

Kazuo Yonenobu

## Cervical radiculopathy and myelopathy: when and what can surgery contribute to treatment?

Received: 9 October 1999  
Accepted: 22 October 1999

K. Yonenobu  
Department of Orthopedic Surgery,  
Osaka University Postgraduate  
Medical School, 2–2 Yamadaoka,  
Suita, 565–0871 Osaka, Japan  
e-mail: yonenobu@ort.med.osaka-u.ac.jp,  
Tel.: +81-6-6879 3551,  
Fax: +81-6-6879 3559

**Abstract** Indications and timing of surgical treatment for cervical radiculopathy and myelopathy, and the long-term results for the conditions, were reviewed. Advances in spinal imaging and accumulation of clinical experience have provided some clues as to indications and timing of surgery for cervical myelopathy. Duration of myelopathy prior to surgery and the transverse area of the spinal cord at the maximum compression level were the most significant prognostic parameters for surgical outcome. Thus, when myelopathy is caused by etiological factors that are either unchangeable by nature, such as developmental canal stenosis, or progressive, such as ossification of the posterior longitudinal ligament, surgical treatment should be considered. When an etiology of

myelopathy is remissible, such as soft disc herniation and listhesis, surgery may be reserved until the effects of conservative treatment are confirmed. When surgery is properly carried out, long-term surgical results are expected to be good and stable, and the natural course of myelopathy secondary to cervical spondylosis may be modified. However, little attention has been paid to the questions “When and what can surgery contribute to treatment of cervical radiculopathy?”. A well-controlled clinical study including natural history should be done to provide some answers.

**Key words** Review · Cervical spondylosis · Treatment outcome · Spinal cord · Spinal nerve root

### Introduction

Cervical spondylosis is one of the most common disorders that ails people over middle-age. Problems brought on by this condition have become more serious in societies where the aged population is expanding. Since Key described encroachments on the spinal cord by ventral ridges of discal origin in 1838 [49], the condition has been an object of study in neurological, neurosurgical, orthopedic, radiological, and other related fields, and have been analyzed from various points of view. With regard to surgical treatment, strenuous efforts made by many surgeons, probably driven by disastrous experiences, resulted in development and improvement of various surgical tech-

niques, and results of surgical treatment have become better and more stable. Various methods of conservative treatment for radiculopathy and myelopathy have also been developed, and choice of treatment for these conditions has become diverse.

However, one of the most important questions, “when and what can surgery contribute to treatment of cervical radiculopathy and myelopathy?” remains unanswered. Namely, when must we change treatment modality for the conditions from conservative to surgical? Can a surgery selected for the patient eradicate their complaints completely, and can the surgery change the natural course of the disease distressing the patient? In this article, indications, timing and results of surgical treatment for cervical radiculopathy and myelopathy are

reviewed, based on etiological, pathological and follow-up studies.

### **Causes of cervical radiculopathy and myelopathy**

The clinical manifestations of cervical spondylosis are quite diverse [5], and the pathomechanisms of some symptoms, such as precordial pain, headache and vertigo, have not yet been clarified. Signs and symptoms observed in a patient with cervical spondylosis can be categorized as core, marginal and inexplicable; the core symptoms are those of discopathy, radiculopathy and myelopathy, according to the origin of the symptoms [37].

A typical radicular syndrome consists of radiating pain and sensory and/or motor deficit localized in an area innervated by a certain nerve root. It is caused by nerve root irritation and/or compression by spondyloarthropathic osteophytes. Radiculopathy is also caused by soft disc herniation, and rarely by spinal and spinal cord tumors, ossification of the posterior longitudinal ligament (OPLL) [22], infectious spondylitis and trauma.

Spinal cord symptoms caused by cervical spondylosis are divided into two groups: long-tract and segmental symptoms. Typical neurological signs of long-tract involvement are exaggerated tendon reflexes, presence of pathological reflexes, spastic quadriplegia, spastic type of myelopathy hand [36], sensory loss with glove and stocking like distribution, and bladder-bowel disturbance. Schematically, segmental sign is motor deficit of an affected segment. However, limited involvement of the gray matter may not manifest clinically. When involvement extends multisegmentally, segmental symptoms appear as cervical spondylotic amyotrophy (CSA) [37]. Myelopathy is also caused by soft disc herniation, OPLL, and rarely by spinal and spinal cord tumors, infectious spondylitis and miscellaneous conditions.

