REVIEW ARTICLE

Degenerative Cervical Spinal Stenosis

Current Strategies in Diagnosis and Treatment

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SUMMARY

Introduction: Cervical spinal stenosis has become more common because of the aging of the population.

There remains much uncertainty about the options for surgical treatment and their indications, particularly in cases of cervical myelopathy.

Methods: In order to provide guidance in clinical decision-making, the authors selectively reviewed the literature, according to the guidelines of the Association of Scientific Medical Societies in Germany.

Results: Cervical myelopathy is a clinical syndrome due to dysfunction of the spinal cord. Its most common cause is spinal cord compression by spondylosis at one or more levels. Its spontaneous clinical course is variable; most patients undergo a slow functional deterioration. Surgical treatment reliably arrests the progression of myelopathy and often even improves the neurological deficits.

Discussion: The available scientific data are too sparse to enable evidence-based treatment of cervical myelopathy. Early surgical intervention is often recommended in the literature. Controversy remains regarding the choice of the appropriate surgical procedure, but there is consensus on the suitable options for many specific clinical situations.

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Key words: cervical spinal stenosis, cervical myelopathy, surgical treatment, anterior decompression, posterior decompression

Diseases of the spinal column are among the most frequent syndromes in modern society and are thought to be caused by ubiquitous degeneration processes, particularly of the intervertebral disks (diskopathy) or of the adjoining vertebral bodies (spondylosis). With increasing age, a large proportion of the population exhibit radiological signs of discopathy or spondylosis, leading to constriction of the spinal canal, usually in the cervical or lumbar spine. Thus, MRI detected cervical lesions affecting the spinal cord of 26% of an asymptomatic group of older patients (1). Because of the development of the age structure of society, the improvements in perioperative medical care, and the higher expectations of our patients – including older patients –, the question of performing surgery is being increasingly raised. Surveys have shown that both cervical and lumbar operations are becoming more common. Thus the frequency of operations on the cervical spine in the USA was as high as 55 per 100 000 in 2000 (2). This means that cervical spinal stenosis is of central importance for neurosurgeons and orthopedic surgeons and an increasingly important syndrome for general physicians, as well as specialists in internal medicine, neurology, and other areas. It has nevertheless often been unclear what the differentiated indications are for surgery, particularly as there are no evidence-based aids to support the decision. In this context, we will provide an overview of current strategies for the diagnosis and treatment of degenerative cervical spinal stenosis.

Methods

This review is based on an extensive and selective literature search, in accordance with the guidelines of the Association of the Scientific Medical Societies in Germany (AWMF).

Pathophysiology

The cervical spinal canal normally provides enough room for the neural elements. The sagittal diameter of the spinal canal varies with height and between individuals. Thus the first cervical vertebral body (C 1) is about 21.8 mm high and the spinal cord makes up about 50% of the spinal canal. On the other hand, C 6 is about 17.8 mm in height and the spinal cord takes up about 75% of the spinal canal (3). Although congenital stenoses are possible, stenoses are usually the secondary consequences of progressive disk degeneration, accompanied by disk protrusion, ventral spondylophyte formation, thickening of the ligamenta flava, and hypertrophy of the dorsal

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facets (*figure 1*). This is reflected in the age distribution of cervical myelopathy, which peaks between the ages of 50 and 60 (4).

The close association between the presence of spinal stenosis and the occurrence of cervical myelopathy has led to the assumption that stenosis is the most important pathophysiological factor in the disease. Nevertheless, this concept is incapable of explaining the spectrum of the disease, particularly myelopathy without stenosis. Spinal stenosis is often accompanied by instability. The spondylotic restriction of the spinal canal results in release and shear forces on the spinal cord. These pathological factors lead to diffuse and focal axonal damage. The diameter of the spinal canal in flexion and extension is reduced. During extension, the ligamentum flavum is folded, which further constricts the spinal canal. Moreover, the changes in length of the spinal canal also affect the length of the spinal cord. For example, the shortening of the spinal cord in extension is linked to an increase in diameter. Particularly if spinal stenosis is present, the spinal cord can be additionally damaged by movement. It is pinched between the pincers of the posteroinferior end of one vertebral body and the lamina or ligamentum flavum of the caudal segment (*figure 1*). These mechanisms not only cause local damage to the spinal cord, but also compress the vessels perfusing it. On the one hand, the anterior spinal artery can be directly compressed. On the other hand, the flattening of the spinal cord can cause torsion in the sulcus vessels, which run transversely. These vessels perfuse the grey matter and the medial white substance, which are typically affected early in the course of the disease (5).

