Table II. Radiological assessment of the C5 palsy and control groups

	C5 palsy	Control	p-value
Plain radiograph			
Lordosis (mean; range)			
α angle (pre-operative) (°)	15.2 (-22 to 41)	16.6 (-8 to 45)	0.59
α angle (post-operative) (°)	14.4 (-27 to 42)	15.3 (-20 to 46)	0.70
β angle (pre-operative) (°)	10.2 (-25 to 40)	10.4 (-21 to 39)	0.98
β angle (post-operative) (°)	9.8 (-33 to 47)	8.3 (-25 to 38)	0.55
MRI			
Number of compression levels	2.9 (1 to 6)	2.7 (1 to 5)	0.60
Most compressive level (C3-4) (cases) (%)	15 (*35*)	27 (*27*)	0.54
HIA* (cases) (%)	33 of 43 (*77*)	78 of 100 (*78*)	0.49
HIA level (C3-4) (cases) (%)	8 (*24*)	21 (*27*)	0.50
Focal HIA (cases) (%)	29 (*88*)	65 (*83*)	0.77
Linear HIA (cases) (%)	4 (*12*)	13 (*17*)	0.20
Posterior shift of the spinal cord (C4-5) (mm)	3.9 (0 to 7.5)	3.0 (0 to 7.5)	0.0091
CT			
Width of the C5 intervertebral foramen (mm)			0.0043‡
Palsy side (C5 palsy)/right side (control)	1.6 (0.5 to 5.0)	4.3 (1.7 to 7.3)	< 0.0001§
Normal side (C5 palsy)/left side (control)	2.1 (0.8 to 7.0)	4.3 (1.0 to 7.2)	< 0.0001¶
Anterior protrusion of the C5 superior articular process (mm)			0.029‡
Palsy side (C5 palsy)/right side (control)	5.1 (0.25 to 9.2)	3.5 (0 to 10.0)	< 0.0001§
Normal side (C5 palsy)/left side (control)	4.3 (1.25 to 8.3)	3.5 (0 to 8.3)	0.048¶
Position of the bony gutter (%)			0.94‡
Palsy side (C5 palsy)/right side (control)	85.6 (50 to 133)	81.6 (50 to 100)	0.19§
Normal side (C5 palsy)/left side (control)	85.6 (61.5 to 175)	82.4 (55.6 to 100)	0.22¶

* HIA, high intensity areas
† values are given as means
‡ palsy side *vs* normal side
§ palsy side *vs* control
¶ normal side *vs* control

ness in the C5 distribution on the affected side, and this remained unchanged after laminoplasty in all except ten patients, in whom the numbness improved slightly. There was no significant difference in the time to motor recovery between these ten patients and the others, but those in whom numbess improved had significantly greater improvement of motor power in the deltoid and biceps ($p < 0.05$).

Radiological. The degree of cervical lordosis remained the same in both groups (Table II). In group P, the mean pre-operative intervertebral angle at C4-5 was 3.9° (-8° to 14°) and the mean post-operative intervertebral angle was 2.9° (-6° to 12°). In group P a post-operative local kyphosis was present in six patients, a posterior listhesis in five, and intervertebral instability in one: these were no worse at final follow-up at a minimum of two years. Post-operatively one patient developed a local kyphosis at C5-6, and two a retrolisthesis of C4. There was no correlation of these with the extent or duration of recovery from the C5 palsy.

Changes in cervical alignment are shown in Figure 3. There were no significant findings.

In group P, MRI showed that the mean number of levels at which the spinal cord was compressed was 2.9 (1 to 6; Table II), most frequently at C4-5 (Fig. 4). Pre-operative T2-weighted MRI demonstrated a high intensity area in 33 patients (77%) at 36 levels. These were at C3-4 in eight patients, C4-5 in 15, C5-6 in six, C6-7 in one and at C3-4+C4-5 in two, and at C4-5+C5-6 in one (Fig. 4). A high intensity area at a level corresponding to the C5 segment (C3-4) was evident in only ten patients (23%). Focal high intensity areas were present in 29 patients (58%) and linear high intensity areas in four (12%) patients in group P. In group C, 78 patients (78%) had a high intensity area. Focal high intensity areas were present in 65 (83%) and linear high intensity areas in 13 (17%). There were no significant differences between the two groups in the number and location of the compressed levels, or the prevalence and type of high intensity area. However, the mean post-operative posterior shift of the spinal cord at C4-5 was 3.9 mm (0 to 7.5) in group P and 3.0 mm (0 to 7.5) in group C ($p = 0.0091$) (Table II). There was no significant relationship between the shift of the spinal cord and the level of recovery from paralysis in group P.