The clinical appearance of cervical spondylotic myelopathy (CSM) is a combination of symptoms of segmental and long-tract impairment. Segmental symptoms mimic radicular symptoms, and thus are sometimes regarded as radicular in origin, and erroneously diagnosed as radiculomyelopathy. In Japan we have many patients with CSM [23], probably due to developmental spinal canal size [31]. Experientially, most patients with CSM have only a combination of long-tract and segmental signs. Naturally, the pathomechanisms of cervical spondylotic radiculopathy and myelopathy are quite different, and thus these conditions should be discussed separately.

### **Cervical myelopathy**

Can we throw some light on the questions of when and what surgery can contribute to treatment from a viewpoint of etiology? Cervical myelopathy is caused by static com-

pression, directly or indirectly, probably through circulatory disturbance. Dynamic factors including normal cervical movement and abnormal movement, so-called spinal instability, also cause myelopathy in association with static compression and/or developmental canal stenosis. Therefore, these etiological factors influence the natural course of cervical myelopathy and thus affect choice of treatment and timing of surgery.

When myelopathy is caused by factors of a progressive nature, such as spinal and spinal cord tumors, surgical treatment is indicated immediately the diagnosis is made. OPLL is a self-limiting condition, but an ossified lesion increases to a certain size and does not decrease in size spontaneously. Thus, surgery must be considered regardless of symptoms, when the occupancy rate (thickness of lesion/developmental anteroposterior canal diameter) is more than 40%, or the space available for the spinal cord is less than 8 mm, because development of myelopathy is strongly anticipated in this situation [27]. A minor trauma may transform the natural course of OPLL. The Japanese investigation committee on OPLL reported that 20.6% of patients with OPLL experienced acute deterioration of neurological symptoms on occasion of trivial trauma such as slipping [46]. This suggests that in OPLL surgical treatment must be considered even for a patient with mild symptoms.

Cervical spondylosis itself is a self-limiting condition. Osteophytes formed at the discovertebral junction, the uncovertebral joint (Luschka), and the zygoapophyseal joint never grow beyond a certain size. However, static compression is not the sole etiological factor of myelopathy in cervical spondylosis, and the etiologically multifactorial origin of CSM makes it difficult to predict the natural course of any particular patient with the condition.

Soft disc herniation is common in the cervical spine as well as in the lumbar spine, and causes cervical radiculopathy or myelopathy. Recently, spontaneous regression of the herniated mass in the lumbar spine has been documented with development of magnetic resonance imaging [45]. Herniated mass seems to be the only static compression factor that can disappear spontaneously. However, regarding regression of herniated disc in the cervical spine, case reports have sporadically been published [44, 48], and only a few reported on the spontaneous regression of the herniated mass based on a follow-up study [20, 52]. No factor predicting spontaneous regression of the herniated mass can be clarified on the basis of these reports, and it is difficult to establish timing of surgery in the reported cases.

Dynamic factors are also an important etiological factor in CSM and OPLL. The dynamic factors include normal neck movement and abnormal movement such as spondylolisthesis. Both normal and abnormal movements are changeable and can therefore be controlled to some degree; thus, immobilization of the spine can be a remedy of conservative treatment. Barnes and Saunders studied

factors predicting long-term prognosis in CSM based on a retrospective study of patients treated conservatively, and reported that those who deteriorated were more likely to have significantly more cervical mobility when compared to those patients whose disability had remained static. They concluded that measurement of cervical mobility may help to select patients who are more likely to deteriorate and thus more likely to benefit from surgical intervention [3]. Spinal instability such as listhesis may remain at its present degree due to the restabilization mechanism. However, the natural course of instability of the spine in a particular patient remains unpredictable, and our knowledge of this subject is insufficient to determine indication for surgical treatment.

The developmental spinal canal size is a predisposing factor of CSM, and is generally believed to remain fixed after maturation. In actual fact, the spinal canal size becomes smaller with age. Hayashi and his co-authors reported a decrease of static and dynamic anteroposterior canal diameter with age [13]. Thus, surgical treatment should be considered if a myelopathy is associated with developmental spinal canal stenosis.

