Eur Spine J (2003) 12 (Suppl. 2) : S181–S187
DOI 10.1007/s00586-003-0631-y

REVIEW

Jiri Dvorak
Martin Sutter
Joerg Herdmann

Cervical myelopathy: clinical and neurophysiological evaluation

Received: 19 August 2003
Accepted: 21 August 2003

J. Dvorak (✉)
Department of Neurology, Schulthess Clinic Spine Unit,
Lengghalde 2, 8008 Zürich, Switzerland
Tel.: +41-1-3857445,
Fax: +41-1-3857574,
e-mail: dvorak@kws.ch

M. Sutter
Department of Neurophysiology, Intraoperative Monitoring,
Schulthess Clinic Spine Unit,
Zürich, Switzerland

J. Herdmann
Department of Neurosurgery, SPINE Unit,
Heinrich-Heine-University,
Düsseldorf, Germany

Abstract The overall frequency of troublesome neck pain is estimated to be about 34%, and it was observed that the frequency of complaints lasting 1 month or longer was higher in women than in men. The prevalence increased with age, with regard to both pain duration and chronic pain. Approximately 14% of a randomly selected population meets the criterion for chronic neck pain: complaints lasting more than 6 months. Epidemiologic data substantiate the importance of morphologic, age-related changes of the cervical spine; however, the incidence and prevalence of cervical myelopathy is not known. It could be that the structural transformation of the intervertebral disc, the uncovertebral processes and the zygapophyseal joints is a process accompanied by disturbed function that ultimately not only induces pain, but can lead to narrowing of spinal canal, with symptoms and signs of cervical myelopathy. For a diagnosis of radicular and myelopathic syndromes, the functional and neurological examination is enhanced by neurophysiological assessment. Electromyography (EMG) performed with needle electrodes is the oldest method for diagnosing nerve root compression and anterior horn cell syndromes, and is claimed to have no false-positive results. For cervical myelopathy, as a routine examination sensory evoked potentials (SEPs) by stimulation of tibial nerve and motor evoked potentials (MEPs) from the upper and lower extremities are recommended.

Keywords Cervical spine · Myelopathy · Diagnosis · Evoked potentials

Introduction

Herbert von Luschka, a German anatomist, first pointed out the developmental changes of the cervical spine's anatomical structures, i.e., the uncovertebral joints, also commonly called Luschka's joints [21]. The processus articularis are covered by a thin layer of cartilage in healthy subjects, and the uneven surfaces in between the zygapophyseal processes are filled in by an infolding of the joint capsule described by Penning and Töndury as meniscoids [31]. These meniscoids consist of connective and fatty tissue, which is highly vascularized and innervated.

In healthy adults, the intervertebral discs in the cervical spine have a structure similar to that of the discs of the lumbar spine, consisting of the annulus fibrosus and nucleus pulposus. However, it has been observed that in the first and second decades of life, before complete ossification occurs, lateral tears do occur in the annulus fibrosus. The tears in the lateral part of the disc tend to enlarge towards the medial aspect of the intervertebral disc.

These anatomical observations by Töndury document the fact that, with increased age, the disc cannot bear or transfer load due to ongoing dehydration, medial splitting of the disc and the disappearance of the nucleus pulposus [40]. With the increased load on the uncovertebral processes, a new cow-horn-like uncovertebral flattening takes over the load bearing function of the intervertebral joint. It is obvious that such transformation of bony structures can lead to irritation or compression of the spinal nerve as well as the vertebral artery, which of course can cause not only intermittent or chronic pain and finally narrowing of the spinal canal due to bony growth, but also demyelisation of ascending and descending spinal pathways, due to a possible deficiency of blood supply to the spinal cord [9].

Those age-related, morphological changes, as described by Töndury, should be clearly differentiated from disc lesions due to trauma, where tears of the intervertebral disc at the vertebral body endplate have been documented [38].

Evidence of radiological degenerative changes of the cervical spine in the aging population are common. By the fourth decade of life, 30% of asymptomatic subjects show degenerative changes of the intervertebral discs, while by the seventh decade, up to 90% have developed degenerative alterations [39]. Similar findings were earlier presented by Kellgren and Lawrence [17, 19]. Therefore, it is always important to interpret these radiological findings in the light of the clinical picture. If symptoms and findings cannot be logically correlated, the presence of a different pathology should be suspected, and appropriate investigations are indicated.