FIGURE 1

Schematic sagittal image of the degenerative changes in the cervical spine. As a result of vertebral disk protrusion (blue), spondylophytes (dark blue) and hypertrophy or folding of the ligamentum flavum (yellow), the spinal canal is restricted, the available space for CSF is reduced (pale blue) and the spinal cord is compressed (green). Slippage in the spine (olisthesis) or instability can also damage to the spinal cord.

Clinical symptoms

The spontaneous clinical course of the disease has still not been adequately studied. The course is highly variable and even spontaneous remissions are possible. However, the literature indicates that most patients' symptoms deteriorate over the years. Deterioration can

TABLE 1

Neurological syndromes with different spinal cord lesions (8)

Syndrome name	Lesion site	Resulting syndrome
Transversal syndrome	Cortico-spinal tract and spinothalamic tract	Gait abnormalities and spasticity of the lower extremities
Motor syndrome	Cortico-spinal tract or anterior horn cells	Exclusively motor disturbance, no sensory deficits
Centromedullary syndrome	Central grey substance of the spinal cord	Weakness of the upper extremities, lower extremities unaffected; possible painful dyesthesia in the hands from anterior horn lesions.
Brown-Sequard syndrome	Monolateral lesion of the spinal cord	Ipsilateral hemiparesis from involvement of the tractus corticospinalis; contralateral anesthesia or thermic anesthesia below the lesion site
Myeloradiculopathy	Combined lesion of the nerve root and the anterior horn cells	Radicular pain combined with lesions of the long tracts
Tandem stenosis	Combined cervical and lumbar spinal stenosis	Neurogenic spinal claudication, complex gait abnormalities, mixed presentation of disturbances of the upper and lower motor neurons
Primary sensory deficits	Posterior cords	Glove shaped distal dyesthesia

Modified from: Crandall D: Cervical spondylotic myelopathy. Journal of Neurosurgery 1966; 25: 57–66, with the kind approval of the Journal of Neurosurgery.

TABLE 2

Diagnosis of cervical myelopathy

Necessary	<ul style="list-style-type: none"> • Medical history, neurological status • MRI of the cervical spine • X-ray diagnosis, including functional images • Neurophysiological diagnostics: SEP • Basic laboratory measurements with inflammation values
Desirable	<ul style="list-style-type: none"> • CSF diagnosis • Residual urine determination
In special cases	<ul style="list-style-type: none"> • Myelography with postmyelo CT • Extended electrophysiological diagnosis, e.g. MEP

SEP, somatosensory evoked potentials; MEP, motor evoked potentials; MRI, magnetic resonance imaging

Figure 2: Sagittal MRI in T2- (left) and T1-weighted imaging. There is stenosis at the level of C 4/5, caused by prolapse of the disk and retrolisthesis. The hyperintense signal in the T2 image (arrow) is a sign of the spinal cord affection. MRI, magnetic resonance imaging; C, cervical vertebral body.

Figure 3: Lateral x-ray of the cervical spine before (left) and after (right) ventral surgery. The retrolisthesis at the level of C 4/5 with dorsal spondylophyte is clearer than in the MRI. After intercorporeal spondylodesis with a titanium cage and plate osteosynthesis, the retrolisthesis is corrected and the spondylophyte resected. MRI, magnetic resonance imaging; C, cervical vertebral body.

occur rapidly and is then mostly irreversible. 75% of patients suffer phases of neurological deterioration (6). There is evidence that about 5% of all patients with asymptomatic spinal cord compression become symptomatic each year (7). There are also patients with an acute clinical course. These are mostly patients with significant but asymptomatic stenosis who suffer acute spinal cord compression after a trivial injury, sometimes leading to high-grade tetraparesis.

