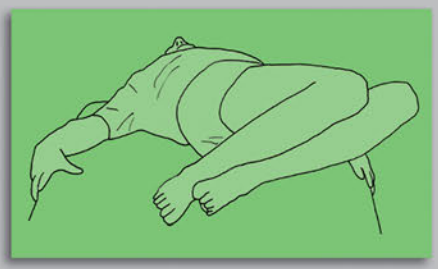
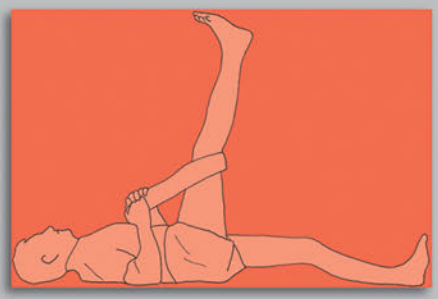
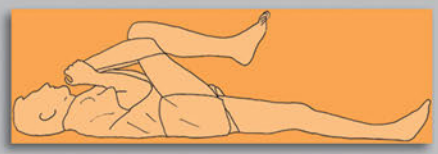
Physical Therapy for Intervertebral Disk Disease

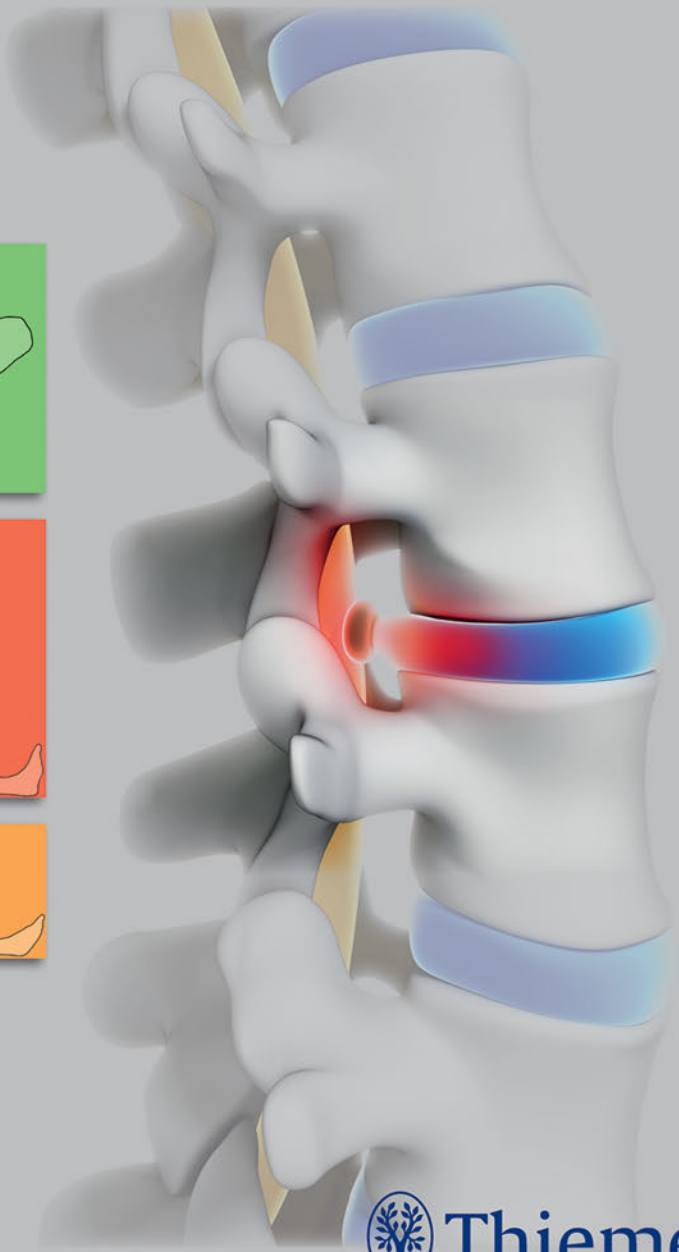
A Practical Guide to Diagnosis and Treatment

Doris Broetz
Michael Weller









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Physical Therapy for Intervertebral Disk Disease

A Practical Guide to Diagnosis and Treatment

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Contents

Foreword

Back in the day, well, I was almost an athlete who'd won quite a few awards. But then there was my profession, and not so much sport. Things went along pretty well that way—for decades—and all the time I was sure that I was still in good condition. I have to confess though that I didn't think about my body very much, and it didn't give me much reason to.

But after a successful disc operation and four weeks of rehab, I suddenly noticed that some of my movements were rather rusty. My physical therapist handed down this decisive judgment: "*What we don't call on constantly—joint, muscle, and even brain—will fade and waste away. Use it or lose it!*", at which point I began to remember long-forgotten movements that I had obviously not performed, correctly anyway, for some time. And I'm not thinking of anything as brutal as knee bends or push-ups—no, just normal things: stooping, getting up out of a chair, and climbing stairs. My movements had clearly become more deliberate but much more limited.

Here was my question: Wouldn't a course of physical training be enough? Now I'm convinced that the answer is no. Even when I try hard to do my exercises correctly, I slack off, get sloppy, simplify them or leave them out altogether. At least for me, even after a few months of self-control, it's important to seek the advice and critical eye of my therapist and accept the new exercises she assigns me. She scolds me for neglecting my exercises and rewards me too, with the laconic comment: "*That was perfect.*" Sometimes I even score a real success, when I notice my progress and see my self-image improving.

But that doesn't mean that written instructions are useless. They help us find out, from someone who knows, how the spinal column is built and how it performs certain movements. Informative illustrations show how to maintain correct posture, avoid poor posture, and protect and train muscles, nerves, and joints. And what makes this book so useful is the presentation of step-by-step procedures and pointers for self-control.