Close collaboration between the orthopedic surgeon, the neurosurgeon and the neurologist is required in the assessment of the patient in the spine unit, in order to optimally indicate and analyze the clinical, radiological and laboratory findings, including neurophysiology, and relate them to the patient's symptoms.

Based on the history and the physical signs, a rational neurological work-up should be designed in order to confirm or reject the indication for spinal surgery. Patients with cervical spine disorders most commonly complain of local and referred pain, headache, dizziness or disturbance of the equilibrium, paresthesias, and weakness in the upper and lower extremities. In addition to the complete neurological assessment, which includes an examination of the cranial nerves and the upper and lower extremities, additional laboratory examinations may be required and may be helpful in the differential diagnosis, including:

- Analysis of cerebrospinal fluid (CSF)
- Electromyography (EMG)
- Electroneurography (ENG)
- Sensory evoked potentials (SEP)
- Motor evoked potentials (MEP)
- Computerized tomography (CT)
- Magnetic resonance imaging (MRI)

In the differential diagnosis of cervical spondylosis with involvement of neural structures leading to cervical spinal myelopathy, the following diseases should be considered:

- Multiple sclerosis
- Amyotrophic lateral sclerosis (ALS)
- Polyradiculitis (Landry-Guillain-Barré)
- Shoulder amyotrophy
- Borreliosis (Lyme disease)
- Syringomyelia
- "Double crush" lesion of the nerve root and peripheral nerve
- Rheumatoid arthritis with involvement of the cervical spine
- Psychogenic disorders (hysteria)

Neurological examination

The neurological examination aims to differentiate between nerve root and spinal cord compression. Examination of cranial nerves, especially the eye movements with the aid of Frentzel goggles, is useful. There is clear evidence showing interaction between the receptors of the cervical joint capsules and the vestibular organ [28, 29]. However, it is well established that the center projection of the cervical spine mechanoreceptors is close to the vestibular nuclei at the region of the brain stem, which makes the clinical differentiation (cervical vs vestibular origin of dizziness) very difficult [26, 27].

Neck pain may be the first clinical symptom of a slowly growing acusticus neurinoma, with absent corneal reflex being the first sign. Patients with referred pain in the region of trigeminus nerve pain commonly present an underlying pathology of the upper cervical spine, often observed in atlanto-axial instability due to rheumatoid arthritis [38, 42].

Electric pain along the spine irradiating to the extremities during maximal flexion and extension of the cervical spine has been described in patients with multiple sclerosis as the Lhermitte sign, but it is also generally observed in patients suffering from compression of the cervical spinal cord [20]. Radicular arm pain during ipsilateral sidebending rotation and manual compression of the head is described as the Spurling test, and expresses itself as a motion-induced radicular irritation/compression radiating pain along the involved dermatoma.

In patients where compression of the spinal cord is suspected, a neurological examination of the upper and lower extremities should be routinely performed.

According to the type of the lesion, the spinal cord will react primarily with demyelinisation of the descending and/or ascending pathways with the classical symptomatology of tetraspastics, pathologically increased muscle tendon reflexes, positive Babinski sign, absent abdominal reflexes and decreased vibratory sense on the lower extremities. It is not always appreciated, however well described by Ebara et al., that some patients with spinal cord compression will present atrophy of the small muscles of the hands, described as "myelopathic hand," as a result of segmental anterior horn cell necrosis [11, 30]. Shimizu et al. systematically observed the blind zone of the upper cervical spinal cord and analyzed the hyperactive scapulohumeral reflex, which was described for the first time by Bechterev in 1900 [36]. Compression of the spinal cord at the level C2/C3 will result in hyperactive scapulo-humeral reflex.

One of the first symptoms and also signs of cervical myelopathy is gait disturbance, especially in dark surroundings, when the optical control should be compensated for by the proprioceptive receptors in the feet.

The European Myelopathy Score (EMS)

To asses the severity of cervical myelopathy, the European Myelopathy Score has been proposed [13], based upon the JOA (Japanese Orthopaedic Association) score.

