11 Neurological Assessment in Spinal Disorders

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Core Messages

- ✔ There is a rather low prevalence of neurological deficits in spinal disorders
- ✔ Neurological deficits can range from very severe and obvious (complete paraplegia) to subtle (radicular sensory deficit)
- ✔ The neurological deficit per se is non-specific to the spinal disorder

The **neurological examination:**

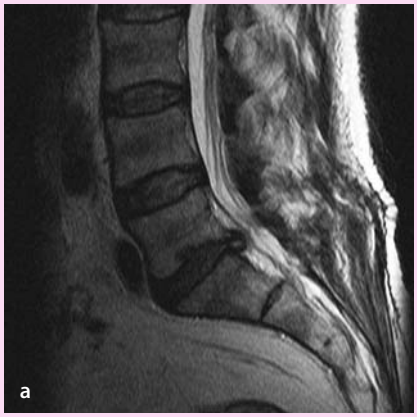
- ✔ Is key to the reliable exclusion of a neurological deficit
- ✔ Complements and influences the diagnostic procedures
- ✔ Has to follow a standardized algorithm to identify the level and extent of a neurological lesion
- ✔ Distinguishes between lesions of the central (cortical, spinal) and peripheral nervous system (nerve roots, plexus, peripheral nerves)
- ✔ Seeks for a somatotopic localization of the lesion
- ✔ Impacts on the treatment decision (conservative versus surgical management) in the presence of a neurological deficit
- ✔ Is insensitive for the assessment of autonomic disorders which require additional testings (e.g. bladder assessment)

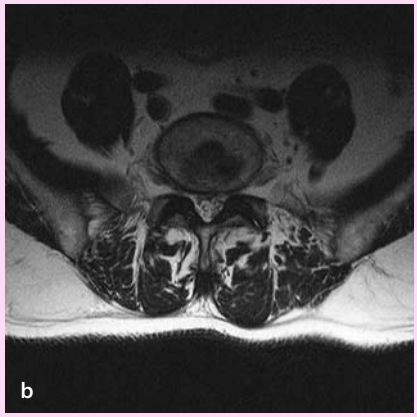
Epidemiology

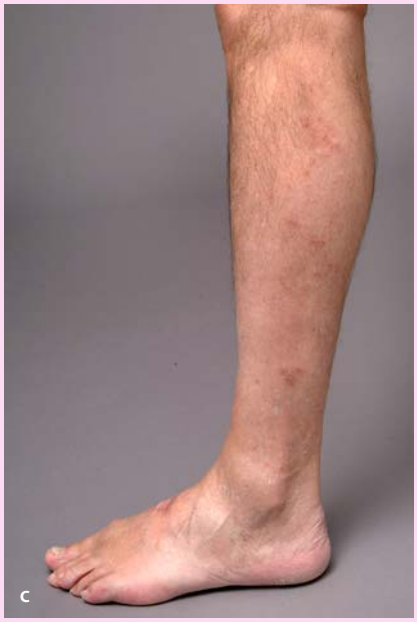
The presence of neurological deficits varies to a large extent in spinal disorders

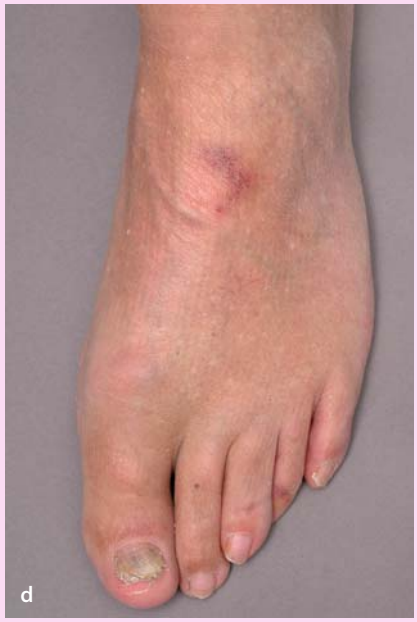
Spinal disorders are associated with neurological symptoms to a very variable extent depending on the underlying pathology. In cervical myelopathy and lumbar spinal canal stenosis, a neurological deficit has been described in about 30 – 50 % of patients depending on the applied clinical measures [3, 33, 65, 76, 105, 117]. Although in general neurological deficits are rather low in frequency, misdiagnosis or failure to detect neurological symptoms may lead to severe sequelae and can result in invalidity if inappropriate management is provided [40]. A knowledge of the typical neurological deficits associated with spinal disorders allows for the management of the diagnostic work-up in timely and comprehensive fashion, and the identification of potential neurological deficits in the treatment of patients with spinal disorders.

Non-traumatic spinal disorders are mainly due to degenerative diseases (e.g. disc herniation and spinal canal stenosis) and occur increasingly in the aging population [11, 24]. Also spine related pain syndromes have a high prevalence which increases with age. For instance, neck and arm pain will have affected about 20 – 34 % of a general population once as shown in a large cross-sectional study and induces actual complaints in about 14 % [16, 47]. However, only in about 4 % of patients suffering from a cervico-cephalic-brachial pain syndrome is an MRI documented radicular lesion present, whereas functional disturbances in conjunction with cervical spondylosis occur in 80 % [61]. Similar findings are reported in patients suffering from low back pain where a focal neurological lesion is present in a comparably low percentage [3, 7, 31, 60].









Case Introduction

A 63-year-old male patient underwent a left-sided discectomy of L5/S1 for an S1 radiculopathy. After a pain free interval of 5 months, he presented again with severe recurrent left sided leg pain predominantly at the posterolateral aspect of the calf. An MRI scan showed a small recurrent sequestrated disc herniation at the level previously operated on (**a**, **b**). The patient was referred to a neurologist because the clinical findings and the imaging study did not completely match. A detailed history revealed that the patient reported pain in the lower back down to the left calf and heel. However, he additionally felt numbness in the thoracoabdominal skin on the left side. The neurological examination revealed an absent left Achilles tendon reflex, hypesthesia of the left T6–T10 and S1 dermatomes but no paresis. The L5 dermatome presented petechial efflorescence (**c**, **d**). The EMG of the gastrognemius muscle confirmed chronic denervation as a sign of a radicular lesion probably caused by the disc herniation of the S1 root. However, prolonged tibial somatosensory evoked potential, hypesthesia of the thoracic dermatomes as well as the dermatomal efflorescence suggested an additional neurological disorder. The suspected diagnosis of a herpes associated myelitis was confirmed by pathological antibody titers against herpes zoster virus, and increased cell count (65/µl) and protein level (1.66 g/l) in the CSF. The patient was treated with acyclovir (i.v. application over 5 days and continued oral medication for 3 months). Three months later the pain had completely subsided and the patient regained full neurological function.

Peripheral neurological disorders may mimic radiculopathy and should be differentiated by the neurological examination and complementary neurophysiological tests.

Always differentiate radiculopathy and peripheral neuropathy

For example, polyneuropathy can cause similar symptoms to lumbar stenosis. While the clinical examination might not be sensitive enough to distinguish between both disorders, neurophysiological testing (nerve conduction and reflex studies) can confirm the presence of a polyneuropathy. There are no reliable data available on the prevalence of polyneuropathy in a general population and the reported percentage ranges between 7 % and 57 % [120]. About 50 % of patients with diabetes and 60 % of patients with alcohol addiction suffer from polyneuropathy, indicating the importance of an extended differential diagnosis in this patient population when patients present with back and leg pain [32, 88, 90, 122].

Entrapment syndromes are easily confused with radiculopathy

Entrapment syndromes frequently show similarities to radicular syndromes. The carpal tunnel syndrome (CTS) is the most frequent entrapment (6 % in a general population) syndrome and occurs twice as often as the compression syndrome of the ulnar nerve [8, 9, 27, 28, 106]. Similar in symptoms, but less common, is the thoracic outlet syndrome (TOS), occurring in not more than 1 % in a general population [79]. The counterpart of the CTS is the tarsal tunnel syndrome of the foot, which is much rarer than the CTS. In electromyography (EMG) laboratories the incidence is reported to be lower than 0.5 % [78, 80].

The C5, C6, L5 and S1 nerve roots are most frequently affected

Due to the different vulnerability of specific nerve fibers and spinal cord tracts, **typical clinical syndromes** are frequently observed both in degenerative and in traumatic spinal disorders. Degenerative disorders, particularly spinal stenosis and disc herniation, most frequently occur in the cervical and lumbar spinal segments due to the biomechanical spine properties (anatomical characteristics) and dynamic/static forces acting on these segments. While a cervical spinal stenosis can result in cervical myelopathy with clinical signs of impaired longitudinal tracts (spasticity of lower limbs, numbness of feet), lumbar spinal stenosis can affect the cauda equina causing neurogenic claudication. Radiculopathies are mainly due to disc herniation and to hypertrophic facet joints. The most frequent cervical radicular lesion is the radiculopathy of C5 and C6, whereas in lumbar radiculopathy the L5 and S1 roots are most frequently involved [17, 38, 102, 128]. Furthermore, in 16 % of patients (study of 585 patients screened in a regional UK clinical neuroscience center) with a non-traumatic para- or tetraparesis, a metastatic or primary spinal tumor could be diagnosed [82, 112].

