Table 1 Demographic data

	Total (n = 135)	A+ (n = 110)	A− (n = 25)	P
Males/females	76/59	61/49	15/10	0.68*
Age (years)	62.7 ± 9.8	61.9 ± 9.7	66.1 ± 9.8	0.05†
Preoperative Frankel				0.92*
A, B or C (non-ambulatory)	93 (68.9%)	76 (69.1%)	17 (68.0%)	
A	1 (0.7%)	0 (0%)	1 (4.0%)	
B	9 (6.7%)	8 (7.3%)	1 (4.0%)	
C	83 (61.5%)	68 (61.8%)	15 (60.0%)	
D	42 (31.1%)	34 (30.9%)	8 (32.0%)	
Operated spine				0.19*
Cervical	18 (13.3%)	15 (13.6%)	3 (12.0%)	
Thoracic	89 (65.9%)	69 (62.7%)	20 (80.0%)	
Lumbar	28 (20.7%)	26 (23.6%)	2 (8.0%)	
Operation time (min)	286 ± 73	289 ± 72	272 ± 79	0.16‡
Estimated blood loss (mL)	872 ± 789	813 ± 757	1128 ± 890	0.06‡
ERT				0.50*
+	94 (69.6%)	78 (70.9%)	16 (64.0%)	
−	41 (30.4%)	32 (29.1%)	9 (36.0%)	

A+ group: those with dural compression from anterior or circumferential lesion

A− group: those with dural compression from posterior and/or lateral lesion

ERT external radiation therapy (preoperative or postoperative)

* Chi square test, † Student's t test, ‡ Mann-Whitney's U test

Table 2 Comparison of postoperative neurological outcomes between the two groups

	Total (n = 135)	A+ (n = 110)	A− (n = 25)	P
Postoperative Frankel				0.47
A, B or C (non-ambulatory)	10 (7.4%)	9 (8.2%)	1 (4.0%)	
A	1 (0.7%)	0 (0%)	1 (4.0%)	
B	0 (0%)	0 (0%)	0 (0%)	
C	9 (6.7%)	9 (8.2%)	0 (0%)	
D or E (ambulatory)	125 (92.6%)	101 (91.8%)	24 (96.0%)	
D	88 (65.2%)	72 (65.5%)	16 (64.0%)	
E (full recovery)	37 (27.4%)	29 (26.4%)	8 (32.0%)	
Gait regain in preoperatively non-ambulatory cases	83/93 (89.2%)	67/76 (88.2%)	16/17 (94.1%)	0.47
Cervical	11/11 (100%)	11/11 (100%)		
Thoracic	61/70 (87.1%)	47/55 (85.5%)	14/15 (93.3%)	
Upper thoracic (T1-4)	13/13 (100%)	9/9 (100%)	4/4 (100%)	
Middle thoracic (T5-8)	27/34 (79.4%)	22/28 (78.6%)	5/6 (83.3%)	
Lower thoracic (T9-12)	21/23 (91.3%)	16/18 (88.9%)	5/5 (100%)	
Lumbar	11/12 (91.7%)	9/10 (90.0%)	2/2 (100%)	
Full recovery in preoperatively ambulatory cases	21/42 (50.0%)	17/34 (50.0%)	4/8 (50.0%)	1.00
Recovery of ≥1 Frankel's grade in all cases	108 (80.0%)	88 (80.0%)	20 (80.0%)	1.00

the rate of surgeries in the middle thoracic spine (T5-8) was the lowest (78.6%) and significantly lower than that of surgeries in all other spinal levels (93.8%, $P = 0.048$).

Discussion

There are two major findings in the present study. First, most paralytic spinal metastases were located anterior to

the spinal canal, and anterior dural sac compression was present in approximately 80% of cases. Second, the neurological recovery after the posterior decompression surgery for anterior lesions treated indirectly was similarly effective compared with direct decompression for posterior and/or lateral lesions, when associated with IORT.

It is believed that the vertebral body is involved in most spinal metastases, but the accurate prevalence of anatomical location has not been established in the literature. Asdourian [10] reported that metastases are located in the vertebral body in 100%, and in the vertebral body and pedicles in 55.6% of cases. Our result is very close to this finding. In the present study, whereas most patients had anterior metastatic lesions, the primary dural compression was not necessarily confined to the anterior location. In fact, approximately one-fifth of the patients with anterior lesions were actually spared from the anterior compression. This discrepancy may be explained by the hypothesis that the posterior longitudinal ligament functions as a strong barrier against the tumoral invasion into the spinal canal [11].

Several authors reported good outcomes with anterior surgery for spinal metastasis [12–15]. The overall neurological recovery rate ranges from approximately 70–100% and our result was comparable to the preceding reports. The rate of the group with indirect decompression was favorable and was not different from that of the group with direct decompression. This finding implies that anterior direct decompression is not always necessary. In the light of our results, more patients than recognized in the progressed stage should be the candidates for indirect decompression surgery via a less invasive posterior approach. This philosophy is similar to the treatment of burst fracture [4, 5]. However, unlike osteoporotic fracture, late progression of spinal metastasis, which causes recurrent dural compression and spinal instability, is problematic and the local control of anterior lesion must be strictly guaranteed. In our institution, we have used IORT for this purpose. The effectiveness of posterior decompression surgery with IORT has been reported with good long-term results [9]. Tumors that react well to the adjuvant therapy including external radiation therapy or chemotherapy can potentially be targeted in other institutions.

We compared recovery results according to morphology of the involved area to clarify the reasons why indirect decompression is usually satisfactory for spinal metastasis and to identify the specific conditions in which indirect decompression should be regarded as suboptimal. The recovery rate in the A+ group was diminished in middle thoracic spine, which has the strongest local kyphosis. This rate was, however, almost 80% and the posterior approach should always be considered as an important option for many patients. In general, the result of posterior surgery for anterior compression due to spondylosis or ossification of posterior longitudinal ligament is worse with local kyphosis [16, 17], because posterior spinal cord shift is not fully achieved by laminectomy and decompression is always indirect. The acceptable result in metastatic spinal surgery can be possibly explained by the fact that the compressive lesion in the metastasis tends to be relatively soft as compared to a bony spur or ossified ligament. Unlike these lesions, soft tumoral compression does not function as a sharp fulcrum to compress the spinal cord and this difference in stiffness may make indirect decompression satisfactory. However, in some cases, spinal metastases result in the pathological vertebral collapse, and paralysis is caused by the extrusion of a destructed posterior wall [18]. In these cases, the anterior compressive lesion partly contains osseous tissue arising from posterior wall disruption and thus becomes harder, and that might be the reason for the lower rate in the kyphotic spinal levels.

There are some limitations in this study. First, this is a retrospective study and we did not directly compare the result of anterior direct decompression for anterior compression and that of posterior indirect decompression. Other potentially important confounders including the primary tumor and comorbidities could not be adjusted either. The postoperative results that are specific to the sort of primary tumor have not been established. Further studies are warranted to clarify them. Second, we only included the cases for which decompression surgery was indicated in the analysis of the anatomical metastases and dural compression locations. This selection bias may have slightly changed the distribution of locations by excluding the patients with small metastases without any symptoms or those who were referred to us too late with the more widespread invasions in which surgery was not indicated.

In conclusion, most spinal metastases cause paralysis by anterior dural compression, and posterior decompression surgery has been proven to be effective and acceptable when local control of anterior lesion is guaranteed. Surgeons should reconsider the indication of posterior surgery for the patients in whom adjuvant therapy is proven to be effective enough to control the local recurrence, for which IORT is one of the best options.

Conflict of interest The authors declare that they have no conflict of interest.

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Prevalence of symptomatic lumbar spinal stenosis and its association with physical performance in a population-based cohort in Japan: the Wakayama Spine Study

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SUMMARY

Objective: The purpose of this study was to investigate the prevalence of symptomatic lumbar spinal stenosis (LSS) and to clarify the association between symptomatic LSS and physical performance using magnetic resonance imaging (MRI) in a population-based cohort.
Design: This cross-sectional study was performed as a part of the Research on Osteoarthritis/osteoporosis Against Disability (ROAD) in Japan and 1,009 subjects (335 men, 674 women, mean age 66.3 years, age range 21–97 years) were analyzed. An experienced orthopedic surgeon obtained the medical history and performed the physical testing for all participants. Symptomatic LSS diagnostic criteria required the presence of both symptoms and radiographic LSS findings. A 6-m walking time, chair standing time, and one-leg standing time were obtained from all participants.
Results: The prevalence of symptomatic LSS was 9.3% (95% confidence interval [CI]: 7.7–11.3) overall, 10.1% (CI: 7.4–13.8) in men and 8.9% (CI: 7.0–11.3) in women. There was a difference in the prevalence with increasing age by gender. The LSS prevalence showed little difference with age greater than 70 years for men, but the LSS prevalence for women was higher with increasing age. Among physical performance measures, 6-m walking time at a maximal pace was significantly associated with symptomatic LSS ($P = 0.03$).
Conclusion: The prevalence of symptomatic LSS was approximately 10% in a cohort resembling the general Japanese population. A 6-m walking time at a maximal pace was a more sensitive index than walking at a usual pace in assessing decreased physical performance associated with symptomatic LSS.

Introduction

Symptomatic lumbar spinal stenosis (LSS) is usually associated with impaired walking and other disabilities in the elderly. Symptomatic LSS has been shown to be the most frequent indication for spinal surgery in patients more than 65 years old[1,2]. However, little is known about the prevalence of symptomatic LSS in the general population. This is because the subjects in previous symptomatic LSS studies were limited to patients who visited the hospital[3,4]. Hence, people with minor symptomatic LSS who did not visit the hospital were not included in those studies. Furthermore, an examination that can capture minute changes of the intervertebral discs and ligaments using a tool like magnetic resonance imaging (MRI) is essential for the diagnosis of symptomatic LSS. This is because the definition of stenosis includes a morphological element. Many previous studies have reported the utility of MRI[5,6], but, to our knowledge, there have been no population-based cohort studies of symptomatic LSS using MRI.