The European Myelophathy Score has five subscores (Table 1). The significance of the each subscore is weighted by the maximal number of points that is achieved if the subscore is normal. All of these subscores are functional criteria that do not require formal testing. They can be obtained by taking the patient's history, or even by questionnaires filled in by the patients themselves. The upper motor neuron is critical in the control of lower limb function. Gait is of major importance for judgment of cervical myelopathy. It is the only subscore that can reach 5 points. Bladder and bowel function (3 points) depend on both motor and sensory integrity. In cervical myelopathy, however, bladder or bowel dysfunction is caused primarily by a bilateral upper motor neuron lesion. Cervical myelopathy is generally due to degenerative changes of the middle and lower cervical spine. Therefore, impairment of hand function can be attributed mainly to lower motor neuron function (4 points), although similar disturbances of precision movements are also seen in upper motor neuron function or cortical lesions. Proprioception and coordination depend on posterior column function (3 points). Posterior column function was included in the European Myelopathy Score instead of the JOA subscores for sensory function – a disturbance which is very difficult to classify into categories. Pain is not a major symptom in cervical myelopathy. Nevertheless, unpleasant sensations such as paresthesia or dysesthesia are often reported, and are mostly caused by a mechanical irritation of the afferent posterior cervical roots (3 points). The maximum number of points a normal subject can reach is 18.

Borrowing from the Glasgow Coma Scale, the worst result is rated with 1 point for each subscore. The minimum score is therefore 5. Depending on the sum reached in the score, cervical myelopathy is classified into three grades: grade III, 5–8 points; grade II, 9–12 points; and grade I, 13–16 points. Subjects with 17 or 18 points are considered free of signs of cervical myelopathy.

The functional character of the criteria used in the European Myelopathy Score allows a critical evaluation of cervical myelopathy from different centers and different countries. The European Myelopathy Score helps to judge the natural course of the disease and to determine the timing of surgery. It also allows a more objective control of postoperative outcome. The European Myelopathy Score is a valuable tool for the evaluation of all conditions involving cervical myelopathy. It will also allow for rapid communication when comparing radiological findings or neurophysiological results in patients with cervical myelopathy. Assessment of EMS on larger patient population with cervical myelopathy is needed.

Neurophysiological investigation of the cervical spine

Patients with spinal disorders, with or without sensorimotor symptoms and signs, often show discrepancies in clinical and neuroradiological (MRI, CT, myelogram) findings, which make it difficult to pinpoint the cause (i.e., particular nerve root or spinal cord segment) of the patient's complaints. Therefore, questions are raised as to which level or nerve root should be surgically approached.

Currently used electrodiagnostic techniques

The spectrum of neurophysiological assessment consists of electromyography (EMG), electroneurography (ENG), and evoked potentials. While somatosensory evoked potentials (SEPs) and motor evoked potentials (MEPs) are most helpful in the investigation of the central nervous system pathways, electromyography, conventional neurography and F-wave studies are more useful for evaluation of the peripheral segments of the sensory and motor pathways.

Somatosensory evoked potentials

For spinal cord evaluation, SEPs are relevant. These are potentials recorded from the lumbar and cervical spine as well as the first components of scalp recordings.

SEPs are generally recorded after electrical stimulation of peripheral nerves or skin. The nerves used are: the posterior tibial, sural, or common peroneal nerves of the lower limbs, and the median radial and the ulnar nerve for the upper limbs. In radicular and spinal disease, several nerves, supplied by different segments, must be stimulated for a level diagnosis. SEPs from tibialis nerve are recommended for the diagnosis of cervical myelopathy [41].

Motor evoked potentials

Somatosensory evoked potentials are delayed in cervical spondylosis and the latency of N11 is significantly delayed statistically. However, similar data have also been reported previously in electrical cortical stimulation studies [1, 12].

A method of painless magnetoelectric transcranial stimulation of the cerebral cortex was introduced in 1985 by Barker et al. [2, 3]. They applied short magnetic pulses, designed to stimulate peripheral nerves, to the scalp, and recorded muscle action potentials from upper and lower limb muscles.

The stimulating coil is placed in such a way as to stimulate the motor cortex, the cervical nerve roots, and the lumbar nerve roots. MEPs are generally recorded at the following muscles: abductor pollicis, adductor minimi, quadriceps, tibialis anterior, gastrocnemius, extensor hallucis, and abductor hallucis [8]. The segmental innervation of these muscles is used for a level diagnosis in analogy to the segmental distribution of the afferent nerves stimulated for SEPs. Surface recording electrodes are placed over the motor end plate [8].