Prof. Erich Koerber (Patient)

Author Biographies

Doris Broetz was born in Ulm, Germany in 1958; she is married and has two children. After completion of her secondary education (1978), she was trained as a physical therapist in Berlin and Tübingen (1978–82). From 1982 to 1986, she worked as a physical therapist at the trauma clinic of the Employers' Liability Insurance Association in Tübingen. Following an extended maternity leave, she worked first at the University Surgical Hospital in Tübingen and in 1993 moved to the Department of General Neurology at the University Hospital in Tübingen where she was chief physical therapist from April 2001 to September 2007 (Chairman: Prof. Dr. Johannes Dichgans; since October 2005 Prof. Dr. Michael Weller). Since October 2007 she has been active in studies on learning at the Institute for Medical Psychology and Behavioral Neurobiology (Chairman: Prof. Dr. Niels Birbaumer) at the Center for Functional and Restorative Neurosurgery (Director: Prof. Dr. Alireza Gharabaghi) and in her own practice. Since 1996, Doris Broetz has planned and conducted several studies of physical therapy diagnosis and therapy in patients with lumbar disk prolapse, working with Prof. Dr. Michael Weller to investigate the efficacy and quality optimization of therapy. On the basis of these studies, a new treatment was developed for patients with disk damage. In addition, the role of muscle relaxants during physical therapy treatment of patients with lumbar disk prolapse and changes in the MRI image of lumbar disk prolapse during physical therapy were investigated.

Doris Broetz is also interested in the analysis and physical therapy of patients with neurological diseases and research in motor learning background and motivation. She has developed diagnostic tests and physical therapy treatments for patients with ataxia, Pusher symptoms, and for the improvement of sensorimotor self-control of patients with hemiparesis.

Prof. Dr. Michael Weller was born in in Rheinbach, Germany in 1962. He is married and has four children. He studied medicine in Cologne (1982–89) and received his training in neurology at the Department of General Neurology in Tübingen (1989–90) and the Department of Psychiatry in Würzburg (1991). He conducted research at the National Institute of Mental Health in Bethesda, Maryland, USA (1992) and the University Hospital, Zürich, Switzerland (1993–94) in the Department of Clinical Immunology. There he studied cerebral cell death processes and, in particular, experimental treatment of malignant brain tumors. In 1995, he returned to the Department of Neurology in Tübingen and completed his postdoctoral thesis (habilitation) in 1997 in the field of neuro-oncology. He became Deputy Chairman in 2001 and was appointed Chairman at the Department of General Neurology in October 2005. In addition to his research and clinical specialty of neuro-oncology, he has been the attending physician for the Pain Clinic of the Neurological Hospital. In collaboration with the Physical Therapy Department (D. Broetz), he established the specialty of conservative physical therapy treatment of disk problems in Tübingen, where he led clinical studies in this field. Since January 2008, he has been Chairman at the Department of Neurology at the University Hospital and University of Zürich.

1 Introduction

At least once in a lifetime, 80–90% of the population have back pain (Loeser and Volinn 1991; Waddell 1998) and about 66% have neck pain (Rao 2002). The causes of such problems originating in the spine are as numerous as the treatment options.

The many possible causes of back pain—tumors, spinal instability, foraminal or spinal stenosis, a prolapsed disk, inflammation, or trauma—can be diagnosed through the medical history, clinical examination, imaging, and laboratory tests. Symptoms that cannot be related to a specific illness by these means are usually interpreted as resulting from tension, sprain, facet pain, blockage of the sacroiliac joint, or as somatic expression of a psychosocial problem.

The usual medical treatment strategies include bed rest, chiropractic treatment, and oral or injectable analgesics and muscle relaxants. Physical therapists use fango treatments, massage, the sling table, and strengthening, usually in combination. Although the efficacy of most therapeutic strategies has not been assured, these treatments generate high healthcare costs (Hildebrandt et al 1996; Cherkin 1998; Chrubasik et al 1998; Williams et al 1998). In addition to the treatments themselves, disability and pension payments are a significant burden on public funds. In spite of increased investment in back pain research, little has changed in recent decades because the data are not reliable (Deyo and Phillips 1996; Van Tulder et al 1997; Cherkin 1998; Krismer and Van Tulder 2007; Vos et al 2012). Currently available guidelines make broad treatment recommendations for patients with back pain and radiating pain. Patient information, encouragement of normal activity, and the prescription of analgesics are cornerstones of these guidelines (Van Tulder et al 2006).

What can be done? First, the concept of "nonspecific back pain or neck pain" must be examined more closely and presented in more specific detail. It is no wonder that even precisely defined treatment methods, applied to a heterogeneous group of patients with the symptoms of back or neck pain in common but not their cause, lead to unsatisfactory results (Faas et al 1995; Malmivaara et al 1995; Cherkin 1998).

But how can disorders be studied and diagnosed if they cannot be unambiguously ascribed to an illness by imaging or laboratory tests? A special approach to this question was described by the New Zealand physical therapist McKenzie (2003, 2006), who used repeated spinal motion as a diagnostic strategy. A hypothesis about the causes of several back pain syndromes was developed on the basis of theoretical considerations regarding anatomy, tissue damage and healing, and knowledge of typical symptoms in certain diseases. McKenzie interpreted the clinical symptoms of *centralization* and *peripheralization* as an indication of disk damage. In this context, centralization means that while the spine is moving, the zone of radiating or radicular pain withdraws rapidly, in a central or proximal direction, toward or as far as the midline of the back. Peripheralization, on the other hand, develops in the opposite direction. That is, the pain radiation is shifted from the back to the periphery or distally.