As this disease is symptomatic spinal cord compression, it may result in several neurological symptoms, depending on the site of the lesion in the spinal cord (table 1) (8).

The symptoms usually develop slowly. Because of the lack of pain, there may be an interval of years between the onset of disease and first treatment. Early symptoms are mostly abnormal sensations in the hands, abnormal gait – particularly in the dark –, and deficiencies in the fine motor skills of the hands. Disturbances in writing mostly occur at advanced stages. Eventually, the hands are totally incapable of grasping. Spasticity, enhanced reflexes and pyramidal tract symptoms are characteristics of this disease, while muscular fasciculation is regarded as untypical. The disturbances in sensitivity are mostly unrelated to the dermatome. The well-known "Lhermitte sign" does not occur constantly. Patients describe the attacks as a sudden generalized electric shock in the arms and trunk, particularly when the head is bent.

Diagnosis

"Cervical myelopathy" is a clinical diagnosis. The additional apparative investigations are intended to throw light on the cause of the disease. Operative treatment is possible for spinal cord compression from stenosis. A good correlation between the medical history, the neurological findings, and neuroradiological or electrophysiological diagnosis is therefore essential to establish an indication for surgery and for differential diagnosis (table 2). It happens quite frequently that patients with cervical myelopathy are only presented after protracted illness and after operations for carpal tunnel syndrome, or even suspected disseminated encephalomyelitis. Magnetic resonance imaging (MRI) is the method of choice to detect both the stenosis and any damage to the spinal cord (figure 2). In addition, this can be used to exclude other possible conditions, such as spinal tumors. Fluid appears hyperintense in the T2-weighted MRI scan. As a result, changes in the spinal cord can often be seen as signal enhancements. On the other hand, this is rather a non-specific sign, which can also be caused by edema, inflammation or myelomalacia, for example.

Conventional x-ray diagnosis is not of primary importance, although functional images can be helpful, as they demonstrate the extent of any instability (figure 3). Computed tomography (CT) of the cervical spine must be regarded as a complementary method to MRI, when it is necessary to establish whether there are any osseous changes. Conventional myelography as an invasive procedure is only useful in exceptional cases. It should then be combined with CT to give postmyelographic

computed tomography. The specificity of this procedure in differentiating osseous space occupying lesions and soft tissue lesions is then higher than with MRI (9).

Electrophysiological investigations can be useful for follow-up and for establishing segmental differentiation. Both the sensory and the motor evoked potentials are important. During the course of the disease, the compression of the spinal cord increases, leading first to delay in the motor sensory potentials, followed by delay in the motor evoked potentials and finally signal changes in the T2 image of the MRI (10). Electrophysiological diagnosis is not important if the symptoms of myelopathy are clear. However, it can confirm or qualify the indication for surgery for patients with a clear finding of stenosis in imaging studies, but with few clinical symptoms. In doubtful cases, CSF diagnosis should be performed, to exclude inflammatory diseases.

Treatment

The underlying problem in deciding whether surgery or conservative treatment is to be preferred is that there are no reliable parameters which are relevant to the prognosis of cervical myelopathy (11). Although the only randomized comparative study (level of evidence II) confirmed that cervical myelopathy markedly improved, particularly soon after the operation, this advantage subsequently disappeared. There were no differences between the groups with respect to other parameters (12). A cohort study (level of evidence III) failed to find any difference between surgery and conservative treatment (13). Thus, there are not enough prospective randomized studies which provide unambiguous proof of the advantage or disadvantage of surgery. *Table 3* provides an overview of possible therapies.