On CT measurement, a difference in width between the intervertebral foramina was confirmed in 20 patients (47%); in 16 (80%) the side of the narrowing corresponded with the side that was paralysed. In group P, the mean width

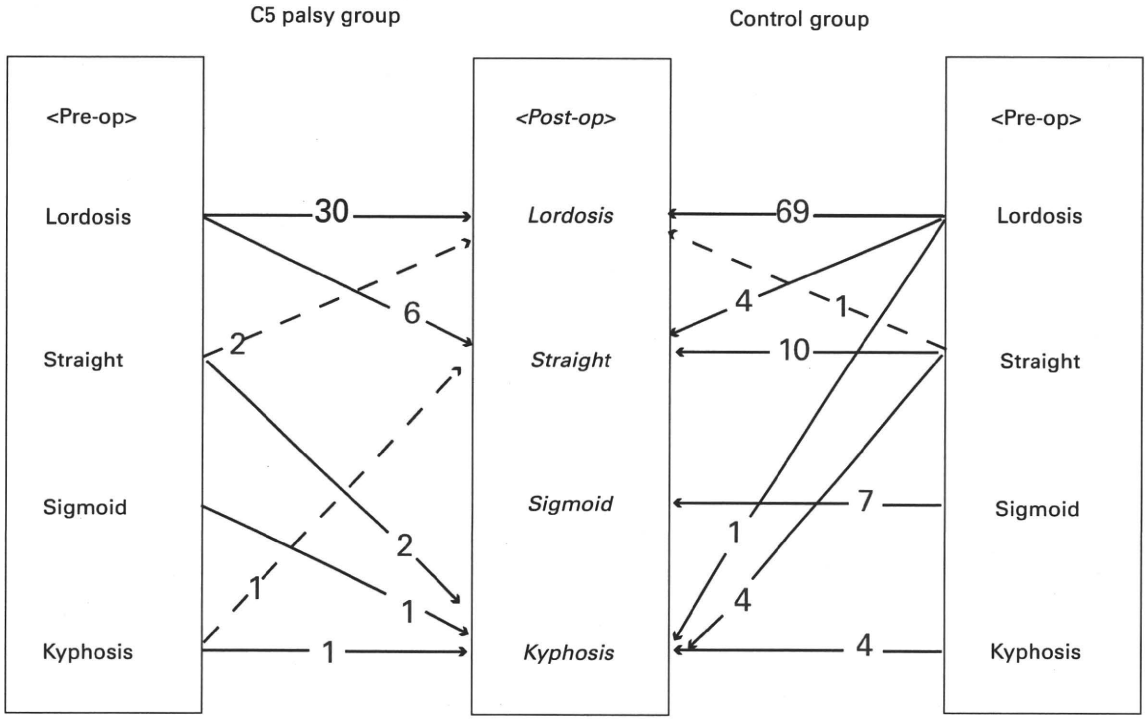

Fig. 3

Diagram showing pre- to post-operative changes in cervical alignment in the two groups. Cervical alignment was maintained in most cases in both groups. The findings were variable and the changes in cervical alignment showed no trend for either group or difference between the groups.

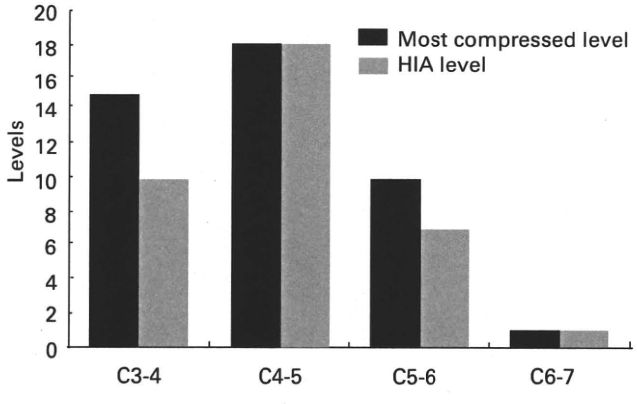

Fig. 4

Graph showing the most compressed level and high intensity area (HIA) level on MRI in cases of C5 palsy. An HIA at a level corresponding to the C5 segment (C3-4) was evident in only ten patients (23%).

of the intervertebral foramen was significantly less on the paralysed than on the normal side (1.6 mm *vs* 2.1 mm, p = 0.0043) (Table II). The mean anterior protrusion of the superior articular process of C5 was significantly greater on the paralysed than the normal side (5.1 mm *vs* 4.3 mm, p = 0.029). In group C, the mean width of the C5 intervertebral foramen (4.3 mm) was significantly greater and the degree of anterior protrusion of the superior articular process of C5 (3.5 mm) significantly less than on either the paralysed or the normal side in group P ($p < 0.0001$) (Table II). In group P the ratio reflecting the position of the bony gutter was 85.4% on the paralysed side and 85.6% on the normal side, whereas in group C these ratios were 81.6% on the right and 82.4% on the left side, with no significant effect on the development of C5 palsy (Table II). There were no cases of dislodgement of the hinge.

