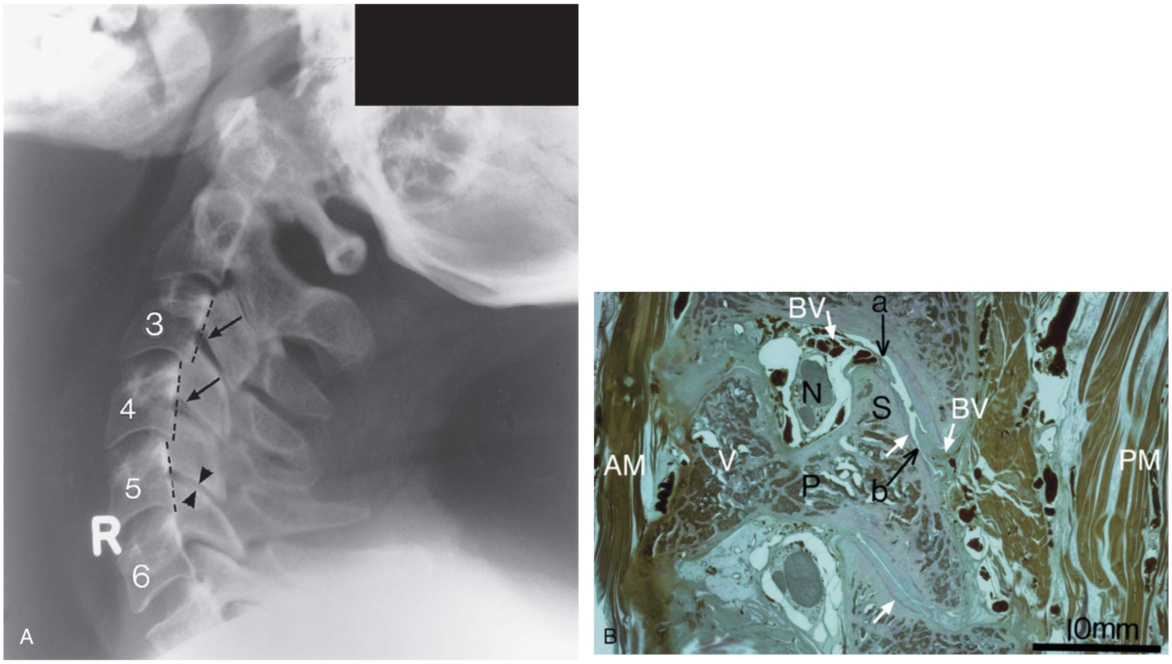

Figure 52.2 (A) Cervical spine lateral extension (functional) plain X-ray image. Note the: (i) retrolisthesis of the C3 vertebral body on the C4 body (as shown by the dotted line on the posterior aspect of the vertebral bodies) suggesting instability due to soft tissue injuries; (ii) retrolisthesis of C4 vertebral body on C5; and (iii) zygapophysial facet joint planes are parallel (for example, see arrowheads at the C5–6 facets) except at the C3–4 and C4–5 levels of instability where the zygapophysial joints 'gap' anteriorly (arrows). The soft tissues associated with the cervical spine are partly represented in (B). (B) Parasagittal section from a cervical spine showing part of the anterolateral aspect of the vertebral body (V) and the superior (S) and inferior (I) articular processes of the zygapophysial joint. Note the superior (a) and inferior (b) highly vascular synovial folds which can be nipped between joint surfaces during injury causing pain and bleeding into the joint. The lower long black arrow shows a joint with normal hyaline articular cartilage on each side of the joint's potential "space". The upper long back arrow shows a joint with osteoarthrotic wear in the hyaline articular cartilage on the superior articular process (S). AM = anterior spinal muscles; PM = posterior spinal muscles, respectively; BV = blood vessels within the intervertebral foramen and in the synovial folds in the upper and lower poles of the joint; N = neural structures in the intervertebral foramen which are surrounded by fatty tissue and many blood vessels. P = pedicle that joins the vertebral body (V) to the posterior bony structures; S = superior articular process and I = inferior articular process, respectively, that form the zygapophysial joint. (Reproduced with permission from Giles L G F 1986 Lumbo-sacral and cervical zygapophyseal joint inclusions. Manual Medicine 2: 89–92.) See also colour plate section Fig. vii.28B.

and C4–5 levels where the zygapophysial joints 'gap' anteriorly in keeping with the slight retrolisthesis of C3 on C4 and of C4 on C5.

She was told how certain soft tissue structures (Fig. 52.2B) may be injured.

DIAGNOSIS

Whiplash-type syndrome due to soft tissue injuries particularly at the C1–2 and C4–5 levels.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was delighted to find that there was indeed some evidence of soft tissue injuries to her neck and that she was not 'imagining the pain'; she was referred back to her general medical practitioner with an explanation of the functional X-ray findings. Her medical practitioner then referred her back for ongoing psychological support and treatment which consisted of needle acupuncture.

The needle acupuncture gave her significant relief, as long as it was performed in the seated position so that hyperextension of the cervical spine was not introduced (which would occur were she to lie prone).

Note

Cervical spine joint dysfunction may be completely missed if flexion and extension functional views are not ordered as suggested by Davis (1945), when no contraindication exists. Flexion/extension radiographs are able to detect disco-ligamentous instability in patients with otherwise normal radiographs. Normal plain radiographs should also be taken prior to obtaining the dynamic motion study (Chapman & Anderson 1997). The dynamic functional techniques should not be performed in the acute post injury phase (Chapman & Anderson 1997).

According to Jackson (1977), when there is no disorder of the cervical spine, flexion and extension result in a smooth curve. The stature of the cervical spine should not be interpreted on CT or MRI examinations because these are performed with the patient supine (Van Goethem et al 1998).

KEY POINTS

1. Flexion and extension functional views should always be taken to determine whether there is any cervical spine instability, unless there is a contraindication such as suspected vertebral artery injury with dizziness and nausea, or joint dislocation.
2. Patients receiving needle acupuncture for cervical spine injuries should preferably be treated in the seated position to eliminate hyperextension of the cervical spine unless the patient is comfortable lying on their side. (N.B. Ensure that the patient cannot fall off the chair.)

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See Case 27 for a further histological understanding of the cervical spine.

Case 53

Vertebral artery tortuosity

COMMENT

When a patient presents with unexplained headaches with apparent sympathetic nervous system involvement, consider intracranial and extracranial causes.

PROFILE

A 41-year-old male sedentary worker who does not smoke cigarettes and only drinks a small amount of alcohol.

PAST HISTORY

Suffered from 'cluster' headaches as a teenager until his late 20s.

PRESENTING COMPLAINT(S) (Fig. 53.1)

Thirteen-year history of suboccipital neck pain, of unknown aetiology, that radiates bilaterally to cause frontal headaches which occur mainly at weekends and can last all day. These are associated with nausea and dizziness. He does not suffer from photophobia or eye problems. The neck pain is worse when he is tired. All neck movements may increase the pain. His general health is very good.

Physiotherapy, traction, acupuncture and chiropractic manipulation had not given any long lasting benefit but non-steroidal anti-inflammatory drugs provide some relief. He said plain X-ray films of his neck and a CT scan were normal.

AETIOLOGY

Unknown.

EXAMINATION

He had undergone a full neurological examination that showed no obvious deficit; he had also had a neurosurgical opinion but a diagnosis was not made. Deep palpation of

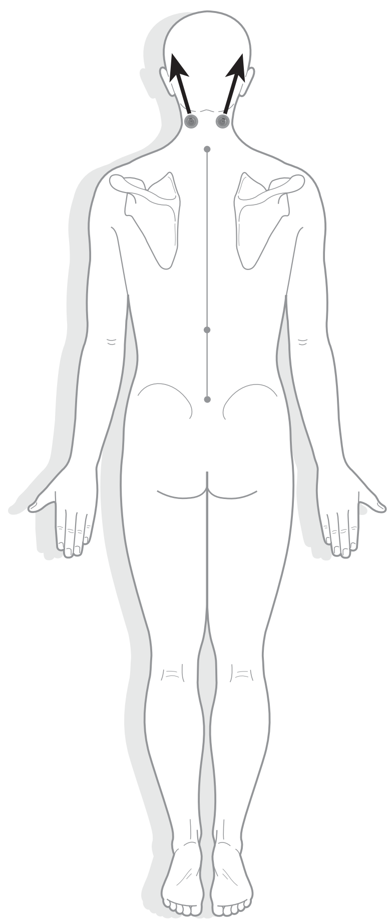

Figure 53.1

the upper cervical spine paravertebral muscles elicited bilateral tenderness. The blood pressure in the seated posture was essentially normal at 120/85.

IMAGING REVIEW

The plain X-ray report stated: ‘Disc spaces and neural foramina are normal. No fracture, bony infiltration or soft tissue abnormalities seen. No instability with flexion or extension, although there is some reduction in the range of flexion’. A review of the plain X-ray films that had essentially been reported as ‘normal’, showed a curvilinear line, indicating bone erosion, at C2 (Fig. 53.2) on the left side that had not been reported. This represented a tortuosity of the vertebral artery.

The CT scan from C3 to T1 showed no evidence of disc protrusion and was reported as a normal study. Unfortunately, the CT scan did not include the area of his main pain, i.e. the upper cervical spine.

CLINICAL IMPRESSION

Vertebral artery tortuosity at C2 with sympathetic plexus involvement.

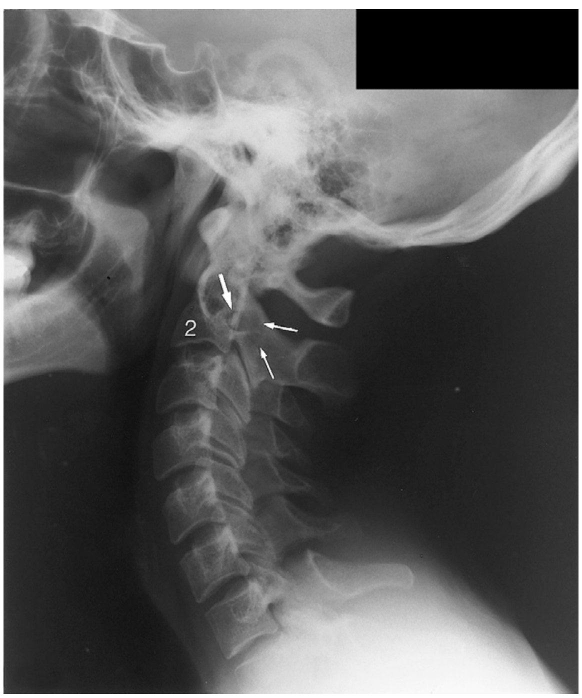

Figure 53.2 Cervical spine lateral plain X-ray image. The large arrow shows the C2 foramen transversarium and the two small arrows indicate the smooth curvilinear line caused by the vertebral artery tortuosity.

WHAT ACTION SHOULD BE TAKEN?

The patient was told that there were early sclerotic changes in the C2–3 and C3–4 zygapophysial joints but that the most likely cause for his headaches was irritation of the sympathetic nervous plexus on the vertebral artery at the area of the tortuosity.

DIAGNOSIS

Vertebral artery tortuosity at C2 on the left side.

TREATMENT AND RESULTS

The patient’s condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was grateful to know that a probable cause had been found, as he was concerned about various types of significant disease processes that may previously have been overlooked.

He was greatly relieved by the diagnosis and managed well on over-the-counter anti-inflammatory medication as required.

Note

It should be noted that the cervicothoracic (stellate) ganglion sends grey rami communicantes to various structures, including the seventh and eighth cervical and first thoracic spinal nerves and the vertebral arteries before extending to the basilar artery and as far as the posterior cerebral arteries, where they meet a plexus from the internal carotid (Harati 1993). Therefore, interference with the vertebral arteries may cause symptoms of vascular insufficiency of the posterior portion of the brain (Jackson 1977). Symptoms due to pressure upon the sympathetic nerve plexus surrounding the vertebral artery constitute a bizarre and confusing clinical picture – the Barre–Lieou syndrome (including headache, nausea, vertigo, nystagmus and suboccipital tenderness) (Hadley 1964).

KEY POINT

When a patient presents with autonomic nervous system symptoms, such as nausea and dizziness, there may be several causes, one of which is vertebral artery tortuosity.

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Case 54

Vertebral artery dissection

COMMENT

Always look carefully for abnormal anatomy.

PROFILE

A 34-year-old male soldier of muscular build who smokes 12–15 cigarettes per day and consumes only a modest amount of alcohol.

PAST HISTORY

Three years ago, while being driven during his work, he was a seat-belted passenger in a motor vehicle that rear-ended the vehicle in front at approximately 50 km/hour. At the time of impact his head was turned to one side. Within 15 minutes he experienced a 'burning' pain posteriorly in the neck. Three days later the pain became so bad that he required pethidine injections.

PRESENTING COMPLAINT(S) (Fig. 54.1)

Constant and significant neck pain with severe headaches and intermittent 'blackouts'; some symptoms of 'weakness' in his legs due to a motor vehicle accident 3 years ago. He recently experienced two blackouts from which it took him several minutes to recover. His symptoms caused him major depression as he had previously been fit and able to lead a perfectly normal and very active life. He had been diagnosed as having a minor 'cervical whiplash injury' due to a 'low-speed impact' and was referred by his medical practitioner for a further opinion.

Sneezing causes a significant increase in neck pain as does lifting relatively minor weights, e.g. 10–12 kg. The neck pain increases severely with all movements of the neck which are extremely limited by pain.

Non-steroidal anti-inflammatory drugs provide only limited relief. Panadeine Forte, Capadex and Valium in large doses, as well as Endone, only provide minor relief. He was also on an antidepressant medication for major depression that resulted from his significant cervical spine syndrome. Physiotherapy treatment was of no benefit. He had consulted a number of general medical practitioners including several specialists. One neurologist had found the fundi were clear, the cranial nerves were intact and examination of his limbs revealed no focal signs; his pulse was regular and he was normotensive and there were no cardiac murmurs or cervical

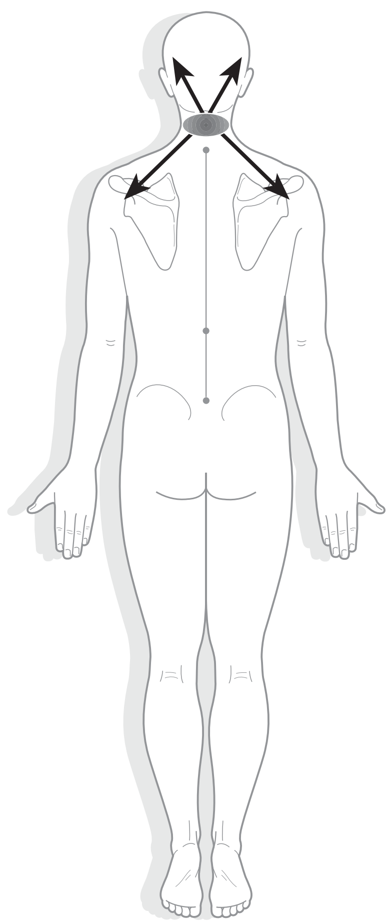

Figure 54.1

bruits. He said a plain X-ray examination of his neck and a CT scan had both been reported as normal.

AETIOLOGY

Motor vehicle accident 3 years ago.

EXAMINATION

All cervical spine ranges of neck movement were severely restricted due to a significant increase in pain. Neurological examination appeared to be normal. Abduction of the arms beyond 90° aggravated his neck pain.

IMAGING REVIEW

Plain X-ray anteroposterior, lateral and oblique views were reported as showing a 'normal examination'. However, on reviewing these films it was noted that there was assimilation of the atlas within the skull base. Because of the severity of his neck pain with restriction of all active ranges of movement, a delayed whole body bone scan with SPECT of the cervical spine had been performed. This was reported as a 'normal examination'.

CLINICAL IMPRESSION

Cervical spine soft tissue injuries including a vascular component that causes the 'blackouts' - query vertebral artery injury.

WHAT ACTION SHOULD BE TAKEN?

Plain X-ray films, including cervical spine extension and flexion functional views, were requested (Fig. 54.2A-E). The functional views (Fig. 54.2A and B) showed assimilation of the atlas within the skull base, with no obvious widening of the gap between the anomalous C1 posterior tubercle and the spinous process of C2 detected between these films; pain permitted only limited extension and flexion. The open mouth view showed incorporation of the atlas within the skull base (Fig. 54.2E), as did a CT reconstruction view (Fig. 54.2F).

A cervical spine MRI was requested and this showed that the vertebral bodies from C2 to T3 and the intervening disc spaces appeared intact. There was no disc protrusion and the cord appeared normal in diameter with no syrinx. However, there was posterior impression of the odontoid peg on the anterior aspect of the brain stem, with early flattening at the C1–2 level and effacement of the CSF with impingement on the anterior aspect of the cervicomedullary junction where the cord/brain stem was sharply angulated at the level of the foramen magnum (Fig. 54.3A and B, small black arrows).

In view of his severe symptoms, a carotid duplex ultrasound study was performed to ascertain whether there was basilar or vertebral artery stenosis. However, the examination was reported as being 'normal'. Therefore, an angiogram was ordered to look for vertebral artery injury but this procedure was denied by the radiologist on the grounds of being 'too risky'. In view of the patient's significant symptoms and depression he was referred to a psychiatrist for support while further steps were taken to have an angiogram performed elsewhere; this was arranged and showed dissection of both vertebral arteries.

DIAGNOSIS

Vertebral artery dissection bilaterally.

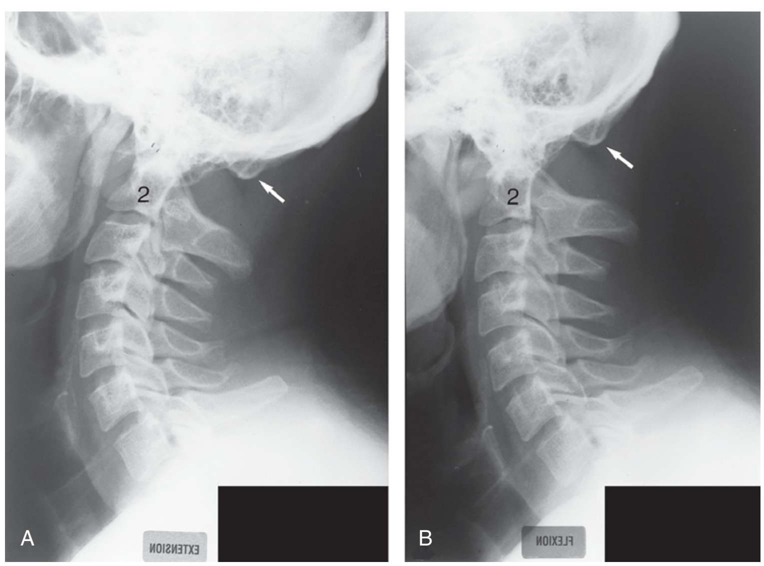

Figure 54.2 (A and B) Lateral cervical spine extension (A) and flexion images (B), respectively. Note the posterior arch of atlas is missing apart from the posterior tubercle (white arrows) that is assimilated into the occiput. Also note the very limited range of cervical spine extension and flexion as well as very limited skull/cervical spine movements.

(Continued)

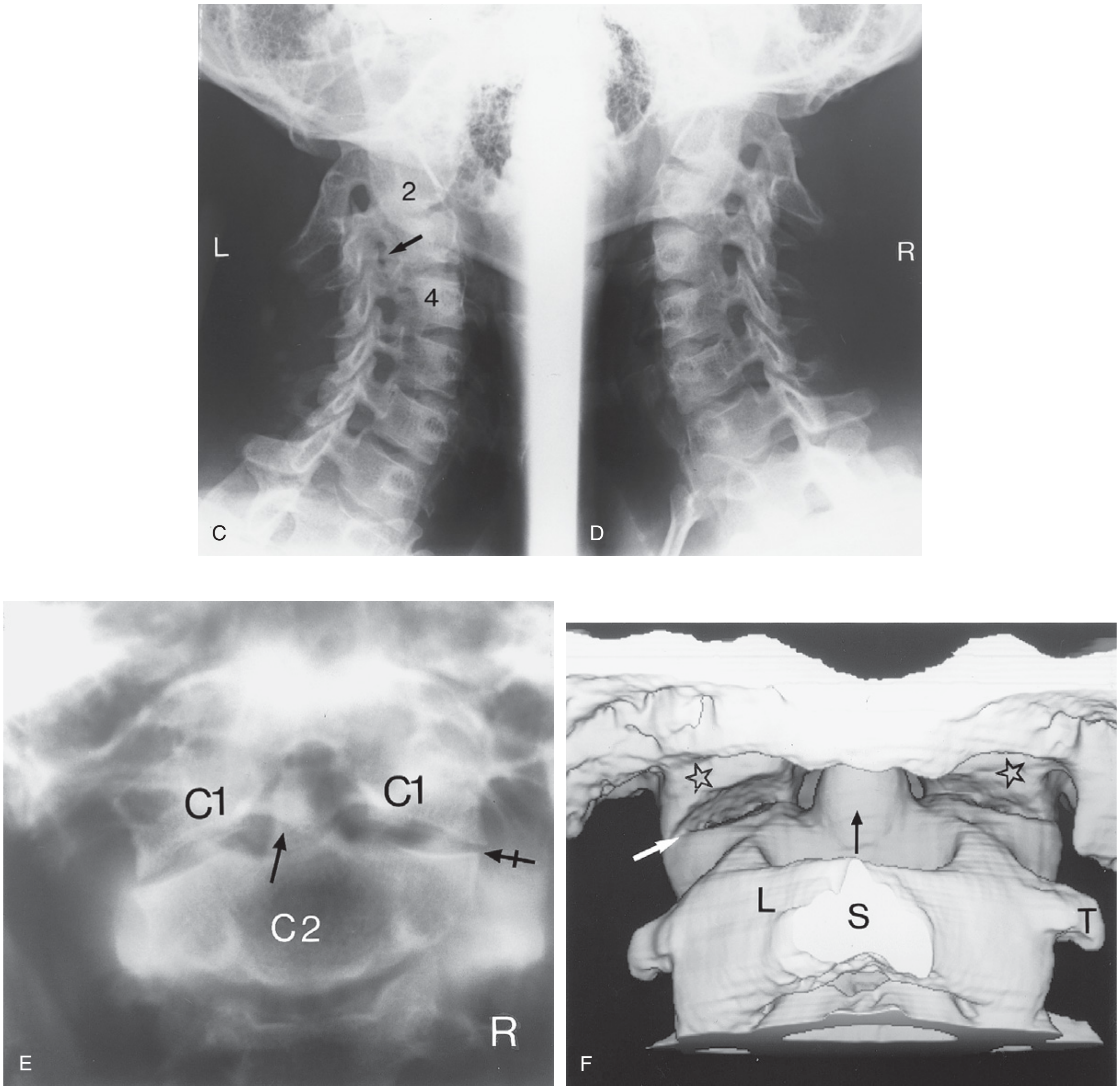

Figure 54.2 Cont'd (C and D) Cervical spine left (C) and right (D) oblique images, respectively. There is some encroachment of the C3–4 intervertebral foramen in (C) (arrow) due to osteophytic encroachment from the uncovertebral joint. (E) A cervical spine open mouth tomogram image that shows the C1 lateral masses (C1). The odontoid peg of C2 is seen (arrow) and the zygapophysial joints are normal between the assimilated atlas lateral masses and the lateral masses of C2 (tailed arrow). R = right side of patient. (F) CT cervical spine reconstruction showing complete incorporation of the C1 lateral masses (*) into the base of the skull. Black arrow = odontoid peg; white arrow = superior articular facet of the C2 vertebra. L = lamina of C2 vertebra; S = spine of C2; T = transverse process of C2.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised that there is no treatment for vertebral artery dissection other than psychological support, analgesics, relaxation techniques and advice to avoid performing head and neck movements that would aggravate his condition. Having provided a diagnosis for the patient this, in itself, was of great therapeutic value as he previously had been labelled as a malingerer.

Unfortunately, his chronic neck pain syndrome persisted but he was grateful that he had been taken seriously, at last, and that a diagnosis of organic pathology was confirmed and that he would no longer be considered a malingerer.

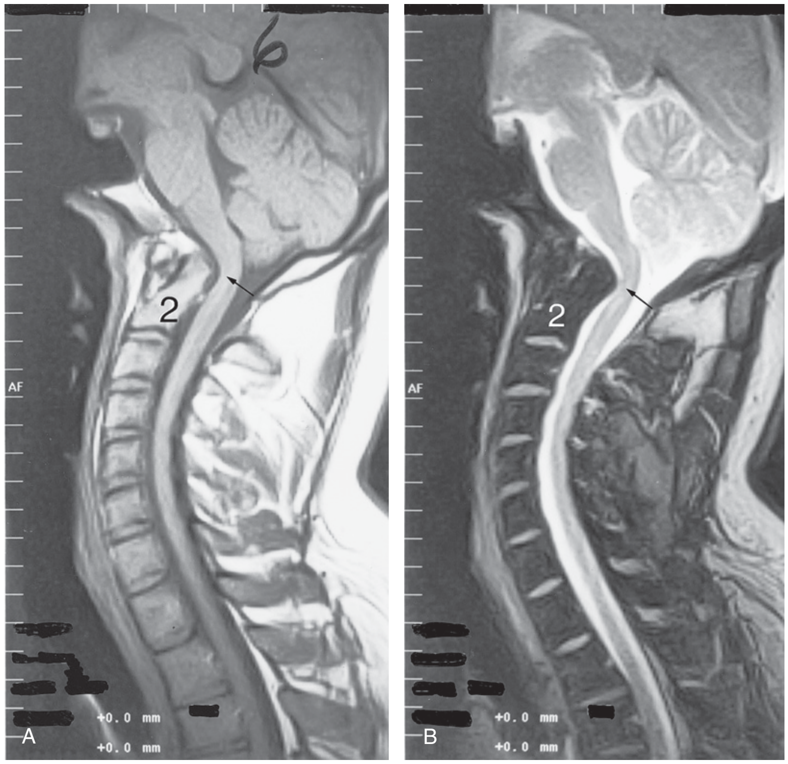

Figure 54.3 (A) Cervical spine MRI sagittal T2-weighted image. The second cervical vertebra (2) is shown with its odontoid peg impressing upon the anterior aspect of the brain stem/cord that is sharply angulated (small black arrow). (B) Cervical spine MRI sagittal T1-weighted image. The second cervical vertebra (2) is shown with its odontoid peg impressing upon the anterior aspect of the brain stem/cord that is sharply angulated (small black arrow).

Note

It seems unlikely that the low-speed impact would have caused any instability of the ligaments attached to the odontoid process. Therefore, it is possible that the posterior impression of the odontoid peg on the anterior aspect of the brain stem was apparently asymptomatic before the motor vehicle accident but it now may have affected the anterior spinal artery on the anterior surface of the spinal cord. Furthermore, the blackouts most likely relate to injury of the vertebral arteries (dissection) and their associated sympathetic nerve plexus.

As Professor Ruth Jackson (1977) pointed out, when a neck injury occurs with the head and neck rotated to one side, the injury will be more severe.

KEY POINTS

1. This again emphasizes how important it is for the clinician to look at the films and not only at the imaging report.
2. To miss the obvious C1 osseous anomaly could lead to disastrous consequences if the spine were considered to be normal and then manipulated. Hence, the wisdom of first looking at spinal structures to examine their integrity before providing manipulative therapy.
3. The vulnerability of the vertebral arteries to rotational movements should always be remembered.
4. When a clinician requests a particular type of imaging, it behoves the radiologist to discuss with the referring clinician the issue of whether the imaging should proceed. To summarily dismiss the request is tantamount to malpractice.

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Case 55

Post-manipulation stroke

COMMENT

Avoid rotational mobilization or manipulation of the cervical spine.

PROFILE

A 35-year-old male office worker of average build who smoked approximately ten cigarettes per day.

PAST HISTORY

He was reported to be very healthy and only occasionally suffered from a ‘stress’ related headache at which time he may take a paracetamol tablet.

PRESENTING COMPLAINT(S) (Fig. 55.1)

He presented to his general medical practitioner with neck pain and left trapezius muscle spasm following lifting at work approximately 4 days previously.

AETIOLOGY

Lifting a typewriter at work approximately 4 days previously.

EXAMINATION

Examination was recorded as showing no neurological signs or symptoms. A cervical spine provocation test was recorded as being negative but caused marked involuntary tremors and shaking with rigidity of the right shoulder and neck region.

IMAGING REVIEW

No imaging was performed.

DIAGNOSIS

The medical records showed a diagnosis of first cervical joint dysfunction with left trapezius muscle spasm. Apparently the patient was told by the medical practitioner that his neck stiffness was related to pinching of the synovial membrane in an upper joint of his neck, in turn causing neck muscle spasm.

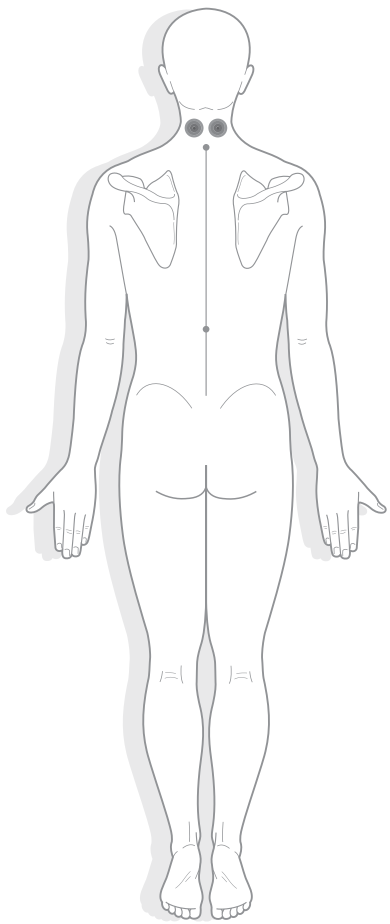

Figure 55.1

TREATMENT AND RESULTS

First visit (Tuesday 8.30 a.m. for approximately 15 minutes)

The treatment plan was for cervical spine mobilization. This was performed as a rotational movement of the cervical spine. He was advised to return the following day for a re-evaluation.

His wife said the first neck treatment had caused her husband great pain and she noticed that he was unsteady while walking. He told her that, following his treatment, he felt so unsteady that he could hardly sign the medical rebate form before he left the surgery; once outside, he fell then got up and went to his car where he sat for some time until he felt capable of driving home. Apparently, he was in considerable pain all night and took a number of paracetamol tablets. The next morning his wife thought that he felt a little better.

Second visit (Wednesday 8.30 a.m. for approximately 30 minutes)

He had continuing left trapezius muscle spasm and a diagnosis was made of dysfunction of the first and second cervical spine joints. The provocation test was repeated and found to be negative. An attempt to mobilize his neck was made but his neck was found to be too stiff and painful. He was given acupuncture treatment, a prescription for 5 mg Valium tablets, and advised to return 2 days later having taken Valium at night.

He told his wife, who had taken him to his appointment, that, although he felt alright, treatment by neck movements had, again, caused great neck pain.

Third visit (Friday – finished at 11.00 a.m.)

The medical records showed that he still had left facet joint problems and that he now presented with a headache. Provocation testing was negative, so cervical spine mobilization was repeated. He experienced a severe headache following the neck mobilization.

His wife took him to this third medical appointment and he told his wife that cervical spine mobilization was performed once again with a loud cracking noise. He told his wife that the treatment had hurt him more than before but that he was pleased that he would not have to return for more treatment. They walked approximately 200 metres to the car then his wife drove him home. He had lunch then, at approximately 12.30 p.m. he made a telephone call but aborted this, telling his wife that he had 'gone deaf' in his left ear during the telephone call. He then told his wife that he felt there was 'something wrong' with the left side of his body. He walked unaided to the bathroom but his wife noted that he kept knocking into the walls. On reaching the bathroom, he vomited several times. He then knelt on the floor and continued vomiting before collapsing at approximately 2.55 p.m. at which time his wife called an ambulance; by the time the ambulance arrived a few minutes later (3.02 p.m.), his eyes had rolled upwards and he was gasping for breath. The ambulance report stated that there was no sign of injury but that he had lost consciousness; he had a Glasgow Coma Scale (GCS) of 3, a pupil diameter of 1–2 mm, his eyes were half open with a glazed appearance, and he had Cheyne–Stokes respiration with poor tidal volume. He was given oxygen and taken to hospital, arriving at 3.30 p.m., Friday. The hospital records showed that he was deeply unconscious (GSC of 3) with the following examination findings being recorded:

1. Mid-size and reactive pupils.
2. No local neurological signs.
3. Flaccid paralysis.
4. No gag reflex.
5. Blood pressure of 165/95.
6. Pulse rate of 84 per minute.
7. Spontaneous breathing.
8. Afebrile.
9. Normal heart sounds without any murmurs.

He was intubated, ventilated and stabilized.

A tentative diagnosis was made of cerebro-vascular accident.

LABORATORY TESTS

The following laboratory tests were commenced at 5.30 p.m. on Friday and the results are reported in Boxes 55.1 to 55.4. Microbiology blood culture showed no organisms were grown, even 6 days later.

Box 55.1 Arterial blood gases/electrolytes

	Friday 5.30 p.m.	Reference range
Patient temp	33.6 °C	
FI O_2 (Fraction of inspired oxygen)	100.0%	
pH	**7.53**	(7.35–7.45)
pO_2	**274** mmHg	(80–100)
pCO_2	**32** mmHg	(36–44)
Bicarbonate	25 mmol/L	(21–28)
Base excess	**3.3** mmol/L	(−2.5 – +2.5)
(AI–art – Alveolar–arterial) pO_2*	**422** mmHg	(<15) *
O_2 Saturation	**99.2**%	(92–99)
Hb CO	0.8%	(<1.5)
MetHb	**0.3**%	(0.4–1.5)
tHb	152 g/L	(130–170)
Potassium	**2.9** mmol/L	(3.5–4.5)
Glucose	**14.1** mmol/L	(3.2–7.5)

*Reference range for AI–art gradient assumes patient is on room air.

Box 55.2 Plasma pathology

	Friday 5.30 p.m.	Saturday 9.30 p.m. 28 hours later	Reference range
Sodium	137	140 mmol/L	(135–145)
Potassium	3.5	3.4 mmol/L	(3.5–4.5)
Chloride	101	106 mmol/L	(100–110)
Bicarbonate	24	26 mmol/L	(22–28)
Anion gap	15	11	(7–16)
Calcium	2.24	2.28 mmol/L	(2.25–2.60)
(total)	2.18	2.27 mmol/L	(2.25–2.60)
(Alb Corr)			
Phosphate	0.64	0.62 mmol/L	(0.80–1.40)
Urea	5.7	4.5 mmol/L	(3.0–8.0)
Urate	0.25	**0.16** mmol/L	(0.25–0.50)
Creatinine	0.08	0.08 mmol/L	(0.06–0.12)
Urea/Creat	65	50	(40–100)
Glucose	**12.5**	7.1 mmol/L	(3.2–7.5)
Osmolality (calc)	293	291	(275–295)
Total bili	18	**31** umol/L	(0–20)
Protein (total)	71	72 g/L	(63–80)
Albumin	48	46 g/L	(35–50)
Globulins	24	26 g/L	(20–35)
Alk phos	86	92 U/L	(35–115)
LD	206	204 U.L	(120–250)
AST	21	33 U/L	(0–40)
ALT	18	21 U/L	(0–40)
Gamma GT	38	38 U/L	(0–60)
Creatine kinase	135	**303** U/L	(30–200)
Amylase	24	35 U/L	(20–100)

Box 55.3 Haematology

	Friday 5.30 p.m.	Friday 9.30 p.m. (4 hours later)	Saturday 9.30 a.m. (24 hours later)	Reference range
HB	166	153	169	(130–170)
RBC	5.31	4.73	5.33	(4.30–5.70)
PCV	0.483	0.433	0.490	(0.38–0.49)
MCV	92	92	93	(83–98)
PLT	360	343	384	(150–400)
WBC	**21.6**	**17.7**	**25.1**	(4.0–10.5)
NEUT	**19.94**	**16.45**		(1.9–6.8)
LYMP	**0.88**	**0.53**		(1.2–3.7)
MONO	0.66	0.72		(0.10–0.75)

The S.I. units used:
Hb g/L, RBC x 10E12/L, MCV fL, PLT x 10E9/L, WBC & Diff x 10E9/L.

Box 55.4 Coagulation profile

	Friday 5.30 p.m.	Saturday 9.30 p.m. (28 hours later)	Reference range
Platelet count	361	169	(150–400)
Prothrombin time	11	10	(Ctrl +/– 3)
PT control	11	11	
APTT	31	26	(25–38s)
Thrombin clotting time	17	12	(12–17s)
Fibrinogen	3.1	**4.7**	(1.5–4.0) g/L

IMAGING

At 7.00 p.m. on Friday, cervical spine (Fig. 55.2) and chest radiology was performed and reported as being normal – a nasogastric tube, a left sided subclavian central venous line and an endotracheal tube were noted. The radiology report stated '*Normal alignment; no evidence of acute trauma*'. A detailed description is shown in the caption for Figure 55.2.

Following the plain X-ray films, a pre- and post-contrast CT scan of the head was performed and reported as a 'normal study' showing 'normal ventricles and basal cisterns, no abnormal areas of a hemiation or contrast enhancement and no midline shift'.

At 6.30 a.m. on Saturday an MRI of the brain and cervical spine was performed that showed tonsillar herniation with the tip of the tonsils at the level of the posterior arch of atlas (Fig. 55.3) and marked mass effect present in the posterior fossa due to extensive oedema involving the cerebellar cortex bilaterally.

At the same time an MRA brain (two scans shown in Fig. 55.4A and B) was performed that showed abnormal signal inferomedially along the right occipital lobe extending medially into the right temporal lobe. Bilateral foci of abnormally increased signal intensity are present in both thalami. Both lateral ventricles and the third ventricle are dilated – the fourth ventricle is normal in size. There was no normal flow void present in the basilar artery as shown by the MRA or the axial T2-weighted scans. The small calibre left vertebral artery, and presumably larger, dominant, right vertebral artery, are both patent to the level of C2. Cervical spine axial T1 scans show an appearance suggesting dissection of the left vertebral artery (shown by an apparent double signal). Conclusion: unequivocally no flow in the basilar artery with extensive cerebellum infarction in the territory of the right posterior cerebral artery. Subsequent mass effect with tonsillar herniation and acute hydrocephalus. Some evidence of blood flow in both vertebral arteries up to the C2 level

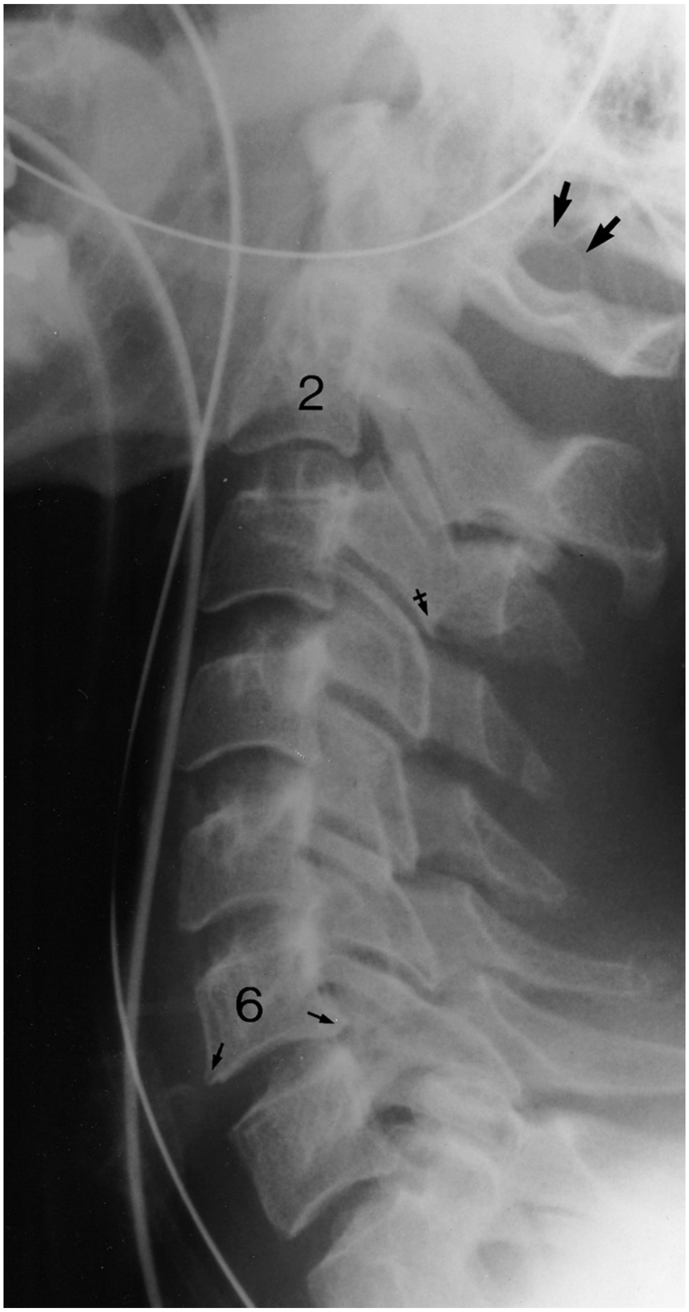

Figure 55.2 Cervical spine lateral plain X-ray image shows (i) a C1 arcuate foramen on at least one side (large black arrows), (ii) early osteophytic lipping on the antero-inferior and postero-inferior margins of the C6 vertebral body (small arrows), and (iii) early osteophytic lipping of the C3 inferior articular facet (small tailed arrow). An endotracheal tube, a left sided subclavian central venous line and a nasogastric tube are seen.

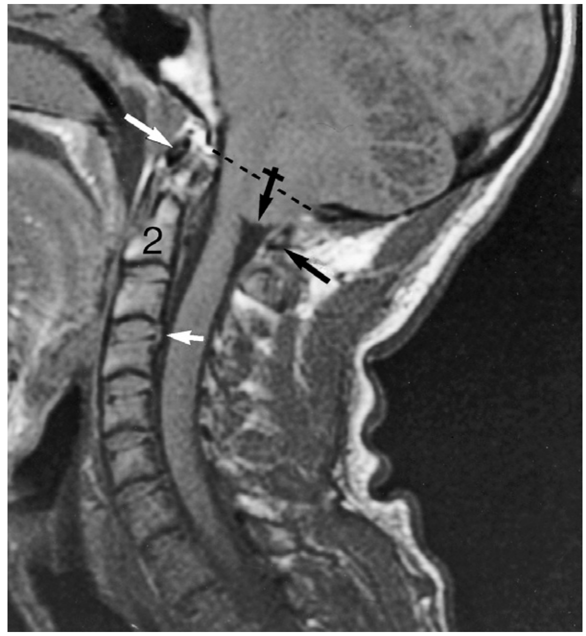

Figure 55.3 Cervical spine MRI sagittal T1-weighted image. Note (i) the anterior and posterior tubercles of atlas (large white and black arrows), (ii) the anterior and posterior margins of the foramen magnum joined by a broken line, (iii) the cerebellar tonsillar herniation through the foramen magnum with the tip of the tonsils at the level of the posterior arch of atlas (tailed black arrow) due to acute hydrocephalus, and (iv) a small posterior disc bulge of the C3–4 intervertebral disc (short white arrow).

with features suggesting possible dissection of the left vertebral artery at the cranio-cervical junction.

On reviewing the MRI and MRA films the radiologist stated that there is 'no evidence for a primary basilar thrombosis or a vertebral artery thrombosis as a cause for the infarction and … a dissection would appear to be entirely possible…'. In fact, the radiologist stated that a dissection was 'more likely' as there was 'no concrete evidence for a thrombosis'. However, because both the MR and MRA imaging were performed within 24 hours of the stroke occurring, it was concluded that there is no 'really reliable signal characteristic to say unequivocally this is a dissection of the vertebral artery'.

The radiologist went on to state that, with MRI it is possible to diagnose dissections of arteries by relying on picking up blood located actually within the arterial wall, as blood that leaks out of the artery into tissues becomes deoxygenated blood and that has very well defined signal characteristics on T1-weighted, T2-weighted and Gradient Echo scans. Therefore, by 3–5 days post dissection, formation of methaemoglobin, (which has very well defined characteristics on T1-weighted images and shows up as white making it very easy to diagnose a dissection at that time as a doughnut appearance, can be seen i.e. a white doughnut appearance.

The patient did not regain consciousness and died at approximately 8.40 a.m. on Sunday, i.e. within 46 hours post neck manipulation (including a 24-hour period on a life support system).

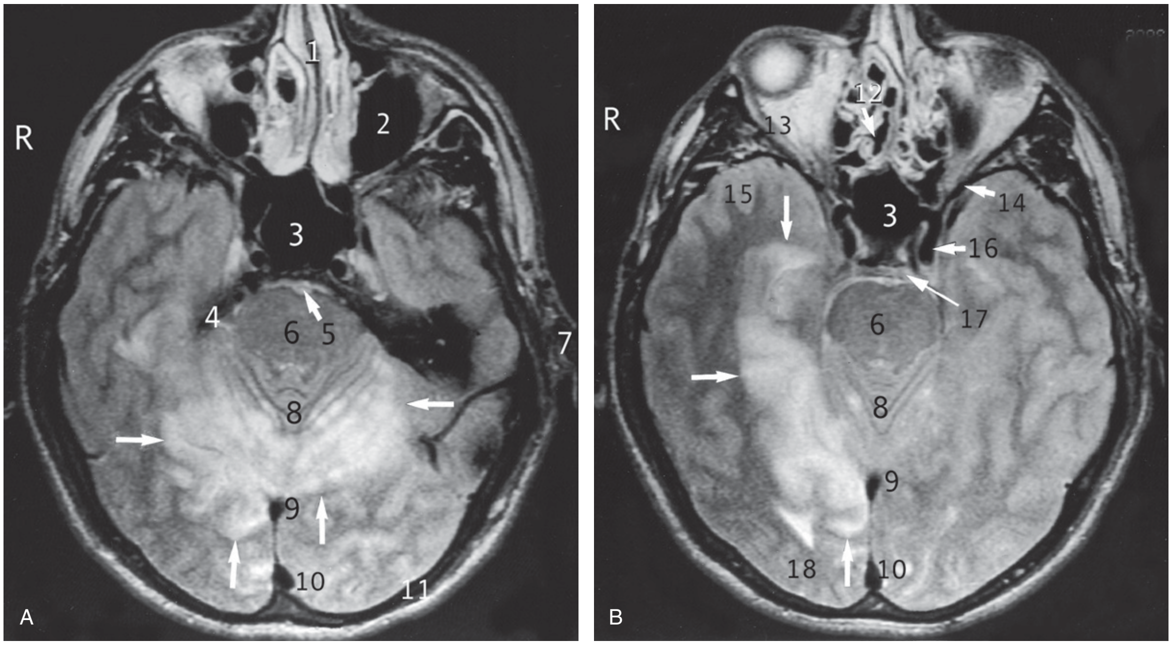

Figure 55.4 (A and B) MRA brain axial T2-weighted images. Arrows without numbers show parts of the brain with abnormal signals. 1 = nasal septum; 2 = maxillary sinus (antrum); 3 = sphenoidal sinus; 4 = posterior cerebral artery; 5 = basilar artery; 6 = pons; 7 = external acoustic meatus; 8 = cerebellum; 9 = straight sinus; 10 = superior sagittal sinus; 11 = occipital bone; 12 = ethmoidal sinus air cell; 13 = lateral rectus muscle; 14 = greater wing of sphenoid bone; 15 = temporal lobe; 16 = internal carotid artery; 17 = dorsum sellae; 18 = occipital lobe.

SUMMARY OF POSTMORTEM EXAMINATION REPORT

Brain macro

1. External appearance:
   a. *Convexities*. Normal leptomeninges, cerebral hemispheres are symmetrical with marked swelling and flattening of the gyri and cleft-like sulci.
   b. *Base of brain*. Normal leptomeninges. Normal basal frontal lobes, with areas of haemorrhagic softening in the inferior temporal gyri bilaterally measuring approximately 1.5 cm in diameter and extending bilaterally backwards to the inferior occipital lobes. Midline structures are displaced downwards approximately 5 mm. Arteries at the base of the brain show a normal anatomical circle of Willis, and are uniformly thin walled and soft. Petechial haemorrhages are seen in the right vertebral artery.
2. Brain sections. Normal sized ventricles. Intact septum pellucidum and normal corpus callosum. Haemorrhagic infarction is noted within the posterior cerebral artery territories bilaterally but more so on the right side. This involves inferior temporal gyri bilaterally and extends into the medial occipital lobe structures. The small area of approximately 5 mm in diameter of haemorrhagic infarction is seen in the superior portion of the right rostral thalamus.
   a. *Brainstem*. The brainstem is softened and haemorrhagic externally. Also there is haemorrhagic infarction on the basis pontis, extending the full length of the pons; at the caudal end there are haemorrhages present within the medulla in the intermediate regions as well as in the floor of the fourth ventricle. Areas of haemorrhagic discoloration and softening are noted in the midbrain in the tectal plate.
   b. *Cerebellum*. The cerebellum is swollen, softened and extremely congested with tonsillar herniation and necrotic cerebellar tonsils pouting down over the medullary spinal junction. There is virtually total haemorrhagic infarction involving both inferior and superior arterial territories.

Brain micro

Brainstem. This shows very early infarction with haemorrhage affecting the medulla, pons and midbrain.
Cerebellum. There is extensive acute haemorrhagic infarction.
Left medial occipital cortex. This shows acute infarction.
Hippocampus. Both sides show extensive acute ischaemic neuronal necrosis and the parahippocampal neocortex is also affected.
Thalamus. Both sides show extensive areas of acute infarction.
Basal ganglia and insula. Intact.

Cingulate gyrus. A small area of acute cortical infarction is seen in the left cingulate cortex.

Arterial border zone territory. Minor cytological changes are seen and may reflect very ischaemic damage or autolysis.

Basilar and vertebral arteries. Arterial structure is normal with no evidence of dissection or thrombosis. (Note: This refers to the vertebral arteries visible within the skull and not the vertebral arteries outside the skull.)

Heart

Very subtle endocarditis consisting of bright red vegetations measuring 1–2 mm in diameter were noted along the line of closure of the mitral valve. Normal coronary arteries were present apart from the origin of the left anterior descending coronary artery that demonstrates approximately 70% narrowing due to yellow atheroma.

Histology

No evidence of myocarditis, ischaemic fibrosis or recent infarction. Focal haemorrhage into a papillary muscle is noted. No evidence is seen of active infective endocarditis. Healing, haemosiderin-rich nodules are seen.

Lungs

The lungs show bilateral congestion and oedema with an area of mucopurulent exudate on the base of the left lung.

Histology

Bilateral pulmonary oedema and congestion.

Larynx, trachea and main bronchi

Mildly congested.

Trachea histology

Congestion and focal haemorrhage.

Pancreas, kidneys, liver, skin

All histologically normal.

Comment

Death resulted from cerebellar and brainstem infarction, the histological appearances of the infarction being consistent with a duration of approximately 1 day. The causative vascular lesion was not found on histological examination but certainly related to the neck manipulation with damage to the vertebral arteries.

Neuropathological diagnosis

Acute brainstem and cerebellar infarction; survival of 46 hours post neck manipulation including 24 hours on life support system.

Cause of death

(1) Trauma to vertebral artery, (2) acute brainstem and cerebellar infarction.

Unfortunately, the hospital chart recorded that the general medical practitioner had performed 'chiropractic manipulation'.

Note

MRI is better than CT for the detection of acute ischaemia and can detect acute and chronic haemorrhage; therefore, it should be the preferred test for accurate diagnosis of patients with suspected acute stroke (Chalela et al 2007). For stroke patients treated 3–6 hours after onset, baseline MRI findings can identify subgroups that are likely to benefit from reperfusion therapies and can potentially identify subgroups that are unlikely to benefit or may be harmed (Albers et al 2006).

KEY POINTS

1. Clinicians performing spinal manipulation therapy (SMT) should avoid performing rotational SMT of the cervical spine, as rotational SMT has been associated with post-manipulation vertebrobasilar stroke (Terrett 1998).
2. Any adverse reaction to cautions cervical spine provocation testing should be taken as a danger sign and no cervical spine SMT should be performed in such cases.
3. Should a patient experience any symptoms suggesting cerebrovascular compromise post-SMT, further SMT is contraindicated.
4. Unfortunately, there is no pre-manipulation screening test that can predict with certainty that a patient is not at risk from such treatment.
5. Should a patient complain of pain during a neck mobilization or manipulation it makes sense not to proceed with that particular form of treatment.
6. There is no substitution for skill, knowledge and finesse and a lack of diagnostic and manipulative skills should be considered a definite contraindication to cervical spine SMT (Gatterman 1998).

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Further reading

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Case 56

Brain injury

COMMENT

Patients involved in neck and closed head injuries may well sustain brain and pituitary stalk injuries.

PROFILE

A 26-year-old female light duties worker who does not smoke cigarettes and occasionally drinks alcohol.

PAST HISTORY

Ten years ago she was involved in a severe motor vehicle accident in which she suffered cervical spine injuries as well as a closed head injury. She had seen a neurologist who found the cranial nerves to function normally and there were no other neurological findings. However, it was subsequently found that, during the accident, the pituitary stalk was transected with the development of associated hormonal problems, e.g. thyroid, corticosteroid, estrogen, testosterone, growth hormone, ACTH, TSH, FSH, and somatocrinin (hypothalamic growth releasing hormone-GHRH) that caused considerable tiredness, apart from other physiological problems.

PRESENTING COMPLAINT(S) (Fig. 56.1)

Chronic bilateral cervical spine pain causing headaches of significant intensity and bilateral cervico-shoulder region pains due to the motor vehicle accident. The headaches are particularly troublesome when her neck is feeling more painful than usual. Her neck pain syndrome is activity related, with increased activity causing an increase in her symptoms. There is no night pain per se. In particular, looking down and looking up aggravate her neck pain syndrome. She is under constant treatment by an endocrinologist but wants an opinion regarding her neck pains in particular as she finds that working at a computer considerably aggravates her neck pain syndrome.

AETIOLOGY

A motor vehicle accident approximately 10 years ago as noted above.

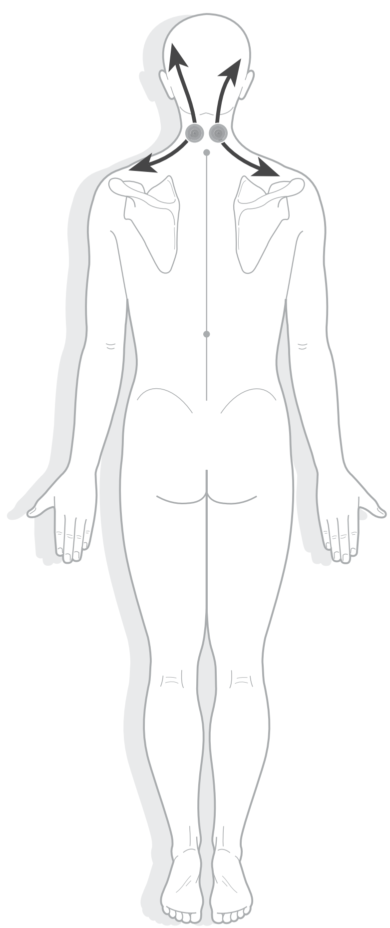

Figure 56.1

EXAMINATION

The deep reflexes in the upper and lower extremities were normal. Pinprick sensation in the upper and lower extremities was normal as was muscle power. Vibration sensation at the elbows and ankles was normal. Deep palpation of the paravertebral muscles elicited local pain. There was no atrophy of muscles in the neck or shoulder girdle regions. The Valsalva manoeuvre did not cause any neck pain. Hoffman's sign was normal. Cervical spine active ranges of movement were of full range but with complaints of pain at the extremes. Cranial nerve function was normal.

IMAGING REVIEW

An initial plain X-ray of the skull and cervical spine did not show any bony injury. However, an MRI brain revealed disruption of the pituitary stalk just distal to its origin. An MRI brain performed 9 years later confirmed that the remainder of the pituitary stalk and the pituitary gland had both undergone profound degeneration, confirming the absolute state of panhypopituitarism. The latter MRI brain also showed symmetrical cortical atrophy of the frontal and parietal lobes of the vertex but no focal areas of encephalomalacia, demyelination, cerebral infarction or prior brain haemorrhage. There was no evidence of prior haemorrhagic contusion or axonal shear type injury and the major intracranial vessels were patent. There was no mass or fluid collection.

CLINICAL IMPRESSION

Musculoligamentous soft tissue injuries of the cervical spine apart from the well documented brain injuries.

WHAT ACTION SHOULD BE TAKEN?

Laboratory tests were not performed as she was in the care of an endocrinologist and a specialist physician who constantly monitor her condition. A set of current plain film cervical spine radiographs, including functional views, and a cervical spine MRI were requested.

The plain film cervical spine radiographs taken approximately 9 years following the motor vehicle accident showed the following.

Neutral lateral view:

- Subchondral sclerosis of the zygapophysial (facet) joints at the C3–4, C4–5 and C5–6 levels (Fig. 56.2A and B).
- Early osteophytic lipping on the vertebral body margins adjacent to the intervertebral disc spaces posteriorly at

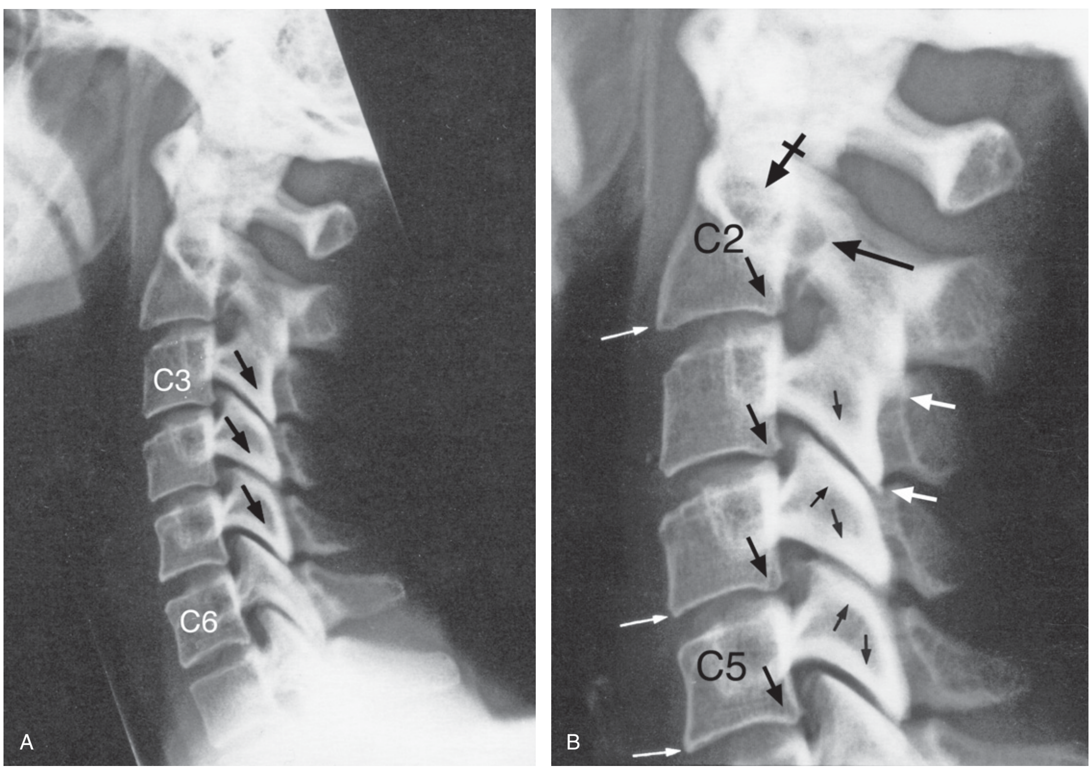

Figure 56.2 (A) Cervical spine neutral lateral image. Note the early subchondral sclerosis of the zygapophysial (facet) joints at the C3–4, C4–5 and C5–6 levels (small arrows) in particular. (B) An enlargement of the levels C1 to C5 to better illustrate the changes. Note (i) the early osteophytic lipping on the vertebral body margins adjacent to the intervertebral disc spaces posteriorly at C2–3, C3–4, C4–5 and C5–6 (large black arrows), (ii) early lipping at the antero-inferior margins of C2, C3, C4 and C5 vertebral bodies (white arrows), and (iii) developing osteoarthrotic spurs at some of the zygapophysial joints, e.g. at C2–3 and C3–4 in particular. The radiolucent circle at the C2 level (long black arrow) shows that the pulsation of the vertebral artery has caused some erosion of the bone. (This is not a superimposed normal foramen transversarium – that is seen over the C2 body (tailed arrow).)

C2–3, C3–4, C4–5 and C5–6 (Fig. 56.2B), with early lipping at the antero-inferior margins of C2, C3, C4 and C5 vertebral bodies (Fig. 56.2B).

- Developing osteoarthrotic spurs at some of the zygapophysial joints, e.g. at C2–3 and C3–4 in particular (Fig. 56.2B).
- There is a radiolucent circle at the C2 level that shows that the pulsation of the vertebral artery has caused some erosion of the bone (Fig. 56.2B).

Extension lateral view:

- There is some retrolisthesis of the C3 body on C4 and of C4 on C5, suggesting instability at these levels due to disc and ligamentous injures (Fig. 56.3).

Flexion lateral view:

- There is a loss of the normal cervical spine contour with an 'angulation' at the C4 level. There is some anterolisthesis of the C3 body on the C4 body, and at C4 on C5 and at C5 on C6, suggesting instability at these levels due to disc and ligamentous injuries (Fig. 56.4).

The MRI cervical spine sagittal T2-weighted image (Fig. 56.5) showed:

- A posterior disc herniation at the C3–4 level.
- Some desiccation causing darkening of the intervertebral discs at C3–4, C4–5 and C5–6 as compared to the discs at C2–3 and below C6.

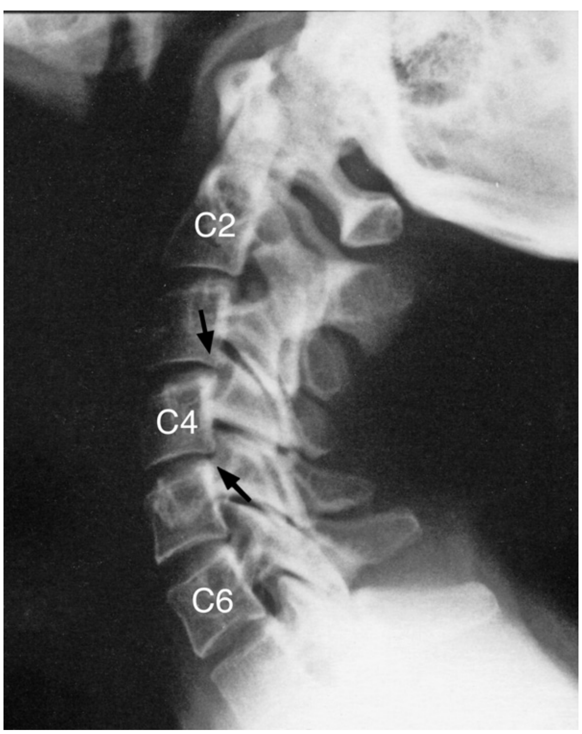

Figure 56.3 Cervical spine extension lateral image. Note the retrolisthesis (black arrows) of the C3 body on C4 and of C4 on C5, suggesting instability at these levels due to disc and ligamentous injures.

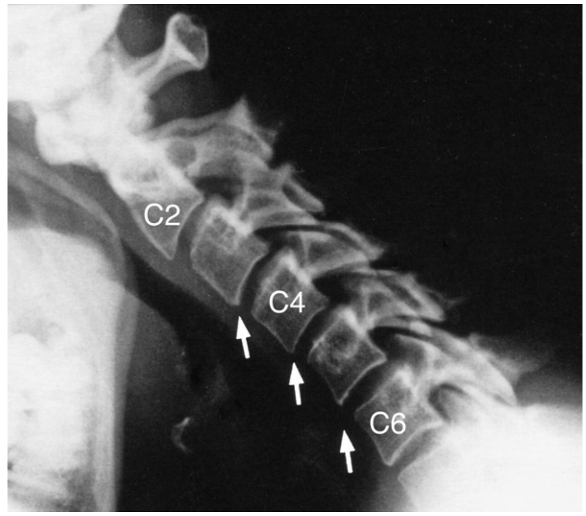

Figure 56.4 Cervical spine flexion lateral image. Note the loss of the normal cervical spine contour with an 'angulation' at the C4 level. There is some anterolisthesis of the C3 body on the C4 body, and at C4 on C5 and at C5 on C6 (white arrows), suggesting instability at these levels due to disc and ligamentous injuries.

Figure 56.5 Cervical spine MRI sagittal T2-weighted image. Note (i) the posterior disc herniation at the C3–4 level (black arrow), and (ii) some desiccation causing darkening of the intervertebral discs at C3–4, C4–5 and C5–6 (white arrows) as compared to the discs at C2–3 and below C6. There is a suspicion of very early posterior disc bulging at the C5–6, C6–7 and T1–2 levels (small black arrows).

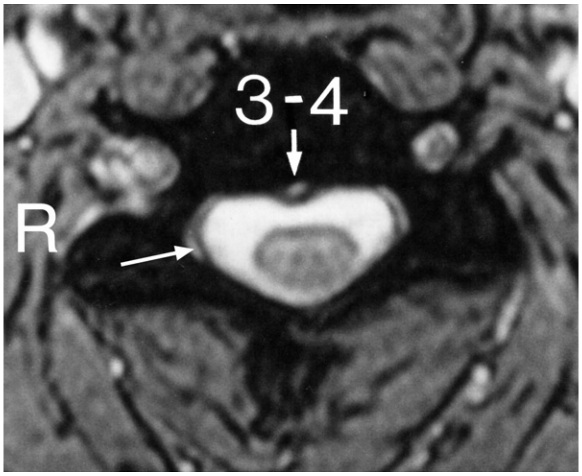

Figure 56.6 Cervical spine MRI axial T2-weighted C3–4 image. Note the central posterior disc protrusion (small white arrow). Although the protrusion does not compress the spinal cord itself, it does efface the pain sensitive anterior part of the dural tube. (The lateral part of the dural tube is shown by the long white arrow.)

- There is a suspicion of very early posterior disc bulges at the C5–6, C6–7 and T1–2 levels.
- A C3–4 central posterior disc protrusion is seen on the axial T2-weighted image (Fig. 56.6). Although this protrusion does not compress the spinal cord itself, it does efface the pain sensitive anterior surface of the dural tube.

DIAGNOSIS

- A transected pituitary stalk and its associated hormonal problems.
- Moderate symmetrical cortical atrophy of the frontal and parietal lobes of the vertex.
- Significant soft tissue injuries and early osteoarthrotic changes in the joints of the cervical spine.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was told that (i) she had been left with early osteophytic degenerative changes in her cervical spine that indicate that the attachment of some discs and ligaments to the vertebral bodies were damaged; (ii) the sclerotic changes in the subchondral bone of the zygapophysial joint facets indicate injury to these joints; and (iii) the C3–4 posterior central disc protrusion, while not touching the spinal cord, presses upon the pain sensitive anterior part of the dural tube, upon the recurrent meningeal nerves, and upon the pain sensitive blood vessels between the herniation and the dural tube, causing pain. The significance of the vertebral artery tortuosity causing some bony erosion at the C2 vertebral level was discussed.

She was told that, because of these plain X-ray and MRI cervical spine findings, her neck is vulnerable to postural stresses and strains such as looking up, or looking down.

Following an explanation of likely pain generators she accepted that she should not perform activities that aggravate her cervical spine pain syndrome, that she should perform gentle cervical spine exercises that do not aggravate her symptoms and that she should consider ergonomic issues with regard to sitting during computer activities, etc. She was advised not to allow the hairdresser to wash her hair with her neck and head extended backwards over the washbasin.

At follow-up 3 months later she said she was coping satisfactorily with her various symptoms by taking her prescribed hormone medication, having massage to her posterior neck muscles, performing gentle neck exercises and being careful not to undertake any activities that may aggravate her cervical spine pain syndrome. This has continued to be the case some 12 years following the motor vehicle accident.

She will require constant medical management in view of her transected pituitary stalk and the associated hormonal problems.

Note

Laboratory tests for pituitary hormone function to exclude any deficit that can occur in people with severe head injury, i.e. tests for FSH, LH, prolactin, thyroid function tests and gonadotrophins, should be considered for patients involved in closed head injuries.

KEY POINT

Patients sustaining severe whiplash injuries in motor vehicle accidents should be screened for pituitary function if there is any suggestion of possible hormonal dysfunction.

Further reading

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Case 57

Intervertebral disc protrusion

COMMENT

This case demonstrates how important it is for clinicians treating spinal pain syndromes to be well versed in the anatomy, symptoms and signs related to a particular part of the spine.

PROFILE

A 46-year-old married man who has always been involved in manual activities. He does not smoke and only drinks beer socially.

PAST HISTORY

Following a motor vehicle accident 10 years ago he developed left upper limb radicular symptoms of pain with 'tingling' in the middle finger (C7). Symptoms came on immediately following the motor vehicle accident and for 3 years he saw several specialists who gave him the impression that the pain was psychosomatic. Eventually, he was referred to a psychiatrist by an orthopaedic surgeon, which the patient told me was 'the best advice he had ever been given'. During the psychiatric evaluation the psychiatrist told the patient that he knew exactly what was wrong, so the patient asked the psychiatrist how he knew this. The reply was that the psychiatrist had also had a motor vehicle accident and ruptured a disc in the lower part of his neck and been told that he was imagining his problem, having seen various specialists, until he found an orthopaedic surgeon who examined him with MRI and found a disc prolapse in his neck! The patient was referred by the psychiatrist to his orthopaedic surgeon who performed a Cloward procedure at the C6–7 level which very successfully resolved the left sided neck pain, radicular symptoms and the tingling in the middle finger.

PRESENTING COMPLAINT(S) (Fig. 57.1)

Right sided neck pain with some left sided neck pain.

He had been referred from interstate with a request that an 'appropriate diagnosis be made without the

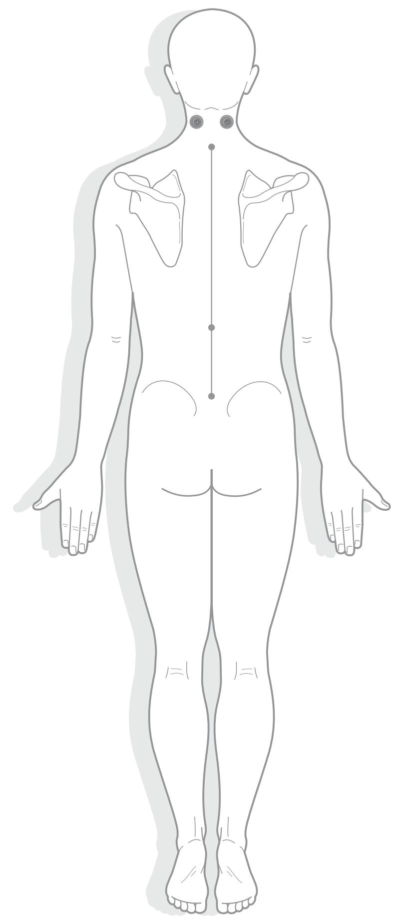

Figure 57.1

patient going through the 3 years of indecision that occurred previously' when the symptoms were on the left side.

AETIOLOGY

A motor vehicle accident 10 years ago when a sedan approaching from the opposite direction hit the patient's four-wheel-drive vehicle head on with such force that the four-wheel-drive's chassis was severely damaged. He immediately felt neck pain with pain extending into his left shoulder/arm.

EXAMINATION

Deep palpation of the paravertebral muscles of the cervical spine elicited pain on the right side at the occiput-C1 level and bilaterally at the C4–7 levels. Toe walking power (S1) and heel walking power (L5) were normal. The deep tendon reflexes in the upper extremities were normal but the ankle jerks (S1) were somewhat diminished bilaterally at one plus (two plus being normal). Pinprick sensation of the upper and lower extremities was normal. Vibration sensation at the elbows and ankles was normal. Power in the upper and lower extremities appeared to be normal.

All cervical spine active ranges of movement were restricted by approximately 50%. Cervical traction and compression did not aggravate his neck pain. The circumference of the forearm, 12 cm below the elbow, was 33.5 cm bilaterally.

IMAGING REVIEW

His previous MRI imaging was reviewed and this showed a large disc protrusion at the C6–7 level prior to surgery (Fig. 57.2A).

CLINICAL IMPRESSION

Central disc protrusion above the spinal fusion level at C6–7 (as there are no unilateral radicular symptoms).

WHAT ACTION SHOULD BE TAKEN?

A new MRI was requested and this showed protrusions at the C5–6 and C3–4 levels, both of which impinge upon the pain-sensitive anterior part of the dural tube (Fig. 57.2B and C).

DIAGNOSIS

- Chronic bilateral neck pain following a whiplash type injury that required a C6–7 Cloward procedure.
- Subsequent C3–4 and C5–6 posterior intervertebral disc protrusion.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised not to perform any

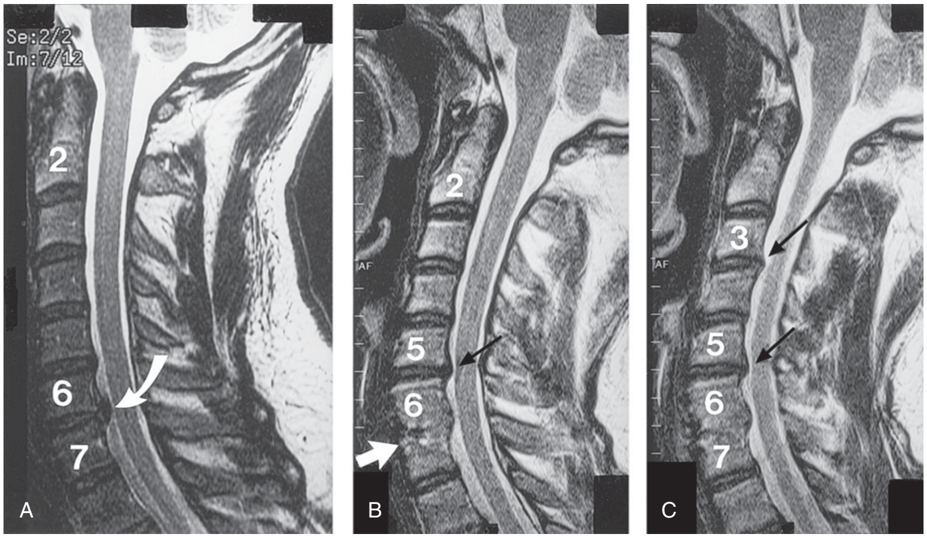

Figure 57.2 (A) Cervical spine MRI sagittal T2-weighted image. The numbers represent the vertebral body level and the curved arrow shows the large C6–7 disc protrusion that considerably indents the pain-sensitive anterior surface of the dural tube and almost touches the spinal cord itself. (B, C) Cervical spine MRI sagittal T2-weighted images. The numbers represent the vertebral bodies and the white arrow shows where surgery was performed at the C6–7 level. The black arrows show the disc protrusions at the C3–4 and C5–6 levels, respectively. Note how the disc protrusions indent the pain-sensitive anterior surface of the dural tube.

activities that would aggravate his neck pain. He was also advised to sleep with a pillow of appropriate thickness, to fill the gap between his shoulder and the side of his neck when he lies on his side in bed. He was told that he should consider consulting the orthopaedic surgeon who had operated on his C6–7 level should his symptoms reach a stage where he required a surgical opinion. Having been shown his MRI images, and advised that prevention was better than cure, he went home satisfied that he knew what was wrong with his neck and he was determined to protect it by not performing activities that could worsen the disc lesions at the C3–4 and C5–6 levels.

Some months post consultation he continues to be active while being careful not to strain or jar his neck.

Note

It is interesting to note that on the slice shown in Fig. 57.2B, the C3–4 disc looks almost normal, whereas in Fig. 57.2C the protrusion is clearly shown. This variation is because the slices are through different sections of the spine and this example shows why sagittal plane scans begin on one side of the spine and finish on the other side of the spine so that any protrusions between the left and right sides can be seen, as not all protrusions are central.

KEY POINTS

1. Bearing in mind the issue of dermatomal and dynatomal pain patterns (Slipman et al 1998) discussed in the Introduction, it should be possible to make a clinical diagnosis of central or posterolateral cervical disc protrusion. MRI is a very useful tool for suspected cervical spine disc protrusion.
2. The clinical examination and the pain pattern should make it possible for the spinal level responsible for symptoms and signs to be established with a considerable degree of certainty.
3. MRI imaging can be used for confirmation of disc bulges and protrusions but may well miss tears in the annular fibres (Schellhas et al 1996).

References

Schellhas K P, Smith M D, Gundry C R, Pollei S R 1996 Cervical discogenic pain. Prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. Spine 21: 300–312.

Slipman C W, Plastaras C T, Palmitier R A et al 1998 Symptom provocation of fluoroscopically guided cervical nerve root stimulation. Are dynatomal maps identical to dermatomal maps? Spine 23: 2235–2242.

Further reading

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Case 58

Failed Cloward procedure

COMMENT

Patients with Lhermitte's sign require at least a thorough CT or MR investigation.

PROFILE

A 54-year-old man of average build who does not smoke cigarettes, only drinks alcohol occasionally, and is self-employed in light work.

PAST HISTORY

He had undergone a C5–6 level Cloward procedure 19 years ago for a posterior disc protrusion at this level that occurred when he fell onto his forehead 4 years prior to surgery. The disc protrusion had caused increasing neck and arm pains as well as headaches.

PRESENTING COMPLAINT(S) (Fig. 58.1)

Nineteen years following the Cloward procedure he presented with ongoing chronic lower neck pain, headaches and intermittent symptoms of tingling in his upper and lower extremities, aggravated by cervical spine flexion, of unknown aetiology. He felt there was a 'pressure' sensation in his neck causing pain to radiate to his head and to behind his eyes. He had seen a number of general medical practitioners and specialists but stated that they could not explain his symptoms.

AETIOLOGY

The original aetiology was falling onto his head 19 years ago.

EXAMINATION

He had some restriction of all active ranges of movement in the cervical spine, with all movements eliciting a minor exacerbation of his lower neck pain; cervical spine flexion aggravated the tingling sensation in his upper and lower extremities that was in a 'patchy' distribution. The deep reflexes in the upper and lower extremities appeared to be normal. The Valsalva manoeuvre aggravated his neck symptoms. Deep palpation of the paraspinal muscles elicited some tenderness with slight muscle guarding, particularly in the lower cervical spine. Pinprick sensation of the upper and lower extremities indicated a patchy distribution of sensation.