For motor root stimulation over the cervical and lumbar spine, the intensity of the stimulator is adjusted so that a potential with a steep negative rise can be recorded. With this, the onset latency is not critically dependent on the positioning of the coil or the stimulation strength [6]. The excitation site of the nerve root is most likely in the region of the root exit from the intervertebral foramen, and does not differ from that suggested for electrical stimulation over the spine [6, 7, 23]. In patients diagnosed as having a lateral compression of the nerve root, the peripheral nerve latency is not delayed, whereas in patients with more medially localized herniations, a prolonged central motor latency (CML) is the most frequent finding [5].

M-wave and F-wave evaluation for the interpretation of MEPs

M-wave

In order to judge the MEP waveform it is also necessary to obtain an M-wave recording by means of conventional neurography. The M-wave is the response to a supramaximal stimulus of the peripheral nerve, and therefore an electric measure of muscle “size” [32]. It is used as a reference signal with which post transcranial stimulation MEP amplitude and duration are compared, i.e., MEP amplitude and duration are expressed as ratios of M-wave amplitude and duration respectively.

F-wave

F-wave recordings allow for the determination of a total peripheral conduction time (peripheral latency: PL) from the anterior horn cell to the muscle, which thus includes the conduction over the motor root to its exit from the intervertebral foramen. hnumber =”Sec12”

The F-wave is usually normal in mild cases of radiculopathy. Distinct delay of the F-wave or a reduced number of clearly distinguishable F-waves after a given number of supramaximal peripheral stimuli, in association with normal distal motor conduction, is a sign of a proximal lesion.

However, as the excitability of the spinal motor neuron fluctuates periodically, the appearance, latency, and amplitude of the F-wave changes in each record. In spite of these limitations, F-waves have a diagnostic value for anterior root lesions. When F-waves are recorded in a chronic neuropathic process, axonal reflexes must be differentiated [18, 33].

Electromyography (EMG)

Needle electromyography examines segmentally affected muscles, chosen based upon the clinical investigation. The needle is repositioned on ten different sites in a muscle in order not to miss denervated parts. Increased insertional activity, spontaneous activity (involuntary) such as sharp positive waves, fibrillations, fasciculations and diminished motor unit recruitment are considered signs of denervation due to deterioration of anterior horn cells (myelopathy hands), or due to compression of nerve root.

In normal muscles, motor unit action potentials (MUAPs) are elicited only in response to neural discharges. Denervated muscle fibers become unstable, as they are no longer under neural control, and individual muscle fibers will fire in the absence of neural stimuli. These signs of denervation in EMG can be spotted at the earliest about 8 days after the nerve lesion, and are termed acute signs of denervation.

EMG performed with needle concentric electrodes is the oldest neurophysiologic method for diagnosing nerve root compression syndrome [35]. EMG is claimed to have almost no false-positive results [43].

Diagnostic reliability

EMG is important in the differential diagnosis of cervical spondylosis. It shows degrees of denervation and the number of roots involved, but it has no prognostic value [25].

The increased latency of MEPs is a sensitive sign; however, the specificity is low. The increased CML can be found in not only degenerative but also inflammatory diseases of the central nervous system, such as multiple sclerosis. Kameyama examined 67 patients with clinically relevant cervical myelopathy, and 24 patients with cervical canal stenosis without myelopathy [15]. A positive correlation was found for the group of myelopathy patients. De Mattei found sensitivity of MEPs in patients with cervical compression myelopathy to be 70% for upper extremity muscles, and 95% for lower extremity muscles [10].

Tanaka et al.[37] examined MEPs in patients with clinically relevant cervical myelopathy who underwent decompressive surgery. Patients who presented a CML longer than 15 ms and/or polyphasic wave pattern of the potential had worse surgical results than the remaining patients.

A comparison of EMGs and SEPs in differentiating anterior horn cell disease from cervical spinal myelopathy showed dermatomal SEPs were clearly superior. They were found to be normal, as expected, in all 12 patients with amyotrophic lateral sclerosis, while in 19 out of 20 patients with cervical myelopathy, a pathological finding was observed.