As mild cervical myelopathy can rapidly lead to persistent deterioration, an operation is favored in these cases. However, it is also acceptable to provide conservative treatment with close clinical monitoring to older patients with mild myelopathy, a relatively wide spinal canal and normal sensory evoked potentials (13, 14).

The results of more recent studies show that the various surgical procedures are capable of preventing the progression of myelopathy and of improving the neurological deficits in a large number of patients. It can be expected that the symptoms will improve in up to 90% of patients (6). The best operative results are expected when the operation is performed within 6 to 12 months of the first mild symptoms and when the transverse area of the spinal canal is greater than 40 mm² (15, 16). Neurological recovery is most marked within the first three months after an operation. Cervical myelopathy is mostly quantified with the scale from the Japanese Orthopedic Association, the JOA scale, ranging from 0 (maximal impairment) to 17 (normal) (*box*) (17). Wada et al. report an impressive postoperative improvement in the JOA, from 7.9 preoperative to 13.9 at five years post-operatively. These authors recorded deterioration in 4% of the operated patients (18). Similar results have been reported by other authors (19). On the other hand, poor results must be expected in patients with advanced

TABLE 3

Therapeutic possibilities for cervical myelopathy

Conservative treatment	
	<ul style="list-style-type: none"> ● Immobilization with a cervical collar ● Drugs: <ul style="list-style-type: none"> – Non-steroidal anti-inflammatories (NSAIDs), – Muscle relaxants ● Intermittent bed rest ● Traction treatment (longitudinal extension of the cervical spine) ● Measures to stabilize the cervicothoracic spine: <ul style="list-style-type: none"> – Strengthening the nuchal musculature – Strengthening the musculature of the upper quadrant – Strengthening the scapula ● Avoidance of activities which stress the cervical spine
Surgery	
Ventral procedures	<ul style="list-style-type: none"> ● Intercorporeal spondylodesis (discectomy + cage +/- plate osteosynthesis) ● Corpectomy + plate osteosynthesis ● Procedures to maintain mobility (intervertebral disk prosthesis)
Dorsal procedures	<ul style="list-style-type: none"> ● Laminectomy ● Laminectomy with fusion ● Laminoplasty
Combined procedures	<ul style="list-style-type: none"> ● Combination of ventral fusion with laminoplasty or laminectomy with or without dorsal stabilization

myelopathy. It must nevertheless be emphasized that the available data were collected in open observational studies and with small groups of patients. Moreover, other authors have failed to confirm these encouraging results (11).

It should be stressed that the risk is increased of suffering irreversible spinal cord damage from a trivial accident, especially for patients with cervical spinal stenosis and Klippel-Feil syndrome (20).

Surgical techniques

The objective of surgery is the decompression of the spinal cord and the neutralization of any instability. To achieve this decompression, the space occupying lesions sketched in *figure 1* must be removed. This can either be achieved ventrally by resection of the vertebral disk protrusion and removal of the spondylophytes or dorsally by removing the ligamentum flava or hypertrophic facets. Instability or spondylolisthesis necessitates stabilizing measures, possibly including osteosynthesis. For this reason, the pathophysiology must be considered when selecting the suitable surgical procedure. The decision whether to perform the decompression dorsally or ventrally depends on many factors: the number of affected segments, the neutral position of the cervical spine, the severity of accompanying neck pain, and the surgeon's confidence in using the different techniques (21). There have been studies comparing ventral corpectomy and dorsal laminoplasty. The long-term results with the two techniques were in principle equivalent, although the initial morbidity was higher with the ventral procedure and laminoplasty gave more neck pain (18). *Table 4* gives an overview of the criteria for selecting a specific surgical procedure.