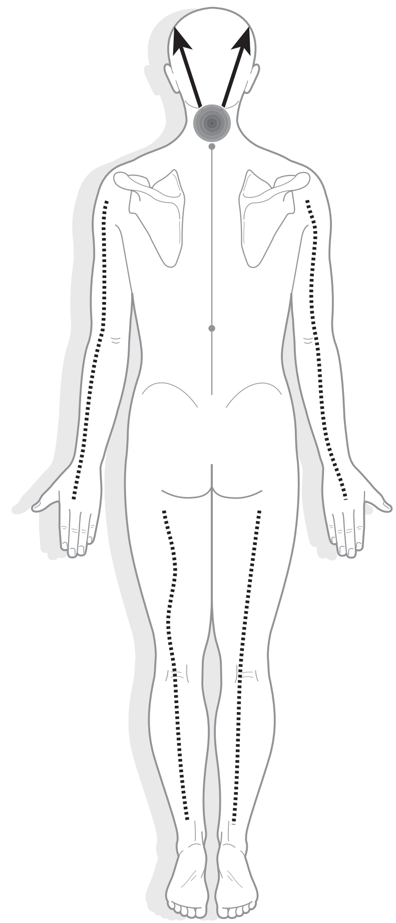

Figure 58.1

IMAGING REVIEW

A CT cervical spine performed approximately 3 years following the Cloward procedure showed the 'fusion bone graft plug' protruding into the spinal canal (Fig. 58.2A and B).

A subsequent MRI investigation 10 years after the Cloward procedure reported: 'Sagittal images show a small posterior protrusion at the C5–6 level with effacement of the CSF and early impingement centrally upon the cord'.

CLINICAL IMPRESSION

Cervical spine canal stenosis at C5–6 due to surgery with subsequent compressive cervical spine early myelopathy symptoms.

WHAT ACTION SHOULD BE TAKEN?

An updated CT scan was requested to re-evaluate his lower cervical spine (Figs. 58.3A and B). The radiologist

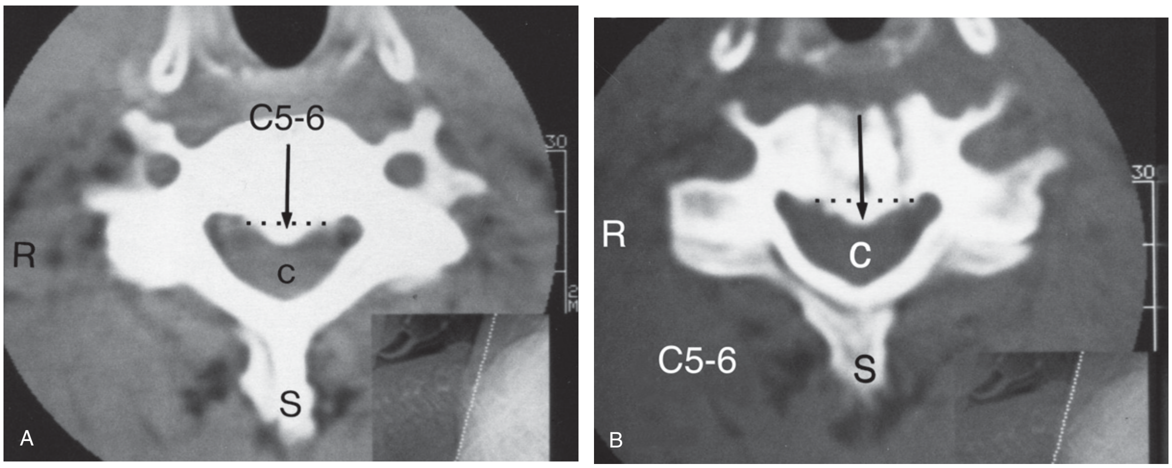

Figure 58.2 (A and B) Cervical spine CT axial image (performed 3 years following the Cloward procedure) through the C5–6 level using a *soft tissue exposure* (A) and *a bone window exposure* (B). Note the bony 'plug' (black arrow) extending beyond the normal posterior margin of the vertebral body (broken line) into the spinal canal and impinging upon the spinal cord (c) (A). The antero-posterior diameter of the spinal canal is 9.5 mm from the lamina junction posteriorly to the bony 'plug' anteriorly and, therefore, is stenotic as a result of the bony 'plug' that causes pressure upon the anterior surface of the spinal cord. S = spinous process.

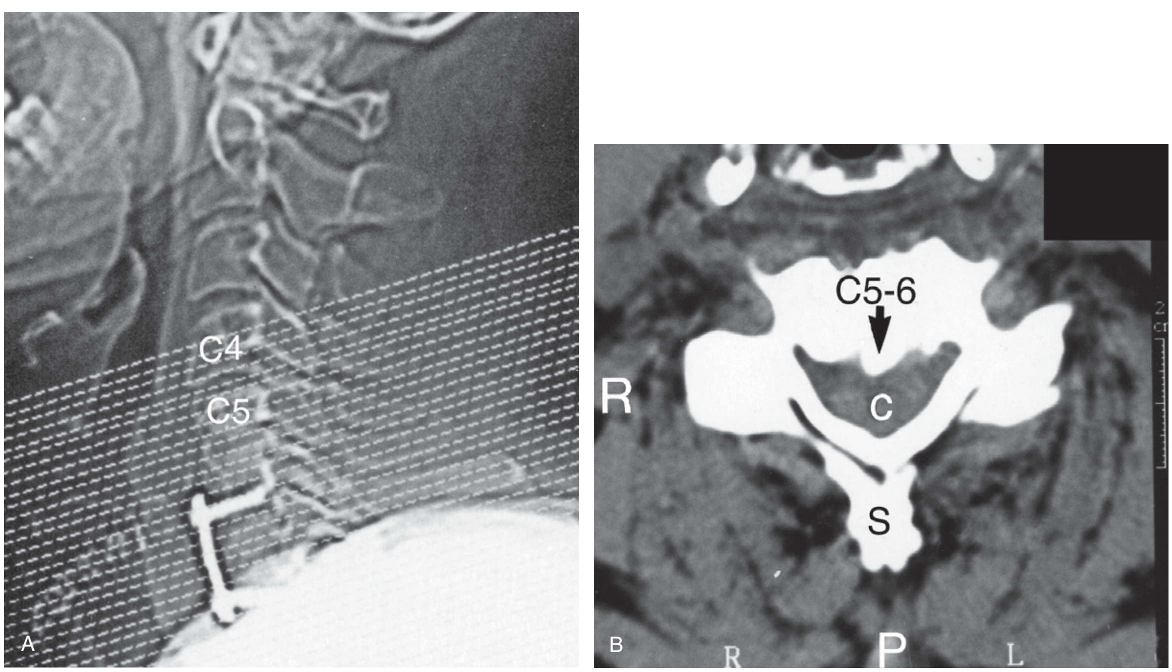

Figure 58.3 (A) CT cervical spine scout image. The anterior fixation plates extend from C6 to C7. (B) CT cervical spine axial image at the C5–6 level. Note the interbody fusion bony 'plug' (black arrow) still impinges upon the spinal cord anteriorly. C = spinal cord; P = posterior; S = spinous process.

stated: 'Anterior fixation plates extend from C6 to C7 (Fig. 58.3A). There is bony fusion between the bodies of C5 and C6. Bony irregularity of the posterior aspect of the body of C5 causes slight stenosis in the dimensions of the spinal canal' (Fig. 58.3B).

DIAGNOSIS

Cervical spine canal stenosis due to a Cloward procedure causing early myelopathy.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was grateful to have had his condition explained to him. He was advised not to flex his cervical spine to where it may reproduce sensations in his upper and lower limbs and he was advised not to work above his shoulders. He was advised to see a spinal surgeon for a further opinion and the spinal surgeon agreed that pressure on the anterior part of the spinal cord would, in all likelihood, be the cause of his symptoms of early myelopathy.

The spine surgeon decided to review the patient in 6 months time as the patient was very reluctant to undergo further surgery. Following that review, the patient decided to take precautions so as not to aggravate his symptoms rather than 'risking' further surgery at his C5–6 level.

Note

The pathophysiology of myelopathy is essentially 'a degeneration of various elements within the spinal cord produced in a complex fashion by the pressure and tension to which the cord is subjected by changes in the vertebral column with the associated ischaemia. Since this is a result of a number of variables, it is easy to understand why there should be great variability in the resulting clinical picture. Moreover, at any particular level the extent to which the gray and white matter are affected (and, within the white matter, the anterior, lateral and posterior columns), can be very variable. Three common initial symptoms that may occur, either alone or in combination, are (i) dysaesthesiae in the hands, (ii) weakness and clumsiness of the hands, and (iii) weakness of the lower limbs. The dysaesthesiae consist of numbness and tingling, especially in the tips of the digits and sometimes with a radicular distribution. It is necessary to remember that symptoms of radiculopathy and myelopathy may often co-exist and indeed be indistinguishable (Brain & Wilkinson 1967, Bohlman & Emery 1988, Bohlman 1995).

The decreased anteroposterior canal dimensions may lead to impingement of the anterior spinal artery system and the compromised blood supply may result in spinal cord ischaemia as the spinal canal diameter is diminished. The cord may also be mechanically deformed and flatten in the anteroposterior diameter, leading to stretching of the terminal branches of the anterior arterial system with exacerbation of the ischaemia (Dunn et al 1998). The spinal cord may also be compressed with dynamic motions of the cervical spine' (Dunn et al 1998).

The normal dimension of the anteroposterior diameter of the cervical spinal canal in male Caucasians at the C5–6 level has been found to be 17.8 mm as measured radiologically by Payne (1959). This reduces to 14.4 mm in patients with cervical myelopathy due to cervical spondylosis (vertebral osteophytosis/'lipping') (Payne 1959). Consequently, pathological stenosis occurs when there is an anteroposterior diameter of the cervical canal less than 15 mm in Caucasians (Hiroshima et al 1998); this Caucasian male patient's anteroposterior diameter was measured as only 9.5 mm.

Apart from the issue of myelopathy, the posteriorly protruding fusion will impinge upon (i) the extensive nerve supply (e.g. recurrent meningeal nerve) (Fig. v) and (ii) other pain sensitive soft tissue structures such as the anterior part of the dural tube and blood vessels. It is known that pressure on these soft tissue structures can cause pain of ischaemic origin (Jayson 1997).

KEY POINTS

1. A complete re-evaluation of patients is often necessary to get a clear impression of their current status.
2. It is important to re-image patients if symptoms re-occur or persist.
3. See also Key points for Case 63.

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Case 59

Persisting symptoms following Cloward procedure

COMMENT

Surgical errors occur and should be considered when a patient's symptoms do not improve after surgery.

PROFILE

A 41-year-old tall, well-built married male manual worker who does not smoke cigarettes and only drinks alcohol socially.

PAST HISTORY

Nothing relevant in his history. He had essentially been fit and active until a work injury 3.5 months ago. Three months prior to consultation he had undergone a Cloward procedure at the C5–6 intervertebral disc level for a right-sided osteophyte and a disc protrusion that followed a work injury.

PRESENTING COMPLAINT(S) (Fig. 59.1)

Neck pain that shoots up to the occipital region of his skull, especially on the right side, and to the shoulder blade and across his shoulders and upper thoracic region. The neck pain particularly radiates to his right arm as far as his hand and he feels a 'stabbing' pain in the right arm. The arm pain goes along the posterior aspect of the arm then crosses to the front of the forearm then radiates along the ulnar distribution to the last three fingers of his right hand. He complained of almost complete 'numbness' and some pins and needles in the fourth and fifth fingers of his right hand.

He was referred by his general medical practitioner because neither the doctor nor the patient could understand why the patient's symptoms felt 'exactly the same before and after surgery'. No one had been able to answer this question.

AETIOLOGY

Work-related injury approximately 3.5 months prior to consultation.

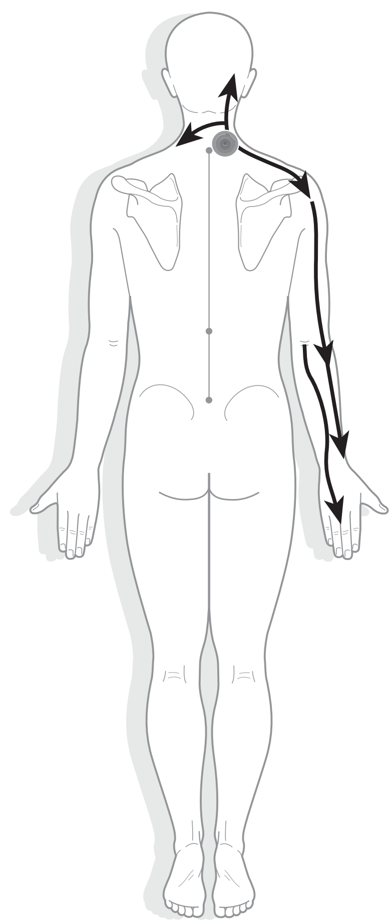

Figure 59.1

EXAMINATION

The right biceps (C5–6) reflex was diminished at one plus (two plus being normal) as was the case with the right triceps (C7) reflex. Pinprick sensation of the upper and lower extremities was normal, apart from hypoaesthesia of the right middle finger (C7) and no sensation for the right fourth and fifth fingers (C8). There was decreased pinprick sensation on the posterior aspect of the right shoulder in the C5 dermatome. There was also an area of no sensation on the lateral aspect of the right leg, approximately 15 cm above the ankle (L5). The circumference of the arms, 10 cm above the elbow joint, was 34 cm (left) and 32 cm (right) showing a 2 cm difference, presumably due to muscle wasting. Vibration sensation was reported as being significantly less when a tuning fork was applied to the right elbow as compared to the left (perhaps indicating some injury to the posterior columns of the spinal cord, although vibration sensation at the ankles appeared to be normal).

Cervical spine active ranges of movement in the seated position were as follows:

1. Flexion – limited by approximately 40% due to pain radiating to the right mid-forearm.
2. Extension – limited by approximately 80% due to slight pain at approximately the C5 level.
3. Left rotation – limited by approximately 80% due to pain on the left side of the cervical spine.
4. Right rotation – limited by approximately 80% due to cervico-shoulder pain on the right side radiating to the right scapula as far as its inferior aspect.

Deep palpation of the cervical paravertebral muscles elicited considerable tenderness, particularly on the right side at approximately the C5 level.

IMAGING REVIEW

The C5–6 disc protrusion was clearly seen on the sagittal and axial (insert) images of an MRI scan performed prior to surgery (Fig. 59.2A).

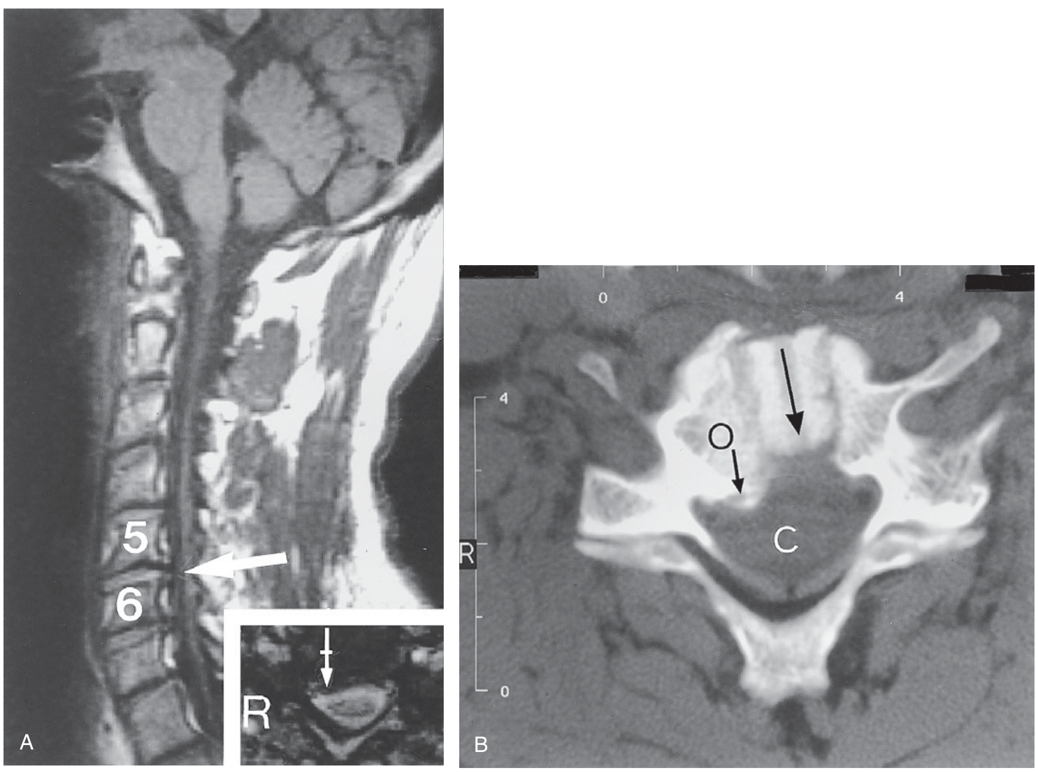

Figure 59.2 (A) Cervical spine MRI parasagittal T1-weighted image showing osteophyte/disc protrusion at C5–6 (white arrow). This right-sided posterolateral disc protrusion is shown in the axial insert (tailed arrow). R = right side of patient. The MRI report read: There is some osteophyte/disc distortion of the cord at C5–6 level. Disc protrusion extends into the intervertebral foramen. There also appears to be a right-sided posterolateral osteophyte at C6–7 but it does not impinge upon the right intervertebral foramen. (B and C) Cervical spine CT axial image of the C5–6 level following the Cloward procedure (performed 3 months previously) to remove the right sided posterolateral disc protrusion and osteophyte seen by MRI as being present at C5–6. Note that the *left* side was decompressed instead of the right side which still shows the disc osteophyte (O) complex on the patient's right side (R); this compresses the anterolateral portion of the dural tube. The surgical approach (large black arrow) was directed to the patient's left side. C = spinal cord within the spinal canal.

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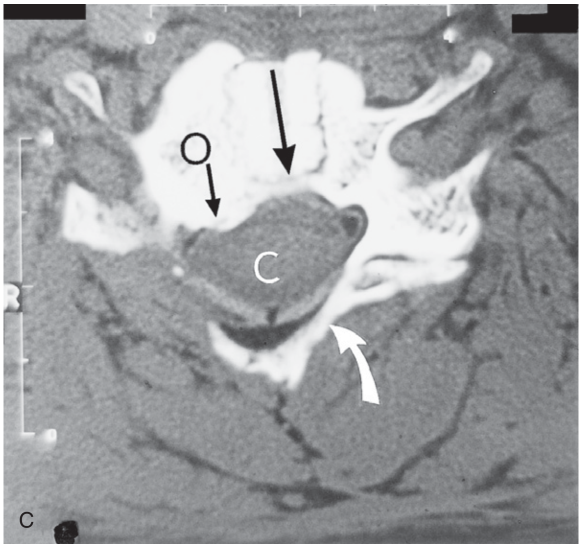

Figure 59.2 Cont'd (C) Cervical spine CT axial image showing the laminectomy that was performed on the right side in an attempt to remove pressure from the disc/osteophyte complex projecting against the pain-sensitive dural tube anteriorly. O = osteophyte; the large arrow indicates the original surgical approach which is directed to the patient's left side. The left lamina is shown by the curved white arrow and the corresponding lamina on the right side has been surgically removed. C = spinal cord within the spinal canal.

CLINICAL IMPRESSION

C5–6 or C6–7 right sided posterolateral disc protrusion.

WHAT ACTION SHOULD BE TAKEN?

In view of the patient's obvious pain, which caused him to keep moving his arm and hand about in order to minimize the pain during the consultation, even though he was taking analgesics, a CT scan was ordered which showed: 'C5–6 anterior interbody fusion. Right posterolateral osteophyte formation at this level with some bony narrowing of the right C6 nerve root exit foramen' (Fig. 59.2B).

DIAGNOSIS

C5–6 right sided neck pain and radiculopathy due to a right sided posterolateral disc protrusion and osteophyte.

TREATMENT AND RESULTS

The patient and his referring medical practitioner were advised that surgery apparently had been directed to the patient's *left* side as demonstrated by the CT scan (Fig. 59.2B).

The patient was referred to another surgeon who performed a right-sided laminectomy (Fig. 59.2C). This was done in an attempt to decompress the neural structures on the right side, as it was now impossible to get to the right posterolateral osteophyte to remove it and any associated disc material because the previous Cloward procedure (Fig. 59.2B) apparently prevented access for a further anterior approach.

The further surgery was unsuccessful and resulted in perineural fibrosis. Unfortunately, the patient developed increasing right upper limb symptoms that led to severe depression and large doses of analgesics. He then began to develop leg symptoms due to cervical spondylosis and early myelopathy at the C5–6 level.

Note

In order to consider similar histopathology at this level in a postmortem specimen, please see Case 49, Figure 49.12.

KEY POINTS

1. Errors do occur with surgery, as they do in any branch of the healing profession. Therefore, it is important to listen carefully to the patient's description of post-surgical symptoms then to perform a thorough clinical and appropriate imaging investigation in order to re-evaluate the patient.
2. Cervical myelopathy is clearly related to spinal cord ischaemia and compression, and stretching and compression of spinal cord tissue will result in a great spectrum of variability in the clinical symptoms (Bland 1987).

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Further reading

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Case 60

Cloward revision surgery

COMMENT

Post-surgical follow-up investigations are essential when symptoms persist or re-occur.

PROFILE

A 54-year-old female of average build who does not smoke cigarettes or drink alcohol and who works in light professional duties.

PAST HISTORY

She had a cervical spine fusion (Cloward procedure) performed approximately 9 years ago following a motor vehicle accident in which she experienced a C5–6 posterior disc protrusion that caused headaches, neck pain and left sided C5–6 radiculopathy.

PRESENTING COMPLAINT(S) (Fig. 60.1)

Chronic bilateral neck pain that causes neck tension with radiation to (i) the occipital area causing occipital headaches, and (ii) the left cervico-shoulder region and left upper limb, including the C6 dermatome of the lateral forearm, thumb and index finger. She said she had not greatly improved after the Cloward procedure. The pain is of variable intensity and an increase in activity causes an increase in her symptoms. She said the pain is similar to the pain that she experienced before the Cloward procedure was performed 9 years ago. Bearing down causes an increase in her neck pain and left upper limb symptoms. Non-steroidal anti-inflammatory medication and analgesics 'take the edge off the pain' but do not provide much relief. In addition, the medication is irritating her stomach, causing pain and indigestion.

AETIOLOGY

Motor vehicle accident 9 years ago.

EXAMINATION

Deep palpation of the paraspinal muscles in the neck elicited pain from the C3 to C7 level bilaterally. The cervico-shoulder region of the left trapezius muscle was tender, particularly in its trigger point. Deep palpation of the paraspinal muscles in the thoracic spine did not elicit any localized significant pain.

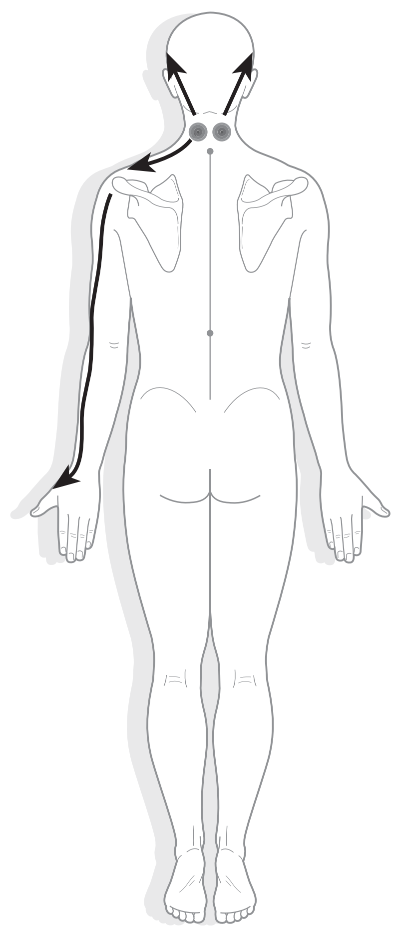

Figure 60.1

The deep reflexes in the upper and lower extremities were normal, apart from the left brachioradialis reflex (C6) that was somewhat diminished in comparison to that on the right side. There appeared to be some weakness (4/5) in muscle power in the left biceps (C5, C6) and the left wrist extensors (C6), as well as in the forearm pronatus and supinatus muscles. Pinprick sensation elicited patchy hypoaesthesia in the left upper limb. All cervical spine active ranges of movement elicited some degree of neck pain with varying degrees of limitation, and movements were performed cautiously as she knows that sudden movements exacerbate her symptoms.

IMAGING REVIEW

Plain X-ray films showed a loss of normal lordosis with a degree of kyphosis at the C5–6 level and that a Cloward procedure had been performed at the C5–6 level.

CLINICAL IMPRESSION

Recurrent C5–6 disc.

WHAT ACTION SHOULD BE TAKEN?

A cervical spine CT scan was ordered from C4 to C7. This showed that there was a significant posterior length of bony graft material compromising the left side of the spinal canal, dural tube and spinal cord with encroachment upon the left C5–6 intervertebral foramen and its C6 nerve root (Fig. 60.2).

DIAGNOSIS

Left sided C6 radiculopathy, neck pain and cervicogenic occipital headaches due to compromise of the spinal and intervertebral canals at the C5–6 level with reflex muscle spasm.

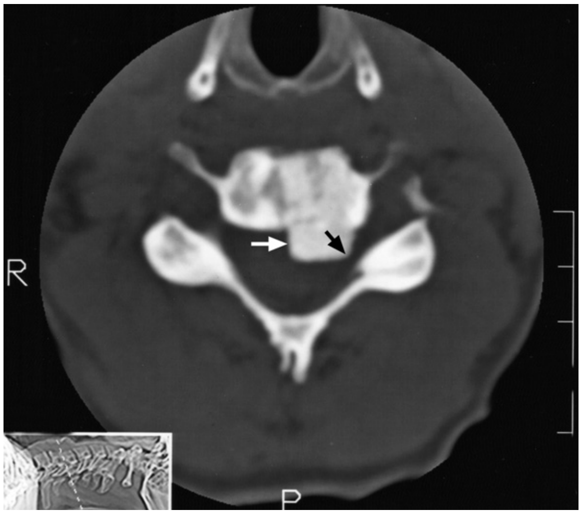

Figure 60.2 Cervical spine CT axial image through the C5–6 level. Note the significant posterior length of bony graft material (white arrow) compromising the left side of the dural tube/thecal sac and spinal cord with encroachment upon the left C5–6 intervertebral foramen (black arrow).

TREATMENT AND RESULTS

The patient was referred back to the neurosurgeon that performed the original Cloward procedure with a request for review of the patient and possible revision of the C5–6 Cloward fusion.

At surgery, the C5–6 graft site was exposed and reported as showing 'hard bone probably osteophytic'. The bone was removed from the dural tube resulting in a dural tear that was closed. A modified graft of reduced length was tapped into place before the wound was closed.

The Cloward revision provided considerable relief from her left upper limb symptoms, neck pain and headaches. However, she experienced intermittent neck pain and headaches, so a cervical spine MRI examination was performed 3.5 years later; this showed a mild reversal of the curvature in the mid-cervical region (Fig. 60.3) with posterior spurring at the C5–6 interspace causing indentation of the anterior pain sensitive part of the dural tube but without any cord or nerve root involvement.

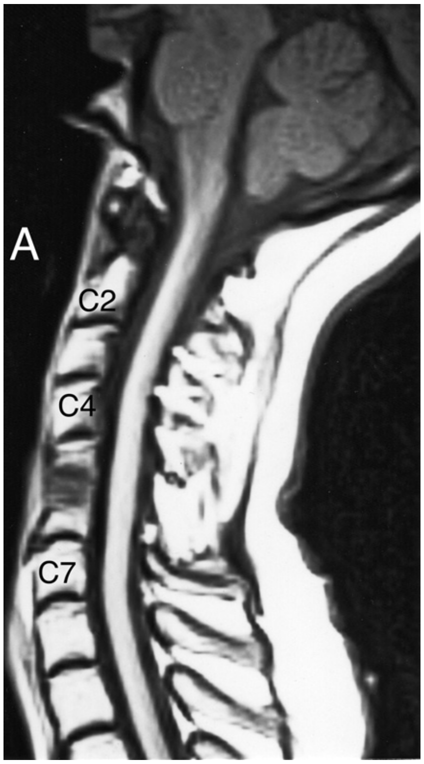

Figure 60.3 Cervical spine MRI sagittal image following the Cloward revision. Note the mild reversal of the curvature of the mid-cervical region.

KEY POINT

The importance of post-surgery follow-up imaging cannot be underestimated when symptoms persist or re-occur.

Case 61

Uncovertebral joint osteoarthrosis

COMMENT

It is well known that there are many causes of headaches – all should be considered in the differential diagnosis.

PROFILE

A 48-year-old married housewife of thin build who does not smoke or drink alcohol.

PAST HISTORY

She had a left C2 neurectomy performed approximately 18 years ago for left sided headaches, without any relief but with resulting numbness involving the entire left occipital region ever since. The neurectomy specimen consisted of a fragment of 'pale tissue measuring 50 cm in length at macroscopic investigation and microscopic sections confirmed the presence of nerve trunks with no evidence of malignancy'. Narcotic injections and migraine prophylaxis had been to no avail for the left sided headaches. She had an appendectomy and a cholecystectomy performed some years ago.

PRESENTING COMPLAINT(S) (Fig. 61.1)

Chronic left sided neck pain and headaches that had flared up during the last few months, having begun 18 months ago and of unknown aetiology. Although she has a history of headaches since her early teens, with multiple investigations and treatments over the years, the present headaches and left sided neck-pain feel 'different'. These headaches appear to be severe vascular/tension type headaches due to severe left sided cervicogenic pain.

She experiences left sided neck symptoms of feeling 'hot'; these are then followed by pain radiating from approximately the left side of C5 vertebra to the upper neck then to behind the left eye, causing severe headaches. She believes that these neck symptoms are precipitated by various neck movements that cause the 'hot' sensation on the left side at approximately the C5 spinal level.

She had been taking analgesics, anti-inflammatory medication and an antidepressant for her chronic neck pain syndrome but had recently ceased all medication as she found that none helped, apart from periodic narcotic injections into the suboccipital region. She had also tried steroid

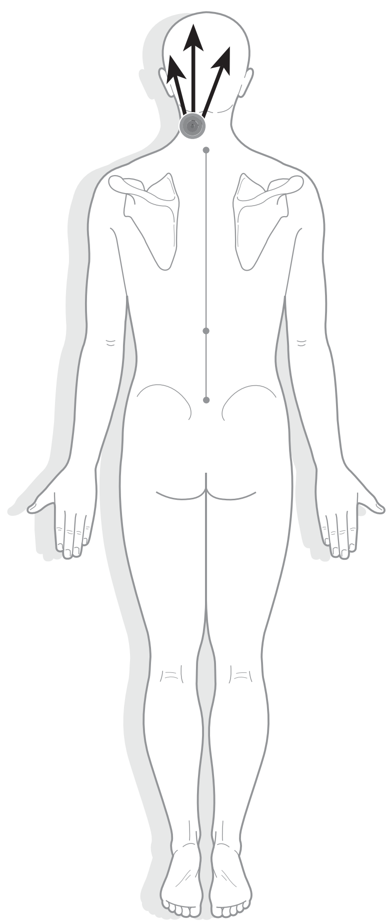

Figure 61.1

and local anaesthetic injections in the paravertebral muscles which provided temporary relief. Spinal manipulation did not help.

AETIOLOGY

Unknown. Apparently suspected of being 'psychosomatic' by previous clinicians.

EXAMINATION

The deep tendon reflexes in the upper and lower extremities were normal as was the case with pinprick sensation and vibration sensation. Power in the upper and lower extremities was normal apart from slight (4/5) diminished power on flexion (C5–6) and extension (C7) at the elbows. All cervical spine ranges of movement were limited by neck pain, particularly extension. She was normotensive.

IMAGING REVIEW

A CT scan of her head and an angiogram had been performed but failed to reveal an aneurysm or any other intracranial pathology. Her plain film radiographs were reported as follows: 'A slight loss of disc space at the C6–7 level. No other bone or joint lesion seen' (Fig. 61.2). However, a review of the plain X-ray films showed a large osteophyte projecting from the left uncovertebral joint inferiorly on C5 vertebra toward the left vertebral artery (Fig. 61.2A). There were also osteophytes involving the C6–7 uncovertebral joints on both sides (Fig. 61.2C and D).

CLINICAL IMPRESSION

1. Left vertebral artery sympathetic plexus compromise due to encroachment by the C5 uncovertebral joint osteophyte upon the sympathetic plexus (suggested by the autonomic symptom of feeling 'hot' on the left side of the neck).
2. A differential diagnosis of a possible neuroma at the site of the left sided C2 neurectomy.

WHAT ACTION SHOULD BE TAKEN?

In view of the large left sided uncovertebral joint osteophyte at C5, a CT cervical spine was performed with contrast which showed 'osteophytic encroachment on the medial

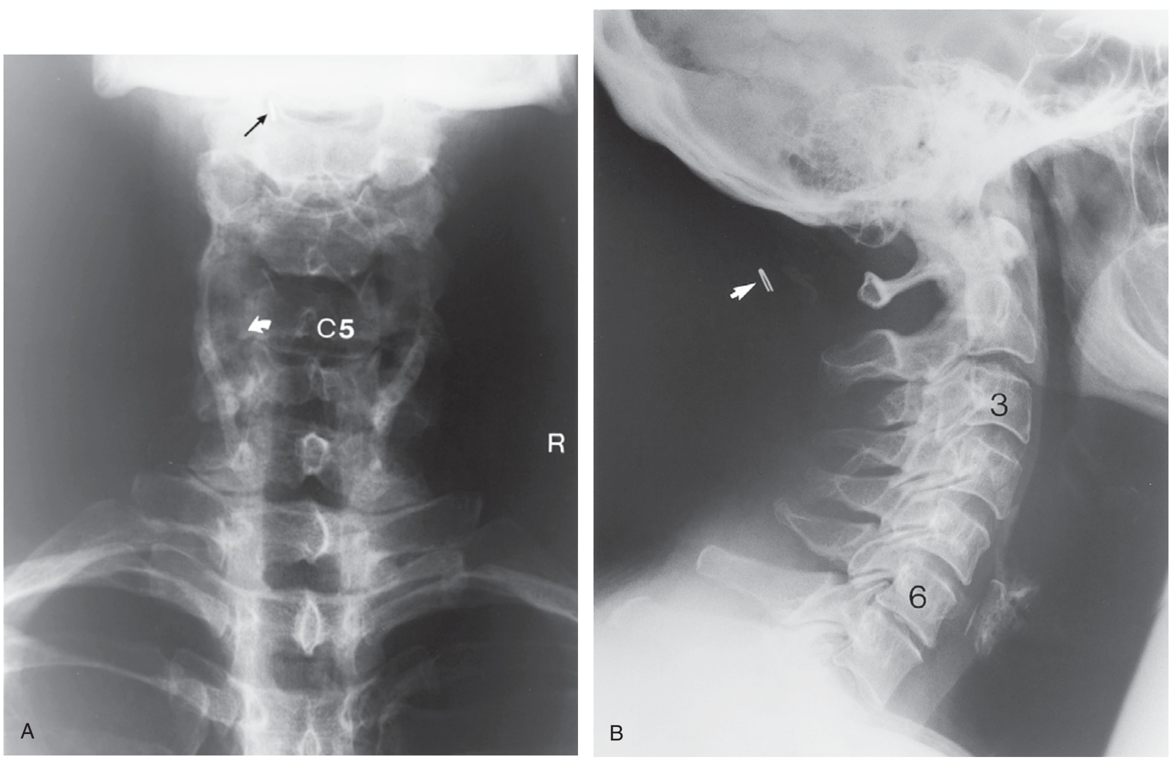

Figure 61.2 (A) Cervical spine anteroposterior plain X-ray image showing the clip (black arrow) in the left suboccipital region where the C2 neurectomy was performed and the C2 nerve was clipped and divided. C5 = fifth vertebra. White curved arrow shows the osteophyte projecting from the left uncovertebral joint toward the vertebral artery in the transverse foramen. R = right side of patient. (B) Cervical spine neutral lateral plain X-ray image showing thinning at the C5–6 disc level and particularly at the C6–7 level. Note the neurectomy clip (white arrow). 3 = third vertebral body; 6 = sixth vertebral body.

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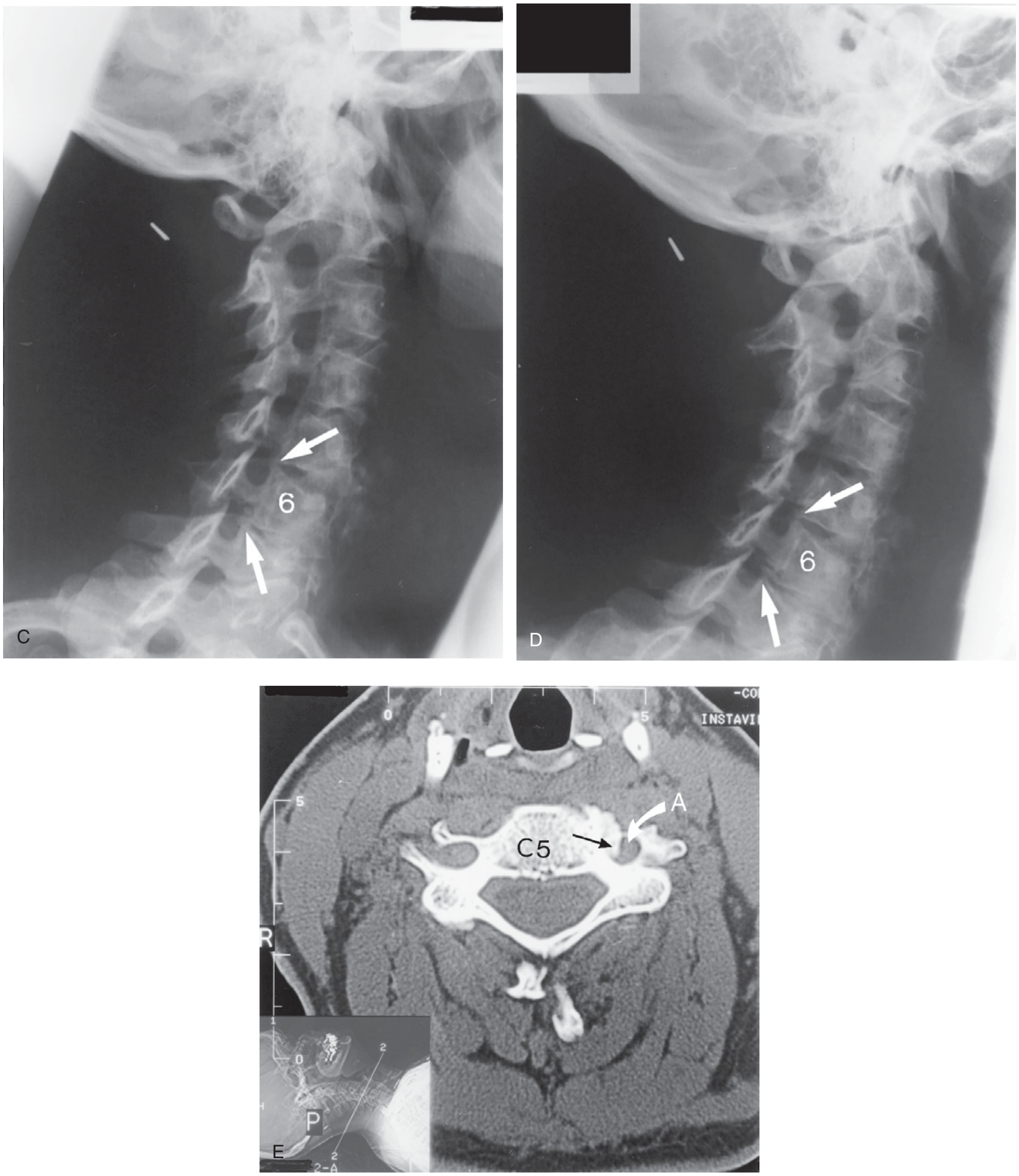

Figure 61.2 Cont'd (C,D) Cervical spine right and left oblique plain X-ray views. The white arrows indicate osteophytic hypertrophy of the uncovertebral joints at C5–6 and C6–7 although the left uncovertebral joint osteophytosis at C5–6 is best visualized on the anteroposterior radiograph (A). (E) Cervical spine CT axial image of the C5 vertebral body showing how the uncovertebral joint osteophytic encroachment (black arrow) at the medial aspect of the left C5 transverse foramen causes reduction in the calibre of the adjacent vertebral artery (A) within the transverse foramen. What cannot be seen is, of course, the effect that this osteophytic hypertrophy has on the sympathetic plexus on the vertebral artery.

aspect of the left transverse foramen at the C5 level with associated reduction in the calibre of the vertebral artery at this level' (Fig. 61.2E). The contrast showed that the vertebral arteries entered the transverse foramen at the C6 level bilaterally.

DIAGNOSIS

Left sided C5 uncovertebral joint osteophytic hypertrophy encroaching upon the vertebral artery and its associated sympathetic plexus causing left sided neck pain and headaches.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

She was advised to change her very active lifestyle to minimize her activities, obtain psychological support and learn relaxation techniques; she was referred to yet another neurologist, a pain management anaesthetist and another neurosurgeon. These consultations resulted in the prescription of Epilim (400 mg twice per day with gradual increase for the neuralgic pain), Valium (2.5–5 mg three times per day as required for tension headaches), and Zomig (2.5 mg twice per day plus or minus Stemetil for migraine headaches). The most helpful of these medications was Epilim and her neurologist suggested that she should gradually increase this to a level where her pain control was manageable – she was not to exceed a maximum dose of 1 g twice per day in her particular case.

The Epilim medication initially gave some relief, so an uncectomy was not considered at the time. However, she developed an adverse reaction to the Epilim (nausea, diarrhoea and anaemia) and, because of the difficulty associated with performing an uncectomy, Neurontin (gabapentin) was trialled with surgery being reserved as a last resort measure should Neurontin fail. She manages to cope on four 400 mg tablets per day but also uses Mersyndol and Zomig as required. Full haematology, serum biochemistry and serum gabapentin levels are monitored in case any abnormalities should occur over time.

Note

It is highly likely that irritation of the sympathetic nerve plexus on the vertebral artery is causing a vascular response that, in turn, causes her incapacitating vascular-type headaches. It is possible that this lady has two types of headaches: a headache with a neurovascular component due to irritation of the left vertebral artery and its sympathetic plexus, and a headache due to a neuroma that may have developed at the site of the C2 neurectomy. Because of the symptoms of feeling 'hot' on the left side of approximately the C5 level, precipitated by various neck movements, irritation of the sympathetic plexus appears to be the most likely cause.

Look at Case 49, Fig. 49.12, to review the anatomy and location of the vertebral artery and vein in the transverse foramen in a histological cross-sectional view of the cervical spine. The intricate sympathetic nerve plexus is not shown at this magnification.

KEY POINT

The issue of irritation of the vertebral artery's sympathetic plexus due to vertebral artery tortuosity has been discussed in Case 53. This case highlights how the sympathetic plexus on the vertebral artery may be irritated by uncovertebral joint osteoarthrosis.

Further reading

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Nagashima C 1970 Surgical treatment of vertebral artery insufficiency caused by cervical spondylosis. Journal of Neurosurgery 12: 512–521.

Case 62

Hangman's fracture of C2 vertebra

COMMENT

Poor quality imaging is a recipe for disaster!

PROFILE

A 50-year-old male manual worker of solid build who does not smoke cigarettes or drink alcohol.

PAST HISTORY

He suffers from mild hypertension that is well controlled by medication. He was known to have had a fracture at the C2 level (Hangman's fracture) 3 years ago due to falling onto his head; he had been hospitalized for approximately 2 weeks while he was further investigated and a halo-thoracic brace was used to stabilize his cervical spine. Eventually, the halo-thoracic brace was replaced with a cervical spine collar until the orthopaedic consultant felt it was safe to remove it.

PRESENTING COMPLAINT(S) (Fig. 62.1)

1. Constant severe bilateral suboccipital pain of 3 years duration that varies in intensity and is activity related. The pain radiates to behind both ears and to the vertex area causing 'bad headaches' that can affect the whole of his head. Some pain radiates to the right cervico-shoulder region then into the right upper limb as far as the thumb; periodically he experiences similar but less severe symptoms in the left arm. He finds it very difficult to get to sleep as he has to sleep on his back because lying on his left and right sides is too painful for his neck. As a result, he gets very little sleep and has to get up at night and walk around. He said his neck movements are very limited. Sometimes he experiences 'dizziness' when coughing or sneezing but he can move his neck within its very limited range of movement and not aggravate the 'dizziness'. Bearing down does not aggravate this pain. The pain is aggravated by coughing or sneezing, work, and being in bed as his neck is very stiff and painful of a morning.

The pain is temporarily relieved by Codalgin (5–6 at a time, taken twice daily), tramadol (200 mg taken three times daily), Valium (15 mg) in an attempt to sleep, and an antidepressant tablet morning and night.
He is concerned about the side effects of medication but cannot cope without it.

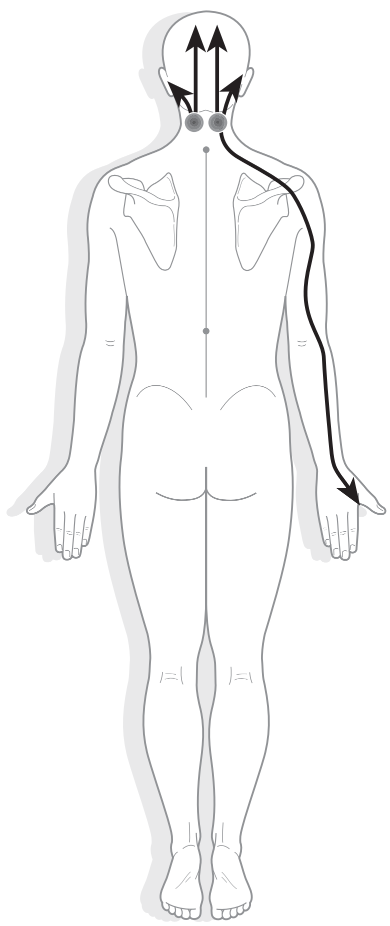

Figure 62.1

2. Approximately T7 level central pain that is constant but is not as severe as his neck pain. The intensity varies depending on his activity at the time, with increased activity causing more severe pain. There is no radiation of pain. Bearing down does not aggravate this pain. The pain is aggravated by work, and being in bed overnight, in spite of getting up and walking about due to his neck pain. The pain is temporarily relieved by the analgesics and anti-inflammatory medication that he takes.

He believes that his mid-thoracic spine pain has not been taken seriously and that it had not been satisfactorily investigated.

AETIOLOGY

Approximately 3 years ago he fell from a height of approximately 2.5 metres, hitting the ground with the left frontal area of his head at which time he was knocked unconscious. On regaining consciousness, he realized that his neck was very painful. He found it difficult to sit up or stand, so he remained lying down until an ambulance arrived and he was provided with a hard collar before being taken to hospital.

EXAMINATION

Deep palpation of the paraspinal muscles in the neck elicited (i) bilateral sub-occipital to C3 pain with generalized neck pain and muscle spasm, and (ii) bilateral pain at approximately the T7 level. Toe walking (S1) and heel walking (L5) were difficult for him to perform as he felt 'off balance'. It appeared that the right triceps jerk (C7) was diminished, as were the left and right brachioradialis reflexes (C6). The deep reflexes in the lower extremities appeared to be normal as was the plantar response. Vibration sensation at the elbows and ankles was normal. Pinprick testing suggested hypoaesthesia in the right thumb (C6) and hypoaesthesia on the lateral aspect of the left calf (L5). There was some weakness (4/5) of right wrist extension (C6) and finger adduction (4/5) (C8, T1).

The circumference of the forearm (10 cm below the elbow) was 28.5 cm (left) and 29 cm (right). The blood pressure in the right arm was somewhat elevated at 180/88 in the seated position.

Active cervical spine ranges of movement were performed cautiously and were measured using a CROM instrument (see Box 62.1).

Other cervical spine and cervico-shoulder region procedures to test for cervical spine pain were: cervical spine compression (painless), downward shoulder pressure (elicited right sided cervico-shoulder pain), trapezius muscle trigger point pressure (elicited greater pain in the right trigger point).

Active thoracic spine ranges of movement were somewhat limited because of his neck pain but did not significantly increase the mid-thoracic spine pain.

Box 62.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	30°	Elicited pain in the mid-cervical spine bilaterally
Extension	60°	30°	Elicited pain in the suboccipital area bilaterally
Lt lateral bending	45°	18°	Elicited pain in the suboccipital area bilaterally
Rt lateral bending	45°	18°	Elicited pain in the suboccipital area bilaterally
Lt rotation	80°	40°	Elicited pain in the suboccipital area bilaterally
Rt rotation	80°	38°	Elicited pain in the suboccipital area bilaterally

IMAGING REVIEW

On the day of his accident, plain X-ray films were taken of his cervical spine and his thoracic spine. The X-ray reports stated. For the cervical spine: '*Note the posterior slip of* C2 on C3 by approximately 3 mm (Fig. 62.2), possibly related to recent bony trauma. Osteophytic lipping is present at the C3–4 and C4–5 levels with disc narrowing and osteophytic lipping at the C5–6 level'. For the thoracic spine the report was: 'There is minor degenerative change (Fig. 62.3). No wedge fractures demonstrated'.

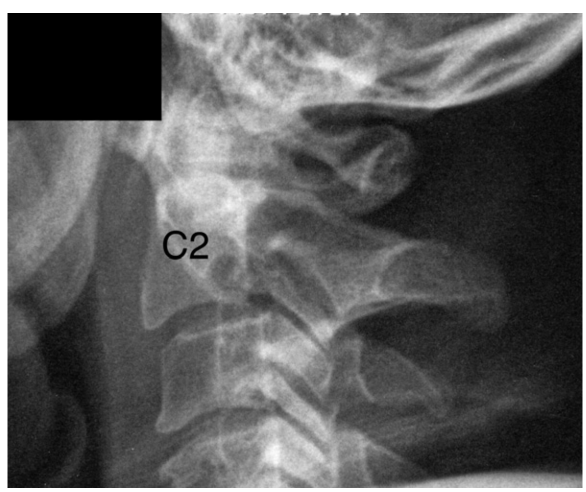

Figure 62.2 Upper cervical spine lateral plain X-ray image reported as showing anterior slip of C2 on C3 by approximately 3 mm, possibly related to recent bony trauma. Osteophytic lipping is seen at the C3–4 level.

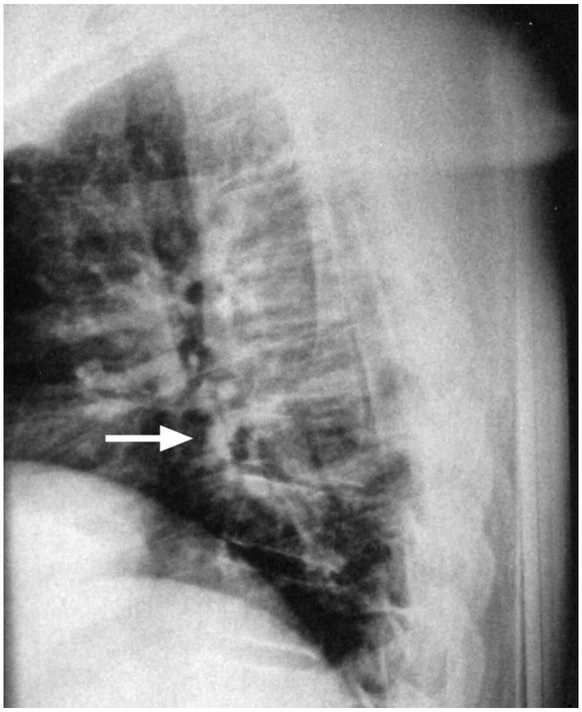

Figure 62.3 Thoracic spine lateral plain X-ray image. Reported as '. . .no wedge fractures demonstrated'. However, note the compression fracture at approximately the T7 vertebral body level (arrow).

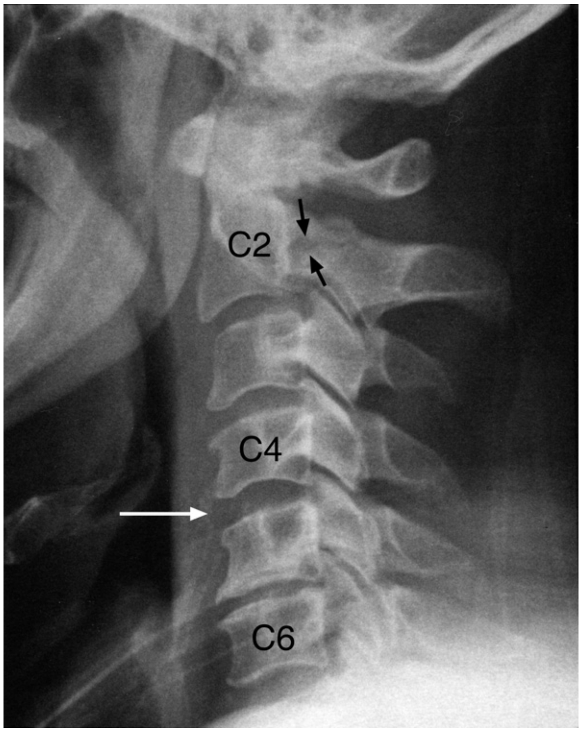

Figure 62.4 Cervical spine lateral plain X-ray image. Note (i) the anterior slip of C2 on C3 in view of the hangman's fracture at C2 (arrows) (due to associated ligamentous and disc injuries at this level), (ii) the possible compression of the antero-superior aspect of the C4 body, (iii) the widening of the C4–5 intervertebral disc space height anteriorly (white arrow) suggesting that injuries occurred to this intervertebral disc and other soft tissue structures at this level, probably due to an extension type injury to his cervical spine, and (iv) the disc narrowing and osteophytic lipping at the C5–6 level.

In view of the poor positioning of the cervical spine lateral view (Fig. 62.2) the lateral view was later repeated and this showed a 'hangman's' fracture (Fig. 62.4).

In view of the above plain film cervical spine imaging, a CT brain and a CT cervical spine had been performed. The CT brain found no abnormality. The CT cervical spine showed an unstable fracture of the body of C2 with a fracture at the junction of the body and the left pedicle with retropulsion of a bony fragment into the vertebral canal (Figs 62.5 and 62.6).

CLINICAL IMPRESSION

1. Hangman's fracture at C2 with associated cervical spine soft tissue injuries.
2. C5–6 posterior intervertebral disc bulge/protrusion causing irritation of the C6 roots.
3. Injury at approximately the T7 level that requires further investigation.

WHAT ACTION SHOULD BE TAKEN?

Cervical to lower thoracic spine MRI to further investigate his mid-thoracic spine pain (Figs 62.7 and 62.8).

The MRI report referred to (i) various cervical spine disc bulges and protrusions, e.g. C3–4, C4–5, C5–6 and C6–7 (Fig. 62.7), and to 'flattening of the dural tube' at the C2–3 level (Fig. 62.8), and (ii) a thoracic spine anterior wedge fracture of the T7 vertebral body (Fig. 62.7).

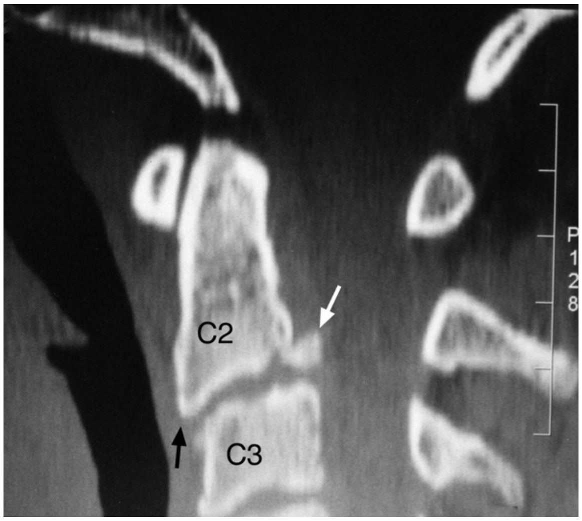

Figure 62.5 Cervical spine CT sagittal image. Note (i) the C2 postero-inferior bony fragment displaced into the spinal canal (white arrow), and (ii) the anterolisthesis (black arrow) of the C2 body on the C3 body below.

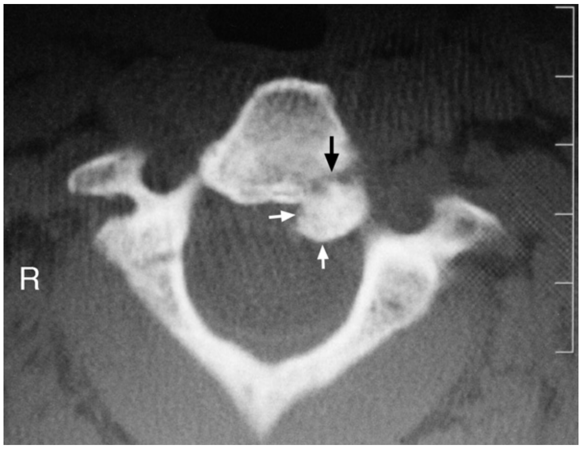

Figure 62.6 Cervical spine CT axial image at the C2 level. Note the fracture line extending into the C2 inferolateral body (black arrow) where significant bone displacement posteriorly into the spinal canal persists (2 short white arrows). The bone fragment appears to press upon the pain sensitive anterior aspect of the dural tube.

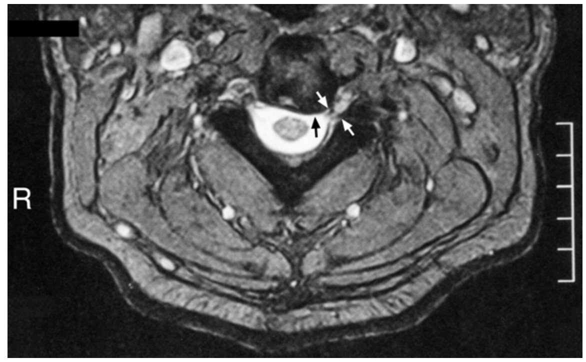

Figure 62.8 Cervical spine MRI T2-weighted axial image through the C2–3 intervertebral foraminae level. Note the left paracentral 'flattening of the thecal sac' (small black arrow). The antero-posterior dimension of the left intervertebral foramen is narrower (white arrows) when compared with the right side (R) of patient.

Figure 62.7 Cervical and thoracic spine MRI sagittal T2-weighted image. Note (i) the posterior disc bulges at the C3–4 and C4–5 levels, (ii) the posterior disc protrusion at the C6–7 level, and (iii) the anterior wedge fracture of the T7 vertebral body (white arrow).

DIAGNOSIS

Cervical spine

- Cervical spine fractures at several levels with a particularly life threatening fracture (Hangman's fracture) of the C2 vertebra with some bony fragments still remaining within the spinal canal.
- Musculoligamentous soft tissue injuries of the cervical spine including posterior disc bulges at the C3–4 and C4–5 levels and a posterior disc protrusion at the C6–7 level.
- Right C6 radiculopathy.

Thoracic spine

- Crush fracture of the T7 vertebral body with approximately 25% loss of anterior vertebral body height.
- Musculoligamentous soft tissue injuries of the thoracic spine including some thinning of the T6–7 and T7–8 intervertebral discs.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was told that there was an anterior wedge fracture of the T7 vertebral body and that this, in all likelihood, was the cause of his central spinal pain at approximately the T7 level.

As he was still undergoing orthopaedic care he was advised to continue with this and to take the new MRI films showing his T7 vertebral body fracture to his next orthopaedic appointment.

KEY POINTS

1. Correct patient positioning for radiology is essential if an appropriate diagnosis is to be made. Incorrect positioning of the patient will lead to serious injuries being missed.
2. Thoroughly investigate complaints of spinal pain to ensure that pathology will not be missed.

Further reading

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Case 63

Cervical cord myelopathy

COMMENT

Beware of neck, arm and leg symptoms as they may be indicative of cervical cord myelopathy.

PROFILE

A 57-year-old male manual worker who smokes cigarettes.

PAST HISTORY

His past history was that he was involved in a motor vehicle accident approximately 7 years ago when the stationary vehicle in which he was sitting was rear-ended. Even though he wore a seat belt, he felt that his neck was 'whipped backwards and forwards' and his head hit the headrest with enough impact to require stitching to the left and right occipital regions. He had been awarded an out-of-court settlement but still had to wear a soft cervical spine collar, in the reversed position, so as not to hyperextend his neck as that caused pain. He said he had always been healthy as a child but required a tonsillectomy at approximately 38 years of age due to chronic tonsillitis that developed in adulthood. He suffered from hypertension that was well controlled by medication.

PRESENTING COMPLAINT(S) (Fig. 63.1)

His main complaints were of neck pain, especially at the C4–7 level, particularly on the left side and which radiated to the left elbow, as well as up to the occipital region causing headaches. He also had some 'foot drop' on the left side.

A minor secondary complaint was of slight low back pain extending to the left buttock and to the lateral aspect of the left thigh and calf, occasionally as far as his foot.

He said his neck and left leg problems were 'completely depressing' in spite of taking an antidepressant, as he could 'not see the light at the end of the tunnel'.

He had seen orthopaedic surgeons, neurosurgeons and a psychiatrist without help.

AETIOLOGY

Motor vehicle accident 7 years ago.

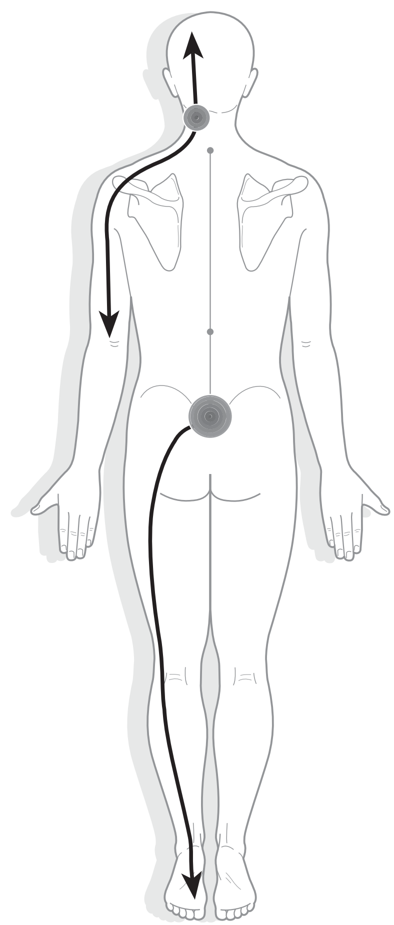

Figure 63.1

EXAMINATION

The deep tendon reflexes in the arms and legs were normal. Pinprick sensation of the upper and lower extremities was normal apart from the left calf laterally (L5) where there was some hyperaesthesia. Vibration sensation at the elbows and ankles was normal. The circumference of the left thigh, 15 cm above the patella, was 1 cm less than on the right side (i.e. just within normal limits); the left calf, 12 cm below the patella, was 2 cm less than on the right side. Both seated and supine SLR indicated limitation to approximately 40° due to slight low back pain and hamstring 'pulling' on elevation of the left leg. Bilateral hip flexion elicited minor low back pain at approximately 100° elevation of the thighs from the examination table. Power in the legs appeared to be normal apart from some weakness (4/5) on dorsiflexion (L4,5) of the left foot. There was some weakness in his left hand on making a fist. The plantar responses appeared to be normal. The foot pulses were normal.

IMAGING REVIEW

Plain X-ray films of the cervical spine showed disc narrowing at the C4–5 and C5–6 levels with moderate degenerative changes in the associated lateral mass articulations and in the uncovertebral joints, with some narrowing of the left C4–5 nerve root foramen. Plain X-ray films of the lumbar spine showed minor degenerative osteophytic changes throughout the lumbar spine and extending into the lower thoracic region. A CT scan of the lumbar spine showed some degenerative change in the zygapophysial joints and at the vertebral body margins but no compromise of the spinal canal or exiting foraminae.

CLINICAL IMPRESSION

Cervical myelopathy in view of the left sided arm symptoms and the left sided 'foot drop'. Differential diagnosis of cervical nerve root impingement with a concomitant and separate condition of left sided L5 nerve root irritation.

WHAT ACTION SHOULD BE TAKEN?

A CT scan was performed of the cervical spine to evaluate the spinal canal. This showed, at the C4–5 level, a 'diffuse annular bulging of the disc and a large left posterolateral osteophyte formation indenting the thecal sac anteriorly with moderate spinal canal stenosis and left C4–5 intervertebral foramen stenosis' (Fig. 63.2A).

DIAGNOSIS

- Cervical cord myelopathy due to C4–5 diffuse disc bulge and large left sided posterolateral osteophyte causing spinal canal and left intervertebral foramen stenosis.
- Minor mechanical low back pain causing slight radiation to the left lower limb due to osteoarthrosis.

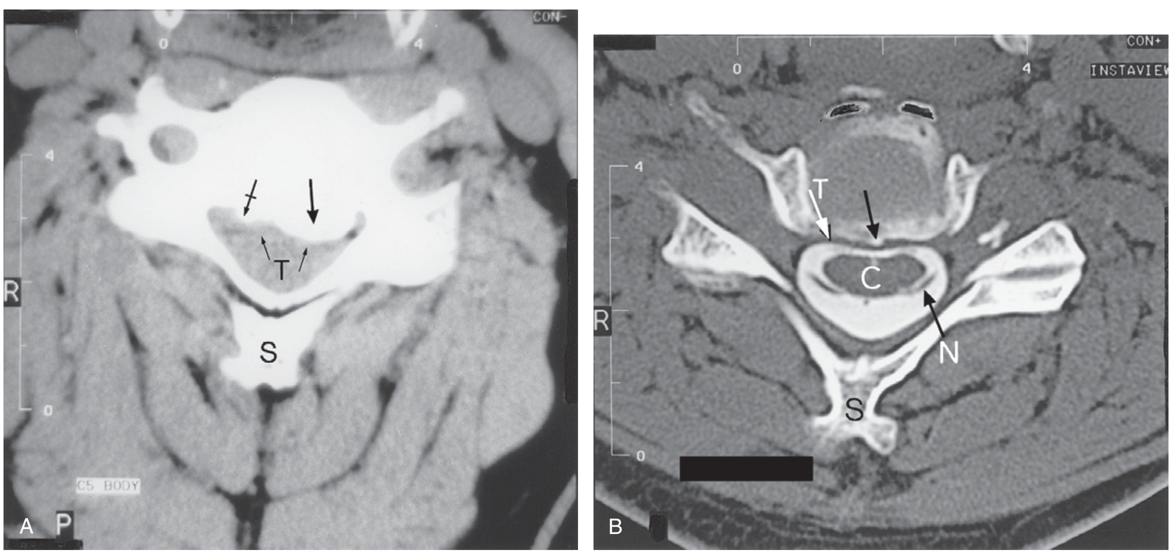

Figure 63.2 (A) Cervical spine CT axial image showing the C4–5 disc diffuse annular bulge and large left posterolateral osteophyte formation (large black arrow) indenting the thecal sac (T) anteriorly (small black arrows) with moderate spinal canal stenosis and left intervertebral foramen stenosis. There is some bony narrowing of the right C4–5 neural foramen due to degenerative changes in the adjacent uncovertebral joint (tailed arrow). R = right side of patient; S = spinous process. (B) Cervical spine CT myelogram from another patient showing how even a small posterior osteophyte (black arrow) will indent the anterior pain-sensitive part of the thecal sac (T) which contains the spinal cord (C) and nerve roots (N). Therefore, a large posterolateral osteophyte with diffuse annular bulging of the disc, causing left canal stenosis, would affect the tracts within the spinal cord with symptoms in the legs due to cervical cord myelopathy. R = right side of patient; S = spinous process.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

He was then referred for a neurosurgical opinion in view of the developing cervical cord myelopathy.

Surgical decompression at the C4–5 level provided relief from his neck and left arm pain and his foot drop.

Note

A relatively normal CT myelogram scan to demonstrate the radiological anatomy of the cervical spine in the axial view is shown in Fig. 63.2B for comparison with the stenotic spinal canal shown in Fig. 63.2A.

KEY POINTS

1. It is important to remember that cervical spondylitic myelopathy has a diverse clinical presentation without any pathognomonic signs or symptoms (Simeone & Rothman 1982, Shelokov 1991).
2. The myelopathic patient has a mixture of abnormalities of both upper and lower extremities with possible subtle gait disturbance with upper motor neuron dysfunction (Shelokov 1991).

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Case 64

Advanced osteoarthrosis in the cervical spine

COMMENT

The effects of appropriate needle acupuncture should not be ignored.

PROFILE

A 69-year-old male retired manual worker who is a non-smoker and only rarely drinks alcohol.

PAST HISTORY

He suffers from hypertension that is well controlled by medication. He had undergone a prostatectomy because of malignancy within the last 5 months.

PRESENTING COMPLAINT(S) (Fig. 64.1)

Since gardening 2 weeks ago he has developed bilateral neck pain, particularly in the suboccipital region and in the lower neck; the latter radiates to the right arm, almost to the elbow. The neck pain also causes headaches.

The neck pain and headaches do not awaken him at a particular time at night but his neck becomes stiff during the night. He can sit and watch the television without neck pain. Coughing and sneezing do not aggravate his symptoms. Periodic prostate-specific antigen (PSA) tests weeks before the consultation had shown that the PSA level had decreased significantly since his surgery 5 months prior to consultation.

AETIOLOGY

Working with a shovel in his garden had caused neck pain and headaches approximately 2 weeks before the consultation.

EXAMINATION

The blood pressure was 130/80 in the right arm in the seated position. All cervical spine movements were limited and elicited some mid to lower cervical spine pain in particular. Neurologically he was intact with power in the upper and lower extremities being normal. The plantar response was normal.

IMAGING REVIEW

No imaging had been performed.

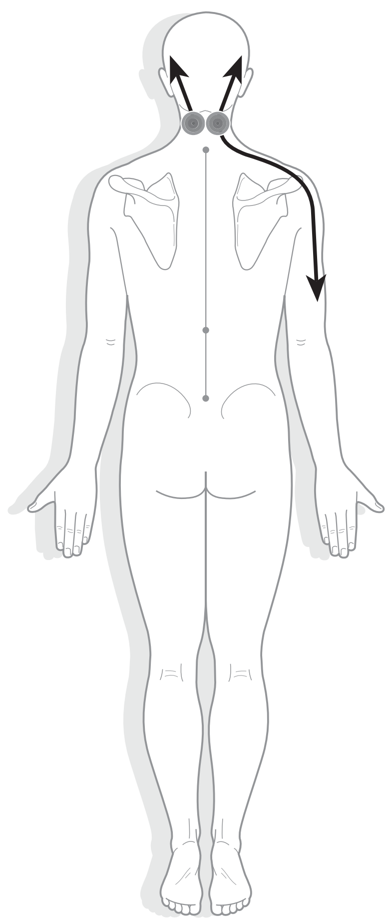

Figure 64.1

CLINICAL IMPRESSION

Osteoarthrotic changes in the cervical spine with a differential diagnosis of possible metastatic disease in view of his history.

WHAT ACTION SHOULD BE TAKEN?

A PSA blood test was performed and found to be 5.3 (i.e. normal for 61–70 years of age: range = 4–5.3 ng/ml). Cervical spine radiographs were requested and these showed 'widespread degenerative changes involving the intervertebral discs, neurocentral and zygapophysial joints. No other bony lesion seen' (Fig. 64.2A, B, C). In view of his history, a bone scan was performed as a precaution to look for any possible bony metastatic disease and this was reported as being normal. In view of the advanced osteoarthrotic changes in the cervical spine, he was advised to have a trial of needle acupuncture.

DIAGNOSIS

Advanced osteoarthrosis of the cervical spine causing headaches and right C5 radiculopathy.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Because of his neck pain and the considerable osteoarthrotic changes in his cervical spine, needle acupuncture was performed with him in the seated position so that he did not strain his neck, or injure the vertebral arteries, by lying prone. Acupuncture needles were placed in various locations where deep palpation of the paraspinal muscles elicited tenderness. Up to 12 needles were used at each visit.

He responded well to acupuncture treatment for his neck pain, headaches and pain radiating to the right arm. After six visits he reported that, since the last visit, he had only experienced one headache, so he was advised to come in as required should his neck pain and headache symptoms reoccur. Periodically he comes for 4–6 acupuncture treatments when his neck pain and headaches trouble him but he goes for considerable periods of time (up to 2 years) without symptoms.

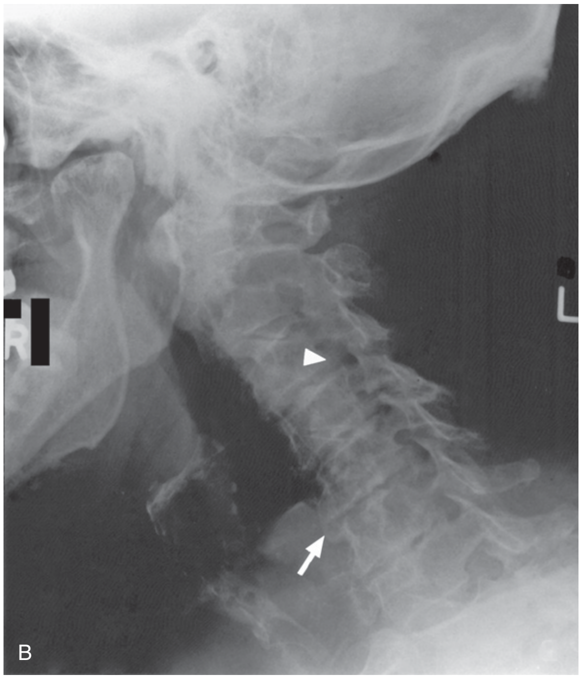

Figure 64.2 (A) Cervical spine lateral plain X-ray image showing the widespread osteoarthrotic degenerative changes involving the intervertebral and zygapophysial joints with disc space narrowing at C4–5, C5–6 and C6–7. Left (B) and right.

(Continued)

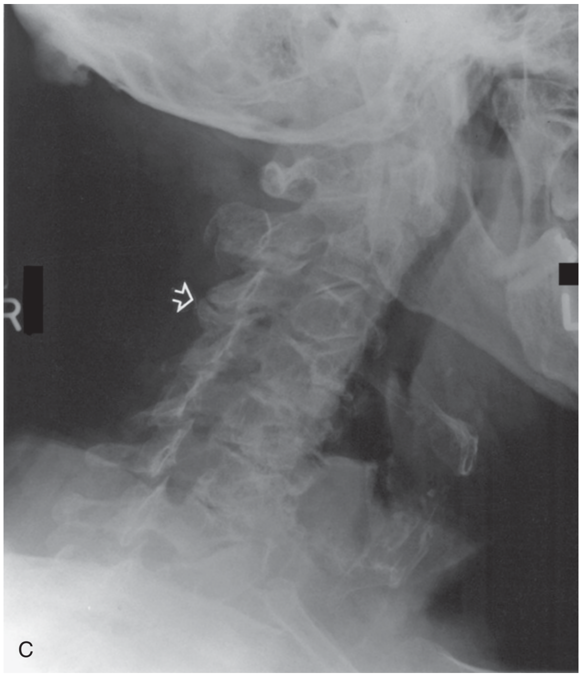

Figure 64.2 Cont'd (C) Oblique cervical spine plain X-ray images showing the widespread osteoarthrotic changes involving the intervertebral joints (white arrows), the zygapophysial joints (open arrow), and the uncovertebral joints with osteophytic encroachment upon some intervertebral foraminae (arrowhead).

KEY POINT

The value of needle acupuncture treatment for mechanical spinal pain in cases where manipulation is contraindicated should not be underestimated. Naturally, strict hygiene measures need to be used including the use of 'sharps' containers for appropriate disposal of needles.

Further reading

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Case 65

Cervical cord ependymoma

COMMENT

Remember that a patient may have a long history of several different serious conditions and yet present with simple symptoms that suggest yet another serious pathology.

PROFILE

A 56-year-old male manual worker who does not smoke or drink alcohol.

PAST HISTORY

Cervical spine pain causing headaches since a motor vehicle accident 20 years ago. Two years ago he underwent a laminectomy and fusion at L5 for sciatica; this gave him relief from his sciatica. A 20-year history of mid-thoracic spine pain since the motor vehicle accident.

PRESENTING COMPLAINT(S) (Fig. 65.1)

1. Frontal headache, originating suboccipitally, with some narrowing of the lateral visual fields.
2. A general feeling of being unwell.
3. Some 'weakness' of his right leg during the last month or so.
4. Right thumb slight weakness making it difficult for him to hold his pen.
5. Some mid-thoracic spine pain during the last 20 years.

There was no history of night pain apart from headaches awakening him during the night.

AETIOLOGY

Unknown.

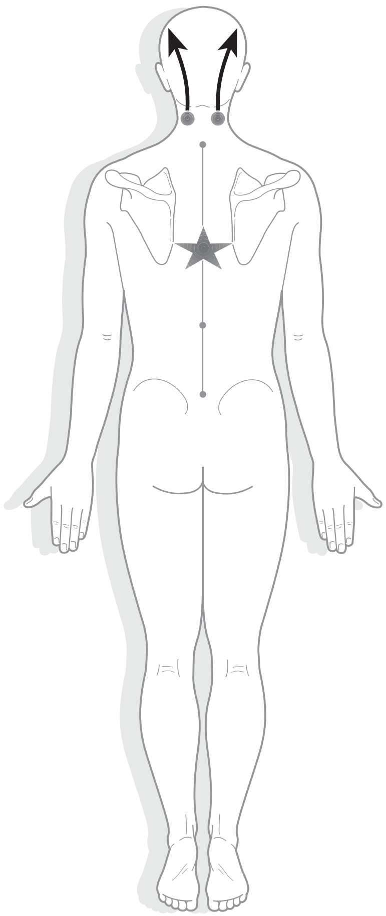

Figure 65.1

EXAMINATION

The deep tendon reflexes, pinprick sensation and vibration sensation were normal in the upper and lower limbs. There was decreased power (4/5) in right (a) shoulder abduction (C5), (b) finger flexion (C8) and extension (C7), (c) hand grip strength, and (d) his right leg. There was some narrowing of the left and right visual fields.

Cervical spine active ranges of movement were decreased by approximately 50% due to 'stiffness'. Thoracic spine active ranges of movement were decreased by approximately 25% on extension due to localized mid-thoracic spine pain.

IMAGING REVIEW

Cervical spine and thoracic spine plain X-ray films and a cervical CT scan taken approximately 2.5 years ago showed decreased C4–5 and C5–6 disc height and T6 body anterior wedging with associated anterior osteophytes. A recent CT of his brain and pituitary fossa showed a pituitary tumour protruding into the suprasella cistern, most probably causing some pressure effect on the adjacent optic chiasm. An MRI brain scan confirmed the pituitary tumour as most likely being a macroadenoma. An MRI thoracic spine examination showed a small disc protrusion at the T5–6 level.

CLINICAL IMPRESSION

1. Pituitary tumour causing headaches and diminished lateral visual fields.
2. Possible cervical cord tumour causing slight weakness of the right thumb and of the right leg.
3. Possible mid-thoracic spine disc causing local pain.

WHAT ACTION SHOULD BE TAKEN?

1. Referral to the neurosurgeon who was to operate on the pituitary tumour.
2. Laboratory tests: these showed an ESR of 26 (normal range <15 mm/hour).
3. Request for new cervical spine X-ray films (Fig. 65.2); these showed the previously known C4–5 and C5–6 decreased disc height (Fig. 65.2A) with only minor joint of von Luschka (uncovertebral joint) osteophytic lipping at the C4–5, C5–6 and C6–7 levels, particularly on the left oblique (Fig. 65.2B) and the anteroposterior view (Fig. 65.2D).
4. The patient was advised to ask the neurosurgeon who was to remove the pituitary tumour to have a cervical spine MRI performed in view of the right thumb and leg weakness. However, this was denied as being unnecessary. Three months later when the patient presented again, following surgical removal of his pituitary tumour, he said his right thumb weakness was progressively becoming worse so a cervical spine MRI study was arranged via referral to an orthopaedic surgeon. The MRI showed a large primary neoplasm (an ependymoma or a cystic astrocytoma) extending from C6 to T1 (Fig. 65.2E and F), thus explaining his right thumb and right leg weakness.