For cervical myelopathy, Vohanka and Dvorak suggest, as a routine examination, SEPs by stimulation of the tibial nerve as well as MEPs from the upper and lower extremities [41].

The value of somatosensory and motor evoked potentials in predicting and monitoring the effect of therapy in spondylotic cervical myelopathy has been prospectively examined by Bednarik et al. [4]. The group changes of some SEP and MEP parameters correlated with the changes in clinical score, which means they could be used as an objective tool for the assessment of the results of therapy. In clinical "silent" cervical cord compression, abnormalities were found in half the subjects (*n*=91) and predicted clinical manifestation of myelopathy in one-third of them during a 2-year period.

Timing for surgery

Guidelines for treatment procedures, either conservative or surgical, in patients with cervical myelopathy do not exist. The literature in this respect presents controversial results. Increasing age, clinical, neurophysiological signs, and the general health condition are relevant factors in the decision-making process.

Sampath et al. [34] presented the results of 62 patients, at less than 1-year follow-up, who were treated for cervical myelopathy by 41 surgeons (members of CSRS), either surgically (*n*=31) or conservatively (*n*=31). Only 43 patients (69%!) were available for follow-up. When medical and surgical treatments are compared, surgically treated patients appeared to have a better outcome. This small, non-randomized study with a large number of surgeons has methodological flaws; the authors acknowledge the fact that randomized studies are necessary to validate the different treatment procedures.

Matsumoto et al. [22] analyzed the increased signal intensity (ISI) in MRI of spinal cord as a predictor for the outcome of conservative treatment in patients with mild myelopathy. Neither ISI nor spinal cord area was significantly associated with outcome. The authors conclude that early decompression for mild cervical myelopathy is not warranted either by ISI or reduced spinal cord area. Yonenobu [44] considers the transverse area of the spinal cord and duration of myelopathy as the most significant prognostic parameters for surgical outcome. Factors that are unchangeable by nature, such as developmental stenosis or progressive degenerative changes of the cervical spine, are parameters to consider or indicate surgical decompression.

A randomized controlled trial (RCT)on patients with mild cervical myelopathy comparing conservative versus surgical results with 3 years' follow-up (*n*=68) did not show surgery to be superior to conservative treatment [14]. The authors of this excellent study, which is the only RCT to have been conducted on cervical myelopathy, are aware of the difficulties and suggest a possible direction to developing this area of investigation:

> "The crucial question in the treatment of mild and moderate nonprogressing SCM is not whether 'to operate or not to operate' because both the conservative and the surgical treatments are potentially useful. The problem is to find the predictive factors for a satisfactory outcome either for the surgical or the nonsurgical approach. It would be desirable to arrange a multicenter study aimed at addressing these questions, as has been mentioned many times. First, however, it would be necessary to validate the scoring systems carefully, probably replacing those currently used to obtain more reliable and reproducible data.
>
> "The current results can serve as a contribution to the theory that conservative treatment has some advantages over surgery in a carefully selected group of patients. The most promising candidates for highly predicted good results from either conservative treatment or surgery could be the transverse area of the stenotic cord, duration of the disease [44], osseous or cartilaginous compression, developmental diameter of the canal, positivity of electrophysiologic findings, low-signal intensity changes on T1-weighted sequences [24], and severity of the neurologic deficit and its dynamics" [14].

The SPINE TANGO of the SSE might be the appropriate platform to search for the answer to this crucial question.

As the indications for surgical decompression of cervical myelopathy are the subject of ongoing discussion, at the authors' institution the following strategy for management of suspected or diagnosed cervical myelopathy has been adopted:

- Obtain a patient's history with respect to the development of symptoms and signs
- Improve awareness of the symptoms of cervical myelopathy among primary care physicians by continuous education
- Conduct a neurological assessment and diagnostic work out to exclude other systemic diseases
- If in doubt, "wait and see", but carry out regular controls
- Neurophysiology, including MEPs/SEPs/EMG, is the most useful way to monitor progression
- Surgery is indicated in progressive and/or severe forms of cervical myelopathy
- Multimodal intraoperative monitoring (MIOM) is required for demanding decompressive surgery, to optimize the surgical procedure