BOX

Severity of cervical myelopathy based on the classification of the Japanese Orthopedic Association (JOA scale), adapted to European conditions (17)

	Points
<ul style="list-style-type: none"> Ia: Function of the upper extremities Incapable of independent eating with spoon and fork; incapable of buttoning even large buttons 0 Capable of eating with spoon and fork, but clumsy 1 Writing possible, although very clumsy; large buttons can be buttoned 2 Writing somewhat restricted, but possible; cufflinks can be fastened 3 Normal 4 Ib: Shoulder and upper arm (evaluation of grade of strength [1–5] of the M. deltoideus or M. biceps brachii [rating for the weaker muscle]) Above grade of strength 3 –2 Grade of strength 3 –1 Grade of strength 4 –0.5 Grade of strength 5 0 II Function of the lower extremities Incapable of standing up and walking 0 Capable of standing up, but not of walking 0.5 Incapable of walking without a walking aid, even on a smooth surface 1 Capable of walking without support, although gait uncertain 1.5 Capable of walking freely on a smooth surface; needs support for climbing stairs 2 Climbs stairs without support; needs supports when descending stairs 2.5 Can walk rapidly, although gait somewhat uncertain 3 Normal 4 III Sensitivity <ul style="list-style-type: none"> Upper extremity Complete loss of sensitivity to touch or pain 0 Up to 50% loss of sensitivity and/or considerable pain or numbness 0.5 Up to 40% loss of sensitivity and/or moderate pain or numbness 1 Numbness without sensory deficit 1.5 Normal 2 Trunk Complete loss of sensitivity to touch or pain 0 Up to 50% loss of sensitivity and/or considerable pain or numbness 0.5 Up to 40% loss of sensitivity and/or moderate pain or numbness 1 Numbness without sensory deficit 1.5 Normal 2 Lower extremity Complete loss of sensitivity to touch or pain 0 Up to 50% loss of sensitivity and/or considerable pain or numbness 0.5 Up to 40% loss of sensitivity and/or moderate pain or numbness 1 Numbness without sensory deficit 1.5 Normal 2 IV Bladder function Complete retention and/or incontinence 0 Feeling that the bladder has been incompletely voided and/or dripping and/or sparse urine stream and/or partial incontinence 1 Delayed bladder emptying and/or pollakiuria 2 Normal 3 	

Modified from:
Fukui et al.:
Pathomechanism,
pathogenesis, and
results of treatment in
cervical spondylotic
myelopathy caused by
dynamic canal stenosis.
Spine 1990; 15: 1149,
with the kind approval
of Lippincott,
Williams & Wilkins.

Ventral procedures – A ventral procedure is generally recommended when the pathology is mono- or bisegmental, with a predominant ventral space occupying lesion from osteophytes or disk tissue. By clearing out the disk through an anterolateral access, the surgeon reaches the spinal canal or lateral to the neuroforamina through the intervertebral space. Depending on the pathology, soft disk prolapses can be removed or osseous osteophytes can be taken off by high-speed burs. Autologous bones from the iliac crest were used traditionally as strut in the intervertebral space, but have now been largely replaced by titanium or plastic cages (22).

A disadvantage of the ventral procedure is that long stretches of ventral osteophytes may have to be removed, even though the spinal canal is very narrow, and this can lead to intraoperative damage to the spinal cord. Another possibility in these cases is to use corporectomy followed by fusion. In this procedure, the middle section of a vertebral body of the same breadth as the spinal canal is removed in addition to the adjoining vertebral disks and then replaced by implantation of a bone graft or a cage.

Another disadvantage is that the risk of fusion failure markedly increases with the number of segments treated, even if plate osteosynthesis is used. Here too the fusion rate can be increased with corporectomy. The accepted advantage of the ventral procedure is that kyphosis can be reduced or even totally corrected (at least for some patients), which is hardly possible with a dorsal procedure alone.

In patients for whom at least three segments with additional kyphotic bend have to be treated, combined dorsal and ventral procedures are used. This leads to a relatively high fusion rate, unfortunately combined with increased morbidity, as the operation is more extensive (23).