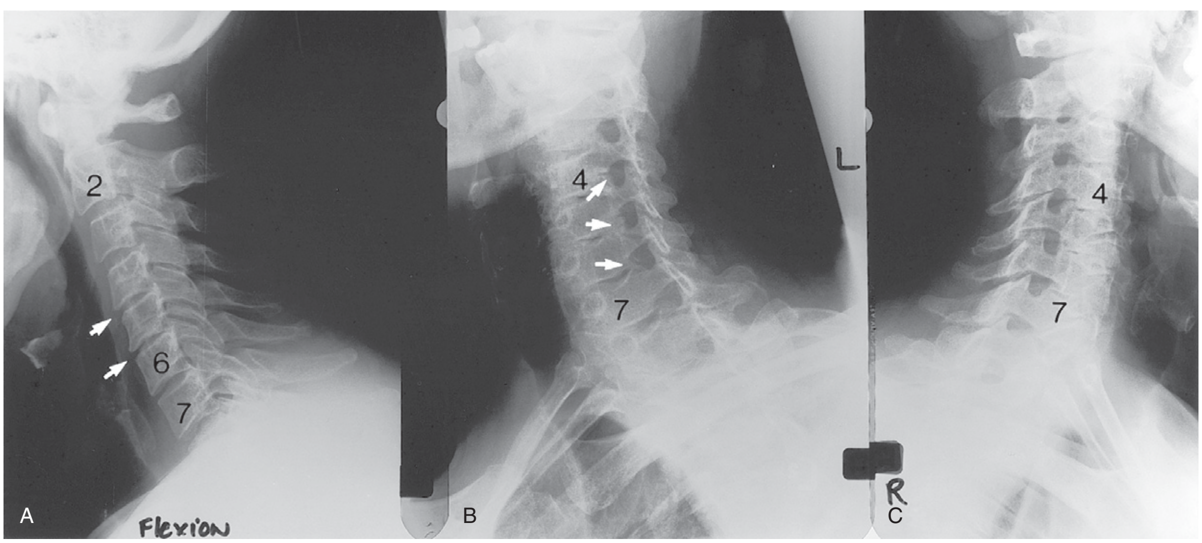

Figure 65.2 (A) Cervical spine flexion plain X-ray image showing the abnormal cervical spine contour, restriction in the range of flexion and loss of disc height at C4–5 and C5–6 (arrows). (B, C) Cervical spine oblique images showing only minor joint of von Luschka (uncovertebral joint) osteophytic lipping at the C4–5, C5–6 and C6–7 levels on the left side in particular (arrows).

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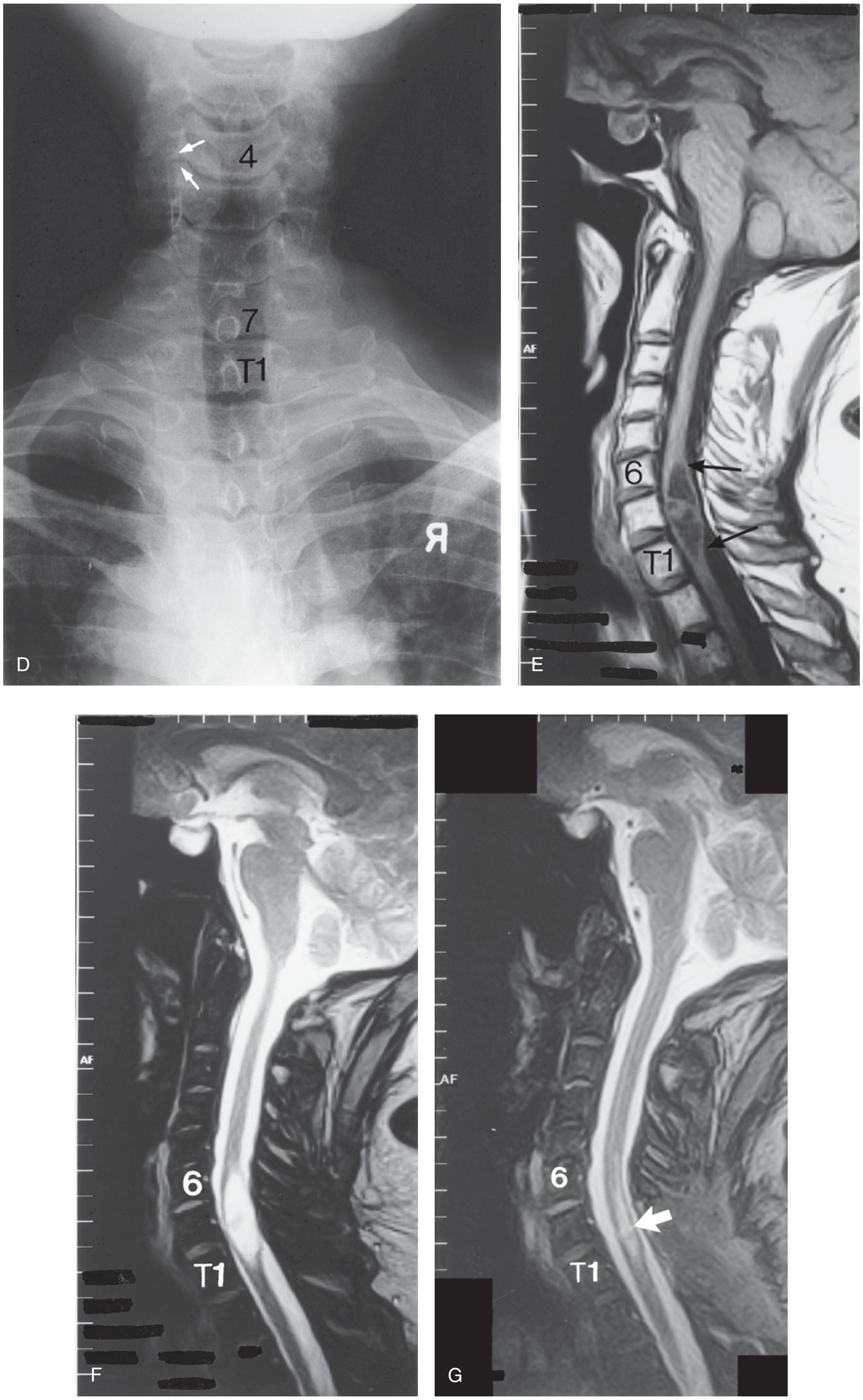

Figure 65.2 Cont'd (D) Cervical spine anteroposterior X-ray image which shows the osteophytic lipping of the joints of von Luschka at the C4–5 (arrows) and C5–6 levels on the left. The lung apices appear to be within normal limits and there are no cervical ribs. 7 = seventh cervical vertebra; T1 = first thoracic vertebra. (E) Cervical spine MRI sagittal T1-weighted image that shows the large ependymoma extending from C6 to T1 (arrows) in the cervical spinal cord. (F) Cervical spine MRI sagittal T2-weighted image that shows the ependymoma. (G) Cervical spine MRI sagittal T2-weighted postoperative image showing the change in the tumour 4.5 months following surgery.

DIAGNOSIS

Ependynoma extending from C6 to T1 levels of the cervical spinal cord.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was referred for urgent surgery for his cervical cord tumour.

The tumour was found to be an ependymoma on histopathology examination.

The cervical cord tumour surgery gave him very good results over some months with an almost complete return to normal strength of his right thumb and leg (Fig. 65.2G). Unfortunately, the tumour recurred 2.5 years later.

> **KEY POINT**
>
> Never think a patient is a malingerer just because more and more symptoms develop. Thoroughly review the history and consider all new symptoms before performing imaging and laboratory tests otherwise the patient will be disadvantaged.

Further reading

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Case 66

Cervical, lumbar, and thoracic spine injuries, as well as a foot injury, following a motorcycle accident

COMMENT

This case illustrates the necessity of listening to the patient and paying attention to detail with respect to evaluating a patient's complaints – there is no room for a clinician to believe arbitrarily that a patient is 'overstating the severity' of their symptoms.

PROFILE

A 21-year-old male of muscular build who is a manual worker. He does not smoke cigarettes or drink alcohol.

PAST HISTORY

Four years ago he was involved in a motorcycle accident in which he was thrown through the air and landed on his back. He had seen orthopaedic surgeons and a neurosurgeon regarding his ongoing symptoms. He was currently taking paracetamol and a non-steroidal anti-inflammatory drug for his spinal pains and headaches but without obtaining much relief. He showed me a letter from one of the consultants that he had seen stating that the consultant believed the patient was 'overstating the severity of his symptoms and that the cervical, lumbar and thoracic symptoms would not be related to the motorcycle accident'. However, the patient told me he was asymptomatic before the motorcycle accident.

PRESENTING COMPLAINT (Fig. 66.1)

1. Constant bilateral neck pain that causes severe headaches in the occipital region of his head and behind his ears. There are no radiating symptoms to his upper limbs. His symptoms are aggravated by coughing, which may cause an increase in his headache, and by looking down while reading.
2. Constant chronic lower back pain with radiation to the right thigh posteriorly that is activity related; for example, arising from the seated position may cause an increase in pain. The symptoms are also aggravated by coughing and by being in bed overnight; on arising in the morning his low back is stiff and painful.
3. Left foot pain located particularly on the lateral side of his ankle and the dorsum of his left foot where he received significant injuries during the accident. He

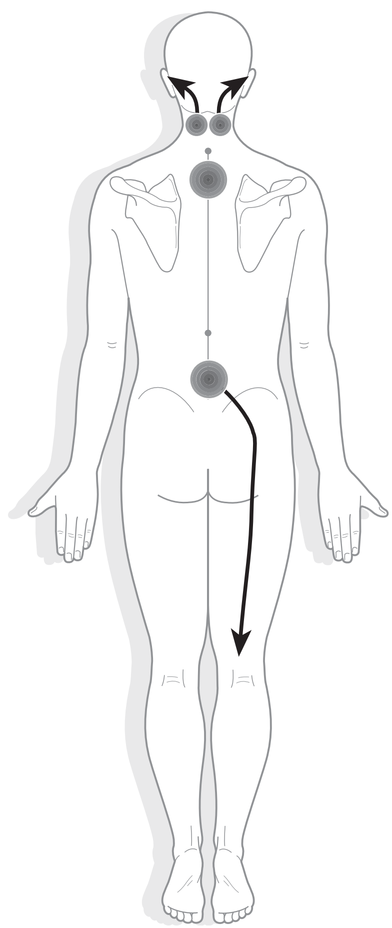

Figure 66.1

cannot tolerate a shoe on his left foot due to the very hypersensitive dorsum of the foot.

4. Intermittent upper thoracic spine 'ache' at approximately the T2 to T4 levels.

AETIOLOGY

Motorcycle accident 4 years ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Deep palpation of the paraspinal muscles elicited pain on the right side at C2–3, and bilaterally at C5–6, and pain at the L4–S1 level. There was muscle spasm on the left and right sides of the lower lumbar spine at approximately the L4–S1 level. Sacroiliac joint strain testing did not elicit any sacroiliac joint pain. Percussion of the lumbar and thoracic spines elicited pain at the L4–S1 level. Toe walking power (S1) and heel walking power (L5) were normal and the only slight weakness (4/5) in motor power in the upper and lower limbs was for right foot dorsiflexion (L4,5). Deep reflexes in the upper and lower extremities appeared to be normal as was the case with vibration sensation at the elbows and ankles. When seated slumped forward, there was no aggravation of his low back pain but the addition of straight leg raising elicited pain on the right side of the low back when the right and left legs, respectively, were raised. Supine straight leg raising was limited to a measured 90° (left) and painless, and to 68° (right) with low back pain. Straight leg raising with foot dorsiflexion was painless for the left side but right foot dorsiflexion caused a slight increase in low back pain. Straight leg raising with foot plantar flexion did not aggravate his low back pain, i.e. a normal response. Lasegue's sign for nerve root tension was painless for the left leg but caused a slight increase in low back pain when testing the right leg. The Milgram active bilateral straight leg raise elicited an increase in low back pain. The circumference of the calf (13 cm below the patella) was 42.5 cm (left) and 43 cm (right). The Fabere sign for hip joint function was essentially normal with only slight pain in the region of the left hip joint. Supine cervical spine flexion elicited some pain in the suboccipital region bilaterally. Hoover's test was normal.

Active cervical spine ranges of movement were measured, using a CROM instrument (see Box 66.1).

Other cervical spine and cervico-shoulder region tests were performed to check for pain of cervical spine origin as shown in Box 66.2.

Active lumbar spine ranges of movement were performed (Box 66.3).

False rotation of the pelvis was painless.

All active thoracic spine ranges of movement were of full range and were reported as being painless.

IMAGING REVIEW

Plain X-ray films taken of his left ankle following the accident showed that he had fractured his ankle. Surprisingly, no spinal radiographs had been taken for a period of approximately 3 years following the accident and then only lumbar spine radiographs were taken. These showed a compression fracture of the L4 vertebral body.

CLINICAL IMPRESSION

- Possible mid-cervical spine central disc bulge.
- L4 vertebral body fracture.
- Upper thoracic spine soft tissue injuries.

WHAT ACTION SHOULD BE TAKEN?

In view of the L4 vertebral body fracture discovered 3 years after his spinal injuries, it was considered necessary to examine his cervical and thoracic spines with plain X-ray

Box 66.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	55°	Elicited bilateral cervical spine pain that radiated to approximately the T3 level
Extension	60°	**50°**	Elicited a minor neck ache
Lt lateral bending	45°	**30°**	Elicited pain in the left suboccipital area that extended to behind the left ear
Rt lateral bending	45°	45°	Elicited pain to the right of approximately the C3–6 level
Lt rotation	80°	80°	Elicited pain to the right of approximately the C1–2 level
Rt rotation	80°	80°	Elicited slight pain on the left of the C1–2 level and on the right at approximately the C3–6 level

Note that there was some restriction of movements and that flexion exceeded the normal range.

Box 66.2 Other cervical spine and cervico-shoulder region tests

	Patient's comments
Lt rotation plus extension	Elicited pain on the left side at approximately the C1–3 level
Rt rotation plus extension	Elicited pain on the right side at approximately the C1–3 level
Cervical spine traction	Painless
Cervical spine compression	Painless
Downward shoulder pressure	Painless
Trapezius trigger point pressure	Elicited pain in the right trigger point

Box 66.3 Active lumbar spine ranges of movement

	Approx range	Patient's comments
Flexion	Fingers reached to his ankles	Elicited lower back pain
Extension	Limited by approx. 15%	Elicited lower back pain
Lt lateral bending	Fingers reached to his knees	Painless
Rt lateral bending	Fingers reached to his knees	Elicited 'tingling' extending from the low back to the back of his right leg and to the right big toe
Lt rotation	Full	Painless
Rt rotation	Full	Painless

images in view of his ongoing neck and thoracic spine pains. The cervical spine radiology report stated: 'No significant bony deformity. Intervertebral disc spaces and intervertebral foramina are intact. Functional views are normal'. However, on review of the films the imaging showed a loss of the normal cervical spine lordosis (Fig. 66.2) with some thinning of the C5–6 intervertebral disc space height. Furthermore, the cervical spine flexion view (Fig. 66.3) showed an angulation at the C5–6 level. The plain film thoracic spine radiograph was not contributory.

In view of the plain X-ray findings an MRI was performed of his cervical, thoracic and lumbar spines in order to fully evaluate this young man's spine in view of his chronic cervical and lumbar spine pain syndromes and his intermittent upper thoracic spine ache. The cervical, lumbar and thoracic spine MRI reports are summarized below.

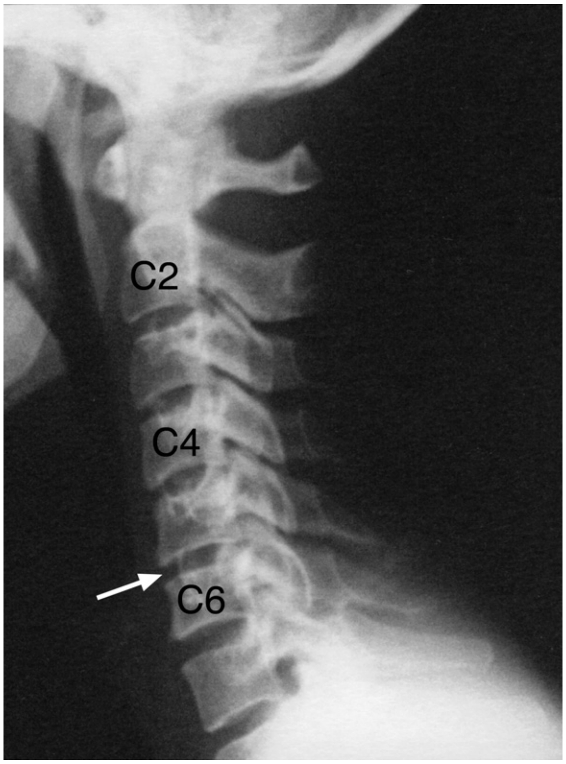

Figure 66.2 Cervical spine lateral plain X-ray image. Note (i) there is a loss of the normal lordosis of the cervical spine with some straightening of the spine from C3 to C6, and (ii) some thinning of the C5–6 intervertebral disc space height (arrow).

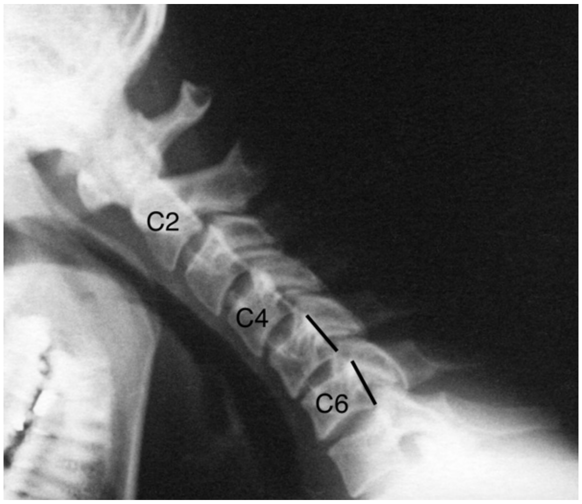

Figure 66.3 Cervical spine flexion plain X-ray image. Note the angulation at the C5–6 level (as indicated by the two black lines).

1 Cervical spine

'There is a posterior disc protrusion at C5/6' (Fig. 66.4) 'that is essentially left paracentral but it probably does not compromise the nerve roots'.

Therefore, this posterior disc protrusion would not cause radicular symptoms. However, it could cause neck pain with resulting muscle spasm and occipital headaches due to disc pressure upon the following structures:

a. The pain sensitive anterior surface of the dural tube.
b. The recurrent meningeal nerves between the protrusion and the dural tube.
c. The blood vessels between the protrusion and the dural tube.

There is a degree of desiccation of all the cervical spine discs strongly suggesting injury to these discs (Fig. 66.4).

2 Lumbar spine

'There is depression of the superior endplate of L4 which results in greater than 20% loss of vertical height of the anterior and mid-portions of L4. Slightly reduced hydration of the L3–4 intervertebral disc.' (Fig. 66.5).

As there is no evidence of oedema in the L4 body, this is an old endplate fracture that, in all likelihood, fits in with the time-frame since his accident 4 years previously.

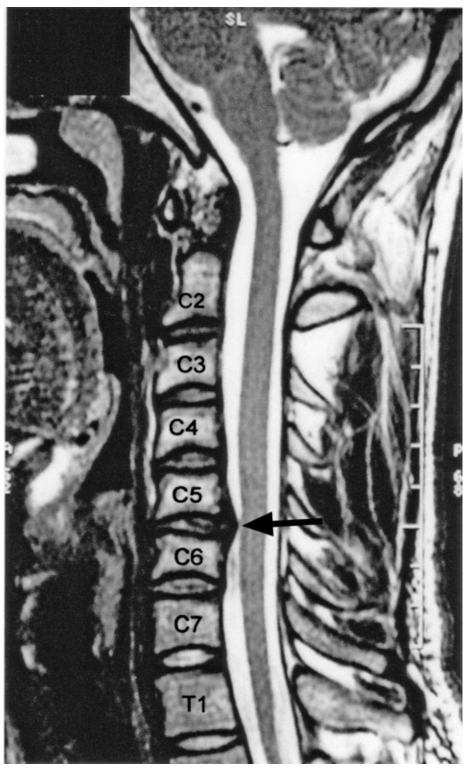

Figure 66.4 Cervical spine MRI sagittal T2-weighted image. Note the considerable C5–6 posterior disc protrusion (arrow). The protrusion presses upon the pain sensitive anterior part of the dural tube and, in all likelihood, presses upon the recurrent meningeal nerves and blood vessels between the protrusion and dural tube.

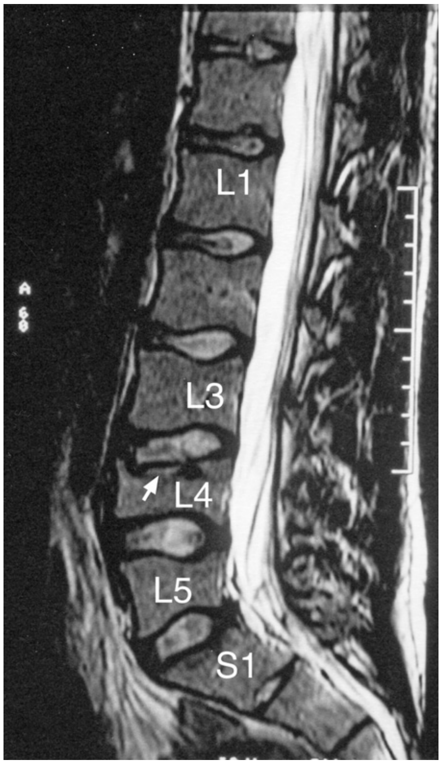

Figure 66.5 Lumbar spine MRI parasagittal T2-weighted image. Note the depression of the superior end plate of L4 (white arrow) that results in greater than 20% loss of vertical height of the anterior and mid-portions of the L4 vertebral body. As there is no evidence of oedema in the L4 body, this is an old endplate fracture that, in all likelihood, fits in with the time-frame since his accident. There is slightly reduced hydration of the L3–4 intervertebral disc.

3 Thoracic spine

'A minor posterior disc bulge is present at T2–3 and at T3–4' (Fig. 66.6). The T3–4 posterior bulge presses upon the pain sensitive anterior surface of the dural tube and there is moderate desiccation of the T2–3, T3–4, T6–7 and T7–8 thoracic discs. In addition, there is a Schmorl's node in the superior endplate of the T6 vertebral body where disc material has herniated through a fracture in the endplate.

DIAGNOSIS

Cervical spine

- Musculoligamentous soft tissue injuries of the neck including a C5–6 intervertebral disc protrusion.
- Occipital headaches of cervicogenic origin.

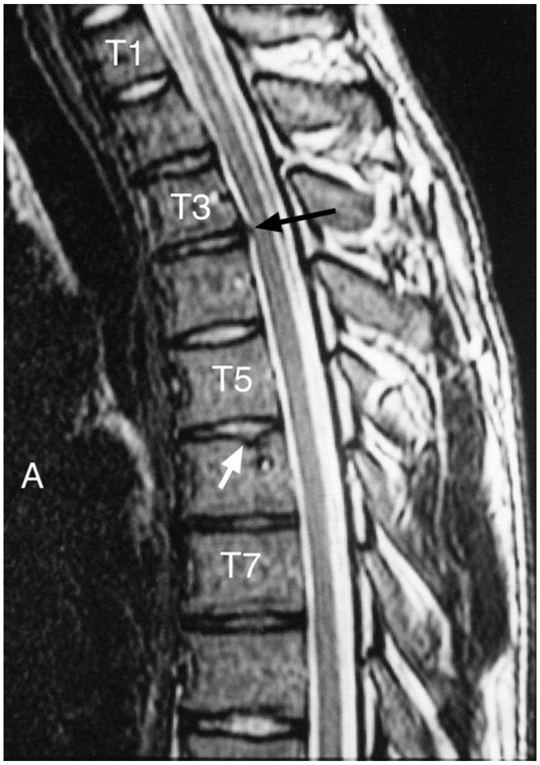

Figure 66.6 Thoracic spine MRI sagittal T2-weighted image. Note (i) there appears to be a small posterior disc bulge at the T3–4 level pressing upon the pain sensitive anterior part of the dural tube/thecal sac (black arrow), (ii) there is moderate desiccation of the T2–3, T3–4, T6–7 and T7–8 thoracic discs, and (iii) there is a Schmorl's node in the superior endplate of the T6 vertebral body (white arrow) where disc material has herniated through a fracture in the endplate.

Lumbar spine

- Musculoligamentous soft tissue injuries, particularly at the L4–S1 level of his spine.
- L4 vertebral body superior endplate compression fracture.
- Schmorl's nodes in the superior endplate of the L4 vertebral body due to disc material rupturing into the vertebral body.

Thoracic spine

- Musculoligamentous soft tissue injuries of the upper thoracic spine.
- Minor posterior disc bulges at the T2–3 and T3–4 levels.
- Moderate desiccation of the T2–3, T3–4, T6–7 and T7–8 thoracic discs.
- A Schmorl's node in the superior endplate of the T6 vertebral body where disc material has herniated through the fracture in the endplate.

Non-spinal injuries

Left foot fractures and soft tissue injuries.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He therefore understood why he had genuine pain in his cervical, lumbar and thoracic spines. He said he knew that he had genuine pain but, apart from the L4 vertebral body fracture discovered approximately 3 years following his accident, when he asked his general medical practitioner to X-ray his low back, no-one had taken him seriously until now. He was advised to:

1. See a spinal surgeon should his C5–6 posterior disc protrusion cause him undue symptoms in the future.
2. Undertake a muscle strengthening programme for the lumbar spine (see Case 1).
3. Lessen his medication as the supporting muscles of his lumbar spine strengthen, so as to avoid possible side effects.

He was told that he would most likely develop osteoarthrotic changes involving his injured spinal joints but that he should not aggravate his injuries by performing activities that could place undue forces upon his spine and that he would have to modify his workplace activities and perhaps undertake job retraining. He said he would deal with his spinal pains using the above conservative approach as he now understood why it was that he had experienced chronic spinal pain syndromes for the last 4 years.

KEY POINTS

1. Even when a patient has seen a 'specialist', be thorough and arrive at your own diagnosis.
2. It is very important to listen to the patient, perform a thorough physical examination, and investigate spinal pain complaints with appropriate imaging. There is no place for a cursory evaluation of a patient's spinal pain syndromes, particularly when there is a history of trauma.

Case 67

Cervical ribs

COMMENT

Always examine X-ray images for rudimentary cervical ribs as they may not be reported.

PROFILE

A 48-year-old male of muscular build who is a self-employed manual worker. He does not smoke cigarettes and only occasionally drinks alcohol. He tries to keep fit and active.

PAST HISTORY

He was involved in a motor vehicle accident approximately 15 years before his symptoms occurred.

PRESENTING COMPLAINT (Fig. 67.1)

Approximately 6 years ago he began to develop bilateral mid-to-lower neck pain that radiated into the left and right cervico-shoulder regions. His symptoms are now chronic, almost constant and are of varying degrees of intensity. His pains frequently cause occipital headaches that he believes begin in his neck. He also experiences chronic pain in the right C7/C8 distribution with paraesthesiae in the fourth and fifth fingers (C8) of his right hand. An EMG study had been performed as well as a CT cervical spine (including a CT angiogram of the subclavian arteries) and these were reported as being normal. He had tried acupuncture without success but had found that occasional cervical spine manipulation gives him temporary relief. However, he wants to know why it is that he has this condition as several X-ray and physical examinations and the EMG have not explained what is causing his symptoms.

AETIOLOGY

No specific event precipitated his symptoms of gradual onset.

EXAMINATION

In the erect posture, there was no evidence of pelvic obliquity or of scoliosis. Deep palpation of the cervical and thoracic spine muscles elicited bilateral tenderness at the cervico-thoracic junction of the spine. The left upper part of the trapezius muscle, i.e. in the left cervico-shoulder

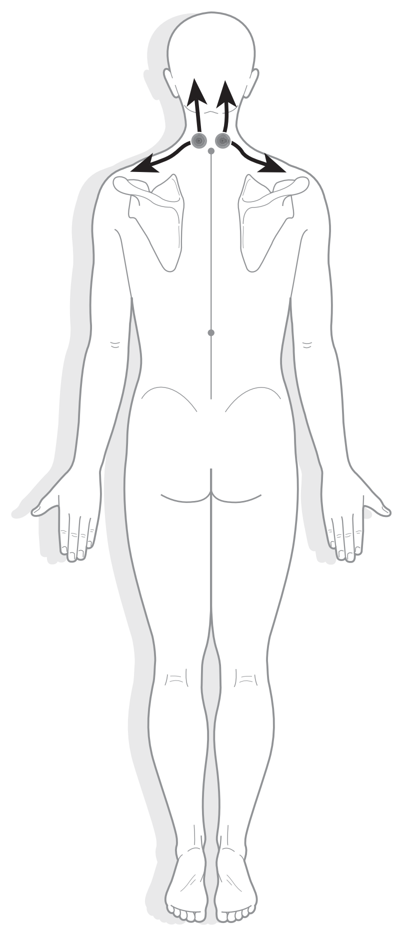

Figure 67.1

region, was painful to deep palpation. Active ranges of cervical spine movement were full and painless. The blood pressure was 140/95 in the seated position in the right arm. There was no bruit over the carotid arteries. The deep reflexes in the upper and lower extremities were normal, as was the case with pinprick sensation. Vibration sensation at the elbows and ankles was normal. The Valsalva manoeuvre did not elicit any pain. Adson's test was positive on the right side. There was no evidence of cyanosis in the upper limbs.

IMAGING REVIEW

Three sets of cervical spine plain X-ray films taken during the last 6 years and reported as being normal were available for review. On review, the anteroposterior (e.g. Fig. 67.2) and oblique views (Fig. 67.3) showed small bilateral bony cervical ribs at C7 level.

CLINICAL IMPRESSION

C7 bilateral rudimentary cervical ribs causing his symptoms.

WHAT ACTION SHOULD BE TAKEN?

As the C7 bilateral cervical ribs were considered to be the cause of his symptoms, no further imaging was performed.

DIAGNOSIS

C7 bilateral rudimentary cervical ribs.

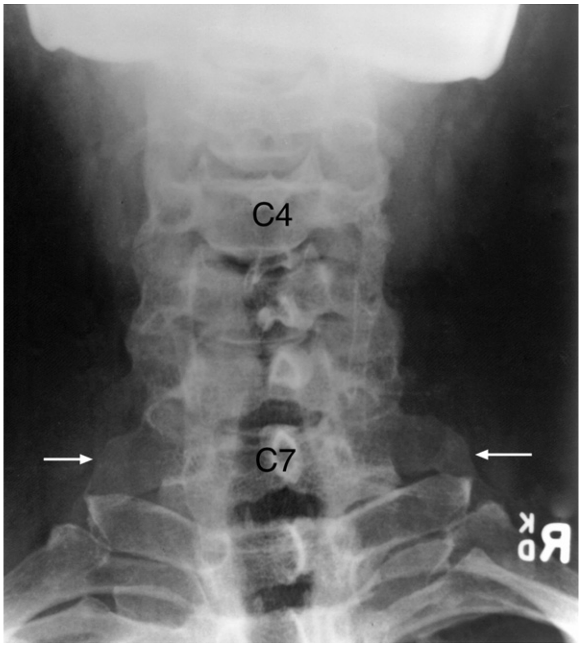

Figure 67.2 Cervical spine anteroposterior plain X-ray image. Note the small bilateral bony cervical ribs (arrows).

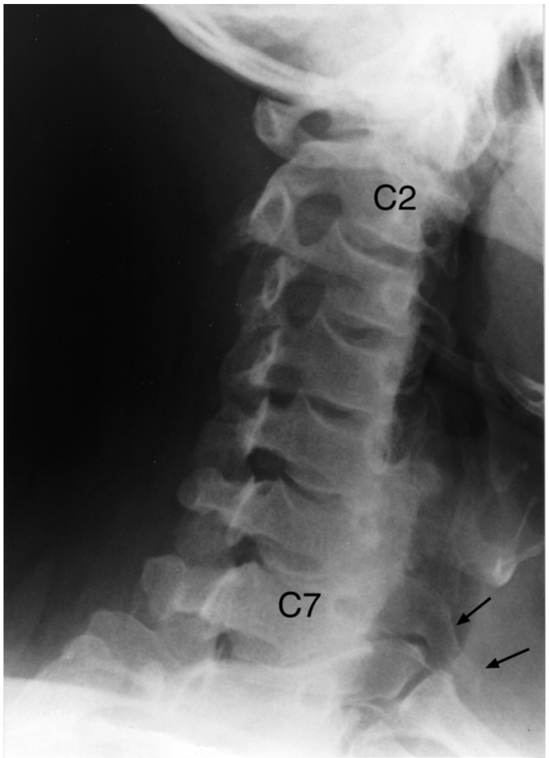

Figure 67.3 Cervical spine oblique plain X-ray image. Note one of the small bilateral bony C7 cervical ribs (arrows).

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was told that the most likely cause of his symptoms was the bilateral bony cervical ribs. In addition he was told that the extent of any fibrous continuation of these bony ribs would not show up on the imaging. He was relieved to know that the most likely cause of his symptoms was the bilateral cervical ribs at C7 as he was becoming concerned about the possibility of a hitherto 'undiagnosed malignancy'.

He was advised not to work above his shoulders and not to lift heavy weights. He was advised to use a pillow of appropriate height between his shoulder and the side of his head so that he did not aggravate his symptoms at night. He responded very well to being reassured and taking a conservative approach to his condition.

Note

It is important to illustrate to patients what is meant by a rudimentary cervical rib and to show its relationship to the associated neurovascular structures. For this purpose, please note the simple anatomical diagram shown in Figure 67.4

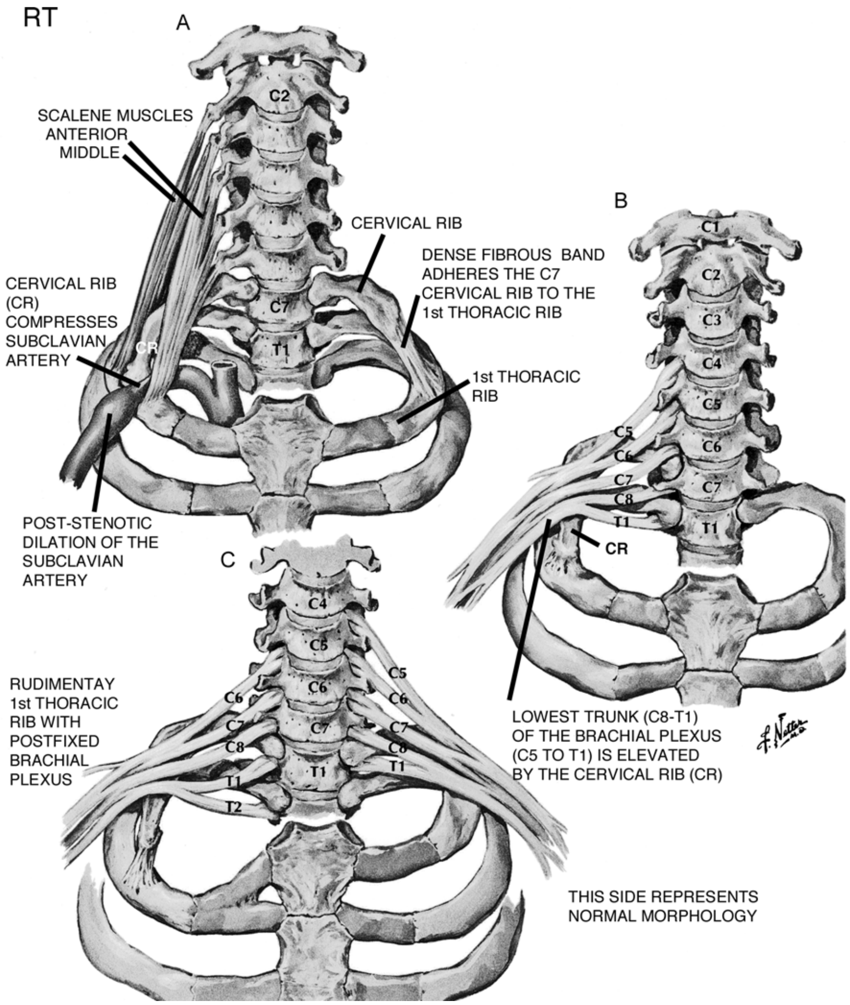

Figure 67.4 (A), (B) and (C) show anteroposterior views of the cervical and upper thoracic spine levels. Note in (A) how the cervical rib (CR) may compress the adjacent blood vessel. In (B) the cervical rib may elevate the lowest trunk (C8–T1) of the brachial plexus. In addition, there may be a dense fibrous band (see A) bridging from the tip of the C7 cervical rib to the 1st thoracic rib. An X-ray will not show this fibrous band. In (C) normal morphology is shown on the left side. RT = right side of figures. Reprinted from Netter Anatomy Illustration Collection, © Elsevier Inc. All Rights Reserved. Labels modified with permission.

According to orthopaedic surgeons Adams & Hamblen (2001), a cervical rib is a congenital over-development (bony or fibrous) of the costal process of the 7th cervical vertebra which often exists without causing symptoms, especially in the young; in adult life the tendency to gradual dropping of the shoulder girdle may lead to it causing neurological or vascular disturbance in the upper limb. According to Sunderland (1968), it is important to remember that most abnormal ribs go unrecognized or show up unexpectedly in a routine film taken for other reasons and it appears that precipitating factors are required to convert an asymptomatic condition into a troublesome one. When symptoms occur they may be *vascular* (e.g. weak or absent radial pulse, dusky cyanosis of the forearm and hand) if the subclavian artery is involved, or *neurological* (pain and paraesthesiae in the forearm and hand); neural and vascular symptoms may be combined as the subclavian artery and the lowest trunk of the brachial plexus arch over the rib (Adams & Hamblen 2001) and result in the condition of thoracic outlet syndrome (Brinker et al 1997). The most common symptom of thoracic outlet syndrome is pain in the neck, upper limbs or signs of vascular occlusion (Brinker et al 1997).

KEY POINTS

Cervical ribs can play an important role in the pathogenesis of cervico-thoracic junction pain and may cause neurological and or vascular disturbance in the upper limb(s).

References

Adams J C, Hamblen D L 2001 Outline of orthopaedics, 13th edn. Churchill Livingstone, Edinburgh.

Brinker M R, Weeden S H, Whitecloud T S III 1997 Congenital anomalies of the cervical spine. In: Frymoyer J W (Editor-in-Chief) The adult spine principles and practice, 2nd edn. Lippincott-Raven, New York, p 1205–1231.

Sunderland S 1968 Nerves and nerve injuries. E and S Livingstone, Edinburgh.

Case 68

Block vertebrae

COMMENT

Beware of cervical block vertebrae, i.e. fusion of adjacent vertebrae.

PROFILE

A 48-year-old married housewife who does not smoke and only drinks alcohol socially.

PAST HISTORY

She has a past history of 15 years of a cervical spine pain syndrome that led to chronic anxiety.

PRESENTING COMPLAINT(S) (Fig. 68.1)

Mid-to-lower cervical spine pain that radiates to the left and right cervico-shoulder regions and was of gradual onset over an approximately 15-year period; the pain is now severe. This has led to some disability regarding neck movements as she is frustrated because she is unable to perform her housework. Her complaint is steadily worsening.

She occasionally experiences 'dizziness' but has no history of fits. Occasionally, she experiences occipital headaches which she believes come from the neck and are increasing in frequency. Bowel and bladder function are normal.

Coughing may aggravate her neck pain. She requires a contoured pillow for supporting her neck at night.

She had previously tried physiotherapy treatment, spinal manipulation and anti-inflammatory medication, without success.

AETIOLOGY

Unknown, but no history whatsoever of trauma.

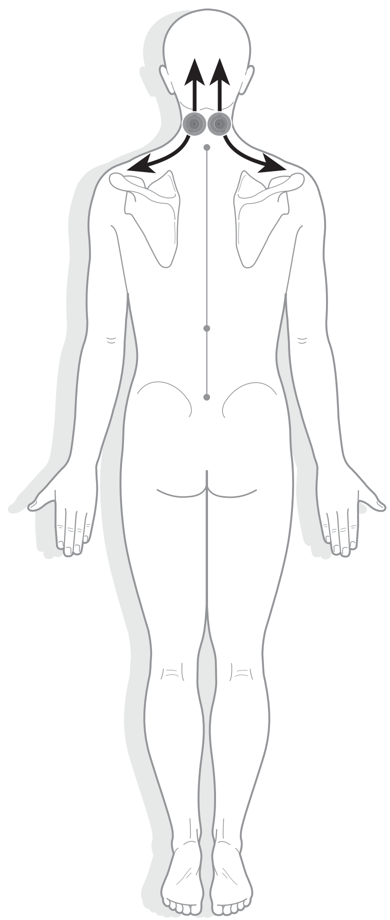

Figure 68.1

EXAMINATION

All cervical spine ranges of movement were limited by approximately 50% due to mid-to-lower neck pain except for extension which was limited by approximately 75% for the same reason. The deep tendon reflexes in the upper and lower extremities were slightly hyper-reflexic. Sensation to pinprick was normal as was vibration sensation. Muscle strength was normal. The plantar response was normal. Deep palpation of the paraspinal muscles of the cervical spine elicited tenderness over the mid-to-lower cervical spine but particularly at the C5–6 level.

IMAGING REVIEW

Previous plain film imaging was reviewed and this showed a partial congenital block vertebra at the C6–7 vertebral level.

CLINICAL IMPRESSION

Cervical spine pain syndrome due to degenerative changes involving the C5–6 level.

WHAT ACTION SHOULD BE TAKEN?

Updated cervical spine radiographs were requested and these showed: 'Disc space narrowing at C5–6 and C6–7 (Fig. 68.2A) with large posterior osteophytes at the C5–6 level. There is uncinate process hypertrophy with prominent osteophytes encroaching into the right neural foramen at C5–6 level. There is partial congenital block vertebra at C6–7 level'. In view of the plain film findings, an MRI examination was performed of the cervical spine. This showed: 'Partial developmental fusion of C6 and C7 vertebral bodies. Degenerative changes at the C3–4, C4–5 and C5–6 levels, most marked at the C5–6 level. There are disc/osteophyte impressions on the dural tube at these levels, most marked at C5–6 level. This causes borderline canal stenosis at the C5–6 level but the canal appears adequate at other levels' (Fig. 68.2B). There is also moderate osteophytic narrowing of the C5–6 intervertebral foramina bilaterally with the remaining foramina appearing adequate.

In addition there is some buckling of the ligamenta flava from C3 to C6. Note that all the degenerative changes are above the block vertebrae.

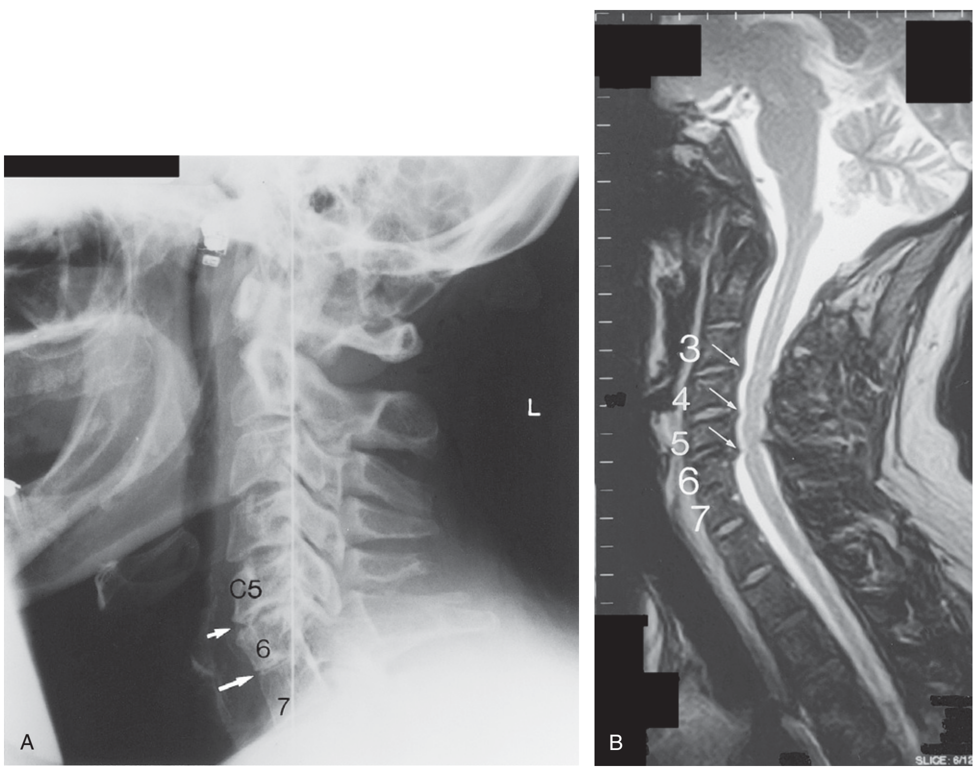

Figure 68.2 (A) Cervical spine lateral plain X-ray image showing the disc space narrowing at C5–6 (small white arrow) and the partial congenital 'block vertebra' at C6–7 (large white arrow). Note the angulation in the cervical spine contour at the C5–6 level which is clearly shown in relation to the vertical plumb line. L shows that the left side of the patient was against the X-ray bucky. (B) Cervical spine MRI sagittal T2-weighted image showing degenerative changes at the C3–4, C4–5 and C5–6 levels (particularly at the C5–6 level) (arrows) with disc/osteophyte impression on the dural tube at these levels with borderline canal stenosis at the C5–6 level. The vertebral bodies have been labelled 3, 4, 5, 6, 7 just to their left side so as not to obliterate the vertebral body with a numeral. Note the partial developmental fusion of the C6 and 7 vertebral bodies.

DIAGNOSIS

C5–6 disc/osteophyte degenerative changes with impression upon the dural tube and borderline spinal and bilateral intervertebral canal stenosis at C5–6 with associated buckling of the ligamenta flava at these levels.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was given the option of medication or acupuncture treatment and she said that, as medication had previously not helped, she would prefer to try needle acupuncture. She did not want a surgical opinion until she had tried acupuncture.

She had 15 acupuncture treatments and reported at that time that she had obtained 'good relief' from her neck pain syndrome. She returned 2 months later and had a further course of 10 acupuncture treatments, again with good results. She then came occasionally, thereafter, during a 4-year period, whenever she experienced a recurrence of symptoms. When seen 4 years later she had experienced a recurrence of her symptoms due to having fallen but again responded well to acupuncture treatment.

Note

This case provides an example of how congenital 'block vertebrae' can cause excessive degenerative changes at levels above the congenital fusion when there is no history of neck trauma.

KEY POINTS

1. This case, with no history whatsoever of trauma, indicates how considerable disc wear and tear changes can occur above the fused i.e. block vertebrae level.
2. When a patient has congenital block vertebrae, it is prudent to realize that there may be associated considerable soft tissue changes above the fused i.e. block vertebrae level.
3. In block vertebrae cases, plain X-ray films may not adequately indicate the soft tissue changes, so an MRI study may be necessary, depending on a patient's age, history, symptoms and signs.

Further reading

Banki Z 1980 Partielle Blockbildung an der Halswirbelsaule. Fortschritte auf dem Gebiete der Roentgenstrahlen und der Nuklearmedizin 133: 637–640.

Bullough P G, Boachie-Adjei O 1988 Atlas of spinal diseases. JB Lippincot, Philadelphia, p 121.

Chandraraj S 1987 Failure of articular process (zygapophysial) joint development as a cause of vertebral fusion (blocked vertebrae). Journal of Anatomy 153: 55–62.

Case 69

Cerebellar tonsil ectopia

COMMENT

Always remember that the arterial blood supply to the anterior inferior cerebellar arteries, the basilar artery and the superior cerebellar arteries may be compromised by pressure upon the vertebral arteries.

PROFILE

A 45-year-old female sedentary worker who does not smoke or drink alcohol.

PAST HISTORY

Motor vehicle accident 3 years ago.

PRESENTING COMPLAINT (Fig. 69.1)

Constant bilateral neck pain that radiates from the lower cervical spine into the cervico-shoulder regions, particularly on the right side, since a motor vehicle accident 3 years ago. The neck pain varies in intensity causing occasional occipital headaches. The neck pain worsens with activity, especially lifting objects and flexing her cervical spine. Because of the neck pain, she can only sit for short periods of time. On looking up she feels slight 'dizziness'.

The neck pain and headaches are made worse with coughing and sneezing but settle with rest. Considerable doses of non-steroidal anti-inflammatory drugs and analgesics provide only temporary relief. She wanted a diagnosis to be made as she was concerned about her hitherto unexplained symptoms.

AETIOLOGY

Motor vehicle accident 3 years ago.

EXAMINATION

Cervical spine flexion and extension were limited due to neck pain with a minor degree of 'dizziness', extension being very limited, i.e. to only approximately 10°. All

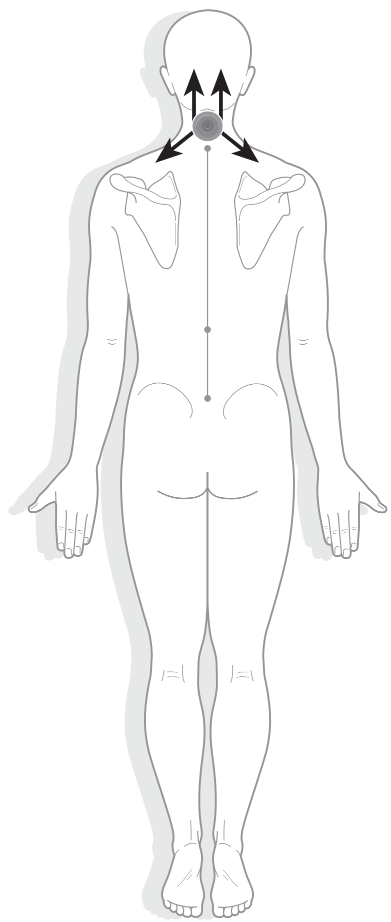

Figure 69.1

other ranges of movement elicited neck pain but were not limited by it. There was generalized tenderness in the paraspinal cervical muscles on deep palpation. Her blood pressure was normal, at 120/80 in the seated position.

IMAGING REVIEW

Plain X-ray films of her neck showed a loss of normal lordosis with a kyphosis above the C5 vertebra. On cervical spine flexion, there was a slight anterolisthesis of C4 on C5. There were osteophytes at the antero-inferior margins of the C4 and C5 vertebral bodies.

CLINICAL IMPRESSION

Possible vertebral artery compression in certain positions of the neck and head causing vertigo.

WHAT ACTION SHOULD BE TAKEN?

A cervical spine MRI was requested and this showed: 'Minimal uncovertebral joint hypertrophy in the lower cervical spine with no significant exit foramen encroachment. C5–6 shallow central lateral disc protrusion extending to the right with early disc narrowing and loss of cervical lordosis' (Fig. 69.2A and B). However, the report did not mention the ectopia of the cerebellar tonsils amounting to

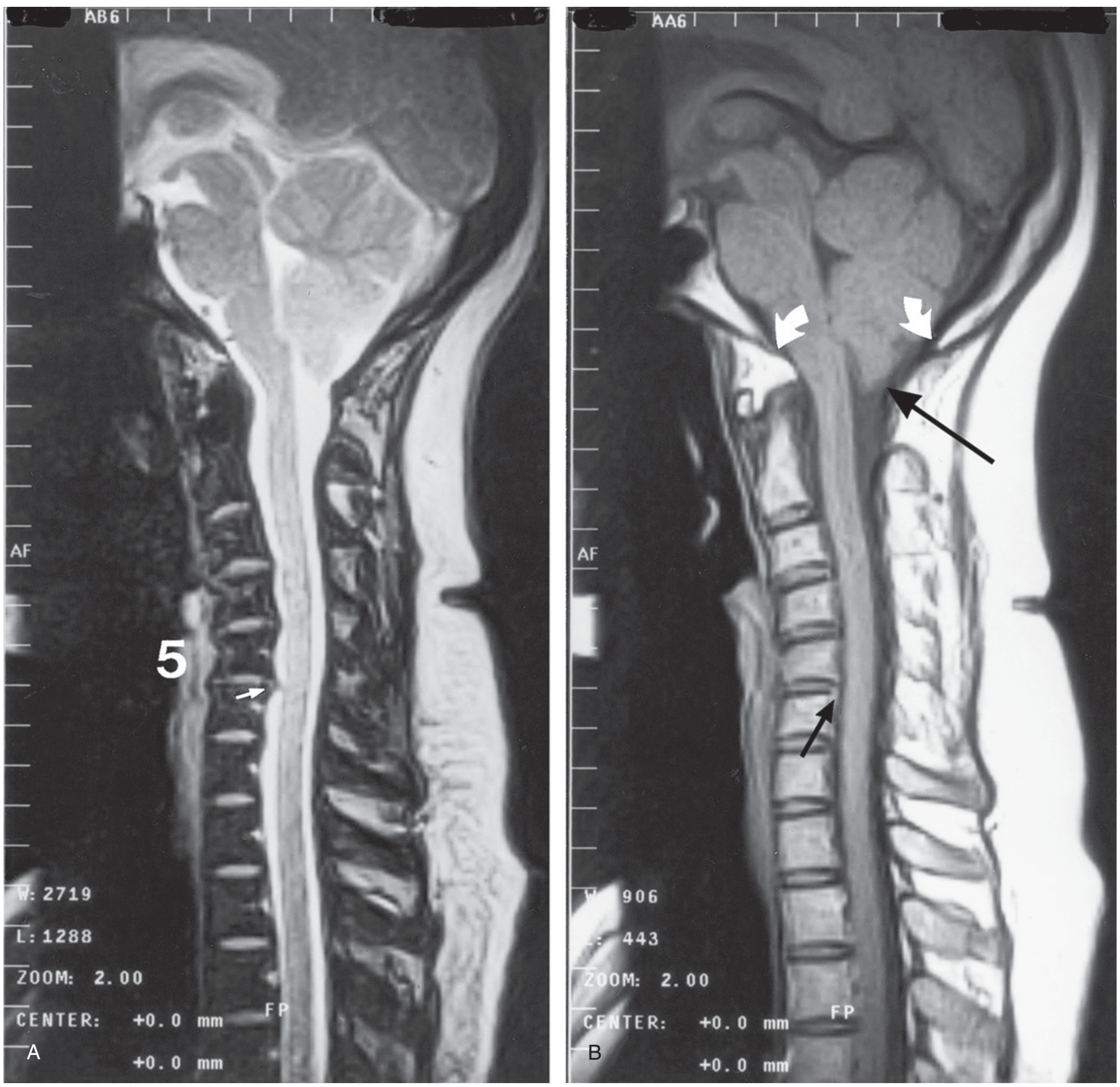

Figure 69.2 (A) Cervical spine MRI sagittal T2-weighted image showing the disc protrusion at the C5-6 level (small white arrow) and loss of the normal cervical lordosis with a mild kyphosis at the C5 level. (B) Cervical spine MRI sagittal T1-weighted image showing loss of the normal cervical lordosis with a mild kyphosis at the C5 level and disc material at the C5-6 level posteriorly (small black arrow). The cerebellar tonsil ectopia is seen more clearly on the T1-weighted image and the anterior and posterior margins of the foramen magnum are shown by the short white curved arrows. Note the cerebellar tonsil ectopia projecting below the foramen magnum (large black arrow); this woman had a mild form of Chiari I maformation.

approximately 10 mm of cerebellar ectopia, a mild form of Chiari I malformation. (A Chiari I malformation is usually not associated with other brain anomalies although spinal cord, skull base and spine lesions are common in this disorder (Osborn 1994).)

DIAGNOSIS

- C5–6 shallow central to lateral disc protrusion causing neck and cervico-shoulder pains.
- Cerebellar tonsil ectopia associated with 'dizziness'.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was told that she had a loss of the normal cervical lordosis with a C5–6 shallow central to lateral disc protrusion with the latter most probably causing most of her bilateral neck and cervico-shoulder pains. She was told that the slight dizziness experienced on looking up may well be due to compression of blood vessels within the foramen magnum (particularly the vertebral arteries) due to the tonsillar ectopia causing a stenotic effect upon soft tissue structures within the foramen magnum during certain neck movements. She was told that she could obtain a surgical opinion regarding the C5–6 disc protrusion but, as she did not have radicular symptoms, she decided not to proceed with a referral. She was advised not to extend her cervical spine as this precipitates the episodes of dizziness. As she was taking considerable doses of non-steroidal anti-inflammatory and analgesic medication, she was offered a trial course of needle acupuncture to see if this would help her neck pain; she agreed to this.

She was very pleased to have had a diagnosis made and she did well with acupuncture treatment apart from one occasion when a locum acupuncturist, instead of giving her acupuncture treatment while she sat in a chair, asked her to lie prone on the examination table with a pillow under her chest; this introduced cervical spine extension and she became very 'dizzy'. She was then asked to sit for a few minutes and the dizziness passed. The acupuncture treatment provided significant relief and enabled her to lessen her medication. (When performing acupuncture in the seated position, ensure that the patient cannot fall off the chair.)

KEY POINTS

1. When a patient feels 'dizzy' on performing certain neck movements, the possibility of arterial compromise should be considered as one likely factor. The probable area of compromise would be of the vertebral arteries.
2. Consider the possibility of a previously asymptomatic Chiari I malformation when a person is involved in a motor vehicle accident (or other neck injury) and experiences neurological symptoms such as vertigo.

Reference

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Further reading

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Case 70

Chiari I malformation

COMMENT

Not only is it imperative for patients to be correctly positioned for imaging but it is imperative that an accurate radiology report be provided so that patients can be treated appropriately. Furthermore, this case highlights how huge unnecessary costs are generated due to inadequate reporting of imaging.

PROFILE

A slim 30-year-old male on a disability pension who smokes approximately 20 cigarettes per day and occasionally drinks alcohol. He was previously very active in athletics until he sustained the injury described below.

PAST MEDICAL HISTORY

He said he had not experienced any unusual illnesses as a child apart from migraines that started at approximately 17 years of age for which he was investigated and told he was a 'hypochondriac'. He said he visited several medical practitioners over a 2-year period regarding his migraines until he found one who diagnosed migraines and treatment was successful in that his migraines 'were cured'. He said he was left with an occasional but different headache, 'like anyone else' if he worked for too long in the heat and did not drink enough fluid.

Two-and-a-half years before consultation, he was in a tree and looking up to enable him to cut off a branch above his head. The branch weighed approximately 20 kg which he then threw backwards; during this activity he felt something 'happen' to his neck with a 'burning' sensation in the neck that became progressively worse. He tried to continue working but developed excruciating neck pain, dizziness and nausea, as well as 'tingling' in his upper and lower limbs. He also felt 'lethargic', so he climbed down the tree and 'stumbled' to his car. However, he had such severe right sided neck pain and neck stiffness that he could not drive, so a friend drove him to a hospital accident and emergency department. He had 'balance' difficulties and could not walk without moving to his right side, causing him to bump into door frames with his right shoulder.

Following a physical examination at the accident and emergency department, plain X-ray films were taken and a carotid duplex scan was performed. The following

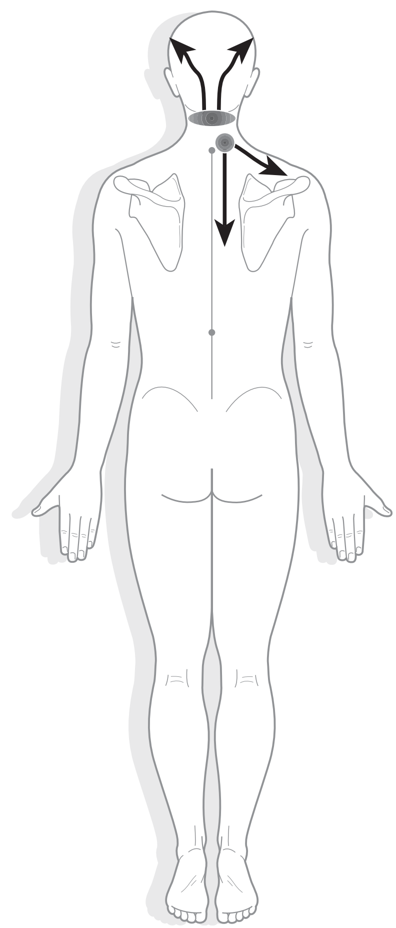

Figure 70.1

day a CT brain scan without contrast was performed. Two days following the injury, MRI and MRA brain scans were performed to determine whether vertebral artery dissection could have occurred. All imaging was reported as being normal, apart from the CT cervical spine report that stated: 'Mild degree of rotation with torticollis deformity'.

As his symptoms worsened, an MRI cervical spine was performed approximately 8 months post injury and this was reported as being 'within normal limits'. Another MRI cervical spine was performed 14 months post injury and this was reported as showing 'no significant abnormality'.

PRESENTING COMPLAINT (Fig. 70.1)

1. Severe suboccipital pain that radiates to the occipital area then to the frontal areas causing a thumping 'migraine' type headache which, in one instance, lasted for approximately 6 weeks.
2. Right sided neck pain that radiates to the right cervico-shoulder region and into the upper thoracic spine between the shoulder blades

He had to take pain killers and rest and, on two occasions, his general medical practitioner sent him to a hospital Pain Clinic for a right sided upper neck facet block injection; these injections provided good but very temporary relief from pain. Physiotherapy had not provided relief.

Medication for his neck pain syndrome now consists of Neurontin (600 mg 3 times per day), morphine (60 mg 3 times per day) and Endep at night. If he cannot sleep, or experiences a severe muscle spasm on the right side of his neck, he takes a 5 mg tablet of Valium and, when necessary, Panadeine Forte (in an attempt to prevent a full blown severe headache). He has tried non-steroidal anti-inflammatory drugs e.g. Voltaren, Brufen, Vioxx, Celebrex etc. but these cause him to 'vomit blood' and he gets blood in the stool as a result of gastric irritation. Medication causes constipation and he has developed a bladder problem in that he has difficulty in starting micturition (sometimes waiting for up to 15 minutes before micturition takes place). He is very frustrated and wants to know why his condition cannot be explained by anyone.

AETIOLOGY

A neck injury 2.5 years ago while cutting a branch off a tree.

EXAMINATION

In the erect posture there was no evidence of pelvic obliquity or of scoliosis. He commented that his slim build was due to a loss of muscle bulk during the period since his injury 2.5 years prior to consultation and that he had lost approximately 12 km in weight. Deep palpation of the paraspinal muscles elicited pain in the C1 to C4 area of his cervical spine (especially on the right) and at approximately the T5 level. Toe walking power and heel walking power were normal. The deep reflexes in the upper and lower extremities were normal. The plantar response was normal as was vibration sensation at the elbows and ankles. Pinprick sensation was patchy in the right upper and lower limbs. There was hyperaesthesia in the right upper limb in the thumb (C6), little finger and 4th finger (C8), the ulnar surface of the forearm (T1) and the medial side of the right arm (T2). Light touch over his face did not suggest any abnormal sensation. The brachial plexus stretch test appeared to be normal. Motor power in the upper and lower extremities appeared to be normal. The temperature of the feet and hands on comparing the left and right sides by palpation appeared to be normal. Hoffman's sign was normal. The Valsalva manoeuvre did not elicit any neck or thoracic spine pain but sneezing caused a sharp pain in his neck.

The circumference of the forearm (10 cm below the elbow) was 24 cm (left) and 25 cm (right) and for the calf (10 cm below the patella) was 33 cm (left) and 32.5 cm (right).

Active cervical spine ranges of movement were measured using a CROM instrument (see Box 70.1).

Active thoracic spine ranges of movement were of full range and painless.

Box 70.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	50°	Elicited 'tightness' on the right side of the neck especially centrally
Extension	60°	**50°**	Elicited 'pinching' at approximately the C5–6 level
Lt lateral bending	45°	45°	Painless
Rt lateral bending	45°	**12°**	Elicited pinching on the right side of approximately the C5–6 level
Lt rotation	80°	**70°**	Painless
Rt rotation	80°	**50°**	He said he could not 'go further' due to 'locking' of his neck

IMAGING REVIEW

The lateral cervical spine view taken on the day of his injury showed a loss of normal cervical spine lordosis in the upper cervical spine (C1–C3 region) where there is a tendency towards a kyphosis of the spine above C4 (Fig. 70.2).

Although the radiologist's Conclusion for the MRI brain study, taken two days following the accident, was 'normal MRI and MRA brain', on review there was in fact an abnormality, i.e. there was a mild projection of the cerebellar tonsil(s) through the foramen magnum, suggesting a small Chiari I malformation (Fig. 70.3).

One of the MRA brain images reported as being normal is shown in Figure 70.4

On reviewing the follow-up cervical spine MRI taken approximately 8 months following his injury, which had been reported as being within normal limits, it was noted that this imaging, unfortunately, did not show the total foramen magnum to best advantage due to suboptimal positioning (Fig. 70.5).

The further follow-up MRI cervical spine taken 14 months following his injury, and which was incorrectly reported as showing '*no significant abnormality*', actually did show the mild cerebellar tonsil ectopia projecting below the foramen magnum (Chiari I malformation) and which measures approximately 6 mm (Fig. 70.6).

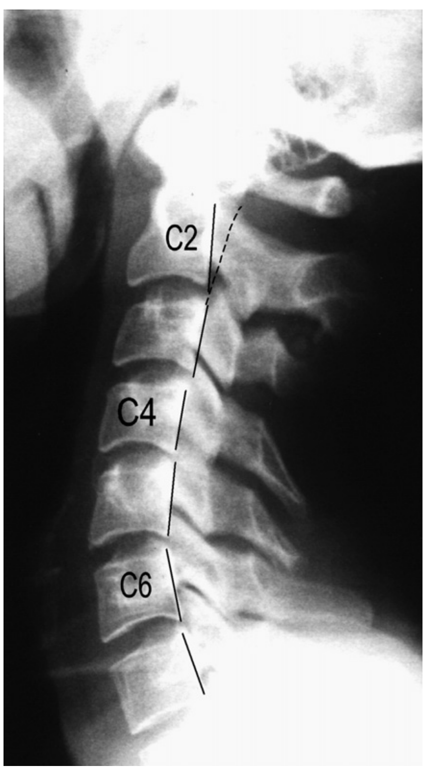

Figure 70.2 Cervical spine lateral plain X-ray image. Note the loss of normal cervical spine lordosis in the upper cervical spine (C1–C3 region) where there is a tendency towards a kyphosis of the spine above C4. The broken line represents what would be a more normal continuation of the lordotic curve in a normal spine.

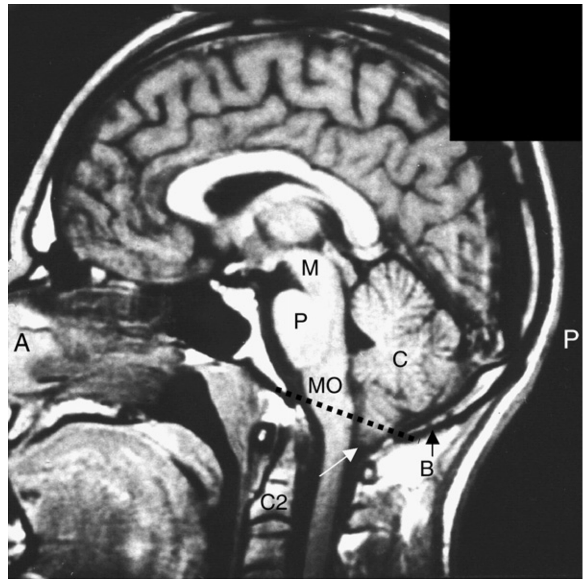

Figure 70.3 Brain MRI sagittal T1-weighted image (approximately centrally through the brain) showing the mild projection of the cerebellar tonsil(s) (cerebellar ectopia) (white arrow) through the foramen magnum, i.e. as a mild Chiari I malformation. This malformation projects below the level of the foramen magnum that is represented by the broken black line across that region. A = anterior; P = posterior; C = cerebellum; B = occipital bone at the back of the skull; C2 = second vertebral body; M = midbrain; MO = medulla oblongata; P = pons. Note how the cerebellum, pons and medulla oblongata are contained within the *posterior cranial fossa* and that the *brain stem* comprises the midbrain, the pons and the medulla oblongata and connects the cerebral hemispheres with the spinal cord.

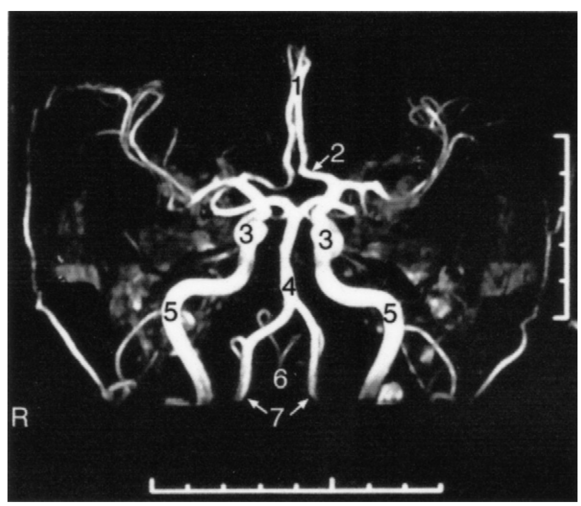

Figure 70.4 One of the MRA brain images reported as being normal shows the following structures: 1 = anterior cerebral artery; 2 = anterior communicating artery; 3 = cavernous portion of the internal carotid artery; 4 = basilar artery; 5 = internal carotid artery; 6 = anterior spinal artery; 7 = vertebral arteries.

Figure 70.5 Cervical spine MRI sagittal T2-weighted image taken approximately 8 months following his injury; the spine, including the cranio-vertebral junction, is positioned too far posteriorly to show the posterior margin of the foramen magnum and its relationship to the cranio-vertebral junction. C2 = second cervical vertebral body; C7 = seventh cervical vertebral body; T1 = first thoracic vertebral body.

CLINICAL IMPRESSION

1. Mild Chiari I malformation.
2. Possible mid-cervical spine soft tissue injury causing radiation into the right cervico-shoulder region and upper interscapular region.

WHAT ACTION SHOULD BE TAKEN?

A current MRI brain was requested as well as an MRI cervical spine.

This MRI brain was correctly reported as showing: 'Mild tonsillar herniation, with cerebellar tonsils extending to approximately 6 mm below the foramen magnum' (Fig. 70.7). The cervical spine MRI report stated: 'At the C5–6 level there is a mild broad-based posterior disc bulge (Fig. 70.8) which is more prominent to the right of the midline which is causing mild indentation of the thecal sac and mild narrowing of the right neural exit foramen' (Fig. 70.9).

DIAGNOSIS

1. A previously *asymptomatic* mild Chiari I malformation that was rendered *symptomatic* by the incident.
2. A posterior and more right sided C5–6 intervertebral disc injury.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

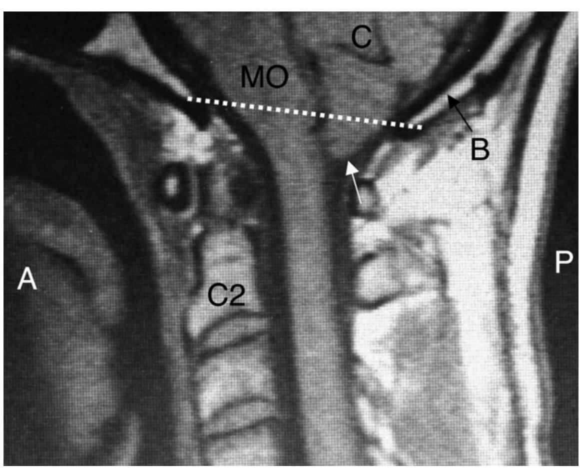

Figure 70.6 Cervical spine MRI sagittal T1-weighted image taken 14 months following his injury that shows the mild (6 mm) projection of the cerebellar tonsil(s) (white arrow) through the foramen magnum that is represented by the broken white almost horizontal line across that region. A = anterior; P = posterior; C = cerebellum; B = occipital bone; C2 = second cervical vertebral body; MO = medulla oblongata.

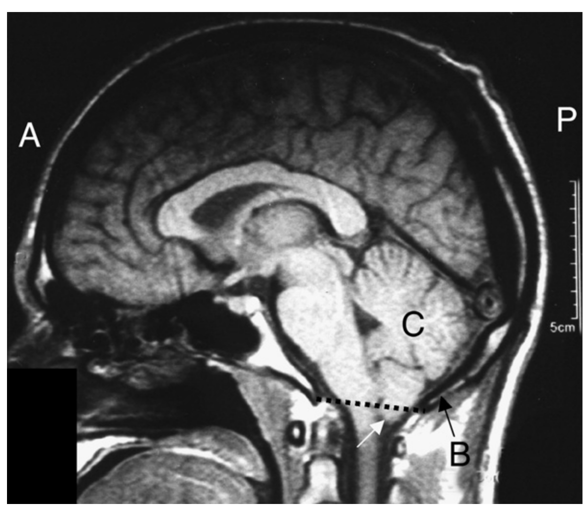

Figure 70.7 Brain MRI sagittal T1-weighted image showing the 'mild tonsillar herniation' (white arrow) that projects approximately 6 mm below the level of the foramen magnum (represented by the dotted line across that region of the skull). A = anterior; P = posterior; C = cerebellum; B = occipital bone.

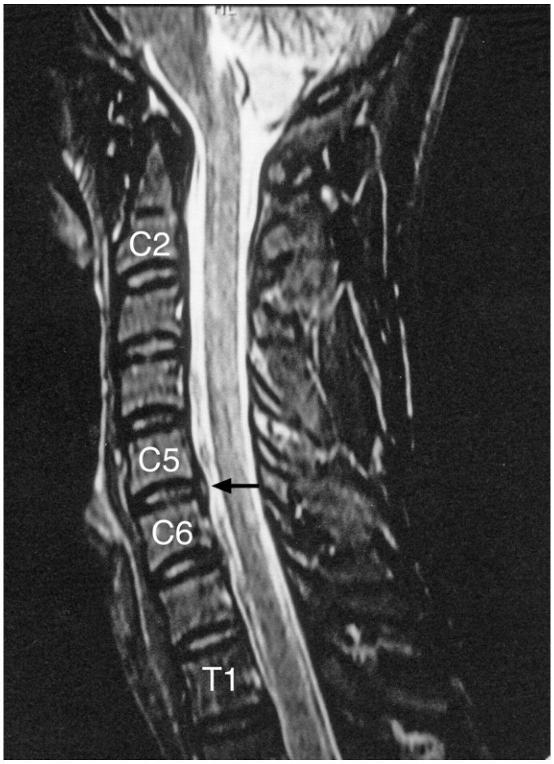

Figure 70.8 Cervical spine MRI parasagittal T2-weighted image. Note the posterior disc bulge at the C5–6 level (arrow). At the same level, an axial image shows the C5–6 posterior disc bulge which is more prominent to the right of the midline (Fig. 70.9).

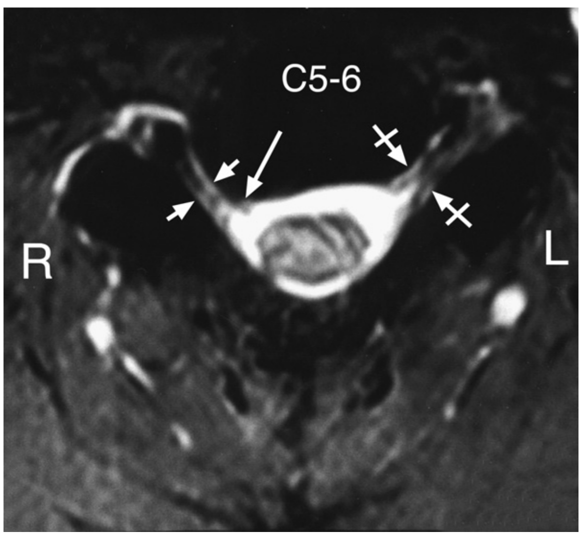

Figure 70.9 Cervical spine MRI axial C5–6 image. Note the posterior disc bulge (white arrow) which is more prominent to the right (R) of the midline causing (i) mild indentation of the dural tube, and (ii) mild narrowing of the right neural exit foramen (two small arrows) which is narrower than the foramen on the left (L) side (two white tailed arrows).

The nature of the C5–6 mild broad based posterior disc bulge, more prominent to the right of the midline causing mild indentation of the dural tube and mild narrowing of the right neural exit foramen, was explained to him. He was advised to perform gentle exercises of his neck in order to retain mobility. He was also advised to sleep with an appropriate pillow and to avoid postures of his neck that could aggravate the C5–6 intervertebral disc lesion.

In particular, the Chiari I malformation was explained to him in great detail. He was referred to a neurosurgeon for an opinion and management for his continuing suboccipital pain syndrome and ataxia. He underwent posterior fossa bony decompression of the foramen magnum for the mild Chiari I malformation. The surgeon found that, at the time of surgery, there was tightness in the cranio-cervical junction. Duraplasty was not performed. A follow-up MRI approximately 8 months after surgery was reported as follows: 'Tonsillar herniation remains unchanged. In the foramen magnum region the cerebellar tonsils are closely apposed to the bony margins. Increased CSF is present posterior to the cerebellar tonsil but there is no evidence of compression around the foramen magnum. The spinal cord appears normal'.

During the postoperative period he experienced good relief from his severe cranio-vertebral junction pain, migraine headaches and ataxia. When he was seen approximately 8 months following surgery he said he had experienced some, but less severe, headaches and virtually no ataxia.

As long as he took the advice provided regarding his right sided neck pain that radiated into the right cervicoshoulder region and into the upper thoracic spine between the shoulder blades, these pains were kept under control. Overall, he was very satisfied with his progress.

On that occasion, he told me that his parents and friends had thought he was a hypochondriac following his injury and that the only person who had believed him had been his partner and that she was the only person that supported him during his 3-year ordeal.

This case provides yet another example of how important it is to listen to the patient, to carefully examine the patient, and to critically evaluate imaging with respect to imaging reports.

Note

1. It is of interest to note that the MRA brain examination did not show any abnormality with the patient lying supine in the magnetic resonance imaging machine. Perhaps an upright MRA examination would have provided a different result when one takes into account the work of Jinkins et al (2003). For example, Jinkins (2007, personal communication) found that in a 45-year-old male with chronic headache and progressive intermittent paroxysmal dizziness only when upright, recumbent MRI showed a Chiari I malformation; however, in the

upright neutral position, MRI showed mobile downward cerebellar tonsil displacement with positional *worsening* of Chiari I malformation. In addition, his patient's episodic dizziness was exacerbated when looking upward, i.e. extending the cervical spine; that posture resulted in positional worsening of the tonsillar displacement with brain stem and 4th ventricle compression.