Some factors mentioned above suggest progression of myelopathy, and can be used as a parameter of decision-making in treatment of CSM. However, in most patients with CSM, the etiology is multifactorial and it is very difficult to tell which factor is most responsible for myelopathy in any particular instance, although efforts have been made to clarify the pathophysiology of the condition [4]. To answer the questions posed in the title, experimental studies should be conducted, including development of animal models with chronic spinal cord compression and clinical investigations using open magnetic resonance imaging (MRI) for study of dynamic factors [8, 29, 30] and positron emission tomography for study of functional imaging [2].

Do studies of the natural history of CSM throw any light on the questions? Several authors have reported on the natural history of CSM [3, 7, 26, 32, 42]. However, most studies lacked inclusion criteria, evaluation of severity of myelopathy was unclear and follow-up periods vary. Therefore, the information on the natural history of CSM from the literature is not sufficient to formulate a precise prognosis, as LaRocca commented in his review of the natural history of CSM [25]. Ideally, prospective studies with sufficient data regarding prognostic factors are indispensable to forecasting the natural history more accurately in any particular patient. However, it is ethically doubtful that this type of prospective study can be carried out, because, with the advances in surgical treatment, delay in surgical intervention has been reported to result in a poor outcome [10, 24, 50].

Does analysis of surgical results provide any answers? Various factors have been reported to influence surgical results: surgical technique, duration of symptoms prior to surgery, severity of myelopathy before surgery, age at sur-

gery, trauma, multiplicity of involvement, anteroposterior canal diameter, transverse area of the spinal cord, high-signal-intensity area on T2-weighted image, etc. The most common and significant factor for prognostication is duration of symptoms prior to surgery [10, 24, 50]. Koyanagi and his co-authors showed that the transverse area of the spinal cord at the maximum compression before surgery and duration of symptoms prior to surgery were the most significant predictors of surgical results, based on multiple regression analysis [24]. Fujiwara and his co-authors reported that when the transverse area of the cord at the maximum compression before surgery was less than 30 mm<sup>2</sup>, surgical results were poor [10]. These findings suggest that delay in surgical treatment for severe myelopathy is associated with poor prognosis. However, these factors are not relevant to decision-making in mild myelopathy. The significance of other factors for prognostication of the outcome of surgical treatment varies across reports. Recently, a high-signal area on T2-weighted MR images has been studied as factor prophesying surgical outcome [1, 6, 28, 34, 47]. However, conclusions were not in agreement; some said the high-signal area was correlated with outcome [6, 34] and others said no [28, 47]. Wada and his co-authors concluded that the high-signal area reflected cystic change in the gray matter, after comparing their findings of MRI and delayed CT-myelography (CTM) with findings of pathological study [33]. They reported that only when the high-signal area extended multisegmentally did its presence indicate poor surgical outcome [47].

Pathological study of the spinal cord in CSM and OPLL helps explain the correlation between clinical manifestations and findings of neuroimaging such as CTM and MRI. Several clinicopathological studies have already been reported elsewhere [12, 18, 35].

In summary, a myelopathic patient experiencing difficulties in the activities of daily life would be a candidate for surgical treatment irrespective of etiology. A patient with myelopathy secondary to soft disc herniation can be treated conservatively, expecting spontaneous regression of the mass, if their symptoms are mild. Indication and timing of surgical treatment for a patient with CSM with mild symptoms is still obscure. When myelopathy is secondary to OPLL and developmental spinal canal stenosis, the risks and benefits of surgery should be assessed, because spontaneous decrease of the size of the lesion cannot be expected.

What, then, can be expected of surgery? Regarding the results of surgical treatment, numerous reports have been published using various evaluation criteria. In Japan, the scoring system for cervical myelopathy proposed by the Japanese Orthopedic Association has been widely used since 1975 [21]. The system consists of three categories, motor function, sensory function and bladder function, and each function is evaluated according to a number of items. Roughly speaking, approximately 60% of recovery,

**Table 1** The efficacy and disadvantages of various surgical procedures in cervical myelopathy (? indicates insufficient data)

Surgical procedure	Anterior approach		Posterior approach	
	Discectomy and interbody fusion	Corpectomy and fusion	Laminectomy	Laminoplasty
Indication and contraindication				
No. of levels involved	2	2	Multiple	Multiple
Developmental canal stenosis	No	Yes?	Yes	Yes
Spinal instability	Yes	Yes	No	Yes?
Kyphotic deformity	Yes	Yes	No	Yes?
Posterior compression	No	Yes?	Yes	Yes
Morbidity				
Neural tissue injury	+	+	+	+
Nerve root tethering	?	+	?	+
Decrease of neck mobility	Less	Less	More	More
Postoperative neck pain	None?	None?	+?	+
Pseudoarthrosis	More	Less	None	None
Donor site pain	+	+	None	None
Postoperative kyphosis	None	Less	More	More?
Long-term results				
Spondylosis adjacent to fused segment	+	+	None	None
Miscellaneous	Brace	Brace		

according to the recovery rate of Hirabayashi [17], can be anticipated, irrespective of surgical technique. Several parameters mentioned above can be predictors of surgical outcome.