References

1. Abbruzzese G, Dall'Agata D, Morena M, Simonetti S, Spadavecchia L, Severi P, Andrioli GC, Favale E (1988) Electrical stimulation of the motor tracts in cervical spondylosis. J Neurol Neurosurg Psychiatry 51:796–802
2. Barker AT, Freeston IL, Alinous R, Merton PA, Morton HB (1985) Magnetic stimulation of the human brain. J Physiol 369:3
3. Barker AT, Jalinous R, Freeston IL (1985) Non-invasive magnetic stimulation of the human motor cortex. Lancet 1:1106–1107
4. Bednarik J, Kadanka Z, Vohanka S, Novotny O, Surelova D, Filipovicova D, Prokes B (1998) The value of somatosensory and motor evoked potentials in pre-clinical spondylotic cervical cord compression. Eur Spine J 7:493–500
5. Bischoff C, Meyer BU, Machetanz J, Conrad B (1993) The value of magnetic stimulation in the diagnosis of radiculopathies. Muscle Nerve 16:154–161
6. Britton TC, Meyer BU, Herdmann J, Benecke R (1990) Clinical use of the magnetic stimulator in the investigation of peripheral conduction time. Muscle Nerve 13:396–406
7. Cadwell J (1989) Principles of magnetoelectric stimulation. In: Chokroverty S (ed) Magnetic stimulation in clinical neurophysiology. Butterworths, Boston, pp 13–32
8. Chomiak J, Dvorak J, Antinnes J, Sandler A (1995) Motor evoked potentials: appropriate positioning of recording electrodes for diagnosis of spinal disorders. Eur Spine J 4:180–185
9. Crock HV, Yoshizawa H (1977) The blood supply of the vertebral column and spinal cord in man. Springer, Vienna New York
10. De Mattei M, Paschero B, Sciarretta A, Davini O, Cocito D (1993) Usefulness of motor evoked potentials in compressive myelopathy. Electromyogr Clin Neurophysiol 33:205–216
11. Ebara S, Yonenobu K, Fujiwara K, Yamashita K, Ono K (1988) Myelopathy hand characterized by muscle wasting. A different type of myelopathic hand in patients with cervical spondylosis. Spine 13:785–791
12. Esteban GR, Lagranja AR, Lopez MAC (1988) Delayed short-latency somatosensory evoked potentials in premature diagnosis of medullary disturbances in cervical spondylosis. Electromyogr Clin Neurophysiol 28:361–368
13. Herdmann J, Linzbach M, Krzan M, Dvorak J, Bock WJ (1994) The European Myelopathy Score. In: Bauer BL, Brock M, Klinger M (eds) Advances in neurosurgery. Springer, Berlin Heidelberg, pp 266–268
14. Kadanka Z, Mares M, Bednanik J, Smrcka V, Krbec M, Stejskal L, Chaloupka R, Surelova D, Novotny O, Urbanek I, Dusek L (2002) Approaches to spondylotic cervical myelopathy: conservative versus surgical results in a 3-year follow-up study. Spine 27:2205–2210; discussion 2210–2211
15. Kameyama O, Shibano K, Kawakita H, Ogawa R (1995) Transcranial magnetic stimulation of the motor cortex in cervical spondylosis and spinal canal stenosis. Spine 20:1004–1010
16. Deleted in production
17. Kellgren J, Lawrence J (1958) Osteoarthrosis and disc degeneration in an urban population. Ann Rheum Dis 17: 288–397
18. Kimura, J. (1974) F-wave velocity in the central segment of the median and ulnar nerves: a study in normal subjects and patients with Charcot-Marie-Tooth disease. Neurology 24:539–546
19. Lawrence J (1969) Disc degeneration: its frequency and relationship to symptoms. Ann Rheum Dis 28:121–136
20. Lhermitte F, Roullet E, Lyon-Caen O, Metrot J, Villey T, Bach MA, Tournier-Lasserve E, Chabassol E, Rascol A, Clanet M, et al (1986) [Double-blind treatment of 49 cases of chronic multiple sclerosis using hyperbaric oxygen]. Rev Neurol (Paris) 142: 201–206
21. Luschka H (1858) Die Halbgelenke des menschlichen Körpers. Reimers, Berlin
22. Matsumoto M, Toyama Y, Ishikawa M, Chiba K, Suzuki N, Fujimura Y (2000) Increased signal intensity of the spinal cord on magnetic resonance images in cervical compressive myelopathy. Does it predict the outcome of conservative treatment? Spine 25:677–682
23. Mills KR, Murray NMF (1986) Electrical stimulation over the human vertebral column: which neuronal elements are excited? Electroencephalogr Clin Neurophysiol 63:582–589
24. Morio Y, Teshima R, Nagashima H, Nawata K, Yamasaki D, Nanjo Y (2001) Correlation between operative outcomes of cervical compression myelopathy and MRI of the spinal cord. Spine 26:1238–1245
25. Negrin P, Lelli S, Fardin P (1991) Contribution of electromyography to the diagnosis, treatment and prognosis of cervical disc disease: a study of 114 patients. Electromyogr Clin Neurophysiol 31:173–179
26. Neuhuber WL, Zenker W (1989) Central distribution of cervical primary afferents in the rat, with emphasis on proprioceptive projections to vestibular, perihypoglossal, and upper thoracic spinal nuclei. J Comp Neurol 280:231–253
27. Neuhuber WL, Zenker W, Bankoul S (1990) Central projections of cervical primary afferents in the rat. Some general anatomical principles and their functional significance. In: Neuhuber WL, Zenker W (eds) The primary afferent neuron. Plenum, New York, pp 173–188
28. Norré ME (1979) Neck torsion nystagmus and neck motility. J Belg Med Phys 2:30–56
29. Norré ME (1985) Otoneurologischer Beitrag zum Problem des zervikalen Schwindels. In: Gutmann G (ed) Arteria vertebralis. Springer, Berlin Heidelberg New York, pp 123–131
30. Ono K (1987) Myelopathy hand. New clinical signs of cervical cord damage. J Bone Joint Surg Br 69:215–219
31. Penning L, Töndury G (1964) Entstehung, Bau und Funktion der meniskoiden Strukturen in den Halswirbelgelenken. Z Orthop Ihre Grenzgeb 98: 1–14
32. Reiners K, Herdmann J, Freund HJ (1989) Altered mechanisms of muscular force generation in lower motor neuron disease. Muscle Nerve 12:647–659
33. Roth G (1978) Intranervous regeneration of lower motor neuron. II. Study of 1153 motor axon reflexes. Second part: contralateral motor axon reflex crossed facial reinnervation. Electromyogr Clin Neurophysiol 18:311–350
34. Sampath P, Bendebba M, Davis JD, Ducker TB (2000) Outcome of patients treated for cervical myelopathy. A prospective, multicenter study with independent clinical review. Spine 25: 670–676
35. Shea P, Woods W, Werden D (1950) Electromyography in diagnosis of nerve root compression syndrome. Arch Neurol Psychiatry 64:93–104