2. It should be noted that the literature reports that the longer the duration of symptoms prior to surgical decompressive treatment for Chiari I malformation, the greater is the negative impact on patient improvement and the literature stresses the need for early diagnosis and treatment of symptomatic patients (Sakamoto 1999). Therefore, as a period of approximately 2 years and 10 months elapsed before surgery was performed to decompress the posterior fossa soft tissues, this raises concerns regarding the prognosis as Paul et al (1983) found that 32% of their patients with a Chiari I malformation had an associated syringomyelia (a slowly progressive condition in which cavitation occurs within the spinal cord resulting in neurological deficits).

KEY POINT

Be thorough and look for the source of a patient's symptoms even in the face of considerable investigations that are reported as 'normal' – patients, on the whole, do not want to be unwell!

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Case 71

Cervical spine plain X-rays versus MRI

COMMENT

For patients' peace of mind, clinicians should be thorough.

PROFILE

A 39-year-old female who does not smoke or drink alcohol and who works as a nurse.

PAST HISTORY

Approximately 5 years ago she was on night duty when an elderly male patient with dementia assaulted her by knocking her to the ground then repetitively hitting her head with his fists. Colleagues eventually came to her assistance and helped her to her feet.

Next morning she saw her general medical practitioner, was examined and referred for a skull X-ray examination before being prescribed a non-steroidal anti-inflammatory drug, Feldene, and being referred for physiotherapy treatment. She commenced a 10-week course of physiotherapy treatment which initially did not help, so cervical spine plain X-ray films were ordered (without cervical spine functional views) that showed 'early wear and tear'. She then began hydrotherapy and this helped considerably for short periods of time following treatment. She stopped taking Feldene as it caused a 'burning pain in the stomach'; she now intermittently takes an analgesic (Panadeine Forte) and a non-steroidal anti-inflammatory drug (Celebrex; 200 mg capsules approximately 3 times per week).

Because her symptoms have persisted for 5 years, she wants to know what is the underlying cause for her continuing symptoms and she is concerned in case she has a 'nasty' condition developing.

PRESENTING COMPLAINT (Fig. 71.1)

Intermittent bilateral mid-to-lower cervical spine pain that may radiate bilaterally to the occipital area of her head, resulting in headaches since the assault 5 years ago. The neck pain is constant but varies in intensity and sometimes has a 'stabbing' and 'burning' quality. She cannot sleep well due to the neck pain and the neck is stiff and painful of a morning.

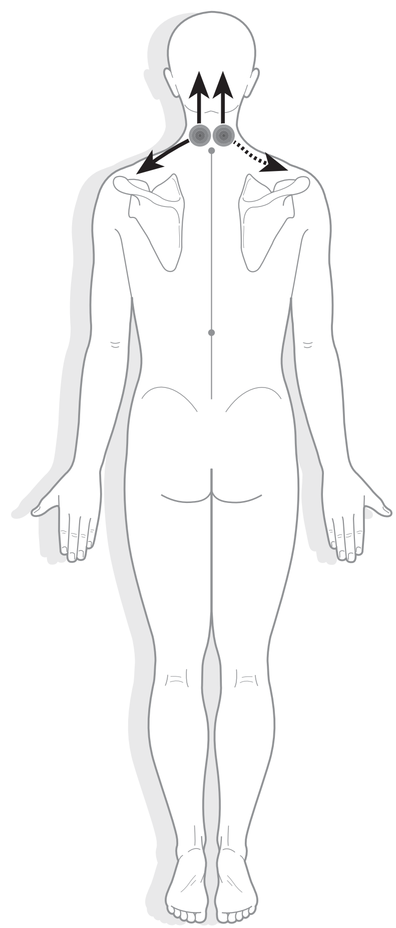


Figure 71.1

She often experiences a 'burning' pain radiating from the cervical spine to the left cervico-shoulder region and very occasionally to the right cervico-shoulder region. She experiences occasional 'dizzy' spells that can be present without a headache and she cannot associate the dizziness with any particular activity.

AETIOLOGY

An assault approximately 5 years ago.

EXAMINATION

Deep palpation of the cervical spine elicited tenderness bilaterally at the suboccipital and mid-to-lower cervical spine regions. The deep reflexes in the arms and legs were normal. The plantar response was normal. Pinprick sensation of the upper extremities and cervico-shoulder regions was normal. Cervical spine traction was painless but compression immediately aggravated the neck pain, especially in the lower cervical to T2 level. Power in the upper and lower limbs was normal, as was the sensation of vibration at the elbows and ankles. There was no evidence of a cervical bruit. The blood pressure in the right arm was 120/80 in the seated position.

Active cervical spine ranges of movement were measured using a CROM instrument (see Box 71.1).

Box 71.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	50°	Elicited bilateral lower cervical spine pain
Extension	60°	–	This was not performed as she has 'found out by experience that this aggravates the neck pain'
Lt lateral bending	45°	41°	Elicited a feeling of 'tightness' on the right of the cervical spine
Rt lateral bending	45°	45°	Painless
Lt rotation	80°	12°	Elicited pain in the right cervico-shoulder region
Rt rotation	80°	12°	Elicited pain in the left cervico-shoulder region

IMAGING REVIEW

A plain X-ray examination of the skull was reported as being normal.

Plain X-ray films of the cervical spine taken 8 days following the assault reported: 'The C5–6 and C6–7 disc spaces show moderate narrowing with minor narrowing of the intervertebral foramina due to early osteophyte formation being present, indicating pre-existing changes' (Fig. 71.2). In addition, it should be noted that there is some thinning and minor retrolisthesis at the C4–5 level and that there is a loss of the normal cervical spine contour.

CLINICAL IMPRESSION

Based on history and imaging, the patient had asymptomatic changes in her cervical spine that had now become symptomatic.

The intermittent 'dizzy' spells may be due to irritation of the sympathetic nervous system in the neck region during the assault, as injury to the cervical spine has been correlated with dizziness (Jackson 1977).

Figure 71.2 Cervical spine lateral plain X-ray image. Note (i) the loss of lordosis, (ii) minor retrolisthesis of C4 on C5 with some disc space height thinning with essentially no anterior lipping of the vertebral body margins, and (iii) the moderate narrowing of the disc space height at the C5–6 and C6–7 levels with very minor lipping anteriorly on either side of the C6–7 intervertebral disc space.

WHAT ACTION SHOULD BE TAKEN?

An MRI was ordered to fully investigate her cervical spine and this was reported as showing: 'Disc protrusion at C4–5 and C5–6 levels with a central disc bulge at C6–7 (Fig. 71.3). There is resultant spinal canal stenosis at the C4–5 level (Fig. 71.4). Narrowing of the neural exit foraminae is seen at C4–5 and C6–7 levels on the left'.

DIAGNOSIS

- C4–5 and C5–6 intervertebral disc posterior protrusions with a C6–7 posterior bulge with left C5 radiculopathy.
- Headaches of vertebrogenic origin.

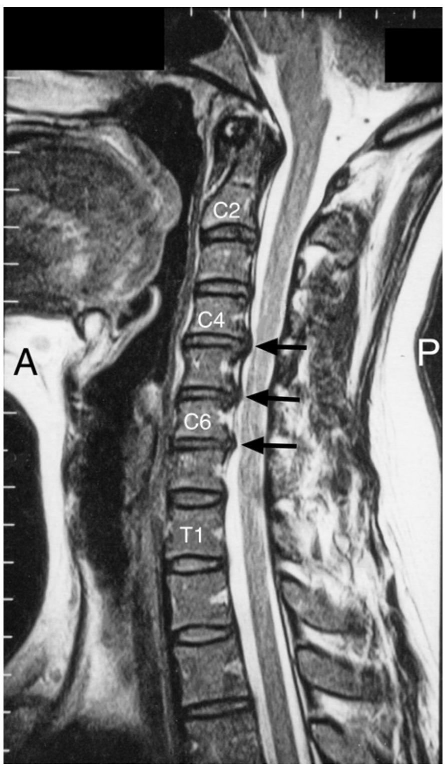


Figure 71.3 Cervical spine MRI parasagittal T2-weighted image. Note the disc protrusion at the C4–5 and C5–6 levels with a bulge at the C6–7 level, impinging on the anterior surface of the dural tube (black arrows). The C3-4 and C4–5 intervertebral discs still retain a reasonable degree of grey colour as compared to the C2-3 and C6–7 discs that are darker and indicate greater desiccation.

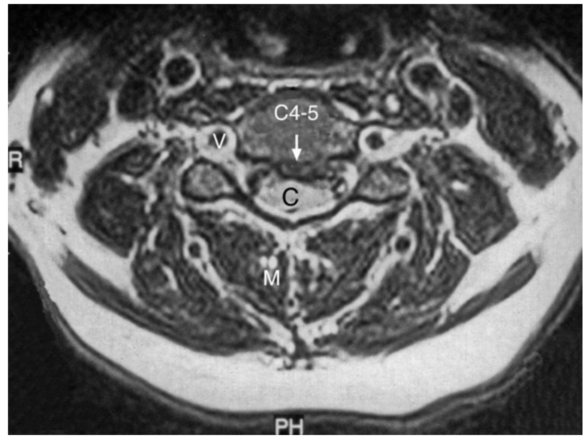


Figure 71.4 Cervical spine MRI axial T2-weighted image through the C4–5 disc. Note the large posterolateral disc protrusion (white arrow) that is central to slightly left paracentral and which impinges on the anterior dural tube and displaces the cord (C) posteriorly. M = multifidus muscle; V = vertebral artery.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was told that she may well require a C4–5 anterior decompression (Cloward procedure) in due course in view of her large posterior and slightly left paracentral disc protrusion at that level that impinges upon the anterior part of the dural tube and displaces the cord posteriorly. She was advised to see a neurosurgeon; however, as she now performed only light duties in her nursing employment and was coping with her considerable C4–5 disc protrusion, she decided not to proceed with a neurosurgical consultation at the time. As a nurse she was aware that there is no guarantee that surgery would be successful, so she decided to put up with her condition and continue with physiotherapy, hydrotherapy and her medicinal regime now that she understood the cause of her symptoms.

KEY POINT

It is important to carefully investigate patient's spinal pain complaints so that they feel a thorough approach has been taken in an attempt to understand their spinal pain syndrome.

Reference

Jackson R 1977 The cervical syndrome, 4th edn. Springfield IL, Charles C Thomas.

Case 72

Cervical and thoracic spine injuries resulting in biomechanical disturbances and pain

COMMENT

Patients with spinal injuries should not routinely be told that their symptoms will resolve spontaneously within 6 weeks of the injury.

PROFILE

A 35-year-old slim housewife who smokes approximately 25 cigarettes per day and drinks alcohol very rarely.

PAST MEDICAL HISTORY

Four years ago she fell from the second step of a ladder 'flat onto her back' and hit the back of her head on the carpeted floor, knocking herself unconscious. She remembers coming to with severe suboccipital and occipital pains and feeling very dazed. Next morning she awoke with considerable neck pain, a severe generalized headache that lasted for 2 days, and right forearm paraesthesiae. She sought a medical opinion, at which time analgesic medication was prescribed.

PRESENTING COMPLAINT (Fig. 72.1)

1. Constant bilateral neck pain that radiates to the occipital region causing severe headaches. There is some radiation to the jaw bilaterally. The neck pain radiates to the cervico-shoulder regions bilaterally, especially on the left side. She sometimes experiences 'numbness, coldness and pins and needles in the right forearm and tingling in all the fingers'. The severity of symptoms is activity related and she experiences 'ups and downs' in her symptoms. Coughing increases the neck and cervico-shoulder pains bilaterally.
2. Mid-thoracic spine pain that radiates towards the scapula on each side; it is activity related, with increased activity causing an increase in pain.

AETIOLOGY

Falling off a ladder 4 years ago.

EXAMINATION

In the erect posture there appeared to be minor pelvic obliquity but no significant scoliosis. Percussion of the spine elicited pain at the T2 and T7 levels. Deep palpation of the paraspinal muscles elicited pain bilaterally at the C2–3, C5–6 and T7 levels. The deep reflexes in the upper and lower extremities were normal. The plantar response

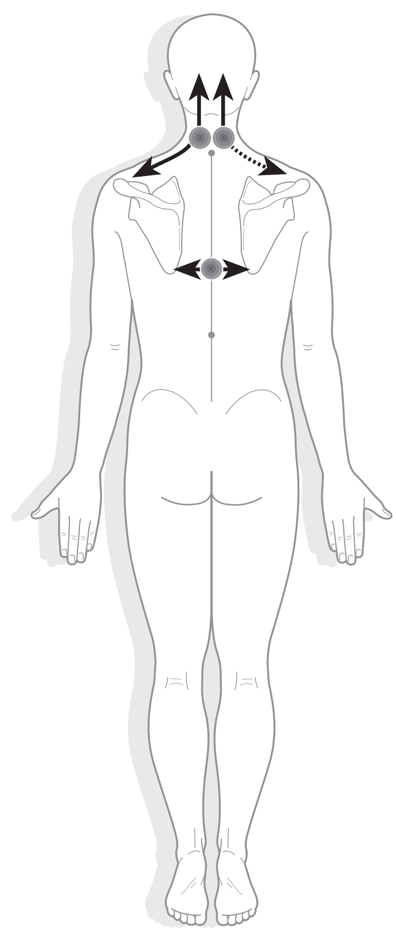


Figure 72.1

Box 72.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comment
Flexion	50°	28°	Elicited pain on the left side of C7–T1.
Extension	60°	60°	Painless.
Lt lateral bending	45°	40°	Elicited pain on the right side of C4 to C7.
Rt lateral bending	45°	30°	Elicited pain on the left side of C4 to C7.
Lt rotation	80°	80°	Painless.
Rt rotation	80°	80°	Painless.

was normal. Pinprick sensation over the upper and lower extremities and the back of the torso was normal, apart from hypoaesthesia in the left cervico-shoulder region, i.e. the left C5 dermatome. Vibration sensation at the elbows and ankles was normal. The brachial nerve stretch test did not elicit any pain. The Valsalva manoeuvre elicited an increase in pain at approximately the T7 level that radiated to the left and right sides. The blood pressure was 130/84 in the right arm (seated). Motor power in the upper and lower limbs was normal.

The circumference of the forearm, 10 cm below the elbow, was 25 cm (left) and 25.5 cm (right), i.e. within normal limits.

Active cervical spine ranges of movement were measured using a CROM instrument (see Box 72.1).

Cervical spine traction and compression did not elicit any neck pain. Compression of the shoulders did not cause any spinal pains.

Active thoracic spine ranges of movement were of approximately full range and painless apart from left rotation that was limited by approximately 10% due to pain on the left side of the mid-thoracic spine. Compression of the thoracic cage did not elicit any joint pain.

In the supine position, cervical spine flexion elicited pain from approximately the C4 to the C7 levels.

IMAGING REVIEW

No imaging had been performed.

CLINICAL IMPRESSION

Possible cervical and thoracic spine soft tissue injuries.

WHAT ACTION SHOULD BE TAKEN?

As no imaging had been performed since her accident 4 years previously, cervical spine plain X-rays were requested, including flexion and extension functional views. Thoracic spine plain films were also requested. The cervical spine X-ray report stated: 'The vertebral bodies and discs spaces appear intact. No fracture or subluxation on flexion and extension views. The atlantodental interval is normal'. The thoracic spine X-ray report stated: 'No fracture or subluxation'.

On reviewing the imaging the following points were noted.

Cervical spine flexion (Fig. 72.2). There is some angulation at the C5 level where there is a loss of the normal cervical spine contour and alignment on forward bending with some anterolisthesis of C4 on C5. The C5 body postero-inferior margin shows an early osteophyte developing. The anterior disc space height at the C5–6 level is less than it is at adjacent disc levels.

Cervical spine extension. This view was not taken in the true lateral projection.

Cervical spine oblique views showed early uncovertebral joint osteoarthrosis formation at the left C5–6 intervertebral foramen level.

Thoracic spine (Fig. 72.3). The lateral view shows some early osteophytes anteriorly adjacent to the discs at approximately the T4–8 levels.

In view of the angulation at the C5 level and a positive Valsalva manoeuvre causing pain at approximately the T7 level, an MRI cervical and thoracic spine was requested. The MRI report stated the following.

Cervical spine: 'Minor reduction in signal intensity of the cervical disc spaces in keeping with some disc desiccation (Fig 72.4). Minor diffuse posterior bulging of the C5–6 disc that does not cause any significant canal stenosis. Both C6 neural foramina appear satisfactory'. However, it should be noted that the C5–6 disc bulge does touch, and indent, the pain sensitive anterior part of the dural tube (Fig. 72.4).

Thoracic spine: 'There are Schmorl's nodes involving the vertebral inferior end-plates at T7 and T12' (Fig. 72.5A and B).

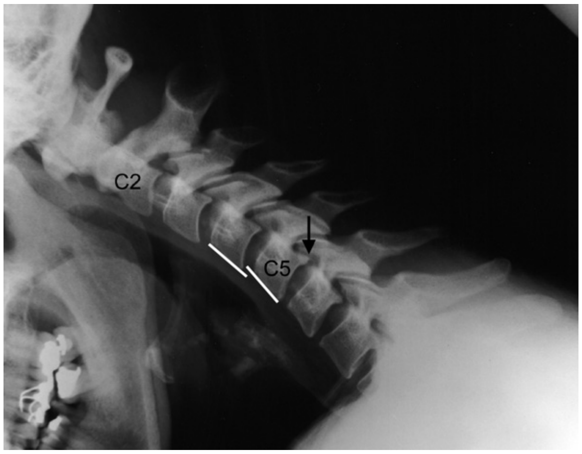


Figure 72.2 Cervical spine flexion plain X-ray image. There is some angulation at the C4–5 level where there is a loss of the normal cervical spine contour and alignment on forward bending with some anterolisthesis of C4 on C5 (measured as approximately 3 mm) suggesting a degree of instability at this level. The C5 body postero-inferior margin shows an early osteophyte developing (arrow). The anterior disc space height at the C5–6 level is less than it is at adjacent disc levels, indicating disc thinning (subsequently supported by the bulging disc at C5–6 on MRI).

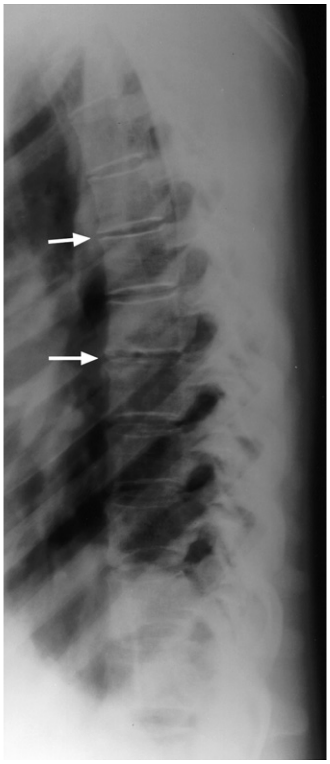


Figure 72.3 Thoracic spine lateral plain X-ray image. This shows some early osteophytes anteriorly adjacent to the discs at approximately the T4–7 levels.

Figure 72.4 Cervical spine MRI sagittal T2-weighted image showing minor desiccation ('blackening') of discs particularly at the C2–3, C3–4 and C5–6 levels, with minor diffuse posterior bulging of the C5–6 disc (arrow). However, the discs are not yet totally 'black' indicating that the MRI films taken 4 years following her fall, reflect early degenerative changes.

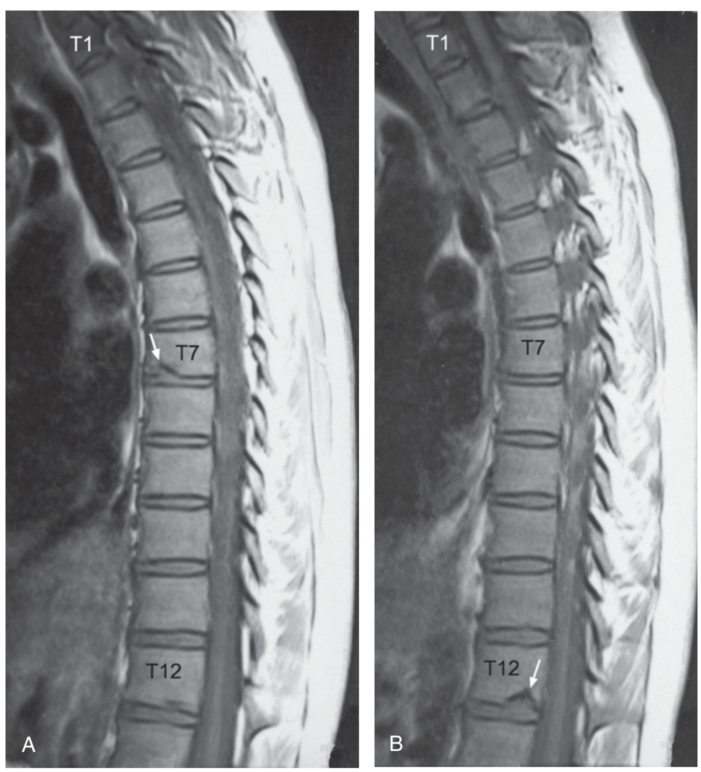


Figure 72.5 (A) Thoracic spine MRI parasagittal T1-weighted image showing a T7 vertebral body inferior endplate fracture (slice = 4.5 mm thick). (B) Thoracic spine MRI sagittal T1-weighted image showing a T12 inferior endplate fracture (Schmorl's node) (slice = 4.5 mm thick).

DIAGNOSIS

Cervical spine

Musculoligamentous soft tissue injuries including a diffuse posterior bulge of the C5–6 disc.

Thoracic spine

1. Musculoligamentous soft tissue injuries.
2. T7 and T12 inferior endplate fractures.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. The plain films and MRI films were explained to her in detail as she had been told that there was no reason why her soft tissue injuries should have caused symptoms to last for 4 years. She now understood that there was an organic reason for her condition as it had previously been intimated that she had experienced 'soft tissue injuries that should have resolved spontaneously within 6 weeks of the injury' and that she should have recovered. Three gentle spinal mobilizations of the cervical and thoracic spines provided considerable relief, so she was advised to perform neck exercises (cervical spine flexion with left and right rotation within a comfortable range of movement) and deep breathing exercises. This approach was very helpful to her.

KEY POINTS

There is no scientific rationale on which to make the statement that 'soft tissue injuries should resolve spontaneously within 6 weeks of the injury'. Teasell (1997) has noted: 'There has long been a misconception that all injuries should heal after 6 weeks. However, clinical experience and follow-up studies clearly demonstrate that not all patients necessarily get better and that there is a significant subset who continue to suffer with chronic symptoms'.

Reference

Teasell R W 1997 The denial of chronic pain. Pain Research Management 2(2): 89–91.

Case 73

C3 lamina fracture

COMMENT

The importance of clearly defining and explaining a patient's injury or injuries should not be underestimated as it is a very important part of treatment.

PROFILE

A 40-year-old male of average build who does not smoke cigarettes and does not drink alcohol who has a manual occupation.

PAST HISTORY

Approximately 7 years ago he had been in a motor vehicle accident in which he experienced a hyperextension injury to his cervical spine. He went to the local hospital where plain X-ray images were taken. The report stated that the lateral view showed a problem at the C3–4 level, so cervical spine flexion and extension functional views were taken. A cervical spine CT scan was then performed approximately 10 days later and this showed a 'fracture' in his neck. He had also had an MR brain and an MR cerebral angiogram and the reports for both investigations stated 'Normal examination'. A follow-up CT cervical spine scan was performed approximately 6 years after the accident.

PRESENTING COMPLAINT(S) (Fig. 73.1)

Chronic mid-cervical spine pain that can radiate up to the occiput causing headaches with some bilateral cervical spine muscle tightness and aching extending into the left and right cervico-shoulder regions following the MVA. The long standing chronic neck pain syndrome had resulted in him becoming depressed, so he had been referred by his general medical practitioner to a psychiatrist who prescribed anti-depressant medication; the patient responded very well to this treatment. However, his psychiatrist felt that this man's main problem was that he had not received a detailed explanation of his injuries and, therefore, could not understand why his symptoms continued, leading the patient to wonder whether he could become 'paralysed due to his broken neck'. His psychiatrist referred him for a cervical spine assessment and report, with a particular request that his patient's condition should be explained, in detail, to the patient with emphasis on the likely long-term prognosis.

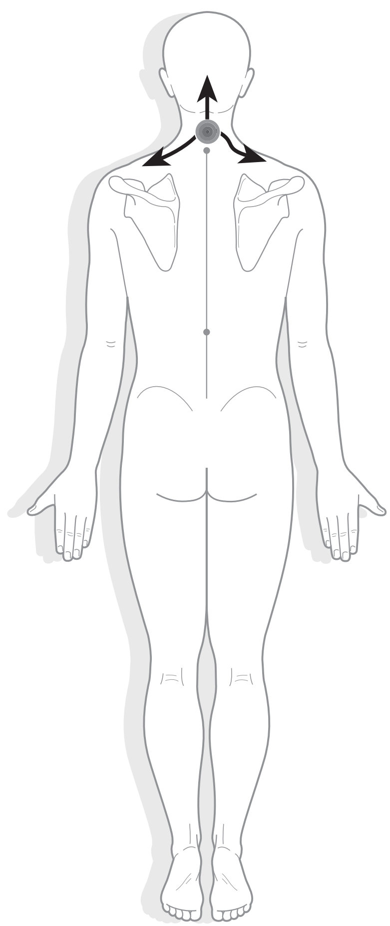


Figure 73.1

AETIOLOGY

A motor vehicle accident approximately 7 years ago.

EXAMINATION

The deep reflexes in the upper and lower extremities were normal. Pinprick sensation in the upper and lower extremities was normal. Deep palpation over the mid-cervical spine elicited a degree of bilateral muscle spasm with some tenderness in the upper regions of the trapezius muscles in both cervico-shoulder regions. Vibration sensation at the elbows and ankles was normal. The Valsalva manoeuvre did not elicit any pain. Power in the upper and lower extremities was normal. There was no wasting of the muscles in the shoulder girdles. Cervical spine active ranges of movement were measured as being slightly limited in all directions with a complaint of slight pain at the extreme of each movement. The blood pressure in the right arm was 120/80 in the seated position. Cervical spine traction provided some relief but compression of the cervical spine caused a slight aggravation of his symptoms.

IMAGING REVIEW

1. The original plain X-ray films taken approximately 7 years ago, i.e. following his motor vehicle accident, showed that there was slight anterior shift of C3 on C4 (Fig. 73.2). However, no mention was made of the apparent loss of height of the C3 articular pillars.
2. Subsequent functional X-ray films showed 'no significant movement of C3 on C4 on flexion' (Fig. 73.3) 'although there was a fixed forward slip of C3 on C4' measuring approximately 4 mm. On extension (Fig. 73.4), no significant movement of C3 on C4 was noted but retrolisthesis of C4 on C5 occurred.
3. The original CT cervical spine CT scan showed: 'There is a fracture of both laminae of C3 with a little displacement and fracture of the left pedicle of C4. Forward slip of C3 on C4 is more obvious than on the plain film radiographs. There is deformity of the left lateral mass of C3 with associated complete ankylosis of the facet joints at this level, an appearance that could be secondary to old healed trauma'. Subluxation is noted of the left C4–5 facet joint with 'possible dislocation of the left C2–3 facet joint.'
4. A follow-up cervical spine CT scan performed approximately 6 years after the accident showed the old healed fracture of the C3 laminae (Fig. 73.5) with deformity of the left lateral mass of C3.

CLINICAL IMPRESSION

Bilateral fractures of the C3 laminae with possible compression fractures of the C3 articular pillars.

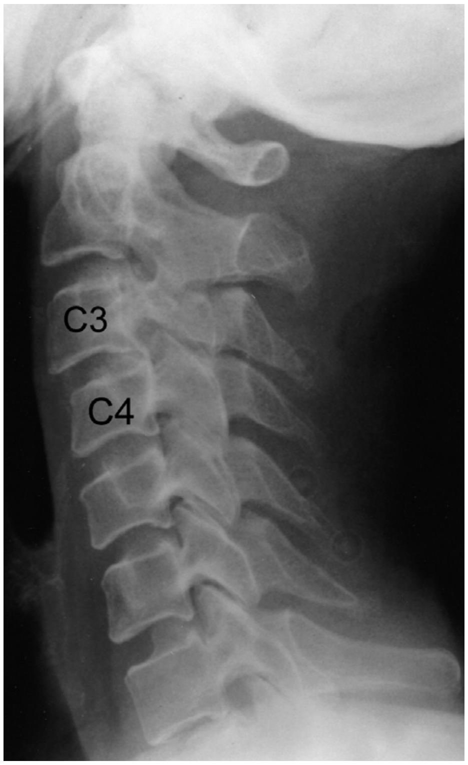


Figure 73.2 Cervical spine lateral plain X-ray image taken soon after the motor vehicle accident. Note the loss of normal cervical spine contour and the fixed anterolisthesis of C3 on C4, measuring approximately 4 mm. In addition, the height of the C3 articular pillars appeared to be decreased.

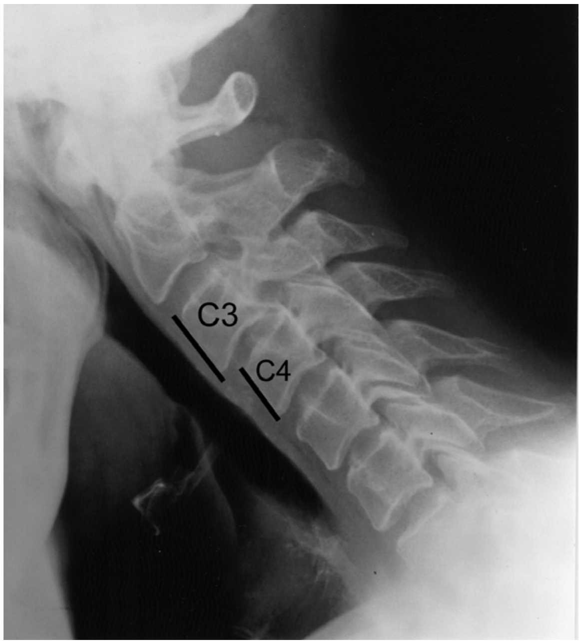


Figure 73.3 A subsequent cervical spine flexion plain X-ray image showed a loss of normal cervical spine contour and fixed anterolisthesis of C3 on C4, measuring approximately 4 mm, as shown by the two lines.

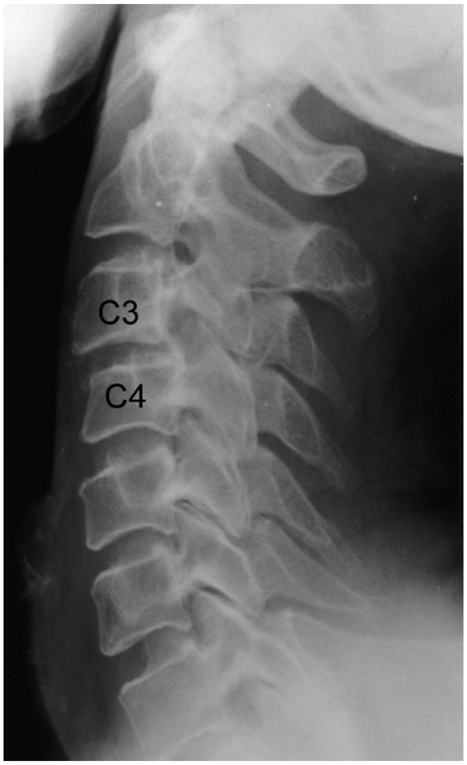


Figure 73.4 The cervical spine extension plain X-ray image showed no significant movement of C3 on C4 but showed that retrolisthesis of C4 on C5 occurred.

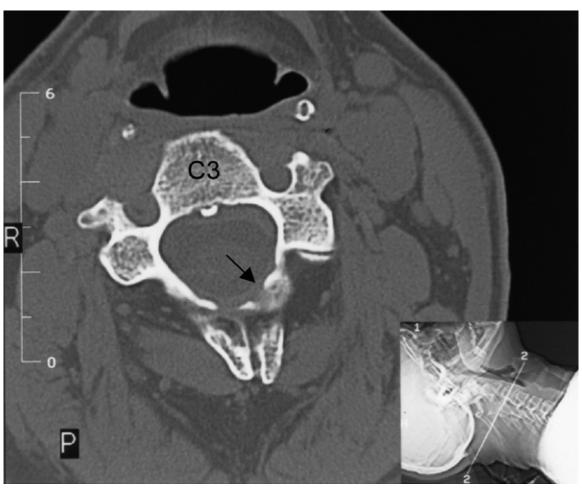


Figure 73.5 Cervical spine CT image at the C3 level. Note the healed fractures of both laminae of C3. The arrow shows the region of the left fracture site. There is some deformity of the left lateral mass of C3.

WHAT ACTION SHOULD BE TAKEN?

No further imaging was considered necessary.

DIAGNOSIS

Fracture of both C3 laminae and the left pedicle of C4 with associated soft tissue injuries.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

He was told that the injuries he had sustained would result in chronic neck pain with associated muscle tension, headaches and cervico-shoulder aching. He was told that he was very fortunate that his spinal canal had not been compressed and that there was definitely no risk of him ending up in a wheelchair. He was told that it would be best to leave well alone and he responded saying that, now that he understood the reason for his chronic pain, he would get on with life and be careful not to further injure his neck.

He was referred back to his psychiatrist with a report detailing what action had been taken and that his patient had been assured that he would not end up in a wheelchair.

Note

Although the C3 fracture would have been unstable initially, there was little displacement of the fracture fragments. Therefore, although this fracture could have led to further complications it did not appear to have done so. However, had it not been fully investigated, manipulation of the cervical spine potentially could have had disastrous consequences.

KEY POINTS

An early detailed explanation of the injuries to the patient may well have prevented the onset of psychiatric sequelae and the considerable associated costs. Had his psychiatrist not taken the course of action that he did, to reassure the patient, ongoing psychological anxiety may well have led to worsening psychiatric sequelae.

Case 74

C5 facet fracture

COMMENT

Individuals thrown out of a moving vehicle are at risk of sustaining serious bony injuries apart from soft tissue injuries.

PROFILE

A 19-year-old male of muscular build who does not smoke cigarettes but drinks alcohol and is a manual worker.

PAST HISTORY

One year ago he was asleep in the back of a truck when the driver apparently lost control and the vehicle hit a tree. As he was asleep, he does not recall the accident but he was told that he had landed approximately 5 metres away, at which time he was knocked unconscious. An ambulance arrived at the scene and put him on a back-board, having first put a supportive cervical spine collar onto his neck. He was transported to the hospital's accident and emergency department where he was thoroughly investigated as he had neck pain and mid-thoracic spine pain. Cervical spine plain X-ray and CT scan images were performed. He also underwent MRI and MRA brain studies for which the radiology report concluded: 'Normal MRI and MRA of the brain'. A chest examination was reported as being normal.

Approximately 2 months following the motor vehicle accident, he underwent internal fixation at the C5–6 level to stabilize that part of his neck.

PRESENTING COMPLAINT(S) (Fig. 74.1)

1. Intermittent lower central neck pain with restriction in ranges of movement, for example when looking back while reversing his motor vehicle. The pain is activity related and increased activity tends to aggravate his neck symptoms. He occasionally experiences occipital headaches that he believes begin in his neck when it is painful. There are no radicular symptoms.
2. Intermittent centrally located mid-thoracic spine aches of varying intensity. There are no radicular symptoms and there is no night pain per se.

AETIOLOGY

A motor vehicle accident 1 year ago.

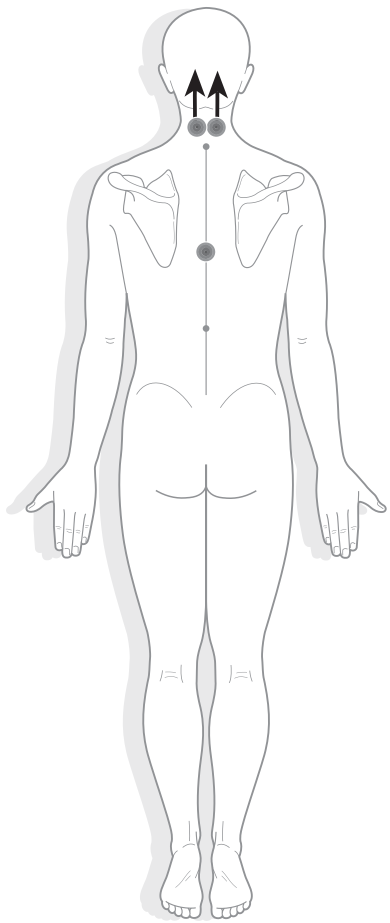


Figure 74.1

EXAMINATION

In the erect posture, there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic and lumbar spines did not elicit any pain nor did deep palpation of the paraspinal muscles in the cervical, thoracic or lumbar regions. The deep reflexes in the upper and lower extremities were normal. Vibration sensation at the elbows and ankles was normal. Pinprick sensation over the torso posteriorly was normal, as it was for the upper and lower extremities. Motor power in the upper and lower extremities was normal. Hoffman's sign was normal. The circumference of the forearm (10 cm below the elbow) was 29 cm (left) and 28.5 cm (right).

Active cervical spine ranges of movement were measured using a CROM instrument (see Box 74.1).

Box 74.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comment
Flexion	50°	40°	Painless
Extension	60°	48°	Painless
Lt lateral bending	45°	30°	Painless
Rt lateral bending	45°	30°	Painless
Lt rotation	80°	62	Painless
Rt rotation	80°	62°	Painless

Other cervical spine tests to check for cervical spine pain were as follows: left rotation with extension, right rotation with extension, cervical spine traction, and cervical spine compression; all these were painless. Trapezius muscle trigger point pressure elicited localized right trigger point pain.

All active thoracic spine ranges of movement were full and painless as was compression of the rib cage.

IMAGING REVIEW

All imaging was reviewed and some findings are shown below.

Cervical spine plain X-ray images: Note that the lateral cervical spine view showed subluxation of the facets at the C5–6 level (Fig. 74.2).

CT cervical spine scan: This showed a fracture through the right inferior articular process and facet of C5 (Fig. 74.3).

A cervical spine lateral plain X-ray taken following his discectomy and internal fixation at the C5–6 level (Fig. 74.4) also showed thinning of the C6–7 disc height and the small avulsion fracture.

Thoracic spine plain X-ray images: The lateral view showed fractures affecting the superior endplate of the T6 and T7 vertebral bodies (Fig. 74.5).

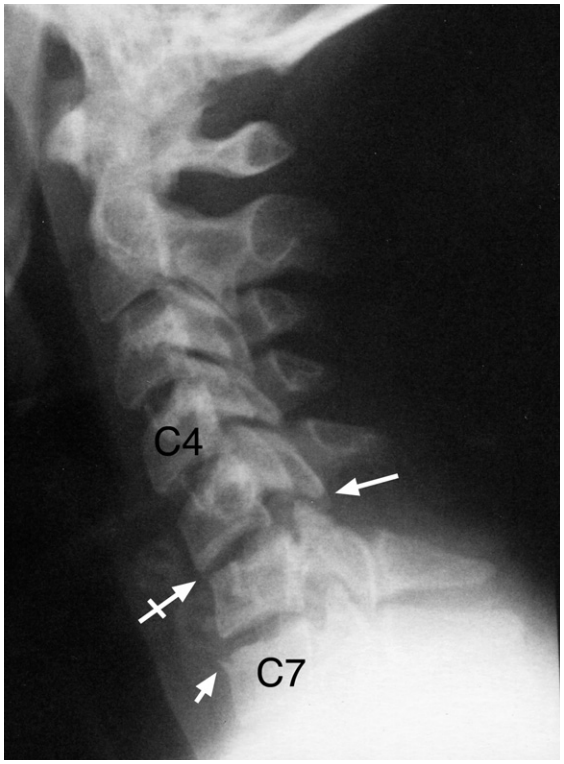


Figure 74.2 Cervical spine neutral lateral plain X-ray image. There is an angulation at the C5–6 level with loss of the normal cervical spine contour. In particular, note the subluxation of the C5–6 facets (long white arrow) with severe anterior wedging of the C5–6 disc that is narrower anteriorly (tailed white arrow). In addition, there was a degree of anterolisthesis of the C5 body on C6 and a small avulsion fracture of the anterosuperior margin of the C7 vertebral body (short white arrow).

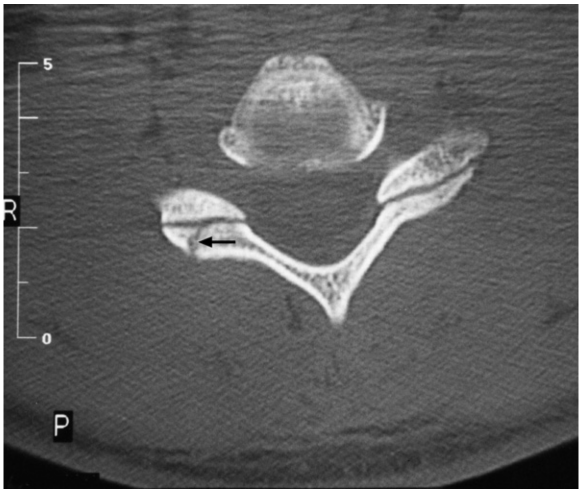


Figure 74.3 Cervical spine CT axial image. Note the fracture (black arrow) extending through the right inferior articular process and facet of C5 on the right.

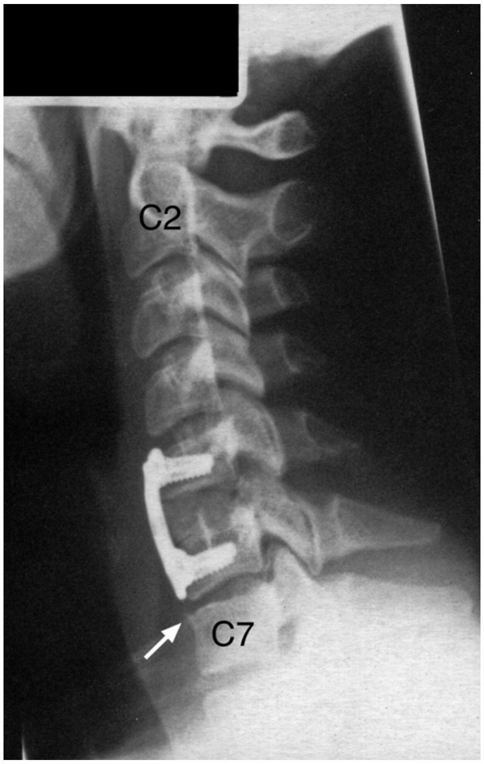


Figure 74.4 Lateral cervical spine image. Note the internal fixation at the C5–6 level. Also note the C6–7 loss of disc space height and the small C7 avulsion fracture (white arrow).

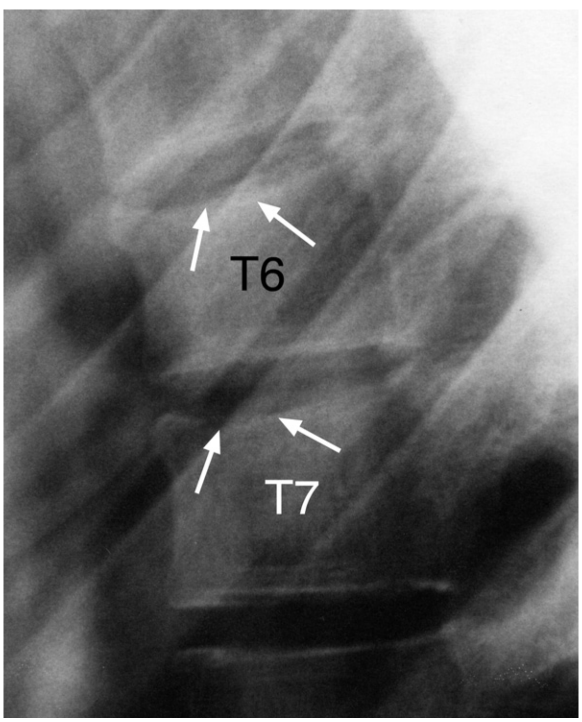


Figure 74.5 Lateral thoracic spine image. Note the fractures of the superior endplates of the T6 and T7 vertebral bodies, respectively (white arrows).

CLINICAL IMPRESSION

Residual symptoms due to (i) cervical spine injuries, and (ii) mid-thoracic spine injuries.

WHAT ACTION SHOULD BE TAKEN?

Time was spent with the patient to fully discuss his case. He was advised that further imaging was not necessary at this time in view of his intermittent lower central neck pain, occasional occipital headaches and intermittent central mid-thoracic spine ache.

DIAGNOSIS

Cervical spine

1. Musculoligamentous soft tissue injuries causing occasional occipital headaches.
2. C5–6 and C6–7 intervertebral disc injuries.

Thoracic spine

1. Musculoligamentous soft tissue injuries.
2. T6 and T7 vertebral body superior endplate fractures.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. In addition, he was told that fusion at the C5–6 level naturally led to restriction in some ranges of movement and that he should not be concerned about this. The possibility of joint degenerative change on each side of the C5–6 level fusion, particularly in view of the C6–7 disc height loss and associated small avulsion fracture of the C7 body, were explained to him but he was advised not to be worried about this as he could have had worse injuries with greater symptoms.

His cervical spine symptoms were not bad enough that he required medication, apart from an occasional flare up of neck pain that settled with paracetamol. He was advised not to work above his head or to take part in activities that may jar his neck. Regarding his mid-thoracic spine pain, two manipulations of the mid-thoracic spine provided him with complete relief from this pain.

Note

This case highlights the necessity for a thorough investigation of individuals subjected to motor vehicle accidents.

KEY POINT

Although the thoraco-lumbar junction is the commonest site for spinal fractures (El-Khoury & Brandser 1997), other levels of the spine are vulnerable to fractures.

Reference

El-Khoury G Y, Brandser E A 1997 Radiography of spinal disorders. In: Frymoyer J W (Editor-in-Chief) The adult spine, principles and practice, 2nd edn. Lippincott-Raven, Philadelphia, p 413–442.

Further reading

Rabb C H, Lopez J, Beauchamp K et al 2007 Unilateral cervical facet fractures with subluxation: injury patterns and treatment. J Spinal Disord Tech 20: 416–422.

Case 75

Fractures of the C6 and C7 spinous processes and of the C7 inferior articular processes

COMMENT

Look for abnormal anatomy when reading imaging films.

PROFILE

A 59-year-old female supervisor of average build who does not smoke cigarettes and only drinks alcohol socially.

PAST HISTORY

Three-and-a-half months ago she was the seat-belted driver of a vehicle when another vehicle, travelling at speed from a side street, cut across the road and hit her vehicle causing it to roll over. She ended up being suspended by her seat belt until someone released it to enable her to crawl out of her vehicle. Because she could not straighten her very painful cervical and upper thoracic spine, she crawled to the side of the road and lay down on her side while waiting for an ambulance. The ambulance crew provided her with a solid neck brace and transported her to the hospital where an X-ray showed a fracture of the C6 and C7 spinous processes.

She was given morphine then analgesic tablets while in hospital where she was kept for a couple of days for observation.

PRESENTING COMPLAINT(S) (Fig. 75.1)

1. Constant bilateral upper and lower cervical spine pains, especially on the right side of her neck. since a motor vehicle accident 3.5 months ago. The pain radiates to the occipital area and causes severe occipital headaches. The pain also radiates to both cervico-shoulder regions. On awakening in the morning she experiences considerable upper cervical spine pain, particularly in the sub-occipital area, even though she wears a neck brace at night to protect her neck and also uses a contoured pillow for support. There is no night pain per se but she does experience aching in the neck and in the cervico-shoulder regions. An increase in activity causes an increase in symptoms. She has noticed that there are restrictions on some movements of her neck; for example, on left and right rotation and on lateral bending to the left and right sides. The symptoms are aggravated by bending her neck forwards for long periods of time, sneezing, working and being in bed overnight. The above

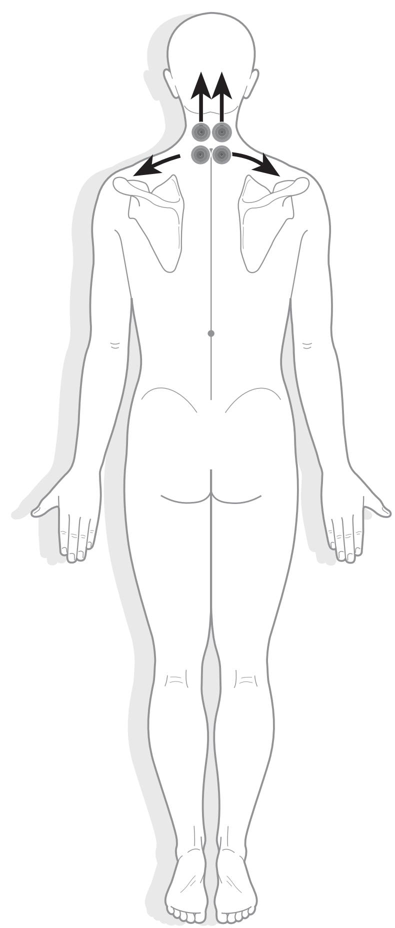


Figure 75.1

symptoms are temporarily relieved by a hot shower, using a hot water bottle behind her neck, bed rest for a couple of hours and Panadol.

2. Anxiety caused by having to constantly protect her neck.

She had not experienced any of the above symptoms before the motor vehicle accident occurred.

Her current treatment consists of Panadol and physiotherapy.

AETIOLOGY

A motor vehicle accident 3.5 months ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of any scoliosis. Percussion of the thoracic spine did not elicit any pain. Deep palpation of the cervical paraspinal muscles elicited pain on the right side of the suboccipital area and pain bilaterally at approximately the C3–4 and C6–T1 levels. Muscle spasm was palpable in the cervical and upper thoracic spine regions. Deep palpation of the thoracic spine elicited pain centrally and on the right extending from T1 to T4. The deep reflexes in the upper and lower extremities were normal. Vibration sensation at the elbows was normal. Pinprick sensation over the back of the upper torso and over the upper limbs appeared to be normal. The left brachial plexus stretch test appeared to cause slight pain. Motor power appeared to be normal in the upper extremities apart from finger adduction between the 3rd and 4th fingers of the left hand (C8, T1). Hoffman's sign did not suggest any upper motor neuron lesion.

The circumference of the forearm (10 cm below the elbow) was 24.5 cm (left) and 24 cm (right). The blood pressure in the right arm, while seated, was 130/92, i.e. somewhat elevated. The Valsalva manoeuvre did not elicit any neck or thoracic spine pain.

Active cervical spine ranges of movement were measured using the CROM instrument (see Box 75.1).

Other cervical spine and cervico-shoulder region tests to check for pain of cervical spine origin were performed as shown in Box 75.2.

Box 75.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	38°	Elicited pain at the C6 and C7 spinous process area
Extension	60°	39°	Elicited pain at the C6 and C7 spinous process area
Lt lateral bending	45°	18°	Elicited pain on the right side of the neck posteriorly
Rt lateral bending	45°	17°	Elicited pain on the right side of the neck posteriorly
Lt rotation	80°	48°	Elicited pain on the right side of the lower neck
Rt rotation	80°	58°	Elicited slight pain on the right side of the lower neck

Note that there was restriction of all movements.

Box 75.2 Other cervical spine and cervico-shoulder region tests

	Patient's comments
Lt rotation plus extension	Elicited pain at the cervico-thoracic and approximately T4–5 levels
Rt rotation plus extension	Elicited pain at the cervico-thoracic region
Cervical spine traction	Painless
Cervical spine compression	Painless
Downward shoulder pressure	Painless
Trapezius trigger point pressure	Elicited slight pain on the left side and greater pain on the right

IMAGING REVIEW

Plain X-ray of the sternum and thoracic spine taken a short time following the accident. The report stated that there may be a fracture of the lower body of the sternum.

Plain X-ray of the cervical spine and chest taken a short time following the accident. The reported stated: 'Fractured spinous process of C6 and C7 with moderate displacement'.

CT cervical spine taken a short time following the accident. The axial study for the cervical spine was from the C3 to the T1 levels. Figure 75.2 shows the fractured spinous processes of the C6 and the C7 vertebrae, with moderate displacement.

However, in addition, there were unreported fractures of the inferior articular processes at the C7 level on the right (Fig. 75.3) and left (Fig. 75.4) sides.

Figure 75.2 Cervical spine CT sagittal image with slices at 2 mm. Note the fractured spinous process of the C6 and the C7 vertebrae (arrows) with moderate displacement.

Figure 75.3 Cervical spine CT right parasagittal image with slices at 2 mm. Note the fracture of the inferior articular process at the C7 level (arrow) on the right.

CLINICAL IMPRESSION

Fractures of the C6 and C7 spinous processes and inferior articular process fractures of C7 on the left and right sides.

WHAT ACTION SHOULD BE TAKEN?

A cervical spine lateral view and flexion and extension functional view X-rays were requested and were reported as follows: 'There is disruption at the base of the spinous process of the C6 and C7 vertebrae consistent with fractures' (Fig. 75.5A). The enlargement of the lower cervical spine (Fig. 75.5B) clearly shows the extent of detachment of the spinous processes that have fractured off the respective vertebrae at the C6 and C7 levels.

There is a loss of the normal cervical spine contour on flexion (Fig. 75.6) with the spine being almost straight and, above the C4 level, there is maintenance of the lordotic curve whereas the curve should reverse in normal cervical spine flexion. There is a degree of anterolisthesis

Figure 75.4 Cervical spine CT left parasagittal image with slices at 2 mm. Note the fracture of the inferior articular process at the C7 level (arrow) on the left.

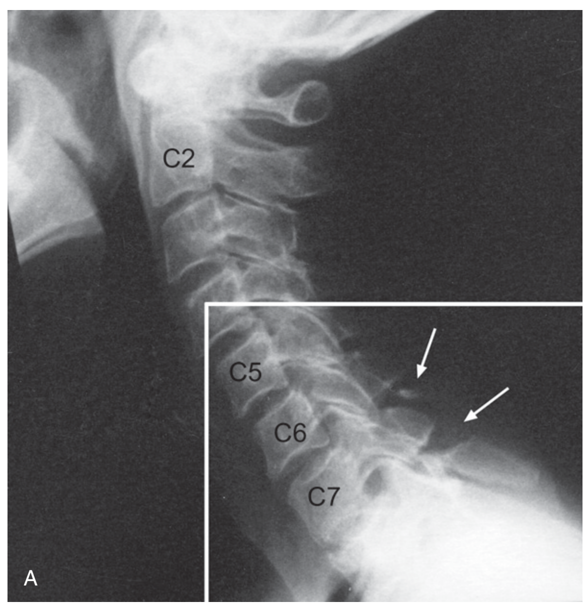


Figure 75.5 (A) Cervical spine lateral plain X-ray image. Note (i) loss of the normal cervical spine contour, and (ii) the fractures of the C6 and C7 spinous processes (white arrows). The area below the white line is shown enlarged in (B).

(Continued)

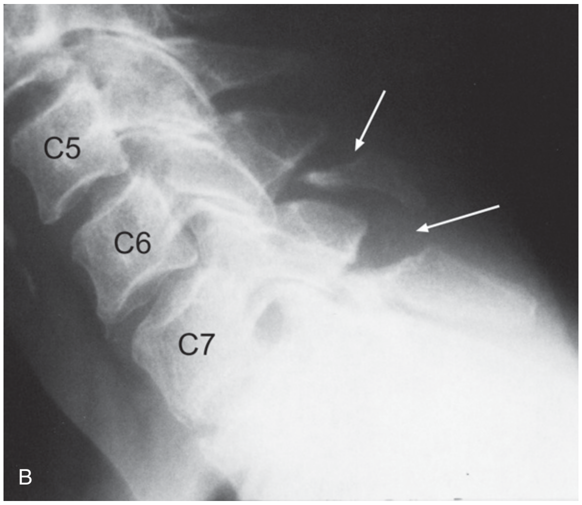


Figure 75.5 Cont'd (B) Enlargement of the lower cervical spine shown in Figure 75.5A. Note the extent of detachment of the spinous processes that have fractured off the respective vertebrae at the C6 and C7 levels (arrows).

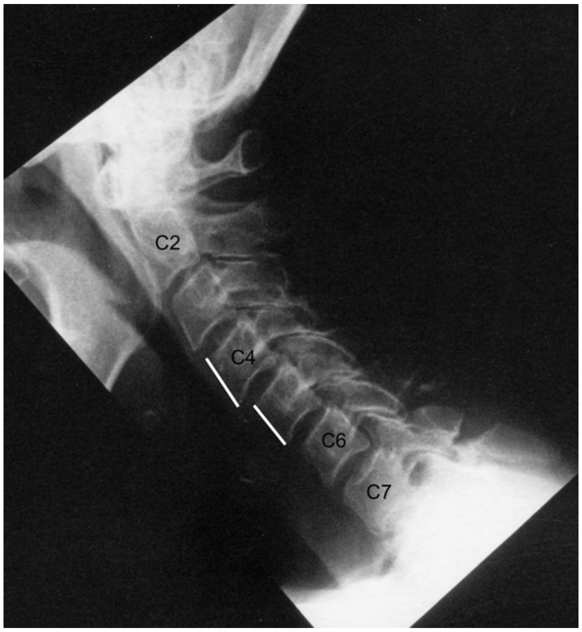


Figure 75.6 Cervical spine flexion plain X-ray image. Note (i) there is a loss of the normal cervical spine contour on flexion, and (ii) there is a degree of anterolisthesis of C4 on C5 (measured at approximately 3.2 mm) indicating instability at this level on forward flexion.

of C4 on C5 (measured at approximately 3.2 mm) indicating instability at this level on forward flexion.

DIAGNOSIS

- Fractures of the spinous process of the C6 and C7 vertebrae.
- Inferior articular process fractures of C7 on the left and right sides.
- Musculoligamentous soft tissue injuries of the cervical spine.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was advised that she should avoid heavy lifting and activities requiring cervical spine extension, e.g. having her hair washed by the hairdresser. She was advised to perform gentle mobilizing neck exercises (cervical spine flexion with left and right rotation within a comfortable range of movement) and deep breathing exercises. She was advised to consider having an MRI study of the cervical and thoracic spines should her symptoms deteriorate, with which she agreed.

KEY POINT

If a clinician 'gets back to basics' i.e. interprets imaging while keeping anatomy in mind, the results of imaging can be more productive.

Case 76

Cervico–thoracic junction and upper thoracic spine aches

COMMENT

Frequently patients merely require reassurance that their symptoms are not associated with an underlying life-threatening condition.

PROFILE

A 30-year-old solidly built male manual worker who does not smoke cigarettes or drink alcohol.

PAST HISTORY

Three-and-a-half years ago he was the seat-belted driver of another car when a car, coming in the opposite direction, crossed onto his side of the road. When he swerved to avoid a head-on collision he lost control of his vehicle and hit a lamp-post. He became unconscious for approximately three-quarters of an hour. A passer-by telephoned the ambulance and the police and he was taken by ambulance to hospital where X-rays were taken and showed that he had fractured the 4th and 5th thoracic vertebrae and some ribs in the right mid-axillary line. He had a right sided haemopneumothorax that required drainage. He was put into the intensive care unit for 2 days then kept in hospital for 5 days before being given a neck-to-pelvis brace and allowed to gradually mobilize before being discharged home. He used the brace for approximately 4 months 'on and off'.

He now wants a re-evaluation of his condition so that he may understand why he gets cervico-thoracic junction and upper thoracic spine aches.

PRESENTING COMPLAINT(S) (Fig. 76.1)

1. Intermittent cervico-thoracic junction aches. Coughing and sneezing cause minor aggravation of his symptoms. He finds that he cannot turn his head as far to the left side as he can to the right. His symptoms do not awaken him at a particular time at night.
2. Intermittent upper thoracic spine aches that may rarely awaken him at night but not at a particular time. Increased activity and coughing or sneezing cause an increase in this ache. Sometimes he experiences discomfort in the upper-thoracic spine that can radiate around the rib cage to the sternum causing him concern in case these symptoms are due to a heart attack. Looking up when hanging up the laundry, or doing housework aggravates his symptoms.

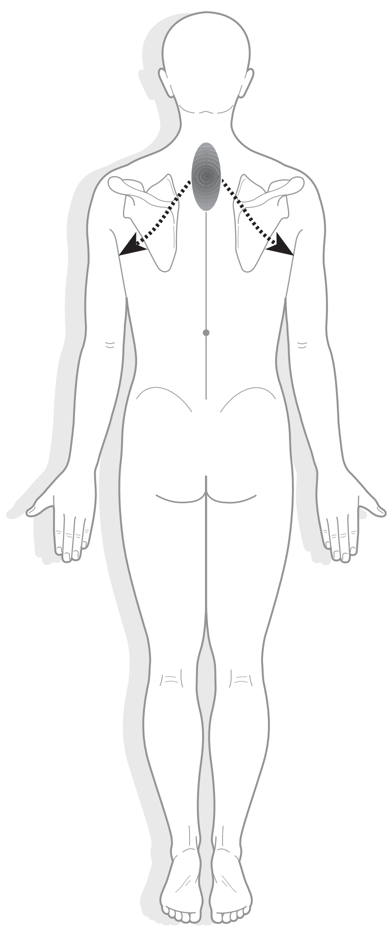


Figure 76.1

He said he had not experienced the above symptoms before being involved in a motor vehicle accident 3.5 years ago.

AETIOLOGY

Motor vehicle accident 3.5 years ago.

EXAMINATION

There was no clinical evidence of pelvic obliquity in the erect posture. There was a minor scoliosis in the thoracic spine. He had a degree of kyphosis in the upper thoracic spine. The left posterior aspect of his thorax was more prominent than the right and he had minor winging of the right scapula (C5–7). Percussion of the thoracic and lumbar spines elicited pain from approximately T4 to T7. Deep palpation of the paraspinal muscles elicited significant pain bilaterally at C4–7 and at T4–6 levels in the thoracic spine.

Toe walking power (S1) and heel walking power (L5) were normal. The deep tendon reflexes in the upper and lower extremities were normal, as was the case with the heel to shin test, rapid hand movements and the finger to nose test. Vibration sensation at the elbows and ankles was normal. Pinprick sensation over the upper and lower extremities, and the back of his torso, did not elicit any sensory disturbance. The plantar response was normal.

Power in the upper and lower extremities appeared to be normal apart from adduction (T1) of the left little finger that was weaker at 4/5.

The circumference for the forearm (8 cm below the elbow) was 28.5 cm bilaterally. The blood pressure was 120/80 in the right arm in the seated position. The cranial nerves appeared to be normal.

In the supine position, cervical spine flexion caused pain in his lower neck and in the upper thoracic spine.

Active cervical spine ranges of movement were measured using a CROM instrument (see Box 76.1).

Active thoracic spine ranges of movement were slightly limited apart from extension that was limited by approximately 25%.

Box 76.1 Cervical spine active ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	45°	Elicited cervico-thoracic junction discomfort
Extension	60°	54°	Elicited cervico-thoracic junction discomfort
Lt lateral bending	45°	40°	Elicited cervico-thoracic junction discomfort
Rt lateral bending	45°	40°	Elicited cervico-thoracic junction discomfort
Lt rotation	80°	56°	Elicited cervico-thoracic junction discomfort
Rt rotation	80°	72°	Elicited cervico-thoracic junction discomfort

IMAGING REVIEW

Plain X-rays of the thoracic and cervical spines were reviewed.

Cervical spine

The report stated 'degenerative changes in the cervical spine are limited to the anterior margins of the C4 and C5 vertebral bodies at the C4–5 disc body junction' (Fig. 76.2A). However, the fractures of the spinous processes of the C7 and T1 vertebrae were not mentioned (Fig. 76.2A and B).

Thoracic spine

These images were reported as showing vertebral body 'crush fractures of T4 and T5 with loss of vertebral body height' (Fig. 76.3).

CLINICAL IMPRESSION

Chronic neck and upper thoracic spine pain syndromes due to bone fractures and soft tissue injuries.

WHAT ACTION SHOULD BE TAKEN?

An MRI investigation of his cervical and thoracic spines was considered but a mutual decision was reached not to proceed at this time in view of his relatively minor symptoms.

DIAGNOSIS

Cervical spine

- Musculoligametous soft tissue injuries.
- C7 and T1 spinous process fractures.

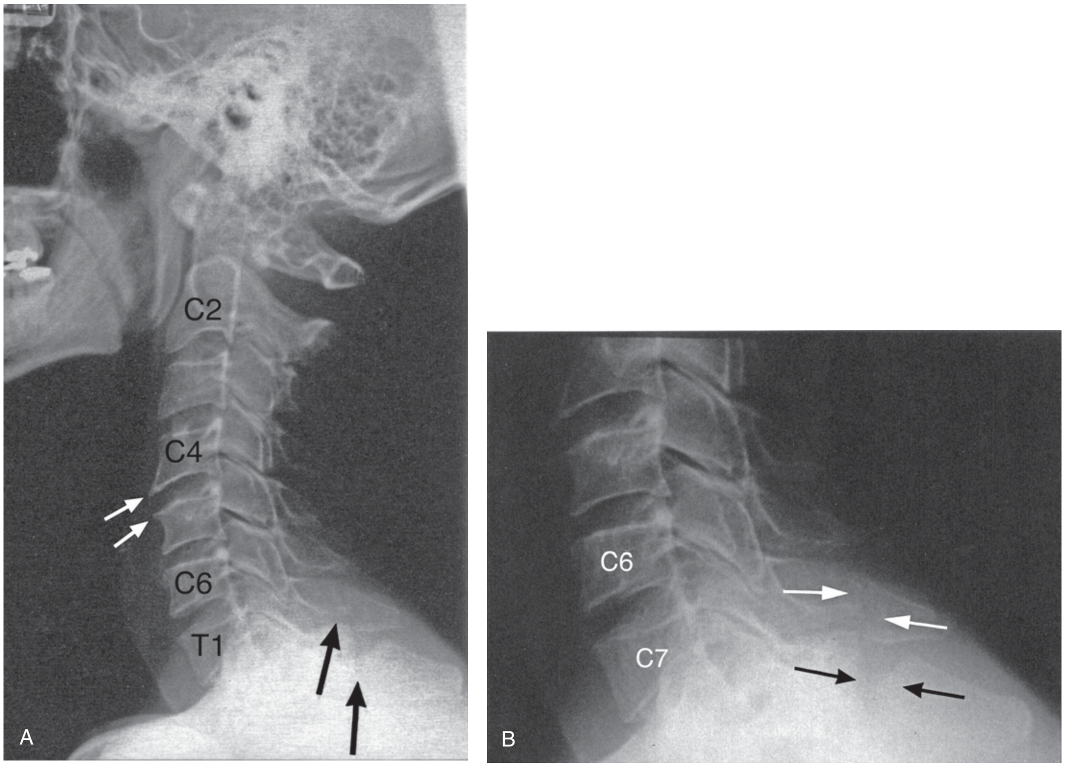


Figure 76.2 (A) Lateral cervical spine plain X-ray image. Note (i) degenerative changes in the cervical spine limited to the anterior margins of the C4 and C5 vertebral bodies at the C4–5 disc body junction (white arrows), and (ii) the C7 and T1 spinous process fractures (black arrows) with significant separation between the fractured fragments and the remaining spinous processes. (B) Enlargement of the C7 and T1 spinous process fractures (arrows) seen in Figure 76.2A.

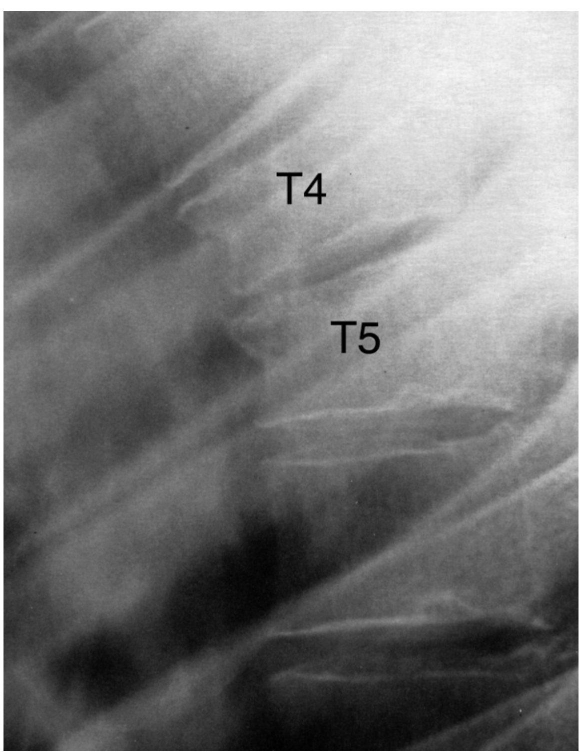


Figure 76.3 Thoracic spine lateral plain X-ray image. Note the vertebral body crush fractures of T4 and T5 with loss of vertebral body height.

Thoracic spine

- Musculoligamentous soft tissue injuries.
- T4 and T5 vertebral body crush fractures.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was reassured that, although his bony and soft tissue injuries cause some discomfort, they are not life-threatening. He was given the option of having active treatment or of first simply performing home exercises (cervical spine flexion with left and right rotation within a comfortable range of movement). He opted for the latter and was given exercises to strengthen his neck and upper thoracic spine muscles. He was advised on how to sleep using a pillow of appropriate height and he was advised not to perform activities that aggravate his symptoms. He was asked to return again should he require active treatment but he made good progress with his home exercise programme.

KEY POINTS

1. This case illustrates how it is necessary to thoroughly investigate patients that have been involved in a motor vehicle accident as, although his T4 and T5 thoracic vertebral body and some right rib fractures were demonstrated radiologically, the C7 and T1 spinous process fractures were overlooked.
2. Patients frequently need reassurance that their condition has been taken seriously and that no sinister underlying life-threatening pathology has been overlooked.
3. Not only do spinal physical injuries have to be thoroughly investigated but patients' psychological wellbeing has to be cared for when treating patients as a whole person.

Case 77

Osseous anomalies

COMMENT

Beware of minor symptoms.

PROFILE

A 24-year-old male of average build.

PAST HISTORY

Nothing contributory.

PRESENTING COMPLAINT(S) (Fig. 77.1)

'Intermittent discomfort' in the left posterior neck muscles for approximately 5 years with no radiation of symptoms. The discomfort is aggravated by bicycle riding. There is no night pain per se. He had physiotherapy treatment some years ago but did not find it helpful.

AETIOLOGY

Unknown.

EXAMINATION

There was no clinical evidence of pelvic obliquity or of scoliosis apart from a slight cervical spine scoliosis that was left convex. There was minor bilateral tenderness on deep palpation of the posterior muscles of the neck. Active ranges of movement were unremarkable. The deep tendon reflexes were normal in the upper and lower extremities as was the case with pinprick sensation and vibration sensation. The plantar response was normal. There was no abnormality in the circumference measurements of the upper and lower extremities. The blood pressure was 120/80 in the right upper limb in the seated posture.

IMAGING REVIEW

A plain X-ray examination taken 1 month ago showed a left convex cervical spine scoliosis (Fig. 77.2), C3 and C5 hemivertebrae (Figs 77.2 and 77.3) and various segmentation anomalies from C2 to C6 with fusion at some levels.

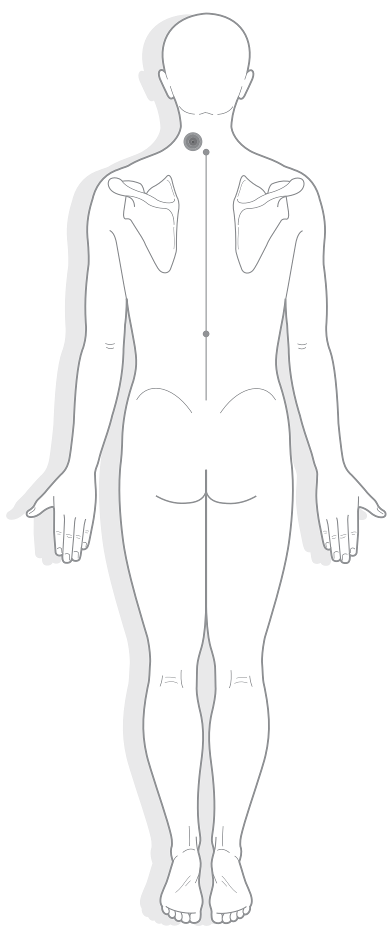


Figure 77.1

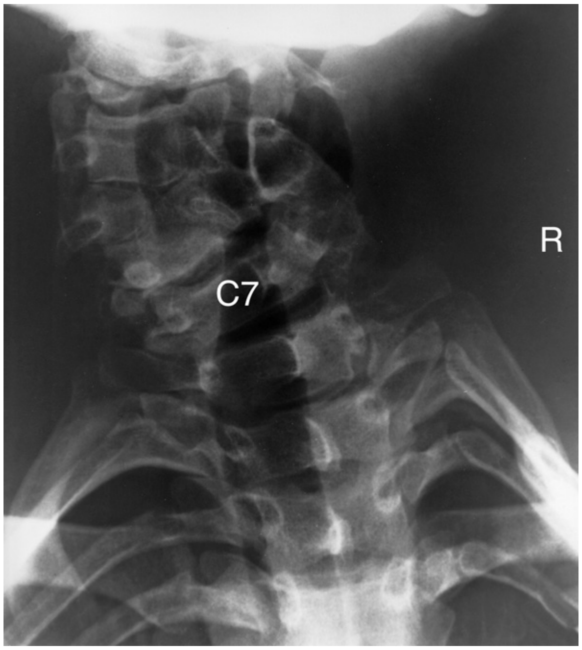


Figure 77.2 Cervical spine anteroposterior plain X-ray image viewed from behind. Note the left convex cervical spine scoliosis with some hemivertebrae.

CLINICAL IMPRESSION

Neck discomfort of mechanical origin due to his various anomalies.

WHAT ACTION SHOULD BE TAKEN?

A cervical spine MRI study was then requested to better visualize the anomalies and the spinal canal. This showed the osseous anomalies in greater detail (Fig. 77.4) and that there was an 'adequate' spinal canal with a 'normal spinal cord' (Fig. 77.5).

DIAGNOSIS

Cervical spine osseous anomalies resulting in neck discomfort of mechanical and postural origin.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised to 'leave well alone' as he only experienced 'discomfort' in his neck. He was advised to sit erect when riding his bicycle and not to ride a racing type bicycle with low handlebars. He was advised on appropriate ergonomics for his daily activities.

Note

This case clearly illustrates how even minor symptoms should be thoroughly investigated in order to make a diagnosis, *particularly* when mechanical intervention such as mobilization or manipulation may be considered as a possible option.

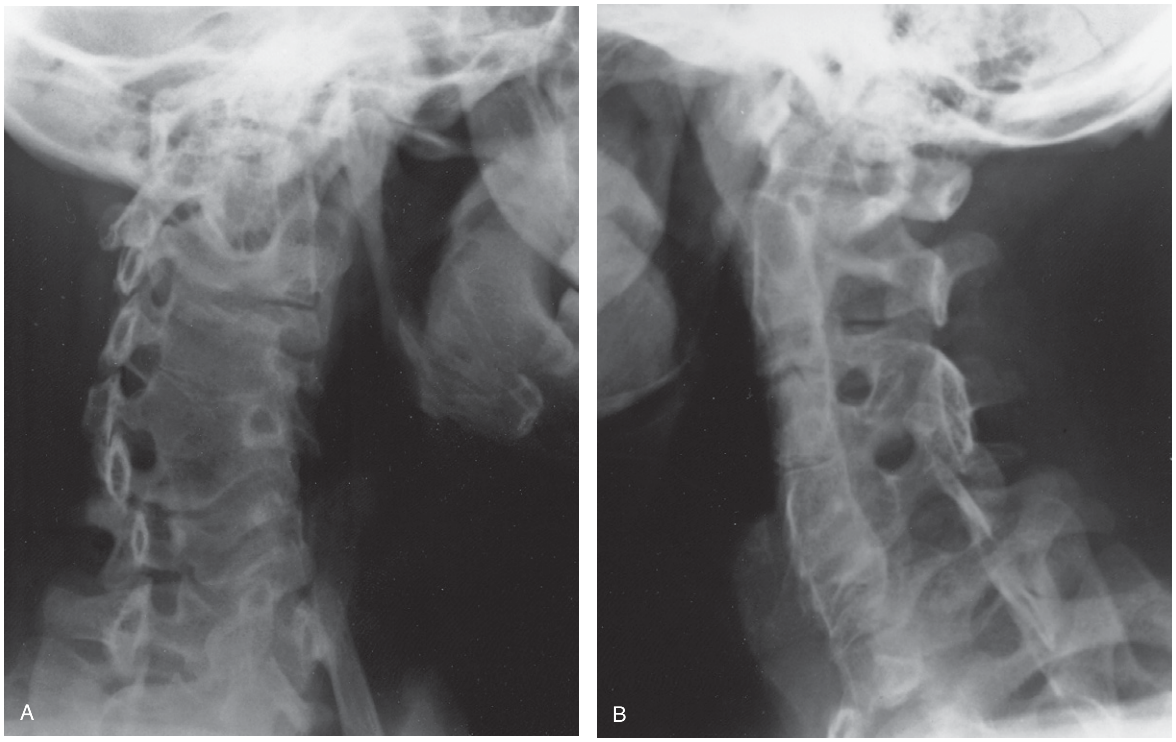


Figure 77.3 (A and B) Cervical spine oblique plain X-ray images showing some of the anomalies.

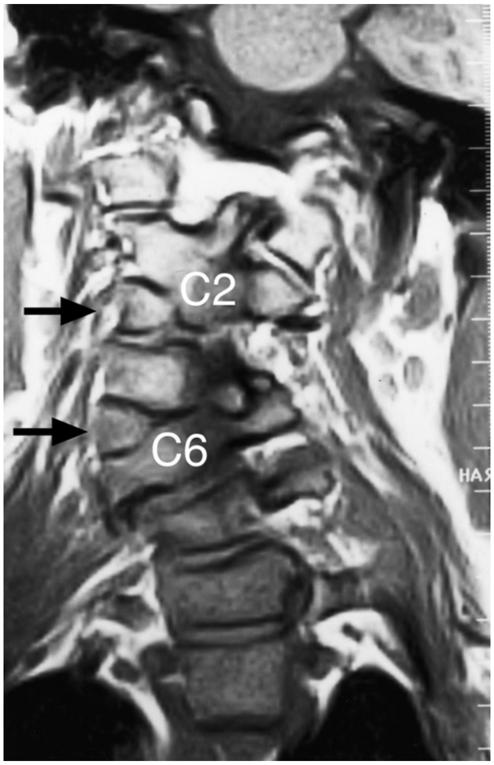


Figure 77.4 Cervical spine MRI coronal image showing some of the osseous anomalies such as the C3 and C5 hemivertebrae (arrows).

Figure 77.5 Cervical spine sagittal MRI T1-weighted image. Note the adequate spinal canal containing the spinal cord.

Such a case may have potentially dangerous vertebral artery tortuosities or impingements associated with the osseous anomalies and a case such as this would best be treated by mild analgesia, soft tissue techniques or appropriate needle acupuncture if symptoms were of an unacceptable level.

KEY POINT

The spine can have hidden anomalous dangers that may go unrecognized unless they are visualized by imaging.

Case 78

C7–T1 posterior central intervertebral disc protrusion

COMMENT

The absence of upper limb symptoms does *not* mean that there is no intervertebral disc posterior protrusion.

PROFILE

A 33-year-old female nurse of medium build who does not smoke cigarettes or drink alcohol.