Traumatic spinal disorders (e.g. spinal cord injury, SCI) are mainly caused [30] by:

- motor vehicle accidents (40 – 50 %)
- sports accidents and falls (20 – 30 %)
- assaults (gunshot and stabbing) (5 – 20 %)
- occupational injuries (10 – 20 %)

Patients suffering from traumatic SCI are mainly young (average age 38 years) and male (male : female ratio = 4 : 1), while there is a second age peak between 60 and 80 years due to predominantly falling injuries [30, 34, 39, 56, 100, 118, 124]. The incidence of traumatic SCI (10 – 30/million) varies between countries with a slightly higher number of incomplete SCI and tetraplegia versus paraplegia (for reference see: www.spinalcord.uab.edu). While spontaneous (osteoporotic) compression fractures of the vertebral column rarely show neurological deficit, burst fractures of the cervical and thoracic spine are commonly associated with severe neurological deficits [4, 12, 21, 71, 72, 119].

About 55 % of patients with SCI suffer from tetraplegia

In patients with SCI, the cervical vertebral column is the most frequently injured spine segment resulting in incomplete tetraplegia in 34.3 % and complete tetraplegia in 22.1 % of cases.

In mid-thoracic traumatic fractures, patients mainly suffer from complete paraplegia while fractures at the thoracic-lumbar junction show an incomplete lesion in more than half of the patients [42, 119].

Anatomy and Somatotopic Background

The spinal cord represents the only connection of neurological structures between body and brain for the conduction of motor, sensory and sympathetic-autonomous information. The **parasympathetic innervation** bypasses the spinal cord via the vagal nerve originating from the brainstem. Longitudinally oriented spinal tracts (white matter) surround central areas (**gray matter**) where neuronal cell bodies are located (Fig. 1). Sensory axons entering the dorsal part of the spinal cord originate in the **dorsal root ganglia**, which are located outside the spinal cord. Along with the motor axons originating from the central part of the spinal cord, they leave the spinal segment through the intervertebral foramen at every segment. Furthermore, it is important to realize that the motor synapses between the first and the second motoneurons are located in the ventral part of the gray matter (alpha-motoneuron), whereas the neuronal cell bodies of the peripheral sensory neuron are situated in the dorsal root ganglion within the intervertebral foramen.

The cell bodies of the motoneurons are located in the gray matter

The cell bodies of the sensory neurons are located in the dorsal root ganglion

In the cervical spine there is one pair of cervical nerve roots more than vertebrae bodies. Therefore, the anatomic relationship changes at the cervicothoracic junction. While in the cervical spine the C4 nerve root exits the C3/4 foramen, the L4 nerve root exits the L4/5 foramen in the lumbar spine. In the cervical spine, the cell bodies of the alpha-motoneuron are located approximately one level higher than the exiting nerve root. This is of clinical relevance as focal damage to the anterior spinal cord can cause a more distal deficit than one would expect from the location [25]. Essential **anatomical landmarks** of the somatotopic organization of the spinal cord are:

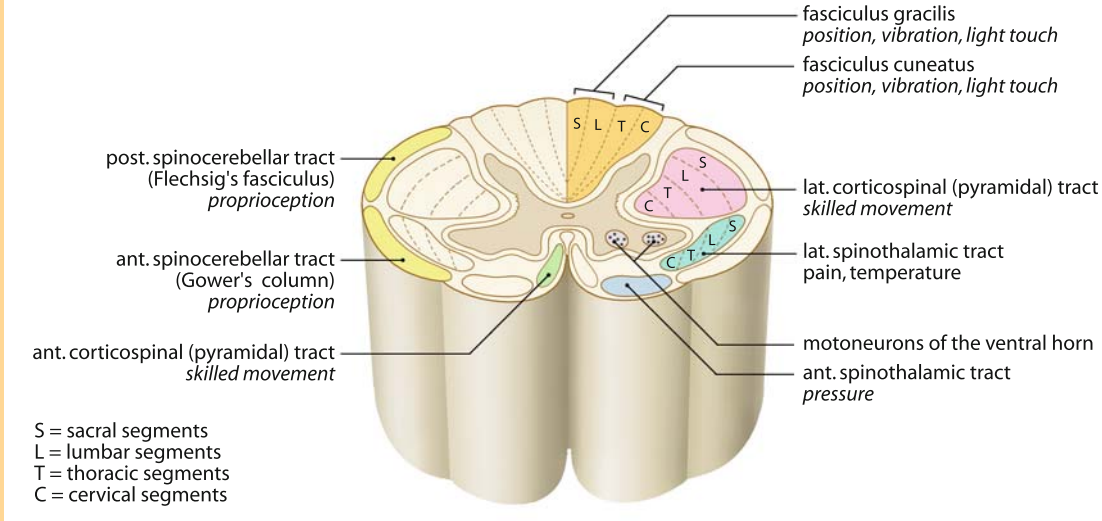


Figure 1. Somatotopic organization of the spinal cord

- the **posterior column** containing sensory nerve tracts conducting position sense (proprioception) and awareness of deep pressure
- the **ventrolateral column** contains spinothalamic tracts for the sensation of pain and temperature
- the **posterior-lateral tract** transmitting voluntary motor control through the pyramidal tract

Classification

A straightforward differentiation of neurological impairment is related to the cause and onset of the disorders and **basically distinguishes** between:

- traumatic injuries
- non-traumatic disorders

Spinal disorders can further be differentiated with regard to the affected **neuronal structures**, i.e.:

- central (CNS) nervous system
- peripheral (PNS) nervous system

A CNS lesion indicates a compromise of the brain or spinal cord, i.e. longitudinal spinal tracts. In contrast, a PNS lesion includes impairment of all the neural structures outlying the spinal cord, i.e. ventral nerve roots and cauda equina nerve fibers within the spinal canal. Therefore, a lesion of the conus medullaris with degeneration of the alpha-motoneurons or the cauda equina shows typical clinical findings of PNS involvement while a lesion higher within the spinal cord mainly presents as a central sensorimotor deficit.

Non-traumatic spinal disorders can be differentiated as listed in Table 1.

Focal compression syndromes predominantly occur in the cervical or lumbar spine

Focal **compression syndromes** of the spinal cord in degenerative disorders are predominantly localized at the cervical and lumbar spinal level [3, 6, 92, 115]. Here, the spine has to cope with the highest biomechanical stress (a high range of motion and being under great strain during daily activities) and is prone to develop a degenerative stenosis resulting either in cervical myelopathy or lumbar spinal canal stenosis and neurogenic claudication. Furthermore, the cervical spinal canal can show a congenitally reduced diameter with increased vulnerability to degeneration or even minimal cervical trauma with severe neurological sequelae [107, 115, 130]. Cervical spinal canal stenosis due to obliterating hypertrophy of the occipital posterior longitudinal ligament (OPLL) and less frequently in the thoracic spine can also induce spinal cord compression even in younger patients [48, 53, 77, 129]. **Spine tumors** of different etiology (intra- or extradural) and dignity always have to be considered in patients assumed to suffer from spinal disorders [1, 44, 66, 81]. **Spinal hemorrhages** predominantly occur acutely/spontaneously in patients undergoing anticoagulation treatment, or suffering from tumors or arteriovenous malformations [37, 58, 83, 91, 114, 116, 126]. While spine compression, tumors and hemorrhages can be reliably diagnosed by imaging (preferably by MRI), the **ischemic, infectious, and degenerative disorders** need a thorough work-up to conclude the specific diagnosis [10, 46].

In atypical cases also consider non-spinal differential diagnosis

Specifically in cases with atypical presentation, disorders other than those of the spinal cord have to be considered in the differential diagnosis. Similarly, in older and multi-morbidity patients, peripheral nerve disorders can be confused with spinal cord disorders and have to be specifically addressed. In patients with a slowly developing polyneuritis, an increasing motor weakness, reduction of walking distance and occurring pain can mimic a lumbar spinal stenosis, while neurophysiological testing can be applied to distinguish between both disorders.

Table 1. Classification of non-traumatic neurological syndromes

Impaired neurological structure	Cause of impairment	Major symptoms
Spinal cord compression	• disc herniation	• severe pain • para-/tetraparesis • bowel/bladder dysfunction
	• congenital cervical stenosis	• clumsy hands with reduced dexterity • ataxic gait
	• degenerative cervical stenosis	• bladder dysfunction micturition problems (urgency, frequency)
	• ossification of the posterior longitudinal ligament (OPLL)	• pain • slowly developing myelopathy • radiculopathy (frequently)
	• lumbar spinal canal stenosis	• neurogenic claudication • low back pain
Spinal cord tumor	• extramedullary intradural tumor (neurinoma, meningeoma, schwannoma) • extramedullary extradural (metastases, lymphoma) • intramedullary tumor (ependymoma, astrocytoma)	• pain syndromes • progressive tetra-/paraparesis • bladder-bowel dysfunction
Spinal hemorrhage	• spontaneous hemorrhage (AV malformation, cavernoma, anticoagulation)	• sudden onset • acute girdle pain • increasing tetra-/paraparesis
Ischemic spinal cord lesion	• ischemia of anterior spinal artery (arteria sulcocommissuralis) • spinal cord malacia (arteria radicularis magna Adamkiewics)	• girdle-like pain prior to weakness • central cord syndrome • acute paraplegia
	• AV malformation	• intermittent claudication
Demyelinating disorders	• multiple sclerosis	• recurrent episodes or primary chronic course of sensorimotor deficits • visual disturbance
	• acute demyelinating encephalomyelitis (ADEM)	• acute onset • cerebral symptoms associated with sensorimotor deficits (mostly after viral infection or vaccination)
	• transverse myelitis	• acute onset with rapid and profound deficits • no clear association with viral infection or other demyelinating CNS disorders
	• neuromyelitis optica (Devic syndrome)	• fulminating progressive para-/tetraplegia • loss of vision
Infectious myelitis	• viral (HSV, HIV, HTLV, EBV, Coxsackie virus, echovirus, poliomyelitis) • bacterial and fungal	• initial girdle-like pain • progressive para- or tetraplegia • spastic spinal paralysis
Physical myelopathy	• radiation/electrical spinal cord damage	• postradiation symptoms (early or late) • beginning with pain • variable syndromes
Hereditary/sporadic degeneration of spinal pathways	• variable mutations of genes, amyotrophic lateral sclerosis	• mainly associated with spastic paraplegia • variable sensory loss • muscle atrophy • bladder dysfunction