Discussion

Laminoplasty is an essentially safe procedure which allows the surgeon to decompress the cervical spine over a wide area. Consequently, it has been used for the treatment of compressive cervical myelopathy with favourable results.[28-31] However, one serious complication is a C5 palsy. In our series of 1858 laminoplasties, we identified 43 such cases. This is a relatively low incidence, but it should be appreciated that we only included severe patients with an MMT score of 2 or less. Other studies have included cases of higher grade, and some also include palsies of C6, C7 and C8 (Table III).

A number of causes have been suggested, including pathology of the spinal cord, nerve root impairment and the surgery itself.[15-19] In our study, the early development of a C5 palsy when the patient was still in bed suggested that it was not associated with loading of the spine by standing

Table III. Reported cases of upper limb palsy after laminoplasty

Authors	Level of palsy	Muscle weakness	Cases	Incidence (%)	Procedure*
Yonenobu et al[22]	C5	MMT = 3 to 5	4/95	*4.2*	Unilateral
Tsuzuki et al[34]	C5 - 8	MMT ≤ 5†	20/188	*10.6*	6 surgical procedures
Satomi et al[30]	C5, C6	MMT ≤ 4	6/51	*11.8*	Unilateral
Uematsu et al[19]	C5 - 7	MMT ≤ 5‡	20/365	*5.5*	Unilateral, French-window
Edwards et al[20]	C5	MMT = 0	1/18	*5.6*	French-door
Wada et al[40]	C5	NA¶	4/24	*16.7*	Unilateral
Chiba et al[15]	C5 - 8	NA	15/208	*7.7*	Unilateral
Hasegawa et al[17]	C5 - 8	NA	21/345	*6.1*	Unilateral, French-window
Current study	C5	MMT < 3	43/1858	*2.3*	Unilateral, French-window

* unilateral, unilateral hinge laminoplasty; French-window, French-door laminoplasty

† including sensory dominant paralysis

‡ including post-operative radiculopathy only

¶ NA, not available

or sitting. It was, however, frequently observed after surgery using a diamond burr. It is our opinion that the nerve root was probably damaged at the time of operation by heat generated by the high-speed drill, which would make it an iatrogenic injury. This risk has been further recently highlighted in an experimental porcine lumbar spine model by Hosono et al.[32]

Chiba et al[15] have suggested that local reperfusion of the spinal cord may cause cellular damage. This is based on the appearance on an MRI of high intensity areas at a significantly higher rate in patients with post-operative paresis of the upper limb than in those without. Furthermore, in their study, the high intensity areas corresponded to the level of the paralysed segment. Previous reports have also suggested that linear high intensity areas are seen more often on MRI at the paralysed level.[26,33] However, we found no difference in the occurrence of these areas between patients with and without a C5 palsy. Also, as most patients (95%; 41 of 43 patients) exhibited unilateral paralysis, we found it difficult to conclude that the cells of the spinal cord had only been damaged on one side. A disorder of the spinal cord cannot be formally excluded, but we found no radiological evidence to support it.

Tsuzuki et al[18,34] have suggested that the pathology of C5 palsy includes some impairment of the C5 nerve root. Using cadavers, they demonstrated that impingement of the nerve root occurs inside the intervertebral joint with backward shifting of the spinal cord after laminoplasty. Furthermore, the superior articular process of C5 protrudes in a more anterior direction than at other levels and the rootlets and root of C5 are shorter than those of other segments and the C5 segment is usually the point at which the extent of posterior shift of the cord is greatest. For these reasons, C5 nerve root impingement might easily occur after laminoplasty. However, radiological evidence of nerve root impingement has not been obtained in previous studies because of the small number of cases and the mixing of patients with C5 palsy who had paralyses of other nerves. In our study, the significantly greater posterior shift of the spinal cord in patients with a C5 palsy indicated tethering of the nerve root, which might be made worse by the tendency of the bony gutter to adopt a more lateral position in these patients. The CT findings also support this argument. We found that cases of C5 palsy accompanied by pain in the distribution of the C5 root accounted for about 80% of those in which impairment of the root was suspected clinically. This suggests that it is more likely to be C5 impairment rather than spinal cord pathology. On this basis, the identification on pre-operative CT of a C5 root that is highly susceptible to injury may be predictive of a C5 palsy after laminoplasty.