PAST HISTORY

She had never had any spinal problems apart from having low back 'strain' approximately 13 years ago that resolved following her taking off two or three days from work.

Six years ago she was the seat-belted driver of a sedan when, without warning, another sedan approached on the wrong side of the road and hit her car head on. She received severe lacerations due to her head hitting the windscreen and her chest hitting the steering wheel. She lost several teeth and sustained fractures of the sternum, nose and second right rib.

PRESENTING COMPLAINT(S) (Fig. 78.1)

Cervical spine pain in general but particularly in the lower cervical spine at the cervico-thoracic junction since the motor vehicle accident 6 years ago. She described this as being constant with variable degrees of intensity of pain. Suboccipital pain can sometimes radiate to the temporal regions bilaterally, causing headaches. Hanging clothes on a clothesline is 'too painful' and makes the arms feel 'heavy'. Lifting her small children causes an aggravation of her neck pain syndrome and even less strenuous activities, such as preparing food, aggravate her neck pain syndrome and make her arms 'feel weak and heavy'. She is wary of taking medication, so only takes paracetamol as required and this provides her with temporary relief.

She tried physiotherapy treatment but this 'stirred up the pains'. She prefers to rely on a TENS device as this provides temporary relief. Her husband massages the muscles posterior to her cervical spine and this provides temporary relief. Coughing and sneezing may aggravate her neck pains.

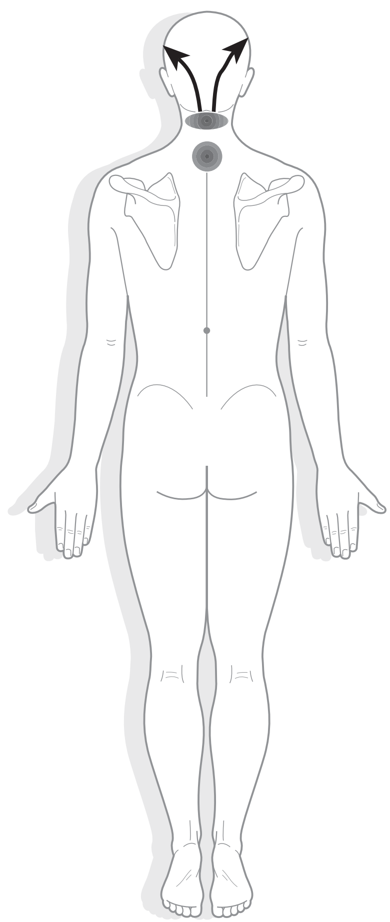


Figure 78.1

AETIOLOGY

A motor vehicle accident 6 yeas ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic spine elicited no pain. Palpation of the cervical spine elicited tenderness bilaterally from C4 to T1. The deep reflexes in the arms and legs were normal. The plantar response was normal. Pinprick and light touch sensations of the shoulders, arms, hands and legs were normal. Vibration sensation at the ankles and elbows was normal. Power in the arms, legs and feet was normal. The circumference of the arms, 12 cm above the left and right elbows, was 26.5 cm bilaterally.

Cervical spine active ranges of movement were measured using a CROM instrument:

1. Flexion – limited to 43° due to pain at the C5–T1 level centrally.
2. Extension – was of full range and painless, as long as extension was maintained for only a short period of time, otherwise extension aggravated her symptoms.
3. Left and right rotation – elicited a 'pulling' sensation on the right side of the neck.

Internal rotation of the left and right arms caused a 'heavy burning pain and pulling' in the C5–T1 region. Adson's manoeuvre did not indicate any compression of the subclavian arteries. Pressure on the sternum resulted in local pain over the sternum. The blood pressure in the right arm was 118/69 (seated). The consensual light and accommodation reflexes were normal as were the optic fundi and eye movements. She was able to smell coffee with no difficulty and was able to make various facial expressions.

IMAGING REVIEW

A cervical spine MRI report, available for an MRI study performed 2 years after the motor vehicle accident, reported some loss of the normal cervical spine lordosis, with a disc bulge posteriorly at the C5–6 and C7–T1 posterior disc protrusion.

CLINICAL IMPRESSION

C5–6 and C7–T1 intervertebral disc injuries apart from other soft tissue injuries of the neck.

WHAT ACTION SHOULD BE TAKEN?

Plain cervical spine imaging, including functional views, were requested to evaluate cervical spine function. An updated MRI was requested to evaluate the current state of the C5–6 and C7–T1 disc bulge.

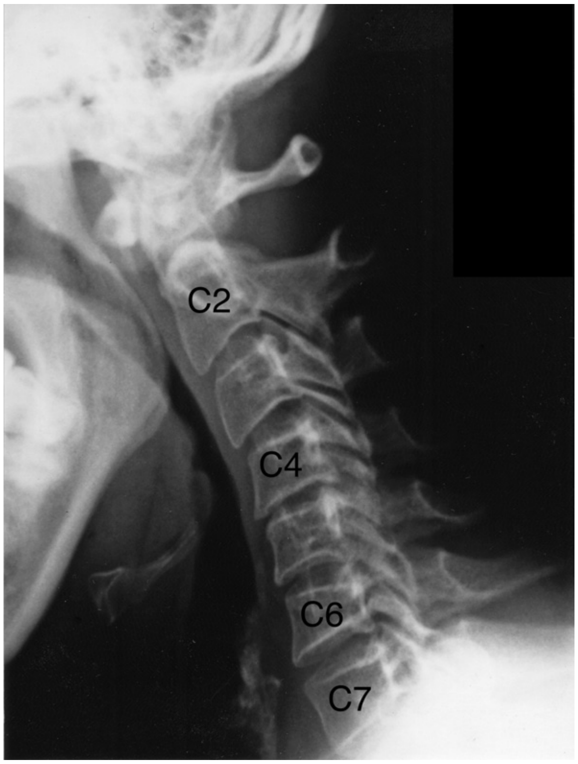


Figure 78.2 Cervical spine flexion plain X-ray image. Note the loss of normal cervical spine contour with restricted movement below the C4 level.

The functional plain X-ray images showed a loss of normal cervical spine contour as seen on both the flexion (Fig. 78.2) and extension views, with a restricted range of movement below the C4 level.

The updated MRI study (performed approximately 6 years following the motor vehicle accident) showed a C7–T1 intervertebral disc posterior protrusion (Fig. 78.3) that was located centrally (Fig. 78.4).

DIAGNOSIS

- C7–T1 central intervertebral disc posterior protrusion causing effacement of the anterior part of the spinal cord.
- Headaches of cervicogenic origin.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

She was advised to obtain a neurosurgical opinion and to minimize or avoid activities that aggravate her neck pain

Figure 78.3 Cervical spine MRI parasagittal T2-weighted image. Note the posterior disc protrusion (arrow) at the C7–T1 level encroaching upon the pain sensitive anterior surface of the dural tube and upon the spinal cord (C) itself.

syndrome. She was given ergonomic advice regarding daily activities. She decided to first modify her daily activities to determine whether she could minimize her symptoms before seeing a neurosurgeon.

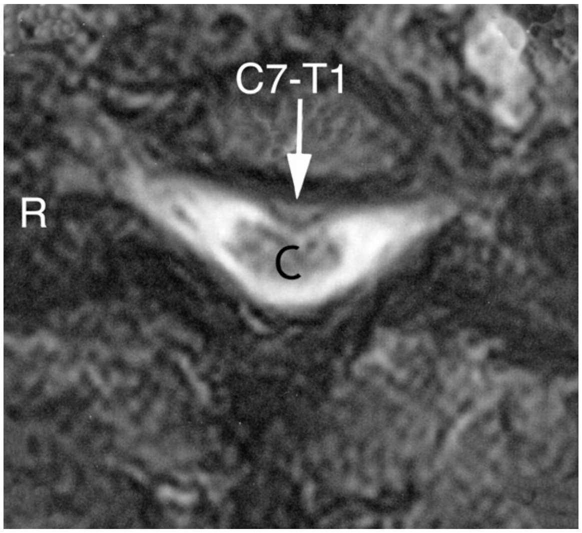


Figure 78.4 Cervical spine MRI T2-weighted axial image at C7–T1. Note the posterior and centrally located C7–T1 intervertebral disc protrusion (arrow) pressing upon the anterior part of the spinal cord (C).

KEY POINTS

A central disc protrusion does not normally cause radicular symptoms but can cause pain due to pressure upon (i) the pain sensitive anterior surface of the dural tube, (ii) the recurrent meningeal nerve, and (iii) the blood vessels between the protrusion and the dural tube.

Case 79

Hyperflexion injury

COMMENT

Craniovertebral junction MRI should be considered in cases where forceful movements occur between the head and the cervical spine.

PROFILE

A 35-year-old male manual worker of average build who smokes approximately 10 cigarettes per day.

PAST HISTORY

Approximately 7 months ago he was bending forward in front of a rock wall that held back a 2 m high bank of sand when the wall gave way and hit him on the back of his head, forcing his chin onto his chest. He immediately felt neck pain and a sensation 'like the neck broke' and, at the same time, he felt a suboccipital 'headache' that radiated to the temporal areas bilaterally.

He took off 1 week from work then saw his general medical practitioner who prescribed Panadol. He then went to see another general medical practitioner who referred him for a neck X-ray and, some time later, for a cervical spine MRI.

He was then referred for an orthopaedic opinion.

PRESENTING COMPLAINT(S) (Fig. 79.1)

Unrelenting constant suboccipital pain that radiates bilaterally to the temporal areas causing a headache. There is no other radiation of pain and he has no symptoms in his upper or lower limbs. Moving his neck cause an increase in his neck pain and headache. The back of his neck is extremely sensitive, even to water from the shower and he cannot bear to have his neck touched. His symptoms are aggravated by coughing, bearing down, work activities and home activities. For example, he can no longer hang up the clothes washing, so he now uses a clothes drier instead. His symptoms have not been relieved by treatment with medication, acupuncture, chiropractic or physiotherapy.

He has tried various prescription medications, e.g. Vioxx, Tramal SR and Panadeine Forte, without success.

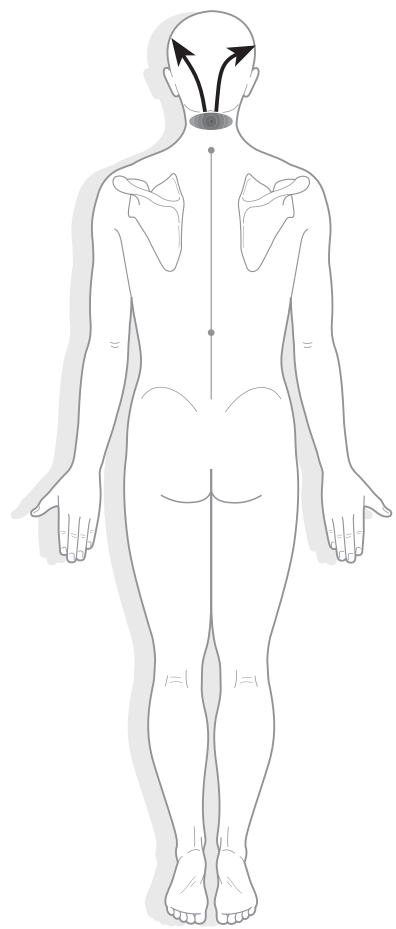


Figure 79.1

AETIOLOGY

Hit on the back of the head 7 months ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic spine did not elicit any pain. Palpation of the paraspinal neck muscles was not possible because his neck was hypersensitive to being touched. Toe walking power (S1) and heel walking power (L5) were normal, as were the deep tendon reflexes in the upper and lower extremities. The plantar response was normal. Vibration sensation at the elbows and ankles was normal. Pinprick sensation over the upper posterior torso and the upper and lower extremities was normal. Motor power in the extremities appeared to be normal. Hoffmann's test was normal.

The circumference of the arm, 13 cm above the elbow, was 28 cm (left) and 29.5 cm (right), a bit greater than one would expect. The blood pressure in the right arm was 110/76 in the seated position.

Active cervical spine ranges of movement were measured using a CROM instrument (see Box 79.1).

Cervical spine traction could not be performed as his neck was too sensitive to touch. Cervical spine compression could not be performed as the slightest pressure on the top of his head caused a significant increase in his symptoms. Palpation over the right cervico-shoulder region of the trapezius muscle elicited pain in the right trigger point.

Box 79.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	10°	He felt suboccipital pain radiating to the temporal areas bilaterally
Extension	60°	10°	He felt suboccipital pain radiating to the temporal areas bilaterally
Lt lateral bending	45°	30°	He felt suboccipital pain radiating to the temporal areas bilaterally
Rt lateral bending	45°	8°	He felt suboccipital pain radiating to the temporal areas bilaterally
Lt rotation	80°	12°	He felt suboccipital pain radiating to the temporal areas bilaterally
Rt rotation	80°	11°	He felt suboccipital pain radiating to the temporal areas bilaterally

Note that there was considerable restriction of all movements.

IMAGING REVIEW

Cervical spine plain X-ray images (without functional images) taken 3 weeks post-injury were reviewed and showed (i) slight retrolisthesis of C2 on C3, with a loss of the normal cervical spine lordosis in that most of the lordosis takes place above the C4 level (Fig. 79.2), and (ii) no lipping of the vertebral body margins, i.e. there is no evidence of a longstanding injury.

An MRI of the brain and cervical spine showed (i) small posterior disc bulges at C4–5 and C5–6 (Fig. 79.3), and (ii) early desiccation of the C4–5, C5–6 and C6–7 intervertebral discs. These bulges press on the pain sensitive anterior surface of the dural tube.

CLINICAL IMPRESSION

Musculoligamentous soft tissue injuries of the cervical spine.

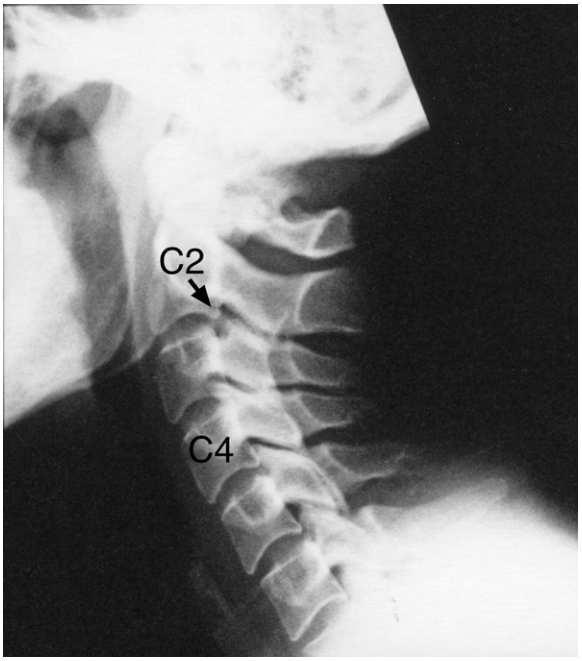


Figure 79.2 Cervical spine lateral plain X-ray image. Note (i) the slight retrolisthesis of C2 on C3 (black arrow), and (ii) the loss of normal cervical spine lordosis (with most of the lordosis taking place above the C4 level) suggesting soft tissue injuries to the neck. There is no lipping of the vertebral body margins and there is no subchondral sclerosis of the zygapophysial 'facet' joints, i.e. there is no evidence of a longstanding injury.

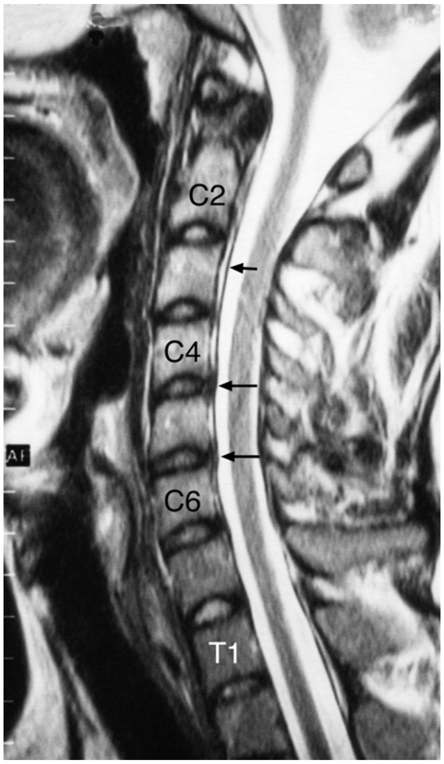


Figure 79.3 Cervical spine MRI sagittal T2-weighted image. Note the small posterior disc bulges (black arrows) at C4–5 and C5–6 that press on the pain sensitive anterior surface of the dural tube (small black arrows). There is early desiccation of some of the discs.

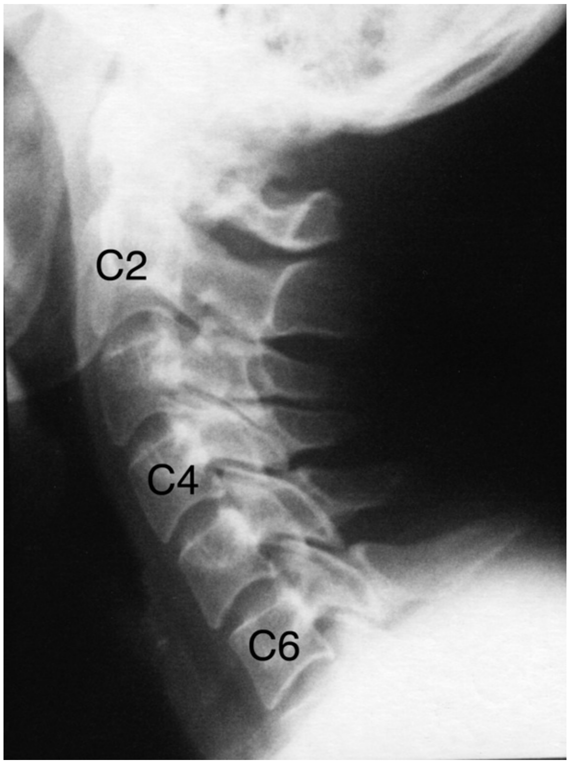


Figure 79.4 Neutral lateral cervical spine view. Note the loss of normal cervical spine lordosis with most of the lordosis taking place above the C4 level, suggesting soft tissue injuries to the neck.

WHAT ACTION SHOULD BE TAKEN?

In view of his ongoing symptoms, cervical spine neutral lateral and functional X-ray views were requested. The lateral view showed a loss of the normal cervical spine lordosis with most of the lordosis taking place above the C4 level, suggesting soft tissue injuries to the neck (Fig. 79.4).

The flexion (Fig. 79.5) and extension (Fig. 79.6) views show marked limitation of ranges of movement with loss of normal contours of the cervical spine.

DIAGNOSIS

Musculoligamentous soft tissue injuries of the cervical spine including posterior disc bulges at C4–5 and C5–6 resulting in a complex regional pain syndrome.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of

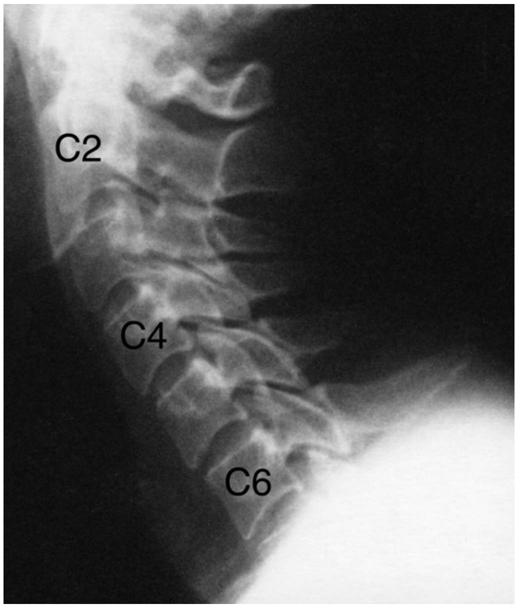


Figure 79.5 Cervical spine flexion plain X-ray image. Note the marked limitation of range of movement with loss of normal cervical spine contour.

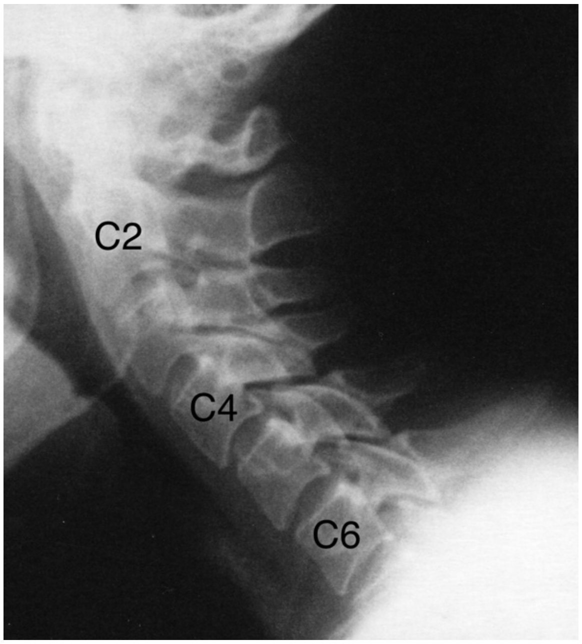


Figure 79.6 Cervical spine extension plain X-ray image. Note the marked limitation of range of movement with loss of normal cervical spine contour.

symptoms was understood. As he could not tolerate his neck being touched he was advised to see a specialist in rehabilitation medicine who prescribed Neurontin which provided some relief. A couple of years later he requested a re-evaluation of his condition at which time his symptoms and signs were much the same. Repeated cervical spine flexion and extension functional X-ray views showed virtually the same cervical spine contours. MR imaging of his cranio-vertebral junction was recommended to thoroughly investigate this area of his neck but that specialized technique (Krakenes et al 2001) was not available locally at that time.

Note

To investigate the cranio-vertebral junction and to assess the appearance of the alar ligaments, the transverse ligament, the tectorial membrane and the anterior and posterior atlanto-occipital membranes by MR imaging requires a high spatial resolution and good contrast between tissues. This can be obtained by using proton-density-weighted (PDW) sequence with 2-mm-thick sections. The examination would need to be performed in three orthogonal planes with the patient's head fixed in the neutral position while using a standard head coil. The axial sections should include the foramen magnum to the base of the dens with coronal sections (from the anterior arch of the atlas to halfway through the spinal canal) and sagittal sections (from one occipital condyle to the other) (Krakenes et al 2001, 2006).

KEY POINT

Frequently MRI cervical spine reports state 'the cranio-vertebral junction is normal' but a routine cervical spine MRI does *not* provide detail to support such an opinion.

References

Krakenes J, Kaale B R, Rorvik J, Gilhus N E 2001 MRI assessment of normal ligamentous structures in the craniovertebral junction. Neuroradiology 43: 1089–1097.

Krakenes J, Kaale B R 2006 Magnetic resonance imaging assessment of craniovertebral ligaments and membranes after whiplash trauma. Spine 31: 2820–2826.

Further reading

Please see References above.

Case 80

Neck pain, headaches and facial injuries

COMMENT

When patients describe an unusual or different symptom, that symptom should be thoroughly investigated.

PROFILE

A 56-year old male delivery truck driver who smokes up to 10 cigarettes per day and does not drink alcohol.

PAST HISTORY

Approximately 4 years ago he was the seat-belted driver of a truck when it rolled onto its hood, causing the cabin to cave in, compressing his head against the steering wheel. He did not lose consciousness so, with difficulty, he climbed out of the crushed cabin. He was taken by ambulance to hospital where skull X-rays were performed; he was told that these showed no fractures. His facial injuries and lacerations were sutured and he was admitted for two nights to hospital for observation. During that time he complained of feeling his facial bones 'moving' when he applied pressure to his teeth. However, he was told that his X-ray examination showed no fractures and that further imaging was not necessary.

Following discharge from hospital, he requested a CT scan of his face from his general medical practitioner and this showed numerous facial bone fractures, so he was referred to a maxillo-facial surgeon who performed surgery for the facial fractures using titanium plates and screws.

PRESENTING COMPLAINT(S) (Fig. 80.1)

Central cervical spine pain of varying intensity at approximately the C5–7 level without any radiation to his arms. The pain may radiate from his neck to the top of his head causing headaches. His work exacerbates his symptoms unless he performs only light duties. He obtains relief from hot water from the shower directed to the back of his neck, lying down, rubbing rubefacients into his neck muscles posteriorly and using an analgesic as required. He has not experienced any bowel or bladder problems. He had not experienced the above symptoms before the motor vehicle accident approximately 4 years earlier.

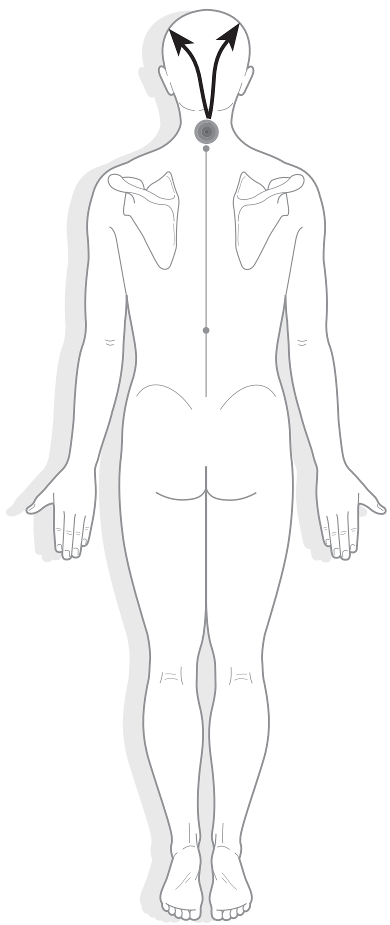


Figure 80.1

AETIOLOGY

Motor vehicle accident approximately 4 years ago.

EXAMINATION

In the erect posture, there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic and lumbar spines did not elicit any pain. There was a palpable lump on the back of his head (occiput area). Deep palpation of the paraspinal muscles elicited pain at approximately the C4–6 level bilaterally and at the cervico-thoracic junction. On applying thumb pressure to the right trapezius trigger point there appeared to be some local pain. The deep tendon reflexes in the upper extremities were normal. The plantar response was normal. Vibration sensation at the elbows and ankles was normal. Pinprick sensation appeared to be normal over the upper and lower extremities, apart from hypoaesthesia in the left little finger (C7). Motor power in the upper extremities appeared to be normal. The Valsalva manoeuvre did not elicit an increase in his neck pain. The blood pressure in the right arm in the seated position was 128/70.

Active cervical spine ranges of movement were measured using a CROM instrument (see Box 80.1).

Box 80.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	42°	Elicited pain at the C5–7 level
Extension	60°	53°	Elicited pain at the C5–7 level
Lt lateral bending	45°	37°	Elicited pain at the C5–7 level
Rt lateral bending	45°	40°	Elicited pain at the C5–7 level
Lt rotation	80°	60°	Painless
Rt rotation	80°	70°	Painless

Left and right rotation plus extension, cervical spine traction, cervical spine compression and downward shoulder pressure were all painless. Trapezius trigger point pressure elicited right trapezius trigger pain. Active thoracic spine ranges of movement were of full range and painless.

IMAGING REVIEW

1. An X-ray of his skull was performed on the day of the accident and was not available for review but it was reported as being 'normal'.
2. A CT scan of the facial bones performed several days after the accident showed facial fractures, i.e. Leforte's type 1 (bilateral horizontal fracture of the maxilla), left zygoma, left orbital floor and the nose (naso-ethmoidal).

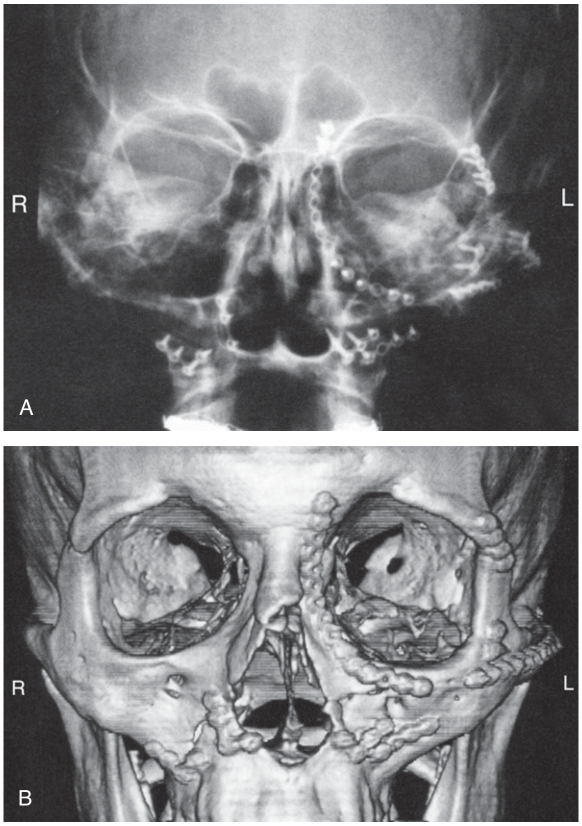


Figure 80.2 (A (X-ray) and B (CT reconstruction)) Note the internal fixation with a plate and screws in relation to the left zygomaticofrontal synostosis, floor and medial wall of left orbit, left zygomatic arch, floor and medial walls of both maxillary antra.

3. An X-ray and CT scan of his facial bones was performed following surgery (Fig. 80.2A and B). The report stated: 'Internal fixation with a plate and screws is noted in relation to the left zygomaticofrontal synostosis, floor and medial wall of the left orbit, left zygomatic arch, floor and medial walls of both maxillary antra.
4. A cervical spine X-ray examination had not been performed until 3 years after the accident and the neutral lateral image (Fig. 80.3) showed: (i) a loss of normal contour with a kyphosis extending from C2 to C6, (ii) a loss in the C5–6 and especially C6–7 disc space heights, (iii) an angulation at the C4 level, and (iv) some anterior lipping of the vertebral bodies at the C5–6 and C6–7 levels.

The flexion view (Fig. 80.4) showed slight anterolisthesis of C2 on C3 and what appears to be a past compression fracture at C6 as suggested by some slight loss in height of the vertebral body when compared to the height of the adjacent vertebral bodies.

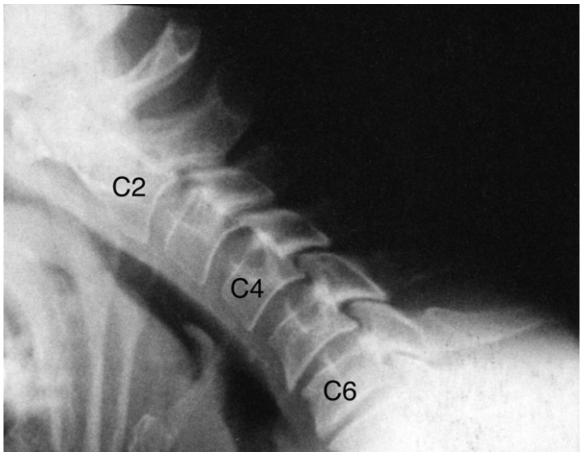


Figure 80.3 Cervical spine neutral lateral plain X-ray image. Note (i) the loss of normal contour with a kyphosis extending from C2 to C6, and (ii) degenerative change at the C5–6 and C6–7 intervertebral disc levels with some loss of disc height at these levels and anterior lipping of the adjacent vertebral bodies.

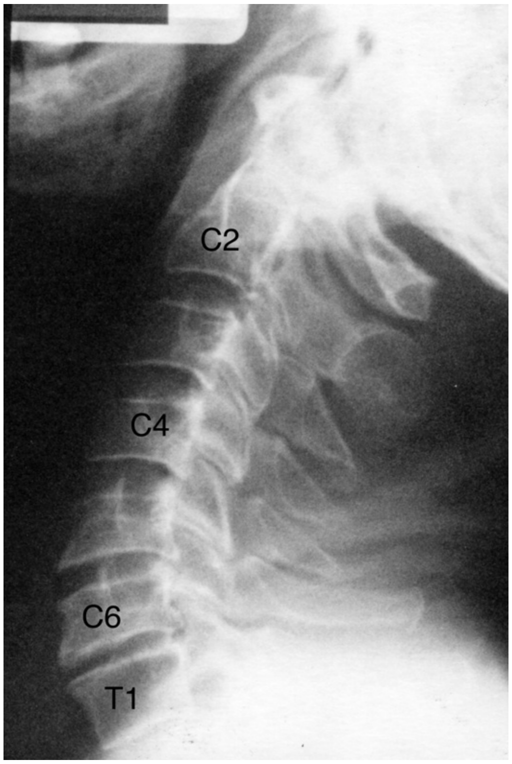


Figure 80.4 Cervical spine flexion plain X-ray image. Note (i) the slight anterolisthesis of C2 on C3, (ii) the angulation at the C4, (iii) what appears to be an old compression fracture at C6 as suggested by some loss in height of the vertebral body when compared to the height of adjacent vertebral bodies, and (iv) anterior lipping of the vertebral bodies at the C5–6 and C6–7 levels.

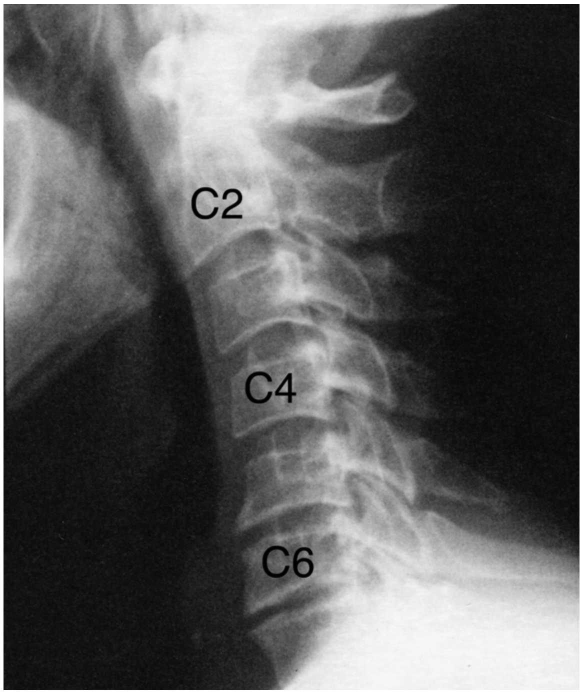


Figure 80.5 Cervical spine extension plain X-ray image. Note the loss in the C5–6 and C6–7 disc space heights as compared to upper disc heights. The C6 vertebral body height appears to be slightly less than that of the adjacent vertebral body heights suggesting a past compression fracture at C6.

The extension view (Fig. 80.5) showed thinning of the C5–6 and C6–7 intervertebral disc space heights, particularly at C6–7.

The oblique views showed early encroachment of the left C5–6 and C6–7 intervertebral foramina and the right C6–7 foramen due to uncovertebral joint early osteoarthrotic changes.

CLINICAL IMPRESSION

Cervical spine musculoligamentous injuries. Headaches of cervicogenic origin.

WHAT ACTION SHOULD BE TAKEN?

An MRI brain scan as a precaution in view of his history of headaches and facial fractures. This MRI was reported as showing an area of old gliosis from previous haemorrhage in the right basal ganglia.

DIAGNOSIS

- Chronic musculoligamentous soft tissue injuries to his neck.
- Headaches of cervicogenic origin.
- Early osteoarthrotic and intervertebral disc degenerative changes at C5–6 and C6–7 levels.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

He was advised not to subject his cervical spine to heavy manual work or awkward positions and to only perform light duty work so as to avoid aggravating his neck pain syndrome. Cervical spine exercises (cervical spine flexion with left and right rotation within a comfortable range of movement) were suggested to mobilize his cervical spine and, should he require active treatment, needle acupuncture may be considered. As he controls his condition using the abovementioned home treatment he was advised to continue with that approach as well as modifying his work. He accepted that there was no sinister underlying condition causing his symptoms. In view of his history of a previous brain haemhorrhage, cervical spine manipulation was not considered.

KEY POINT

When an initial imaging examination does not find evidence of injury to explain a patient's symptoms but symptoms persist, it is prudent to re-evaluate a patient's condition.

Introduction

Before reading the thoracic spine cases it is important to consider the following summary of some possible causes of thoracic spine pain, with or without radiculopathy (Table viii).

PHYSICAL EXAMINATION

The physical examination should be orderly and systematic and should include the following thoracic examinations as indicated by the patient's presenting complaint(s) (Table ix).

The symptoms and signs caused by a herniated thoracic disc depend on the location of the herniation:

- **Figure xv shows the clinical features of a *posterolateral* thoracic disc herniation that may cause nerve root impingement, as summarized in the figure.**
- ***Midline* herniation may cause symptoms in the arm, thoracic spine and/or in the legs as well as sensory loss, bladder incontinence and upper motor neuron lesion signs.**

Table viii Some possible causes of thoracic spine pain

Acute spinal pain
Dissecting aortic aneurysm
Myocardial infarction
Febrile disorders
Injury

Chronic spinal pain

1. **Traumatic, mechanical or degenerative**
   - Thoracic spine strain; fatigue, obesity, pregnancy causing altered biomechanics, avulsion fracture of T1 spinous process (clay shoveller's fracture)
   - Injuries of bone, joint, intervertebral disc or ligaments
   - Degenerative or traumatic changes of the spine (osteoarthrosis; spondylosis)
   - Scoliosis: primary and secondary; kyphoscoliosis
   - Spinal or intervertebral canal stenosis
   - Spinal origin – cervical spine osteoarthrosis and disc herniation (especially C4–C7), cervicothoracic junction injury, thoracic spine osteoarthrosis including joints (e.g. costovertebral, zygapophysial, intervertebral with disc), thoracolumbar junction injury
   - Other thoracic joints – sternoclavicular, manubriosternal, costochondral, sternocostal
   - Disorders of ribs (including 12th rib syndrome, rib tip syndrome, slipping rib syndrome), muscles, ligaments, e.g. ossified ligamenta flava
   - Other – costochondrites, Tietze's syndrome, xiphoidalgia, myofascial syndrome, thoracic outlet syndrome, intercostal neuralgia. Post-nephrectomy syndromes with entrapment of 12th thoracic intercostal nerve in the scar tissue. Serratus anterior nerve palsy
   - Subscapular bursitis
2. **Joint dysfunction**
   - Zygapophysial
   - Intervertebral disc
   - Costochondral
3. **Metabolic**
   - Osteoporosis
   - Osteomalacia
   - Hyper- and hypo-parathyroidism
   - Ochronosis
   - Fluorosis
   - Hypophosphataemic rickets
4. **Unknown causes**
   - Inflammatory arthropathies of the spine, such as ankylosing spondylitis, the spondylitis of Reiter's (Brodie's) disease, psoriasis, ulcerative colitis, Whipple's and Crohn's diseases; diffuse idiopathic skeletal hyperostosis
   - Rarely polymyositis and polymyalgia rheumatica
   - Paget's disease of bone
   - Scheuermann's 'disease'

table continues

Table viii Some possible causes of thoracic spine pain—Cont'd

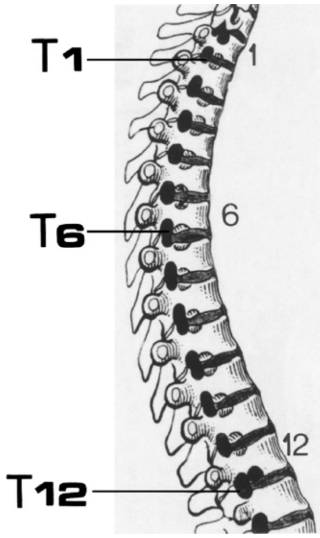
5. **Infective conditions of bone, joint and theca of the spine**
   - Osteomyelitis
   - Tuberculosis
   - Melioidosis
   - Undulant fever (abortus and melitensis)
   - Typhoid and paratyphoid fever and other *Salmonella* infections
   - Syphilis
   - Yaws
   - Very rarely Weil's disease (leptospirosis icterohaemorrhagica)
   - Spinal pachymeningitis
   - Chronic meningitis
   - Subarachnoid or spinal abscess
   - Herpes zoster
   - Post-herpetic neuralgia
6. **Psychogenic**
   - Anxiety
   - Depression
   - Hysteria
   - 'Compensation neurosis'
   - Malingering
7. **Neoplastic – benign or malignant, primary or secondary**
   - Osteoid osteoma
   - Eosinophilic granuloma
   - Metastatic carcinomatosis
   - Bronchial carcinoma
   - Oesophageal carcinoma
   - Sarcoma
   - Myeloma
   - Primary and secondary tumours of spinal canal and nerve roots: ependymoma; neurofibroma; glioma; angioma; meningioma; lipoma; rarely cordoma
   - Reticuloses, e.g. Hodgkin's disease
   - Neoplasms of the chest wall and pleura
8. **Cardiac and vascular**
   - Coronary insufficiency
   - Pericarditis
   - Pulmonary embolism
   - Subacute bacterial endocarditis
   - Grossly enlarged left atrium in mitral valve disease
   - Luetic or dissecting thoracic aorta aneurysm; aneurysm with thoracic vertebral involvement
   - Enlarged thoracic aortic aneurysm
   - Subarachnoid or spinal haemorrhage
   - Arteriovenous malformations
9. **Pulmonary**
   - Embolus
   - Pneumothorax
   - Pneumonia
   - Pleurisy
   - Pleurodynia (Bornholm's disease)
10. **Oesophageal**
    - Spasm
    - Rupture
    - Oesophagitis
    - Aerophagy
    - Hiatus hernia
11. **Acute subdiaphragmatic**
    - Stomach disorders, e.g. peptic ulcer, carcinoma
    - Biliary, renal, duodenal, pancreatic and subphrenic disorders
12. **Blood disorders**
    - Acute haemolytic states
13. **Drugs**
    - Corticosteroids
    - Methysergide
    - Compound analgesic tablets

Modified from Hart (1985) and Bland (2000).

Table ix Some elements of the thoracic spine physical examination

Erect posture examination	
Observe for Fluidity of movement Body build Skin markings – café-au-lait spots, lipomata, melanoma Posture Deformities Scoliosis Idiopathic Postural with pelvic obliquity Spine alignment *Test spinal column motion for* Flexion Extension Side bending Rotation	*Compression of rib cage* *Intercostal expansion at nipple line* a. 5–7.6 cm (normal) b. <2.6 cm (ankylosing spondylitis) *Palpate for* Muscle spasm Myofascial trigger points Supraspinous and interspinous ligament tenderness Adjacent muscle tenderness Relative motion between adjacent vertebrae (by motion palpation) in an attempt to find restricted movement *Observe gait* Steady or unsteady
Seated	
Neurological tests Knee jerk, ankle jerk Pinprick sensation on torso and lower limbs	Vibration sensation at ankles Straight leg raising
Measure	
Thigh circumference bilaterally Calf circumference bilaterally	
Supine	
Kernig test (spinal cord stretch)* Tests to increase intrathecal pressure: Naffziger test* Valsalva manoeuvre*	Superficial abdominal reflexes (upper T7–10; lower T10–L1) Test sensation and motor power Palpation of chest, cervical and axillary lymph nodes Auscultation
Prone – Palpate thoracic spine, over related joints, and trigger points	

*See Definitions and abbreviations chapter.
Adapted from Hoppenfeld (1976), Hart (1985), Mackenzie (1985) and Keim & Kirkaldy-Willis (1987).



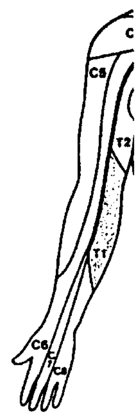
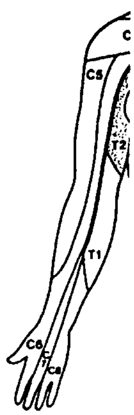
HERNIATION	T1-2	T2-3	T3-4	T4-5	T5-6, T6-7	T7-9/10	T10-12
NERVE ROOT	T1	T2	T3	T4	T5, T6	T7-9/10	T10-12
SENSORY SUPPLY							
				Nipple line level		T7 at xiphoid process level, T10 at umbilicus level	T10 at umbilicus level; T12 at groin level
PAIN	Medial arm and shoulder (deep ache)	Axilla	Intercostal dermatome	Intercostal dermatome; T4 syndrome: vague pain & paraesthesiae in upper limbs, not derma-tomal, diffuse & vague head & posterior neck pain (may also involve upper thoracic vertebrae)	Intercostal dermatome	Intercostal dermatome	Intercostal (T10 & T11) dermatomes; Thoraco-lumbar T12 & L1 ventral rami (subcostal and iliohypogastric nerves respectively) ► lower abdominal wall, skin of groin, lateral iliac crest (as the lateral cutaneous branch)
MOTOR WEAKNESS	All small muscles of hand	-	The intercostal muscles are segmentally innervated and are difficult to evaluate individually				
SCREENING EXAM	Thumb abduction, Finger Abduction	-	-	T4 syndrome: spinal tender-ness & joint stiffness (T4-T5)	Beevor's sign (T5-12) to test rectus abdominis muscle's segmental innervation integrity (umbilicus should not move when patient does a quarter sit-up)		
REFLEXES	-	-	-	-	-	Superficial upper abdominal	Superficial lower abdominal

Figure xv The clinical features of a posterolateral thoracic disc herniation that may cause nerve root impingement. Adapted from: Hoppenfeld (1976, 1977), Wilkinson (1986), Kenna & Murtagh (1989), McGuckin (1986), Maigne (2000), Lawrence & Bukkum (2000), Pauchet & Dupret (1937) and Chusid (1985).

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Case 81

T9–10 intervertebral disc herniation

COMMENT

This lady was considered a malingerer due to her various complaints.

PROFILE

A 40-year-old married housewife.

PAST HISTORY

She had a fall 10 years ago and 'banged' her head on the floor. Six years ago she fell heavily onto her buttocks and back when she slipped and fell.

PRESENTING COMPLAINT(S) (Fig. 81.1)

Thoracic spine pain in the vicinity of T9–10, particularly on the right side, i.e. in the region in which a laminectomy was performed approximately 4 years ago. This pain has worsened in the last 3–4 weeks and now radiates, intermittently, from the T9–10 level along the adjacent right intercostal nerve to the front of her chest, at approximately the lower region of the sternum and below her right breast. This pain can last for about 45 minutes during which period she takes very 'shallow breaths' until the pain passes. She also complains of some 'hypersensitivity' of the right anterolateral part of her upper chest; this sensation extends as far as approximately the second rib level.

She is frustrated because she has been labelled a 'malingerer' and wants to know why she has debilitating symptoms in spite of having had surgery at the T9–10 level.

AETIOLOGY

Slipped and fell 6 years ago.

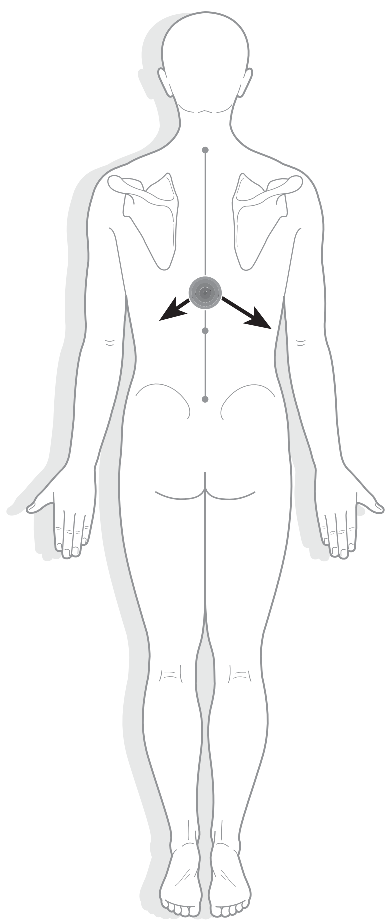

Figure 81.1

EXAMINATION

Deep tendon reflexes in the upper extremities were normal but it was difficult to elicit the knee jerks (L2,3,4), although these were elicited using a reinforced patella reflex test; the reflexes bilaterally were only one plus (two plus being normal). The ankle jerks (S1) were normal as was vibration sensation to a tuning fork applied to her left and right ankles. Pinprick sensation of the arms and hands was normal, as was the case in all parts of the legs and feet, apart from a feeling of hypoaesthesia on the lateral aspect (L2) of her right thigh. The circumference of her thighs was the same on the left and right sides. Percussion of her spine elicited considerable tenderness at approximately T4–5 and particularly at T9–10 where the laminectomy was performed approximately 4 years ago. Power in the lower extremities was normal. Coughing did not elicit any pain and the Naffziger test (compression of the jugular veins for 15 seconds before the patient was asked to cough) did not elicit any pain.

Active thoracic spine ranges of movement in the seated position were as follows:

1. Flexion – limited by approximately 15% due to pain on the right side of T9–10.
2. Extension – approximately of full range but elicited pain on the right side of T9–10.
3. Left rotation – limited to approximately 15% due to pain on the right of T9–10.
4. Right rotation – limited to approximately 15% due to pain on the right side of T9–10.

Auscultation of the heart and lungs was within normal limits. The blood pressure in the right arm was 134/88 and in the left arm 132/82, in the seated position, indicating no coarctation of the aorta.

IMAGING REVIEW

The report of a CT scan of her abdomen and pelvis before and after intravenous contrast (Fig. 81.2A) made no reference to the central to left sided calcified extruded disc material in the spinal canal at the T9–10 level. As her symptoms persisted, further thoracic spine plain X-ray films had been taken 5 months later and these showed a small area of calcification posterior to the T9–10 disc space within the intervertebral foramen (Fig. 81.2B). This finding led to a thoracic myelogram where dye was introduced by a lumbar puncture and clear CSF was obtained. This myelogram showed a 'minimal hold-up of the contrast at the T9–10 level' due to an extradural indentation (Fig. 81.2C). This was followed by a CT thoracic spine scan which showed a central to left sided calcified extradural mass at the T9–10 level; this represented calcified extruded disc material (Fig. 81.2D).

The patient had then undergone a laminectomy for removal of the T9–10 disc material.

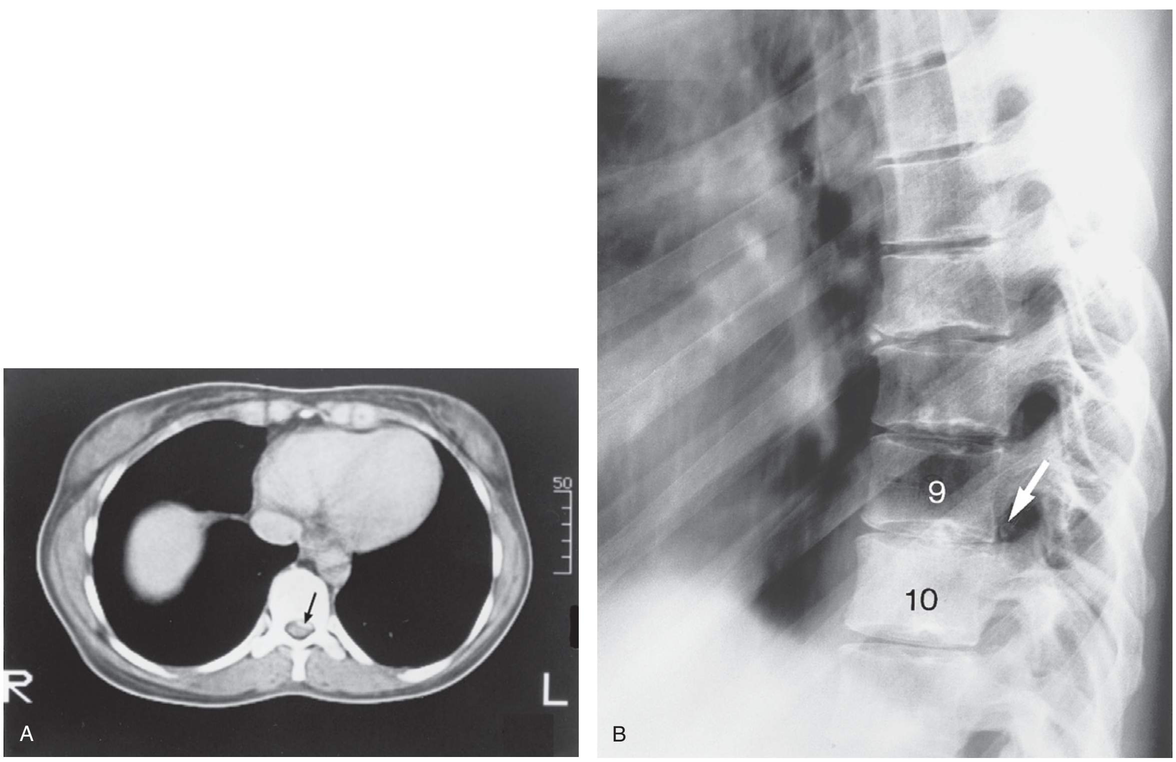

Figure 81.2 (A) A CT image at the T9–10 level after intravenous contrast. Note the central to left sided calcified extruded disc material in the spinal canal (black arrow). R and L = right and left sides, respectively, of the patient. (B) A lateral thoracic spine plain X-ray image taken 5 months after the CT scan shows a small area of calcification posterior to the T9–10 disc space within the intervertebral foramen (white arrow). 9 = ninth thoracic vertebra; 10 = tenth thoracic vertebra.

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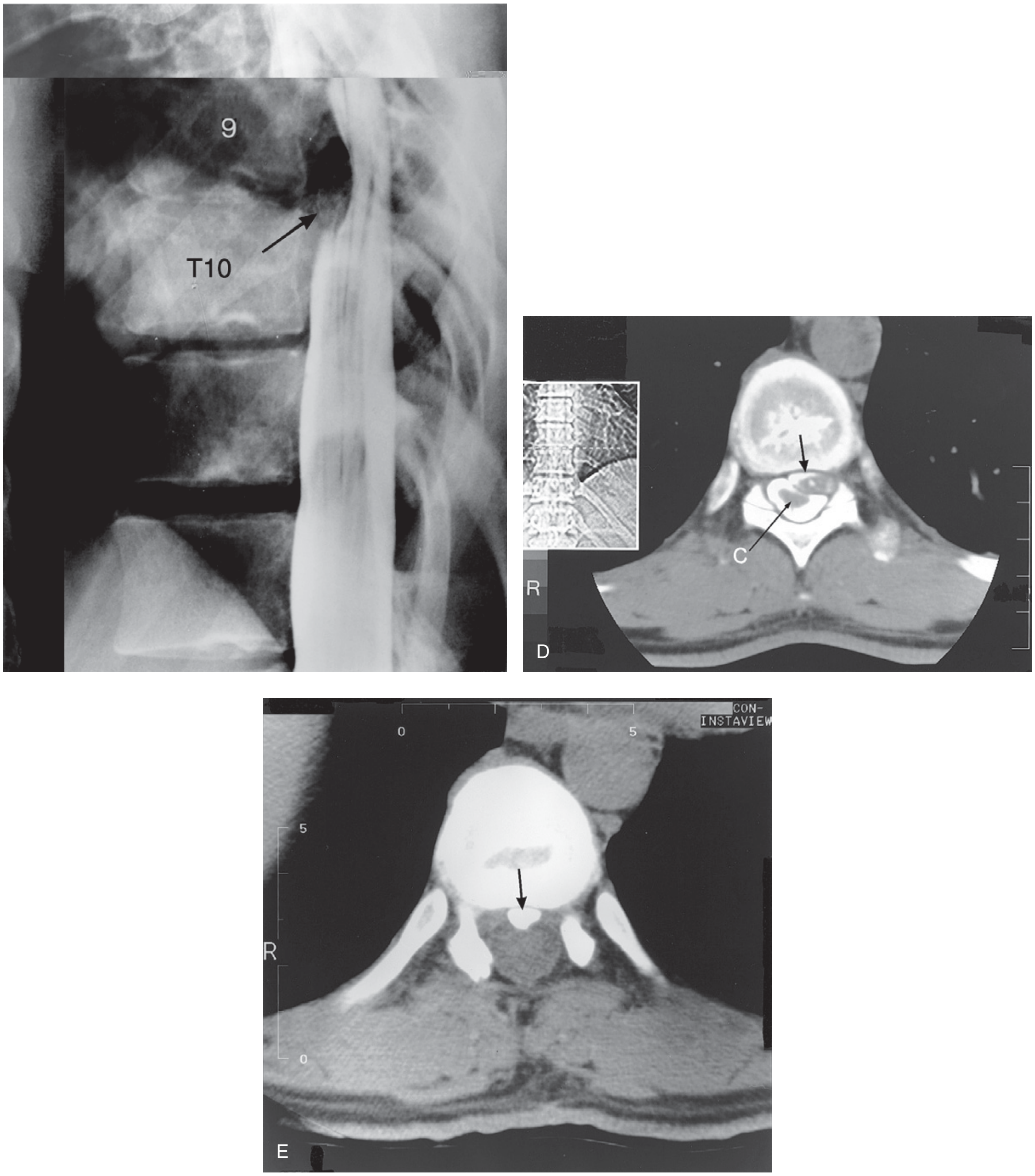

Figure 81.2 Cont'd (C) A thoracic spine myelogram showing a 'minimal hold-up of the contrast at the T9–10 level' due to an extradural indentation (black arrow). 9 = ninth thoracic vertebra; 10 = tenth thoracic vertebra. (D) A post-contrast thoracic spine CT image showing a central to left sided calcified extradural mass at the T9–10 level (arrow), i.e. a calcified disc protrusion. C = spinal cord within the cerebrospinal fluid. Note that the cord is somewhat displaced. R = right side of patient. (E) A post-surgery thoracic spine CT image showing that there was still 'a prominent piece of ossification centrally and anteriorly in the spinal canal' thought to be causing some degree of cord compression at this level (arrow). R = right of patient.

CLINICAL IMPRESSION

Possibly residual or recurrent disc at the T9–10 level.

WHAT ACTION SHOULD BE TAKEN?

As she still had a thoracic spine pain syndrome at presentation, a post-surgery CT thoracic spine scan was requested; this showed that there was still 'a prominent piece of ossification centrally and anteriorly in the spinal canal', thought to be causing some degree of cord compression at this level (Fig. 81.2E). An MRI of the thoracic spine showed 'residual calcified disc material at the T9–10 level which indents the anterior thecal sac and abuts and mildly flattens the anterior cord, with some narrowing of the cord at this level'.

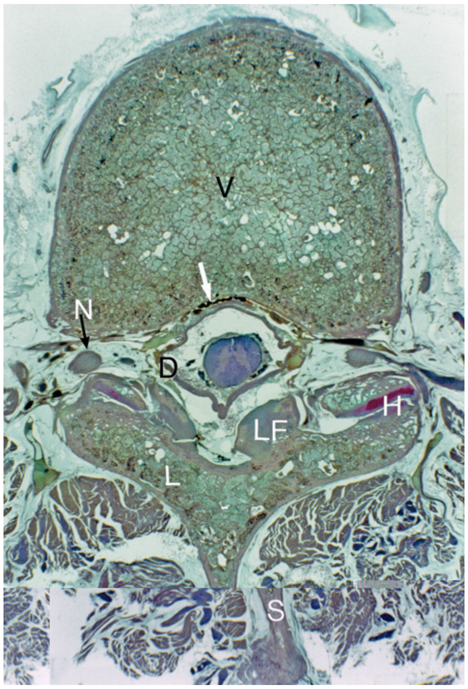

Figure 81.3 Superior to inferior horizontal view of a 200-micron thick postmortem histopathology section through the T10–11 level of the thoracic spine of a 40-year-old male. N = spinal nerve ganglion; LF = ligamentum flavum; L = lamina; D = dural tube containing the spinal cord; H = hyaline articular cartilage on the facet surfaces of the zygapophysial joints; S = spinous process; V = vertebral body. The small space between the pain sensitive anterior surface of the dural tube and the posterior part of the vertebral body, or intervertebral disc, has small blood vessels in it (white arrow). In addition, there is a recurrent meningeal nerve from each side (not visible at this magnification). See also colour plate section Fig. vii.30.

In order to understand the significance of this finding, Fig. 81.3 shows a horizontal histology section through a thoracic vertebra. This shows the vertebral body, the spinal canal, the left and right intervertebral foramina, the left and right zygapophysial joints with their facet cartilage, the lamina and the spinous process. Figure vi (General Introduction) shows the complex innervation of the nerve supply of the thoracic ventral compartment at the level of the vertebral body and intervertebral disc.

DIAGNOSIS

T9–10 central post-surgical calcification/ossification with a degree of cord compression.

TREATMENT AND RESULTS

The findings of the post-surgical CT and MRI scans were explained to the patient, as were the possible pain mechanisms such as: (i) pain resulting from irritation of the small blood vessels being compressed between the residual calcified disc material and the dural tube; (ii) compression of the recurrent meningeal nerves between the calcified disc material and the dural tube; and (iii) direct pressure upon the pain-sensitive anterior part of the dural tube. She was advised not to undergo further surgery unless her symptoms deteriorated and she was also advised to avoid movements that irritated her symptoms. She was told that it would be prudent to keep fit and to take part in exercise, such as walking, that did not aggravate her symptoms. She was particularly pleased to know why she still had residual symptoms following surgery and that her condition was real and not imagined.

She accepted her post-surgical condition and heeds the advice provided as a result of a thorough reappraisal of her symptoms. The conservative approach to her pain syndrome with a detailed explanation of her condition, which was indeed ‘real’, made her feel very much better.

Note

It should be noted that dural sympathetic innervation, if irritated at one particular level, e.g. T9, which has led to nociceptive stimulation, may enter the spinal cord at many levels (up to about 9, i.e. from T5 to L1) (Groen & Stolker 2000).

KEY POINTS

1. Thoracic disc herniation has long been a difficult entity to diagnose and treat and there is no typical presentation (Errico et al 1997).
2. Never label a patient as being a malingerer unless you have made a thorough appraisal of all facts – not many patients wish to be invalids!

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Case 82

T7–8 intervertebral disc posterior protrusion

COMMENT

A posterior central intervertebral disc protrusion does not cause radiculopathy.

PROFILE

A 31-year-old overweight man who is a manual worker.

PAST HISTORY

There is nothing contributory.

PRESENTING COMPLAINT(S) (Fig. 82.1)

Mid to lower thoracic spine pain, essentially centrally located, that is sometimes accompanied by 'chest' pains. His symptoms have been present for 6 years. An increase in activity causes an increase in his thoracic spine pain. There is no night pain per se. To date he has seen various medical practitioners but has not received a diagnosis for his symptoms. Treatment has consisted of non-steroidal anti-inflammatory drugs and analgesics but these did not provide long lasting relief. He then became depressed about his continuing symptoms as he had been given the impression that his condition was 'psychosomatic' and was put onto antidepressant medication. He had been to a hospital Pain Clinic and been told that he would have to 'live with' his problem. He said he was frustrated because his attempts to define the underlying problem had not resulted in an explanation being received by him.

AETIOLOGY

There was no known incident but he said he had always worked in heavy manual work.

EXAMINATION

In the erect posture there was no clinical evidence of leg length inequality, pelvic obliquity or postural scoliosis. Deep palpation of the paraspinal muscles elicited some

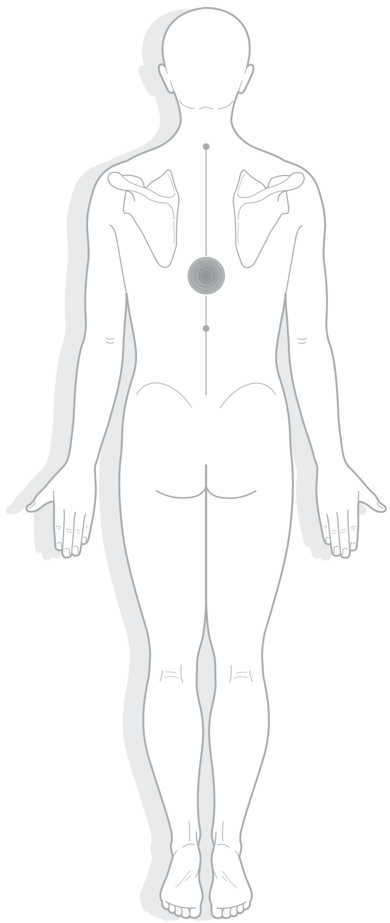

Figure 82.1

tenderness at approximately the T6–9 level bilaterally. Deep tendon reflexes in the upper and lower extremities were normal. Pinprick sensation over the posterior aspect of the torso appeared to be normal. The Valsalva manoeuvre aggravated his mid to lower thoracic spine pain.

Active ranges of thoracic spine movements were painless apart from flexion; this aggravated the mid to lower thoracic spine pain.

IMAGING REVIEW

Plain film radiographs of his thoracic spine and chest had been reported as 'normal'.

CLINICAL IMPRESSION

Possible mid to lower thoracic spine posterior central disc bulge or protrusion. A differential diagnosis of a benign space-occupying lesion within the spinal canal was considered.

WHAT ACTION SHOULD BE TAKEN?

A thoracic spine MRI was requested to further investigate his spine and the report stated: 'A small anterior extradural defect at approximately the T7–8 level is noted (Fig. 82.2).

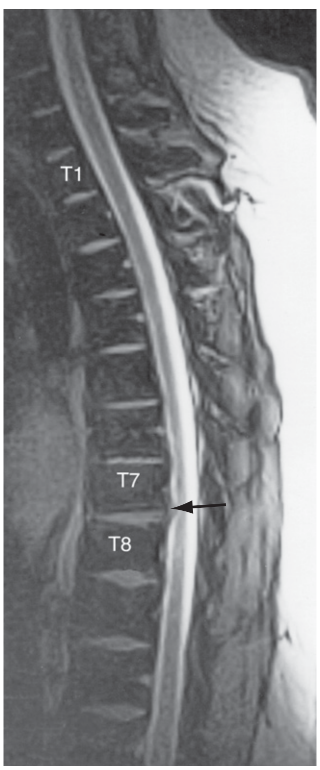

Figure 82.2 Thoracic spine MRI sagittal T2-weighted image showing the 'small anterior extradural defect at approximately the T7–8 level' probably representing a small area of central disc protrusion (arrow).

This probably represents a small area of disc protrusion. This protrusion indents the anterior surface of the dural tube (Fig. 82.3). The visualized portions of the cord are normal. There are no other findings of significance.'

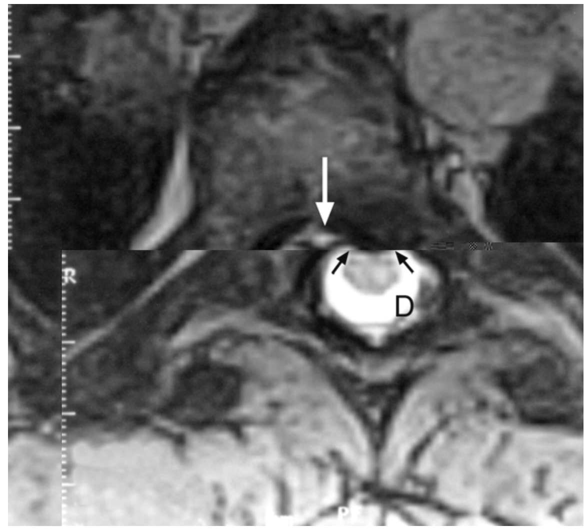

Figure 82.3 Thoracic MRI axial T2-weighted image through the T7–8 level showing that the 'protrusion (arrow) indents the anterior aspect of the thecal sac' (small arrows). D = dural tube. A similar posterior disc protrusion from a postmortem specimen is shown in a histological section in Figure 82.4.

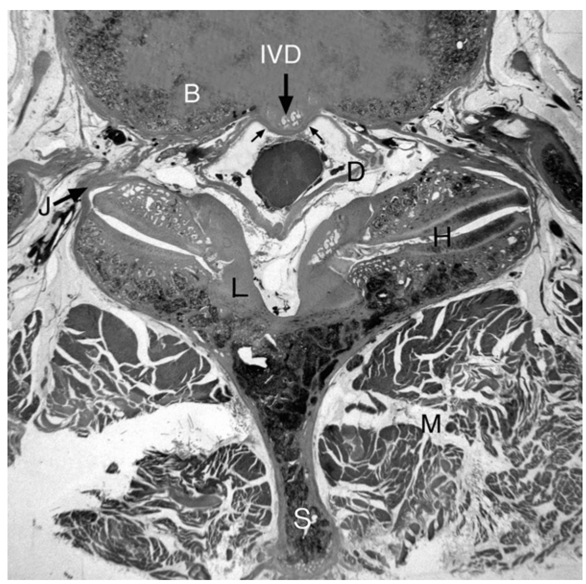

Figure 82.4 Histological axial view, from postmortem material at the intervertebral foramen level showing a central posterior disc protrusion (arrow) similar to this patient's. Note the indentation small (arrows) of the anterior aspect of the dural tube (D). B = blood vessel forming part of Batson's venous complex; H = hyaline articular cartilage on the zygapophysial joint facet surfaces; IVD = intervertebral disc; J = joint capsule (fibrous portion); L = ligamentum flavum; M = muscles; S = spinous process.

DIAGNOSIS

T7–8 level posterior central disc protrusion.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. The patient was told that he had a physical condition and not a psychosomatic one.

He was advised (i) not to perform heavy manual work or to play any contact or jarring sports, (ii) to swim in order to obtain exercise, and (iii) to lose weight in order to lessen the load on his spine. He was told that he could trial spinal mobilization or acupuncture to see if that would help his symptoms and that surgery would only be an option if his symptoms became unbearable in view of the considerable risks associated with surgery. He said that now that he knew that he had a physical problem, and knew what it was, he would take the abovementioned advice regarding changing his work, sports activities and losing weight as he said he could deal with the problem positively and 'get on with life' now that his condition had been explained to him.

KEY POINT

Thoracic spine disc bulges/protrusions should always be considered in cases of thoracic spine pain (as should be 'frank' pathology and rib head osteoarthrosis or dysfunction).

Case 83

Pancoast's tumour

COMMENT

Beware of joint pain that cannot be reproduced or aggravated by joint movements designed to strain the joint.

PROFILE

A 72-year-old unmarried retired stockman of short stature. He smokes approximately 25 cigarettes per day and drinks beer.

PAST HISTORY

No unusual conditions and he said he had 'always been fit and healthy' until being knocked over by a motor vehicle 1 year ago.

PRESENTING COMPLAINT(S) (Fig. 83.1)

Pain on the left side of his neck with radiation to behind the left eye causing 'dull and constant' headaches with intermittent 'acute' headache episodes. Pain at the cervico-thoracic junction extending to approximately the T4 level. Pain in the right shoulder that is only slightly aggravated when he moves his right arm across his chest; the shoulder was not painful on any other active movements of the shoulder joint.

On turning his head to the left side he feels 'dizzy', although he does not feel he will lose his balance but rather that 'objects move in front of the eyes'.

AETIOLOGY

He was knocked over by a motor car 1 year ago but did not require hospitalization.

EXAMINATION

The deep tendon reflexes in the upper and lower extremities were normal as was the case with pinprick sensation. Vibration sensation at the ankles and elbows was normal. Cervical spine left rotation was limited by approximately 50% due to left cervical and cervico-shoulder pain and 'dizziness'. Passive right shoulder movements were of full range and painless; there was minor pain on active right arm internal rotation.

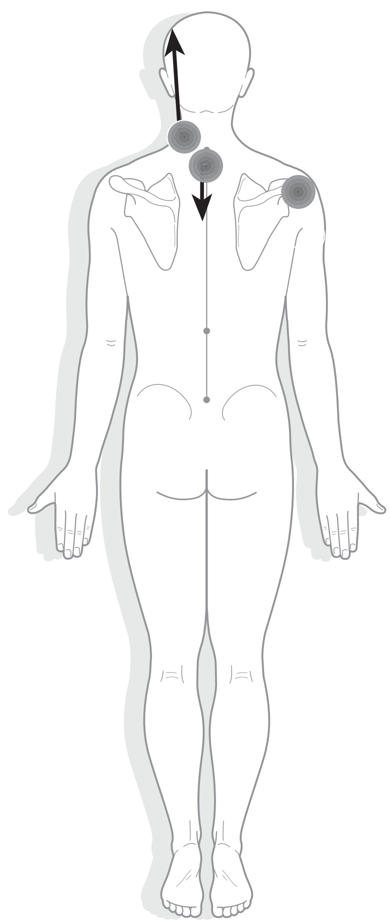

Figure 83.1

IMAGING REVIEW

The patient did not have any previous imaging.

CLINICAL IMPRESSION

Cervical spine spondylosis. Referred pain in the right shoulder – query Pancoast's tumour in right lung.

WHAT ACTION SHOULD BE TAKEN?

In view of his symptoms a series of cervical and thoracic spine films was requested as well as chest posteroanterior (P-A) and lateral films. The cervical spine films showed: 'extensive spondylitic changes throughout the cervical spine with disc narrowing and marginal osteophyte formation; disc narrowing is most severe at the C3–4 and the C6–7 levels and the marginal osteophyte formation is also most prominent at these levels' (Fig. 83.2A). The thoracic spine X-ray report read: 'No destructive bone lesions are identified. There are minor degenerative osteophytes forming at all thoracic levels. The disc spaces appear satisfactory' (Fig. 83.2B). The chest P-A film (Fig. 83.2C) and lateral film showed: 'A Pancoast's tumour in the right lung apex extending down to the right superior mediastinum, widening the mediastinum slightly with pleural thickening and a small effusion at the base of the right lung.' A CT scan of the chest, to better assess the right apical lung mass, was immediately ordered (Fig. 83.2D). A plain X-ray was taken to look at his ribs but there was no radiological evidence of metastatic disease. However, a whole body bone scan was performed (Fig. 83.2E) and this showed increased tracer uptake at the following sites: T10 vertebral body, left 5th and 6th ribs at the costochondral junction, the right 6th rib anteriorly, the right wrist, and increased tracer activity in the lumbar spine.

The conclusion was: 'Increased activity at T10 is non-specific. It could represent bony metastasis or an osteoporotic crush fracture. Plain film correlation in the first instance is recommended. Increased rib activity is most likely metastatic; however in this location it could be traumatic. The right wrist activity is most likely degenerative. Plain film correlation is recommended'.

DIAGNOSIS

- Pancoast's tumour in the right lung apex with metastatic disease.
- Cervical spine spondylosis.

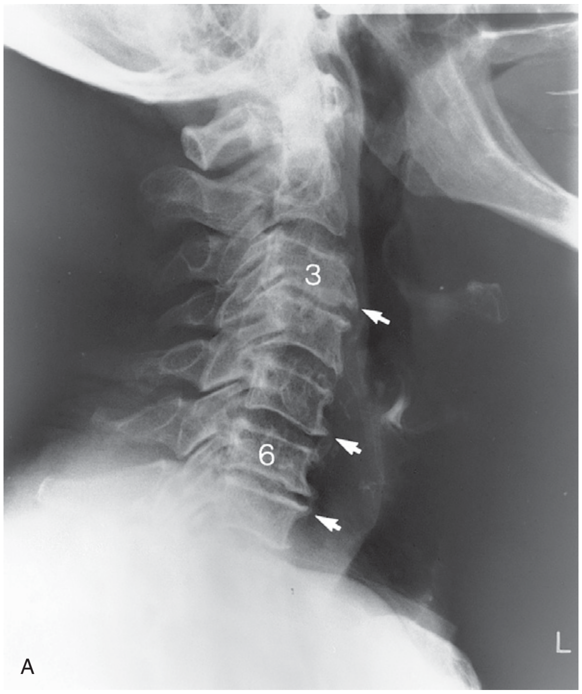

Figure 83.2 (A) Cervical spine lateral plain X-ray image showing 'extensive spondylitic changes throughout the cervical spine with disc narrowing and marginal osteophyte formation. The disc narrowing is most severe at the C3–4 and the C6–7 levels and the marginal osteophyte formation is also most prominent at these levels' (e.g. white arrows). (B) An anteroposterior thoracic spine plain X-ray view showing minor degenerative osteophytes forming at all thoracic levels and a suspicion of a space-occupying lesion in the right lung apex.

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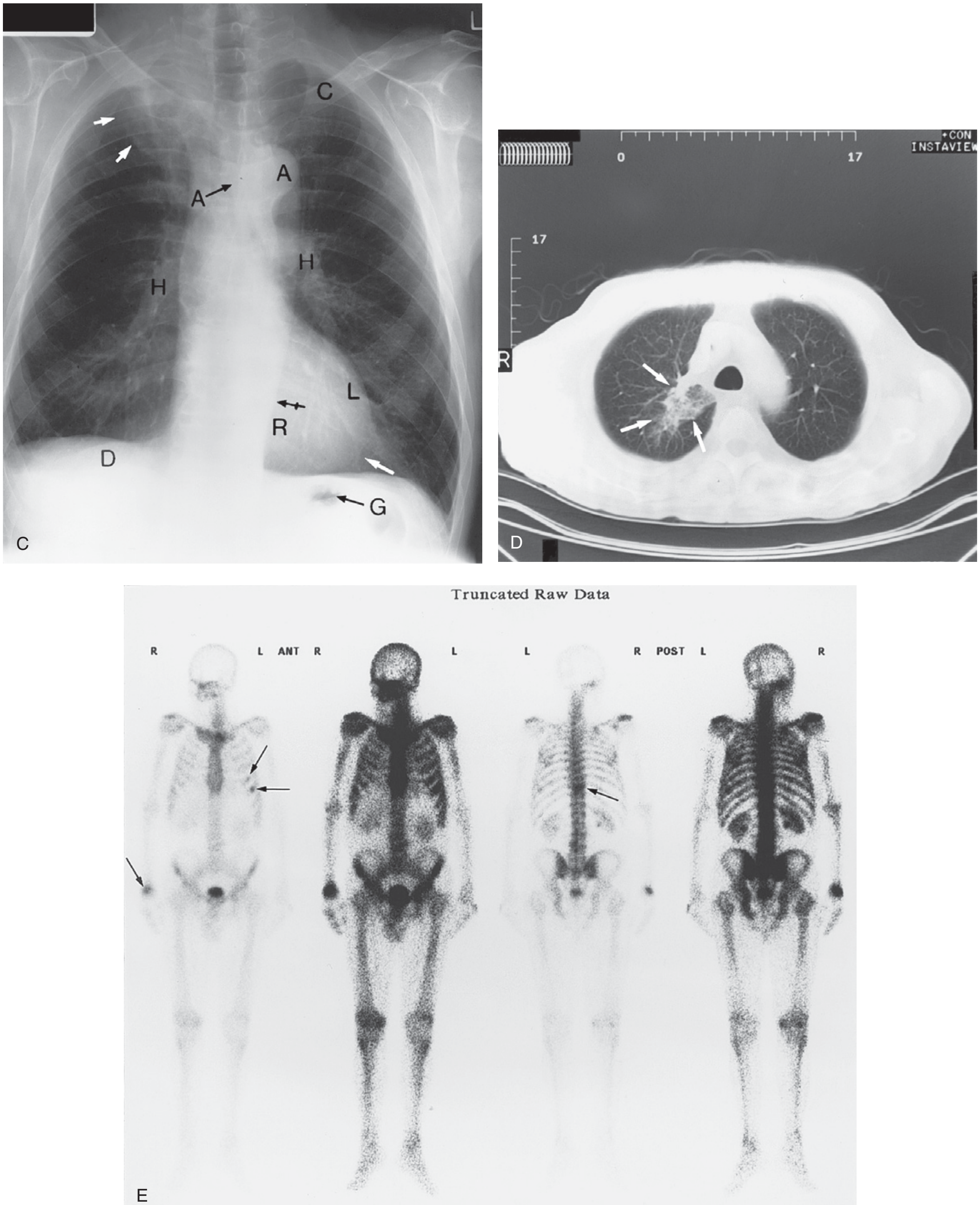

Figure 83.2 Cont'd (C) A chest posteroanterior plain X-ray view that clearly shows the Pancoast's tumour (white arrows show the approximate border) in the apex of the right lung. A = aortic knuckle; D = dome of right diaphragm; A arrow = aortic arch; L = left ventricle; H = hilar shadow; C = clavicle; single white arrow = apex of the heart; tailed black arrow = descending thoracic aorta; G = gas in the upper part of the stomach ('magenblase'). (D) A CT scan of the chest showing the Pancoast's tumour on the right side (arrows). (E) Whole body bone scan truncated raw data. Arrows show areas of increased tracer uptake.