A mismatch of clinical findings and imaging studies must prompt a thorough neurological assessment

Therefore, in patients where the radiological and clinical findings are not fully in line with the patient complaints or imaging findings, a thorough neurological work-up should be initiated (**Case Introduction**). For example, the first clinical symptom of a diabetic neuropathy can appear as a severe painful affection of the femoral nerve with a marked paralysis of the quadriceps muscle. This symptom can be easily confused with an L3 radiculopathy and the mismatch between an extensive clinical picture (weakness, loss of reflexes and sensory deficit) and normally appearing lumbar imaging should indicate a further work-up.

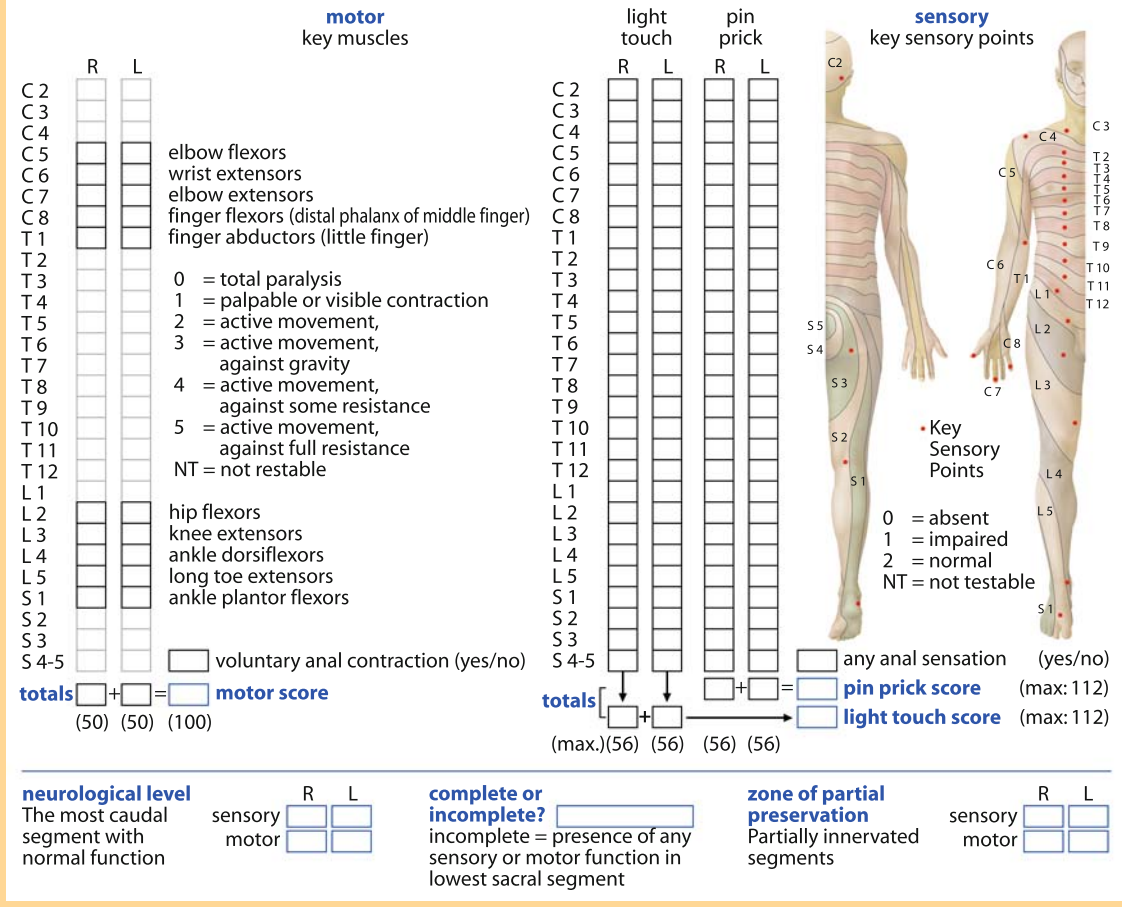


Figure 2. Standard neurological classification of spinal cord injuries (ASIA)

In **traumatic spinal cord injury** the main classification distinguishes between:

- paraplegia
- tetraplegia

The term "paraplegia" refers to the impairment or loss of motor and/or sensory function in the thoracic, lumbar or sacral (but not cervical) neural segments (T2–S5). Impairment or loss of motor and/or sensory function in the cervical segments (C0–T1) is called tetraplegia. In accordance with the standard neurological classification of spinal cord injury (Fig. 2) of the American Spinal Injury Association (**ASIA**), the defined muscles and sensory examination points should be assessed for diagnosis [68].

A further differentiation is made with regard to the **completeness of the lesion** as:

- complete
- incomplete

The distinction between complete and incomplete is based on the preservation of any sensory or motor function within the last sacral segments S4–S5. The ASIA impairment scale (AIS) allows a further grading (Table 2) of the completeness of the lesion [67, 70].

The preservation of lower sacral segments indicates an incomplete lesion

Table 2. ASIA Impairment Scale

ASIA A	• sensory and motor complete
ASIA B	• sensory incomplete, motor complete
ASIA C	• sensory and motor incomplete, motor function below the level of lesion in mean M3
ASIA D	• sensory and motor incomplete, motor function below the level of lesion in mean >M3
ASIA E	• no relevant sensorimotor deficit, minor functional impairments of reflex-muscle tone changes

Neurological Assessment

Complementary to the physical and radiological examination of the spine, the neurological examination focuses on identifying:

- the level of the lesion
- the extent of neural compromise

A detailed history enables an initial broad diagnosis (involvement of upper versus lower limbs, time of onset, trauma) and the neurological examination determines more precisely any possible spinal cord damage. The clinical examination can be complemented by additional neurophysiological studies particularly when the clinical examination is limited due to poor cooperation by the patient. The following **clinical symptoms** should be distinguished by the examiner:

- motor weakness
- sensory deficit
- altered reflexes (cave: spinal shock)
- pain syndromes
- autonomic functions (bowel and bladder dysfunction)

The **examination** can allocate the symptoms to neurological syndromes such as:

- radiculopathy
- polyneuropathy
- myelopathy
- central paresis

Neurological syndromes are non-specific for the underlying pathology

However, neurological syndromes are non-specific with regard to their spinal cause, e.g. a radiculopathy can be caused by a disc herniation, an osseous spur, or a synovial facet joint cyst. From a practical point of view, it is reasonable to differentiate the assessment of patients with and without trauma and the course of symptom onset (acute versus slowly progressive). This differentiation is not always self-evident and has to be specifically identified.

Pain

Pain is the most frequently complained of symptom which can lead one to the impaired neurological structure [49, 95, 108]. The pathophysiology and diagnostic assessment of pain are covered in Chapters 5 and 40.

Sensory Deficits

Distinguish the sensory qualities (light touch, pin prick, proprioception)

Although multiple sensory qualities (heat–cold, pain, touch, pressure, static and dynamic two-point discrimination, vibration sensation) can be distinguished, the **examination of:**

- light touch
- pinprick
- proprioception

is most frequently applied in clinical practice to assess spinal cord dysfunction [13, 41, 51, 62, 84, 89, 99, 101]. While the light touch sensation assesses the perception of touch as applied by the finger or cotton wool, the pinprick sensation identifies the ability to sense a sharp needle tip. The latter function is transmitted via the spinothalamic spinal pathway and the actual examination does not produce different levels of pain. The **key** is that the patient identifies a sharp sensation, which is not necessarily painful. The vibration sense is reliably tested with a tuning fork that allows different grades of vibration recognition to be distinguished [45, 86, 98, 99].

It is important to be aware that particularly incomplete lesions of the spinal cord can cause more diffuse distributed sensory deficits whereas radicular and peripheral lesions result in circumscribed changes. Patients with cervical myelopathy often complain of pain, clumsiness and numbness of the whole hands and/or feet.

Consider central lesions in diffuse/dissociated sensory deficits

In ischemic lesions of the central part of the spinal cord, the predominant clinical finding is an impairment of pain and temperature sensation. In such cases, sensation to touch remains preserved while pain and temperature sensation is abolished, which is typically distributed in a segmental pattern. The affection of the posterior column as induced by a B_{12} hypovitaminosis or rarely due to trauma causes a reduction of the vibration sense with predominant gait disturbance.