It is well-known that the principal symptoms for LSS are sciatica and intermittent claudication (IC)[1,2]. Although most patients with MRI evidence of radiographic LSS are asymptomatic[7,8], when symptoms are present, severe symptoms are probably associated with poor physical performance. There have been few reports concerning physical performance of patients with symptomatic LSS[9,10]. According to a previous report concerning walking ability of

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subjects with three different degenerative musculoskeletal disorders (knee osteoarthritis, hip osteoarthritis, and symptomatic LSS) who were scheduled for either joint replacement or spinal decompression surgery, walking ability was limited in all three groups compared to healthy controls[9]. However, patients with symptomatic LSS showed the greatest restrictions in walking ability. In another report regarding subjects with symptomatic LSS in an orthopedic clinical practice, subjects in the healthy group showed greater functional mobility than those in the symptomatic LSS group[10]. The subjects included in the previous studies had enough symptoms to have visited the hospital, however, the association of physical performance measures with symptomatic LSS in subjects with minor symptoms who do not visit the hospital has not been well characterized. Although there may be a latent diminished physical functioning in symptomatic LSS with even minor radiographic changes and symptoms, there have been no population-based studies on symptomatic LSS that have included people with minor signs and symptoms of LSS.

Symptomatic LSS in this study was diagnosed by the presence of both clinical symptoms and radiographic LSS findings consistent with the clinical presentation. The aim of the present study was to clarify the prevalence of symptomatic LSS by gender and age strata using a population-based cohort. In addition, the association of symptomatic LSS with physical performance measures (walking speed, chair standing time, and one-leg standing time) was evaluated.

Methods

Participants

The present study, entitled "the Wakayama Spine Study: population-based cohort", was a population-based study for degenerative spinal disease and performed in a subcohort of the large-scale population-based cohort study called Research on Osteoarthritis/osteoporosis Against Disability (ROAD). ROAD is a nationwide, prospective study of bone and joint diseases consisting of population-based cohorts established in several communities in Japan. As a detailed profile of the ROAD study has already been described elsewhere, only a brief summary is provided here[11–14]. To date, creation of a baseline database including clinical and genetic information for 3,040 inhabitants (1,061 men, 1,979 women) in the age range of 23–95 years (mean, 70.6 years) has been completed. Participants were recruited from listings of resident registrations in three communities: an urban region in Itabashi, Tokyo; a mountainous region in Hidakagawa, Wakayama; and a coastal region in Taiji, Wakayama. All participants provided written informed consent, and the study was conducted with the approval of ethical committees of the University of Tokyo and the Tokyo Metropolitan Institute of Gerontology. Participants completed an interviewer-administered questionnaire of 400 items that included lifestyle information, underwent anthropometric measurements, and physical performance measures were recorded. A second visit of the ROAD study to the mountainous region of Hidakagawa and the seacoast region of Taiji was performed between 2008 and 2010. From inhabitants participating in the second visit of the ROAD study, 1,063 volunteers were recruited to undergo MRI examinations. Fifty-two of the 1,063 volunteers declined the MRI examination, therefore, 1,011 were registered in the present study. All participants provided another written informed consent for the MRI examination. Among those 1,011 participants, two participants with LSS symptoms for whom MRI was contraindicated (due to presence of a pacemaker) were excluded, because a final diagnosis of symptomatic LSS could not be made (Fig. 1). Thus, 1,009 participants (335 men and 674 women, mean age 66.3 years, age range of 21–97 years) were analyzed in the present study. Similar to the baseline study, participants in the second visit of the ROAD study completed an interviewer-administered questionnaire of 400 items that included lifestyle information such as smoking habits, alcohol consumption, family history, past history, physical activity, reproductive variables, and health-related quality of life (QOL). Anthropometric measurements included height, weight, bilateral grip strength, and body mass index (BMI) (weight [kg]/height^2 [m^2]). The ankle-brachial index (ABI) was measured using PWV/ABI (OMRON Co., Kyoto, Japan) for all participants. A timed 6-m walk at the participant's usual pace in a hallway was recorded to measure physical performance. Similarly, 6-m walking time at a maximal pace was measured[15–18]. The time taken for five consecutive chair rises without the use of hands was also recorded[18–20]. One-leg standing time with each leg was measured using a stopwatch (upper limit, 60 s) and the time adopted was the mean value of both legs[21,22].

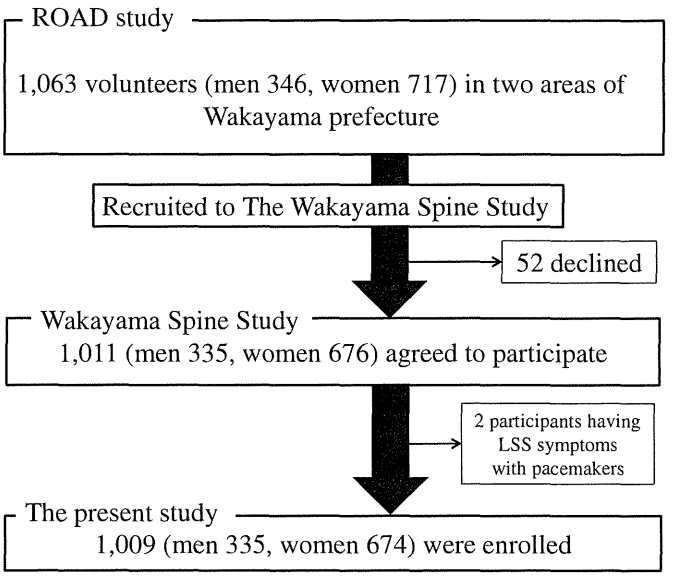

Fig. 1. Flow diagram depicting participants recruited to the Wakayama Spine Study from the ROAD study.

MRI

A mobile MRI (Excelart 1.5 T, Toshiba, Tokyo, Japan) unit was used in the present study, and total spinal MRI was performed for all participants on the same day as the examination. MRI exclusion criteria included presence of a cardiac pacemaker, claustrophobia, or other contraindications. The participants were positioned in supine during the MRI, and those with rounded backs used triangular pillows under their head and knees. The imaging protocol included sagittal T2-weighted fast spin echo (FSE) (repetition time (TR): 4,000 ms/echo, echo time (TE): 120 ms, field of view (FOV): 300 × 320 mm), and axial T2-weighted FSE (TR: 4,000 ms/echo, TE: 120 ms, FOV: 180 × 180 mm). Sagittal images were taken for the entire spine, but axial images were done at each lumbar intervertebral level (L1/2–L5/S1) parallel to the vertebral endplates.

Symptomatic LSS diagnosis

An experienced orthopedic surgeon (YI) consistently took the medical history and performed the physical testing for all the participants in this study. The history included information on the

presence of low back, buttock and leg pain, the area of pain or other discomfort, the presence of IC and its distance, and a modified Zurich Claudication Questionnaire[23] (excepting six items about satisfaction and a history of lumbar surgery for symptomatic LSS). Physical examinations included symptoms induced by lumbar extension, symptoms improved or induced with lumbar flexion, floor finger distance (cm), peripheral circulation (good or poor), a straight leg raising test, manual muscle testing of both upper and lower extremities, tendon reflex testing for both upper and lower extremities, and Babinski reflex testing. In addition, the MRI study of the entire spine was performed on all participants on the same day as the physical examination.

The diagnostic criteria for symptomatic LSS used in the present study were based on the LSS definition from the North American Spine Society (NASS) guideline, which requires presentation of both LSS symptoms and radiographic signs of LSS[24]. The orthopedic surgeon (YI) made the diagnosis of symptomatic LSS using this definition. The diagnosis for LSS symptoms required one or more of the following symptoms: pain, numbness and neurological deficits in the lower extremities and buttocks, and bladder/bowel dysfunction. The symptom characteristics should be induced or exacerbated with walking or prolonged standing and relieved with lumbar flexion, sitting and recumbency. The severity of radiographic LSS was assessed by qualitative measurements, which were performed by a well-experienced orthopedic surgeon (YI) and images were provided on films. The features assessed for LSS included severity of central, lateral recess, and foraminal stenosis, rated as four grades: none, mild, moderate and severe. The lateral recess was defined, as per Fardon and Millette[25], as extending from the medial edge of the facet to the edge of the neural foramen. We applied the general guideline classification of a[26] mild stenosis as narrowing of the normal area by one-third or less, moderate stenosis as narrowing between one-third and two-thirds, and severe stenosis as narrowing of more than two-thirds. Central and lateral recess stenosis was rated on the axial images and foraminal stenosis on the sagittal images. We used the most severe side for the rating of lateral and foraminal stenosis at each level. The same observer scored 50 randomly selected lumbar MRI films more than 1 month after the first reading to evaluate the intraobserver variability of the severity rating. Two experienced orthopedic surgeons also scored 50 different lumbar MRI films (YI & KN) for interobserver variability. The intraobserver variability was confirmed by a kappa analysis which dichotomized radiographic LSS severity as no/mild stenosis vs moderate/severe stenosis, and showed sufficient reliability for assessment of central, lateral and foraminal stenosis (0.77, 0.70 and 0.65, respectively). Interobserver variability was also sufficient for assessment using the kappa analysis (0.71, 0.65 and 0.65, respectively).

Radiographic LSS also required the severity to be more than moderate and the radiographic finding needs to be consistent with the symptoms as outlined above. An experienced orthopedic surgeon (YI) made the final diagnosis of symptomatic LSS using this definition, which requires presentation of both LSS symptoms and radiographic LSS findings. There were no participants with LSS symptoms due to tumor, inflammatory, or traumatic pathologies.