TREATMENT AND RESULT

This man was referred to the oncology department where biopsy confirmed the diagnosis of a Pancoast's tumour. He had radiotherapy but it was not successful and his symptoms became worse with the development of severe pain in his right shoulder and upper arm, particularly on the medial aspect, and he complained of allodynia (pain resulting from a non-noxious stimulus to normal skin) and hyperpathia (abnormally exaggerated subjective response to painful stimuli) which made it very difficult for him to sleep at night. The neuropathic pain that he developed was most likely related to the involvement of his T1 nerve root and perhaps sympathetic chain at the apex of his right lung. He remained in hospital on various analgesics that were given in an attempt to relieve his symptoms. However, the result was that, when he was actually pain free, he was completely asleep.

Chemotherapy was not helpful and he passed away within 3 months of the original diagnosis.

KEY POINTS

1. When a patient presents with joint pain that cannot be reproduced by passive or active joint movement, one must consider the possibility of referred visceral pain.
2. Remember that a Pancoast's tumour may damage the brachial plexus as carcinoma of the lung apex extends through the apical pleura (Patten 1996).
3. Pancoast's tumours must be considered in the differential diagnosis of patients presenting with neck and upper extremity pain as well as neurological symptoms (Clark 1997).

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Case 84

Zygapophysial joint facet fracture

COMMENT

Always look carefully at the imaging and not just at the report.

PROFILE

A 43-year-old male executive of muscular build.

PAST HISTORY

He had always been healthy and fit and he had no recollection of trauma apart from a motorbike accident approximately 10 years ago following which he was not immediately aware of this pain.

PRESENTING COMPLAINT(S) (Fig. 84.1)

A 10-year history of chronic mid to lower thoracic spine pain that radiates to the left side of his chest as far as the mid-clavicular line, often associated with paraesthesiae and occasional pain referral to his left arm. He initially felt pain and 'pins and needles' at approximately the T8 level, then the left sided chest pain occurred during long drives in the car. The pain intensity varies depending on what he does. He now experiences recurrences of the pain after short drives in the car and he finds this very frustrating.

He was referred with an extensive background of investigations including MRI studies of the thoracic spine. He had also had stress tests performed to rule out a cardiac problem; there was no indication of heart problems. He had also been investigated for an oesophageal/gastric cause and a pulmonary cause, all to no avail. There was no night pain. Coughing and sneezing were painless. He was frustrated and depressed as the cause of his pain could not be found.

AETIOLOGY

Possible motorbike injury approximately 10 years ago.

EXAMINATION

On deep palpation of the paraspinal muscles he was tender over the T6–10 level and there was increased tone of the paraspinal muscles. The deep tendon reflexes in the upper and lower extremities were normal, as was the case with pinprick sensation. Vibration sensation at the ankles and

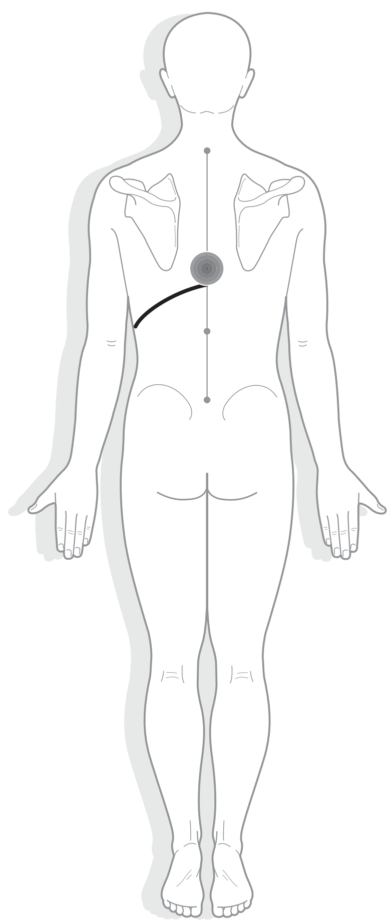

Figure 84.1

elbows was normal. Active ranges of thoracic spine movement were limited by approximately 20% in forward bending and in left and right lateral bending.

IMAGING REVIEW

Plain X-ray films showed minor degenerative lipping at the T6–9 vertebral bodies anteriorly. The MRI thoracic spine was non-contributory.

CLINICAL IMPRESSION

'Mechanical' spinal pain as the pain is reproducible on thoracic spine flexion and left and right lateral bending. There is no night pain to suggest a serious pathology and MRI had not shown any disease process or space occupying lesion.

WHAT ACTION SHOULD BE TAKEN?

Bearing in mind that an MRI study can be less useful than a CT scan for looking at bones, a CT scan from T3 to T10 was requested; this was reported as showing 'minimal degenerative change in the rib articulations at T8 and T9 with small anterior vertebral osteophytic change at the T8–9 level'. However, on carefully reviewing the CT slice images, it was noted that the left zygapophysial synovial joint facet at the T8–9 level was abnormal and probably represented a fracture of the superior articular process facet of T9 (Fig. 84.2). As a result, an isotope bone scan was performed of the thoracic spine and this showed 'mildly increased isotope accumulation in the region of the left T8–9 facets, typical of degenerative or post-traumatic changes'.

DIAGNOSIS

T8–9 left zygapophysial fracture involving the superior articular process of T9.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was greatly relieved to find that the cause of his problem was benign and that there was not an underlying more sinister pathological reason for his chronic pain syndrome. Because it was considered that the left T8–9 zygapophysial joint was probably irritating the intercostal nerve root on the left side, he was

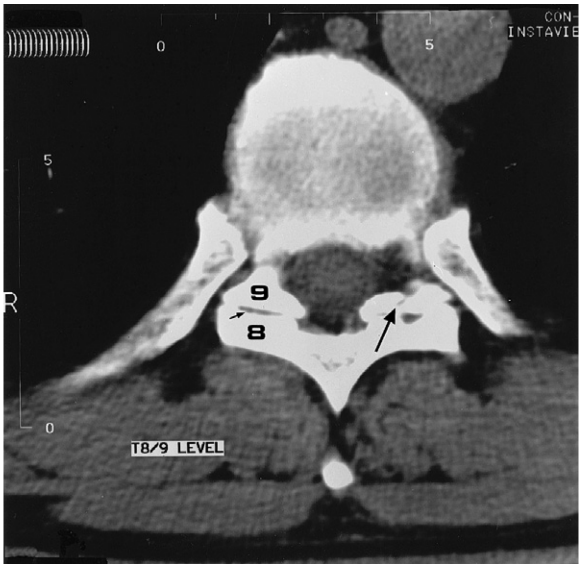

Figure 84.2 Thoracic spine CT axial image at the T8–9 level clearly shows the fracture (large black arrow) of the superior articular process and facet of the T9 vertebra. (For comparison with 'normal' histology, please see Fig. 81.3.) The inferior articular process of the T8 vertebra is shown by the numeral 8 and the superior articular process of the T9 vertebra is shown by the numeral 9. On the right side (R) there is a normal zygapophysial (facet) joint with hyaline articular cartilage (small arrow) between the facet surfaces.

sent for an orthopaedic opinion. The orthopaedic surgeon agreed with the diagnosis and suggested a facet block injection.

The facet block injection gave considerable pain relief. He was left with a painful 'trigger point' in the mid-axillary line after prolonged sitting, so an anaesthetic with steroid injection into the trigger point was performed; this gave him dramatic relief. He later required a further facet block injection for the left T8–9 zygapophysial joint and he was very satisfied with the result.

KEY POINTS

1. Zygapophysial (facet) joint pain can occur due to injury of these joints as various structures, such as the joint capsule and the synovial folds, have nociceptors in them.
2. Inflammation of these joints may cause joint capsule distension resulting in pressure on the adjacent nerve root or the spinal nerve itself.
3. Be wary of pain that appears to be mechanical in nature but is actually due to osseous spinal injury.

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Case 85

Osteoporosis

COMMENT

Repeat imaging, when dictated by the history, symptoms and signs, should be considered even when previous images are relatively recent.

PROFILE

A retired 68-year-old overweight male who was perspiring at consultation.

PAST HISTORY

His past medical history included an 11-year history of low back pain for which he had undergone decompressive L4–5 laminectomy that failed to give him relief and exacerbated his symptoms. Therefore, he underwent a coccygectomy but this did not help his symptoms either. He had had a spinal cord stimulator (epidural catheter) implanted in an attempt to control his chronic low back pain but this did not produce any worthwhile pain relief. Therefore, the electrodes were repositioned, with the most superior tip being placed around the thoracolumbar junction. He also has a 14-year history of Crohn's disease, hypertension and chronic, stable angina.

PRESENTING COMPLAINT(S) (Fig. 85.1)

His consultation was specifically only for constant and severe chronic mid to lower thoracic spine pain which is not associated with any upper limb symptoms. Approximately 12 months ago he suddenly experienced acute thoracic pain that lasted for approximately 4 months then recurred, prompting his consultation.

Movement aggravates his thoracic spine pain but the pain does not awaken him at night. Coughing aggravates

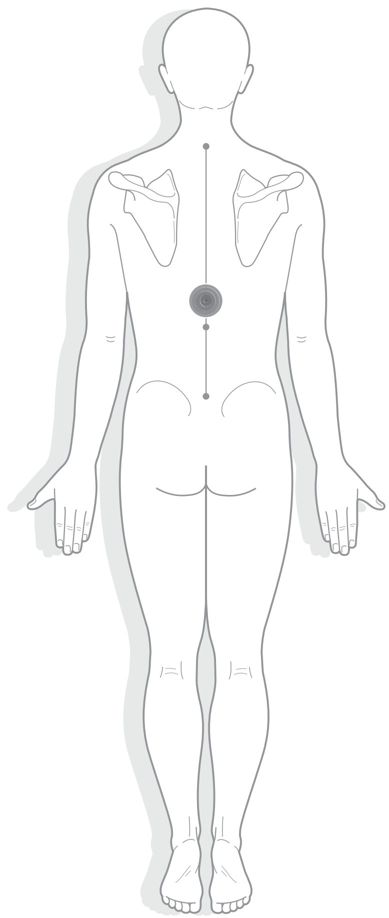

Figure 85.1

his pain, as does changing position. He had tried narcotic analgesia but this apparently 'caused memory loss' so he stopped taking it.

AETIOLOGY

Unknown.

EXAMINATION

He is considerably overweight and somewhat hypertensive with a blood pressure of 170/90. Examination of the cardiovascular system was otherwise unremarkable. The deep tendon reflexes in the lower extremities were normal apart from an absent right knee jerk. Pinprick sensation of the lower limbs was unremarkable. There was slight tenderness on palpation from the lower thoracic region to the sacrum.

IMAGING REVIEW

Thoracic and lumbar spine plain film radiographs taken approximately 3 years previously showed moderate compression of the T6 body and some compression of the superior margin of the T12 body, both being consistent with past trauma. Slight degenerative osteoarthritic changes were present in the mid-thoracic spine. The spinal cord stimulator was noted from the T9 level down; this had been inserted in an attempt to control his low back pain.

CLINICAL IMPRESSION

Possible mid-thoracic spine vertebral body compression fractures due to osteoporosis. Differential diagnosis to include bone lytic disease.

WHAT ACTION SHOULD BE TAKEN?

New plain film thoracic spine radiographs were ordered and showed 'osteoporosis with severe loss of height in the T6 and T7 vertebral bodies and associated degeneration of the T7–8 disc as evidenced by gas in this disc space. The upper margin of a spinal cord stimulator is noted at the T9 level (Fig. 85.2A and B). A CT thoracic spine scan was ordered from T5 to T9. This showed: 'Loss of T6 and T7 vertebral body height is associated with marginal osteophyte formation and on the bone windows the cortex of these bones remains intact suggesting that these changes are due to previous trauma rather than to an infiltrative and destructive bone process'.

A three-phase bone scan was ordered as a precaution to exclude lytic disease and this showed 'diffusely increased tracer uptake in the midthoracic spine at approximately T6–8 but the plain X-ray films suggest that this is not due to metastatic disease'.

Laboratory tests were performed as a precaution (ESR, serum alkaline phosphatase and serum calcium) and were within normal limits.

DIAGNOSIS

Osteoporotic crush fractures of the T6 and T7 vertebral bodies.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. As the patient had tried various types of medication, and the spinal cord stimulator had been inserted for his low back pain, it was decided that a trial of needle acupuncture may be helpful as an initial attempt to stop his chronic mid-thoracic spine pain but this was of no help. Therefore, a right T6–7 'facet' joint block injection was arranged under CT control (Fig. 85.2C) as this was the main level for his pain on deep palpation.

The facet joint block injection resulted in a good response for his thoracic spine pain. He was advised to try taking paracetamol if symptoms dictated the need for analgesic medication periodically. The right T6–7 facet block injection accurately localized the source of his mid-thoracic spine pain and was useful as a diagnostic and therapeutic procedure. This injection gave relief for approximately 2 years then he returned for a further evaluation. At this time he was advised to lose a considerable amount of weight and to have a further facet block injection at the same level. On that occasion, he received less effective pain control so he decided to take paracetamol periodically for pain relief.

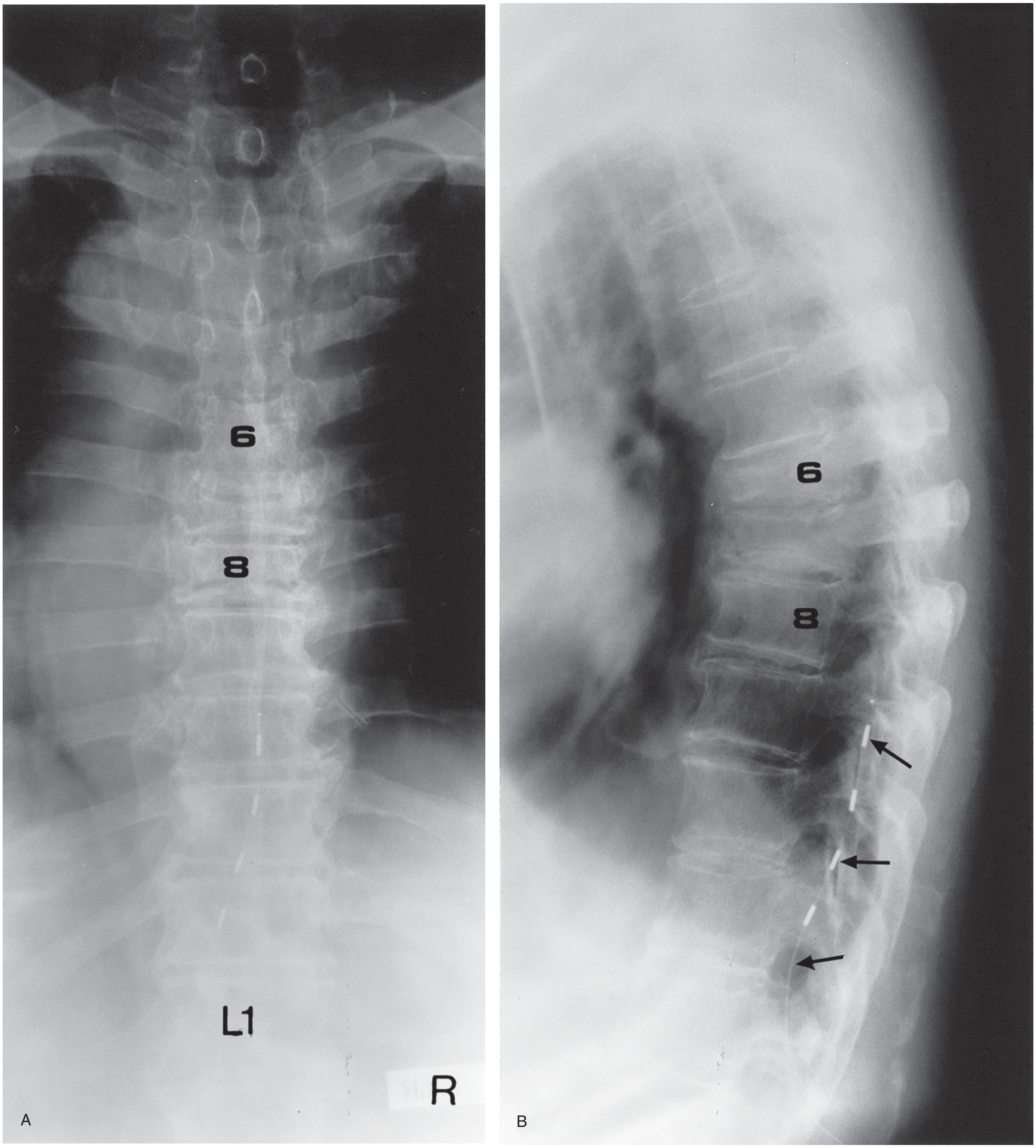

Figure 85.2 Plain film radiographs of the thoracic spine in (A) the anteroposterior and (B) lateral projections. Note the osteoporosis within associated significant vertebral body crush fractures at the T6 and T7 levels. Also note the gas in the T7–8 disc and the epidural spinal cord catheter terminating at the level of T9 (see arrows).

(Continued)

Figure 85.2 Cont'd (C) Thoracic spine CT scan at the T6–7 zygapophysial 'facet' level showing needle placement for the facet block injection. R = right side of patient. Note the marginal osteophyte formation on the vertebral body (small white arrows). The dural tube shows an abnormally narrowed configuration behind both the T6 and T7 vertebral bodies but the cause is not evident and there is generous fat around the dural tube within the bony spinal canal.

KEY POINTS

1. Osteoporosis is a clinical condition characterized by decreased skeletal bone mass in which the bone is otherwise normal (Kostuik & Heggeness 1997).
2. Common fracture sites in patients over 60 years of age are in the hip, proximal tibia, wrist and the thoracic and lumbar spines (Kostuik & Heggeness 1997).

Reference

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Case 86

T10–11 intervertebral disc posterior protrusion

COMMENT

It is a fallacy to believe that, once a patient receives a significant cash settlement following litigation for a work-related injury, the patient's symptoms will automatically disappear. This case is just one of many examples of symptoms persisting following settlement of a work-related claim.

PROFILE

A 43-year-old, somewhat overweight, married man who has worked as an electrician for approximately 28 years. He does not smoke cigarettes and only drinks alcohol socially.

PAST HISTORY

He had a tonsillectomy at approximately 6–7 years of age. He broke the right tibia at approximately 12 years of age. As an adult, he injured his right knee while playing basketball and required two surgical procedures to rectify this problem. Seven years ago he suffered some transient cervical and upper thoracic spine strain while carrying a heavy electrical object. His general medical practitioner sent him for cervical and thoracic spine X-ray films but they did not indicate any skeletal abnormality.

Six years ago he was working in a ceiling, in a twisted position, when the ladder on which he was standing slipped, causing him to become jammed in the ceiling. This incident caused some transient bruising in the thoracic spine and around his ribs but no long-lasting sequelae.

PRESENTING COMPLAINT(S) (Fig. 86.1)

Constant pain in the lower thoracic spine at approximately the T10 level which varies in intensity depending on activity. He experiences some radiation to the ribs bilaterally from T10; sometimes this will go to the front of the rib cage when severe.

Coughing, sneezing or bearing down increase the pain at T10. If he rakes the yard, he experiences an increase in his pain. He can no longer play basketball, golf or cricket because of his pain. On getting into a motor vehicle, the twisting action can aggravate the T10 level pain.

The pain does not wake him up at a particular time during the night and it is not a deep-seated bone pain. However, he does wake up in the early hours and has to move around periodically during the night to obtain some relief. Heat from the hot shower gives temporary relief. When he arises in the morning, the pain is more noticeable.

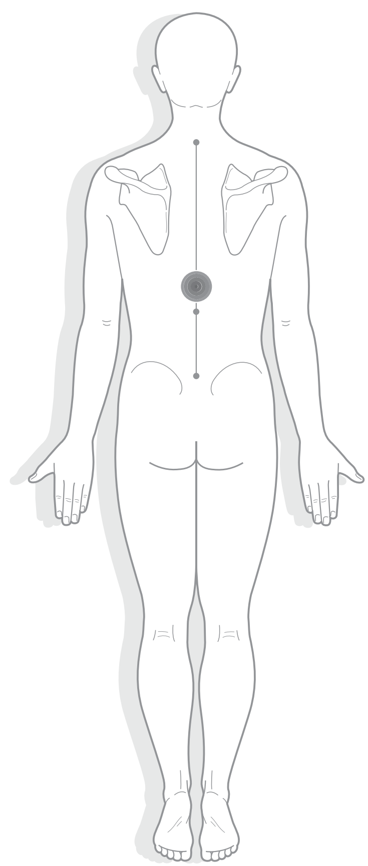

Figure 86.1

He has tried anti-inflammatory medication, physiotherapy and local anaesthetic and cortisone injections with no relief.

AETIOLOGY

Six years ago he moved a heavy (approximately 65 kg) battery for a locomotive from the back of a utility vehicle. As he slid the battery forward with his left arm stretched forwards, while trying to keep the trolley still with one foot, the battery slid off the back of the utility and caught the top edge of the trolley with the result that it fell. He instinctively grabbed it as he had previously seen a battery explode when someone dropped it. He immediately felt pain in the T10 region and some pain in the right buttock and leg.

EXAMINATION

In the erect posture, there was no evidence of pelvic obliquity or scoliosis. Percussion of the spine elicited some tenderness at approximately the T10 level. The sacroiliac joint strain test did not elicit any sacroiliac joint pain. Deep palpation of the paraspinal muscles elicited pain at the T10 level. Toe walking power (S1) and heel walking power (L5) were normal but the latter caused some ‘jarring’ at approximately T10. Deep tendon reflexes in the upper and lower extremities were normal. Pinwheel sensation of the upper and lower extremities, as well as the posterior aspect of the torso, was normal. Vibration sensation at the elbows and ankles was normal.

Active ranges of spinal movement were as follows.

Thoracic spine

1. Flexion – full range and painless.
2. Extension elicited some pain at approximately T10 at full range.
3. Left lateral bending elicited some pain at T10 at full range.
4. Right lateral bending elicited pain at T10 with approximately 20% restriction of mobility.
5. Left rotation – limited by approximately 10% due to T10 pain.
6. Right rotation – limited by approximately 30% due to T10 pain.

Cervical spine

1. Flexion – at full range elicited some pain and ‘tightness’ at approximately T10.
2. Extension – full range and painless.
3. Left and right lateral bending elicited pain at approximately T10 at full range.
4. Left and right rotation – full range and painless.

Lumbar spine

1. Flexion – finger tips reached to within 5 cm of the floor.
2. Extension elicited some pain at T10 at full range.
3. Left lateral bending – full range but caused T10 pain.
4. Right lateral bending – limited by approximately 20% due to T10 pain.
5. Left rotation – limited by approximately 10% due to T10 pain.
6. Right rotation – limited by approximately 30% due to T10 pain.

The Valsalva manoeuvre (bearing down) elicited an increase in pain at the T10 level.

IMAGING REVIEW

Original plain thoracic spine X-rays reported: ‘No skeletal abnormality is demonstrated’. Plain thoracic spine X-rays taken 2 years later reported: ‘Minor degenerative spurring at one or two levels’ of the thoracic spine with ‘minimal early spurring’ in the lumbar spine. A further thoracic spine plain X-ray film examination taken 4 years later reported: ‘Mild degenerative changes are present in the thoracic spine with marginal osteophytosis and loss of disc height at multiple levels. Focal loss of disc height seems most marked at the T10–11 level . . .’.

A thoracic spine CT scan reported: ‘Some minor degenerative changes . . . with vertebral body anterior osteophytosis and some minor degenerative changes at the costovertebral joints. A Schmorl’s node is present in the superior endplate of T8’.

An MRI thoracic spine scan showed Schmorl’s nodes from T6 to L1 (Fig. 86.2A); because of their frequency it was considered that they may be associated with a congenital developmental anomaly known as persistent notochord (which can remain asymptomatic unless injury is superimposed). It also showed ‘some encroachment upon the thoracic canal, slightly eccentrically towards the left side at T10–11, by a disc protrusion’ (Fig. 86.2B).

CLINICAL IMPRESSION

T10–11 posterior disc protrusion.

WHAT ACTION SHOULD BE TAKEN?

No further imaging was necessary. The patient was reassured that his pain was genuine and he was greatly relieved to find out that there was indeed a reason for his spinal symptoms. He was advised not to lift heavy weights or bend his spine into awkward positions during work and he was advised to perform only light duties. He was referred for an orthopaedic opinion, which confirmed the T10–11 disc protrusion and associated symptoms but surgery was not considered as an option at that time.

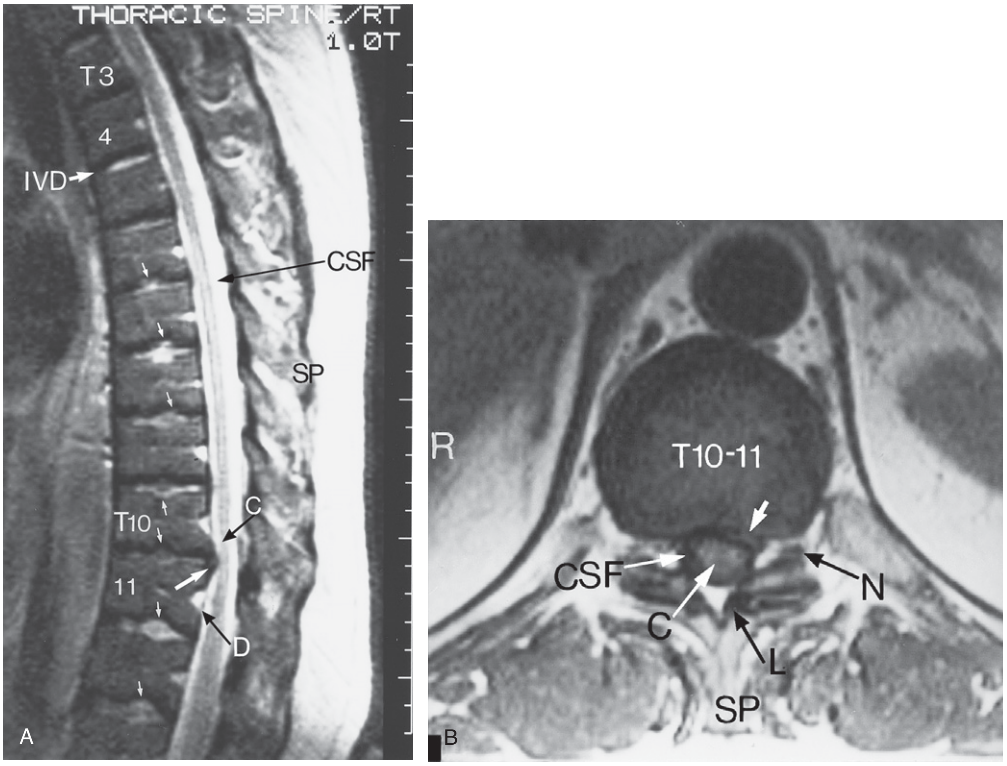

Figure 86.2 (A) MRI thoracic spine sagittal T2-weighted image which shows the Schmorl's nodes from T6 to T12 (small white arrows). Note the T10–11 disc protrusion compressing the anterior part of the dural tube (D) and apparently touching the spinal cord (C) which lies within the dural tube surrounded by cerebrospinal fluid (CSF) that is white on the T2-weighted MRI image. IVD = disc with normal hydration (as compared to the T3–4 disc that shows desiccation (blackening); SP = spinous process. The numerals 3, 4, 10 and 11 indicate thoracic vertebral bodies. (B) Thoracic spine MRI axial T1-weighted image through the T10–11 intervertebral disc showing the left sided disc protrusion (small white arrow) that impresses upon the dural tube with some distortion of the spinal cord (C) itself as it lies within the cerebrospinal fluid (CSF) that appears as a dark area around the cord on this T1-weighted image. L = ligamentum flavum; N = spinal ganglion within the intervertebral foramen; SP = spinous process.

DIAGNOSIS

T10–11 left sided posterior disc protrusion.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was informed that he may require surgery, in due course, in spite of doing sedentary work, but that this should not be undertaken lightly as this type of surgery has a high risk. It was suggested that it would be better for him to keep fit by going to a gymnasium and to spend time swimming, to see if this would help him to maintain his present levels of activity As Panadeine Forte caused side effects, such as constipation, he was referred back to his general medical practitioner to consider a simple analgesic such as paracetamol instead.

The advice provided gave considerable relief but, as symptoms still persisted 5 years after settlement of his work injury claim, he returned for a further consultation. That consultation led to the MRI scan seen in Fig. 86.3, which clearly showed that he still had a T10–11 disc protrusion pressing upon the dural tube and touching the spinal cord.

He was referred for a neurosurgical opinion with the result that his condition was confirmed but, again, surgery was not contemplated due to risk factors. He manages his pain with paracetamol, exercise and by performing only light duties.

Note

This is only one of many cases where a patient went through the process of litigation for a work-related injury, but his symptoms persisted after a significant cash settlement.

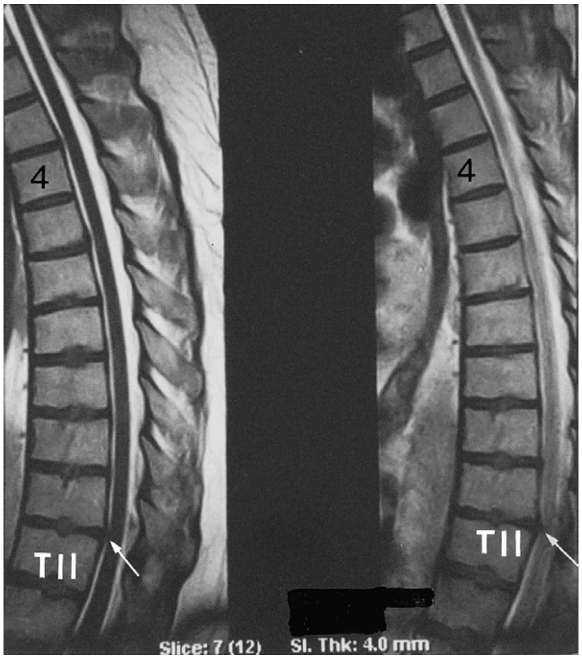

Figure 86.3 These two T2-weighted sagittal MRI images of the thoracic spine were taken 5 years after the scan shown in Fig. 86.2A. Note that the T10–11 disc protrusion is still present (arrows).

KEY POINTS

1. Thoracic spine disc protrusions are notoriously difficult to diagnose.
2. Unfortunately, it is often thought that individuals with work-related disability exaggerate their complaints and suffer from psychiatric disorders such as depression and neuroticism only until they receive a 'curative' financial settlement, at which time they are able to return to work (Margoshes & Webster 2000), even though recent studies have refuted this concept (Mendelson 1982, Burns et al 1995).

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Case 87

Burst fracture

COMMENT

Beware of patients who apparently sustain a seemingly simple vertebral body compression fracture.

PROFILE

A 41-year-old male of muscular build who currently does not work as a result of a motorbike accident. He does not smoke and only drinks alcohol socially.

PAST HISTORY

He said he had not suffered from any unusual childhood illnesses or unusual adult illnesses and had experienced 'perfect health all [his] life' prior to a motorbike accident that occurred 12 months ago. He said he had always kept active and that this had kept his muscles well toned.

PRESENTING COMPLAINT(S) (Fig. 87.1)

Constant pain, that varies in intensity, at approximately the T12 area; this pain can go 'up and down' a few segments and it radiates bilaterally around the rib cage to the lower sternum.

Coughing and sneezing significantly increase his pain, which is activity related. Bearing down also increases this pain. He says he has great difficulty sleeping at night as his thoracolumbar region is very painful and he finds this is distressing as he has to 'toss and turn all night'. He says he has some pain intermittently that radiates into the right lower abdomen and then into the right testicle; he believes this pain comes from the thoracolumbar region.

He had never had any of the above pains before the motorbike accident. He does not take medication as he is wary of possible side effects.

AETIOLOGY

He was riding his motorbike toward a roundabout when he was knocked off the motorbike when a car hit him at speed. During the accident, he bounced off the bonnet of the car and over the windscreen then ended up lying in the street.

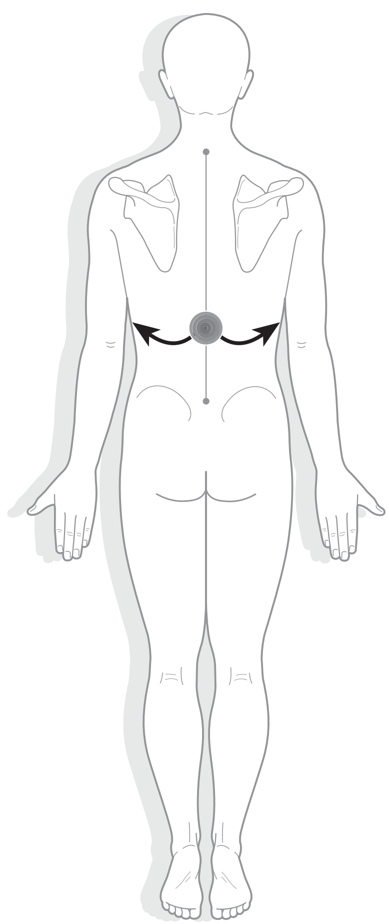

Figure 87.1

EXAMINATION

In the erect posture, there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic and lumbar spines did not indicate any particular tenderness. Deep palpation of the paraspinal muscles elicited pain bilaterally in the cervical spine at the C4–7 level and at the thoracolumbar junction region.

The deep tendon reflexes in the upper and lower extremities were normal, as was the case with vibration sensation at the elbows and ankles. Pinprick sensation over the arms, hands and legs was normal but there was some hypoaesthesia on the medial aspect of his left foot (L5). Motor power in the upper and lower extremities was normal, as was the case with the foot pulses. The temperature of both feet appeared to be equal and normal on palpation. The superficial upper abdominal reflexes (T7–10) and lower abdominal reflexes T10–L1 appeared to be normal.

Active thoracic spine ranges of movement were all limited by 50% due to 'stiffness' and, on flexion, he moved with a lumbar 'flat back' due to protective muscle splinting.

IMAGING REVIEW

Plain X-ray films showed what appeared to be a simple uncomplicated compression fracture of the T12 vertebral body (Fig. 87.2A) with anterior wedging.

CLINICAL IMPRESSION

T12 burst fracture.

WHAT ACTION SHOULD BE TAKEN?

A thoracic spine CT scan was performed. This showed that the fracture was much more serious, with a burst fracture of the body of T12 vertebra with considerable retropulsion of a posterior bony fragment into the spinal canal causing a reduction of cross-sectional area of approximately 35% (Fig. 87.2B).

He saw an orthopaedic surgeon who advised him not to undergo surgery and, as he was coping well with his condition, this view was supported.

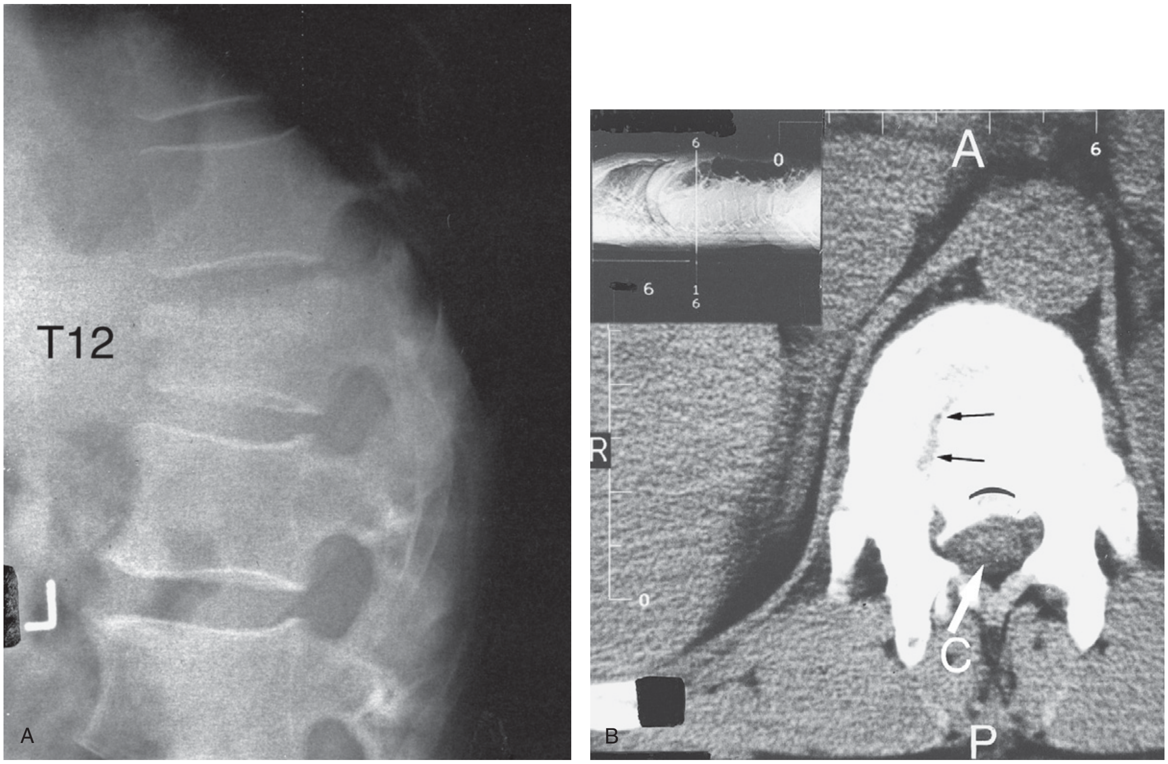

Figure 87.2 (A) Thoracic spine lateral plain X-ray image showing the compression fracture of the T12 vertebral body with anterior wedging and disruption of George's line. (B) Thoracic spine CT axial image through the T12 vertebral body that shows the burst fracture of the body of the T12 vertebra, with considerable retropulsion of a posterior bony fragment into the spinal canal causing a reduction of cross-sectional area of approximately 35%. Small arrows show part of the fracture line. The retropulsed posterior fragment in the spinal canal lies posterior to the semicircular black line that represents the approximate area at which the anterior part of the spinal canal would normally be seen. A = anterior; C = spinal cord; P = posterior; R = right side of patient. The insert shows the level of the axial scan slice.

DIAGNOSIS

T12 burst fracture with vertebral body compression.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

He was advised not to take part in any excessively flexed, extended or rotated spine activities that would place heavy loads on his spine. He was also advised to swim as much as possible to maintain muscle tone as he already had a good physique. In addition he was told to seek immediate orthopaedic or neurosurgical advice should he experience any symptoms such as leg pain or weakness, bowel or bladder dysfunction, or any increase in his current symptoms. An MRI scan was considered but was declined at this time as surgery was not planned.

He continues to remain fit and active and has managed to adapt to his injury with resulting impairment.

KEY POINTS

1. The thoracolumbar junction (T11–L2) is the commonest site for spinal fractures (El-Khoury & Brandser 1997).
2. When a vertebral body compression fracture appears uncomplicated on lateral plain X-ray films, remember that a burst fracture may be present with bony fragment(s) in the spinal canal.

Reference

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Case 88

Thoracic vertebral body fracture

COMMENT

Individuals can experience serious osseous and musculoligamentous injuries due to motor vehicle accidents and should be taken seriously following such accidents.

PROFILE

A slim 19-year-old female office worker who does not smoke cigarettes and only drinks alcohol very rarely.

PAST HISTORY

Four months ago she was the seat-belted driver of a small car and, as she was slowing her vehicle to stop, without any warning her car was subjected to a 'huge' rear end impact from another vehicle at approximately 80 km per hour. Her head and neck were jerked about and, when her head hit the steering wheel, she was knocked unconscious. Her seat back collapsed backwards and her head hit the side panel in the car as evidenced by some blood and hair on that structure. She was taken by ambulance to hospital where she was examined and X-ray images were taken of her painful cervical and thoracic spines while she lay on the ambulance 'flat back' board. She was given a morphine injection and her occipital cuts were cleaned and stitched. The X-ray images were considered to be 'normal', so she was discharged 5 hours later; as she walked, supported, through the hospital foyer, she collapsed into a chair due to acute mid-thoracic spine pain. She was re-admitted to hospital for approximately 1.5 hours, then allowed to go home; she was advised to see her general medical practitioner within 24 hours which she did and was given analgesics and a non-steroidal anti-inflammatory drug.

PRESENTING COMPLAINT(S) (Fig. 88.1)

Four months following a motor vehicle accident she presented with:

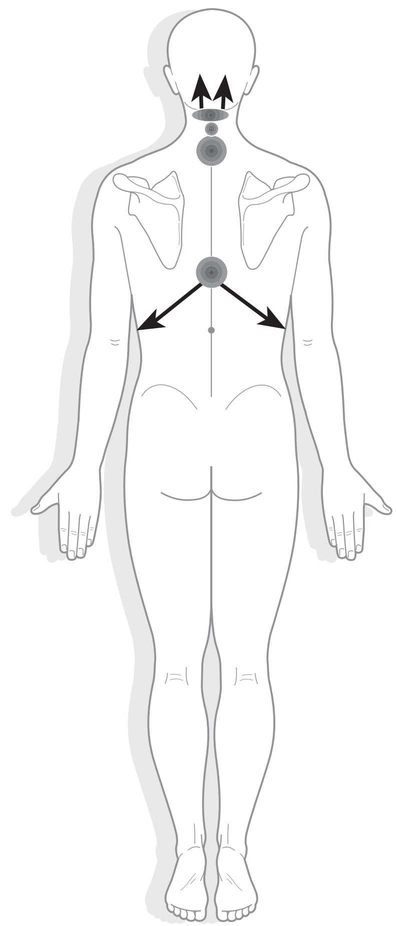

Figure 88.1

1. Constant mid-thoracic spine pain (she points to the T7–8 level of her spine) that varies in intensity from mild to severe; it is activity related i.e. increased activity causes an increase in pain. She also experiences (i) bilateral, but especially right sided, intercostal pain at the T7–8 level, (ii) 'pins and needles' on the left side of her upper thoracic spine and

on the left side of her torso (in the mid-axillary to mid-clavicular line) at approximately the T6–T12 level and pain at approximately the T4 level centrally.

The above symptoms are aggravated by coughing and sneezing, bearing down, work activities, arising from the seated position, being in bed overnight (this necessitates the use of hot shower water in the morning to try to obtain some relief), sitting, particularly if she sits still, bending forward to file documents, filing papers in high or low pigeon holes, twisting her torso to the left or right sides, and sitting up from lying supine (she has to roll onto her side before sitting up).

The above symptoms are relieved by medication (Nurofen), heat, as a result of using hot water from the shower, or a heat pack and bed rest, although she finds that she cannot remain on her back and has to lie on one side to minimize the T7–8 level pain.

2. Intermittent neck pain that occurs daily and is felt in the suboccipital, mid-cervical and cervico-thoracic junction regions; it may radiate to the occiput bilaterally. Approximately once per week she experiences a 'really sore neck plus headache'. Her neck pain is activity related and becomes worse as the day progresses.

   The above symptoms are aggravated by: work, for example bending her neck forwards to look down causes pain to extend from the upper neck region to approximately the T7–8 level; lying prone, therefore she no longer does this; looking behind her when driving; turning her head to the left or right side causes a 'pinching' sensation in the neck that is always accompanied by some 'stiffness'; and bearing down causes bilateral cervico-shoulder pain.

   The above symptoms are relieved by being in bed overnight, heat as a result of using hot water from the shower or a heat pack, and medication; she obtains some relief from Nurofen and rubbing in Deep Heat.

AETIOLOGY

A motor vehicle accident 4 months ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic spine elicited pain at the T1 and T6–8 levels; there was no pain in the lumbar spine. Deep palpation of the paraspinal muscles elicited pain bilaterally, but especially on the right side, at C3–5 and bilaterally at the T1 and T6–10 levels; no pain was elicited in the lumbar spine. Deep palpation centrally between the spinous processes elicited pain at the C4–5, T3–4 and T7–8 levels; no pain was elicited in the lumbar spine. The sacroiliac joint strain test did not elicit any sacroiliac joint strain. Toe walking power (S1) and heel walking power (L5) were normal. The deep tendon reflexes in the upper and lower extremities were normal, apart from a somewhat diminished (one plus instead of two plus) left triceps (C7) reflex. Vibration sensation was normal at the elbows and ankles. Pinprick sensation over the upper posterior part of the torso appeared to be normal as was the case with the upper and lower limbs, apart from an area of hyperaesthesia on the left side of the spine at approximately the T7 to T9 level. The brachial plexus stretch test did not cause any brachial plexus pain. Motor power in the upper and lower extremities appeared to be normal except for left little finger adduction (C8, T1) where the power appeared to be slightly diminished (4/5). When seated slumped forwards there was no low back pain but this caused a 'pulling' and 'stretching' sensation from approximately T4 to T10. Hoffman's sign was normal.

The circumference for the following regions was measured (Box 88.1).

Box 88.1 Forearm and calf circumferences

Circumference	Left	Right	Comments
Forearm (9 cm below the elbow)	21 cm	21 cm	Normal
Calf (10 cm below the patella)	36 cm	36.5 cm	Normal

The Valsalva manoeuvre (bearing down) aggravated the cervico-thoracic junction and T7–8 level pains. The blood pressure in the right arm in the seated position was 120/72. Auscultation of the heart appeared to be normal and there was no cervical bruit. Supine straight leg raising was to a measured 80° bilaterally and painless. Bilateral hip flexion did not elicit any pain but combining bilateral hip flexion and cervical spine flexion caused an increase in pain in the mid to lower cervical spine and at the T7–8 level.

Active thoracic spine ranges of movement were measured as shown in Box 88.2.

Box 88.2 Active thoracic spine ranges of movement

	Approx range	Patient's comments
Flexion	Full	Elicited slight pain at approximately the T7–8 level
Extension	Full	Elicited slight pain at approximately the T7–8 level
Lt lateral bending	Limited by approx 10%	Elicited pain on the left side at approximately the T7–8 level
Rt lateral bending	Limited by approx 10%	Elicited greater pain on the left and right sides at T7–8
Lt & Rt rotation	Limited by 25%	Elicited an increase in pain at the T7–8 level

Box 88.3 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	40°	Elicited approximately C4–5 level pain, especially on the right side
Extension	60°	50°	Elicited slight pain at approximately the C4–5 level
Lt lateral bending	45°	40°	Elicited 'stiffness' but no pain
Rt lateral bending	45°	42°	Elicited 'stiffness' and a 'stretching' sensation on the left side
Lt rotation	80°	52°	Elicited very slight 'aching' and 'stiffness' on the right side at approximately the C5–6 level
Rt rotation	80°	60°	Elicited very slight 'aching' and 'stiffness' on the left side at approximately the C5–6 level

Note that there was measured restriction of all movements.

Thoracic anteroposterior, lateral and oblique compression tests to elicit pain in the rib cage articulations was painless.

Active cervical spine ranges of movement were measured using CROM instrument (see Box 88.3).

Other cervical spine and cervico-shoulder region tests were performed to check for cervical spine pain as shown in Box 88.4.

Active lumbar spine ranges of movement were measured as shown in Box 88.5.

IMAGING REVIEW

The hospital imaging was not available. However, thoracic spine plain X-ray films taken approximately 2 months post injury showed: 'Anterior wedging of the bodies of T7 and T8 (Fig. 88.2) with the body of T8 showing anterior height loss of approximately 20%. Some wedging to the right of the body of T7. Slight narrowing of the T6–7 and T7–8 disc spaces. The remaining disc spaces are preserved'.

Box 88.4 Other cervical spine and cervico–shoulder region tests

	Patient's comments
Lt rotation plus extension	Elicited a 'pinching' sensation on the left side at approximately the C4–5 level and general 'stretching' on the right side of her neck
Rt rotation plus extension	Elicited a 'pinching' sensation on the right side at approximately the C4–5 level and general 'stretching' on the left side of her neck
Cervical spine traction	Painless
Cervical spine compression	Painless
Downward shoulder pressure	Painless
Trapezius trigger point pressure	Elicited pain in the left and right trapezius trigger points

Box 88.5 Active lumbar spine ranges of movement

	Approx range	Patient's comments
Flexion	Fingers reached to her ankles	Painless in the lumbar spine
Extension	Full	Painless in the lumbar spine
Lt lateral bending	Fingers reached to approx 5 cm above the knee (i.e. somewhat restricted)	Painless in the lumbar spine
Rt lateral bending	Fingers reached to approx 5 cm above the knee (i.e. somewhat restricted)	Painless in the lumbar spine
Lt & Rt rotation	Somewhat limited due to approximately mid-thoracic spine pain	Painless in the lumbar spine

CLINICAL IMPRESSION

1. Vertebral body fractures at the T7 and T8 levels with associated musculoligamentous soft tissue injuries.
2. Musculoligamentous soft tissue injuries of the cervical spine.

WHAT ACTION SHOULD BE TAKEN?

A CT thoracic and cervical spine was performed to look at the osseous structures in detail. These showed the following results.

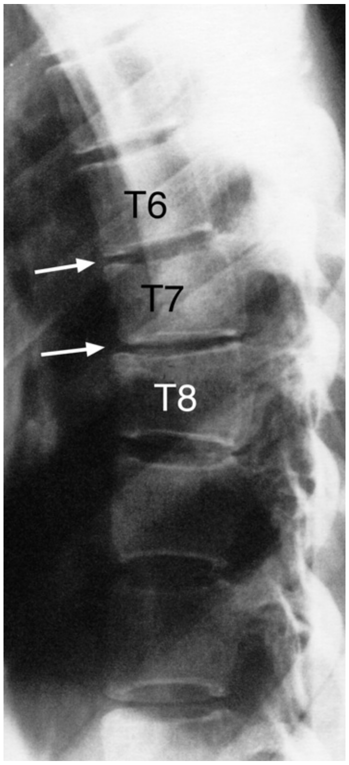

Figure 88.2 Thoracic spine lateral plain X-ray image showing anterior wedging of the bodies of T7 and T8 indicating compression fractures of these vertebral bodies. Also note the slight narrowing of the T6–7 and T7–8 disc spaces (white arrows).

Thoracic spine: 'Stellate fractures to the bodies of T7 (Fig. 88.3) and T8 (Fig. 88.4), which show evidence of some bony union, although union is incomplete. There is no significant displacement of any fracture fragments, and the spinal canal and intervertebral foramina appear normal in size and shape. There is no evidence of encroachment upon the thecal sac or exiting nerve roots'.

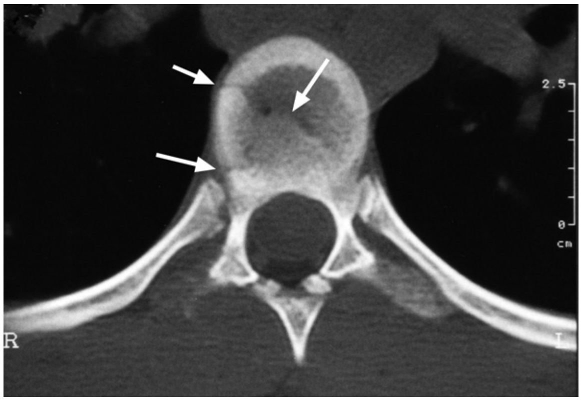

Figure 88.3 CT axial image through the T7 vertebra. Note the incomplete body union at the fracture sites (white arrows). R = right side of patient.

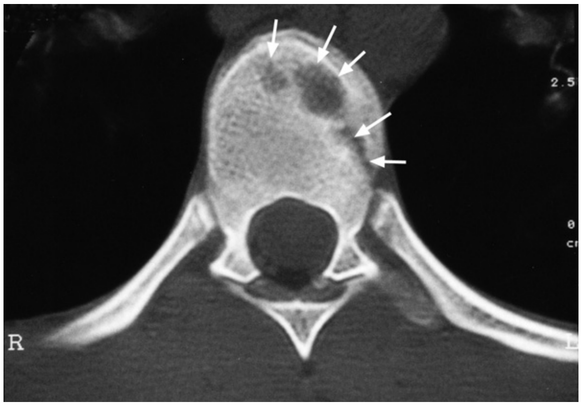

Figure 88.4 CT axial image through the T8 vertebra. Note the incomplete body union at the fracture sites (white arrows).

Cervical spine: Reported as normal. However, the lateral scout view shows a likely loss of normal cervical spine lordosis with a cervical spine kyphosis with an apex at approximately the C5 level (Fig. 88.5). In addition, there appears to be a degree of anterolisthesis of C2 on C3. There is no lipping of the vertebral body margins to suggest an injury of longstanding.

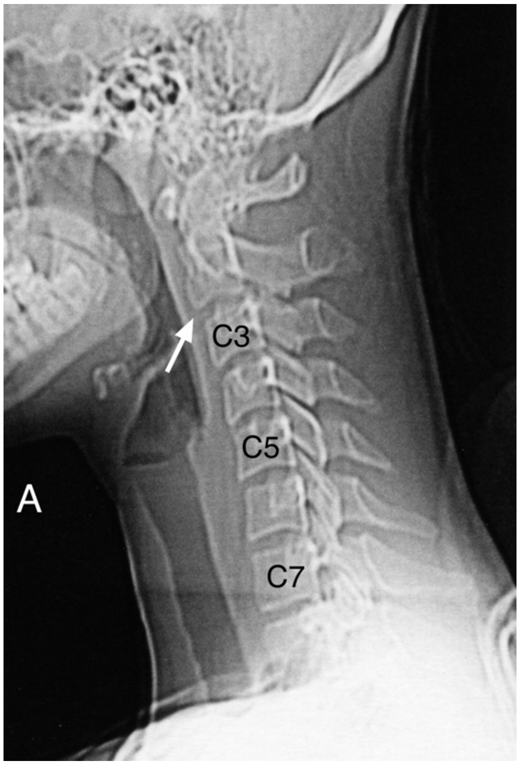

Figure 88.5 Cervical spine CT lateral scout image. Note the loss of normal cervical spine lordosis with a cervical spine kyphosis and an apex at approximately the C5 level. In addition, there appears to be a degree of anterolisthesis of C2 on C3 (white arrow). There is no lipping of the vertebral body margins to suggest an injury of longstanding.

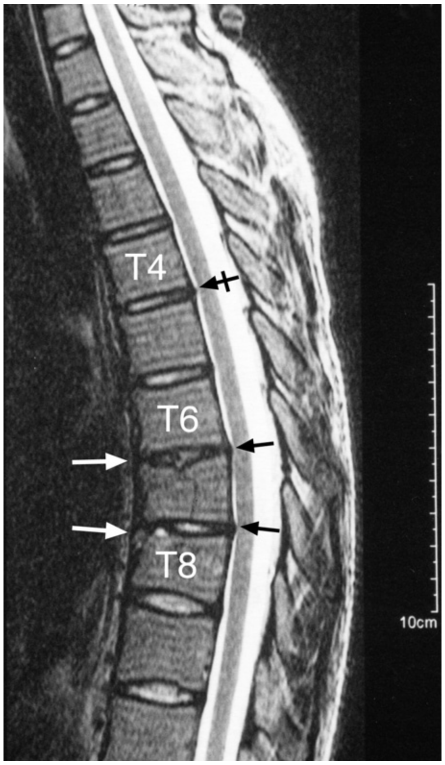

Figure 88.6 Thoracic spine MRI sagittal T2-weighted image. Note the 'wedge compression fractures involving the superior end-plates of T7 and T8', the traumatic 'degenerative narrowing (white arrows) and signal loss involving the T6–7 and T7–8 intervertebral discs associated with minimal central posterior disc bulges (black arrows) . . . and slight angular kyphosis at the level of the compressed vertebral bodies'. In addition, the T4–5 intervertebral disc shows early desiccation with a minimal central posterior disc bulge (tailed black arrow). These disc bulges press on the pain sensitive anterior part of the thecal sac and, due to the slight angular kyphosis, appear to be causing the T6–7 and T7–8 disc bulges to be in close proximity to the spinal cord.

As a result of the CT scan, a thoracic and cervical spine MRI was requested to investigate the soft tissue structures. These images showed the following.

Thoracic spine: 'Wedge compression fractures involving the superior end-plates of T7 and 8 (Fig. 88.6). Degenerative narrowing and signal loss involving the T6–7 and T7–8 intervertebral discs associated with minimal central posterior disc bulges and slight angular kyphosis at the level of the compressed vertebral bodies. The thoracic cord is of normal signal intensity with no increased signal intensity in this area to suggest myelomalacia'.

Cervical spine. Reported as '*Normal*'. However, the cervical spine sagittal images show evidence of central posterior disc bulging at C5–6 (Fig. 88.7). There is early desiccation of the intervertebral discs at the C4–5 and C5–6 levels.

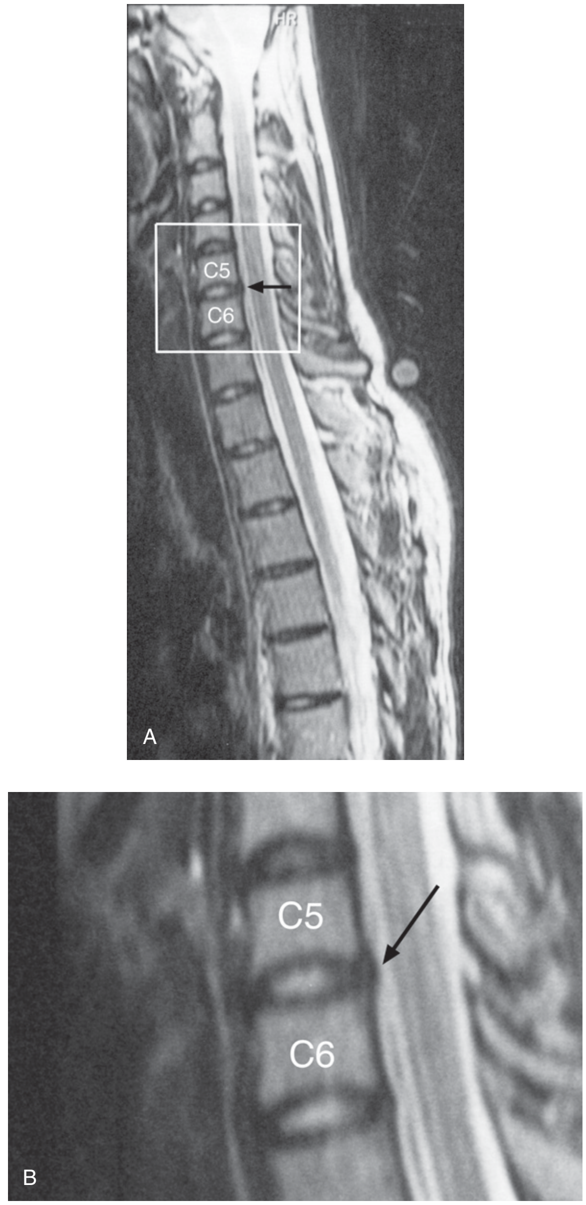

Figure 88.7 (A) Cervical spine MRI sagittal T2-weighted image. Note the central posterior disc bulge at C5–6 (black arrow). The white rectangle represents the magnified view (B) shown above.

DIAGNOSIS

- C5–6 central posterior disc bulge.
- T7 and T8 vertebral body superior endplate compression fractures with minimal central posterior disc bulges at T4–5, T6–7 and T7–8 levels.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

She now fully understood why she had ongoing mid-thoracic spine pain and intermittent neck pain. She was advised not to perform any activities that would add additional forces to her thoracic and cervical spines. As she had been on non-steroidal anti-inflammatory medication since the motor vehicle accident 4 months ago, needle acupuncture treatment was advised for pain control as the long-term use of non-steroidal anti-inflammatory drugs may well result in side effects. She was advised to see an orthopaedic surgeon to monitor the progress of the incomplete union of the T7 and T8 vertebral body fractures and for advice on how to minimize the development of the thoracic kyphosis. This approach resulted in satisfactory control of her symptoms and no orthopaedic intervention was recommended as a result of the orthopaedic review.

KEY POINT

The usefulness of MR imaging for motor vehicle patients should not be underestimated.

Further reading

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Case 89

T6–7 intervertebral disc posterior protrusion

COMMENT

Thoracic spine disc bulges and protrusions may cause straightforward symptoms and signs, as in this case, or they may present as a complex group of symptoms and signs as presented in previous cases.

PROFILE

A 39-year-old male who does not smoke or drink alcohol and is physically very fit and works in the armed forces.

PAST HISTORY

At approximately 15 years of age he injured his mid-thoracic spine while playing football, but he recovered over a period of time. He then fell off a horse and caused an aggravation of his mid-thoracic spine pain. This pain usually resolves itself within a few days after seeing a chiropractor who treats him with heat packs and massage. He has had remissions and exacerbations for some 20 years.

PRESENTING COMPLAINT(S) (Fig. 89.1)

Acute on chronic mid-thoracic spine pain that does not radiate to either side. He pointed to approximately the T6–7 level.

Coughing, sneezing and bearing down markedly increase the mid-thoracic spine pain. He is not awakened at a particular time during the night, i.e. there is no night pain. Bowel and bladder function appear to be normal, although he wonders if bowel motions are affected when the pain is severe. He cannot take a deep breath because this aggravates his pain. He also has to be careful with lifting, sitting and bending his spine forward, backward and laterally, as these movements increase his pain.

He does not like to take medication as he found that a non-steroidal anti-inflammatory was of no help, but caused gastric pain. He said he had seen four or five general medical practitioners and each time he was given a non-steroidal anti-inflammatory, which he did not take after realizing that the first one had caused gastric pain.

His chiropractor referred him for a further evaluation because of a clinical suspicion of a disc lesion and wanted to have a thoracic spine MRI performed and also requested that treatment be provided as deemed appropriate.

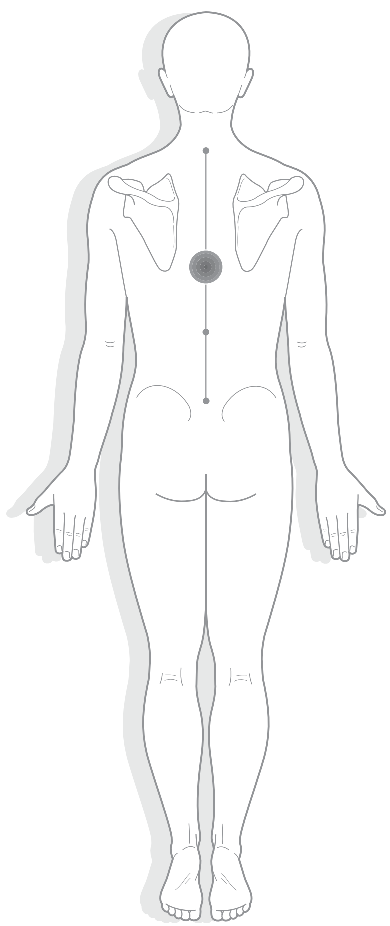

Figure 89.1

AETIOLOGY

His current acute mid-thoracic spine pain was due to bending forwards 2 days prior to consultation to pick up, from the floor, a light object; he felt a 'stabbing pain' in the mid-thoracic spine with radiation to both scapulae and 'stiffness' in the thoracic spine.

EXAMINATION

The deep tendon reflexes in the arms and legs were normal as was pinprick sensation. Power and muscle tone in the legs and feet were normal and the plantar response was normal, i.e. downward. The pulses and the temperature of both feet were normal and equal. Percussion of the thoracic spine did not elicit any pain but deep palpation of the paraspinal muscles elicited pain at approximately the T6–7 level. Vibration sensation was normal.

Active thoracic spine ranges of movement were as follows:

1. Flexion elicited pain in the mid-thoracic spine when his fingers reached to his knees.
2. Extension – limited by approximately 50% due to mid-thoracic spine pain.
3. Lateral bending – limited by approximately 30% to each side due to mid-thoracic spine pain.
4. Rotation to the left and right sides – full and painless.

Ribcage compression elicited the following:

1. In the anteroposterior plane, there was an increase in mid-thoracic spine pain.
2. In the left to right coronal plane there was no pain.
3. On oblique compression from the left anterior to the right posterior side of the ribcage there was an increase in mid-thoracic spine pain.

In the seated and slumped forward position there was an increase in mid-thoracic spine pain; the addition of left and right SLR, respectively, elicited a further increase in mid-thoracic spine pain. Supine SLR was of full range and painless. Supine cervical spine flexion elicited an increase in mid-thoracic spine pain.

IMAGING REVIEW

Thoracic spine plain film anteroposterior and lateral views were unremarkable.

CLINICAL IMPRESSION

Posterior disc protrusion at the T6–7 level.

WHAT ACTION SHOULD BE TAKEN?

A thoracic spine MRI was requested in view of his symptoms and the clinical findings to determine whether there was a disc bulge or protrusion in the mid-thoracic spine. The MRI showed: A focal centrolateral disc protrusion at the T6–7 level, extending to the left of the midline. There is effacement of the CSF and early impression on the anterior aspect of the thoracic cord (Fig. 89.2). There is minimal disc narrowing at this level. No bony abnormality. The remainder of the cord appears intact.

DIAGNOSIS

T6–7 focal centrolateral posterior disc protrusion.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised not to perform any heavy lifting or activities where he would have to hyperflex or

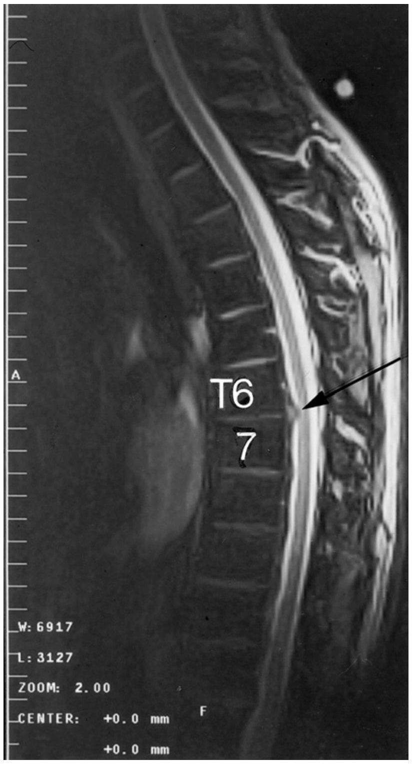

Figure 89.2 Thoracic spine MRI sagittal T2-weighted scan showing a focal posterior disc protrusion at T6–7 with impression on the thoracic cord.

hyperextend his thoracic spine. He was offered the option of seeing an orthopaedic surgeon or of trying needle acupuncture treatment. He chose the acupuncture treatment.

At the third acupuncture treatment session he reported that he now had only a 'low-grade dull awareness' of mid-thoracic spine pain, so he was advised to return if further treatment was indicated. He has subsequently been well and not needed further treatment 3 years following his acupuncture treatment.

KEY POINTS

1. The symptom of increased pain on coughing, sneezing and bearing down strongly suggests a disc protrusion if there is no night pain to suggest an overt pathological process.
2. A central disc protrusion is suspected if the pain is centrally located with no radiation of pain affecting the nerve roots.

Further reading

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Case 90

Spinal cord syrinx

COMMENT

If the cervicothoracic spine is subjected to forceful trauma and symptoms and signs consistent with a spinal cord injury develop, a complete diagnostic work-up, including an MRI study, is warranted.

PROFILE

A 39-year-old somewhat overweight married man who worked as a miner and who does not smoke and only drinks alcohol socially.

PAST HISTORY

He has an allergy to Digesic that causes 'blurring of vision and nausea'. He had osteomyelitis in the left 'hip' region at approximately 6–8 years of age from which he made a complete recovery following surgery.

Two-and-a-quarter years ago he was working late at night in a mine; he had 'fired' the rock face before hosing it down to settle the dust. He looked up and saw a large rock slab falling towards him, so he turned and started to run away from it but it hit his upper thoracic spine, knocking him face down onto other rocks. He immediately felt 'numbness' from approximately his 'nipple level down'. His legs felt 'paralysed', particularly his right leg but he gradually recovered and was then able to walk again; however, he gradually developed the above presenting symptoms.

PRESENTING COMPLAINT (Fig. 90.1)

A 'burning' sensation from approximately the T5 level 'down to the feet' due to a mining accident 2.25 years ago. When lying in bed he may get 'spasms in the legs which shake a lot', usually one leg being affected at a time; the symptoms may then affect the other leg. He cannot sit or stand for more than a few minutes without moving because of the mid-thoracic spine pain. He also experiences a 'stabbing' sensation at approximately the T5 level which can radiate around his torso. He periodically experiences headaches which begin in the lower cervical spine and radiate up over the occipital and temporal regions of his skull without any definite time interval or frequency.

He walks with a slightly spastic gait and says the injury has resulted in some sexual disturbance in that he cannot maintain an erection. He also has a bowel problem in that

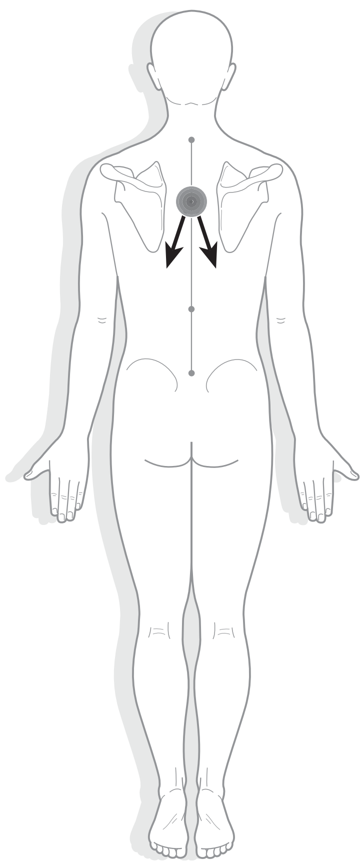

Figure 90.1

he sometimes has 'soiling' on his underpants due to a lack of bowel control. He has to empty his bladder regularly, i.e. by timing, rather than by urge, otherwise he cannot control his bladder.

AETIOLOGY

Mining accident 2.25 years ago.

EXAMINATION

In the erect posture there was no obvious clinical evidence of pelvic obliquity or of scoliosis. Percussion of his spine at the T4–6 level elicited local pain with a sensation of 'numbness' lower in his thoracic spine. Deep palpation of the paraspinal muscles elicited considerable tenderness over the T4–6 level. Toe walking power (S1) and heel walking power (L5) were somewhat weak. The deep tendon reflexes in the upper extremities were normal but the knee jerks (L4) were considerably hyper-reflexive bilaterally, suggesting an upper motor neuron lesion. The ankle jerks appeared to be approximately normal. The plantar response (Babinski test) was strongly upward turning on the right and, to a lesser degree, on the left (indicating a pathological response, i.e. an upper motor neuron lesion). The upper (T7–10) and lower (T10–L1) superficial abdominal reflexes could not be elicited.

Light touch and pinprick sensation of the cervicoshoulder region (C4, C5, T1–2 dermatomes), the arms and hands was normal. Pinprick sensation below the nipple line on the front and back of the torso was diminished; on the lateral aspect of his left thigh (L2) he expressed pinpricking as feeling like a 'burning' sensation but on the medial aspect of his left thigh (L3) there was a subjective 'numbness'. Pinprick on the left and right calves, laterally, elicited a 'burning' sensation and the same sensation was reported for light touch using cotton wool over the same area. His feet were particularly hypersensitive to pinpricking and he reported the pinpricking as feeling as if his feet were 'strongly burning' on the lateral (S1) and medial aspects (L5). There was a patchy distribution of sensation in his legs and feet. (These findings most likely indicate injury to his spinal cord.) Applying a vibrating tuning fork to the left ankle gave an approximately normal response but there was considerable subjective diminution in sensation when the tuning fork was placed on the right ankle. (This suggests an injury to the posterior columns of his spinal cord.) Power in the upper and lower limbs appeared to be normal, apart from difficulty with toe and heel walking. The pulses in his feet appeared normal. The temperature of his feet was equal on palpation, apart from his toes which felt cold on each foot; both feet had a bluish colour (suggesting some possible difficulty with his circulation).

In the supine position, cervical spine flexion elicited pain at approximately the T5 level. Supine left and right SLR, respectively, elicited some mid-thoracic spine pain, as did SLR plus foot dorsiflexion. Bilateral knee flexion caused mid-thoracic spine pain. (All these tests suggested that tractioning the spinal cord was causing pain in the upper to midthoracic spine).

Active thoracic spine movements in the seated position were as follows:

1. Flexion elicited pain at approximately T5.
2. Extension elicited similar pain but also an ache at the thoracolumbar region.
3. Left and right rotation did not elicit any thoracic spine pain but elicited some thoracolumbar junction 'ache'.
4. Lateral bending did not elicit any thoracic spine pain but elicited some thoracolumbar junction 'ache'.

The blood pressure in the left arm was 112/80 in the seated position.

IMAGING REVIEW

Plain film radiographs of his cervical and thoracic spines were non-contributory.

CLINICAL IMPRESSION

Upper thoracic cord injury.

WHAT ACTION SHOULD BE TAKEN?

A thoracic spine MRI scan. This showed 'post-traumatic cord abnormality at T1 with syrinx and posterior spinal stenosis' with effacement of the posterior aspect of the thecal sac and perhaps the thoracic spinal cord at this level (Fig. 90.2A and B).

In order to determine whether decompression of the spinal cord was necessary a neurological opinion was sought.

DIAGNOSIS

Post traumatic syrinx of the cord at the C7–T1 level.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

He was advised not to perform any heavy manual work. In addition, he was advised not to work underground as this could be dangerous to him and to his work colleagues. The neurosurgeon advised the patient that, if there were any suggestion of neurological deterioration, he should immediately present for review.

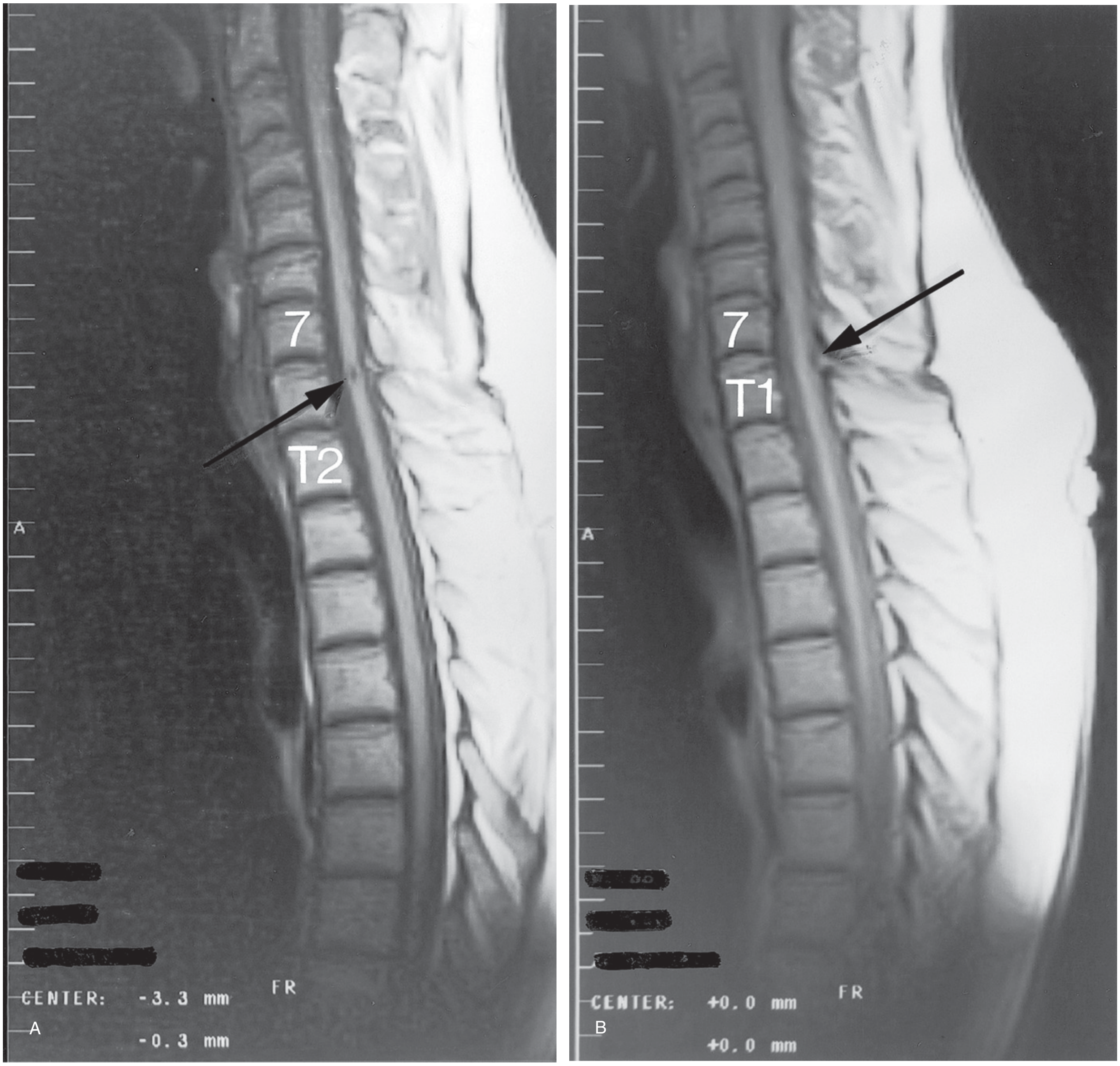

Figure 90.2 (A) Thoracic spine MRI sagittal T1-weighted image showing the small post-traumatic syrinx at the T1 level (arrow) which indicates an injury to the spinal cord itself. A = anterior; T2 = second thoracic vertebra; 7 = seventh cervical vertebra. (B) Thoracic spine MRI sagittal T1-weighted image showing the effacement of the posterior aspect of the dural tube and the thoracic spinal cord at this level (arrow). A = anterior; T1 = first thoracic vertebra; 7 = seventh cervical vertebra.

KEY POINTS

1. It is important to consider the possible development of a post-traumatic syrinx in patients with various symptoms and signs following a spinal injury.
2. Note that the presentation of a post-traumatic syrinx may be quite subtle (Zdeblick & Ducker 1997).
3. Note that the onset of symptoms from a post-traumatic syrinx can be quite delayed from the time of injury with delays from 6 months to 16 years having been reported (Barnett et al 1971, Shannon et al 1981, Marshall et al 1987).
4. It is interesting to note that the symptoms were mainly felt at approximately the T5 level, whereas the syrinx and posterior spinal stenosis were at the T1 level. Therefore, in complex cases it is wise to perform imaging that extends above and below the symptomatic area.

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Case 91

Extradural cystic lesion

COMMENT

Be suspicious of spinal pain that cannot be aggravated by mechanically stressing the spine on performing various movements.

PROFILE

A 57-year-old housewife.

PAST HISTORY

She had kept fit and healthy during her busy life.

PRESENTING COMPLAINT(S) (Fig. 91.1)

Chronic mid-thoracic spine pain. Coughing sometimes aggravates the symptoms as does sneezing. Plain X-ray films had been non-contributory.

AETIOLOGY

Unknown.