Motor Deficits

The differentiation of the causes of muscle weakness can sometimes cause diagnostic difficulties. In general the following lesions should be distinguished:

- peripheral lesion
- radicular lesion
- central lesion

The muscle force should be assessed according to a standardized protocol either following the guidelines of the British Medical Research Council or as modified by the ASIA Standards (see Chapter 8) [70].

A monoparesis of upper or lower limbs is frequently caused by a plexus lesion. Radicular lesions are typically associated with pain emanating into the respective dermatomes and show paresis of the innervated muscles. The differentiation between radicular and peripheral nerve lesion is sometimes difficult (see below).

Painless muscle atrophy demands a detailed neurological differential diagnosis

A **painless atrophy** of hand or foot muscle always demands a neurological work-up and an extended differential diagnosis has to be considered:

- amyotrophic lateral sclerosis
- spinal muscular atrophy
- myelopathy
- neuropathy (hereditary motor neuropathies)

Reflex Deficits

The clinical examination of upper and lower limbs as well as sacral reflexes is mandatory in the assessment of spinal disorders. Reflexes are not only helpful in defining the level of lesion but also in distinguishing acute versus chronic changes. Besides the muscle tendon reflexes, various signs (Figs. 3, 4) and muscle tone testing (clonus, stiffness) are used to screen for **pyramidal tract or conus lesions** [5, 18, 23, 36, 43, 54, 64, 75, 85, 104, 127].

Screen for central lesions using reflex assessments

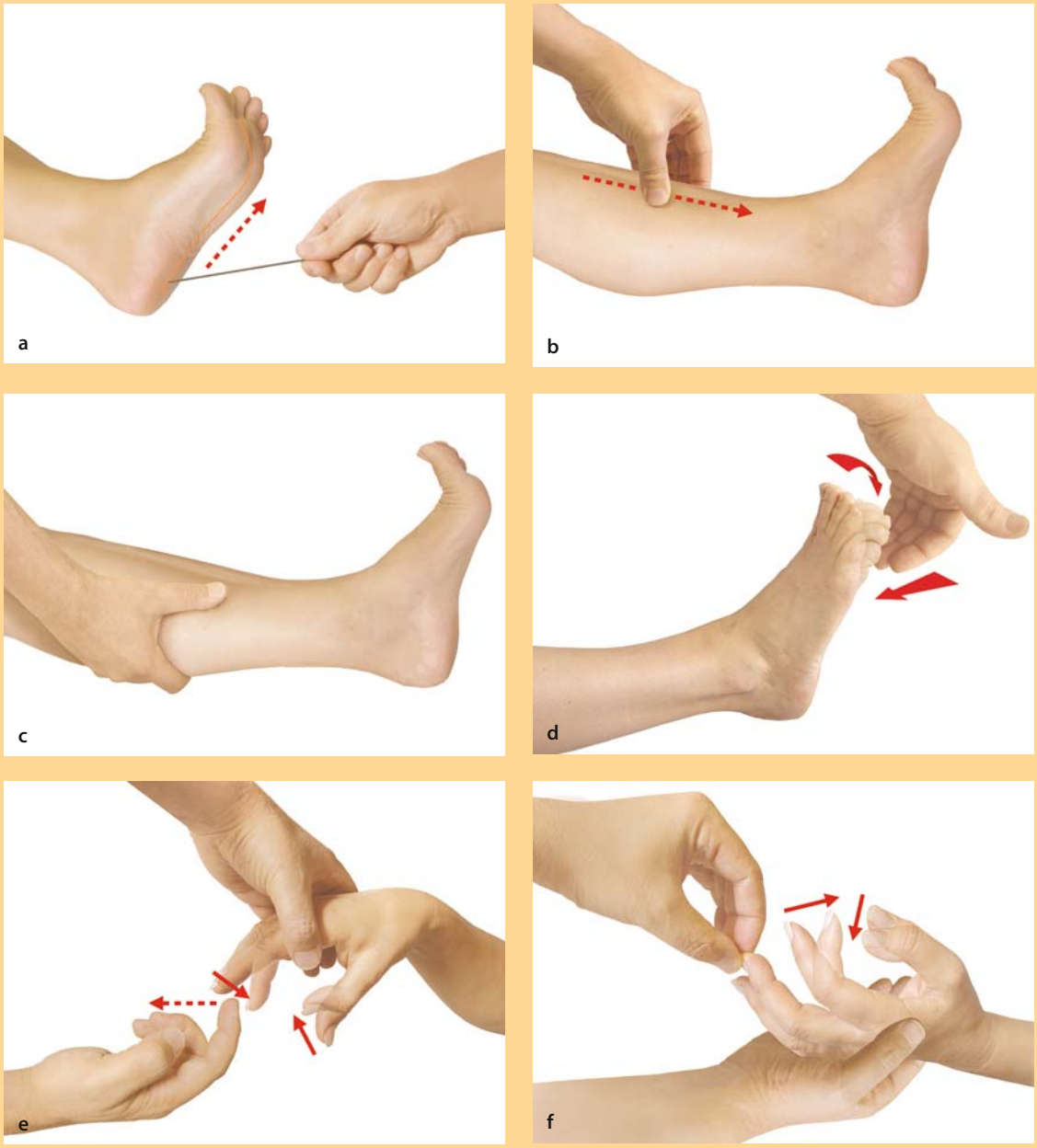


Figure 3. Signs (reflexes) indicating pyramidal tract lesions

a Babinski sign. **b** Oppenheim sign. **c** Gordon sign. **d** Rossolimo sign. **e** Trömner sign. **f** Hoffmann sign. The Hoffmann and Trömner signs can be observed in healthy individuals with hyperexcitability and are only pathologic if they occur unilaterally or in very pronounced fashion.

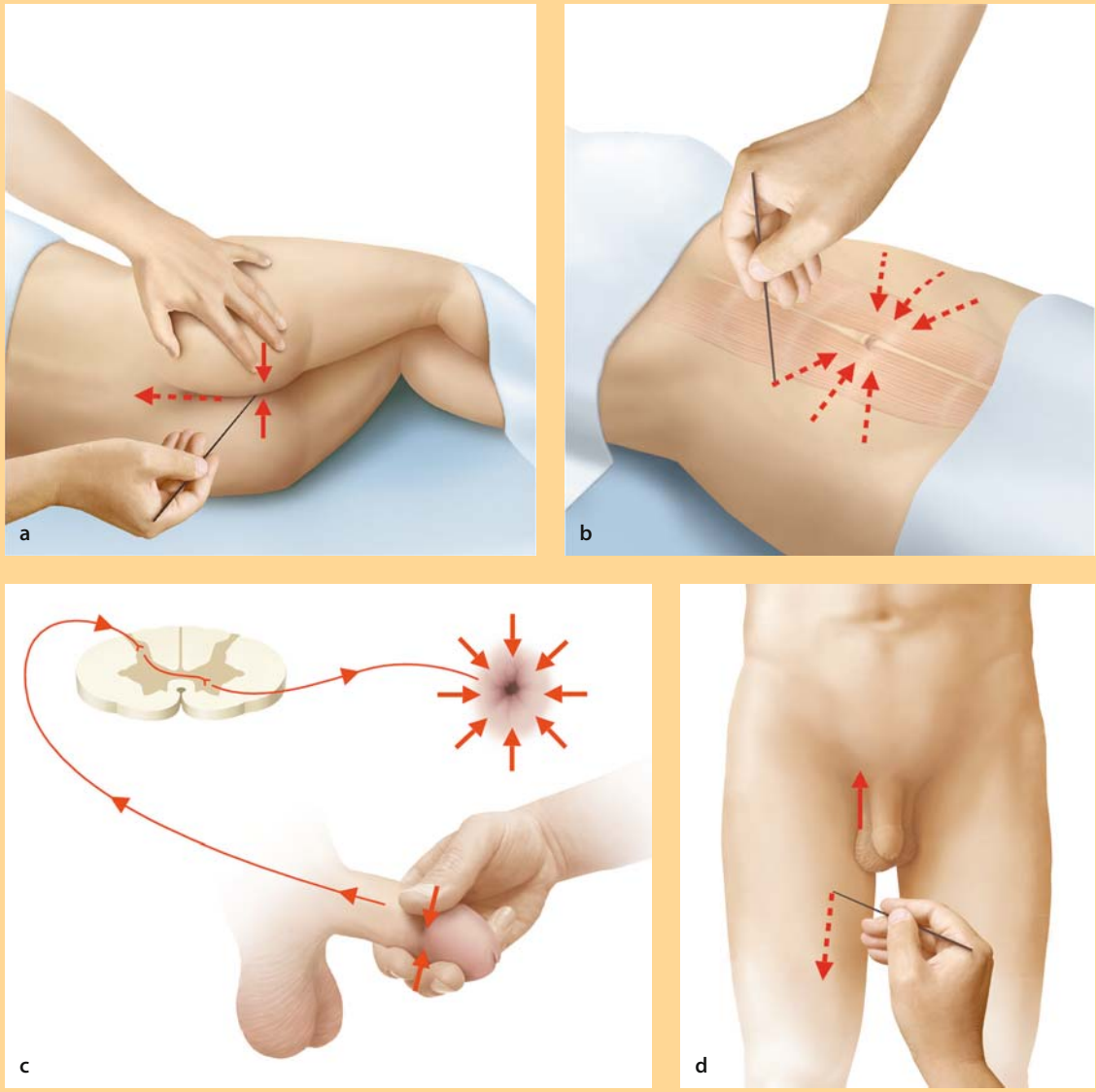


Figure 4. Polysynaptic reflexes

a The absence of the **anal reflex** indicates a lesion at S3 – 5. b Absence of the **abdominal reflex** indicates a lesion at T7 – 12 (screening test for patients with putative idiopathic scoliosis). c Absence of the **bulbocavernosus reflex** indicates a conus medullaris injury. After acute spinal cord injury, the bulbocavernosus reflex can be elicted within 72 h even in spinal shock in contrast to the lower limb tendon reflexes. Recovery of the bulbocavernosus reflex without sensory or motor function indicates a complete spinal cord lesion. d Absence of the **cremaster reflex** indicates a lesion at the level of L1/2.