Donelson et al (1997) described a significant correlation between the clinical sign of centralization in end-range movements and diskographically diagnosed tears in the anulus fibrosus. The clinical sign of peripheralization correlated with tears in the outer annulus (see Chapter 12 Selected Studies on the Topic of Spinal Disorders).

In diskography, after percutaneous injection of contrast medium into the nucleus pulposus, leakage of contrast medium into the anulus fibrosus is taken as evidence of structural damage. A diskogram is evaluated as positive if the pain familiar to the patient can be reproduced and if imaging shows tears in the outer third of the anulus with the outer edge still intact or completely torn.

Thus, disk damage might be diagnosed by means of repeated end-range movements of the spine, even if noninvasive imaging procedures such as computed tomography (CT) or magnetic resonance imaging (MRI) did not identify any damage. In this way, the patients with diagnosable disk damage could be filtered out from the large group of those with nonspecific back and neck pain.

Schwarzer et al (1995) used diskography to identify lumbar disk damage as a probable cause of problems in 39% of patients in whom the CT scan did not demonstrate lumbar disk prolapse. In a study by Laslett et al (2005) on patients with chronic back pain, some with radiating pain, a diskogram showed disk damage in 75% of patients. Consequently, in performing and interpreting diagnostic tests, experienced physical therapists may be able to filter out about half the patients in the group with nonspecific back pain and give them a specific diagnosis so that they can receive targeted physical therapy. This could also apply to patients with nonspecific neck pain, because of the anatomical and mechanical similarity of the different spinal segments. Up to now, the appropriate studies have not been conducted.

In patients with neuroradiologically confirmed disk prolapse, it can also be determined, by means of repeated end-range spinal movements, whether they can be expected to benefit from conservative treatment (Brötz et al 2001, 2003; Brötz, Burkard, Weller 2010).

The treatment described here is governed by changes in symptoms during the acute phase, and its success is evaluated on the basis of defined milestone targets. The measures used are spinal movements performed by the patients themselves. After the acute phase, movements of the extremities are added, with the objective of preserving or improving the mobility of the nerve roots and nerves. Spinal stability is restored by means of exercises that activate the tonic, stabilizing muscles. All therapeutic movements are very simple and can be easily performed by every patient. Only in very rare cases does the therapist supplement the effect of the exercises with passive movements.

Once the symptoms have largely subsided and no worsening occurs on loading, treatment aims at free mobility of the spine and extremities. Strengthening, coordination exercises, and fitness training finally bring the patient to normal movement, normal capacity, and ability to work.

Because patients experience rapid changes in their symptoms during the exercises they perform themselves, they are motivated to share the work and the responsibility for their own recovery. The job of the therapist is as much to inform the patient explicitly about the pathogenesis of a disk prolapse as to instruct them precisely how to exercise and observe their symptoms. The patient learns how to modify their movement patterns and how to stress their body evenly, purposefully, and appropriately, so that the structures of the spine are not damaged but acquire stability as needed for a given load. In this way, the therapy described here can prevent development of a fear of pain that could result from movement or stress. This prevents chronification and relapses.

With time, the authors have changed and supplemented the strategies presented by McKenzie (1981, 1986, 1990) and for this reason the therapeutic program described here is called the BASE PT (behavioral, active, self-determined, evidence-based physio therapy). The possibilities and results of physical therapy according to the BASE PT have been systematically studied at the Neurology Clinic of the University of Tübingen in Germany from 1997 to 2010, in patients with lumbar disk prolapses. Patients with neurological deficits resulting from prolapsed disks were also included in the physical therapy studies.

The order of test movements was changed and now depends on which movement relieves the symptoms of the disorder assumed in the preliminary suspected diagnosis. Spinal rotation, both as a diagnostic test movement and as an exercise performed by the patient, is very important. In addition to the spinal movements, the concept includes movements of the extremities to prevent and treat limitation of nerve-gliding capacity and exercises to stabilize the spine. Observation and documentation of neurological disorders, signs of nerve stretching, and development of pain are given major attention.

The treatment design includes strengthening, coordination exercises, and fitness training, and brings the patient back to normal movement, load-bearing capacity, and ability to work.

2 General Principles

For the understanding and interpretation of disk prolapse symptoms, this summary presents the fundamentals of the *functional anatomy* of the spine and the nervous system, the *pathophysiology of disk damage* and its effect on nerve roots, and the *healing* process.

Pain and limited function in activities of daily living are the chief problems caused by disk damage. Assessment of pain and disability are necessary for evaluation of the effect of treatment and the course of the disease. Various measurement scales for this purpose are introduced.

The predisposing factors are of interest for prevention. Section 2.5 Epidemiology and Risk Factors sheds light on the controversial questions of whether certain activities lead to disk damage with particular frequency and which physical conditions or social circumstances promote these problems.

2.1 Anatomy of the Spine and Nervous System

The spine fulfills two different functions: it *carries the head and thorax* and *protects the spinal cord*. At the same time, a high degree of mobility is needed. The requirements of stability and mobility are in constant conflict. In order to perform both functions, the spine must display good interaction between its bearing and moving structures.

The *passive holding apparatus* consists of the vertebrae, disks, vertebral joints, ligaments, and articular capsules, while the *active holding apparatus* is made up of muscles and tendons. The *nervous system* registers the position, load, and stresses on the spine and controls the active system in order to fulfill the stability and mobility requirements (Waddell 1998). Malfunction in one of the three systems leads to a reaction in the other two systems. Adaptation, incorrect loading, pain, or loss of function can result.

2.1.1 Musculature

The active holding apparatus consists of numerous muscles and tendons. The muscles that move and actively stabilize the spine can be roughly grouped into back, abdominal, posterior and anterior neck muscles. Here we will discuss only the largest and most important muscles (**Table 2.1**).