EXAMINATION

There was no clinical evidence of pelvic obliquity or of postural scoliosis. Deep palpation of the paraspinal muscles elicited pain over the T4–6 level of the thoracic spine. The deep tendon reflexes in the upper and lower extremities were normal, as was pinprick sensation. Active ranges of thoracic spine movement were of full range and did not elicit any increase in pain. The Valsalva manoeuvre elicited a slight increase in her pain (suggesting a space-occupying lesion). Bowel and bladder function were normal.

IMAGING REVIEW

There was no previous imaging.

CLINICAL IMPRESSION

A space-occupying lesion within the spinal canal. Intervertebral disc bulge/protrusion. Benign or malignant intraspinal lesion.

WHAT ACTION SHOULD BE TAKEN?

A thoracic spine MRI. This found: 'There is a small area located posterior to the thecal sac at the level of T5 that

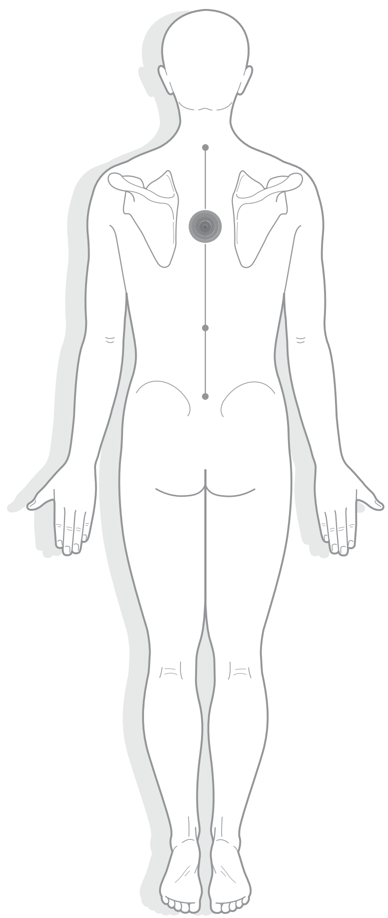

Figure 91.1

measures approximately 5–10 mm. It is oval in shape, behaves like spinal fluid and it does not enhance. It probably represents a small arachnoid cyst abutting the posterior aspect of the thecal sac but it does not appear to put pressure on the thecal sac' (Fig. 91.2A-C).

In view of the imaging findings, the following blood tests were performed as a precaution (Box 91.1). These results were within normal limits.

In view of the previous MRI findings, a further MRI was taken 6 months later. This reported: 'The small cystic

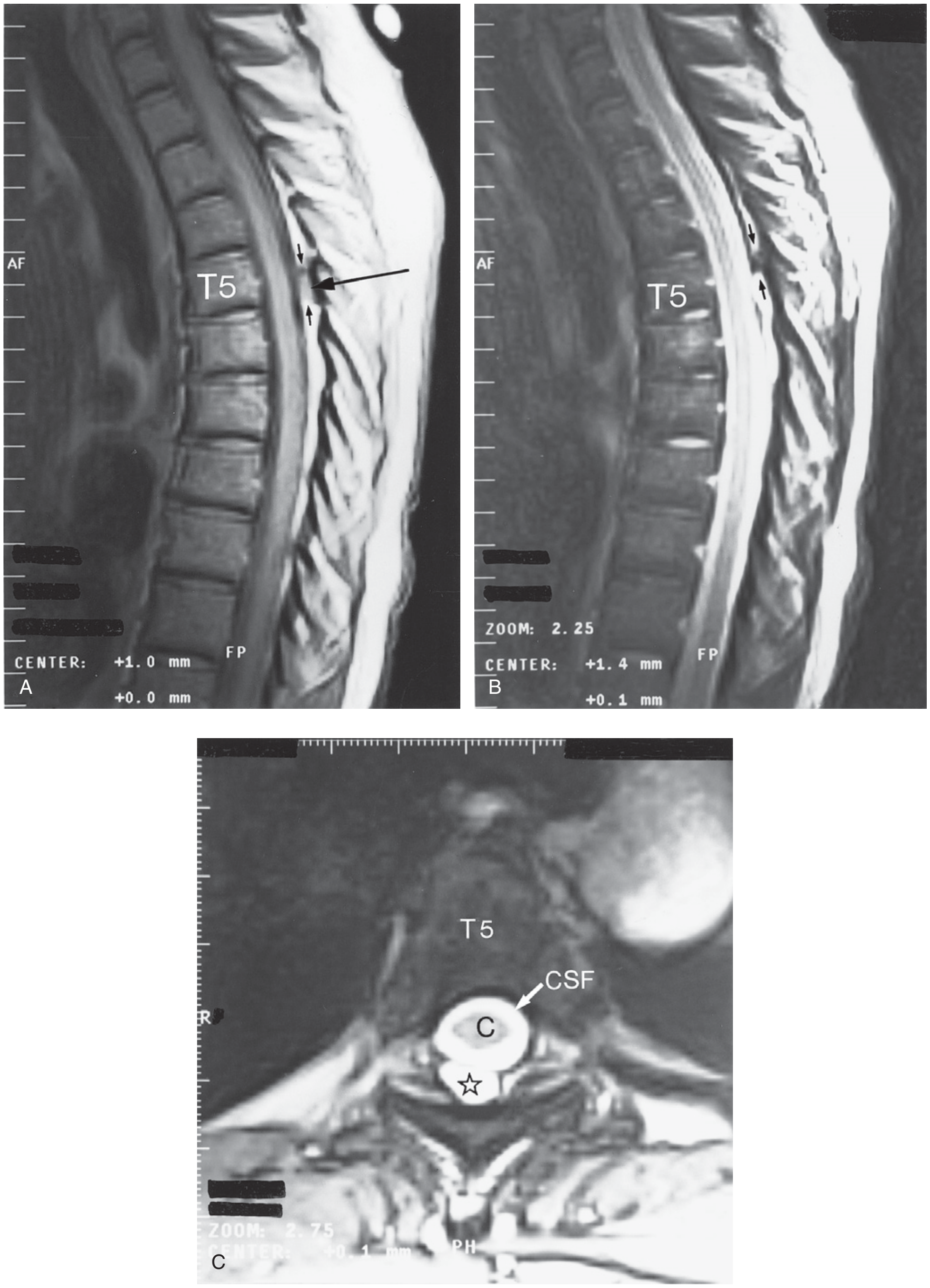

Figure 91.2 (A) Thoracic spine MRI sagittal T1-weighted image that shows the small cystic lesion (arrows) lying posterior to the thecal sac at the T5 level. There is no obvious mass effect on the thecal sac or spinal cord. (B) Thoracic spine MRI sagittal T2-weighted thoracic spine MRI scan that shows the small cystic lesion (arrows) lying posterior to the thecal sac at the T5 level. (C) Thoracic spine MRI axial T2-weighted image through the small cystic lesion showing the cross-sectional area of the lesion (star). C = spinal cord lying within the cerebrospinal fluid (CSF). T5 = fifth thoracic vertebral body.

Box 91.1 Haematology

Specimen type		Units	Reference range
Haemoglobin	127	g/l	(115–160)
Red cell count	4.39	$\times 10^{12}$/l	(3.90–5.60)
Red blood count dist width	13		(12–15)
Haematocrit	0.39		(0.37–0.47)
MCV (mean corpuscular volume)	88	fl	(75–95)
MCH (mean corpuscular haemoglobin)	28.9	pg	(27.0–32.0)
MCHC (mean corpuscular haemoglobin concentration)	330	g/l	(310–350)
Platelet count	240	$\times 10^{9}$/l	(150–400)
White cell count	8.1	$\times 10^{9}$/l	(3.5–10.0)
Neutrophils	35.7	$\times 10^{9}$/l	(2.0–7.5)
Lymphocytes	1.7	$\times 10^{9}$/l	(1.0–4.0)
Monocytes	0.7	$\times 10^{9}$/l	(0.0–0.8)
Eosinophils	0.1	$\times 10^{9}$/l	(0.0–0.4)
Basophils	0.0	$\times 10^{9}$/l	(0.0–0.1)
ESR (erythrocyte sedimentation rate)	12	mm/hour	(5–15)

lesion lying posterior to the thecal sac at the T5 level remains unchanged in size and signal characteristics since the previous MRI examination. There is no pressure on the thecal sac, spinal cord or on the adjacent nerves of the T5–6 intervertebral foramina'.

DIAGNOSIS

Posterior extradural cyst at the T5 level.

TREATMENT AND RESULT

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

The patient was reassured that the thoracic spine lesion was benign and harmless. She was seen by a spinal surgeon at which time the option of surgery was discussed and it was decided that the lesion should be left as it was unless her symptoms increased.

Some years later she still had no increase in symptomatology.

KEY POINT

Small extradural (epidural) benign cystic lesions initially cause uncharacteristic spinal pain (Rickenbacher et al 1982). Therefore, be wary of thoracic spine pain that cannot be reproduced by mechanical stressing of spinal joints, particularly when the Valsalva manoeuvre causes a slight increase in pain.

Reference

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Further reading

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Case 92

Post-traumatic anterior longitudinal ligament calcification

COMMENT

Remember the possible role of the sympathetic nervous system when patients present with autonomic type symptoms.

PROFILE

A 51-year-old married male sedentary worker who does not smoke cigarettes or drink alcohol.

PAST HISTORY

He had not had any unusual childhood or adult illnesses, apart from some lower sternal pain since an injury approximately 2 years ago; he had been told that this pain was due to 'indigestion'.

PRESENTING COMPLAINT(S) (Fig. 92.1)

Lower thoracic spine pain that radiates to the right side as far as the subscapular region since being assaulted approximately 2 years ago. When this pain is severe, he feels 'breathless'.

Medication, spinal manipulation, physiotherapy, and acupuncture treatment have all been of no help.

AETIOLOGY

He was assaulted by a large person who hit his head with such force that he was propelled backwards and hit the lower back part of his thoracic spine against a fixed shelf. His thoracic spine arched backwards and 'twisted over the shelf', and he 'felt and heard a tearing sensation' in the lower thoracic spine. He thought his shirt had been torn but later found it had not. He fell to the floor unconscious for a few seconds; upon regaining consciousness, he felt 'breathless' and dazed but apparently managed to

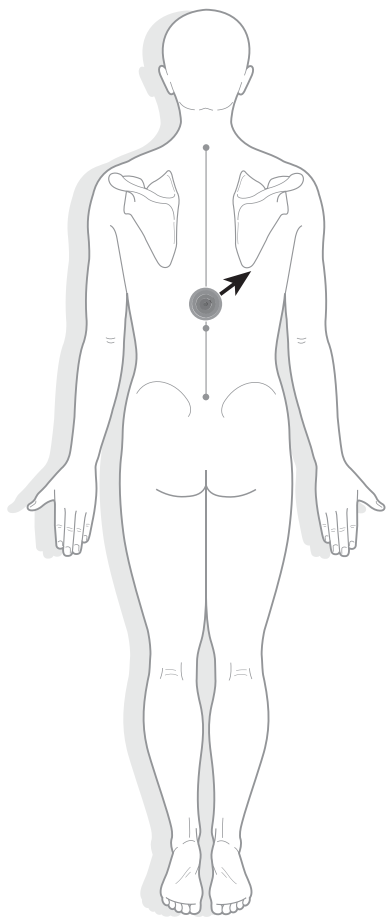

Figure 92.1

defend himself. He felt he could not breathe properly, so his general medical practitioner sent him for thoracic spine radiographs and gave him cortisone medication followed by physiotherapy treatment. He never had any lower thoracic spine pain before the injury described above.

EXAMINATION

Deep tendon reflexes in the upper and lower extremities were normal. Pinprick sensation was normal for the arms and legs except for diminished sensation (hypoaesthesia) in the right big toe (L5). Light touch was normal over the extremities and torso. Auscultation of the heart and lungs was normal. Vibration sensation at the ankles and elbows was normal. Percussion of the lower thoracic and upper lumbar spines elicited an increase in local pain at the T7–10 level. Deep palpation over the paraspinal muscles elicited pain at the T7–10 level bilaterally. The Valsalva manoeuvre (bearing down) did not increase his pain, so a space-occupying lesion was considered to be unlikely. Hand grip strength was normal. Power in the lower limbs was normal. Plantar responses were normal.

Passive left lateral bending was limited by approximately 10% due to some T10–L1 pain. Right lateral bending was limited by approximately 10% due to pain at approximately the T8 level that radiated to below the right scapula. Left and right rotation were limited by approximately 20% due to pain in the T7–8 region. Thoracic spine active ranges of movement were as follows:

1. Flexion – limited by approximately 10% due to a feeling of 'shortness of breath'.
2. Extension – limited by approximately 50% due to pain at approximately the T7–8 level.

IMAGING REVIEW

Plain X-ray films of the thoracic spine were reported as showing 'idiopathic calcification in the T6–7 disc with early calcification appearing in the T7–8 disc. Tiny osteophytes at the vertebral body margins at the T7–8, T8–9 and T9–10 levels'. A very important finding was overlooked, i.e. the slight calcification, probably in the anterior longitudinal ligament, as seen on the lateral thoracic spine X-ray film (Fig. 92.2A). A further thoracic spine plain X-ray examination had reported on the T6–7 disc calcification and 'some aortic calcification'.

CLINICAL IMPRESSION

Mechanical spinal pain due to soft tissue injuries associated with the lower thoracic spine.

Autonomic nervous system involvement in view of his 'shortness of breath' at times.

WHAT ACTION SHOULD BE TAKEN?

In order to further investigate the apparent calcification in the anterior longitudinal ligament, a thoracic CT scan was performed through the mid T9 to mid T12 levels. The CT report stated: 'There are very minor degenerative changes noted with small osteophytes beginning to form at the vertebral body margins at all levels examined. No evidence of fractures is seen. There are no paravertebral soft tissue masses to suggest adjacent haematomas. The spinal canal is not compromised. The exiting foramina are all satisfactory'.

Unfortunately, the calcification in the anterior longitudinal ligament, that probably indicates tearing of the anterior longitudinal ligament with subsequent calcification, was not reported (Fig. 92.2B and C).

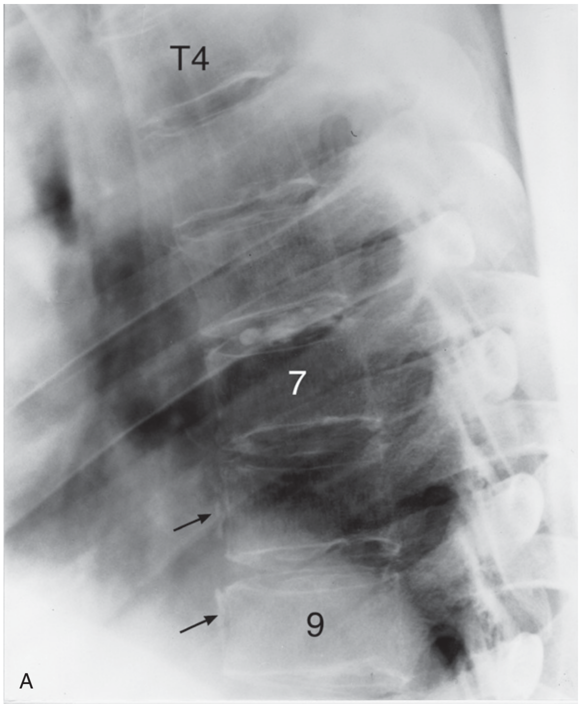

Figure 92.2 (A) Thoracic spine (T4–T9) lateral plain X-ray image showing the 'idiopathic calcification in the T6–7 disc with early calcification appearing in the T7–8 disc'. Also note the slight calcification probably located within the anterior longitudinal ligament (arrows). T4 = fourth thoracic vertebra; T6 = sixth thoracic vertebra; T9 = ninth thoracic vertebra.

(Continued)

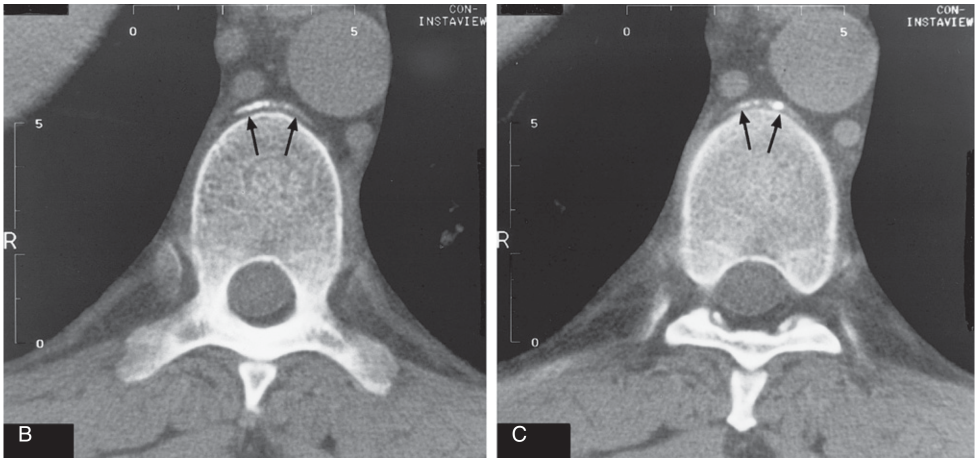

Figure 92.2 Cont'd (B and C) Thoracic spine CT axial images through the 8th thoracic vertebra showing the calcification in the anterior longitudinal ligament (arrows).

DIAGNOSIS

Soft tissue injuries including post-traumatic anterior longitudinal ligament calcification.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

The patient was reassured that he had a benign condition causing his symptoms of mid to lower thoracic spine pain with radiation to the right subscapular region. He was told that the innervation of the spine is very complex and that it was likely that he had injured the sympathetic plexus on the vertebral bodies at the time that the anterior longitudinal ligament and possibly other adjacent soft tissues, were injured when his thoracic spine was hyperextended and that this may be the cause of his periodic shortness of breath when he experiences exacerbations of his thoracolumbar pain. As mentioned in Case 81, Groen & Stolker (2000) have stated that injuries to the thoracic spine may refer pain sensation up and down several segments, so this neurological mechanism may be involved in his shortness of breath.

He responded well to the explanation of his soft tissue injuries and possible sequelae and was pleased to know that an organic cause for his condition had been found as he had been told to stop malingering. He has now learned to live with his condition.

KEY POINT

Hyperextension of the thoracic spine can cause tearing of the anterior longitudinal ligament and probable injury to the autonomic sympathetic plexus adjacent to the vertebral bodies and discs

Reference

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Case 93

Neurofibroma

COMMENT

Remember there may well be more than one condition producing localized symptoms.

PROFILE

A 26-year-old male manual worker of average build who smokes approximately 12 cigarettes per day but does not drink alcohol.

PAST HISTORY

Approximately 8 years ago he was injured when a grenade exploded, causing a couple of small pieces of shrapnel to penetrate the muscles in the back of his neck and upper thoracic spine. Two years ago he fell, causing a fracture of the T6 vertebral body for which he was given rehabilitation treatment during an off-work period of 5 months.

PRESENTING COMPLAINT (Fig. 93.1)

He has suffered from intermittent approximately T4–8 thoracic spine pain and non-specific neck pain since falling and injuring his mid-thoracic spine 2 years ago. His pain is mild to severe and interferes with his sleep. Twisting causes an increase in symptoms. He had previously been seen at a hospital Pain Clinic and had tried non-steroidal anti-inflammatory and analgesic medication, as well as needle acupuncture privately. Medication provided no real relief while acupuncture provided good relief from pain but, as he required intermittent ongoing acupuncture treatment, he wanted to know why he had persisting mid-thoracic spine pain interfering with his sleep. Coughing and sneezing can aggravate his symptoms.

AETIOLOGY

A fall 2 years ago.

EXAMINATION

In the erect posture there was no clinical evidence of leg length inequality or of pelvic obliquity or scoliosis. There was a generalized tenderness over the mid-thoracic spine region. Deep tendon reflexes in the upper and lower extremities were normal as was the case with pinprick sensation

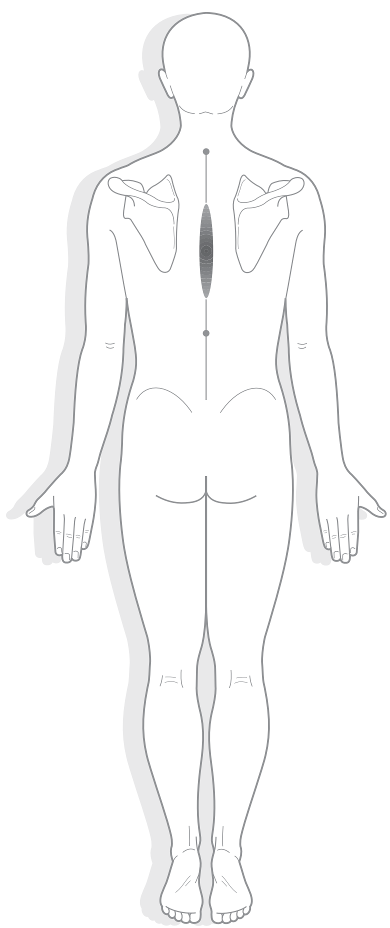

Figure 93.1

over the upper and lower extremities and the torso. Thoracic spine active ranges of movement were within normal limits and did not aggravate his mid-thoracic spine pain. Cervical spine active ranges of movement were of full range and only elicited slight central neck pain at full range. The Valsalva manoeuvre caused a slight increase in his thoracic spine pain.

IMAGING REVIEW

Plain film thoracic spine radiographs had been reported as follows: 'A minimal scoliosis, left concave, is present which may be due to muscle spasm/pain. The vertebral bodies and pedicles are intact'.

CLINICAL IMPRESSION

Possible T 4–8 spinal canal pathology due to the localized thoracic spine pain that troubles him at night as well as during the day.

WHAT ACTION SHOULD BE TAKEN?

A thoracic spine CT scan was requested to further evaluate his mid-thoracic spine pain. Images were performed from T4 to T8. The report stated :'Abnormal increased soft tissue is noted in the epidural fat outside the thecal sac in all of the scanned sections, more prominent towards the right side. This increased soft tissue abnormality appears to be serpiginous (having wavy margins). In some areas it appears more diffuse around the thecal sac. There is no significant paraspinal soft tissue mass and no related bone or joint destruction. Differential diagnosis includes multiple neurofibromas and a vascular malformation' (Fig. 93.2A and B).

In view of the above, a thoracic myelogram and CT was performed. The radiology report stated: 'The myelographic images appear unremarkable. The thoracic cord does not appear expanded and there are no abnormal filling defects identified within the thecal sac. The thecal sac is not significantly displaced. On the CT images no focal abnormality is detected in the cord or in the thecal sac. The abnormal increased tissue in the extra thecal fat persists unchanged when compared with the previous CT examination. Comment: Findings remain non- specific. Although the absence of evidence of a vascular malformation within the thecal sac does not exclude such a pathology, it is slightly less likely and more likely that the lesions are due to neurofibromas. Because neither lesion is likely to be treatable and may be of questionable significance in relation to his clinical symptom of mid-thoracic spine pain, the risks of angiography do not warrant this rather difficult and more risky examination'.

When the myelogram was performed, CSF was obtained for laboratory testing, as a precaution, and showed the results given in Box 93.1.

The results were normal apart from the very slightly increased protein level, thought not to be significant.

Box 93.1 CSF Studies

		Units	Reference range
Protein	0.47	g/L	0.15–0.45
Glucose	3.7	mmol/L	1.8–4.2
Chloride	119	mmol/L	120–130

Rapid plasma reagin test: non–reactive
Treponema pallidum haemagglutin test: non–reactive
Microscopy: no malignant cells seen

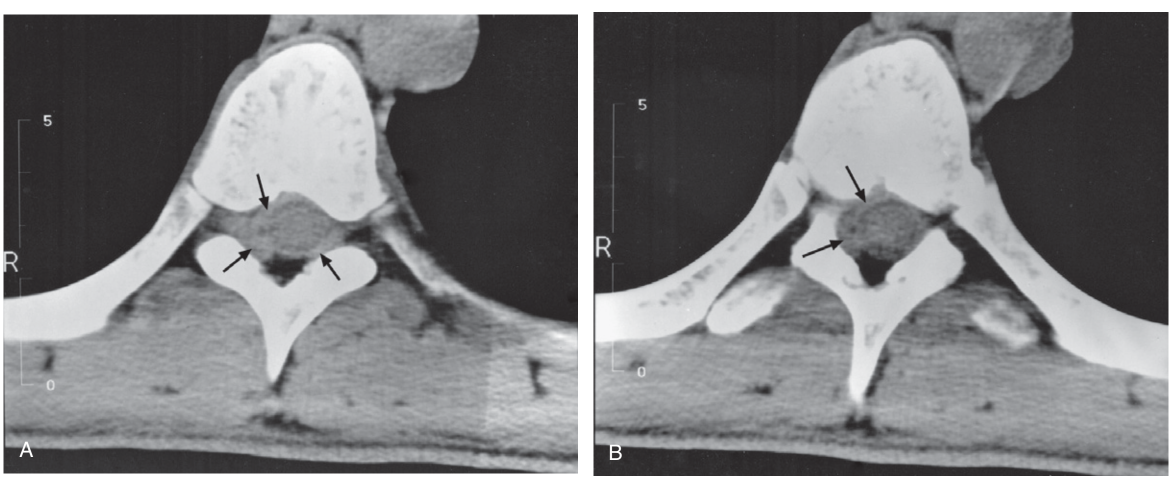

Figure 93.2 (A and B) Thoracic spine CT axial images showing the abnormal increased soft tissue (arrows) in the epidural fat outside the thecal sac, more prominent to the right side.

An MRI investigation of his thoracic spine was considered in an attempt to clarify the pathology. However, in view of his history of having a couple of small pieces of shrapnel in his posterior neck and thorax muscles, routine cervical spine and thoracic spine plain film radiographs were first undertaken as a precaution. These examinations showed small areas of shrapnel posteriorly in the neck (Fig. 93.3) and thorax muscles.

He was referred to a neurosurgeon for a further opinion in view of concerns that the shrapnel may prevent him from safely undergoing an MRI examination of his cervical and thoracic spines. The neurosurgeon stated that a thoracic spinal arteriovenous malformation (AVM) or possibly neurofibromas were present, so he had spoken to radiology colleagues who agreed with him that the small pieces of shrapnel metal in the soft tissue structures were unlikely to cause any significant disturbance of the MRI image. Therefore, an MRI of the cervical and thoracic spines was requested but the examination had to be aborted because the patient felt considerable heat in the muscles posterior to his cervical and upper thoracic spines and the MRI technician noted that there was gross distortion of the initial image (Fig. 93.4).

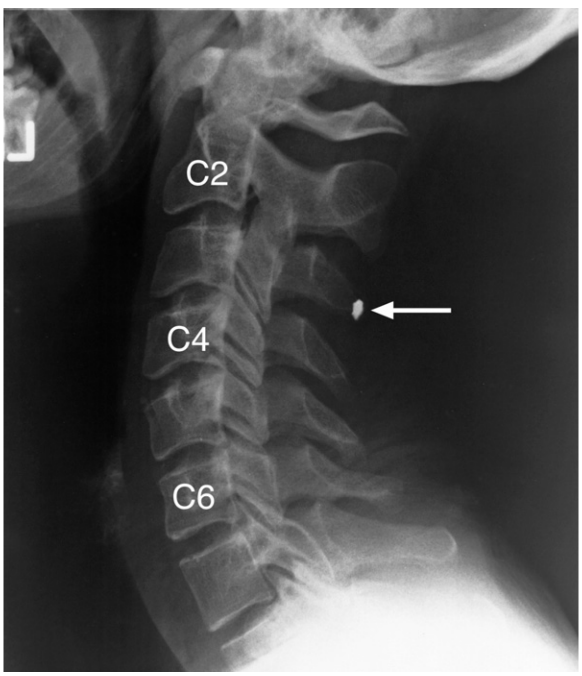

Figure 93.3 A cervical spine lateral plain X-ray image showing a piece of shrapnel (arrow).

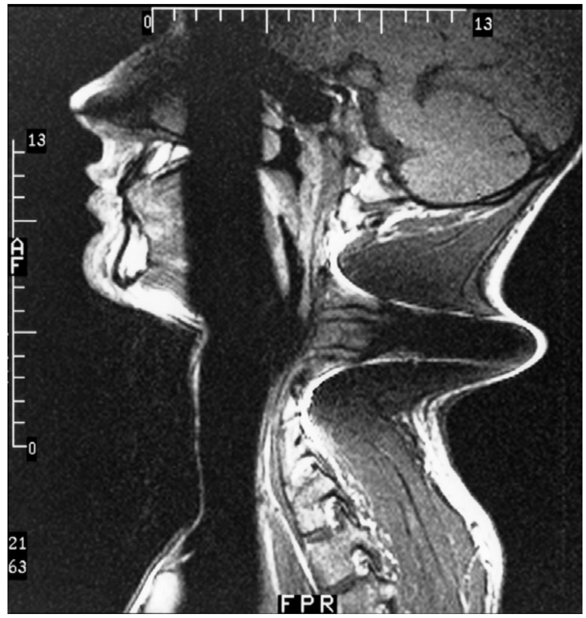

Figure 93.4 Cervical spine MRI sagittal image showing gross distortion of the image due to the shrapnel.

DIAGNOSIS

A tentative diagnosis of thoracic spine arteriovenous malformation with a differential diagnosis of neurofibroma.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. The patient was told that a specific diagnosis for his possible vascular malformation or more likely neurofibromas, could not be made categorically. However, he was reassured that there was no malignancy.

As intermittent needle acupuncture treatment had helped him previously, he was advised to continue with needle acupuncture treatment on an as required basis.

KEY POINTS

A spinal injury history does not mean that spinal pain following the injury should not be thoroughly investigated. The investigations will depend upon the history and the physical examination as exemplified by this case.

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Case 94

Posterior central osteophyte encroaching upon the dural tube

COMMENT

When patients describe unusual symptoms, look to the spine as one possible source.

PROFILE

A 32-year-old married female sedentary worker.

PAST HISTORY

She had undergone a gastroscopy for her ongoing and unexplained symptoms; this procedure revealed slight oesophagitis but there was nothing else of relevance in her history.

PRESENTING COMPLAINT(S) (Fig. 94.1)

For 1 year she has suffered from intermittent severe 'stabbing pains' in the upper thoracic spine that radiate to the left side and to under the left breast; sometimes the pain radiates into the left arm.

Standing erect relieves the pain and sleeping with one pillow is better than using more than one pillow. There is no increase in pain on coughing or sneezing.

She is not aware of what precipitates the symptoms that may occur at any time during the day or night. There is no night pain per se but she may feel pain on turning over during the night. She thinks that picking up her small child is an aggravating factor. She is very concerned that she may have a serious pathological condition.

AETIOLOGY

She could not recall having had any injuries to her upper thoracic spine or neck.

EXAMINATION

Deep palpation over the paraspinal muscles in the upper thoracic spine (particularly T2–4) elicited local pain. The deep tendon reflexes in the upper and lower extremities were normal, as was the case with vibration sensation at

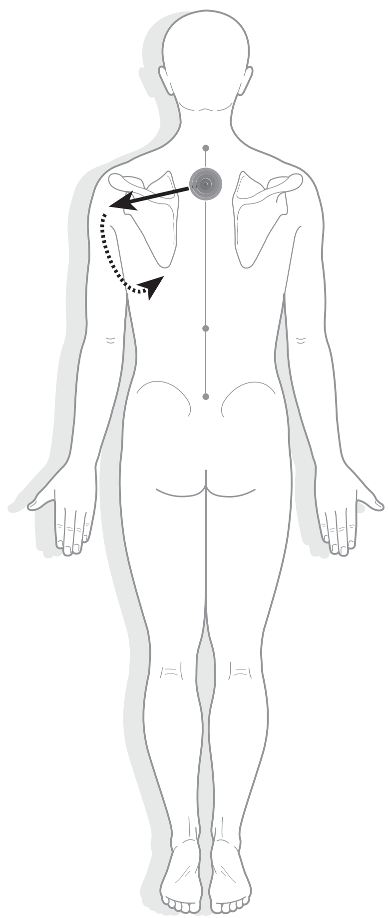

Figure 94.1

the elbows and ankles. Power in the upper and lower extremities was normal. Pinprick sensation did not elicit any areas of altered sensation. Bending forwards elicited an increase in her symptoms.

IMAGING REVIEW

Plain film cervical spine and chest films, respectively, showed mild straightening of the upper cervical lordosis and slight old post-traumatic deformity of the C5 vertebral body. No other changes were noted.

CLINICAL IMPRESSION

Upper thoracic spine disc protrusion. Differential diagnosis – spinal canal space-occupying lesion or bone pathology (considered less likely as there is no pattern of night pain).

WHAT ACTION SHOULD BE TAKEN?

A CT thoracic spine was requested from the T2 to T7 levels. This showed 'Midline T3–4 disc level small posterior osteophyte beginning to encroach upon the spinal canal' (Fig. 94.2A). In order to better visualize the midline posterior osteophyte, a three-dimensional reconstruction oblique tunnel view, looking from above, was also obtained (Fig. 94.2B). This more clearly defined the extent of the osteophyte.

DIAGNOSIS

T3–4 disc level posteior central osteophyte encroaching upon the dural tube.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood.

The patient was told that the midline posterior osteophyte was most likely the cause of her symptoms and she was reassured that there was no more significant pathology. She was advised to sleep on her side using only one pillow to fill the gap between her shoulder and the side of her head and neck and she was told that it would be best to avoid all movements where she might bend forwards and cause the osteophyte to further impinge upon the pain sensitive dural tube and associated blood vessels and the recurrent meningeal nerves.

The issue of whether surgery should be performed was discussed and it was decided that, unless her symptoms deteriorated, then it would be more appropriate to control them by avoiding postures that aggravate her symptoms, to which she agreed now that she had been reassured that there was no 'dangerous' pathology.

She responded well to the above advice.

To better understand the anatomy of the thoracic spinal canal and related structures, see the cadaveric histopathology section in Fig. 94.3.

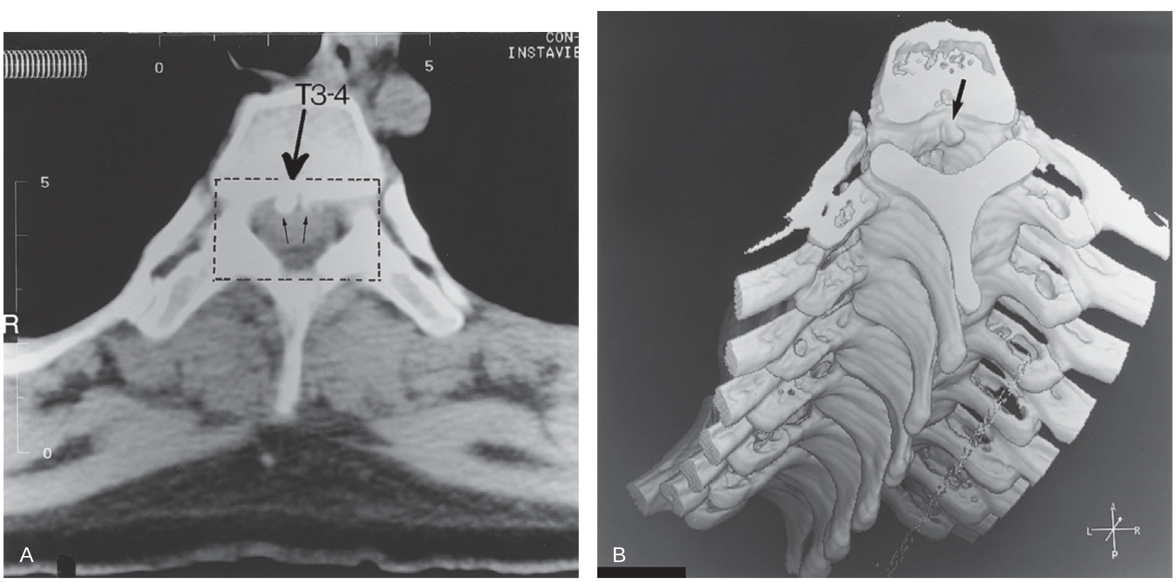

Figure 94.2 (A) Thoracic spine CT axial image at the T3–4 level showing the posterior central osteophytes projecting from the disc-vertebral body junction. Note how the osteophytes encroach upon the pain sensitive anterior part of the dural tube (small arrows). The area in the rectangle is represented in an essentially normal thoracic spine by the histological section in Fig. 94.3. (B) Thoracic spine three-dimensional reconstruction oblique tunnel view looking from above. Note the midline posterior osteophyte (arrow) at the T3–4 level.

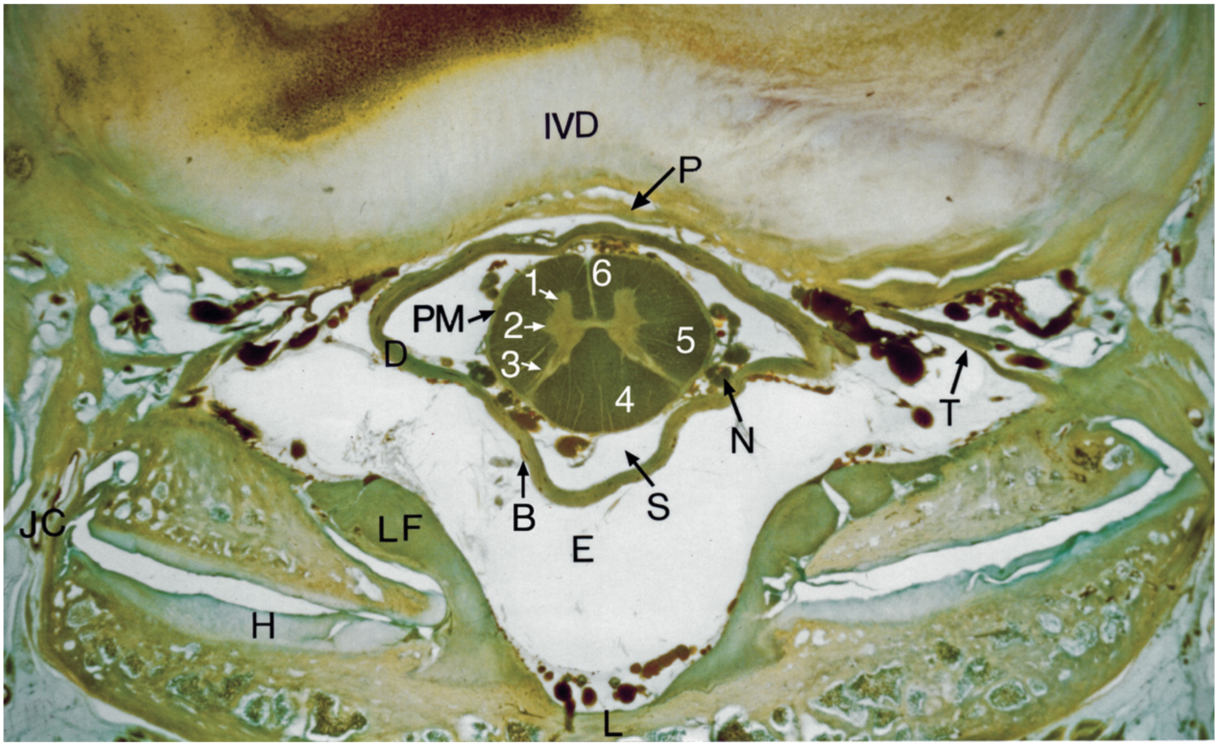

Figure 94.3 A 200-micron thick horizontal (axial) histological section through the thoracic spine of a 40-year-old male cadaver representing the approximate area in the rectangle in Fig. 94.2 A. This shows the anatomy of the spinal canal and related structures at this level but without osteophytes. B = blood vessels within the dural membrane; D = dural tube; E = extradural (epidural) space; H = hyaline articular cartilage on the inferior articular process facet of the vertebra above; IVD = intervertebral disc; JC = zygapophysial (facet) joint capsule; L = lamina junction; LF = ligamentum flavum; N = dorsal (posterior) nerve root; P = posterior longitudinal ligament; PM = pia mater; S = subarachnoid space; T = transforaminal ligament crossing the intervertebral foramen; 1 = anterior grey column; 2 = lateral grey column; 3 = posterior grey column; 4 = posterior funiculus; 5 = lateral funiculus; 6 = anterior funiculus. (Ehrlich's haematoxylin and light green counterstain.)

KEY POINT

When a patient is concerned about a serious pathology possibly causing the symptoms, a CT scan or an MRI scan can be used not only as a diagnostic test, but also as a therapeutic measure which can allay patient fears of a serious pathology being present.

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Case 95

T4 and T6 vertebral body fractures

COMMENT

Lhermitte's sign always needs to be urgently and thoroughly investigated.

PROFILE

A 35-year-old man of muscular build who works as a trainer. He does not smoke cigarettes or drink alcohol.

PAST HISTORY

Two years and four months ago he fell off his horse when it stumbled and fell while cantering; he immediately felt excruciating thoracic and sternal pain. He was taken to hospital by ambulance where imaging showed fractures of his T4–T6 vertebral bodies and his manubrium.

PRESENTING COMPLAINT(S) (Fig. 95.1)

1. Constant pain of varying intensity located centrally in the T3 to T6 area of his thoracic spine. His pain is aggravated by Increased activity, lying supine in bed and coughing. He awakens at various times during the night but there is no night pain per se.
2. On looking down, he experiences sudden 'jabs of pins and needles' that radiate from approximately the mid-thoracic spine to the lumbar region, the buttocks and to both lower regions of the calves.
3. Depression due to his symptoms that he feels have not been explained to him in spite of numerous inpatient and outpatient consultations.

He has normal bowel and bladder function. He takes non-steroidal anti-inflammatory drugs and analgesics for pain control.

AETIOLOGY

He fell off a horse 28 months ago.

EXAMINATION

In the erect posture, there was no clinical evidence of pelvic obliquity or of scoliosis. However, he had a noticeable gibbus deformity at approximately the T4 level. Percussion of the thoracic and lumbar spines was painless. Deep palpation of the paraspinal muscles elicited pain at the T3–6 level. Toe walking power (S1) and heel walking power (L5) were normal. There was a visual and palpable

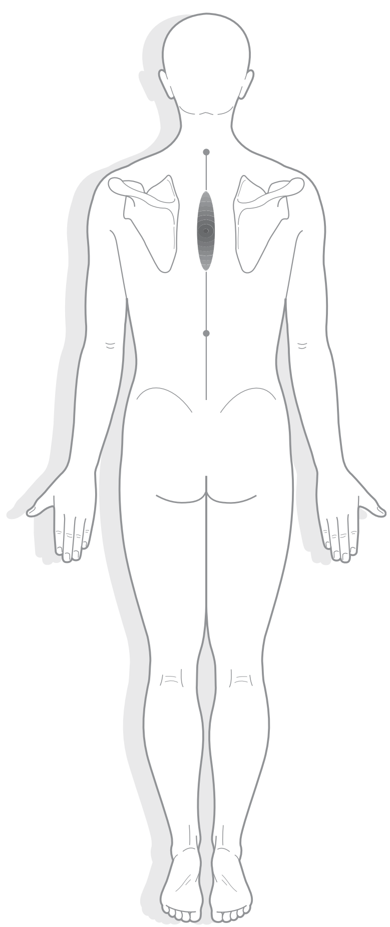

Figure 95.1

deformity of the upper sternum (manubrium) and this was very painful on palpation. Inspiration/expiration measurements across his upper torso indicated a difference of 2 cm, which is lower than would be expected for a fit person of his age. Pinprick sensation over the upper and lower extremities and the back of the torso was normal, apart from a vague sensory cut-off at the T4 level. Motor power and deep reflexes in the upper and lower limbs were normal. The foot pulses were normal. The Valsalva manoeuvre caused upper sternal pain but no significant spinal pain.

Active thoracic spine ranges of movement for extension, left and right lateral bending and left and right rotation were of approximately full range and caused approximately T3 to T6 level pain; flexion reproduced his symptoms of sudden jabs of pins and needles radiating from the mid-thoracic spine inferiorly.

IMAGING REVIEW

The following imaging was performed by the hospital following his injury.

Thoracic spine plain film images including the sternum were reported as showing: 'Compression fractures at T4 and T6' (Fig. 95.2). 'There is a complex fracture of the manubrium sternum dividing the manubrium into three fragments. Anterior displacement of the distal manubrial fragment and body of the sternum is present. No pneumothorax is noted. No mediastinal haematoma is noted. There is no cardiac or pulmonary lesion'.

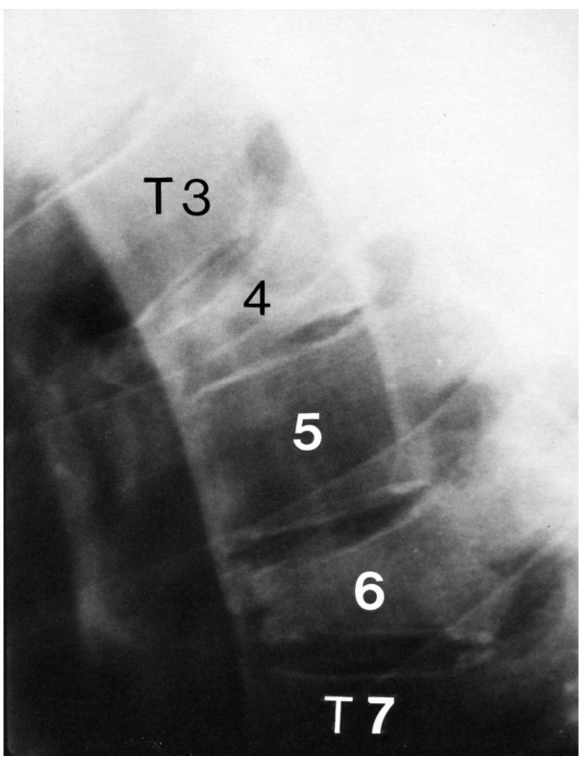

Figure 95.2 Thoracic spine lateral plain X-ray image showing thoracic vertebrae T3 to T7. Note the compression fractures at T4 and T6.

A CT thoracic spine was reported as follows: 'A bursting fracture of the T4 vertebral body (Fig. 95.3) is present with some minimal retropulsion of the posterior aspect of the body but no significant compromise of the spinal canal. In addition, there is a less significant bursting fracture anteriorly at T6.' (Fig. 95.4). In addition there were other

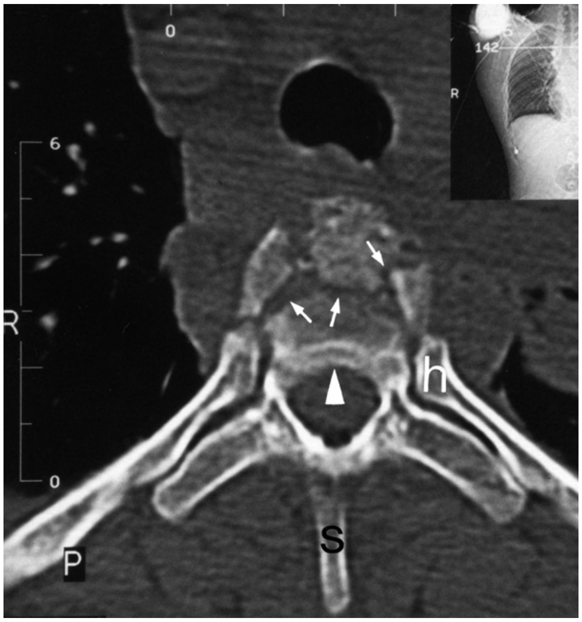

Figure 95.3 Thoracic spine CT axial image at the T4 vertebral body level. Note the fracture lines in the vertebral body (white arrows) and the retropulsion posteriorly into the spinal canal (arrow head). H = left rib head adjacent to the costovertebral joint, S = spinous process.

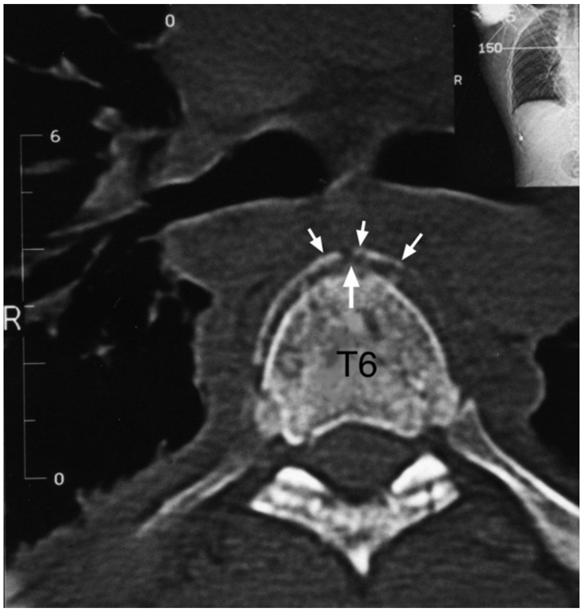

Figure 95.4 Thoracic spine CT axial image at the T6 vertebral body level. Note the bursting fracture anteriorly at this level (white arrows).

fractures that are not illustrated here, i.e. 'there is an undisplaced transverse fracture across the T3 lamina that extends into the transverse processes bilaterally and into the approximate area of the right third rib. At T5 there is a fracture of the left lamina and pedicle extending inferiorly into the inferior articulating process and facet but the facet joint does not appear to be disrupted'.

A CT of the thorax was reported as follows: 'A mediastinal haematoma is present in the para and prevertebral region extending around the aortic arch into the anterior mediastinum. It would be difficult to exclude any actual vascular injury on this examination. The left upper lobe shows some collapse and consolidation medially, representing a pulmonary contusion, as well as a small similar patch of pulmonary opacification medially in the upper lobe on the right; this probably represents pulmonary contusion. Minor basal atelectasis is noted bilaterally with small plural effusions bilaterally'.

An aortic arch angiogram was performed and the report stated: 'No aortic or arch vessel injury is demonstrated'.

CLINICAL IMPRESSION

Spinal cord compromise on cervical spine flexion causing a type of Lhermitte's sign (the development of sudden, transient, electric like shocks spreading down the body on cervical spine flexion; seen mainly in multiple sclerosis but also with compression, inflammatory and other disorders affecting the cervical cord).

WHAT ACTION SHOULD BE TAKEN?

In view of his ongoing symptoms some 2 years 4 months since his accident, an MRI cervical and thoracic spine investigation was requested. The report was as follows.

Cervical spine: 'There is slight loss of overall height of the vertebral bodies at C5 and C6 suggesting minor old compression fractures. There are broad-based posterior disc protrusions at the C5–6 and C6–7 levels (Fig. 95.5) that very slightly impress the thecal sac but appear not to compromise the cord or exit foraminae'.

Thoracic spine: 'Anterior wedge fractures of T4 and T6 are associated with a significant kyphosis (Fig. 95.6A and B). Minor retropulsion of the postero-inferior aspect of the T4 vertebral body is seen impressing the thecal sac very slightly but not significantly compromising it in this neutral position. However, it could well increase its compromise of the spinal canal with significant flexion of the neck and upper thoracic spine and may well account for this man's mid-thoracic paraesthesia radiating to the lumbar spine, buttocks and legs with neck flexion. At the T4–5 intervertebral disc level, a small focus of decreased signal intensity is seen compressing the thecal sac in the T2-weighted images that is not identified on the T1-weighted images. This suggests the presence of a small traumatic disc protrusion in association with the wedge fracture in all likelihood further compromising the spinal canal at this level during flexion. No focal intrinsic cord lesions are identified'.

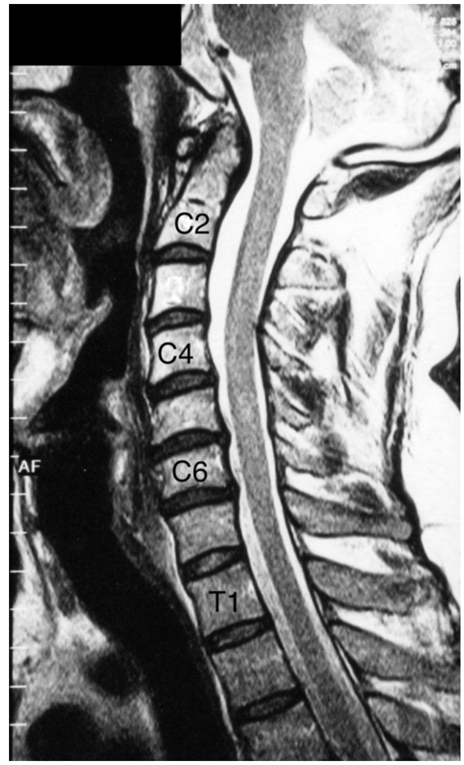

Figure 95.5 MRI sagittal T2-weighted image of the cervical spine showing the 'slight loss of overall height of the vertebral bodies at C5 and C6 suggesting minor old compression fractures' and the 'posterior disc protrusions at the C5–6 and C6–7 levels (arrows) that very slightly impress the thecal sac'. In addition, note the minor posterior disc bulges at the C3–4 and C4–5 levels as well as the desiccation of the C5–6, C6–7 and T2–3 intervertebral discs.

DIAGNOSIS

- T4 and T6 vertebral body anterior wedge fractures with minor retropulsion of the postero-inferior aspect of the T4 vertebral body impressing the thecal sac and abutting the anterior surface of the spinal cord.
- C5–6 and C6–7 posterior disc protrusions; minor posterior bulges at C3–4 and C4–5 levels.
- C5 and C6 vertebral body minor compression fractures.

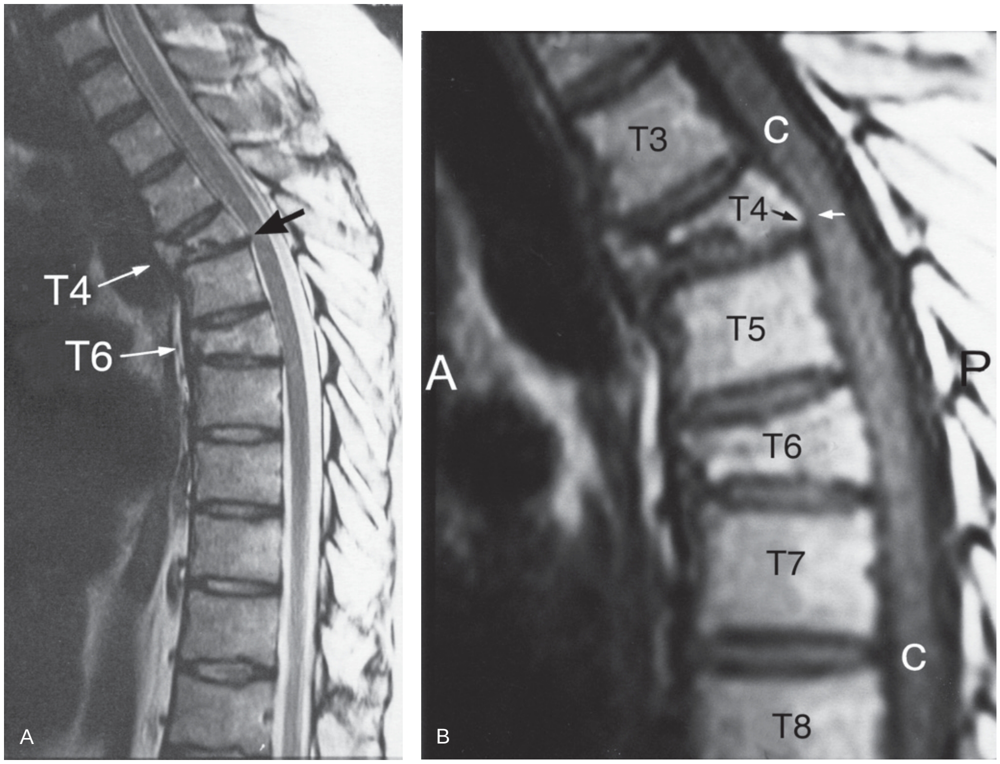

Figure 95.6 (A) Thoracic spine MRI sagittal T2-weighted image showing the severely compressed T4 and T6 bodies with a significant kyphosis and minor retropulsion of the postero-inferior aspect of the T4 vertebral body slightly impressing the thecal sac. The black arrow shows the T4–5 posterior disc protrusion. A = anterior, P = posterior. (B) Thoracic spine MRI sagittal scout image showing the severely compressed T4 and T6 bodies and how the retropulsed postero-inferior aspect of the T4 body compresses the anterior surface (white arrow) of the spinal cord (C). A = anterior, P = posterior.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. He was advised to avoid flexing his cervical and thoracic spines and he was referred to a specialist in rehabilitation medicine who, in turn, referred him to a hospital with a Spinal Injuries Unit for further assessment; he was placed under the care of an experienced spinal surgeon who decided the risks of surgery were not warranted at this time.

KEY POINT

Cervical or upper thoracic spine flexion causing Lhermitte's sign suggests compromise of the spinal cord.

Further reading

Siebenga J, Segers M J M, Elzinga M J et al 2006 Spine fractures caused by horse riding. Eur Spine J 15: 465–471.

Case 96

T7 vertebral body fracture

COMMENT

Carefully scrutinize all imaging.

PROFILE

An 18-year-old female secretary of average build who does not smoke cigarettes and only drinks alcohol socially.

PAST HISTORY

Three months ago she was a passenger, not wearing a seat belt, in a vehicle travelling at speed when it rolled over. She was thrown out of the vehicle and was unconscious for approximately 2 minutes. She was taken by ambulance to hospital where chest X-ray films were taken followed by a CT scan of the abdomen (following oral and intravenous contrast) in view of her right upper quadrant pain and tenderness. She was told no fractures had been found but she was kept overnight for observation then discharged the next day. However, her mid-thoracic spine pain persisted with some radiation to the left mid-axillary line.

PRESENTING COMPLAINT(S) (Fig. 96.1)

1. Constant pain at approximately the T6–8 level where she has noted a bony prominence (slight gibbus). She said this prominence followed the motor vehicle accident 3 months ago. Coughing and sneezing do not aggravate her pain but may cause her to experience some pain in the left mid-axillary line at approximately the T8–10 rib level. Bearing down does not aggravate her symptoms. If she lies on her left side, the ribs become painful in the mid-axillary line at approximately the T8–10 level and slightly anteriorly in the chest wall. Working aggravates her symptoms. Cold weather makes her thoracic spine painful. When she arises of a morning, her thoracic spine feels reasonably good after a night's rest.
2. Intermittent mid-to-low back pain, without radiation, that begins approximately 1 hour after starting her office work.

AETIOLOGY

A motor vehicle accident 3 months ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or scoliosis. There was a gibbus at approximately the T7 level. Percussion of the thoracic and

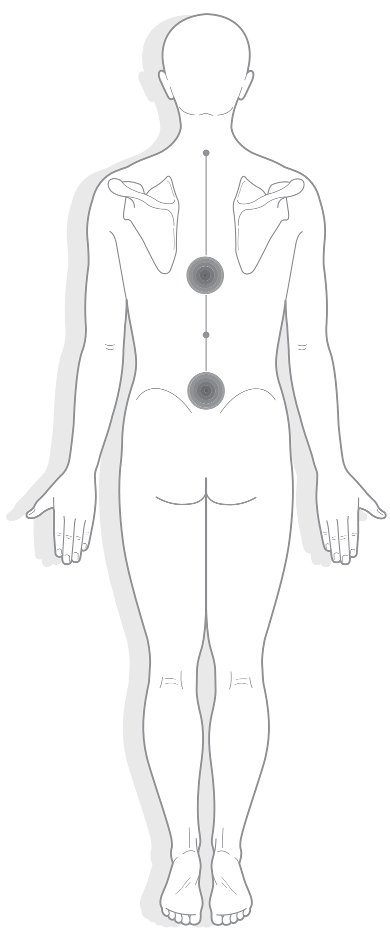

Figure 96.1

lumbar spines did not elicit any local pain. Deep palpation of the paraspinal muscles in the cervical, thoracic and lumbar regions elicited pain at approximately the T6–8 level with tenderness at approximately the mid lumbar spine. Straining the sacroiliac joints did not cause any sacroiliac joint pain but elicited some lumbosacral pain. Toe walking power (S1) and heel walking power (L5) were normal. The deep reflexes in the upper and lower extremities were normal apart from a diminished (one plus) left ankle jerk (S1). Vibration sensation was normal. Pinprick sensation over the back of the torso and the upper and lower extremities was normal. When seated slumped forward there was some pain at approximately the T6–8 level but no low back pain; the addition of straight leg raising did not elicit low back pain. The brachial plexus nerve stretch test did not elicit any pain. Motor power in the upper and lower limbs appeared to be normal. The foot pulses were normal as was the temperature of both feet on palpation. The Milgram active bilateral straight leg raise elicited low back pain. Supine straight leg raising was measured to 65° for the left and right sides respectively, and was limited due to a 'pulling' sensation in the hamstring muscles. Lasegue's sign was normal. Bilateral hip flexion did not cause any low back pain. The Fabere test did not elicit any hip joint pain nor did rolling the femur head in the hip joint.

Active thoracic spine ranges of flexion, extension, left and right lateral bending, and right rotation were of full range and painless; left rotation was of full range but elicited pain on the right of approximately the T8 vertebra. Rib cage on compression in the anteroposterior and lateral planes was painless; oblique plane compression caused pain on the left anterolaterally at approximately the T10 rib level.

Active lumbar spine ranges of flexion and left and right rotation were of full range and painless; extension and left and right lateral bending elicited pain at approximately the L4–S1 level.

Active cervical spine movements were of full range and painless.

IMAGING REVIEW

The hospital imaging was reported as follows. Chest films: 'Lungs clear with cardiac size normal. No fracture seen'. However, on review there appeared to be some unreported anterior wedging of the T7 vertebral body.

CT abdomen full length: 'There is some atelectasis of the left base. The liver, pancreas, kidneys, spleen and bowel appear normal. No free fluid within the abdomen or pelvis. There is a small cortical crack in the anterior alar surface of the left side of the sacrum. The fracture line does not extend to the sacroiliac joint or to the sacral foramina'. On reviewing the CT scan, there appeared to be a fracture of the L3 vertebra's left pars interarticularis with a possible fracture of the pars on the right side (Fig. 96.2).

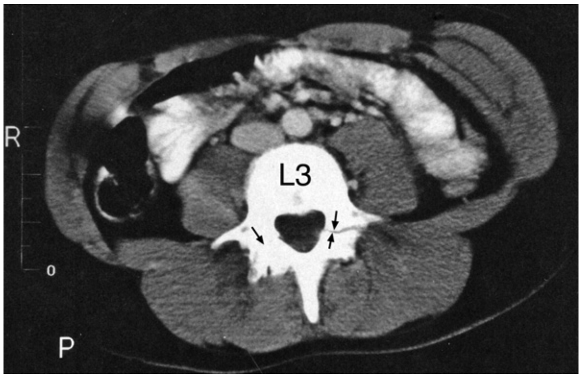

Figure 96.2 CT abdominal image. Note what appears to be a fracture of the left L3 pars interarticularis (short arrows) with a possible fracture on the right side (long arrow).

CLINICAL IMPRESSION

- Compression fracture of the T7 vertebral body.
- Left L3 pars interarticularis fracture; possible right pars fracture.
- Left sacral alar anterior cortical fracture.

WHAT ACTION SHOULD BE TAKEN?

A thoracic and lumbar spine plain film investigation was requested. The radiology report stated: 'A gross compression fracture with flattening of the T7 body is present (Fig. 96.3) with a mild kyphosis at this level. Other thoracic levels appear normal'. The lumbar spine was reported as being normal. However, there appeared to be a pars interarticularis fracture at the L3 vertebral level (Fig. 96.4).

In view of the above findings, a thoracic and lumbar spine MRI was requested that was reported as follows:

A. *Thoracic spine*. 'A wedge compression fracture of the T7 body is present with localized kyphosis at this level and effacement of the thecal sac' (Fig. 96.5). The T2-weighted image (Fig. 96.6) shows 'minimal impression on the dural tube at T7–8 level thought to be related to bony elements at the apex of the kyphosis, rather than a traumatic disc'.

B. *Lumbar spine*. 'A diffuse annular bulge is present at the L4–5 level with slight central prominence; a similar appearance is noted at the L5–S1 level (Fig. 96.7). No nerve root compression'.

DIAGNOSIS

- T7 vertebral body wedge-shaped compression fracture.
- T7–8 level dural tube compression due to bony/disc elements.
- L3 pars interarticulares fractures.
- L4–5 and L5–S1 posterior annular bulges.
- Left sacral alar anterior cortical fracture.
- Musculoligamentous soft tissue injuries.

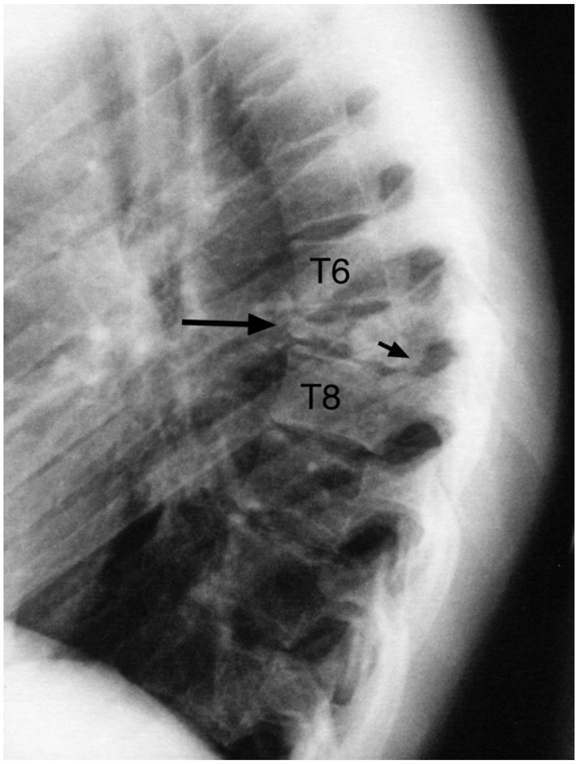

Figure 96.3 Thoracic spine lateral plain X-ray image. Note the grossly compressed T7 vertebral body that appears wedge shaped causing a kyphosis at this level. Also, there is an opacity in the T7–T8 intervertebral foramen (black arrow) suggesting that there may be a burst fracture with retropulsion of bone into the spinal canal. There also appears to be a fracture of the superior endplate of the T8 vertebra.

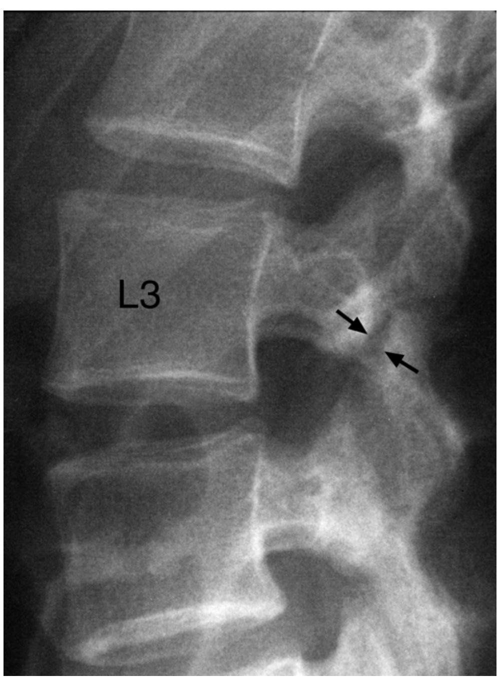

Figure 96.4 Lumbar spine lateral plain X-ray image. Note the pars interarticularis fracture at the L3 vertebral level (arrows).

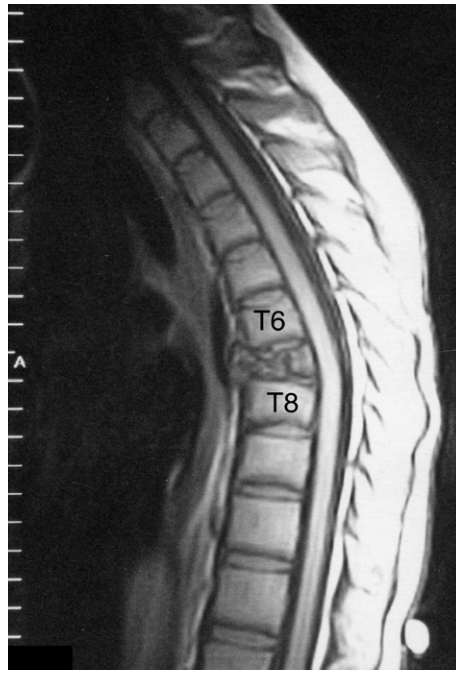

Figure 96.5 Thoracic spine MRI sagittal T1-weighted image showing the wedge compression fracture of the T7 body with localized kyphosis at this level and effacement of the dural tube. Note the kyphosis that has developed in the thoracic spine due to the T7 compression fracture.

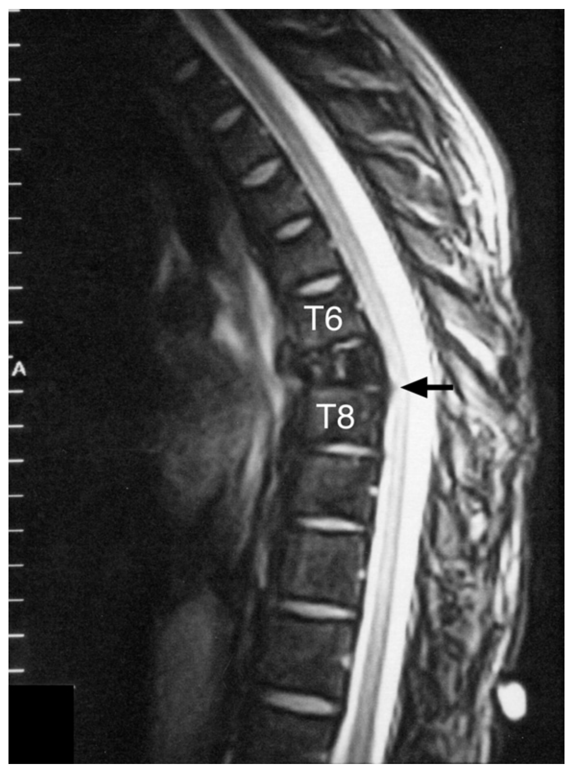

Figure 96.6 Thoracic spine MRI sagittal T2-weighted image showing the minimal impression on the dural tube at T7–8 level (arrow) thought to be related to bony elements at the apex of the kyphosis, rather than a traumatic disc. This may represent a combination of retropulsion of bone fragment and an acute traumatic posterior disc injury. Note the kyphosis that has developed in the thoracic spine due to the T7 compression fracture.

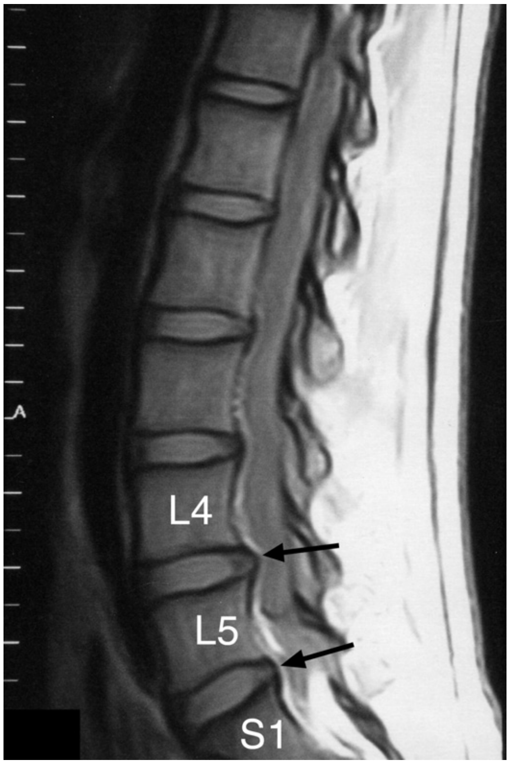

Figure 96.7 Lumbar spine MRI parasagittal T1-weighted image showing the L4–5 and L5–S1 annular bulges (arrows) elevating the posterior longitudinal ligament and pressing upon the pain sensitive anterior surface of the dural tube.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. After the imaging findings were explained to her, she was referred to an orthopaedic surgeon for a consultation and examination. He advised conservative treatment using non-steroidal anti-inflammatory drugs and swimming for exercise. When I saw her some 3 years later, she said she still experienced pain at approximately the T6–8 level where she had originally noted a bony gibbus. However, there was less radiation of pain to her left side.

KEY POINTS

1. When a patient is flung out of a vehicle at high speed it is important to thoroughly investigate all painful areas of the spine and to include, for example, the ribs, contents of the chest and abdomen when symptoms and signs are present.
2. Equally important is that imaging should be carefully scrutinized and reported upon.

Case 97

Spinal canal cyst

COMMENT

Beware of symptoms of gradual onset.

PROFILE

A 50-year-old slim female who works as a librarian. She stopped smoking cigarettes approximately 3 months ago and only occasionally drinks wine.

PAST HISTORY

There is a history of recurrent depressive disorder that is well controlled by her psychiatrist prescribing. However, she is concerned about her interscapular pain that was thought to be of 'idiopathic' origin.

PRESENTING COMPLAINT (Fig. 97.1)

Worsening central interscapular thoracic spine pain of a couple of years duration that came on gradually, i.e. without any known aetiology, and sometimes radiates anteriorly and bilaterally (right greater than left) to the chest. A neurosurgical opinion had resulted in a diagnosis of mechanical back pain and referral for physiotherapeutic mobilization but this treatment aggravated her symptoms. The thoracic spine pain is aggravated by exercise, 'prolonged' sitting, lifting, walking and housework. Although her symptoms are relieved to some extent by lying down, they are increasing in severity and awaken her at night, at which time she has to walk around before going back to sleep, i.e. she has night pain.

She derived marginal benefit from non-steroidal anti-inflammatory drugs (Vioxx 12.5 mg morning and night) and an analgesic (Di-Gesic). She was referred by her psychiatrist who felt that her concerns regarding her interscapular pain were well founded and that previously undertaken plain X-ray films of her chest and thoracic spine were not an adequate examination for her interscapular pain.

AETIOLOGY

The mid-thoracic spine pain began insidiously.

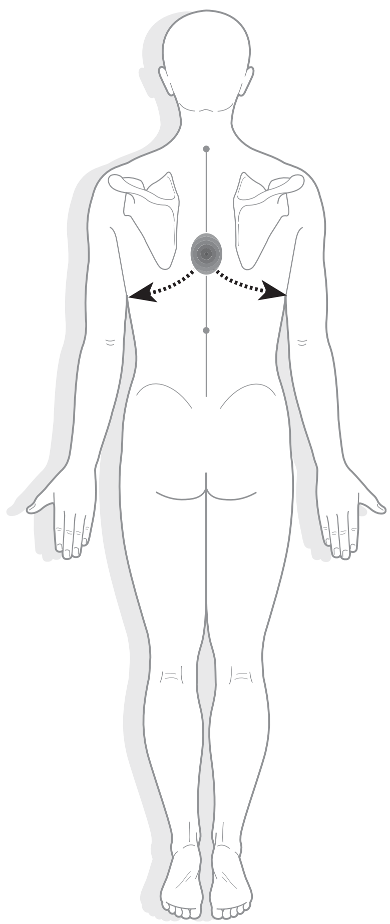

Figure 97.1

EXAMINATION

On examination there was no clinical evidence of pelvic obliquity or of leg length inequality or scoliosis. There was some minor increase in the thoracic kyphosis. Thoracic spine ranges of movement appeared to be reasonable for her age. Deep palpation of the paraspinal muscles in the mid-thoracic spine elicited some pain as well as muscle spasm bilaterally. The deep reflexes in the upper and lower extremities were normal as was the case with pinprick sensation. Pinprick sensation over the torso did not elicit any abnormality. The Valsalva manoeuvre caused some aggravation of her symptoms. Bowel and bladder function were reported as normal.

IMAGING REVIEW

Chest and thoracic spine plain X-ray films were reported as showing: 'Hyper expanded lungs with features suspicious of emphysema in both upper lobes. No pulmonary mass is demonstrated. No focal infection or pleural fluid. No hyla or mediastinal lymphadenopathy. Heart size is normal and there is no evidence of failure. Multilevel osteophytic marginal lipping in the thoracic spine with no evidence of bony infiltration or compression fractures'.

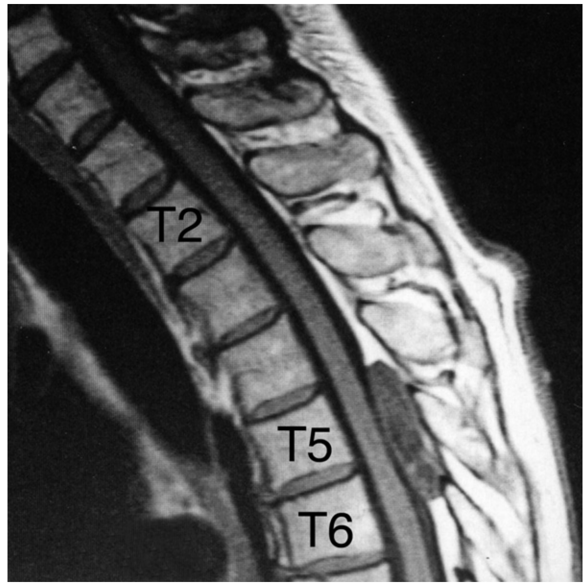

Figure 97.2 Thoracic spine MRI sagittal T1-weighted image showing the T5–6 level ovoid lesion measuring 3.7 cm in length × 0.9 cm in its anteroposterior diameter involving the posterior epidural space.

CLINICAL IMPRESSION

A possible space-occupying lesion in the mid-thoracic spine in view of the history and positive Valsalva manoeuvre.

WHAT ACTION SHOULD BE TAKEN?

An MRI of the thoracic spine was requested. The report stated: 'At T5–6 level the T1-weighted sagittal images show an ovoid lesion measuring 3.7 cm in length × 0.9 cm in its anteroposterior diameter involving the posterior epidural space (Fig. 97.2). On T2-weighted images the lesion is hyper-intense (Fig. 97.3) and shows a small hypo-intense component inferiorly. On T2-weighted axial images (Fig. 97.4) the lesion is seen to encroach the T5–6 neural exit foramina, particularly on the right side. Diffusion images show the lesion appears hypo-intense – therefore it is unlikely to be an epidermoid cyst. Post contrast sagittal and axial images do not indicate any significant enhancement in the lesion. Impression: Posterior epidural space-occupying lesion at T5–6, unlikely to represent an epidermoid cyst and represents an arachnoid cyst'.

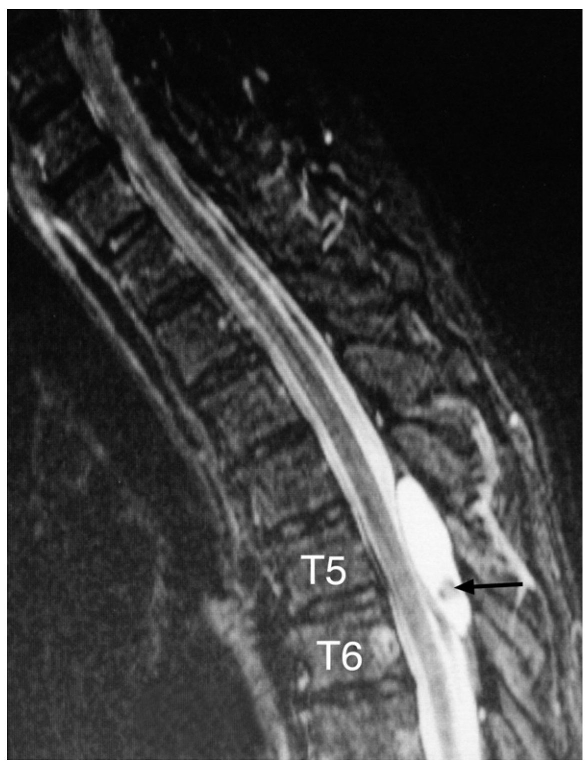

Figure 97.3 Thoracic spine MRI sagittal T2-weighted image. Note that the lesion is hyperintense compared to the surrounding CSF and has a small hypo-intense component inferiorly (arrow).