More recently, monosegmental pathologies have been treated with intervertebral disk prostheses, to circumvent the possible problem of later adjacent segment instability or degeneration. It must be said that the postulated advantage of this procedure, especially the advantage of avoiding adjacent level disease, has never been demonstrated. Moreover, there is uncertainty about the indication for cervical disk prostheses. Although instability, kyphotic deformities, and marked segmental degeneration are accepted as contraindications, the actual indications are still controversial. Some authors favor a restricted indication, as they think that a prosthesis will fail to neutralize any dynamic factors (24). Other authors propagate prosthesis implantation even for cervical myelopathy and osteophytic spinal stenosis (25).

Dorsal procedure

Dorsal procedure – The objective of all dorsal procedures is indirect decompression of the spinal cord. After such an operation, the spinal cord is displaced dorsally, so that ventral space occupying lesions can be indirectly treated. Care must however be taken that the dorsal release is over a long stretch, as otherwise this effect does not occur. The use of a dorsal procedure therefore assumes that there is no fixed kyphotic malposition, as

TABLE 4

Selection criteria for a specific surgical procedure

What is the sagittal profile of the cervical spine?	<ul style="list-style-type: none"> Fixed or severe kyphosis ($>10^\circ$); ventral or dorsoventral Normal profile, multisegmental stenosis: most probably dorsal
Is there instability?	<ul style="list-style-type: none"> If yes, non-fusion procedures, such as intervertebral disk prosthesis are excluded
What is the bone quality?	<ul style="list-style-type: none"> With severe osteoporosis and multisegmental stenosis: probably dorsal
How many levels are affected?	<ul style="list-style-type: none"> Mono- or bisegmental: probably ventral Multisegmental: probably dorsal
What sort of stenosis is it?	<ul style="list-style-type: none"> Congenitally narrow spinal canal: probably dorsal Predominantly ventral space-occupying lesion: probably ventral

this cannot or can only inadequately be treated dorsally, since the spinal cord cannot properly move dorsally. Pure laminectomies without additional stabilization are now regarded very critically, as the risk is relatively high of postoperative kyphotic malposition, so-called swan neck deformity. This complication can be effectively prevented by additional stabilization with internal instrumentation.

So-called laminoplasty is often being used in eastern Asia. In this procedure, the vertebral arches are not resected, but split and forced apart. This expands the spinal canal and the individual motion segments are retained. Although this reduces the risk of kyphotic malposition, this procedure leads to markedly increased rates of neck pain. Moreover, although the vertebral motion segments are preserved, the mobility of the cervical spine is usually restricted.

Conflict of interest statement

The authors declare that no conflict of interest exists according to the guidelines of the International Committee of Medical Journal Editors.

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REFERENCES

1. Teresi LM, Lufkin RB, Reicher MA, Moffit BJ, Vinuela FV, Wilson GM et al.: Asymptomatic degenerative disc disease and spondylosis of the cervical spine: MR imaging. *Radiology* 1987; 164: 83–8.
2. Patil PG, Turner DA, Pietrobon R: National trends in surgical procedures for degenerative cervical spine disease: 1990–2000. *Neurosurgery* 2005; 57: 753–8.
3. Lang J: Funktionelle Anatomie der Halswirbelsäule und des benachbarten Nervensystems. In: Hohmann D, Kügelgen B, Liebig K, Schirmer M (eds.): *Neuroorthopädie Band 1*. Berlin, Heidelberg, New York, Tokyo: Springer 1983; 1–118.
4. Chiles BW, Leonard MA, Choudhri HF, Cooper PR: Cervical spondylotic myelopathy: patterns of neurological deficit and recovery after anterior cervical decompression. *Neurosurgery* 1999; 44: 762–9.
5. White AA, Panjabi MM: Biomechanical considerations in the surgical management of cervical spondylotic myelopathy. *Spine* 1988; 13: 856–60.