So which procedure produces the best outcome? Ideally, surgical procedures for compression myelopathy must be tailored to the individual patient. On the other hand, the efficacy and disadvantages of each procedure, as summarized in Table 1, need to be taken into account in reaching a decision. A lively discussion in this field is choice of surgical procedure for CSM with multiple-level involvement and a relatively narrow spinal canal. Several surgical procedures, including discectomy and fusion, subtotal corpectomy and fusion, laminectomy and laminoplasties have been applied for this condition. However, no single procedure has been proved to be superior to others on statistical evidence. Only a few comparative studies have been conducted [14, 51]. A prospective, multicentric, controlled trial may answer the question [41].

The last question in this section is “Can surgery change the natural course of disease distressing the patient?” Long-term follow-up studies may give some clues to the answer. In Japan, the health insurance system allows us to follow a patient up for a long postoperative period, and symposia on long-term follow-up results of surgery for cervical compression myelopathy were held at the 1999 annual meeting of the Japanese Spine Research Society. More than 20 papers on this subject, with minimum follow-up of 10 years, were presented, and conclusions were as follows. Generally, surgical benefits were maintained but deterioration of activities developed gradually with age, and frequent causes of the deterioration in the mus-

culoskeletal system were disorders related to aging, such as lumbar spondylosis and osteoarthritis of the knee. A few patients showed regression of symptoms due to spondylosis at levels adjacent to fusion. As to symptomatic adjacent-segment disease, In a follow-up of 374 patients using Kaplan-Meier survivorship analysis, Hilibrand and co-authors found that 25.6% of patients who had an anterior cervical arthrodesis developed new disease at an adjacent level within 10 years after the surgery [16]. At the moment, conclusions cannot be drawn on whether surgery can change the natural course of the disease, because we have no data on the “true” natural history of cervical spondylosis. However, surgical benefits of a satisfactory level are generally maintained for a long postoperative period.

### Cervical radiculopathy

Similarly to CSM, static compression and dynamic factors are believed to cause CSR. However, CSR rarely transforms to CSM [26]. CSR is not usually associated with spinal canal stenosis [38]. Osteophytes come from the discovertebral junction and the uncovertebral joint (Luschka), and the zygoapophyseal joint, which forms the neuroforamen, is visible with roentgenography. Humphreys and co-authors reported that foraminal heights, widths, and areas were larger in asymptomatic patients than in symptomatic patients [19]. However the incidence of radiculopathy does not increase with age, while the incidence of roentgenographical spondylotic changes does increase with age. Dynamic factors may be etiological to



CSR, because immobilization of the cervical spine can cure symptoms. An abnormal movement, such as listhesis, can be visualized clinically to be a possible etiological factor, while with a normal movement, it is hard to tell its etiological significance. It is difficult to throw any light on the questions in the title from the standpoint of etiology..

In the lumbar region, it is argued that chemicals from and/or induced by herniated mass play an important role in development of radiculopathy, and the same mechanism may occur in the cervical region. Inflammation caused by mechanical and/or chemical factors serves as a major component of the disease process of CSR. Thus, anti-inflammatory therapies can be a remedy of CSR, and choice of treatment for the condition is more varied than for CSM.

The natural history of CSR has been investigated mostly as a part of follow-up study of conservative treatment, because conservative treatment is generally believed to alleviate symptoms of CSR in the short term. However over a long period it has no effect on the natural history. Only a few reports taking an epidemiological approach have been published [40]. Thus, authors have proposed a variety of outcomes of this condition, probably due to different inclusion criteria for patients, different evaluation criteria for symptoms and differences in duration of follow-up. An optimistic result was 90% remission, while a pessimistic result indicated one-third suffered persistent symptoms [11, 40]. At the same time, no parameter has yet been clarified to predict prognosis. Thus, a stepwise procedure of choice of remedies for the condition is unavoidable in most patients with CSR. The indications of surgery for CSR are (1) failure of conservative treatment and (2) progressive neurologic deficit. As for period of conservative treatment, a trial of at least 3 months is recommended [15].