Gait Disorders

Gait disorder should be detailed by questioning and clinical tests. Ataxic gait with increased danger of falls (impaired balance and ability for line walking), need for an enlarged support base, and increased difficulty in walking in darkness are signs of disturbed proprioception. That may be caused (with decreasing frequency) by:

Gait disorders must be thoroughly differentiated

- polyneuropathy
- posterior column disorders
- cerebellar lesion

Several clinical tests can be applied to distinguish between these disorders.

In **polyneuropathy** the most specific finding is a pattern of loss of reflexes and sensory deficit in a distal and **sock like distribution** (below the knee and/or in the area covered by socks) of impaired light touch sensation and reduction of proprioception. The latter is clinically tested by passively moving the foot or toes up and down and asking the blindfolded patient to describe the direction of movement.

The impairment of **dorsal column function** is clinically tested by **Romberg's test**. This test is named after the German neurologist Moritz Heinrich Romberg (1795 – 1873).

Romberg's test is performed in two stages:

- First, the patient stands with feet together, **eyes open** and hands by the sides.
- Second, the patient **closes the eyes** while the examiner observes **for a full minute**.

Because the examiner is trying to elicit whether the patient falls when the eyes are closed, it is advisable to stand ready to catch the falling patient. For large patients, a strong assistant is recommended. Romberg's test is **positive** if, and only if, the following two conditions are both met:

- The patient can stand with the eyes open; **and**
- The patient falls when the eyes are closed.

The test **is not positive** if either:

- The patient falls when the eyes are open; **or**
- The patient sways but does not fall when the eyes are closed.

Maintaining balance while standing in the stationary position relies on intact sensory pathways, sensorimotor integration centers and motor pathways.

The main **sensory inputs** are:

- joint position sense (proprioception), carried in the dorsal columns of the spinal cord
- vision

Crucially, the brain can obtain sufficient information to maintain balance if either the visual or the proprioceptive inputs are intact. Sensorimotor integration is carried out by the cerebellum. The first stage of the test (standing with the eyes open) demonstrates that at least one of the two sensory pathways is intact, and that sensorimotor integration and the motor pathway are intact. In the second stage, the visual pathway is removed by closing the eyes. If the proprioceptive pathway is intact, balance will be maintained. But if proprioception is defective, both of the sensory inputs will be absent and the patient will sway then fall. Romberg's test is not a test of cerebellar function, as it is commonly misconceived. Patients with cerebellar ataxia will generally be unable to balance even with the eyes open: therefore, the test cannot proceed beyond the first step and no patient with cerebellar ataxia can correctly be described as *Romberg's positive*. Rather, Romberg's test is sensitive to an affection of the proprioception receptors and pathways caused by sensory peripheral neuropathies (such as polyneuropathy) or disorders of the **dorsal columns of the spinal cord**.

Romberg's test is not a test of cerebellar function

Unterberger's stepping test is a simple means of identifying labyrinth dysfunction, which can induce vertigo and dysbalance during walking and standing. During the clinical testing the patient is asked to perform stationary stepping for 1 min with their eyes closed and the arms lifted in front. A positive test is indicated by rotational movement of the patient towards the side of the lesion.

Unterberger's test identifies labyrinth dysfunction

Cerebellar dysfunction is clinically searched for by the **heel-to-knee test** and the **finger-to-nose test**. These tests assess dysmetric and ataxic lower and upper

limb control, which is independent from the impairment of the deep sensory system (proprioception). Patients move the right heel to the left knee and then move the heel with contact to the skin along the tibia bone to the ankle, or point with the tip of the index finger to the tip of the nose (with eyes closed and then opened). The performance of a dysmetric and ataxic movement indicates a cerebellar dysfunction which is not completely corrected with open eyes.

The finger-to-nose and heel-to-knee tests screen for cerebellar dysfunction

Bowel and Bladder Dysfunction

In spinal disorders, bowl and bladder dysfunction are frequently underestimated and patients do not report these problems immediately because they do not realize there is any connection with their spinal problems. Patients have to be **specifically asked for** changes in:

- frequency of micturition
- urgency of voiding
- any kind of urine or bowel incontinence

Asking about **frequency** addresses the question of whether a patient has to visit the bathroom more frequently than they used to. **Urgency** describes whether a patient is able to withhold voiding after the first desire to void or has to visit the bathroom very quickly to avoid incontinence. **Incontinence** can describe a stress incontinence where a physical activity (lifting a heavy object or coughing) that increases the intra-abdominal pressure induces a non-voluntary urine loss or a neurogenic bladder dysfunction with non-voluntary urine loss due to uncontrolled bladder activity (hyperreflexive detrusor). Besides these questions the neurological examination of **sacral segments** is indispensable. After testing the perianal sensitivity for light touch and pinprick (segments S4/S5), the sacral reflexes, **bulbocavernosus reflex** (BCR) and **anal reflex** (AR) have to be examined [5, 104]. Both the BCR and the AR represent the sacral segments S2–S4 (Fig. 4).

A detailed history is needed for bladder dysfunction

It is most important to acknowledge that the function of the bladder (detrusor muscle) cannot be clinically assessed. The clinical diagnosis of urine retention along with the possibility of overflow as a typical finding in an areflexive bladder cannot be reliably distinguished from a reflex bladder activity with incontinence by clinical inspection. Only a full **urodynamic examination** is able to diagnose in detail the bladder function (areflexive versus hyperreflexive detrusor, bladder capacity and compliance) and interaction with the sphincter functions (detrusor sphincter dyssynergia) [29, 76, 103]. The latter test should be considered when the clinical examination shows a pathological finding (sacral motor and reflex disturbance) or the patient describes pathological micturition behavior.

Suspected bladder dysfunction should be investigated by urodynamic assessment

Disorders of the Autonomic System

Deterioration of **autonomous column and sympathetic fibers** which are conducted through the spinal cord becomes obvious in changed hidrosis. Patients may report skin areas with increased (wheat) or reduced (dry skin) sweating (hidrosis). However, these symptoms have to be specifically explored because patients usually do not report these alterations spontaneously. Areas of reduced sweating can be tested by the so-called **spoon test**: A teaspoon is lightly stroked over the skin. On the line of demarcation between the normal (wheat) and impaired (dry) skin region, the spoon has a reduced friction as the skin with reduced hidrosis shows a lower adhesion [15, 20, 22, 74, 96, 97, 109, 121].

The spoon test indicates areas of altered hidrosis

Spinal Cord Injury

SCI is assessed according to the ASIA protocol

For spinal cord injury (SCI), the **Standard for Neurological Classification of SCI** (Fig. 2) as developed by the American Spinal Injury Association (**ASIA**) provides a standardized assessment protocol that can be applied in patients with acute and chronic traumatic SCI [67–69].

The ASIA protocol allows important information to be obtained about the level and extent of lesions in a reasonably short time [35, 67, 68]. It is important to acknowledge that assigning one key muscle and one dermatome (defined by a specific point) to represent a single spinal nerve segment is a simplification. However, it could be shown that the ASIA testing allows for a reliable assessment of the level and extent of lesions [73]. The **neurological level** refers to the lowest segment of the spinal cord with normal sensory and motor function. Differentiation between complete (ASIA A) and incomplete SCI (ASIA B–E) is given by the absence (complete) or preservation (incomplete) of any sensory and motor function in the lowest sacral segment (S4/S5).

The ASIA protocol is not approved for non-traumatic SCI

In the ASIA protocol, appreciation of pinprick (algesia) and of light touch (esthesia) is scored semiquantitatively on a three point scale (absent, impaired, normal). The dermatomal key points defined by ASIA help to perform the sensory examination in a standardized form. The involvement of sacral segments is of predictable value for neurological outcome [125].

However, the ASIA protocol is not a suitable tool with which to guide the diagnosis of disorders affecting extraspinal neuronal structures, e.g. polyneuropathy, plexus lesions or other peripheral neurological lesions. Furthermore, it does not enable central lesions of spinal cord and brain disorders to be distinguished.