Statistical analysis

All statistical analyses were performed using JMP version 8 (SAS Institute Japan, Tokyo, Japan). Differences in age, height, weight, BMI, 6-m walking time at a usual pace, 6-m walking time at a maximal pace, chair standing time, and one-leg standing time between men and women were examined by the non-paired Student's *t*-test. The non-paired Student's *t*-test was also used to compare age between participants with and without symptomatic LSS. The prevalence of symptomatic LSS was also compared between men and women by the chi-square test. Differences in physical performance measures (6-m walking time at a usual pace, 6-m walking time at a maximal pace, chair standing time, and one-leg standing time) between participants with and without symptomatic LSS were examined by the non-paired Student's *t*-test. Furthermore, logistic regression analysis was used to estimate the odds ratios (ORs) of physical performance measures (6-m walking time at a usual pace, 6-m walking time at a maximal pace, chair standing time, and one-leg standing time) for symptomatic LSS after adjustment for age, gender and BMI.

Results

Table I shows the characteristics of 1,009 participants (335 men and 674 women, mean age 66.3 years, age range of 21–97 years) including age, anthropometric measurements, and physical performance in the present study. Two-thirds of the 1,009 participants were women. Mean age was not significantly different between men and women. BMI was significantly lower in women than in men ($P = 0.005$). Physical performance measures of the 6-m walking time at a usual pace and at a maximal pace were significantly shorter in men than in women ($P < 0.05$ for both), while chair standing time and one-leg standing time were not significantly different between men and women.

The prevalence of radiographic LSS findings was much greater than the prevalence of symptomatic LSS for the participants in this study. The percentage of participants with moderate or severe radiographic central stenosis was 76.5% (95% confidence interval [CI]: 73.7–79.0) in total, while the prevalence of symptomatic LSS was 9.3% (95% CI: 7.7–11.3) in total, 10.1% (CI: 7.4–13.8) in men, and 8.9% (CI: 7.0–11.3) in women. There was no significant difference between men and women ($P = 0.52$). The prevalence in men less than 39 years, 40–49, 50–59, 60–69, 70–79, and 80 years and older was 0%, 3.8% (CI: 0.7–18.9), 9.8% (CI: 4.6–19.8), 11.8% (CI: 6.1–21.5), 11.7% (CI: 6.7–19.8), and 10.7% (CI: 5.6–19.7), respectively, while that in women was 0%, 1.4% (CI: 0.2–7.3), 5.7% (CI: 2.8–11.3), 9.3% (5.7–14.8), 11.9% (CI: 7.9–17.5), and 13.3% (CI: 8.4–20.6), respectively (Fig. 2). The prevalence of both genders

Table I
Characteristics of participants

	Total	Men	Women	*P* value for gender
No. of participants	1009	335	674	
Age group (years)				
≦39	30	11	19	–
40–49	100	26	74	–
50–59	184	61	123	–
60–69	229	68	161	–
70–79	271	94	177	–
≧80	195	75	120	–
Demographic characteristics				
Age, years	66.3 ± 13.6	67.3 ± 13.8	65.9 ± 13.4	0.11
Height, cm	155.9 ± 9.4	164.5 ± 7.1	151.6 ± 7.2	<0.0001
Weight, kg	56.8 ± 11.5	64.4 ± 11.7	53.1 ± 9.4	<0.0001
BMI, kg/m^2	23.3 ± 3.6	23.7 ± 3.5	23.1 ± 3.6	0.005
Physical performance				
Six-meter walking time at a usual pace, s	5.7 ± 2.2	5.5 ± 1.5	5.8 ± 2.4	0.04
Six-meter walking time at a maximal pace, s	3.9 ± 1.4	3.6 ± 1.1	4.0 ± 1.6	<0.0001
Chair standing time, s	8.9 ± 4.0	8.8 ± 3.4	8.9 ± 4.2	0.61
One-leg standing time, s	36.0 ± 23.7	35.7 ± 24.0	36.1 ± 23.6	0.82

Non-paired *t*-test was used to determine differences in demographic characteristics and measurements of physical performance between men and women. Values are the means ± standard deviation.

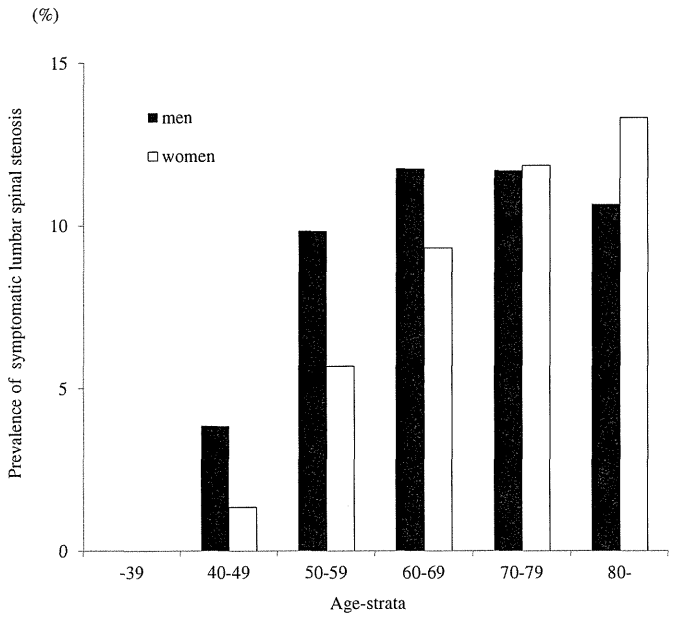

Fig. 2. Prevalence of symptomatic LSS classified by age and gender among 1,009 participants from a community cohort in Japan.

increased until reaching the 60–69 year old age group in which the prevalence in men was higher than that of women. However, the prevalence for women was higher than that of men after age 70. The prevalence of symptomatic LSS in men demonstrated little difference between age groups 60–69 years to over 80 years, but the prevalence for women became significantly higher with increasing aging ($P = 0.036$).

Fifty-five (58.5%) of 94 participants defined as having symptomatic LSS had IC. Five of these 55 participants presented with an ABI < 0.9. However, these five participants also had symptomatic LSS and their leg symptoms were positionally dependent. In this study, there were fifty neurogenic IC cases. There were five cases of unspecified IC, which was caused by both neurogenic and vascular claudication.

Table II shows the physical performance measures in participants with and without symptomatic LSS. In the overall population, 6-m walking time at a usual pace, 6-m walking time at a maximal pace, chair standing time, and one-leg standing time were significantly worse in participants with symptomatic LSS than those without symptomatic LSS ($P < 0.01$ for all). When analyzed in men and women separately, the results were similar to those overall, although the significant differences disappeared in some physical performance measures in men. The significant differences of 6-m walking time at a usual pace in both genders and one-leg standing time in men disappeared after a Bonferroni adjustment. Logistic regression analysis after adjustment for age, gender and BMI showed that 6-m walking time at a maximal pace was significantly associated with symptomatic LSS (OR: 1.17, 95% CI: 1.01–1.34). The physical performance measures of 6-m walking time at a usual pace, chair standing time, and one-leg standing time were not significantly associated with symptomatic LSS (OR: 1.04, 95% CI: 0.94–1.13, OR: 1.03, 95% CI: 0.97–1.09 and OR: 1.00, 95% CI: 0.98–1.01, respectively).

Discussion

The present study is the first to clarify the prevalence of symptomatic LSS by gender and age strata and the association of symptomatic LSS with physical performance measures using a population-based cohort. The prevalence of symptomatic LSS was found to be 9.3% in the general Japanese population, 10.1% in men, 8.9% in women, and there were no significant differences between genders. Interestingly, although the prevalence in women was higher with increasing age, the prevalence in men was the highest at 60–69 years, and little difference in prevalence was seen in men aged 60–69 years to 80 years or older. The prevalence of radiographic LSS was much greater than the prevalence of symptomatic LSS, with only a small proportion of participants with radiographic LSS actually showing symptoms suggestive of the clinical syndrome. The 6-m walking time at a maximal pace was significantly associated with symptomatic LSS, while the 6-m walking time at a usual pace was not.

We have identified no previous studies of symptomatic LSS prevalence. Johnsson[4] reported that the incidence of symptomatic LSS was 50/million person-years in southern Sweden in a study of patients who consulted the orthopedic department in two cities. However, as the author of that report described, the incidence of symptomatic LSS could be underestimated, because the studies did not include patients with minor symptoms who did not visit the hospital. This study is the first to clarify the prevalence of symptomatic LSS using a population-based cohort study.

Reported differences in prevalence of symptomatic LSS between men and women are mixed[27–29]. Verbiest reported a preponderance of symptomatic LSS in men as compared to women among his patients diagnosed by clinical symptoms and myelography[28]. However, Getty reported an equal gender distribution of symptomatic LSS prevalence in a series in which subjects were treated surgically for symptomatic LSS[29]. It is important to note that the subjects in those studies were patients who visited hospitals. In the present study, differences in the prevalence of symptomatic LSS between men and women in the general population were clarified. The prevalence of symptomatic LSS in men was slightly higher than in women, but there was no significant difference between genders. There was a difference in distribution of symptomatic LSS between men and women. The prevalence in women was higher with increasing age, but that in men was the highest at 60–69 years and

Table II
Measurements of each physical performance in participants with and without symptomatic LSS

	Total			Men			Women		
	LSS	Non-LSS	*P* value	LSS	Non-LSS	*P* value	LSS	Non-LSS	*P* value
Number of participants	94	915		34	301		60	614	
Physical performance									
Six-meter walking time at a usual pace, s	6.3 ± 2.7	5.6 ± 2.1	0.003	6.0 ± 1.6	5.4 ± 1.5	0.03	6.5 ± 3.1	5.7 ± 2.3	0.02
Six-meter walking time at a maximal pace, s	4.5 ± 2.1	3.8 ± 1.3	<0.0001	3.9 ± 1.1	3.6 ± 1.1	0.09	4.8 ± 2.4	3.9 ± 1.5	<0.0001
Chair standing time, s	10.1 ± 4.0	8.8 ± 3.9	0.002	9.7 ± 2.8	8.7 ± 3.4	0.10	10.3 ± 4.6	8.8 ± 4.1	0.008
One-leg standing time, s	27.9 ± 23.5	36.8 ± 23.6	0.0005	27.7 ± 25.4	36.7 ± 23.7	0.04	28.0 ± 22.6	36.9 ± 23.5	0.006

Values are the means ± standard deviation.
Non-paired *t*-test was used to determine differences in measurements of physical performance between LSS and non-LSS.

little different in men aged 60–69 years to 80 years and older. The prevalence of lumbar spondylosis (LS) diagnosed as Kellgren/Lawrence (KL) grade two or greater (defined as osteophyte formation with and without disc space narrowing) was found to be significantly higher in men than in women[30]. The prevalence of LS in women was found to be higher with increasing age, while that in men found little difference over 60 years[13]. Interestingly, these distribution patterns are similar to the prevalence of symptomatic LSS in the present study. Anatomical LSS arises from degenerative LS, and facet osteoarthritis and/or hypertrophy, which is associated with narrowing of the space available for the neural elements[1]. This may be one reason for the similarity between LS and symptomatic LSS prevalence.