Treatment for CSR consists of conservative and surgical therapy. The former is composed of rest, immobilization of the cervical spine and various physical therapies, including traction and medical treatment. The latter consists of anterior surgery (discectomy with or without fusion, foraminotomy) and posterior surgery (facetectomy, foraminotomy). Only a few studies aimed directly at establishing choice and timing of treatment have been conducted so far [39, 43]. Persson and co-authors reported results of three therapies, surgery, collar immobilization and physiotherapy, based on a prospective study, and at 16 months after treatment no difference was found. Results of surgical treatment vary, and, as in CSM, no single procedure has been proved with statistical significance to be better than others.

Finally, what symptoms can be cured by surgery? Symptoms of radiculopathy can be categorized into core and marginal symptoms, the latter including Barre-Lieou syndrome, blurred vision, tinnitus, cervical migraine, cervical angina, etc [37]. At same time, a patient with CSR is sometimes associated with axial pain. Radicular pain is usually alleviated better than axial pain [9, 53]. The core symptoms of radiculopathy are likely to be more curable by surgery than marginal symptoms, although results of surgery with special reference to the marginal symptoms have been rarely reported.

Accumulation of experience on treatment of cervical radiculopathy and myelopathy has provided some clues as to when and what surgery can contribute. However, clear answers to the question, backed by statistical evidence, have not been found. A prospective, well-controlled study, especially on radiculopathy, which is more common and is treated in a variety of ways, would help to shorten the suffering period of patient and cut the medical expenses of society.

## References

1. Al-Mefty O, Harkey LH, Middleton TH, Smith RR, Fox JL (1988) Myelopathic cervical spondylotic lesions demonstrated by magnetic resonance imaging. *J Neurosurg* 68:217-222
2. Baba H, Uchida K, Sadato N, Yonekura Y, Kamoto Y, Maezawa Y, Furusawa N, Abe Y (1999) Potential usefulness of  $^{18}\text{F}$ -2-Fluoro-Deoxy-D-Glucose positron emission tomography in cervical compressive myelopathy. *Spine* 24:1449-1454
3. Barnes MP, Saunders M (1984) The effect of cervical mobility on the natural history of cervical spondylotic myelopathy. *J Neurol Neurosurg Psychiatry* 47:17-20
4. Bleasel K, Connelley TJ, Dan NG (1973) Cervical spondylotic myelopathy. The use of cine-radiography to select certain cases for surgery. *Proc Aust Assoc Neurol* 9:213-218
5. Brain WR, Northfield D, Wilkinson M (1952) The neurological manifestations of cervical spondylosis. *Brain* 75:187-225
6. Bucciero A, Vizioli L, Carangelo B, Tedeschi G (1993) MR signal enhancement in cervical spondylotic myelopathy. Correlation with surgical results in 35 cases. *J Neurosurg Sci* 37:217-222
7. Clarke E, Robinson PK (1956) Cervical myelopathy: a complication of cervical spondylosis. *Brain* 79:483-510
8. Condon BR, Hadley DM (1988) Quantification of cord deformation and dynamics during flexion and extension of the cervical spine using MR imaging. *J Comput Assist Tomogr* 12:947-955
9. Dillin W, Booth R, Cuckler J, Balderson R, Simeone F, Rothman R (1985) Cervical radiculopathy. A review. *Spine* 11:988-991
10. Fujiwara K, Yonenobu K, Ebara S, Yamashita K, Ono K (1989) The prognosis of surgery for cervical compression myelopathy. An analysis of the factors involved. *J Bone Joint Surg Br* 71:393-398
11. Gore DR, Sepic SB, Gardner GM, Murray MP (1987) Neck pain. A long-term follow-up of 205 patients. *Spine* 12:1-5
12. Hashizume Y, Kameyama T, Mizuno J, Nakagawa H, Yanagi T, Yoshida M (1997) Pathology of spinal cord lesions caused by ossification of the posterior longitudinal ligament. In: Yonenobu K, Sakou T, Ono K (eds) *Ossification of the posterior longitudinal ligament*. Springer, Berlin Heidelberg New York, pp 59-64