A pitfall in the diagnostic assessment of SCI is exhibited by the syndrome of **spinal shock**. This initial state of transient depression of spinal cord function below the level of injury is associated with loss of:

- all sensorimotor functions
- flaccid paralysis
- bowel and bladder dysfunction
- abolished tendon reflexes

Spinal shock can last from several days to weeks. The sacral reflexes [bulbocavernosus (BCR) and anal (AR) reflexes] can be reliably assessed within 72 h after injury and can be applied to search for an involvement of the conus medullaris and cauda equina [5, 123] (Fig. 4).

The **neurophysiological examination** enables valid information to be obtained about the functional deficit of the spinal cord at an early time point after SCI (see Chapter 12) [26, 55].

Spinal Cord Syndrome

Impairment of the intraspinal neural structures, i.e. the myelon and cauda equina, results in typical clinical syndromes. These syndromes may occur with any cause of an incomplete spinal cord lesion and describe by clinical means the primarily affected areas of the spinal cord (Table 3).

- **Brown-Séquard syndrome** (spinal hemisyndrome). This is caused by the deterioration of only half of the spinal cord and results in ipsilateral proprioceptive and motor loss and contralateral loss of pain and temperature perception (dissociated sensitive disorder).
- **central cord syndrome.** This lesion affects the central gray structures of the spinal cord with deterioration of alpha-motoneurons and the crossing

Table 3. Spinal cord injury syndromes

Syndrome	Paresis	Reflexes			Sensory function		Vasomotor dysfunction	Bladder/bowel	Frequent cause
		Tendon tap	Babinski	AR and BCR	Deep pressure	Pain			
Complete lesion									
spinal shock	flaccid	–	+/–	+	–	–	+	flaccid	trauma
C1–T1	spastic tetra	++	+/–	+	–	–	+	spastic	trauma
T2–T12	spastic para	++	+/–	+	–	–	+	spastic	trauma, tumor
conus	spastic and/or flabby	(+)–	(+)	–	–	–	–	spastic/flaccid	trauma
cauda	flaccid	–	–	–	–	–	–	flaccid	trauma, disc herniation
Incomplete lesion									
Brown-Séquard syndrome	spastic hemiparesis	++ ipsilateral	+ ipsilateral	+	– ipsilateral	– contralateral	+/–	–/spastic	trauma
central cord syndrome	spastic tetra (flaccid paresis of upper limbs)	++	+	+	+/–	–	+	spastic	trauma, cervical stenosis, syrinx, disc herniation, OPLL
anterior cord syndrome	flaccid paresis	–	+/–	+	+	–	–	spastic	ischemia
posterior cord syndrome	spastic or no paresis	+/++	+/–	+	–	+	–	spastic	vitamin B_{12} deficiency syndrome

+ positive, ++ increased, – abolished

segmental spinothalamic fibers. The syndrome occurs most frequently in the cervical region.

- **anterior cord syndrome.** This syndrome refers to the disturbance of the anterior spinal artery with consecutive affection of the anterior part (bilateral) of the cord. Thus, there is loss of motor function and of sensitivity to pain and temperature (ventrolateral column).
- **posterior cord syndrome.** This syndrome occurs relatively seldom in trauma and is more frequently seen in non-traumatic disorders (such as B_{12} deficiency). It produces primarily proprioceptive impairment as a result of impaired posterior column.
- **conus medullaris syndrome.** As a result of a compromise of the conus medullaris (sacral spinal enlargement approximately at the spinal level L1–L2 vertebrae) and/or cauda equina (lumbar nerve roots within the spinal canal), a distinct pattern of bladder-bowel dysfunction and lower limb impairment can be observed. Frequently a clear distinction between conus medullaris and/or cauda equina lesion cannot be achieved. A pure cauda equina lesion presents a remaining areflexive bladder dysfunction with loss of sacral reflexes (BCR and AR) and saddle anesthesia. The lower limbs show a flaccid paresis and in time a severe muscle atrophy. A conus medullaris lesion can present a mixture of flaccid and spastic symptoms of both the bladder and lower limbs depending on the localization within the conus. Impotence accompanies both syndromes. The extent of symptoms depends on the degree of damage (complete or incomplete) of the conus medullaris and cauda equina.

Differential Diagnosis

Differentiation of Central and Peripheral Paresis

Spasticity differentiates central and peripheral lesions

The neurological examination should not only confirm if there is any neurological deficit but provide a somatotopic assessment of the location of the lesion. A frequent problem is the **differentiation** between (Table 4):

- central paresis (spastic paresis)
- peripheral paresis (flaccid paresis)

Differentiation between spastic and flaccid paresis allows the distinction of central from peripheral lesions

The differentiation into spastic and flaccid paresis is one of the most significant factors for distinguishing between central and peripheral lesions.

A flaccid paresis indicates reduced or abolished muscle tone, while spastic paresis is described by increased muscle tone with resistance to passive extension, brisk jerks and cloni. The muscle resistance is especially present in fast passive extension and at the start of movement. In the presence of spasticity, the muscle tone should be assessed by the adapted Ashworth score (Table 5) [93, 110, 111].

Differentiation of Radicular and Peripheral Nerve Lesions

If a peripheral lesion is assumed, differentiation of a radicular and peripheral nerve lesion is required. Differences in the dermatomal area of the roots and peripheral nerves as well as differences in the key muscles may be helpful. However, the sensory examination can be very challenging particularly in elderly and young patients, as well as in patients with impaired consciousness and psychiatric disorders. Also the muscle strength testing depends on the cooperation of the patient and is influenced by pain. The somatotopic relation between nerve root and peripheral nerve is summarized in Tables 6 and 7. Because of the similarity of symptoms, the clinical differentiation between some radicular syndromes and peripheral or plexus lesions can be difficult.

Table 4. Clinical differentiation of central and peripheral paresis

Central paresis	Peripheral paresis
• brisk tendon reflexes, muscle cloni	• diminished or absent tendon reflexes
• uni- or bilateral increased stretch reflexes and enlarged reflex zones	• reduced or absent polysynaptic reflexes
• pathological reflexes (Babinski sign, Gordon and Oppenheimer reflexes), uni- and/or bilateral	• no evidence of pathological reflexes
• increased muscle tone	• flaccid muscle tone
• para- or hemi-like distribution of motor deficit	• distribution related to peripheral nerve innervation
• spinal lesions from C1 to L1 (conus medullaris)	• lesions below L2

Table 5. Assessment of spasticity

Ashworth score	Degree of muscle tone
0	• no increase in muscle tone
1	• slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
2	• slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the reminder (less than half) of the ROM
3	• more marked increase in muscle tone through most of the ROM, but affected part(s) easily moved
4	• considerable increase in muscle tone passive, movement difficult
5	• affected part(s) rigid in flexion or extension

Table 6. Peripheral and segmental innervation of upper extremity muscles

	Peripheral innervation	Segmental innervation
Muscles of the shoulder		
trapezius	• accessory n.	• C3–4
latissimus dorsi	• thoracodorsal n.	• C6–8
rhomboids	• dorsal scapular n.	• C5
levator scapulae	• dorsal scapular n.	• C3–5
serratus posterior (superior and inferior)	• thoracic n.s	• T1–12
deltoideus	• axillary n.	• C5–6
supraspinatus	• suprascapular n.	• C4–6
infraspinatus	• suprascapular n.	• C4–6
teres minor	• axillary n.	• C5–6
teres major	• subscapular n.	• C5–6
subscapularis	• subscapular n.	• C5–6
Muscles of the arm		
biceps brachii	• musculocutaneous n.	• C5–7
brachialis	• musculocutaneous n.	• C5–7
coracobrachialis	• musculocutaneous n.	• C5–7
triceps brachii	• radial n.	• C7–8
anconeus	• radial n.	• C7–8
pronator teres	• median n.	• C6–7
flexor carpi radialis	• median n.	• C6–7
palmaris longus	• median n.	• C6–7
flexor digitorum superficialis	• median n.	• C7–T1
flexor carpi ulnaris	• ulnar n.	• C8–T1
flexor digitorum profundus	• ulnar n. (ulnar side) • median n. (radial side)	• C8–T1
flexor pollicis longus	• anterior interosseous branch of median n.	• C8–T1
pronator quadratus	• anterior interosseous branch of median n.	• C8–T1
brachioradialis	• radial n.	• C5–6
extensor carpi radialis longus	• radial n.	• C6–7
extensor carpi radialis brevis	• radial n.	• C6–7
extensor digitorum	• deep branch of radial n.	• C6–8
extensor digiti minimi	• deep branch of radial n.	• C6–8
extensor carpi ulnaris	• deep branch of radial n.	• C6–8
extensor pollicis longus	• deep branch of radial n.	• C6–8
extensor indicis longus	• deep branch of radial n.	• C6–8
abductor pollicis longus	• deep branch of radial n.	• C6–8
extensor pollicis brevis	• deep branch of radial n.	• C6–8
supinator muscle	• deep branch of radial n.	• C6
Muscles of the hand		
palmaris brevis	• superficial branch of ulnar n.	• C8–T1
abductor pollicis brevis	• median n.	• C8–T1
opponens pollicis	• median n.	• C8–T1
flexor pollicis brevis	• median n. (superficial head) • ulnar n. (deep head)	• C8–T1
adductor pollicis	• deep palmar branch of ulnar n.	• C8–T1
lumbricales	• median n. (1st and 2nd) • ulnar n. (3rd and 4th)	• C8–T1
abductor digiti minimi	• deep palmar branch of ulnar n.	• C8–T1
flexor digiti minimi brevis	• deep palmar branch of ulnar n.	• C8–T1
opponens digiti minimi	• deep palmar branch of ulnar n.	• C8–T1
palmaris brevis	• deep palmar branch of ulnar n.	• C8–T1
interosseous	• deep palmar branch of ulnar n.	• C8–T1