DIAGNOSIS

A posterior epidural space arachnoid cyst.

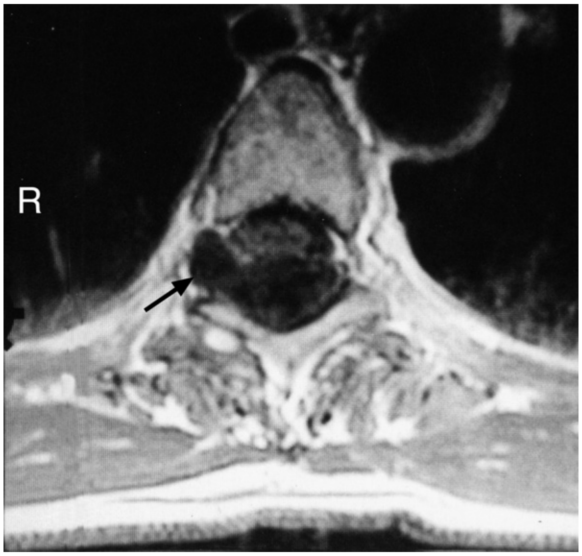

Figure 97.4 MRI axial T1-weighted post gadolinium image showing that the lesion is seen to encroach upon the T5–6 neural exit foramina, particularly on the right side (black arrow).

TREATMENT AND RESULTS

The MRI findings were explained to the patient and she was told that the lesion appeared to be non-malignant. She was pleased to know that she had a genuine condition causing her symptoms as she felt that she had been considered as a malingerer in view of her psychiatric history. She was referred to a neurosurgeon for a neurosurgical opinion and surgery was performed with a successful outcome.

KEY POINT

Clinicians need to consider a person as a whole and not categorize them as being a malingerer until the clinician is quite certain that there is no underlying hitherto undiagnosed cause.

Case 98

Scheuermann's disease – or is it?

COMMENT

Frequently, imaging reports incorrectly make a diagnosis of Scheuermann's disease to the detriment of patients, particularly in the context of medicolegal cases.

PROFILE

A slim 28-year-old female who smokes approximately 30 cigarettes per day but does not drink alcohol. She works in a managerial capacity.

PAST HISTORY

One year before consultation she was a rear seat-belted passenger in a car, sitting with her head turned to the right side, when the vehicle in which she was travelling hit the car in front. At the time of impact she felt 'something happen to her neck and between the shoulder-blades, resulting in spinal pain at these levels. She was given a neck collar when the ambulance came and took her to hospital where she was examined and an X-ray was taken of both her neck and thoracic spines; she was told that these were 'normal'. She was given an analgesic medication and discharged.

PRESENTING COMPLAINT(S) (Fig. 98.1)

She was referred for an opinion only to determine (i) whether she has Scheuermann's disease, and (ii) what is the cause of her symptoms of:

1. Severe mid-scapula thoracic spine pain that is essentially central. There is no radiating intercostal pain. Pain severity is activity related, with various activities causing a considerable increase in symptoms, e.g. vacuuming, sweeping or mopping. Bending forwards, for example to cut her toe nails, causes a considerable increase in the pain and will also cause some pain in the sternal region. The thoracic spine pain is temporarily relieved by three Panadeine Forte tablets at a time, as required. There are no bowel or bladder problems.
2. Bilateral neck pain approximately once per week that may radiate up to the vertex and frontal areas causing headaches. Looking up may cause a sharp pain in the neck, so she normally avoids this. There is no associated nausea or dizziness. Bending her neck forward can cause pain to go from approximately the T4–6 level up to the occipital area.

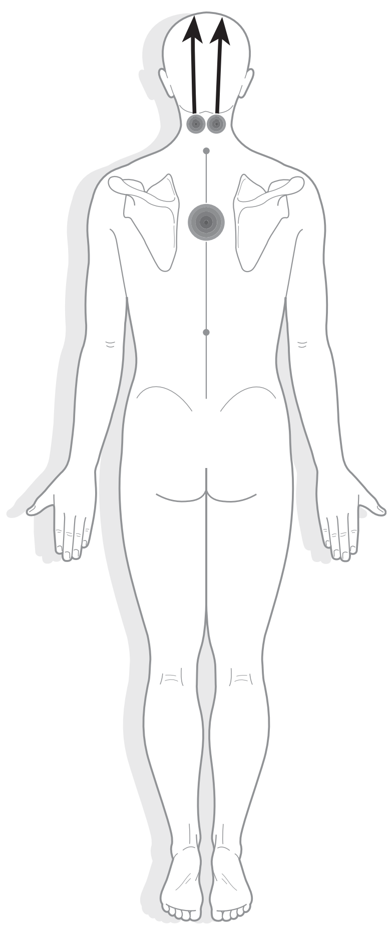

Figure 98.1

AETIOLOGY

A motor vehicle accident 1 year ago.

EXAMINATION

In the erect posture there was no evidence of pelvic obliquity or scoliosis. Percussion of the spine elicited pain at the T2–6 level. Deep palpation of the cervical paraspinal muscles elicited pain on the right side of C2–3 and at the T2–6 level of the thoracic spine. Deep palpation centrally between the spinous processes elicited pain at the T2–6 levels. The deep reflexes in the upper and lower extremities were normal. Vibration sensation at the elbows and ankles was normal. Pinprick sensation over the back of the upper torso and in the upper extremities was normal. Motor power in the upper and lower extremities appeared to be normal. The Valsalva manoeuvre caused an increase in spinal pain in the mid-scapula region.

Active thoracic spine ranges of movement were of full range and only left and right lateral bending and left and right rotation elicited pain at approximately the T4–6 levels.

Anteroposterior, lateral and oblique thoracic cage compression to check for pain in the rib cage were all painless.

Active cervical spine ranges of movement were measured as follows, using a CROM instrument (see Box 98.1).

Box 98.1 Active cervical spine ranges of movement

	Normal range	Measured range	Patient's comments
Flexion	50°	50°	Elicited pain on the right side of approximately C7–T1
Extension	60°	40°	Elicited a sharp twinge at approximately the C3–4 level
Left and right lateral bending	45°	30°	Painless
Left rotation	80°	68°	Painless
Right rotation	80°	72°	Painless

In order to further check for pain of cervical spine origin, the following tests were performed:

1. Left rotation plus extension; this elicited pain at approximately the T4–6 level on the right.
2. Right rotation plus extension – painless.
3. Cervical spine traction – painless.
4. Cervical spine compression – painless.
5. Downward shoulder pressure – painless.
6. Trapezius trigger point pressure; this elicited considerable pain in the left and right cervico-shoulder trigger points.

IMAGING REVIEW

Plain X-rays of the thoracic and cervical spines were reported as follows. Thoracic spine: 'No significant bony or disc space abnormality is detected'. Cervical spine: 'No significant bony abnormality is detected. The disc spaces and intervertebral foraminae are intact'.

However, on reviewing the cervical spine imaging the following were noted: (1) the cervical spine neutral lateral view (Fig. 98.2) shows loss of the normal lordosis, with slight kyphosis at the C4–5 level; (2) the flexion view (Fig. 98.3) appears to show a loss of normal contour with some angulation at the C4–5 level and slight anterolisthesis (3 mm) of C3 on C4 and of C4 on C5; and (3) the extension view (Fig. 98.4) shows a loss of normal contour with slight retrolisthesis of C2 on C3 and of C3 on C4.

CLINICAL IMPRESSION

Musculoligamentous soft tissue injuries of the upper to mid-thoracic spine and of the cervical spine.

WHAT ACTION SHOULD BE TAKEN?

An MRI of the thoracic spine was ordered. The report stated: 'Minor posterior bulging is present in the mid to upper

Figure 98.2 Cervical spine neutral lateral plain X-ray image. Note the loss of normal lordosis (forward bending) with some subtle kyphosis at the C4–5 level.

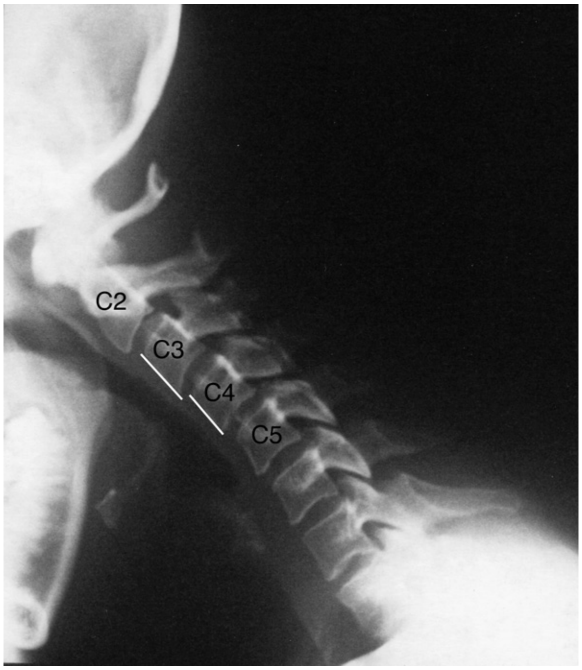

Figure 98.3 Cervical spine flexion plain X-ray image. Note the loss of normal contour with some angulation at the C4–5 level and anterolisthesis (see lines) of C3 on C4 and of C4 on C5.

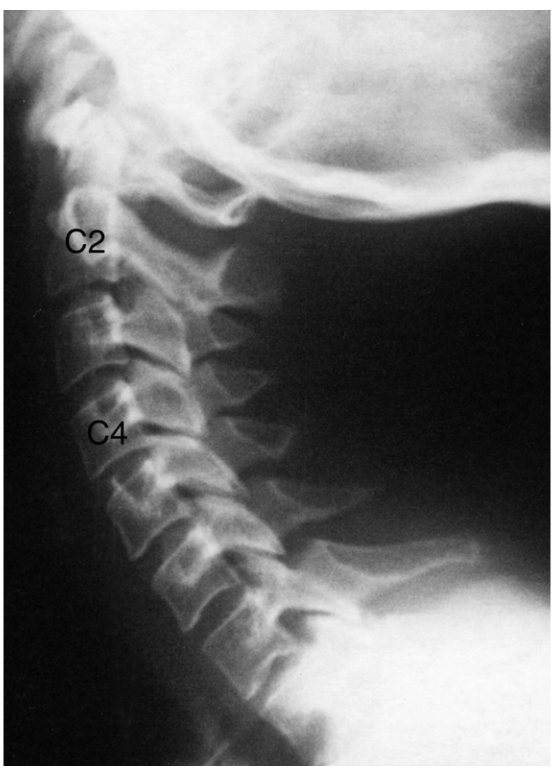

Figure 98.4 Cervical spine extension plain X-ray image. Note the loss of normal contour with slight retrolisthesis (arrows) of C2 on C3 and of C3 on C4.

thoracic spine (Fig. 98.5). The irregularity of the endplates in the mid and lower thoracic spine indicates Scheuermann's disease. The spinal canal is widely patent and the spinal cord appears normal'.

However, it should be noted that certain criteria have to be met for a diagnosis of thoracic spine Scheuermann's disease to be made. Scheuermann's disease usually occurs in at least three contiguous vertebrae, involving the anterior one-third of such vertebrae (Noakes 1998, Adams & Hamblen 2001), with wedging of 5° or more of each participating vertebral body (Sorensen 1964). Also, Scheuermann's disease is usually associated with an increase in the anteroposterior diameter of the vertebral bodies (Heitoff et al 1994).

According to Noakes (1998), the radiographic appearances of thoracic spine Scheuermann's disease include undulating irregular endplates, Schmorl's nodes which are anterior in position with sclerotic margins, loss of intervertebral disc height, kyphosis due to wedging and separated secondary ossification centres (limbus vertebra). Occasionally, a single vertebral body is involved (Adams & Hamblen 2001).

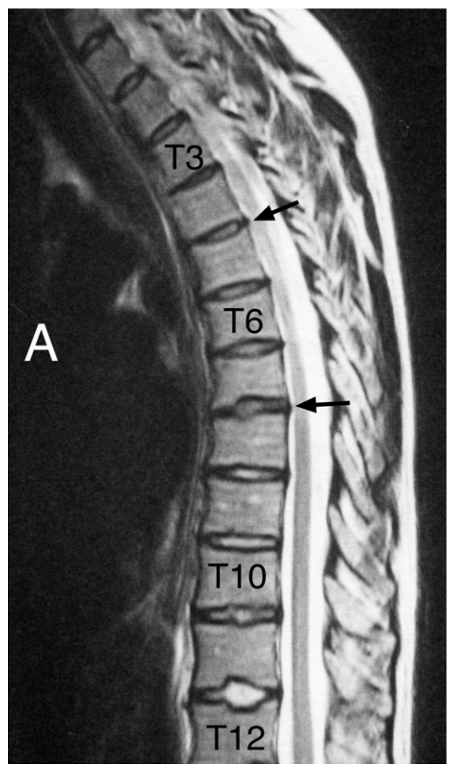

Figure 98.5 Thoracic spine MRI T2-weighted sagittal image. Note (i) the 'minor posterior bulging' of discs 'in the mid to upper thoracic spine' (black arrows), (ii) early desiccation of these discs indicating that these changes are not of longstanding, (iii) the very slight irregularity of the superior endplate of T11 centrally, and (iv) Schmorl's nodes centrally to posteriorly in the endplates adjacent to the T11–12 disc. There is no evidence to suggest Scheuermann's disease. A = anterior (front) side of patient.

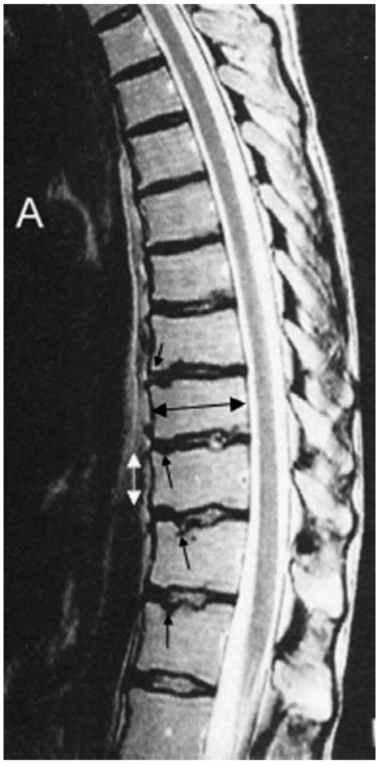

Figure 98.6 An example of Scheuermann's disease of the thoracic spine. Sagittal T2-weighted MR image showing (i) irregularity of three contiguous vertebral body end-plates anteriorly (short black arrows), (ii) some loss in vertebral body height anteriorly (white arrows), and (iii) an increase in the anteroposterior diameter (long black arrow) of three contiguous vertebrae. With permission from: McCall IW. Radiology of the thoracic spine. In: Giles L G F, Singer K P (eds) Clinical anatomy and management of thoracic spine pain. Oxford, Butterworth-Heinemann (2000) p 188.

Figure 98.5 is an MRI sagittal image of this patient's thoracic spine while Figure 98.6 is an MRI sagittal image of a patient who *does* have Scheuermann's disease. A comparison between the two figures, that takes into account the above criteria for Scheuermann's disease, shows that there is Scheuermann's disease in Figure 98.6; the thoracic spine does have (i) at least three contiguous vertebral bodies with endplate irregularities, (ii) anterior wedging of 5° or more of vertebral bodies, and (iii) an increase in the anteroposterior diameter of the vertebral bodies.

There is very slight irregularity of the superior endplate of T11 centrally with Schmorl's nodes centrally to posteriorly in the endplates adjacent to the T11–12 disc.

DIAGNOSIS

Cervical spine

Musculoligamentous soft tissue injuries.

Thoracic spine

- Musculoligamentous soft tissue injuries.
- Minor posterior disc bulges in the mid-to-upper thoracic spine.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. She was told that she does not have Scheuermann's disease.

Note

According to Coventry et al (1945), Fahey et al (1998) and Jensen et al (1994): 'A Schmorl's node refers to herniation of the disc material through the cartilaginous endplate into the adjacent vertebral body. Most consider Schmorl's nodes to be asymptomatic. However, some believe that Schmorl's nodes may be associated with pain (Hamanishi et al 1994). In this regard, most believe that the Schmorl's node itself does not cause pain, but that symptoms may occur as a result of damage to associated structures (Coventry et al 1945). The association between trauma and the subsequent development of a Schmorl's node may be particularly important in the medicolegal setting. Also, it has been reported that Schmorl's nodes may give rise to disc degeneration (Hilton et al 1976), therefore, Schmorl's nodes may be an important predictor of degenerative spine disease in later life.

KEY POINTS

Scheuermann's disease of the thoracic spine is reported frequently when, in fact, there is no justification for such a diagnosis, i.e. the criteria for Scheuermann's disease are not present. This may have profound adverse repercussions for patients involved in litigation over thoracic spine injuries as they may be classified as having a 'pre-existing condition'.

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Case 99

T6 intervertebral disc anterior protrusion and posterior bulge

COMMENT

To tell injured patients who feel genuine pain that they should have recovered within 6 weeks of an accident is offensive to genuine patients.

PROFILE

A 44-year-old male manual worker of muscular build who does not smoke cigarettes and only occasionally drinks alcohol.

PAST HISTORY

Ten months ago he was the seat-belted driver of a vehicle when he realized that another vehicle would hit his vehicle so, just before the impact, he tried to lie down across the car seats as he thought this may protect him. Immediately following the impact he experienced severe pain in his lower thoracic spine and in the left lower ribcage. He was taken by ambulance to hospital where plain X-ray films were taken of his chest and ribcage, which showed: 'a possible fracture of the left 8th rib and linear shadowing at the left base which may well indicate the presence of a pulmonary contusion'. Urinalysis was performed and found to be normal. The seat belt abrasions on the lower left side of his chest were disinfected, he was given analgesic medication, then discharged.

PRESENTING COMPLAINT (Fig. 99.1)

Constant pain of 10 months duration and of varying intensity on the left side of his lower thoracic spine that radiates periodically into the lower ribcage on that side and to the mid-clavicular line since a motor vehicle accident. Bearing down causes a slight increase in his pain. Lying down does not provide significant relief. On turning over at night, the pain may awaken him but there is no night pain per se. On arising of a morning, his left sided lower thoracic and lower ribcage pains are more pronounced. Work aggravates his symptoms. An intravenous pyelogram examination indicated that his kidneys were normal. He did not have any bowel or bladder problems. Physiotherapy manipulations of his thoracic spine apparently aggravated his symptoms so he stopped having this treatment and was prescribed non-steroidal anti-inflammatory drugs; these provided only temporary relief. He was frustrated as he had been told that he 'should have recovered within 6 weeks of the accident' and he got the impression that he was being thought of as a 'malingerer'. He said that all he wants to know is what is the cause of his symptoms and how he should avoid aggravating the symptoms.

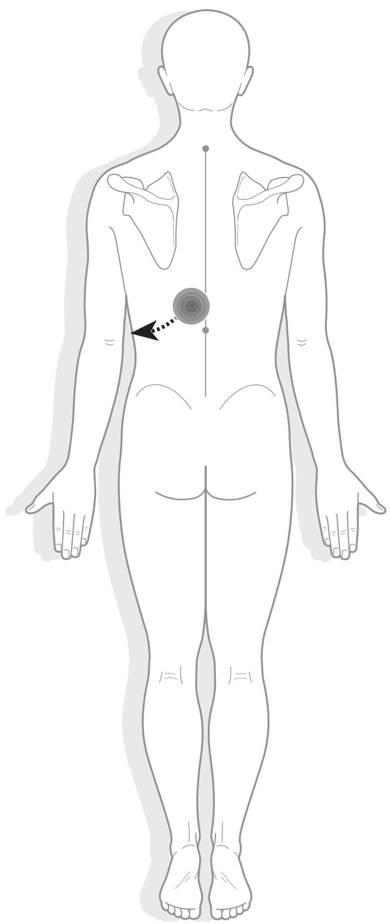

Figure 99.1

AETIOLOGY

A motor vehicle accident 10 months ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. A semicircular brown pigmented scar on the lower left side of his ribcage was noted (Fig. 99.2).

Percussion of the thoracic spine elicited pain at approximately the T9–10 level. Deep palpation of the paraspinal muscles elicited slight pain on the left side at approximately the T9–L1 levels. The deep tendon reflexes in the lower extremities appeared to be normal, as was the plantar response. Vibration sensation at the ankles was normal. Pinprick sensation over the posterior and lateral sides of the torso and the lower limbs was normal. In the supine position, straight leg raising was to a measured 90° (right) and painless; left straight leg raising was to 70° and aggravated the pain in the left lower ribcage area. Bilateral hip flexion aggravated his left lower ribcage pain and the addition of cervical spine flexion elicited a significant increase in this pain. The Milgram active bilateral straight leg raising test elicited an increase in his left lower ribcage pain. The superficial abdominal reflexes appeared to be normal as was Beevor's sign.

Active thoracic spine ranges of movement were of full range but elicited pain on the left side of the lower thoracic spine and in the left lower ribcage as far as the mid-axillary line. Ribcage anteroposterior and lateral compression were painless; oblique compression elicited pain on the left anterior side of his lower ribcage.

Figure 99.2 Seatbelt abrasion on the lower left side of his ribcage.

IMAGING REVIEW

Plain X-rays of the chest and left ribs were reported as showing: 'a possible fracture of the left 8th rib and linear shadowing present at the left base which may well indicate the presence of a pulmonary contusion'. Plain X-rays of the cervical, thoracic and lumbosacral spine were reported as showing 'no fracture or dislocation. Minor degenerative changes are present in the thoracic and lumbar spines although the disc spaces are all of normal height'. Plain X-ray films taken approximately 1 month post the accident showed the following: 'The heart and mediastinum are normal. Both lungs are clear. No abnormality is seen in the pleural space. No rib fracture seen'.

CLINICAL IMPRESSION

Musculoligamentous soft tissue injuries to his lower thoracic spine and to the left side of his lower thorax.

WHAT ACTION SHOULD BE TAKEN?

A thoracic spine MRI was requested and the report stated: 'No disc herniation or spinal canal stenosis. Thoracic nerve roots appear to exit satisfactorily. The thoracic cord appears to be normal'. No mention was made of the early modic changes in the inferior endplate of the T9 vertebral

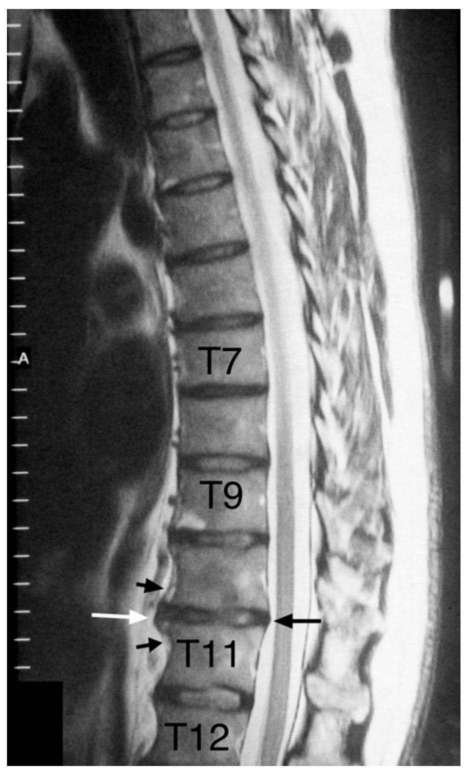

Figure 99.3 MRI thoracic spine sagittal T2-weighted image. Note the T10–11 invertebral disc changes, i.e. (i) early desiccation, (ii) anterior disc protrusion (white arrows) that elevates the anterior longitudinal ligament (small black arrows), (iii) very small posterior disc bulge (long black arrow) that indents the pain-sensitive anterior surface of the dural tube, and (iv) early thinning of the disc.

body (Fig. 99.3) or of the T10–11 intervertebral disc changes, i.e. (i) early desiccation, (ii) anterior disc protrusion, (iii) very small posterior disc bulge, and (iv) early thinning of the T10–11 disc.

DIAGNOSIS

1. Musculoligamentous soft tissue injuries on the left side of his lower thorax.
2. T10–11 intervertebral disc anterior protrusion and posterior bulging.
   a. The anterior protrusion will, in all likelihood, press upon the sympathetic nervous plexus (Fig. vi). This would most likely result in the vertebrogenic autonomic pain syndrome described by Jinkins et al (1989).
   b. The posterior bulging disc pressing upon the pain sensitive anterior surface of the dural tube will, in all likelihood, cause local pain which could be referred up and down a few segments as described by Groen (1986) and Groen et al (1988).
3. Possible costovertebral joint injuries involving the lower thoracic spine on the left side.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Once the situation was explained to him he said he knew there was a reason for his genuine pain and that it was offensive to have previously been told that he should have recovered within 6 weeks of the accident. He said he would avoid aggravating factors and would be careful to work within his limitations.

KEY POINTS

1. Clinicians should consider patients as being honest unless the clinician has clear-cut findings to the contrary (Giles 2005).
2. Clinicians should not draw the conclusion that a patient is psychoneurotic as this is a reflection on the examiner's diagnostic ability and not on the patient, until proven otherwise (Jackson 1977).

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Case 100

T5–6 and T11–12 intervertebral disc posterior protrusions

COMMENT

Thoracic spine pain usually is not given much coverage in orthopaedic texts in spite of it causing distress to many patients.

PROFILE

A 30-year-old female manual worker who is a non-smoker and only occasionally drinks alcohol.

PAST HISTORY

Approximately 16 months ago she lifted a heavy item at which time she felt a strain in her thoracic spine. The following day she felt severe pain in her chest and mid-to-upper thoracic spine, so she went to see her medical practitioner where an ECG was performed and an analgesic was prescribed. She was told her symptoms were due to 'indigestion'. Three days later she again had to go to the medical centre regarding her chest and thoracic spine pains. This time she was told that she had a musculoskeletal thoracic spine problem, so she was given a non-steroidal anti-inflammatory drug.

PRESENTING COMPLAINT(S) (Fig. 100.1)

Constant central thoracic spine pain at approximately the T5–6 and T11 levels since lifting heavy equipment approximately 1 year 4 months ago before seeing me. The T5–6 level pain is the source of greatest pain. The pain does not radiate to the left or right sides but extends down a few segments from the T5–6 level. When her T5–6 level thoracic spine is particularly painful, her chest anteriorly can feel 'tender' at about this same level. Pain intensity varies depending on her activity. She experiences broken sleep due to the T5–6 level spinal pain. Lying on a bed, rather than sitting, is less aggravating for her mid-thoracic spine pain.

Sitting or standing for more than 30 minutes, and bearing down aggravate her mid-thoracic spine pain. On awakening in the morning, her mid-thoracic spine is very painful. However, lying down, moving about, and heat applied on the painful area provide temporary relief as does Panadeine Forte. Walking of a morning, then getting under the hot shower also provides temporary relief.

She said she had not experienced any bowel or bladder problems.

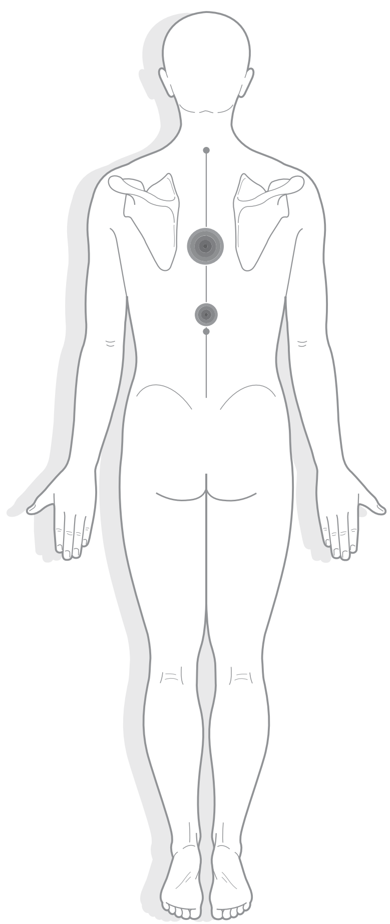

Figure 100.1

AETIOLOGY

Lifting heavy items 16 months ago.

EXAMINATION

In the erect posture there was no clinical evidence of pelvic obliquity or of scoliosis. Percussion of the thoracic and lumbar spines did not elicit any pain. Deep palpation of the paraspinal muscles elicited pain at the T5–6 and T11 levels. Motor power in the upper and lower extremities was normal. The deep reflexes in the upper and lower extremities were normal, as was the plantar response. Vibration sensation at the elbows and ankles was normal. Pinprick sensation across the back of the thorax did not elicit any sensory abnormality. When seated in the slumped forward position, this aggravated the pain at the T5–6 level. The Valsalva manoeuvre elicited an increase in pain at the T5–6 and T11 levels. Supine straight leg raising did not elicit any pain. The Milgram active bilateral straight leg raise elicited some discomfort in the mid-thoracic spine. Bilateral hip flexion did not elicit any pain but the addition of cervical spine flexion elicited an increase in pain at the T5–6 level. Naffziger's jugular compression plus cough test elicited slight pain at the T5–6 level.

Active thoracic spine ranges of movement appeared to be of full range and painless apart from left and right rotation both of which elicited pain at the T5–6 level. Thoracic spine compression in the anteroposterior, lateral and oblique planes did not elicit any pain.

Figure 100.2 Thoracic spine MRI sagittal T2-weighted image. Note (i) the midline posterior disc protrusion at T5–6 (black arrow) effacing the pain sensitive anterior surface of the dural tube, (ii) the anterior (large white arrow) and posterior (small white arrow) disc bulges at the T11–12 level, and (iii) desiccation at the T4-5, T5–6 and T11–12 intervertebral disc levels.

IMAGING REVIEW

A chest X-ray was reported as follows: 'Lung fields and pleural spaces are clear. The cardiomediastinal contour is normal. No chest wall abnormality is identified'.

A CT thoracic spine concluded: 'Normal examination'.

CLINICAL IMPRESSION

Posterior intervertebral disc bulge injury. (The Valsalva manoeuvre, Naffziger and Milgram tests are positive for a space-occupying lesion within the spinal canal at the T5–6 level.)

WHAT ACTION SHOULD BE TAKEN?

An MRI investigation was requested. The sagittal T2-weighted image showed a midline posterior disc protrusion at T5–6 (Fig. 100.2) effacing the pain sensitive anterior part of the dural tube. There is also a small posterior disc bulge at the T11–12 level with greater anterior bulging. Early desiccation is present at the T4-5, T5–6 and T11–12 levels.

The axial image through the T5–6 intervertebral disc level (Fig. 100.3) shows the central to slightly right sided posterior disc protrusion impinging upon the dural tube anteriorly.

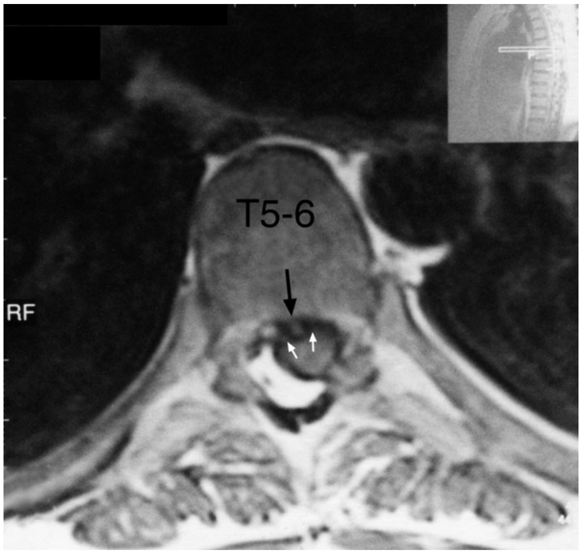

Figure 100.3 Thoracic spine MRI axial T1-weighted image at the T5–6 level. Note the protrusion (arrow) that is essentially central and impinges upon the dural tube anteriorly (small white arrows).

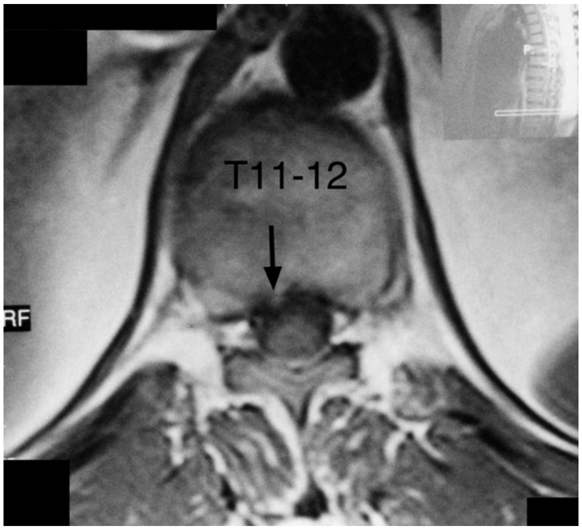

Figure 100.4 Thoracic spine MRI axial T1-weighted image at the T11–12 level. Note the right paracentral disc protrusion (arrow).

The small right paracentral disc protrusion at the T11–12 level is shown in Figure 100.4.

DIAGNOSIS

Musculoligamentous soft tissue injuries with objective evidence of a small posterior disc protrusion at T5–6. A T11–12 posterior disc protrusion impinging upon the dural tube anteriorly with an anterior disc bulge at the T11–12 level.

TREATMENT AND RESULTS

The patient's condition was clearly explained to the patient using the approach outlined in the Introduction, under Treatment, to ensure that the likely cause of symptoms was understood. Analgesic and non-steroidal anti-inflammatory drug treatment did not appear to have been of much benefit but her main concern was to determine what was causing her pain rather than seeking treatment as she thought there could be a malignancy because previous imaging had been reported as being 'normal' in spite of her genuine pain. She was advised to change her job to a light duties only occupation so she obtained a secretarial position.

Note

Pain in the thoracic spine represents a diagnostic and treatment challenge; often appearing as localized somatic pain, symptoms must be interpreted with the consideration that pain may be referred from other tissues, i.e. in the neck, shoulder, pulmonary, or subdiaphragmatic structures (Erwin 2005).

KEY POINTS

1. Because the small right paracentral disc protrusion at T5–6 is essentially central it impinges on the pain sensitive anterior part of the dural tube and not on the nerve roots. Therefore, one would not expect her to have neurological signs, e.g. pinprick sensation. However, she did have positive signs in that tests to increase pressure within the spinal canal, e.g. the Valsalva manoeuvre, did aggravate her T5–6, and to some extent T11, pain.
2. Because the dural nerves on the anterior part of the dural tube may extend up to eight spinal segments, with a great amount of overlap between adjacent nerves, this may provide an anatomical basis for extra-segmentally referred dural pain (Groen et al 1988).
3. The work of Willen et al (1997) showed that, when CT or MR images were performed of patients in the supine position, images obtained in that position illustrated smaller disc bulge or protrusion than when the spine was under loading. The same principle applies in this case as her MRI was performed in the supine position without loading of her thoracic spine.

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Questions and answers

QUESTIONS REGARDING (I) SPINAL PAIN SYNDROMES, AND (II) SPINAL ANATOMY FROM AN IMAGING AND A HISTOPATHOLOGY PERSPECTIVE

1. List five possible causes of pain arising from the zygapophysial 'facet' joints.

ANSWER:

a. Osteoarthrosis.
b. Synovial fold pinching.
c. Osteophytic encroachment upon the exiting nerve root.
d. Adhesions within the fibrous joint capsule.
e. Synovial cyst.

2. List five possible causes of pain arising from the intervertebral disc.

ANSWER:

a. Posterior central bulging or protrusion effacing the dural tube.
b. Posterolateral bulging or protrusion compromising the dural tube and/or the nerve root.
c. Far lateral disc protrusion compromising the adjacent spinal nerve.
d. Leakage of nuclear material to contact a nerve root.
e. Internal disc disruption.

3. Which structure comprising part of the motion segment is not innervated?

ANSWER:

The hyaline articular cartilage of the zygapophysial joints.

4. List the two main branches of the lumbar posterior primary ramus of the spinal nerve.

ANSWER:

a. Lateral branch.
b. Medial branch.

5. What is the minimum innervation of the zygapophysial joint's fibrous capsule?

ANSWER:

At least the articular branch from the posterior primary ramus from the adjacent intervertebral foramen and from the descending branch from the spinal nerve emitting from the intervertebral foramen one level above.

6. What is the significance of a positive Valsalva manoeuvre, i.e. if bearing down causes pain in the spine or radiating pain down the legs?

ANSWER:

A possible space-occupying lesion within the spinal canal causing intrathecal pressure or involving the theca itself.

7. When a large central posterior intervertebral disc protrusion occurs above the cauda equina, what seven structures are likely to be involved, progressing posteriorly from the protrusion?

ANSWER:

a. Posterior longitudinal ligament.
b. Recurrent meningeal nerve.
c. Blood vessels within the anterior epidural space.
d. The anterior surface of the dural tube.
e. Sub-arachnoid space containing the cerebrospinal fluid.
f. The anterior spinal artery and vein.
g. The anterior surface of the spinal cord.

8. A patient presents with a history of acute low back pain with left buttock pain, sciatica and paraesthesiae in the left foot with left leg cramp following a twisting injury to his low back. Examination elicited a loss of pinprick sensation in the S1 dermatome of the left foot and hypoaesthesia to pinprick sensation in the perianal region and a decrease in anal sphincter tone. What is the likely diagnosis?

ANSWER:
Diagnosis = Cauda equina syndrome.

9. A patient presents with spinal pain and 'night sweats'. What are two serious conditions to be considered?

ANSWER:
a. Malignancy, e.g. lymphoma.
b. Spinal tuberculosis.

10. What signs would alert you to the possibility of adolescent tethered cord syndrome in a patient presenting with non-specific low back pain and leg aches?

ANSWER:
a. No paravertebral muscle spasm.
b. No lumbosacral spine tenderness.
c. Exaggerated lumbar lordosis.
d. Scoliosis.
e. Deformity of foot or leg.
f. Weakness of (i) extensor hallucis longus, (ii) peroneus longus, (iii) posterior tibialis, (iv) anterior tibialis, or (v) gastrocnemius muscles.
g. Flabby or atrophic muscles in the lower extremities.
h. Ankle joint instability on toe or heel walking.
i. Pinprick sensation diminished (i) on the dorsum of the foot, (ii) perianal area and groin, and (iii) patchy sensation in the lower limbs.
j. Diminished or lost anal wink reflex.
k. Diminished sphincter tone reflex on voluntary contraction during digital insertion.
l. Hypoactive deep tendon reflexes.
m. Normal straight leg raising test.
n. Is post-void residual urine present?

11. A patient presents with pain in the right shoulder but passive movements of the shoulder are painless. What should be the first and second considerations?

ANSWER:
a. Referred pain.
b. Pain referral most likely from a visceral pathology, e.g. Pancoast's tumour.

12. Night pain in the spine or pelvis, without fever, may be a sign of serious pathology such as?

ANSWER:
Malignancy.

13. Can a vertebral body haemangioma become symptomatic and, if so, when?

ANSWER:
a. Yes
b. When a haemangioma expands posteriorly into the spinal canal and causes compression of the spinal cord.

14. What is the name of a stress fracture that can occur in the sacrum of an osteoporotic person?

ANSWER:
An insufficiency fracture.

15. What can be the pathological consequence of vascular compromise to the spinal cord?

ANSWER:
Myelomalacia as a result of demyelination.

16. Which MR imaging technique is best for showing a stress fracture in the pars interarticulares?

ANSWER:
Short-time inversion recovery (STIR) technique.

17. What is the sensitivity of (i) plain X-ray, and (ii) MRI for bone pathology causing bone loss?

ANSWER:
a. Plain X-ray approximately 40% bone loss.
b. MRI 2–3% bone loss.

18. What is the likely importance of retrolisthesis of L5 on S1? Give two possibilities.

ANSWER:
a. A posterior bulge or protrusion of the L5–S1 intervertebral disc.
b. Congenitally short pedicles.

19. Is scanography a precise method for measuring overall leg length inequality? Please qualify your answer.

ANSWER:
a. No.
b. Scanography accurately measures the length of the femur and tibia on each side but does not take into account compression of soft tissues within the lower limbs or foot pronation.

20. A woman presents with low back pain and 'bloating'. What diagnosis should be considered?

ANSWER:
Ovarian cancer.

21. Is there any known pre-manipulative test that can be performed to categorically prevent a stroke occurring?

ANSWER:
No.

22. What appear to be the 3 most useful methods for preventing a stroke?

ANSWER:
a. To make an appropriate diagnosis to exclude cases where manipulation is contraindicated.
b. Never to perform rotational manipulation of the cervical spine.
c. For manipulation to be performed only by appropriately trained clinicians.

23. What imaging findings are necessary for making a definitive diagnosis of thoracic spine Scheuermann's disease?

ANSWER:
a. Scheuermann's disease usually involves the anterior one-third of at least three contiguous vertebrae.
b. $\geq 5°$ anterior wedging of each participating vertebral body.
c. An increase in the anteroposterior diameter of the involved vertebral bodies.
d. Loss of associated intervertebral disc height.
e. Occasionally a single vertebral body is involved.

24. When an appearance of a large intervertebral disc protrusion or extrusion is seen on MRI, what differential diagnosis should be considered?

ANSWER:
An epidural haematoma.

25. What spinal level is the commonest site for fractures?

ANSWER:
The thoracolumbar junction (T11–L2).

26. In a closed head injury, what structure can be severed?

ANSWER:
The pituitary stalk.

27. List two tests for an upper motor neuron lesion.

ANSWER:
a. Hoffmann's sign.
b. Babinski response.

28. When an intervertebral disc posterior protrusion occurs, what is likely to be the main cause of (i) central spinal pain only, and (ii) unilateral radiculopathy?

ANSWER:
a. A central protrusion.
b. A protrusion compressing a spinal nerve root.

29. What would be the likely clinical findings for posterolateral intervertebral disc protrusion at the following lumbar levels?

1. L3–4 posterolateral protrusion impinging upon the L4 nerve root.

ANSWER:
a. Sensory loss on the medial side of the ankle.
b. Decreased patellar reflex.
c. Motor weakness for knee extension (squat and rise).

2. L4–5 posterolateral protrusion impinging upon the L5 nerve root.

ANSWER:
a. Sensory loss over the dorsum of the foot.
b. No reliable deep tendon reflex.
c. Weakness of heel walking and big toe dorsiflexion.

3. L5–S1 posterolateral protrusion impinging upon the S1 nerve root.

ANSWER:
a. Sensory loss along the lateral side of the foot.
b. Decreased/absent Achilles reflex.
c. Weakness of toe walking/plantar flexion of big toe.

30. A patient presents with constant mild-to severe lumbar pain of unknown aetiology and of approximately 1 year's duration and she feels unwell. What diagnoses should be considered and what initial diagnostic tests should be performed?

ANSWER:
a. Possible diagnoses – pancreatic carcinoma or ovarian carcinoma.
b. Tests:
  i. A full blood count including liver function tests, ESR/CRP and tumour markers.
  ii. Upper and lower abdomen ultrasound.
  iii. Abdominal CT scan.

31. What are the anatomical structures labelled on the following MR images (Figs A to D)?

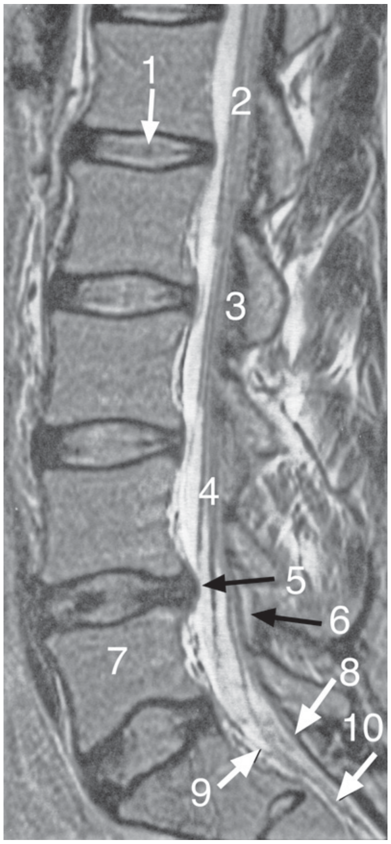

Figure A

ANSWER (Fig. A):

1 = Intranuclear cleft.
2 = Conus medullaris.
3 = Ligamentum flavum.
4 = Cauda equina.
5 = Posterior bulging intervertebral disc (raising the posterior longitudinal ligament and effacing the anterior surface of the dural tube).
6 = Retrothecal fat.
7 = Fifth lumbar vertebral body.
8 = Posterior surface of the dural tube.
9 = Anterior surface of the dural tube.
10 = Filum terminale.

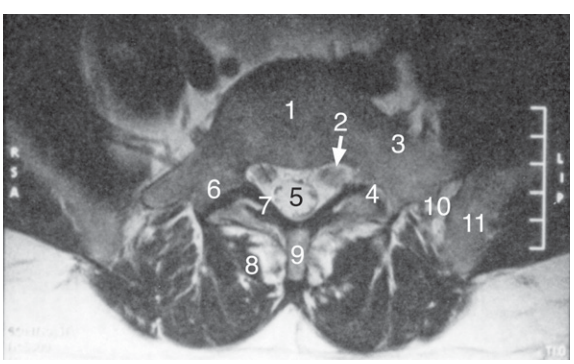

Figure B

ANSWER (Fig. B):

1 = Body of the 1st sacral segment.
2 = Nerve root.
3 = Ala of sacrum.
4 = Zygapophysial facet joint at L5–S1 level.
5 = Dural tube containing some cauda equina nerve roots.
6 = Superior articular process of sacrum.
7 = Ligamentum flavum.
8 = Multifidus muscle showing some atrophy and replacement with fibro-fatty tissue.
9 = Spinous process.
10 = Sacroiliac joint.
11 = Ilium.

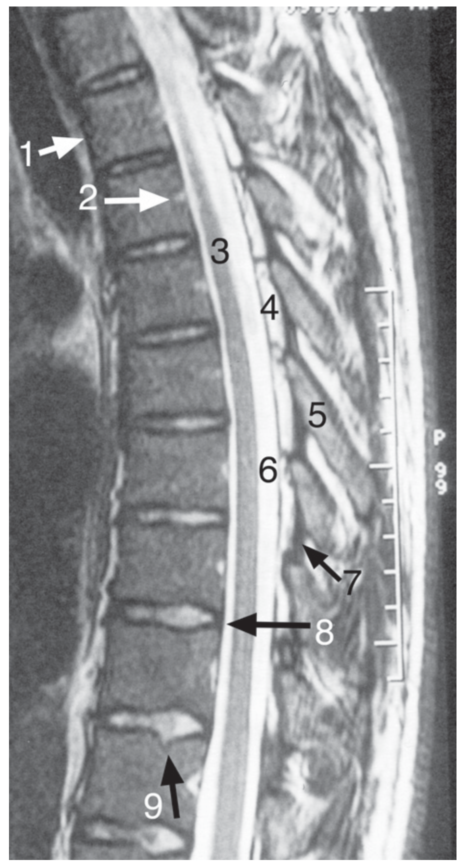

Figure C

ANSWER (Fig. C):

1 = Anterior longitudinal ligament.
2 = Basivertebral vein.
3 = Spinal cord.
4 = Epidural fat.
5 = Spinous process.
6 = Subarachnoid space.
7 = Ligamentum flavum.
8 = Posterior longitudinal ligament and annular fibres.
9 = Schmorl's node.

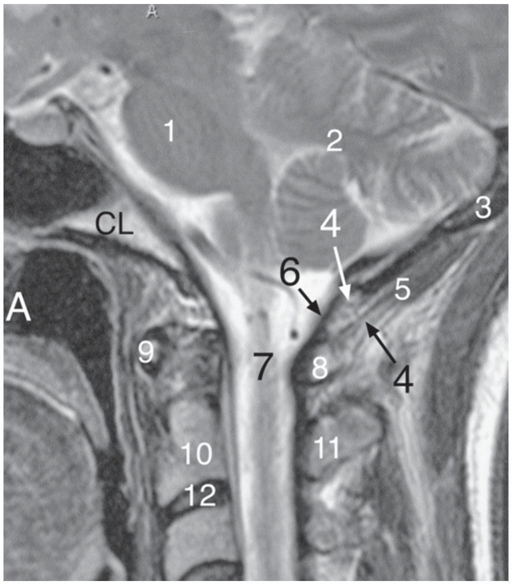

Figure D

ANSWER (Fig. D):
1 = Pons.
2 = Cerebellum.
3 = Occiput.
4 = Connective tissue bridge between the rectus capitis posterior minor muscle and the posterior cervical dura.
5 = Rectus capitis posterior minor muscle.
6 = Posterior cervical dura.
7 = Spinal cord.
8 = Posterior tubercle of atlas.
9 = Anterior tubercle of atlas.
10 = Body of C2 vertebra.
11 = Spinous process of C2 vertebra.
12 = Intervertebral disc at C2–3 level.
A = Anterior.

32. What are the anatomical structures labelled on the following histopathology sections (Figures E to I)?

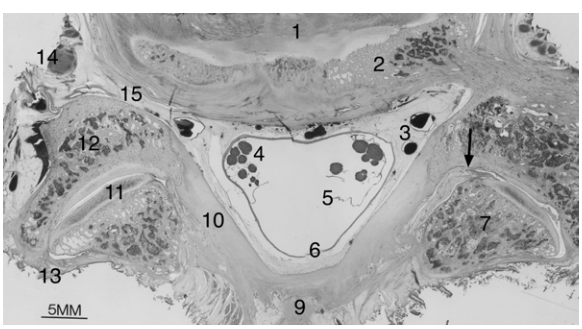

Figure E

ANSWER (Fig. E):
1 = Intervertebral disc.
2 = Sacrum.
3 = Batson's venous plexus.
4 = Cauda equina.
5 = Arachnoid membrane.
6 = Dural membrane.
7 = Inferior articular process of L5 vertebra.
8 = Epidural space.
9 = Spinous process remains.
10 = Ligamentum flavum.
11 = Hyaline articular cartilage on the facet surface of the zygapophysial joint.
12 = Fibrous capsule of the zygapophysial joint.
13 = Superior articular process of the sacrum.
14 = Spinal ganglion.
15 = Intervertebral canal.
Arrow = intra-articular synovial fold.

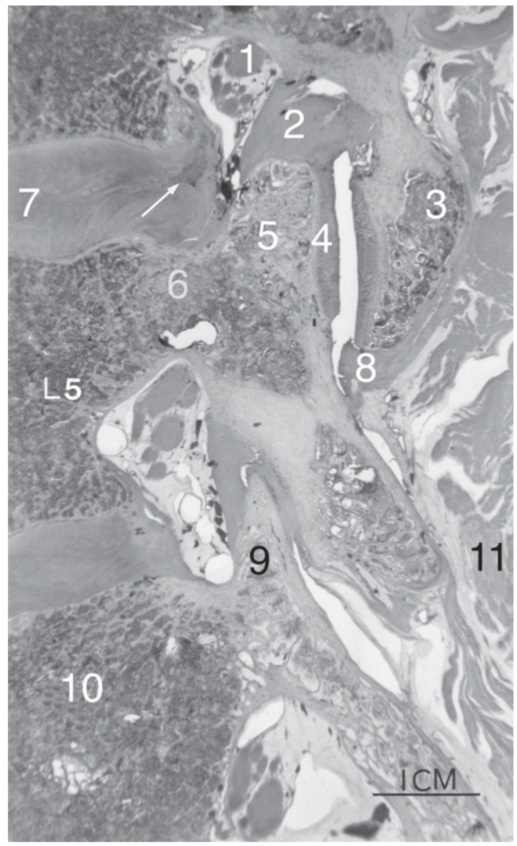

Figure F

ANSWER (Fig. F):
1 = Neural structures.
2 = Ligamentum flavum.
3 = Inferior articular process of L4 vertebra.
4 = Hyaline articular cartilage on the zygapophysial joint facet.
5 = Superior articular process of L5 vertebra.
6 = Pedicle.
7 = Intervertebral disc.
8 = Fibrous joint capsule inferiorly.
9 = Superior articular process of sacrum.
10 = Body of the 1st sacral segment.
11 = Muscles.
Arrow = intervertebral disc nuclear material moving posteriorly.

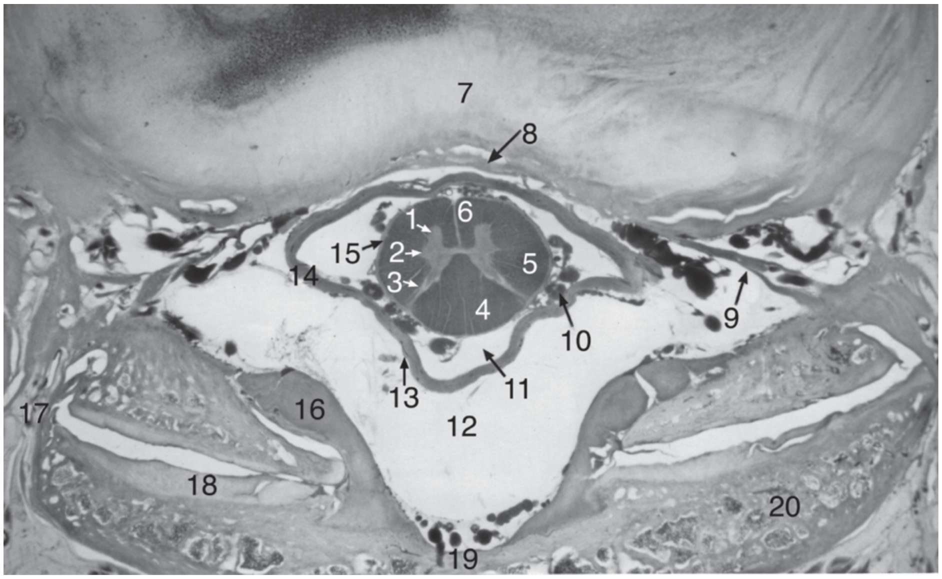

Figure G

ANSWER (Fig. G):

1 = Anterior grey column of the spinal cord.
2 = Lateral grey column.
3 = Posterior grey column.
4 = Posterior funiculus.
5 = Lateral funiculus.
6 = Anterior funiculus.
7 = Intervertebral disc.
8 = Posterior longitudinal ligament.
9 = Transforaminal ligament.
10 = Dorsal (posterior) nerve root.
11 = Subarachnoid space.
12 = Epidural/extradural space.
13 = Blood vessels within the dural membrane.
14 = Dural membrane.
15 = Pia mater.
16 = Ligamentum flavum.
17 = Fibrous joint capsule of the zygapophysial joint.
18 = Hyaline articular cartilage on the zygapophysial joint facet.
19 = Lamina junction.

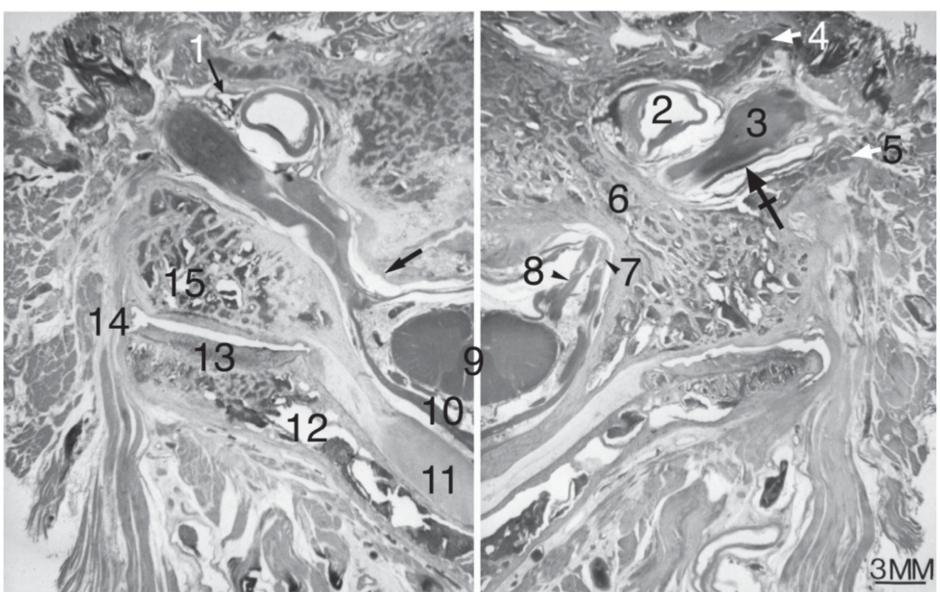

Figure H

ANSWER (Fig. H):

1 = Vein adjacent to the vertebral artery.
2 = Vertebral artery lumen.
3 = Spinal ganglion.
4 = Anterior tubercle of the transverse process.
5 = Posterior tubercle of the transverse process.
6 = Pedicle.
7 = Sensory root.
8 = Motor root.
9 = Spinal cord.
10 = Dural tube.
11 = Ligamentum flavum.
12 = Lamina.
13 = Hyaline articular cartilage on the zygapophysial joint facet.
14 = Fibrous capsule.
Tailed arrow = Intermediate neural branch blood vessel.
Arrow = early osteophytic spur.

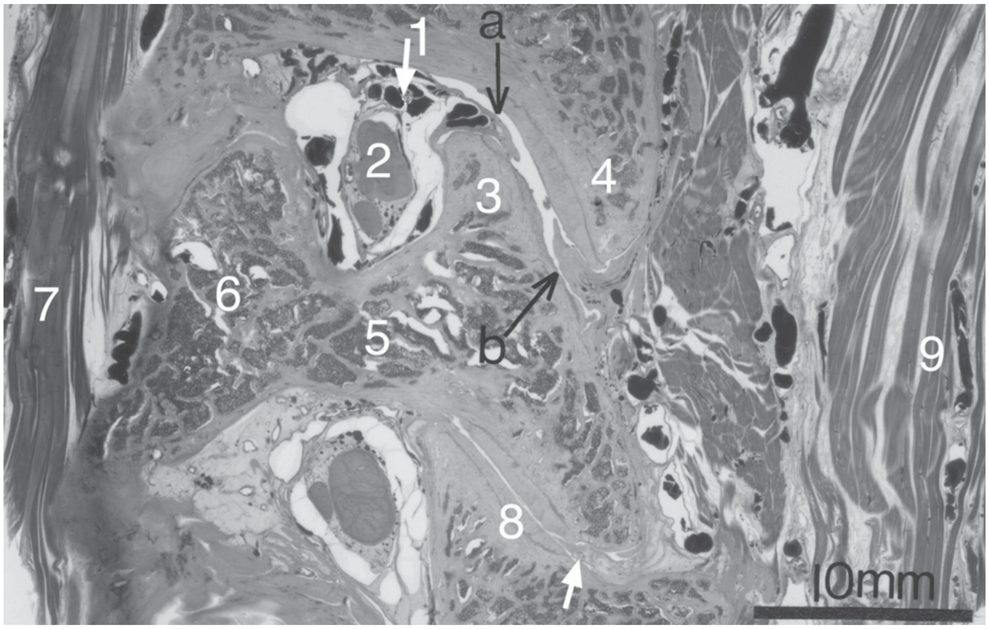

Figure I

ANSWER (Fig. I):

a = Synovial fold in the superior pole of the zygapophysial joint.
b = Synovial fold in the inferior pole of the zygapophysial joint.
1 = Blood vessels in the intervertebral foramen.
2 = Neural structures in the intervertebral foramen.
3 = Superior articular process.
4 = Inferior articular process.
5 = Pedicle.
6 = Vertebral body.
7 = Anterior spinal muscles.
8 = Hyaline articular cartilage on the zygapophysial joint facet.
9 = Posterior spinal muscles.
White arrow = synovial fold.

Conclusion

The reader will have realized the value of taking a thorough history, performing an appropriate physical examination and carefully looking at available or new imaging in an attempt to help the patient.

Patients do not normally like to be unwell. In my experience, there are very few malingerers, so one should be on one's guard not to be suspicious of a patient's motives without evidence to substantiate an impression of malingering.

Most clinicians are decent ethical practitioners but clearly errors occur in many fields of health care. We all make diagnostic or treatment errors from time to time but these errors should readily be acknowledged so that aggrieved patients can get a fair deal. I trust this book will give helpful insight to clinicians and make them aware of potential diagnostic pitfalls.

Patients presenting for medicolegal consultation and examination following a work-related injury, or a motor vehicle injury, require a thorough investigation and should not arbitrarily be classified as having a 'zero percent permanent incapacity', based on a superficial examination, particularly when they sustain a serious injury such as a moderately large spontaneous disc prolapse (Giles 2001). Obviously, the consulting clinician must be unbiased when providing evidence as an expert witness to the Court and that evidence should be based on a thorough investigation of the patient.

It must be remembered that it is a privilege to care for patients and that every endeavour should be made to make an appropriate diagnosis and to alleviate the physical pain and any psychological distress that many patients endure.

It is essential to perform an appropriate and thorough diagnostic work-up on which to base efficient best practice management. Patient management should include obtaining a signed informed consent form once treatment options and risks have been discussed. In addition, patient cooperation should be sought regarding their involvement in, for example, a healthy lifestyle including taking part in appropriate exercises and adhering to a proper diet for their particular needs.

Finally, clinicians require an intimate knowledge of the gross and histological anatomy of the spine upon which to base their diagnosis and subsequent treatment. Furthermore, clinicians dealing with spinal pain syndromes require a thorough understanding of normal and abnormal anatomy on imaging. They should also be aware of the limitations of imaging procedures, particularly with respect to recumbent versus upright and functional imaging procedures and the interpretation of such images.

The gross anatomical, histological and histopathological examples used in this text would not have been available were it not for the tremendous gift made by so many individuals when they bequeath their bodies to university departments where anatomical and histological/histopathological studies can be performed. Students of anatomy should remember the privilege associated with working on human material in order to better comprehend the human body.

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Giles L G F 2001 Medicolegal reporting for spinal injuries. Plaintiff 44: 21–23.

Definitions and abbreviations

Antalgic posture a posture assumed by patients experiencing acute low back pain, with or without leg pain, in which they lean away from the painful area.

Anteroposterior (A-P) the *position* of patients when an X-ray beam is directed to their anterior surface and an X-ray plate is positioned behind them. In this text, the A-P radiographs are *viewed* from behind the patient; the patient's right side is indicated by a right marker (R).

Cervical Range of Motion instrument (CROM) product of Performance Attainment Associates, St Paul, MN, USA.

Cloward procedure Cloward (1958) used an anterior interbody fusion operation for treating affected protruding discs and/or osteophyte complexes in the region of C4 to C7.

Coarctation of aorta a fibrous constriction in the aorta: 98% are distal to the origin of the left subclavian artery, but 2% may be abdominal or in the lower thoracic aorta. Usually presents as proximal hypertension due to mechanical obstruction and a low renal perfusion pressure. It is associated with congenital berry aneurysms in the cerebral circulation, and 60% of patients have a bicuspid aortic valve. Patients are often asymptomatic, but may experience leg fatigue or claudication (Pumphrey 1996). Coarctation causes systolic and diastolic hypertension in the upper extremities (Verrier 1994).

Cobb's method (1948) A method for measuring the angle of scoliotic spinal curvature. The angle of curvature is measured by drawing lines parallel to the superior surface of the most upper vertebral body of the curvature and to the inferior surface of the lowest vertebra of the curvature (Fig. xvi).

Degenerative spondylolisthesis (Pseudospondylolisthesis) is secondary to longstanding degenerative arthrosis of the lumbar zygapophysial joints and discovertebral articulations, without a pars separation (Yochum et al 1996).

Disability an alteration of an individual's capacity to meet personal, social, or occupational demands because of an impairment (Cocchiarella and Anderson 2000). Disability refers to an activity or task the individual cannot accomplish (Luck & Florence 1988).

Dura mater also known as dura or dural membrane.

Dural tube also known as dural sac or thecal sac. The dura mater forms the spinal dural sac, a long tubular sheath within the vertebral canal (Moore & Dalley 2006). The dura is the outermost of three spinal meninges, i.e. dura mater, arachnoid mater and pia mater.

Dysaesthesia an unpleasant abnormal sensation produced by normal stimuli (*Dorland's Illustrated Medical Dictionary* 1994).

EMG *Electromyography*: an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions and during electrical stimulation; performed using any of a variety of surface electrodes, needle electrodes, and devices for amplifying, transmitting, and recording the signals (*Dorland's Illustrated Medical Dictionary* 1994).

Haemangioma a general term denoting a benign or malignant vascular tumour that resembles the classic type of haemangioma but occurs at any age (*Dorland's Illustrated Medical Dictionary* 1994).

Hemiation brain hemiation results from increased intracerebral pressure.

Impairment a loss, loss of use, or derangement of any body part, organ system, or organ function (Cocchiarella & Andersson 2000) or any loss or abnormality of psychological, physiological, or anatomical structure or function (WHO 1980).

Intervertebral canal (intervertebral foramen, lateral canal, nerve root tunnel, radicular canal, root canal, interpedicular canal) is clinically a very important structure that exits from the spinal canal via the lateral recess (Dorwart & Genant 1983). Some authors use the term intervertebral foramina to describe both the osseous nerve root canals and the medial and lateral 'openings'; however, Dommisse (1975) correctly suggests that the term 'foramen' should only be used to describe the

Figure xvi An erect posture radiographic image of a 19-year-old male showing a right leg length deficiency of 21 mm, sacral base obliquity and postural scoliosis with a 17° angle of curvature. R = right side of the patient. Note the vertical plumb-line shadow that is used for measuring leg lengths by drawing a horizontal line from the top of each femur head to meet the plumb-line at right angles Sacral base obliquity is measured by drawing a horizontal line from each superior sacral notch to meet the plumb-line at right angles. The vertical difference between paired horizontal lines gives the difference in leg lengths and the difference in height between the superior sacral notches. (Reproduced with permission from Giles L G F 1989 Anatomical basis of low back pain. Williams & Wilkins, Baltimore.)

inner and outer boundaries of intervertebral canals. Therefore, the term intervertebral canal is used in this text.

Intervertebral disc conditions

Annular bulge refers to a concentric extension of the margins of the disc circumferentially beyond the vertebral margins (Hodges et al 1999).

Broad-based protrusion refers to protrusion of disc material extending beyond the outer edges of the vertebral body apophyses over an area greater than 25% (90°) and less than 50% (180°) of the circumference of the disc (Fardon & Milette 2001).

Contained herniation is when nuclear material does not escape from the confines of the annular fibres.

Extrusion is the extension of the nucleus completely through the outer annulus into the epidural space (Hodges et al 1999).

Herniation is defined as a localized displacement of disc material beyond the limits of the intervertebral disc space (Fardon & Milette 2001).

Protrusion is a focal area of extension of the nucleus beyond the vertebral margin that remains beneath the outer annular and posterior longitudinal ligament complex (Hodges et al 1999).

Sequestration is a specific type of extrusion in which there is a free disc fragment (Hodges et al 1999).

Intra-articular synovial fold a fibrous or highly vascular fat-filled zygapophysial joint synovial fold which is covered by the synovial lining membrane.

Intranuclear cleft A hypointense linear signal seen on a T2-weighted sagittal MR image as a continuous smooth band within the centre of the intervertebral disc. The intranuclear cleft represents normal appearance of central fibres inside the nucleus (Yrjama et al 1997).

'Leg length' inequality the absolute inequality in length of the lower limbs. In this text a 'significant leg length inequality' is referred to when an inequality of 9 mm or more is found using an accurate method for erect posture radiography (Fig. xvi).

Lhermitte's sign the development of sudden, transient, electric-like shocks spreading down the body on performing cervical spine flexion; it is seen mainly in multiple sclerosis but also in compression, inflammatory and other disorders of the cervical cord (*Dorland's Illustrated Medical Dictionary* 1994).

Manipulation (Cassidy & Kirkaldy-Willis 1988) The definition given by Sandoz (1976, 1981) is both clear and concise. A manipulation or lumbar intervertebral joint 'adjustment' is a passive manual manoeuvre during which the three-joint complex is suddenly carried beyond the normal physiological range of movement without exceeding the boundaries of anatomical integrity. The usual characteristic is a thrust – a brief, sudden, and carefully administered 'impulsion' that is given at the end of the normal passive range of movement. It is usually accompanied by a cracking noise.

Motion (Mobile) Segment of Junghanns all the space between two vertebrae where movement occurs: the intervertebral disc with its cartilaginous plates, the anterior and posterior longitudinal ligaments, the zygapophysial joints with their fibrous joint capsules and the ligamenta flava, the contents of the spinal canal and the left and right intervertebral canal, and the supraspinous and interspinous ligaments (Figure xvii)

Neuro-orthopaedic tests

Adson's test – used to determine the state of the subclavian artery, which may be compressed by an extra cervical rib or by tightened scalenus anticus and scalenus medius muscles, which can compress the artery where it passes between them on its way to the upper extremity. Take the patient's radial pulse at the wrist and while continuing to feel the pulse, abduct, extend, and externally rotate the arm; then instruct the patient to take a deep breath and to turn the head toward the arm being tested. If there is compression of the subclavian artery, a marked diminution or absence of the radial pulse will be felt (Hoppenfeld 1976).

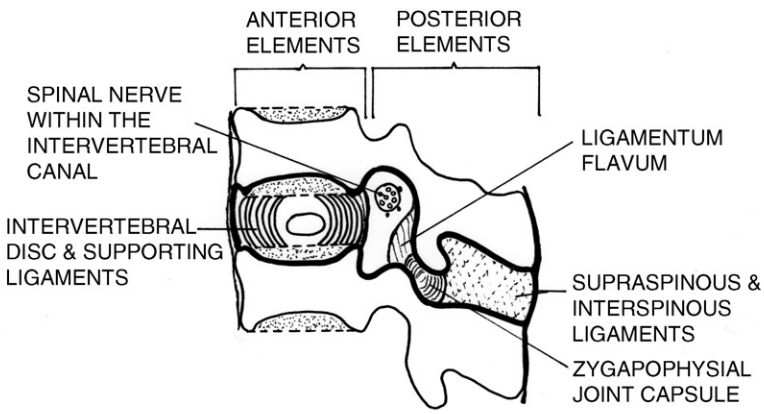

Figure xvii The motion (mobile) segment. (Modified from Schmorl G, Junghanns H 1971 The human spine in health and disease, 2nd edn. Grune and Stratton, New York, p 37, and reproduced with permission from Giles L G F 1989 Anatomical basis of low back pain. Williams & Wilkins, Baltimore.)

Babinski test – run a pointed instrument across the plantar surface of the foot from the calcaneus along the lateral border to the forefoot. In a negative reaction, the toes either do not move at all or bunch up uniformly or turn down. If there is a positive reaction, the great toe extends while the other toes plantar flex and splay. A positive Babinski reflex indicates an upper motor neuron lesion. In the newborn, a positive Babinski is normal; however, the reflex should disappear soon after birth (Hoppenfeld 1976).

Cervical spine compression test – narrowing of the neural foramen, pressure on the facet joints, or muscle spasm can cause increased pain upon compression. In addition, the compression test may faithfully reproduce pain referred to the upper extremity from the cervical spine, and, in doing so, may help locate the neurological level of any existing pathology. Press down upon the top of the seated patient's head. If there is an increase in pain in either the cervical spine or the extremity, note its exact distribution and whether it follows any previously described dermatome (Hoppenfeld 1976).

Fabere test – a test for detecting pathology in the hip, as well as in the sacroiliac joint. The patient lies supine on the table and places the foot of the painful side on the opposite knee. This causes the hip joint to be flexed, abducted, and externally rotated. In this position, inguinal pain gives a general indication of pathology in the hip joint or the surrounding muscles. At full ranges of flexion, abduction, and external rotation, the femur is fixed in relation to the pelvis. To stress the sacroiliac joint, place one hand on the flexed knee joint and the other hand on the opposite anterior superior iliac spine. Press down on each of these points and if the patient complains of increased pain, there may be sacroiliac joint pathology.

Femoral stretch test – a nerve root tension test performed with the patient in either the prone or lateral decubitus position; the test involves extension of the thigh at the hip with the knee flexed. Reproduction of the patient's pain (usually anterior or anterolateral thigh pain) is a tension sign suggesting involvement of the upper (L2, L3, and L4) nerve roots (Klein & Garfin 1997).

Gaenslen's sign – the patient lies supine on the table, and is asked to draw both legs up to the chest; the patient is then shifted to the side of the table to enable one buttock to extend over the edge of the table. Allow the unsupported leg to drop over the edge, while the opposite leg remains flexed. Complaints of subsequent pain in the sacroiliac joint area gives an indication of pathology in that area.

Hoffman's sign – briefly pinch the nail of the middle finger; normally there should be no reaction at all. A positive reaction produces flexion of the terminal phalanx of the thumb and of the second and third phalanx of another finger. If present, this is an indication of an upper motor neuron lesion (Hoppenfeld 1977).

Hoover test – this test helps to determine whether the patient is malingering when stating that the leg cannot be raised. The examiner's hands are put under the patient's heels and, as the patient tries to raise one leg, the opposite heel is used to gain leverage; this causes downward pressure to be felt on the examiner's hand in genuine cases. If the patient does not bear down while attempting to raise one leg, the patient is probably not really trying.

Kernig test – this procedure applies tension to the spinal cord and can reproduce pain. Ask the patient to lie supine, then to place both hands behind the head and to forcibly flex the head onto the chest. The patient may complain of pain in the cervical spine, and, occasionally, in the low back or down the legs, an indication of meningeal irritation, nerve root involvement, or irritation of the dural coverings of the nerve roots. Ask the patient to locate the area from which the pain originates.

Lasegue's sign – flex the hip with the knee flexed, followed by the knee being gradually straightened.

Milgram test – the patient lies supine on the examining table with legs straight, then actively raises them to a position about 5 cm from the table and holds this position for as long as possible. This manoeuvre stretches the iliopsoas muscle, the anterior abdominal muscles, and increases the intrathecal pressure. If the patient can maintain this position for 30 seconds without pain, intrathecal pathology may be ruled out. If the patient cannot hold the position, or cannot lift the legs at all, or experiences pain in the attempt, there may be intrathecal or extrathecal pathology (e.g. herniated disc).

Naffziger test – a compression test designed to increase intrathecal pressure by increasing the intraspinal fluid pressure. The jugular veins are gently compressed for about 10 seconds until the patient's face begins to flush. The patient is asked to cough; if coughing causes pain, there is probably pathology pressing upon the theca. The patient is asked to locate the painful area.

Slump test – the patient sits with the back straight and the legs hanging over the edge of the examination table then slumps the cervical and thoracic spines forward; then straighten one leg at a time to traction the dura. If further dural traction is necessary, dorsiflex the foot. Ask the patient to extend the neck and, if low back or leg pain is relieved, the pain arises from the spine (Kenna & Murtagh 1989).

Swallow test – difficulty or pain upon swallowing can sometimes be caused by cervical spine pathology such as bony protuberances, bony osteophytes, or by soft-tissue swelling due to haematomas, infection, or tumour in the anterior portion of the cervical spine (Hoppenfeld 1976).

Valsalva manoeuvre – the patient is asked to bear down as if trying to move the bowels; this increases the intrathecal pressure. If bearing down causes pain in the spine, or radiating pain, there is probably pathology either causing intrathecal pressure or involving the theca itself.

Obliquity

Pelvic obliquity – this is a lateral inclination of the pelvis which is tilted downward to the short leg side (Fig. xvi).

Sacral base obliquity – a lateral inclination of the sacral base (Fig. xvi).

Osteoarthritis (degenerative joint disease, degenerative arthritis, hypertrophic arthritis) characterized by degeneration of articular cartilage, hypertrophy of bone at joint margins, and synovial membrane changes; usually associated with pain and stiffness (Hellmann 1992).

Osteoarthrosis chronic non-inflammatory arthritis.

Persistent notochord in rare instances the notochord, instead of being entirely confined to the intervertebral discs, persists in whole or in part (Tondury 1958, Diethelm 1974). The result may be an unossified central canal in the vertebral body or conical depressions in the upper and lower endplates (Rickenbacher et al 1982).

Scoliosis

Angle of curvature – the angle between lines drawn parallel to the superior surface of the upper vertebra of the curvature and to the inferior surface of the lowest vertebra of the curvature.

Postural (compensatory) – this is a lumbar or thoracolumbar scoliosis (lateral curvature) which is an adaptation of the vertebral column to pelvic obliquity and which is convex on the short leg side. The intervertebral discs are wedged from the concave to the convex sides on the A-P radiograph with the discs being wider on the convex side of the scoliosis (Fig. xvii).

Structural idiopathic – a lateral curvature with fixed rotational deformity of the spine.

Shoe-raise therapy the provision of a shoe-raise on the side of the short leg. The raise on the heel is equal to the difference in leg lengths and the raise on the sole can be 5 mm less.

Spinal pain

Acute spinal pain refers to severe pain of recent onset (less than 4 weeks) with marked limitation of spinal movements (Skoven et al 2002).

Sub-acute spinal pain refers to pain of greater than 4 weeks and less than 13 weeks.

Chronic spinal pain refers to pain of long duration (13 weeks or more) without marked limitation of spinal movements.

Spondylosis osteophytosis secondary to degenerative intervertebral disc disease (Weinstein et al 1977).

Subluxation in this text, the term is used when apposing facet surfaces of the zygapophysial joint are no longer congruous, as demonstrated by imbrication (telescoping) of the zygapophysial joint facet surfaces (Hadley 1964) (Fig. xviii).

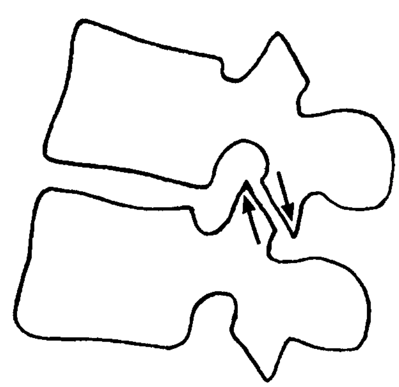

Figure xviii Note the subluxation (imbrication telescoping) of the zygapophysial joint facet surfaces as indicated by the arrows. (Reproduced with permission from Giles L G F 1989 Anatomical basis of low back pain. Williams & Wilkins, Baltimore.)

Tropism asymmetry in the horizontal plane of paired left and right zygapophysial joints.

Vertebroplasty a procedure where bone cement is percutaneously injected into a fractured vertebral body to stabilize it; typically used for an osteoporotic vertebral body fracture causing severe spinal pain with decreased height and spinal deformity. The objective of vertebroplasty is to reduce pain by stabilizing the fractured bone.

Zygapophysial joint the diarthrodial synovial joint between adjacent vertebral arches (apophysial joint, 'facetal' joint, interlaminar joint).

Zygapophysial joint cartilage according to Hadley (1964) this is of the hyaline articular cartilage variety and it lines the facet surfaces; extensions of cartilage beyond the facet surface, known as 'bumper-fibrocartilage', are not composed of hyaline cartilage (Hadley 1964).

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