The present study was the first to show that, among the general population, 6-m walking time at a maximal pace was significantly associated with symptomatic LSS, while 6-m walking time at a usual pace was not. This may mean that participants with symptomatic LSS appeared to have no disadvantage concerning activities of daily living compared to those without symptomatic LSS. However, when requiring greater functional reserve, such as 6-m walking time at a maximal pace, differences between participants with and without symptomatic LSS appeared. This is also the first study to indicate that tasks requiring greater functional reserve, such as walking at a maximal speed, could be a more sensitive index in assessment of decreased physical performance due to symptomatic LSS.

There are several limitations in the present study. First, although the present study included more than 1,000 participants, these participants may not represent the general population as they were recruited from only two areas. However, anthropometric measurements were compared between participants and the general Japanese population, and no significant differences were found in BMI (men: 23.71 (3.41) and 23.95 (2.64), $P = 0.33$, women: 23.06 (3.42) and 23.50 (3.69), $P = 0.07$)[31]. In addition, the proportion of current smokers and current drinkers (those who regularly smoked or drank more than one drink/month) in the general Japanese population was compared with that in the study population. Proportions of current smokers and drinkers in men and that of current drinkers in women were significantly higher in the general Japanese population than in the study population, but there were no significant differences in that of current smokers in women (smokers: men, 32.6% in the Japanese population, 25.2% in study participants, $P = 0.015$; women, 4.9% in the Japanese population, 4.1% in study participants, $P = 0.50$; drinkers: men, 73.9% in the Japanese population, 56.8% in study participants, $P < 0.0001$; women, 28.1% in the Japanese population, 18.8% in study participants, $P < 0.0001$), suggesting that it is likely that the participants (both men and women) had healthier lifestyles than the general Japanese population. Second, this is a cross-sectional study, so any causal relationship between symptomatic LSS and physical performance cannot be clarified. The Wakayama Spine Study is a longitudinal survey, so further progress will help to elucidate any causal relationships. Thirds, total walking distance/duration was not measured, and this metric for walking would likely have been of greater relevance to symptomatic LSS than speed of walking. In addition, this study only represents the Japanese population, hence, prevalence in other countries may be quite different.

In conclusion, the present study clarified that the prevalence of symptomatic LSS was about 10% in a cohort resembling the Japanese general population. There was a difference in the prevalence of symptomatic LSS distribution by age strata between men and women. The 6-m walking time at a maximal pace was a more sensitive index for assessing decreased physical performance due to LSS than the 6-m walking time at a usual pace. Further longitudinal surveys of the Wakayama Spine Study will help to further clarify the incidence and risk factors for symptomatic LSS.

Author contributions

All authors worked collectively to develop the protocols and methods described in this paper. YI, SM, KN, NO, HO, TA, and NY were principal investigators responsible for the fieldwork in the Wakayama Spine Study. YI and SM performed the statistical analysis. YI, HY, SM, KN, HH, HO, TA, MY, and NY contributed to the analysis and interpretation of results. YI wrote the report. All authors read and approved the final report.

Role of the funding source

The study sponsors played no role in the study design, the collection, analysis, and interpretation of data, writing of the report, or the decision to submit the paper for publication. The corresponding author had full access to all the data and had the final decision to submit for publication.

Conflict of interest

The authors declare that we have no conflicts of interest.

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CERVICAL SPINE

Presence of Anterior Compression of the Spinal Cord After Laminoplasty Inhibits Upper Extremity Motor Recovery in Patients With Cervical Spondylotic Myelopathy

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Study Design. A retrospective single-center study.

Objective. To investigate how functional recovery is influenced by anterior compression of the spinal cord (ACS) and instability at the level of ACS after laminoplasty in patients with cervical spondylotic myelopathy.

Summary of Background Data. There have been many reports that patients whose spinal cord cannot be decompressed sufficiently after laminoplasty are likely to show unsatisfactory neurologic outcomes. Notably, postoperative ACS is well known to cause problems. Clinically, however, it remains unknown how functional recovery is inhibited by postoperative ACS.

Methods. Sixty-four consecutive patients who underwent expansive laminoplasty for the treatment of myelopathy at our hospital between 1998 and 2005 were reviewed. All 64 patients were available for follow-up. The average follow-up period was 97 months (60–156 months). Patients were divided into 2 groups: the ACS(+) group comprised 16 patients who had ACS 3 years postoperatively, and the ACS(–) group comprised 48 patients with no ACS. Clinical outcome was compared in terms of the Japanese Orthopaedic Association score (mean total score, mean score of each item, and recovery rates).

Results. Demographics were similar between the 2 groups. Mean Japanese Orthopaedic Association score at final follow-up was 12.1 points (recovery rate 34.0%) in the ACS(+) group and 13.8 points (recovery rate 56.6%) in the ACS(–) group, and there was a significant difference in recovery rate between the groups ($P < 0.05$). Notably, a significant difference was found between the 2 groups in improvement of upper extremity motor function ($P < 0.05$). In addition, we found that not only the presence of ACS but also postoperative hypermobility of the intervertebral segment with ACS influenced clinical outcome negatively.

Conclusion. These results demonstrate that ACS after laminoplasty could be a risk factor for clinical outcome and might prevent improvement in upper extremity motor function in patients with myelopathy.

Key words: anterior compression of the spinal cord, anterior horn, cervical spondylotic myelopathy, laminoplasty, upper extremity motor function. **Spine 2012;37:377–384**

Laminoplasty (LAMP) has been adopted by many surgeons[1–5] as an effective and safe treatment for patients with cervical spondylotic myelopathy (CSM). Although it is an easy, relatively fast, and cost-effective surgical method for most patients with cervical myelopathy, it is associated with several problems including the development of postoperative kyphosis, closure of the opened laminae, C5 palsy, and residual cord compression. Seichi *et al*[6] reported that patients with residual cord compression due to ossification of the posterior longitudinal ligament (OPLL) after LAMP can be at risk of mild cord injury from minor trauma. The presence of anterior compression of the spinal cord (ACS) will prevent improvements in neurologic outcome and cause the patients considerable distress. Although a few articles have reported on the presence of postoperative ACS as detected using magnetic resonance imaging (MRI), the etiology and frequency of this complication remain unclear. In addition, although it is well known that the symptoms of myelopathy consist of segmental and long tract disorders, how functional recovery is prevented by ACS also remains unknown. The purpose of our study was to investigate how functional recovery can be influenced by ACS after LAMP for treatment of patients with CSM.

MATERIALS AND METHODS

Patients and Methods

We conducted a retrospective, observational, single-center study of posterior decompression with LAMP for the

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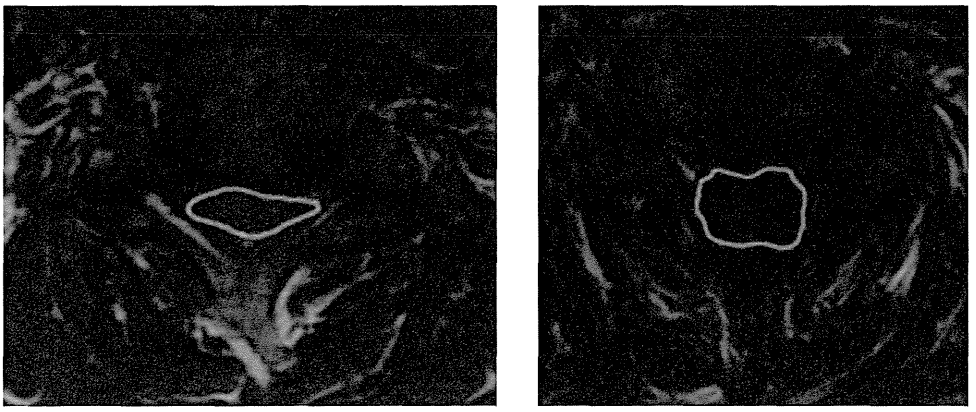

A B

Figure 1. Cross-sectional area within the cervical spinal canal at the responsible level was measured on the basis of T1-weighted magnetic resonance imaging using Adobe Photoshop CS4 and Scion Image before (**A**) and 3 years after surgery (**B**).

treatment of CSM. The study was carried out with the approval of the institutional ethics committee of Tokyo Medical and Dental University. Patients with cervical myelopathy caused by spondylosis were included in the study. Exclusion criteria were myelopathy caused by single-level disc herniation or OPLL, amyotrophy caused by spondylosis, and a history of previous cervical spine surgery. Patients with preoperative cervical kyphosis of more than 10° were also excluded. Sixty-four consecutive patients who underwent LAMP for the treatment of CSM between 1998 and 2005 were reviewed for this study. All 64 patients were available for follow-up. The average follow-up period was 97 months (60–150 months).