2.1.2 Bony Spine and Ligaments

The spine is made up of seven cervical, 12 thoracic, and five lumbar vertebrae and the sacrum. The highest cervical vertebra is called the atlas and, in

Table 2.1 The chief muscle groups for stabilization and movement of the spine

Muscle	Origin	Attachment	Function	Innervation
Neck musculature				
Short, dorsal neck muscles	Atlas Axis	Linea nuchae Transverse process of atlas Jugular process of the occipital bone	Extension, rotation, and lateral flexion of the head	N. suboccipitalis (C1)
Scalene muscles	Ventrally at the transverse processes of the cervical vertebrae	1st–2nd ribs	Lateral flexion of the cervical spine with fixed head: elevation of the 1st and 2nd ribs	Cervical plexus Brachial plexus (C3–C8)

▶ (*continued*)

Table 2.1 The chief muscle groups for stabilization and movement of the spine (*continued*)

Muscle	Origin	Attachment	Function	Innervation
Prevertebral muscle group M. longus colli M. longus capitis M. rectus capitis anterior	Ventrally at all cervical vertebral bodies and the upper thoracic vertebral bodies Transverse processes of the cervical vertebrae	Atlas ventrally on all cervical vertebral bodies Transverse processes of the caudal cervical vertebrae Occipital bone	Flexion, rotation, and lateral flexion on the ipsilateral side of the cervical spine	Cervical plexus (C1–C6)
M. sternocleidomastoideus	Sternum Clavicle	Mastoid process of the temporal bone Linea nuchae	Flexion of the caudal and extension of the cranial cervical vertebrae and the head joints (protraction) Rotation of the head to the contralateral side with fixed head: help in inspiration	N. accessorius Cervical plexus (C1–C3)
Back muscles				
Autochthonous back muscles: M. erector spinae (consists of many small muscle groups that connect transverse processes, spinous processes, and ribs)	Sacrum Iliac crest	Occipital bone	Extension, rotation, lateral flexion in individual sections and the entire spine Assurance of erect posture	Dorsal branches of spinal nerves (C2–L4)
M. trapezius	Linea nuchae Spinous processes of the cervical and thoracic vertebrae	Clavicle Acromion Spine of scapula	Cranial fibers: – Elevation of the shoulder blade – Rotation of the head to the contralateral side Medial fibers: – Retraction of the shoulder blade Caudal fibers: – Depression of the shoulder blade	N. accessorius Cervical plexus (C2–C4)
M. latissimus dorsi	Spinous processes from T7 to the sacrum 8th–12th ribs Iliac crest	Crista tuberculi minoris (humerus)	Inward rotation, "adduction," extension in shoulder joint with fixed arm (support): elevation of the pelvis	N. thoracodorsalis (C6–C8)
M. quadratus lumborum	Iliac crest Iliolumbal ligament	12th rib Lumbar vertebrae L1–L4	Pulls the 12th rib caudally Lateral flexion of the lumbar spine with fixed thorax: elevation of the pelvis	Muscular branches of the lumbar plexus N. intercostalis 12 (T12–L3)
Abdominal muscles				
M. rectus abdominis	5th–7th ribs Xiphoid process	Cranial edge of the pubic bone	Pulls the thorax toward the pelvis Bending the trunk or lifting the pelvis Antagonist of the long back extensor	Middle and caudal intercostal nerves (T5–T12)

▶ (*continued*)

Table 2.1 The chief muscle groups for stabilization and movement of the spine (*continued*)

Muscle	Origin	Attachment	Function	Innervation
M. obliquus externus	Exterior surfaces of 5th–12th ribs	Iliac crest Inguinal ligament Sheath of the rectus	Abdominal press Trunk bending Elevation of the pelvis Rotation of the trunk to the contralateral side	Caudal intercostal nerves (T5–T12)
M. obliquus internus	Iliac crest Thoracolumbar fascia Inguinal ligament	9th–12th ribs Linea alba	Abdominal press Trunk bending Elevation of the pelvis Rotation of the trunk to the ipsilateral side Lateral flexion of the trunk	Caudal intercostal nerves Branches of the lumbar plexus (T10–L2)
M. transversus abdominis	7th–12th ribs Transverse processes of the lumbar vertebrae Iliac crest Inguinal ligament	Sheath of the rectus abdominis	Retraction and tightening of abdominal wall Abdominal press	Intercostal nerves (T5–L2)

contrast to the other vertebrae, has no vertebral body. The second vertebra is called the axis and has an anterior projection (dens axis) that articulates with the atlas.

The vertebral bodies with the disks form the anterior portion of the spine. The vertebral arches with the transverse processes and the spinous processes enclose and protect the spinal cord laterally and posteriorly and form the posterior portion of the spine. The articulations between the vertebrae are formed anteriorly by the disks and posteriorly by the small vertebral joints (facet joints). The end plates of the vertebrae form the contact surfaces for the disks (**Fig. 2.1**).

The spaces between the vertebral bodies and the small vertebral articulations are the intervertebral foramina through which the nerve roots emerge from the spinal cord and run from the cervical spine to the arms, from the thoracic spine to the trunk, and from the lumbar spine to the legs. The sacrum forms the connection between the spine and the pelvis.

The spine is stabilized by three longitudinal ligaments:

- Anterior longitudinal ligament: runs anteriorly over vertebrae and disks.
- Posterior longitudinal ligament: runs posteriorly over vertebrae and disks.
- Ligamentum flavum: connects the vertebral arches of two adjacent vertebrae.

2.1.3 Intervertebral Disks

There are intervertebral disks between all vertebral bodies from cervical vertebral bodies C2–C3 to the transition between the lowest lumbar vertebral body (L5) and the first sacral vertebra (S1). They are attached to the end plates of the vertebral bodies (Bogduk 2000).