13. Hayashi H, Okada K, Hashimoto J, Tada K, Ueno R (1988) Cervical spondylotic myelopathy in the aged patient. A radiographic evaluation of the aging changes in the cervical spine and etiologic factors of myelopathy. *Spine* 13:618–625
14. Herkowitz HN (1988) A comparison of anterior cervical fusion, cervical laminectomy, and cervical laminoplasty for the surgical management of multiple level spondylotic radiculopathy. *Spine* 13:774–779
15. Herkowitz HN (1989) The surgical management of cervical spondylotic radiculopathy and myelopathy. *Clin Orthop* 239:94–108
16. Hilibrand AS, Carlson GD, Palumbo MA, Jones PK, Bohlman (1999) Radiculopathy and myelopathy at segments adjacent to the site of a previous anterior cervical arthrodesis. *J Bone Joint Surg Am* 81:519–528
17. Hirabayashi K, Miyakawa J, Satomi K, Maruyama T, Wakano K (1981) Operative results and postoperative progression of ossification among patients with ossification of cervical posterior longitudinal ligament. *Spine* 6:354–364
18. Hiroshima K, Ono K, Fujiwara K (1998) Pathology of cervical spondylosis, spondylotic myelopathy, and similar disorders—Is clinicopathological correlation verified? In: Ono K, Dvorak J, Dunn E (eds) *Cervical spondylosis and similar disorders*. World Scientific, Singapore, pp 89–139
19. Humphreys SC, Hodges SD, Patwardhan A, Eck JC, Covington LA, Sartori M (1998) The natural history of the cervical foramen in symptomatic and asymptomatic individuals aged 20–60 years as measured by magnetic resonance imaging. A descriptive approach. *Spine* 23:2180–2184
20. Iwasaki M, Ebara S, Miyamoto S, Wada E, Yonenobu K (1996) Expansive laminoplasty for cervical radiculomyelopathy due to soft disc hernia. A comparative study between laminoplasty and anterior arthrodesis. *Spine* 21:32–38
21. Japanese Orthopaedic Association (1994) Scoring system (17–2) for cervical myelopathy. *J Jpn Orthop Assoc* 68:490–503
22. Kawai S (1997) Clinical manifestation of cervical ossification of the posterior longitudinal ligament. In: Yonenobu K, Sakou T, Ono K (eds) *Ossification of the posterior longitudinal ligament*. Springer, Berlin Heidelberg New York, pp 81–84
23. Kokubun S, Sato T, Ishii Y, Tanaka Y (1996) Cervical myelopathy in Japan. *Clin Orthop* 323:129–138
24. Koyanagi T, Hirabayashi K, Satomi K, Toyama Y, Fujimura Y (1993) Predictability of operative results of cervical compression myelopathy based on preoperative computed tomographic myelography. *Spine* 14:1958–1963
25. LaRocca H (1988) Cervical spondylotic myelopathy: natural history. *Spine* 13:854–855
26. Lee F, Turner JWA (1963) Natural history and prognosis of cervical spondylosis. *BMJ* 2:1607–1610
27. Miyasaka H (1975) The consideration on pathophysiology of ossification of the posterior longitudinal ligament (in Japanese). *Rinsho Seikeigeka* 10:1091–1096
28. Morio Y, Yamamoto K, Kuranobu K, Murata M, Tada K (1994) Does increased signal intensity of the spinal cord on MR images due to cervical myelopathy predict prognosis? *Arch Orthop Trauma Surg* 113:254–259
29. Muhle C, Metzner J, Weinert D, Falliner A, Brinkmann G, Mehdorn HM, Heller M, Resnick (1998) Classification system based on kinematic MR imaging in cervical spondylotic myelopathy. *Am J Neuroradiol* 19:1763–1771
30. Muhle C, Metzner J, Weinert D, Schon R, Rautenber E, Falliner A, Brinkmann G, Mehdorn HM, Heller M, Resnick D (1999) Kinematic MR imaging in surgical management of cervical disc disease, spondylosis and spondylotic myelopathy. *Acta Radiol* 40:146–153
31. Murone I (1974) The importance of the sagittal diameters of the cervical spinal canal in relation to spondylosis and myelopathy. *J Bone Joint Surg Br* 56:30–36