Surgical Technique

LAMP

Expansive LAMP, as described by Miyazaki and Kirita,[7] was performed, and decompression was extended from C3 to C7 in 38 patients, C3 to C6 in 25 patients, and C2 to C7 in 1 patient. Briefly, LAMP at C3 to C7 included removing the processes at C4 to C6, undercutting the cranial edge of the C7 laminae, splitting the laminae at the center, and making bilateral gutters using a high-speed air-burr drill. The bilateral laminae were kept open by anchor sutures to the deep fascia, and small bone chips made from the spinous processes were inserted into the gap between the laminae and the facets on the hinge side. Patients were instructed to wear a neck collar for 3 to 4 weeks postoperatively.

EVALUATION

Clinical Outcome

The Japanese Orthopaedic Association (JOA) scoring system, which consists of 7 categories including upper extremity motor function, lower extremity motor function, sensation of upper or lower extremity and trunk, and bladder functions, was used to evaluate cervical myelopathy before and at 3 and 6 months after surgery and annually thereafter. The recovery rate (RR) was calculated using the method of Hirabayashi *et al*[8] to compare pre- and postoperative JOA scores. Each item of the JOA score was also investigated pre- and postoperatively, and the RR of each of the 7 functions was calculated as we previously reported[9] to evaluate recovery of each function.

Radiologic Evaluation

Radiographic studies were conducted in all patients, and results were evaluated by 3 independent surgeons. Cervical sagittal alignment (C2–C7 lordotic angle) was determined as tangential lines on the posterior edge of C2 and C7 bodies and preoperatively available anteroposterior canal diameter of the cervical spine.

MRI scans were obtained before and at 3 months after surgery in all patients and annually thereafter. The presence of preoperative signal intensity change in the cord was evaluated on sagittal view T2-weighted MRI. The cross-sectional areas within the space available for the spinal cord (SAC) at the responsible level and at the C4 vertebral level, which was decompressed in all cases, were measured on axial view T1-weighted MRI. The preoperative space available for the spinal cord was defined as the area enclosed by the inner margins of the ligamentum flavum and anterior factors (*i.e.*, spur formation of the vertebral body and disc bulging) before surgery (Figure 1A). The postoperative space available for the spinal cord was defined as the area enclosed by the inner bony margin of the opened lamina and inner margin of anterior factors 3 years after surgery (Figure 1B). These areas were digitally analyzed using Photoshop CS4 (Adobe Systems Incorporated, San Jose, CA) and Scion Image (National Institutes of Health, Bethesda, MD). Expansion rate of the area of the spinal canal (ER; %) was defined as (postoperative area – preoperative area) × 100 / preoperative area.

ACS after LAMP was also evaluated. The criteria for defining significant ACS, as described by Bapat *et al*,[10] were as follows: (1) effacement of anterior cerebral spinal fluid buffer on the T2 sagittal and axial images; and (2) evidence of anterior compression of cord substance on the T1 sagittal and axial images. If both these criteria were satisfied, the cord was considered to be ACS(+). In addition, we hypothesized that not only static but also dynamic factors of ACS influence neural recovery after LAMP. Therefore, we investigated the associations between clinical outcome and either the presence

TABLE 1. Demographic Data of Patients Who Underwent LAMP for Treatment of CSM

	ACS(+) group (n = 16)	ACS(–) group (n = 48)	P
Age (yr)	64.8 ± 10.6	62.0 ± 10.2	ND
Male (%)	75.0	75.0	ND
Preoperative JOA score	9.9 ± 2.3	10.0 ± 2.9	ND
Duration of symptom (mo)	14.8 ± 8.8	15.8 ± 11.3	ND
Canal diameter (mm)	11.8 ± 1.0	11.9 ± 1.6	ND
Positive ratio of signal intensity change (%)	50	45.8	ND
Segments (case)	C3–C7: 13	C3–C7: 25	
	C3–C6: 3	C3–C6: 22	
		C2–C7: 1	

Data are expressed as mean ± standard deviation

ACS indicates anterior compression of spinal cord; CSM, cervical spondylotic myelopathy; JOA, Japanese Orthopaedic Association; LAMP, laminoplasty; ND, not significant difference.

of ACS or the range of motion in flexion-extension, calculated using x-ray, at the level falling under the definition of ACS (ROM_{ACS}) at the 3-year time point.

The 64 patients were divided into 2 groups: the ACS(+) group comprising 16 patients who had ACS at 3 years postoperatively and the ACS(–) group comprising 48 patients with no ACS.

Statistical Analysis

The Student unpaired *t* test was used to compare the differences in each item between the 2 groups, except for the RR of each function, for which the Mann-Whitney *U* test was used. A linear regression model was used to determine whether there was any correlation between the RR of the JOA score and the ROM_{ACS} in the ACS(+) group. A *P* value of less than 0.05 was considered significant.

RESULTS

The mean patient age was 64.8 years (range: 48–82 years) in the ACS(+) group and 62.0 years (range: 42–86 years) in the ACS(–) group. Average duration of symptoms before surgery was 14.8 months (range: 0.5–36.0 months) in the ACS(+) group and 15.8 months (range: 0.75–36.0 months) in the ACS(–) group. None of the 64 patients underwent secondary spinal surgery due to deterioration of myelopathy. Mean JOA score before surgery was 9.9 points in the ACS(+) group and 10.0 points in the ACS(–) group. There was no significant difference in demographic data between the 2 groups (Table 1).

Clinical Results

Clinical and radiologic outcomes are summarized in Table 2. There was a significant difference in RR of the total JOA score at the final visit (Figure 2). The best JOA score within the 5 years after surgery was almost the same as that at the final visit. The JOA score improved in 62 of 64 patients immediately after treatment, and no patients deteriorated after surgery in either group. However, no improvement (RR of 0%) was seen in 2 patients in the ACS(–) group, both of whom had suffered from myelopathy for more than 3 years preoperatively and whose symptoms were unchanged from immediately after surgery until the final visit. RR for each item of the JOA score in the ACS(+) group *versus* the ACS(–) group were as follows: upper extremity motor function (36.5% *vs.* 71.7%), lower extremity motor function (33.8% *vs.* 54.7%), sensation of upper extremity (56.4% *vs.* 45.1%), sensation of trunk (80.8% *vs.* 82.4%), sensation of lower extremity (66.7% *vs.* 88.7%), and bladder function (63.2% *vs.* 75.0%). There was a significant difference between the 2 groups only for improvement in upper extremity motor function ($P < 0.05$, Table 2).

Radiographic Results

Mean C2–C7 lordotic angle in the neutral position changed from 14.0° to 12.0° in the ACS(+) group and from 15.8° to 13.1° in the ACS(–) group, indicating no significant difference between the groups in postoperative kyphotic change. The segment with ACS was at C3/C4 in 2 cases, C4/C5 in 6 cases, and C5/C6 in 8 cases. This result showed that ACS was likely to be seen at C4/C5 or C5/C6. The mean ROM_{ACS} on x-rays was 5.2° (range: 0°–13.2°, Table 2). ROM_{ACS} was not directly associated with preoperative mobility. In terms of positive ratio of signal intensity change on preoperative MRI, there was no significant difference between the ACS(+) and ACS(–) groups (Table 1). However, patients with a change in signal tended to have unsatisfactory surgical outcomes (data not shown). Mean ER at the responsible level was 59.6% in the ACS(+) group and 94.5% in the ACS(–) group, which represented a significant difference between the 2 groups ($P < 0.001$, Table 2), whereas mean ER at the C4 level was similar in the 2 groups.

Next, we investigated the cause of ACS after LAMP. Eleven patients had ACS immediately after surgery—that is, residual ACS. Of these 11 cases, preoperative local kyphosis was observed at the responsible level in 6 patients, local kyphosis plus spondylolisthesis in 3 patients, and disc bulging in 2 patients (Table 3). In addition, ACS occurred from 2 years postoperatively in 5 other patients; ACS was induced by the progression of local kyphotic change in 2 of these patients, by spondylolisthesis in 1 patient, by both local kyphosis and spondylolisthesis in 1 patient, and by new disc bulging in the remaining patient (Table 3). In all 5 of these patients, the occurrence of ACS due to progressive degenerative change could not be foreseen before surgery because they had preoperative lordotic or straight alignment that should prevent postoperative ACS. The cause of ACS was local kyphosis in 12 of 16 patients (Table 3). We also found that the greater the mobility

TABLE 2. Clinical Outcomes in the ACS(+) and ACS(–) Groups

		ACS(+) Group (n = 16)		ACS(–) Group (n = 48)	*P*
JOA score (pts)					
Preoperative		9.9 ± 2.3		10.0 ± 2.9	ND
Postoperative		12.1 ± 2.2		13.8 ± 2.4*	<0.05
RR of JOA score (%)		34.0 ± 18.6		56.6 ± 25.4†	<0.005
RR of each function in JOA score (%)					
Motor function	Upper extremity	36.5 ± 39.6		71.7 ± 33.6*	<0.05
	Lower extremity	33.8 ± 29.3		54.7 ± 38.7	ND
Sensation	Upper extremity	56.4 ± 35.7		45.1 ± 46.2	ND
	Trunk	80.8 ± 39.6		82.4 ± 34.0	ND
	Lower extremity	66.7 ± 45.1		88.7 ± 44.8	ND
Bladder function		63.2 ± 49.8		75.0 ± 50.0	ND
C2–C7 angle (°)	Preoperative	14.0 ± 11.7		15.8 ± 9.5	ND
	Postoperative	12.0 ± 13.0		13.1 ± 12.3	ND
Postoperative kyphotic change		2.0 ± 7.5		2.4 ± 9.8	ND
Segment with postoperative ACS (case)		C3/C4	2		
		C4/C5	6		
		C5/C6	8		
ROM_{ACS} (°)		5.2 ± 3.8			
Cross-sectional area within the cervical spinal canal					
At C4 vertebral level					
Preoperative (mm^2)		178.6 ± 11.9		172.8 ± 12.3	ND
Postoperative		296.5 ± 23.1		290.1 ± 28.1	ND
Expansion rate (%)		67.1 ± 23.4		68.6 ± 25.4	ND
At responsible level					
Preoperative (mm^2)		139.0 ± 5.3		140.8 ± 7.6	ND
Postoperative		221.5 ± 27.3		273.1 ± 38.7†	<0.005
Expansion rate (%)		59.6 ± 20.5		94.5 ± 27.3†	<0.005

Data are expressed as mean ± standard deviation.