6. Edwards CC, Riew KD, Anderson PA, Hilibrand AS, Vaccaro AF: Cervical myelopathy. Current diagnostic and treatment strategies. *Spine J* 2003; 3: 68–81.
7. Bednarik J, Kadanka Z, Dusek L, Novotny O, Surelova D, Urbanek I et al.: Presymptomatic spondylotic cervical cord compression. *Spine* 2004; 29: 2260–9.
8. Crandall PH, Batzdorf U: Cervical spondylotic myelopathy. *J Neurosurg* 1966; 25: 57–66.
9. Baron EM, Young WF: Cervical spondylotic myelopathy: a brief review of its pathophysiology, clinical course, and diagnosis. *Neurosurgery* 2007; 60: 35–41.
10. Kanchiku T, Taguchi T, Kaneko K, Fuchigami Y, Yonemura H, Kawai S: A correlation between magnetic resonance imaging and electrophysiological findings in cervical spondylotic myelopathy. *Spine* 2001; 26: 294–9.
11. Kadanka Z, Mares M, Bednarik J, Smrcka V, Krbec M, Stejskal L et al.: Approaches to spondylotic cervical myelopathy: conservative versus surgical results in a 3-year follow-up study. *Spine* 2002; 27: 2205–10.
12. Sampath P, Bendebba M, Davis JD, Ducker TB: Outcome of patients treated for cervical myelopathy. A prospective, multicenter study with independent clinical review. *Spine* 2000; 25: 670–6.
13. Kadanka Z, Mares M, Bednarik J, Smrcka V, Krbec M, Chaloupka R et al.: Predictive factors for mild forms of spondylotic cervical myelopathy treated conservatively or surgically. *Eur J Neurol* 2005; 12: 16–24.
14. Mazanec D, Reddy A: Medical management of cervical spondylosis. *Neurosurgery* 2007; 60: 43–50.
15. Lesoin F, Bouasakao N, Clarisse J, Rousseaux M, Jomin M: Results of surgical treatment of radiculomyelopathy caused by cervical arthrosis based on 1000 operations. *Surg Neurol* 1985; 23: 350–5.
16. Kadanka Z, Mares M, Bednarik J, Smrcka V, Krbec M, Chaloupka R et al.: Predictive factors for spondylotic cervical myelopathy treated conservatively or surgically. *Eur J Neurol* 2005; 12: 55–63.
17. Fukui K, Kataoka O, Shio T, Sumi M: Pathomechanism, pathogenesis, and results of treatment in cervical spondylotic myelopathy caused by dynamic canal stenosis. *Spine* 1990; 15: 1148–52.
18. Wada E, Suzuki S, Kanazawa A, Matsuoka T, Miyamoto S, Yonenobu K: Subtotal corpectomy versus laminoplasty for multilevel cervical spondylotic myelopathy: a long-term follow-up study over 10 years. *Spine* 2001; 26: 1443–7.
19. Sevik K, Mehmet T, Ufuk T, Azmi H, Mercan S, Erkal B: Results of surgical treatment for degenerative cervical myelopathy: anterior cervical corpectomy and stabilization. *Spine* 2004; 29: 2493–500.
20. Bailes JE: Experience with cervical stenosis and temporary paralysis in athletes. *J Neurosurg Spine* 2005; 2: 11–6.
21. Witwer BP, Trost GR: Cervical spondylosis: ventral or dorsal surgery. *Neurosurgery* 2007; 60: 130–6.
22. Thome C, Leheta O, Krauss JK, Zevgaridis D: A prospective randomized comparison of rectangular titanium cage fusion and iliac crest autograft fusion in patients undergoing anterior cervical discectomy. *J Neurosurg Spine* 2006; 4: 1–9.
23. Douglas AF, Cooper PR: Cervical corpectomy and strut grafting. *Neurosurgery* 2007; 60: 137–42.
24. Nabhan A, Ahlhelm F, Shariat K et al.: The ProDisc-C prosthesis: clinical and radiological experience 1 year after surgery. *Spine* 2007; 32: 1935–41.
25. Mummaneni PV, Burkus JK, Haid RW, Traynelis VC, Zdeblick TA: Clinical and radiographic analysis of cervical disc arthroplasty compared with allograft fusion: a randomized controlled clinical trial. *J Neurosurg Spine* 2007; 6: 198–209.

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