Each vertebral body is covered by an end plate consisting of hyaline and fibrous cartilage. Insertions of collagenous fibers from the intervertebral disk form the connection between the end plate and the disk. This connection is more stable than that between the end plate and the vertebral body.

Moreover, the disks are the connection between the end plates of two adjacent vertebral bodies. They consist of two parts (**Fig. 2.1**): the outer fibrous ring (anulus fibrosus) and the inner mucoprotein gel (nucleus pulposus). The two components are not sharply distinct from each other since the outer regions of the nucleus pulposus blend smoothly into the inner region of the anulus fibrosus (Bogduk 2000).

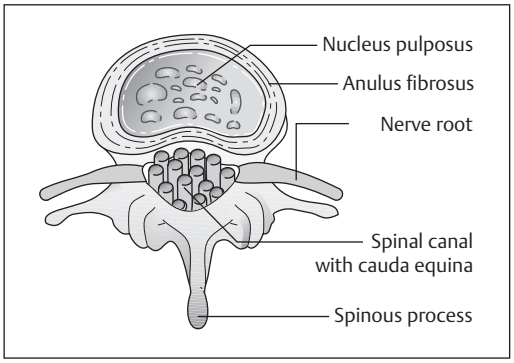

Fig. 2.1 Lumbar vertebra with disk. In the thoracic and cervical spines, the spinal canal contains the spinal cord instead of the cauda equina.

With increasing age, the structural and functional differentiation of the disk areas decreases. Both portions contain water, collagen, and proteoglycans in different concentrations.

The anulus fibrosus is 60% connective tissue (Type 1 collagen) arranged in an oblique ring. It creates the firm connection between the vertebral bodies, retains the gel mass between the vertebral bodies, and can cushion tensile stresses like axial spinal rotation (Hadjipavlou et al 1999).

The gel core consists largely of proteoglycans and glycosaminoglycans, which take up water and can carry loads and absorb shocks.

Physiological Compressive Stress

When the spine moves, the nucleus pulposus changes its shape and distributes pressure to the end plates and the anulus fibrosus. The nucleus pulposus is squeezed like a sponge by loading, and when pressure is released it absorbs water again (McMillan et al 1996; Wilke et al 1999; Race et al 2000).

Examples

- Wilke et al (1999) measured pressure for almost 24 hours in the middle of the disk between vertebral bodies L4 and L5, in a healthy 45-year-old subject. The patient performed various activities during this time and assumed a variety of positions. During a 7-hour sleep period, a pressure increase from 1 to 24 bar was measured. This was explained by regenerating water absorption by the disks.
- The increase of spinal pain and limitation of motion during the night in patients with disk damage could be related to this pressure increase and the greater volume occupied by the hydrated disk. At zero gravity, because of decompression, astronauts were up to 5 cm taller than under normal pressure conditions (Urban and McMullin 1988).
- Nachemson and Elfström (1970) investigated the pressure on intervertebral disks in nine subjects who assumed various body positions. Of these, six had had no back pain in the past, two had pains in the past, and one suffered from scoliosis. In each case, a probe was positioned in the middle of the L3–L4 disk.

Wilke et al's (1999) results were in many respects identical with Nachemson and Elfström's (1970): the lowest reclining disk pressure and the highest disk pressure while lifting loads with straight knees and bent spine, measured in both studies, were in agreement. Activity of the back muscles in all positions was associated with an increase in pressure (Nachemson and Elfström 1970; Wilke et al 1999). The intradiskal pressure also rises when traction is applied if the back muscles are activated in compensation at the same time (Andersson et al 1983).

In contrast to Nachemson and Elfström (1970), Wilke et al (1999) found no increase in pressure when the subject was sitting upright, as compared to when standing upright. When the subject was sitting in dorsal flexion, against a back rest, Wilke et al (1999) even observed a significantly lower pressure than when standing upright or sitting without a back rest.

This knowledge led to a wide-ranging discussion of the erect seated posture favored by physical therapists and in back schools (Reinhardt 1992; Brügger 1997; Nentwig et al 1997). Clinically, it has proven advantageous for patients with disk damage to avoid sitting as much as possible (see Chapter 9 Rehabilitation and Prevention).

Metabolism

Disks are the largest avascular structures in the body; they are characterized by low metabolism. Exchange of nutrients and metabolic products in the disk takes place passively by diffusion and

osmosis through the blood vessels of the end plates and the anterior and posterior longitudinal ligaments (Holm et al 1981; Van den Berg 1999).

Repeated movement of the spine is thought to improve the exchange of nutrients and metabolic products in the disk (Holm and Nachemson 1983). This hypothesis is based on an animal experiment in which a group of dogs that performed repeated active movements of the spine during a controlled training program was compared with a control group (ibid.). In the group that performed repetitive spinal movements, there was increased oxygen consumption in the external portion of the anulus fibrosus and the nucleus pulposus, with a lower lactate concentration than in the control group.

Innervation

The question of whether disks have sensory innervation was controversial for a long time. Free nerve endings that make the sensing of pain possible were at first found only in the skin, the facet joints, the sacroiliac joint, the anterior and posterior longitudinal ligaments, the ligamentum flavum, the periosteum of the vertebrae and the vertebral arches, the fasciae and tendons, the dura mater, and the dural sheaths of the nerve roots.

Bogduk et al (1981, 1983, 1988) described sensory innervation in the anulus fibrosus as well. The posterior portion is supplied by the sinuvertebral nerve, the lateral areas by the communicating gray branches of the ventral branches of the spinal nerves.