**P < 0.05.*

†P < 0.005 versus ACS(+) group by Student unpaired t test or Mann-Whitney U test.

ACS indicates anterior compression of spinal cord; JOA, Japanese Orthopaedic Association; ND, not significant difference; ROM, range of motion; RR, recovery rate.

of the segment with local kyphosis, the more likely it would be classified as newly acquired ACS (data not shown). On the other hand, patients with residual ACS had poorer outcomes than those with newly acquired ACS ($P < 0.05$, Table 3).

We also investigated whether, in addition to the presence of ACS, the RR of the JOA score was influenced by the mobility of the segment with ACS. A negative correlation was found between clinical outcome and ROM_{ACS} ($y = -3.769x + 53.96$; where y is the RR of the JOA score, and x is the ROM_{ACS}, $P < 0.0001$, Figure 3).

CASE PRESENTATIONS

Case 1. A 67-year-old woman presented with bilateral hand clumsiness (2 points) and a spastic gait (2 points). Sagittal view on MRI showed that both anterior and posterior factors at C4/C5 and C5/C6 were related to severe compression of

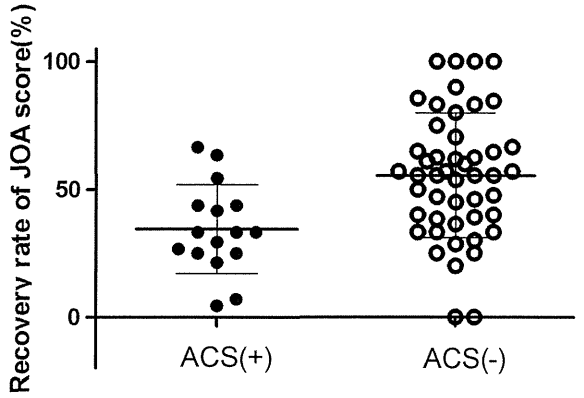

Figure 2. The recovery rate of the Japanese Orthopaedic Association score was significantly higher in the group with anterior comparison of the spinal cord (ACS) at 3 years postoperatively (ACS[+]) than in the group without ACS (ACS[–]) ($P < 0.05$).

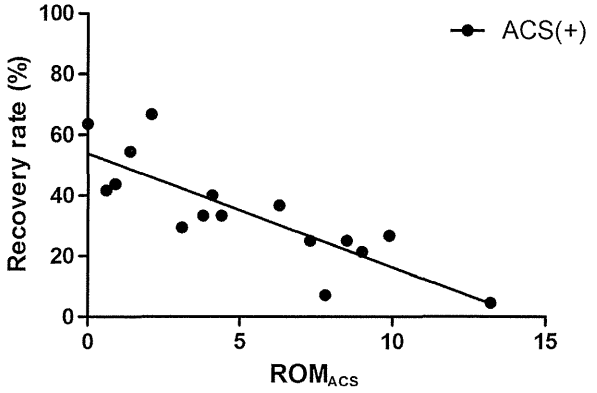

Figure 3. In the group with anterior compression of the spinal cord (ACS(+)), there was a statistically negative correlation between clinical outcome and the range of motion in flexion-extension at the level falling under the definition of ACS (ROM_{ACS}; $y = -3.769x+53.96$; R^2: 0.70, R: 0.834, $P < 0.001$).

the cervical spinal cord (Figure 4A). LAMP at C3–C7 was performed, because her cervical alignment had sufficient lordosis (C2–C7; 24°). A postoperative MRI at 3 months showed persistent anterior impingement of the cord at C4/C5 and C5/C6 and insufficient posterior shift of the cord (Figure 4B). At 3 years postoperatively, ROM_{ACS} was 8.6°, and the RR of upper extremity motor function was 25% (2.5 points).

Case 2. A 64-year-old man had been suffering for approximately 3 years from progressive impairment of fine motor skills in his upper limbs (1.5 points). Six months after surgery, the fine motor skills of his hands had slightly improved (2 points), and MRI showed sufficient decompression around the cord with new local kyphosis at C4/C5 (Figure 5A). Two years after surgery, however, he reported a worsening of bilateral hand clumsiness (1.5 points) despite physiotherapy. MRI at 3 years after surgery showed ACS at C4/C5 with both local kyphotic change and spondylolisthesis (Figure 5B), and ROM_{ACS} was 8.1°. There was no recovery of upper extremity motor function.

TABLE 3. Causes of Postoperative ACS in 16 Patients

Cause of ACS	Residual ACS (n = 11)	Newly Acquired ACS (n = 5)	*P*
Local kyphosis alone	6	2	
Spondylolisthesis alone		1	
Local kyphosis + spondylolisthsis	3	1	
Disc bulging	2	1	
RR of JOA score (%)	27.9 ± 15.1	49.2 ± 19.6*	<0.05

**P < 0.05 versus residual ACS by Student unpaired t test.*

ACS indicates anterior compression of spinal cord; JOA, Japanese Orthopaedic Association; RR, recovery rate.

DISCUSSION

It has been reported that the decompression effect of LAMP consists of 2 distinct mechanisms: a direct posterior decompression effect and an indirect anterior decompression effect resulting from the posterior shift of the spinal cord from the anterior compressive lesion. Whether insufficient indirect decompression after LAMP is associated with the risk of poor clinical outcome has been widely debated. Baba *et al*[11] showed that neurologic improvement is associated with posterior cord shift on MRI. Sodeyama *et al*[12] reported that a posterior shift of the spinal cord of more than 3 mm leads to good recovery of myelopathic symptoms. Based on these facts, it is clear that not only direct decompression but also an indirect effect is very important for the surgical treatment of myelopathy. In our study, the ER at the responsible level on MRI showed a significant difference between the 2 groups, whereas there was no such difference at the C4 level and no obvious closure of the laminae on postoperative computed tomography in either group (data not shown). Therefore, we found that indirect anterior decompression was not sufficient at the level of ACS despite sufficient opening of the lamina. However, there are still very few reports of studies that have analyzed the decompression status in relation to ACS. Mihara *et al*[13] evaluated the relationship, using intraoperative sonography, between the subarachnoid space ventral to the cord and postoperative neurologic recovery after LAMP. They concluded that restoration of the anterior subarachnoid space could be a significant factor for neurologic improvement after posterior decompression surgery. Our clinical and radiologic results support this. Breig *et al*[14–15] revealed that filling arterial defects to the spinal cord occur in cervical flexion, leading to a reduction in blood supply to the spinal cord. Likewise, Shinomiya *et al*[16] showed that anterior compression of the cord leads to symptoms of myelopathy if there is a loss of the posterior cervical epidural ligaments that anchor the posterior dura mater to the ligamentum flavum. According to these findings, it is speculated that anterior impingement of the cord does not allow for either adequate cerebrospinal fluid circulation or blood supply from the

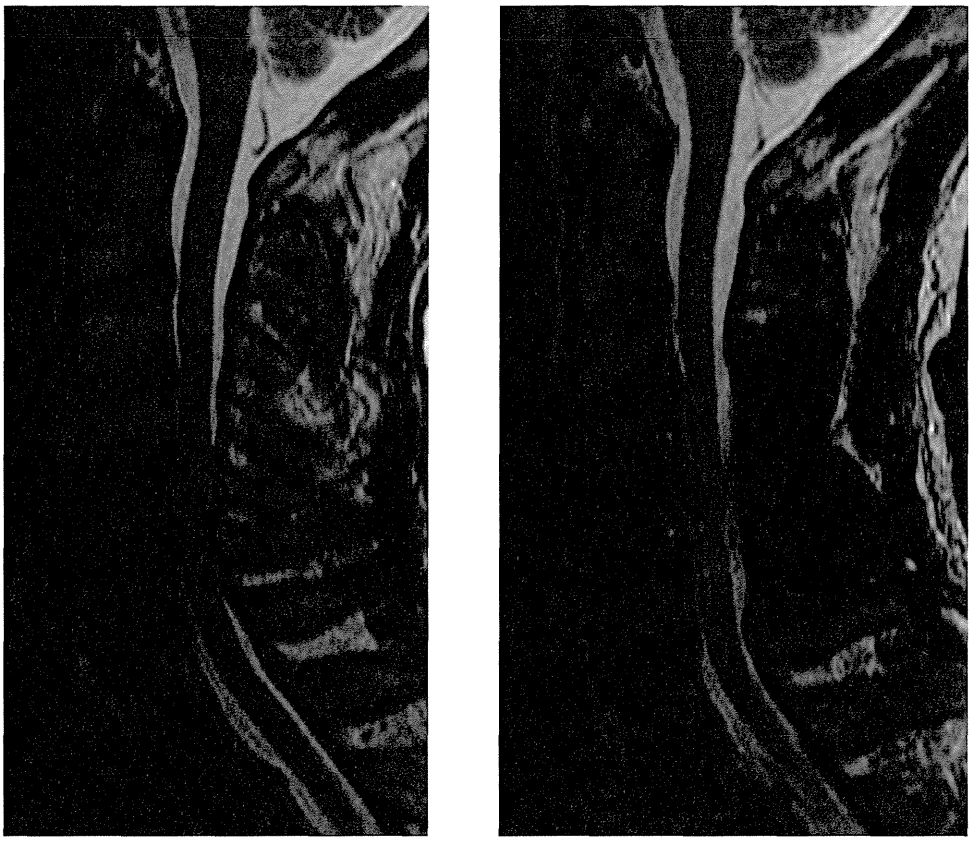

Figure 4. Preoperative magnetic resonance imaging in Case 1 showed that both anterior and posterior factors at C4/C5 and C5/C6 were related to severe compression of the cervical spinal cord (**A**). Postoperative magnetic resonance imaging after 3 months showed persistent anterior impingement of the cord at C4/C5 and C5/C6 and insufficient posterior shift of the cord (**B**).