36. Shimizu T, Shimada H, Shirakura K (1993) Scapulohumeral reflex (Shimizu). Its clinical significance and testing maneuver. Spine 18:2182–2190
37. Tanaka M, Dvorak J (1999) The evaluation of motor evoked potentials (MEPs) by magnetic stimulation in cervial spondylotic myelopathy. Neuroorthopedics 125:75–89
38. Taylor J, Tworney L (1993) Acute injuries to cervical joints: an autopsy study of neck sprain. Spine 18:1115–1122
39. Tepe HJ (1985) Die Häufigkeit osteochondrotischer Röntgenbefunde der Halswirbelsäule bei 400 symptomfreien Erwachsenen. Röfo 6:659–663
40. Töndury G, Theiler K (1990) Entwicklungsgeschichte und Fehlbildungen der Wirbelsäule, 2nd edn. Hippokrates-Verlag, Stuttgart
41. Vohanka S, Dvorak J (1993) Motor and somatosensory evoked potentials in cervical spinal stenosis. Presented at the 40th Congress of the Czech and Slovak Neurophysiology, Brno
42. Wälchli B, Dvorak J (1998) Axial symptoms including cervical migraine and cervical angina. In: Ono K, Dvorak J, Dunn E (eds) Cervical spondylosis and similar disorders. World Scientific, Singapore, New Jersey, London, pp 167–180
43. Wilbourn A, Aminoff M (1988) AAEE Mini Monograph 32. The electrophysiological examination in patients with radiculopathies. Muscle Nerve 11: 1011–1014
44. Yonenobu K (2000) Cervical radiculopathy and myelopathy: when and what can surgery contribute to treatment? Eur Spine J 9:1–7