32. Nurick S (1972) The natural history and the results of surgical treatment of the spinal cord disorder associated with cervical spondylosis. *Brain* 95:101–108
33. Ogino H, Tada K, Okada K, Yonenobu K, Yamamoto T, Ono K, Namiki H (1983) Canal diameter, anteroposterior compression ratio, and spondylotic myelopathy of the cervical spine. *Spine* 8:1–15
34. Okada Y, Ikata T, Yamada H, Sakamoto R, Katoh S (1993) Magnetic resonance imaging study on the results for surgery for cervical compression myelopathy. *Spine* 18:2024–2029
35. Ono K, Ota H, Tada K, Hamada H, Takaoka K (1977) Ossified posterior longitudinal ligament: a clinicopathological study. *Spine* 2:123–138
36. Ono K, Ebara S, Fuji T, Yonenobu K, Fujiwara K, Yamashita K (1987) Myelopathy hand. New clinical signs of cervical cord damage. *J Bone Joint Surg Br* 69:215–219
37. Ono K, Ohwada T, Ohkohchi T, Ebara S (1998) Radicular symptoms, myelopathic symptoms, including spastic and amyotrophic hand. In: Ono K, Dvorak J, Dunn E (eds) *Cervical spondylosis and similar disorders*. World Scientific, Singapore, pp 181–222
38. Payne EE (1957) The cervical spine and spondylosis. *Neurochirurgia* 1:178–196
39. Persson LC, Moritz U, Brandt L, Carlsson CA (1997) Cervical radiculopathy: pain, muscle weakness and sensory loss in patients with cervical radiculopathy treated with surgery, physiotherapy or cervical collar. A prospective, controlled study. *Eur Spine J* 6:256–266
40. Radhakrishnan K, Litchy WJ, O'Fallon WM, Kurland LT (1994) Epidemiology of cervical radiculopathy. A population-based study from Rochester, Minnesota, 1976 through 1990. *Brain* 117:325–335
41. Rowland LP (1992) Surgical treatment of cervical spondylotic myelopathy: time for a controlled trial. *Neurology* 42:5–13
42. Sadasivan KK, Reddy RP, Albright JA (1993) The natural history of cervical spondylotic myelopathy. *Yale J Biol Med* 66:235–242
43. Sampath P, Bendebba M, Davis JD, Ducker T (1999) Outcome in patients with cervical radiculopathy. Prospective, multicenter study with independent clinical review. *Spine* 24:591–597
44. Song J, Park H, Shin K (1999) Spontaneous regression of a herniated cervical disc in a patient with myelopathy. *J Neurosurg* 90:138–140
45. Teplick JG, Haskin ME (1985) Spontaneous regression of herniated nucleus pulposus. *AJR* 145: 371–375
46. Tsuyama N, Terayama K, Ohtani K, Yamauchi Y, Yamaura I, Kurokawa T, Kaneda K, Harata S, Inoue S, Kirita Y, Ono K, Kataoka O, Ikata T, Sako T, Hattori S, Tsuzuki N, Hirabayashi K, Sasaki T, Yanagi T, Tominaga S, Tezuka A, Nagai Y (1981) The ossification of the posterior longitudinal ligament of the spine. *J Jpn Orthop Assoc* 55:425–440
47. Wada E, Yonenobu K, Suzuki S, Kanazawa A, Ochi T (1999) Can intramedullary signal change on magnetic resonance imaging predict surgical outcome in cervical spondylotic myelopathy? *Spine* 24:455–461
48. Westmark RM, Westmark KD, Sonntag VKH (1997) Disappearing cervical disc. Case report. *J Neurosurg* 86:289–290

- 
49. Wilkinson M (1967) Historical introduction. In: Lord Brain, Wilkinson M, (eds) *Cervical spondylosis*. W.B. Saunders, Philadelphia, pp 1–9
  50. Yonenobu K, Fuji T, Ono K, Okada K, Yamamoto T, Harada N (1985) Choice of surgical treatment for multisegmental cervical spondylotic myelopathy. *Spine* 10:710–716
  51. Yonenobu K, Hosono N, Iwasaki M, Asano M, Ono K (1992) Laminoplasty vs. subtotal corpectomy—a comparative study of surgical results in multisegmental cervical spondylotic myelopathy. *Spine* 17:1281–1284
  52. Yoshida M, Tamaki T, Kawakami M, Hayashi N, Ando M (1998) Indication and clinical results of laminoplasty for cervical myelopathy caused by disc herniation with developmental canal stenosis. *Spine* 23:2391–2397
  53. Yu YL, Huan CY (1987) Cervical spondylotic myelopathy and radiculopathy. *Acta Neurol Scand* 75:367–373