anterior spinal artery or nerve root arteries and may, thus, inhibit the degree of postoperative neurologic recovery. In addition, we found a negative correlation between clinical outcome and ROM_{ACS} (Figure 3). This suggests that clinical outcome might be influenced negatively by both the existence of ACS (static factor) and the postoperative segmental

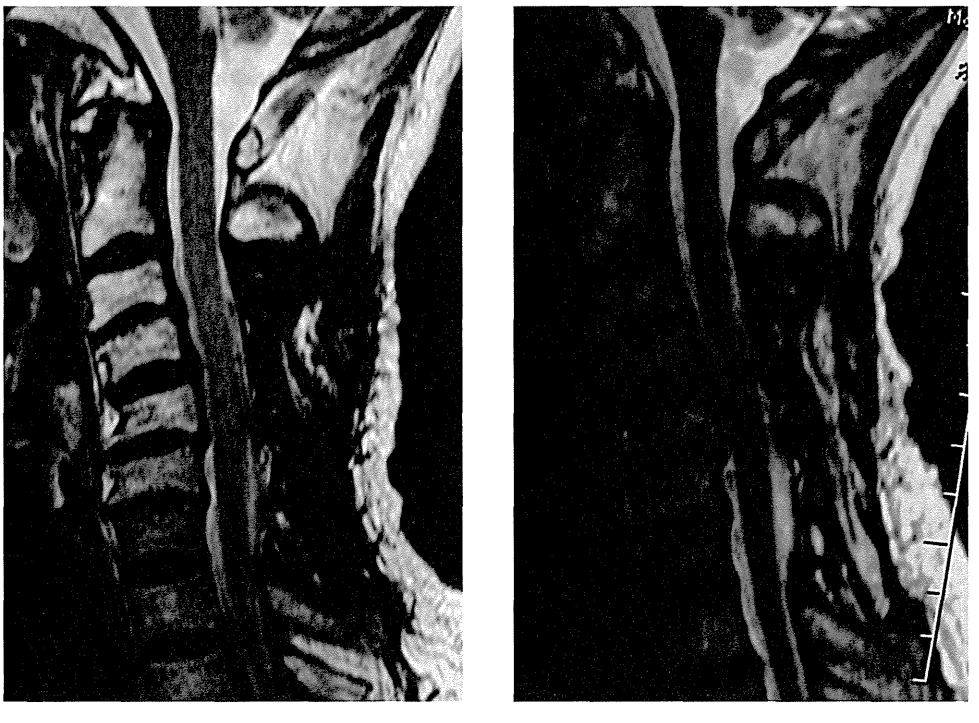

Figure 5. Preoperative magnetic resonance imaging (MRI) in Case 2. MRI obtained 3 months after LAMP (**A**) showed mild kyphosis with no anterior compression of spinal cord. At 3 years postoperatively, however, MRI (**B**) showed anterior compression of spinal cord at C4/C5 with both local kyphotic change and spondylolisthesis.

mobility (dynamic factor). Compared with ACS in patients treated with LAMP for OPLL,[6] we speculated that ACS in our patients treated with LAMP for CSM, which preserves the mobility of the cervical spine, may damage the anterior aspect of the cord mechanically and inhibit the blood supply such that the greater the mobility at the responsible level, the poorer the outcome for patients in this series (Figure 3). Based on these results, ACS after LAMP for CSM should be considered as a complication after posterior decompression. We proposed salvage surgery for 16 ACS(+) patients to eliminate ACS. However, the patients rejected it because they were satisfied with their condition.

On the other hand, the improvements in lower extremity motor and sensory functions in the ACS(−) group tended to be better than those in the ACS(+) group, but the differences were not significant. The anterior structure of the cord includes gray and white matter, so that not only the anterior horn but also the pyramidal tract and afferent sensory fibers from the lower extremities can be damaged by ACS after LAMP. Li *et al*,[17] using the finite element method as a 3-dimensional modeling technique, demonstrated that the anterior horn in the gray matter was more likely to be vulnerable than the long tracts in the white matter. These mechanisms can explain why there was a significant difference found only in upper extremity motor function in the present study.

It is very important for surgeons to find a preoperative index to predict the presence of ACS after LAMP. Fujiyoshi *et al*[18] suggested that the K-line could be a candidate for evaluating the relationship between cervical alignment and OPLL. Therefore, we attempted to determine whether a line connecting the midpoints of the spinal canal at C2 and C7 on preoperative MRI could be useful for deciding the appropriate surgical treatment for CSM. However, ACS after LAMP could be predicted in only 5 of 11 patients who suffered from residual ACS; thus, this line could not accurately predict ACS after LAMP for the treatment of patients with CSM. These 5 patients, in whom anterior decompression should be selected as surgical treatment, required a less invasive procedure because of their poor general health. Indeed, the postoperative alignment of the cord is not usually dependent on skeletal morphology of the cervical spine, whereas it is likely that patients with sufficient lordotic alignment of the cervical spine, allowing the cord to shift dorsally, can obtain indirect decompression after LAMP. Jokich *et al*[19] reported that the spinal cord moves very little at the site of maximal compression after posterior decompression, as the posteriorly directed force from the anterior mass is counteracted by restraining dentate ligaments and nerve roots. Consistent with the report, findings of the present study demonstrated that the degree of posterior shift of the cord after posterior decompression alone in patients with a high degree of anterior compression was likely to be less than expected (Figure 4B). Thus, it is difficult to predict ACS after LAMP by preoperative radiologic examination alone.

The results of this study show that ACS was induced by preoperative local kyphosis in 12 (75%) of 16 patients who had ACS after LAMP. Notably, we also found that local kyphosis with preoperative instability increases the incidence of newly acquired ACS after LAMP, a finding consistent with the results of Suda *et al*.[20]

This study demonstrates that there is a limitation of LAMP for the treatment of patients with local kyphosis with instability or a high degree of anterior compression. We recommend in patients with such preoperative factors that other treatment options such as posterior decompression with fusion or anterior procedures should be considered to avoid postoperative anterior impingement or to stabilize the mobility of the segment with ACS.

➢ Key Points

- ❑ The results of this study showed that the improvement in JOA score in patients with no ACS was significantly better than in those with ACS.
- ❑ Among the individual items of the JOA score, only a statistically significant difference in improvement of upper extremity motor function was found between the ACS(+) and ACS(−) groups.
- ❑ The results revealed that clinical outcomes after LAMP could be influenced not only by the presence of ACS but also by postoperative mobility of the intervertebral segment with ACS.
- ❑ Posterior decompression alone is a limitation for treating patients with a lack of stability at the level with local kyphosis or large anterior compression.

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CERVICAL SPINE

Warning Thresholds on the Basis of Origin of Amplitude Changes in Transcranial Electrical Motor-Evoked Potential Monitoring for Cervical Compression Myelopathy

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Study Design. A retrospective analysis of prospectively collected data from consecutive patients undergoing transcranial electrical motor-evoked potential (TCE-MEP: compound muscle action potentials) monitoring during cervical spine surgery.

Objective. To divide the warning threshold of TCE-MEP amplitude changes on the basis of origin into the spinal tract and spinal segments and decide warning thresholds for each.

Summary of Background Data. The parameter commonly used for the warning threshold in TCE-MEP monitoring is wave amplitude, but amplitude changes have not been examined by anatomical origin.

Methods. Intraoperative TCE-MEP amplitude changes were reviewed for 357 patients with cervical myelopathy. Most of the patients were monitored by transcranial electrical stimulated spinal-evoked potential combined with TCE-MEP. The warning threshold of TCE-MEP was taken as waveform disappearance. For each patient, amplitude changes were separated, according to origin, into the spinal tract and spinal segments and compared with clinical outcome.

Results. Assessable TCE-MEP waves were obtained in 350 cases. Disappearance of TCE-MEP waves, which were innervated by the spinal levels exposed to the surgical invasion, was seen in 11 cases. Disappearance of TCE-MEPs, which were innervated by the spinal levels inferior to them, was seen in 43 cases. There was no postoperative motor deficit in those cases. However, such deficits caused by spinal segment injury were seen in 2 cases, which showed that intraoperative amplitude decreased to 4.5% and 27%.

Conclusion. If we had established the warning threshold as 30% of the control amplitude, we would likely have prevented both cases of postoperative motor deficits, but 106 (30.3%) cases would have become positive cases. If we had established the warning threshold separately as wave disappearance for the spinal tract and 30% of the control amplitude for the spinal segments, sensitivity and specificity would have been 100% and 83.7%, respectively. Dividing the warning threshold on the basis of origin of amplitude changes could reduce false-positive cases and prevent intraoperative injuries.

Key words: motor-evoked potential, intraoperative monitoring, warning threshold. **Spine 2012;37:E913–E921**

Transcranial electrical motor-evoked potential (TCE-MEP) monitoring has become widely used during spine surgery to monitor motor function. Among its advantages, TCE-MEP monitoring requires no invasive procedures and can monitor each bilateral and segmental function of gray as well as white matter by measuring multiple electromyograms.[1,2] However, the waveform amplitude of TCE-MEP is changeable because it is susceptible to anesthetic agents and is itself a compound electromyogram.[3,4] To date, no reliable warning threshold for TCE-MEP monitoring has been reported.