Palmgren et al (1996) found both sensory and autonomic nerve endings in surgically removed disk tissue. The fact that the anulus fibrosus contains pain receptors endorses the interpretation that back pain can be triggered by injury to the anulus fibrosus.

Indahl et al (1997) triggered action potentials in the longissimus and the multifidus muscles of 23 sedated pigs by applying electrical stimuli to the posterolateral anulus fibrosus. After local stimulation of the small vertebral joints by injection of physiological saline, the action potentials triggered by electrical stimulation of the anulus fibrosus were weakened. This indicates that there is a reflex mechanism between the anulus fibrosus, the back muscles, and the facet joints such that stimulation of the small vertebral joints decreases muscle tone rather than increasing it. We will return later to the possible significance of this mechanism for the interpretation and treatment of back pain (see 2.2.3 Disk Damage and Muscle Tension).

The disks have the following functions:

- Firm connection of two vertebral bodies.
- Facilitation of movement.
- Bearing the weight that is transmitted by the vertebrae lying above.
- Shock absorption.

2.1.4 Nervous System

Depending on its location and function, the nervous system is divided into the *central* and the *peripheral nervous system*. The central nervous system (CNS) consists of the brain and the spinal cord. It serves to receive and process information and initiate appropriate reactions.

As soon as they emerge from the spinal cord, structures become part of the peripheral nervous system. This system serves to conduct sensory impulses from the periphery to the central nervous system and motor impulses from the central nervous system to the periphery.

In addition, depending on the function, a distinction is made between the *somatic* and the *autonomic nervous system*. This classification applies chiefly to the peripheral nervous system but also to the central nervous system.

The somatic nervous system serves to control voluntary motion and conscious perception of sensory stimuli. The autonomic (visceral, vegetative) nervous system consists of the sympathetic and the parasympathetic systems and controls (unconscious) events in the internal organs (e.g., breathing, digestion, blood pressure). It plays only a subordinate role in disk prolapse and will therefore not be discussed further.

Spinal Cord, Cauda Equina, and Nerve Roots

The spinal cord is part of the central nervous system and runs through the spinal canal. It is connected to the brain, specifically the medulla oblongata, at the level of the foramen magnum of the occipital bone and extends down as far as the first lumbar vertebra. Below this point, the nerve roots run inside the vertebral canal and only emerge through

the intervertebral foramina in lower segments. They resemble a horse's tail, reflecting the anatomical Latin designation, *cauda equina*.

The anterior (motor) roots emerge between the anterior and lateral strands of the spinal cord and the posterior (sensory) roots emerge between the lateral and posterior strands. They join at the level of the intervertebral foramina to form the spinal nerves. The sensory spinal ganglion associated with each spinal nerve is located in the corresponding intervertebral foramen.

In the cervical region, the nerve roots are named for the vertebra that forms the lower boundary of the intervertebral foramen. Thus the root between the fifth and sixth cervical vertebrae is called C6; yet the root between the seventh cervical vertebra and the first thoracic vertebra is called C8. Consequently, the nerve roots from the first thoracic vertebra are numbered after the vertebra forming the upper boundary of the intervertebral foramen. The root between L5 and S1 is called L5.

Spinal Nerves and Peripheral Nervous System

Eight cervical, 12 thoracic, five lumbar, and five sacral spinal nerves on both sides emerge from the various spinal cord segments. The thoracic nerves run throughout the thorax and provide the sensory and motor supply for the back, chest, and abdomen. The spinal nerves from the cervical and lumbar spine, as well as those from the sacral segments, unite in the paravertebral plexuses that divide into individual peripheral nerves further along their course. The skin area supplied by the fibers of a given nerve root is called a *dermatome.*

As a result of the interconnection of nerve root fibers in the plexuses with the formation of peripheral nerves, in cases of sensory disorders it is possible to distinguish between areas corresponding to a root lesion (segmental innervation) or to the area of innervation of a peripheral nerve (peripheral innervation; **Fig. 2.2a, b**). Most muscles