According to Sobotta [113]

Table 7. Peripheral and segmental innervation of lower extremity muscles

	Peripheral innervation	Segmental innervation
Muscles of the hip and thigh		
iliopsoas	• muscular branch of the lumbar plexus	• L1–4
sartorius	• femoral n.	• L2–3
quadriceps	• femoral n.	• L2–4
pectineus	• femoral n.	• L2–4
adductor longus	• anterior branch of obturator n.	• L2–4
adductor brevis	• anterior branch of obturator n.	• L2–4
gracilis	• anterior branch of obturator n.	• L2–4
obturator externus	• anterior branch of obturator n.	• L3–4
adductor magnus	• posterior branch of obturator n. • tibial part of sciatic n.	• L2–4 • L4–S1
gluteus maximus	• inferior gluteal n.	• L5–S1
gluteus medius	• superior gluteal n.	• L4–S1
gluteus minimus	• superior gluteal n.	• L4–S1
tensor fascia lata	• superior gluteal n.	• L4–S1
piriformis	• 1st and 2nd sacral n.s	• S1–2
obturatus internus	• n. to obturator internus	• L5–S2
gemelli	• n. to obturator internus	• L5–S2
quadratus femoris	• n. to quadratus femoris	• L5–S2
Muscles of the leg		
biceps femoris	• tibial portion of the sciatic n. (long head) • peroneal portion of the sciatic n. (short head)	• S1–3 • L5–S2
semitendinosus	• tibial portion of the sciatic n.	• L5–S2
semimembranosus	• tibial portion of the sciatic n.	• L5–S2
tibialis anterior	• deep peroneal n.	• L4–S1
extensor hallucis longus	• deep peroneal n.	• L4–S1
extensor digitorum longus	• deep peroneal n.	• L4–S1
triceps surae	• tibial n.	• S1–2
soleus	• tibial n.	• S1–2
plantaris	• tibial n.	• S1–2
popliteus	• tibial n.	• L4–S1
tibialis posterior	• tibial n.	• L5–S1
flexor digitorum longus	• tibial n.	• L5–S1
flexor hallucis longus	• tibial n.	• L5–S1
peroneus longus	• superficial peroneal n.	• L4–S1
peroneus brevis	• superficial peroneal n.	• L4–S1
Muscles of the foot		
extensor digitorum brevis	• deep peroneal n.	• L5–S1
extensor hallucis brevis	• deep peroneal n.	• L5–S1
abductor hallucis	• medial plantar n.	• L5–S1
flexor hallucis	• medial plantar n.	• L5–S1
adductor hallucis	• lateral plantar n.	• S2–3
abductor digiti minimi	• lateral plantar n.	• S2–3
flexor digiti minimi	• lateral plantar n.	• S2–3
opponens digiti minimi	• lateral plantar n.	• S2–3
flexor digitorum brevis	• medial plantar n.	• L5–S1
quadratus plantae	• lateral plantar n.	• S2–3
interossei	• lateral plantar n.	• S1–2

According to Sobotta [113]

Radiculopathies

The clinical presentations of the radicular syndromes are summarized in Table 8.

The exact differentiation between radicular and peripheral nerve damage may demand neurophysiological studies, i.e. EMG to show denervation of root- and/or nerve-specific muscles as well as neurography to exclude conduction delay of the peripheral nerve. **Entrapment syndromes** are an important differential diagnosis of radicular lesions. Knowledge of the characteristic symptoms is mandatory (Table 9).

C5 Radiculopathy

In contrast to an isolated lesion of the **musculocutaneous nerve**, a C5 lesion causes not only a paresis of the biceps muscle, but also of the scapular muscle

Table 8. Radicular syndromes and differential diagnosis

Root	Dermatome	Muscle	Reflex	Important differential diagnoses
C1–4	• neck and collar	• neck muscles • diaphragm (paradoxic abdominal muscle movements)	–	• lung carcinoma • neuritis of brachial plexus • lymphoma • thymome
C5	• lateral shoulder	• deltoid muscle	• biceps reflex	• frozen shoulder • Erb's palsy • neuralgic amyotrophy of the shoulder • palsy of axillary nerve
C6	• lateral arm and thumb	• extensors of hand, flexors of elbow	• biceps reflex • brachioradial reflex	• carpal tunnel syndrome • radial nerve palsy • musculocutaneous nerve palsy
C7	• dorsum of shoulder and arm into the long finger	• triceps, wrist flexors, finger extensors	• triceps reflex	• palsy of posterior interosseus nerve, brachial plexus paralysis (middle part)
C8–T1	• medial arm into ulnar two digits	• intrinsic hand muscles	• Trömner's reflex	• palsy of anterior interosseus nerve • brachial plexus paralysis (Klumpke type) • thoracic outlet syndrome • ulnar palsy
L2	• inguinal ligament	• iliopsoas	• cremaster reflex	• femoral palsy • hip osteoarthritis • pelvic disorder (i.e. psoas muscle)
L3	• medial femoral and knee	• femoral adductors, vastus medialis of quadriceps muscle	• adductor reflex	• paralysis of obturator nerve • pelvic disorder (aseptic necrosis of symphysis) • hip osteoarthritis
L4	• lateral femoral and medial shank	• vastus lateralis of quadriceps muscle	• patellar reflex	• paralysis of femoral nerve
L5	• lateral shank	• tibialis anterior muscle	• tibialis posterior reflex	• peroneal paralysis
S1	• dorsal shank, along heel into fifth digit of foot	• gastrocnemius muscle	• Achilles tendon reflex	• tibial paralysis • tarsal tunnel syndrome
S2	• dorsal femoral	• ischiocrural muscles	• biceps femoris reflex	• sciatic pain syndrome
S3	• proximal medial femoral	• bulbocavernosus muscle and anal sphincter	• bulbocavernosus and anal reflex	• palsy of cutaneus posterior femoral nerve (sacral plexus)
S4–5	• perineum	• bulbocavernosus muscle and anal sphincter	• bulbocavernosus and anal reflex	• palsy of clunium medii • palsy of anococcygei nerves (coccygeal plexus)

Table 9. Frequent entrapment syndromes

Syndrome	Findings
Carpal tunnel syndrome	• pain of hand and forearm, frequently at night (antebrachialgia nocturna) hypesthesia of digits 1 to 3 including the radial side of digit 4 paresis and atrophy of the thenar muscles positive Tinnel sign over the carpal tunnel
Sulcus ulnaris syndrome	• numbness of digits 4 and 5 paretic intrinsic hand muscles and hypothenar muscles positive Tinnel sign over the ulnar sulcus
Thoracic outlet syndrome	• paresis of the intrinsic hand muscles worsening of symptoms by elevating the shoulder frequently associated with cervical rip or ligamental hypertrophy pain of hand and forearm
Fibularis syndrome	• paretic foot elevation numbness of the dorsal foot often history of repeating pressure over the fibular caput
Tarsal tunnel syndrome	• paresis of short foot muscles numbness of the plantar foot atrophy of abductor hallucis muscle

group (supra- and infraspinatus, teres major and minor muscles). The sensory deficits of a C5 radiculopathy are located at the posterolateral upper arm while the musculocutaneous nerve also innervates the ventral aspects (see Chapter 8).

C6 Radiculopathy

The sensory deficits in a C6 lesion may mimic median nerve lesion. However, in median nerve lesion neither is the biceps tendon reflex (BTR) diminished nor the biceps muscle paretic. Similarly, the middle finger is typically not involved in a C6 hypesthesia but in a median nerve lesion.

C8/T1 Radiculopathy

This radiculopathy must be distinguished from an **ulnar nerve lesion**. In C8/T1 radiculopathy, the ulnar side of the forearm is hypesthesic and all intrinsic hand muscles are affected. The ulnar nerve is mostly compressed within the sulcus, resulting in paresis of the hypothenar and only those intrinsic hand muscles innervated by the ulnar nerve. The sensory deficit affects the two ulnar fingers.

L3/4 Radiculopathy

In a neuropathy of the **femoral nerve** and in L3/4 radiculopathy, the patellar tendon reflex (PTR) is reduced or abolished with a predominant weakness of the quadriceps muscles. However, detailed testing in femoral nerve neuropathy shows a sensory deficit restricted to the ventral aspect of the thigh with paralysis of hip flexion (iliopsoas muscle) while in L3/4 radiculopathy the sensory deficit is extended to the medial site and below the knee with weakness of the thigh adduction (adductor muscles).

L5 Radiculopathy

Paresis of foot elevation can be due to a L5 radiculopathy and/or a lesion of the peroneal nerve (see Chapter 8, Case Introduction). Clinical differentiation is

possible by proving the hip abduction, which is also affected in a L5 radiculopathy with weakness of the gluteal muscles (gluteus medius, tensor fasciae latae).