We had initially taken disappearance of the TCE-MEP wave as the warning threshold at our institution on the basis of our clinical experience that, in around 500 cases, postoperative motor deficit presented only when there had been disappearance of the TCE-MEP wave intraoperatively (unpublished data) and because some articles had suggested establishing such a threshold.[5–10] However, we subsequently experienced several cases of postoperative motor deficit that involved deficit in only the upper limb muscles innervated by the spinal segment, which might have had direct surgical damage. Meanwhile, we had experienced no postoperative muscle weakness of the lower limbs due to injury to the spinal tract. TCE-MEP monitoring is thought to be able to evaluate

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functioning of the entire corticospinal motor system below the brain stem,[1,9,11–16] and as such intraoperative changes seen in the TCE-MEP waveform may be caused by injury either to the spinal tract or the spinal segments. We therefore aimed to distinguish between motor deficit caused by spinal segment injury and that caused by spinal tract injury.

In this study, we reviewed intraoperative changes in TCE-MEP amplitude in our past cases for the purpose of establishing the warning threshold of impending neurological deficits caused by spinal tract injury and spinal segment injury, respectively, in surgeries for compressive myelopathy.

MATERIALS AND METHODS

We had performed spinal cord monitoring for all 357 patients undergoing cervical spine surgery for compression myelopathy at our institution between April 2003 and April 2010. Details of the clinical diagnoses are listed in Table 1. There were no cases of intramedullary tumor. Intraoperative TCE-MEP monitoring was applied in all 357 cases. Moreover, transcranial electrical simulated spinal cord-evoked potential (TCE-SCEP) monitoring was combined with TCE-MEP in all cases except 1 case of cervical arachnoid cyst complicated with a lower thoracic lesion and 2 cases of cervical cord injury due to trauma.

Total intravenous anesthesia was used for all patients. Anesthesia was maintained with intravenous remifentanil (0.25–0.5 μg/kg/min) or fentanyl (1.3 μg/mL: blood level) and with propofol (4–6 mg/kg/h) through the use of a constant infusion pump. Anesthetic depth was controlled so as to keep the bispectral index level in the range of 40 to 60. Vecuronium (Vecuronium bromide [0.08–0.1 mg/kg]) or rocuronium bromide (0.6 mg/kg) was used as a muscle relaxant only at the time of induction of anesthesia. Intraoperative continuous infusion of muscle relaxant was used exceptionally in earlier cases at a fixed minimum level (Vecuronium bromide, 0.5–1.5 mg/h) only when body movement due to electrical stimulation disturbed the surgical position. The level of muscle relaxant was not changed during surgery in any cases. Body temperature was maintained at more than 35°C intraoperatively in all patients.

TABLE 1. Details of Clinical Diagnoses

Diagnosis	No. of Patients
Cervical spondylotic myelopathy	202
Ossification of posterior longitudinal ligament or yellow ligament	80
Extramedullary spinal cord tumor	27
Cervical disk herniation	20
Congenital anomaly of spine	8
Atlantoaxial subluxation with rheumatoid arthritis	9
Other	11

Neuropack MEB-2200 (Nihon Kohden, Tokyo, Japan) was used for the measurement and analysis of evoked potentials. The stimulating electrodes were a pair of needle electrodes bent in a hook shape of 90° and placed on the scalp 5 cm lateral and 2 cm anterior to Cz (International 10–20 system) in the symmetric position. Stimulation for TCE-MEP and TCE-SCEP was performed under the 2 conditions in which the right electrode was designated as anode or cathode. Measurement of both evoked potentials was performed before and after the invasive surgical procedure. Periodical measurement was also performed once every 15 to 30 minutes even when no invasive procedure was performed. Changes in amplitude were determined by comparing the intraoperative waveform with the waveform measured one time before that provided a control amplitude. The stimulating condition of TCE-MEP was 200 mA intensity, a train of 5 rectangular pulses of 0.5-ms duration with an interstimulus interval of 0.2 ms, and 1-Hz frequency for each stimulus unit. In general, TCE-MEPs were recorded from bilateral biceps brachii (biceps), abductor digiti minimi (ADM), and flexor hallucis brevis. The deltoid muscles were also used if decompression of higher cervical levels was needed. TCE-MEPs were measured in belly-tendon derivation, using surface electrodes. The average of 5 to 10 waves from consecutive repetitive stimulations was calculated and recorded. For TCE-SCEP recording, a single rectangular pulse of 200-mA intensity, 0.5-ms duration, and 3-Hz frequency was used. TCE-SCEP was recorded by bipolar derivation from epidural electrodes (Unique Medical, Tokyo, Japan) placed before surgery, using a Tuohy needle in the lower thoracic epidural space (Th11–12). The distance of the poles was 15 mm. Consecutive potentials (20–50) were averaged and recorded. We regarded a decrease in amplitude to less than 50% as a significant change in TCE-SCEP.[8,9,12,17,18]

TCE-MEP and TCE-SCEP were measured and analyzed in the operating room in real time by 1 of 4 orthopedic surgeons who received training, and the results were reported orally to the operating surgeon within 1 minute of the measurement. The operating surgeon was warned only when the TCE-MEP wave disappeared concomitant with significant changes in TCE-SCEP or a surgical procedure, which might cause spinal injury (specifically, spinal decompression, installation of the implant, or changing the patient's position). When the requirement for warning was met, before alerting the surgeon, we first checked whether the wave disappearance was due to technical problems of measurement, anesthetic dose, body temperature, or blood pressure. We excluded the changes due to these external factors from the warning requirements and acted to improve them. If improvement of the amplitude did not occur despite elimination of these external factors, a warning was given, and the surgeon suspended the procedure or treated the factor thought to be the cause of the wave disappearance.

In this study, TCE-MEP of the flexor hallucis brevis was used to monitor the spinal tract. TCE-MEPs of the deltoid, biceps, and ADM were used to monitor the corresponding spinal segments when the spinal level innervating them (deltoid: C5–C6, biceps: C5–C6, ADM: C8–T1) was within a

decompressed spinal level; when the spinal level innervating these segments was lower than the decompressed level, the same TCE-MEPs were used to monitor the spinal tract.

Intraoperative amplitude changes in TCE-MEP waves, presence of warning, and presence of postoperative motor deficits were investigated retrospectively in all patients, and each correlation was evaluated. Muscular strength was evaluated using the 6 (0–5) grades of the manual muscle testing (MMT) just before and after surgery, and we considered postoperative degradation of MMT as motor deficit.

RESULTS

Assessable and reproducible TCE-MEP waves were obtained in 350 (94.6%) of the 357 cases. We investigated the number of the cases with intraoperative changes in wave amplitude (Table 2). When amplitude change was noted in several muscles, the largest change was used in the analysis. Disappearance, including temporal disappearance, of the wave was seen in 48 (13.7%) of the 350 cases. We gave a warning to the surgeon in 15 of these 48 cases and the surgeons acted accordingly, recovering the waveform in 10 cases and not being able to recover it in the remaining 5 cases until surgery was completed. However, no postoperative muscle weakness was seen in either set of cases. Also, there were no postoperative motor deficits in the 33 cases in which no warning had been required. Moreover, postoperative motor deficits considered to be due to spinal segment injury became apparent in 2 cases, which showed no wave disappearance intraoperatively and in which no warning was therefore given.

The first of these 2 cases, case 1, had undergone anterior decompression and fusion with instrumentation of C2–C7 for ossification of the posterior longitudinal ligament of the cervical spine (Figure 1). In this case, TCE-MEP amplitude of the left biceps decreased to 4.5% during exposure as compared with before skin incision (Figure 2). We did not alert the surgeon, as there was no wave disappearance and the change occurred before decompression. However, the muscular strength of the left deltoid and left biceps decreased from a preoperative MMT of 5 to 1–2 just after the surgery. This amplitude change occurred before decompression procedures, and severe stenosis of C3–C4 and C4–C5 had been apparent on preoperative computed tomographic scan. Therefore, we assume that cervical hyperextension in the surgical position had additionally compressed the C5 and/or C6 spinal segments.

Case 2 had undergone laminectomy of C2–C5 and tumor removal for a cervical meningioma (Figure 3). When we resected the tumor around the left C5 root and C3–C4 spinal cord, TCE-MEP amplitude of the ipsilateral deltoid and biceps decreased to 27% and 21%, respectively, compared with before resection (Figure 4). We monitored the situation without warning the surgeon because waveform disappearance did not occur. However, muscular strength of the left deltoid and biceps was decreased from a preoperative MMT of 5 to 3 just after the surgery. This was suspected to be due to injury of the C5 spinal segment.

For the purpose of studying the cause of these 2 false-negative cases of segment origin, we retrospectively classified the intraoperative amplitude changes in all 350 cases into changes in waveform originating in the spinal tract or in the spinal segments and re-evaluated the classified changes and postoperative motor deficits. The number of cases with intraoperative amplitude change originating in the spinal tract

TABLE 2. Intraoperative Changes of Wave Amplitude Caused by Spinal Tract and Spinal Segments

Change in TCE-MEP Amplitude	Overall MEP Changes Caused by Spinal Tract and Segment Invasion			MEP Changes Caused by Spinal Tract Invasion			MEP Changes Caused by Spinal Segment Invasion		
	No. of Cases	No. of Warnings	Postoperative Motor Deficit	No. of Cases	No. of Warnings	Postoperative Motor Deficit (Tract)	No. of Cases	No. of Warnings	Postoperative Motor Deficit (Segment)
≥50%	196	0	0	223	0	0	287	0	0
40%–49% amplitude	20	0	0	21	0	0	8	0	0
30%–39% amplitude	28	0	0	20	0	0	18	0	0
20%–29% amplitude	25	0	1	15	0	0	15	0	1
10%–19% amplitude	21	0	0	18	0	0	7	0	0
0%–9% amplitude	12	0	1	10	0	0	4	0	1
Disappearance of waveform	48	15	0	43	14	0	11	3	0

TCE-MEP indicates transcranial electrical motor-evoked potential; MEP, motor-evoked potential.