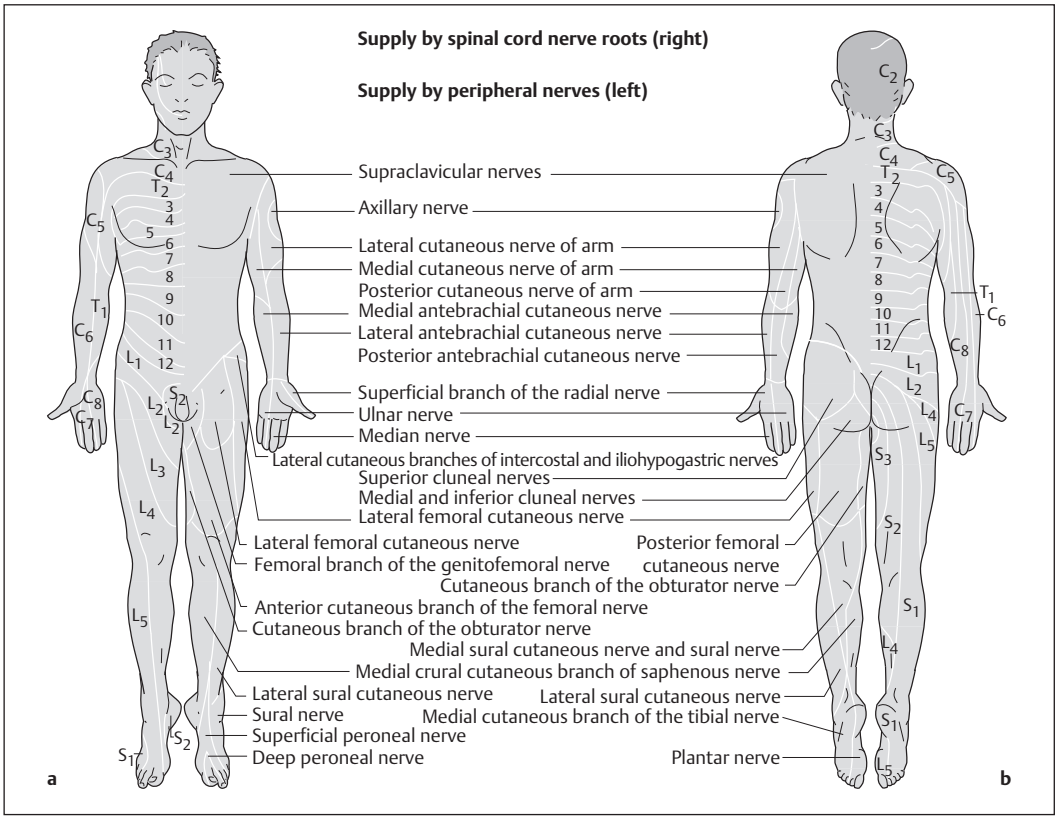

Fig. 2.2 Sensory innervation of the skin. The right half-body shows the sensory innervation by nerve roots (segmental innervation); the left half-body the sensory innervation by peripheral nerves (peripheral innervation). **(a)** Front. **(b)** Back.

are supplied by fibers from several nerve roots. Sometimes a single nerve root predominates to such an extent that a *segment-indicating* muscle can be ascribed to it (**Table 2.2**).

In a nerve root syndrome, the associated pain and sensory disorders occur in the dermatome that is supplied by the affected nerve root and paresis and reflex weakening occur in the corresponding segment-indicating muscles (**Table 2.3**). Pain and sensory disorders can extend laterally over the neck or back from the corresponding spinal segment.

Table 2.2 Segment-indicating muscles

Segment-indicating muscles	Segments
Deltoid muscle	C5
M. biceps brachii M. brachioradialis	(C5) C6
M. triceps brachii	C7
Muscles of the hypothenar eminence Mm. interossei	C8
M. quadriceps femoris M. tibialis anterior	(L3) L4
M. extensor hallucis longus	L5
M. triceps surae	S1

Table 2.3 Signs and symptoms of root syndromes (predominant pareses and impaired functions are printed in bold type)

Root	Pain and hypesthesia area	Paresis	Functional impairment	Reflex
C2–C4	Between shoulder blades Back of neck	**Trapezius**	**Elevation** and retraction of the shoulder blade	
C5	Outside of upper arm (upper third)	**Deltoid** **Biceps brachii**	**Abduction of shoulder joint more than 30°** Flexion in elbow joint	Deltoid reflex Biceps tendon reflex
C6	Shoulder Arm to radial side of lower arm Fingers I and II	**Biceps brachii** **Brachioradialis**	**Flexion in elbow joint**	Biceps tendon reflex Brachioradialis reflex
C7	Shoulder Arm to fingers II–IV volar and dorsal, especially finger III	Pectoralis major **Triceps brachii** Opponens pollicis	Adduction in shoulder joint **Extension in elbow joint** Opposition of thumb	Triceps tendon reflex
C8	Shoulder Arm Lower arm ulnar side Fingers IV–V	Flexor carpi ulnaris Abductor digiti minimi **Interossei dorsales**	Volar flexion with ulnar abduction in wrist Abduction of the little finger **Spreading fingers**	Finger flexor reflex
T1	Inner side of upper and lower arm	–	–	–
T2–12	At corresponding level in the trunk	–	–	–
L1	Groin	**Iliopsoas**	**Hip flexion**	
L2	Groin	**Iliopsoas** Hip adductors	**Hip flexion** Hip adduction	Adductor reflex
L3	Front of thigh to knee	Iliopsoas Adductor magnus and brevis **Quadriceps femoris**	Hip flexion Hip adduction **Knee extension**	Adductor reflex Patellar tendon reflex
L4	Front of thigh Knee Inner side of lower leg Inner ankle Medial edge of foot	Quadriceps femoris **Tibialis anterior**	Knee extension **Dorsal extension in ankle**	Patellar tendon reflex

▶ (*continued*)

Table 2.3 Signs and symptoms of root syndromes (predominant pareses and impaired functions are printed in bold type) (*continued*)

Root	Pain and hypesthesia area	Paresis	Functional impairment	Reflex
L5	Posterior exterior thigh Outer lower leg Medial dorsum of foot Toes I–II	**Extensor hallucis longus** **Gluteus maximus, gluteus medius, gluteus minimus** Tibialis posterior Tibialis anterior	**Dorsal extension of great toe** **Hip abduction and extension** Plantar flexion/supination in ankle Dorsal extension in ankle	Tibialis posterior reflex
S1	Posterior thigh Posterior lower leg Heel Sole of foot Outside edge of foot to toes III–V	 Peroneal muscles **Triceps surae** **Gluteus maximus, gluteus medius, gluteus minimus**	 Pronation and plantar flexion in ankle **Plantar flexion/supination in ankle** **Hip abduction and extension**	 Achilles tendon reflex

All root syndromes of roots C5–T5 can cause pain between the shoulder blades, and syndromes of roots L1–S1 can cause pain in the lumbar spine, lateral back, and buttocks. **Table 2.3** shows only the distal pain and hypesthesia areas typical for individual nerve root syndromes.

Tissues of the Nervous System

Nervous tissue is made up of nerve cells (neurons) and glial cells. Neurons conduct and process excitation. For this purpose, they have special processes that are not found in other cell types. Each neuron has a neurite (axon) and several dendrites. Neurites conduct signals from the cell (efference) and dendrites receive signals from other cells (afference).