S1 Radiculopathy

In suspected S1 radiculopathy, damage of the **tibial nerve**, e.g. tibial tunnel syndrome or partial sciatic lesion, has to be excluded. While S1 radiculopathy is signaled by diminished Achilles tendon reflex and weak foot extension, the tibial nerve affection involves the toe and ankle extensor muscles while the peroneal nerve lesion shows paresis of the toe and ankle flexor muscles.

Differential Diagnosis of Spinal Cord Compression Syndromes

This group of syndromes is due to obliteration of the spinal canal resulting in compression of the neural structures. Both cervical and lumbar stenosis frequently originate from degenerative (secondary) changes of the spine. Also a congenitally narrow spinal canal (primary spinal canal stenosis) can be present, which exposes the patient to an increased risk of compression syndromes and a greater danger of neuronal damage in minor spine trauma. In Asian people (e.g. Japanese individuals), an ossified posterior longitudinal ligament (OPLL) can cause spinal cord compression, which is only rarely described in Caucasian people. Although all compression syndromes present with distinct symptoms, differential diagnosis from other disorders is mandatory in equivocal cases (Table 10).

Table 10. Spinal cord compression syndromes

Compression syndrome	Symptoms	Differential diagnosis
Cervical stenosis	• clumsy painful hands • disturbed fine motor skills • imbalance of gait • numb feet • urinary urgency	• multiple sclerosis • Myelitis • B_{12} hypovitaminosis • spinal tumors • polyneuropathy (PNP) • arteriovenous malformations
Thoracic stenosis	• lower limb sensory deficit • thoracic sensory level • spastic paraparesis • bladder-bowel dysfunction	• disc herniation (often calcified) • OPLL • arteriovenous malformations • spinal tumors
Lumbar stenosis	• tired legs and weakness on walking • lumbar pain on walking • pain relief during sitting, lying and forward bending	• vascular claudication • spinal metastasis • polyneuropathy
Cauda equina syndrome	• severe leg pain • flaccid paraparesis • sensory loss of legs • urinary and bowel incontinence • saddle anesthesia	• cauda equina radiculitis (Elsberg's syndrome) • lesion of pelvic plexus

Miscellaneous Differential Diagnoses

Neurovascular Disorders

Non-traumatic acute paraplegia may be due to spinal ischemic or hemorrhagic disorders. Typically, the first symptom is girdle-like pain in the dermatome referring to the involved level. Thereafter, motor paresis and sensory deficits appear, mostly within minutes to a few hours. A very special but not so uncommon disor-

Girdle-like pain may be an initial symptom of a spinal ischemic or hemorrhagic disorder

der is the spinal decompression syndrome, which can be seen in scuba divers. When the time requirement for decompression after deep diving is not adequately followed (decompression sickness), microembolisms of non-resolved nitrogen gas emboli can obstruct small branches of the anterior spinal artery and cause a spinal ischemia. This can induce an anterior/central cord syndrome or even complete SCI and represents one of the most serious complications in diving [2, 19, 57, 59, 87]. In contrast hemorrhagic disorders are mostly based on arteriovenous malformation or spontaneous spinal bleeding in patients with anticoagulation treatment and often result in complete paraplegia.

Neurodegenerative Disorders

Neurodegenerative disorders can be easily confused with spinal disorders particularly in the early stages

Based on its frequency, **multiple sclerosis** is the most important differential diagnosis in suspected disorder of the spinal cord. Increased reflexes, ataxia, numbness and paresis of limbs and bladder dysfunction can occur in both multiple sclerosis and myelopathy. However, the presence of MRI signal changes (white spots in T2 weighted images) in the brain and of the spinal cord without or with only minor spinal cord compression indicating neurodegenerative-immunologic disorders should be taken into the differential diagnosis. The definitive differential diagnosis demands further diagnostics, particularly the examination of evoked potentials and the CSF [14, 50, 52, 63, 94].

Also very rare neurodegenerative disorders, e.g. **amyotrophic lateral sclerosis** (ALS), in combination with minor degenerative spinal disorders can potentially mimic a spinal disorder.

Inflammatory Disorders

A number of infectious diseases can be associated with myelitis. Various viruses, i.e. herpes virus, human immune deficiency virus or poliomyelitis, may affect the spinal cord, roots or peripheral nerves. With regard to the opportunities for therapy, the diagnosis of a bacterial or viral infection of the spinal cord is particularly important. Inflammatory disorders are often associated with systemic signs of infection such as fever or respiratory infection and can show cutaneous efflorescences particularly in herpes zoster infection (Case Introduction). In patients with assumed herpes zoster infection, immediate treatment with antiviral medication (acyclovir) is recommended.

Recapitulation

Epidemiology. Even though neurological symptoms in spinal disorders are not frequent, the neurological examination is most important for the planning of further diagnostic assessments and therapy. In contrast to patients with traumatic spinal disorders, who are mainly young patients suffering from non-traumatic spinal disorders, most patients are elderly. The most frequently involved nerve roots are C5, C6, L5 and S1. In SCI about 45 % of patients suffer from tetraplegia.

Classification. Neurological symptoms should be related to the involved neural structures and differentiate lesions of the central and peripheral nervous system. Depending on the impaired spinal segments, spinal cord injury is classified as paraplegia or tetraplegia and complete or incomplete.

Pathogenesis. Traumatic and non-traumatic spinal lesions are distinguished while the neurological symptoms are non-specific to the cause of lesion. Therefore, in spinal disorders with unknown pathology, a broad differential diagnosis has to be considered. In patients with acute onset of symptoms, spinal, radicular and peripheral nerve disorders should be distinguished.

Clinical presentation. The **medical history** focuses on the time of onset and duration of actual complaints, dependence on physical activities as well as other disorders that might impact spinal cord function. Radicular and peripheral lesions mostly cause localized pain, muscle paresis and sensory disorders in the related dermatomes. In contrast, deterioration of spinal cord function results in more bilateral and complex symptoms (impaired upper limb – hand function, gait disorder, bladder and bowl dysfunction). Duration of symptoms is important for the definition of etiology and urgency of therapy (e.g. cauda equina syndrome). While acute traumatic disorders are most obviously degenerative, metabolic and infectious diseases have be considered carefully.

Neurological examination. In spinal disorders it is absolutely mandatory to exclude any neurological lesions. Depending on the neurological deficit, further diagnostic assessments should be initiated. To assure a timely and thorough assessment, the clinical examination has to follow an appointed algorithm.
After observing the **gait**, **proprioceptive reflexes** and **pathologic reflexes** have to be assessed. In peripheral lesions, proprioceptive reflexes are absent or diminished, while in central lesions they might be increased (cave: spinal shock). Pathological reflexes indicate central (spinal and supraspinal) lesions. **Motor strength** is subdivided into six grades (M0 – M5), and key muscles both for radicular and spinal lesions should be examined. The **muscle tonus** has to be tested to differentiate spasticity (modified Ashworth scale 1 – 5) from flabby paresis. Subsequently, a **sensory examination** for touch and pinprick sensation is performed. Impairment of posterior column is diagnosed by assessing the sense of vibration. Deterioration of **sympathetic fibers** appears in changed hidrosis. In every case with or without complained of bladder or bowel dysfunction, the **sacral segments** have to be examined. However, the neurological examination is not sensitive to the assessment of autonomic disorders (bladder, bowl, sexual and cardiovascular dysfunction). In SCI the ASIA protocol enables the neurological examination to be performed in a standardized form. Further neurological tests depend on the results of the clinical examination (detailed examination of hand function, exclusion of cerebral damage, peripheral nerve lesion, etc.).

Key Articles

Maynard FM, Jr, Bracken MB, Creasey G, Ditunno JF, Jr, Donovan WH, Ducker TB, et al. (1997) International Standards for Neurological and Functional Classification of Spinal Cord Injury. American Spinal Injury Association. Spinal Cord 35(5):266 – 74
This article describes the internationally standardized classification of a neurological deficit after a traumatic spinal cord injury to score the extent (complete–incomplete) and level of the spinal cord damage. It is the standard used in almost all SCI studies since 1996.

Siddall PJ, Loeser JD (2001) Pain following spinal cord injury. Spinal Cord 39(2):63 – 73
For the distinction of the frequently present different pain syndromes after SCI, the paper presents the first internationally accepted clinical algorithm to qualify the complained of pain and to distinguish the potential different causes.

Priebe MM, Sherwood AM, Thornby JI, Kharas NF, Markowski J (1996) Clinical assessment of spasticity in spinal cord injury: a multidimensional problem. Arch Phys Med Rehabil 77(7):713 – 6
The clinical description and quantification of spasticity in SCI can be semiquantitatively documented by a standardized score and allows for monitoring changes over time.

Vroomen PC, de Krom MC, Wilmink JT, Kester AD, Knottnerus JA (2002) Diagnostic value of history and physical examination in patients suspected of lumbosacral nerve root compression. J Neurol Neurosurg Psychiatry 72(5):630 – 4
This paper demonstrates that the medical history provided by the patient about the onset and characteristics of radicular pain is of highest value for the diagnosis of a lumbar-sacral nerve root compression. The study outlines that clinical tests and neuro-imagine provide additional information but are only relevant in combination with a thoroughly taken medical history.

Verbiest H (1954) A radicular syndrome from developmental narrowing of the lumbar vertebral canal. J Bone Joint Surg 36:230 – 237
Landmark paper describing the clinical characteristics of the neurogenic claudication due to lumbar spinal canal stenosis.

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