Glial cells have a supporting function that provides structure; they also participate in exchange of nutrients between neurons and blood as well as in conduction of stimuli. A specialized form of glial cell—oligodendrocytes of the central nervous system and the Schwann cells of the peripheral nervous system—surrounds the axons and forms myelin sheaths.

The inner subarachnoid spaces are lined with ependymal cells. The musculature is innervated by motor neurons of the spinal cord, whose neurites form the motor anterior roots (**Fig. 2.3**).

Peripheral nerves consist of nerve fibers and the connective tissue that surrounds them. Several axons and dendrites, enclosed in myelin sheaths, are called nerve fibers (fascicles) and are embedded in the endoneurium. The perineurium gathers the nerve fiber bundles. Several nerve fiber bundles are embedded in the epineurium and form the peripheral nerve (**Fig. 2.4**).

Elasticity and extendability of the nerves depend on the sheath tissues of the fascicle, while the epineurium protects against *compression* like a cushion. Every nerve contains several bundles of fascicles, each of which provides sensory or motor innervation to a specific area. Individual axons run wavelike within the fascicles, the fascicles run in waves in the epineurium, and the nerves run in waves in their sheaths (Sunderland 1990). This makes adaptation possible in case of extension during physiological movement sequences.

The thickness of the epineurium varies along the course of the different nerves. Nerves consisting of only a few fascicles and copious epineurium (e.g., the tibial nerve) are better protected against pressure and mechanical stress than those with many fascicles and less epineurium (e.g., the peroneal nerve). The epineural and perineural tissue structures of nerve roots are not distinct; the nerve fibers are parallel and the endoneurium is finer than along the course of the nerves. These structural conditions make nerve roots particularly sensitive to compression and extension.

In the spinal canal, the nervous system is surrounded by a hard covering referred to as the dura

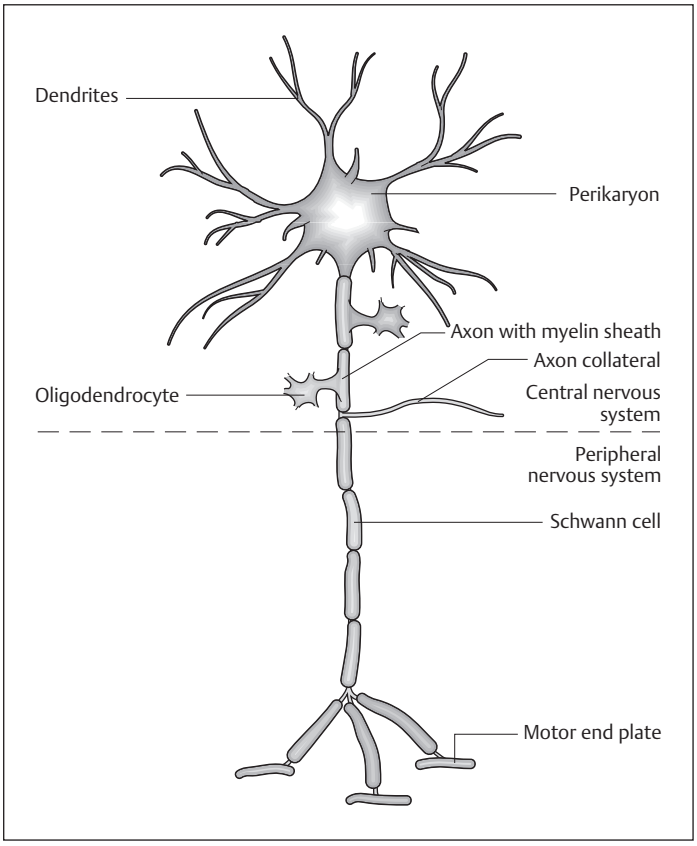

Fig. 2.3 Motor neuron from the anterior horn of the spinal cord. The dotted line marks the boundary between the central and peripheral nervous systems.

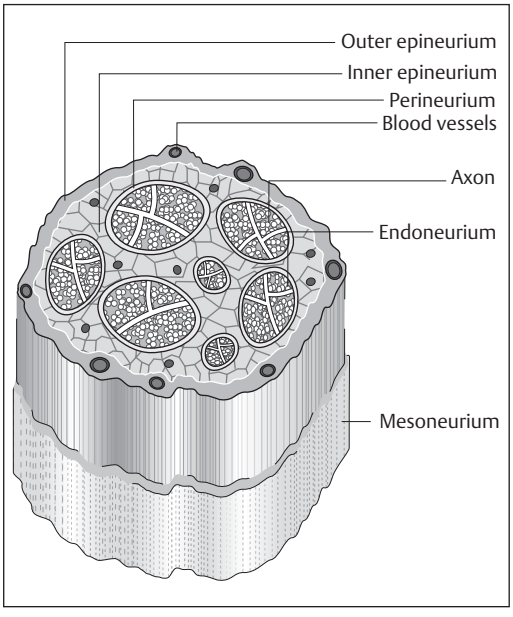

Fig. 2.4 Connective tissue of a nerve.

mater. Cranially, it is attached to the foramen magnum and caudally it is attached to the coccyx by the filum terminale. The dura mater is connected to the spinal cord internally by ligaments, via the arachnoid membrane and a soft cerebrospinal coverage referred to as the pia mater, and externally to the bony surroundings (**Fig. 2.5**). In this way, the spinal cord is safely suspended and protected from elastic stress by ligaments and the dura mater. The spinal fluid in the subarachnoid space is a flexible, protective cushion for the nervous system. Anteriorly, the dura mater is connected to the posterior longitudinal ligament and posteriorly, to the ligamentum flavum.

Stimulus Conduction

The chief function of the nerve cells is reception, processing, and communication of signals from the periphery (afference) as well as transmission back to the periphery of signals generated