There is no reliable way of preventing a C5 palsy, but its occurrence may be lessened by the selective use of concomitant foraminotomy after pre- or intra-operative electrophysiological tests.[35-38] The pathology of the palsy may be multifactorial, but if a patient has a stenotic C4-5 intervertebral foramen and a prominent articular process, a palsy may be avoided by carrying out a concomitant foraminotomy. Excessive expansion by laminoplasty should be avoided to prevent the C5 nerve root becoming tethered by a posterior shift of the spinal cord.

Spontaneous recovery from a C5 palsy has been recorded,[19,30,39] but motor paralysis (33%; 14 of 43) and pain in the upper extremity (18%; 6 of 34) were present at final follow-up in some of our patients, making it difficult to conclude that a satisfactory recovery had been achieved with conservative treatment. As a significantly worse recovery occurred if the patient was severely paralysed at the onset (mean MMT score in deltoid = 1.2 (0 to 2)), conservative treatment may not be indicated for such cases. An additional foraminotomy at an early stage should be considered in those with a severe paralysis or unbearable pain.

A C5 palsy does not appear to be associated with the type of laminoplasty, sagittal alignment of the cervical spine, or the presence of a high intensity area in the spinal cord. We have demonstrated impairment of the C5 nerve root on CT scanning. Stenosis of the C4-5 intervertebral foramen and prominence of the superior articular process

of C5 on pre-operative CT are predictive factors for the development of a palsy. Significant posterior shift of the spinal cord from excessive expansion can easily lead to such a lesion. Foraminotomy may be useful to treat patients with a C5 palsy and severe pain or motor paralysis (MMT = 0 or 1), even after laminoplasty.

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Supplementary material

Graphs showing the posterior shift of the spinal cord on MRI was significantly greater in group P, ‡the width of the C5 intervertebral foramen was narrowest on the paralysis side, †the anterior protrusion of the C5 superior articular process was most prominent on the paralysis side and the position of the bony gutter in group P tended to be more lateral than that in group C, but there was no significant relationship between the position of the bony gutter and development of C5 palsy and a figure showing an example of how we adjusted the radiographs for actual length are available with the electronic version of this article on our website at www.jbjs.org.uk

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ORIGINAL ARTICLE

C5 palsy following anterior decompression and spinal fusion for cervical degenerative diseases

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Abstract Postoperative C5 palsy is a common complication after cervical spine decompression surgery. However, the incidence, prognosis, and etiology of C5 palsy after anterior decompression with spinal fusion (ASF) have not yet been fully established. In the present study, we analyzed the clinical and radiological characteristics of patients who developed C5 palsy after ASF for cervical degenerative diseases. The cases of 199 consecutive patients who underwent ASF were analyzed to clarify the incidence of postoperative C5 palsy. We also evaluated the onset and prognosis of C5 palsy. The presence of high signal changes (HSCs) in the spinal cord was analyzed using T2-weighted magnetic resonance images. C5 palsy occurred in 17 patients (8.5%), and in 15 of them, the palsy developed after ASF of 3 or more levels. Among ten patients who had a manual muscle test (MMT) grade $\leq$2 at the onset, five patients showed incomplete or no recovery. Sixteen of the 17 C5 palsy patients presented neck and shoulder pain prior to the onset of muscle weakness. In the ten patients with a MMT grade $\leq$2 at the onset, nine patients showed HSCs at the C3–C4 and C4–C5 levels. The present findings demonstrate that, in most patients with severe C5 palsy after ASF, pre-existing asymptomatic damage of the anterior horn cells at C3–C4 and C4–C5 levels may participate in the development of motor weakness in combination with the nerve root lesions that occur subsequent to ASF. Thus, when patients with spinal cord lesions at C3–C4 and C4–C5 levels undergo multilevel ASF, we should be alert to the possible occurrence of postoperative C5 palsy.

Keywords C5 palsy · Cervical spine · Anterior surgery · Decompression · Fusion

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Introduction

Postoperative C5 palsy is well known as a common complication after decompression surgery at the cervical spine [12]. Previous reports have indicated that C5 palsy occurs not only after posterior cervical decompression surgery, such as laminoplasty [1, 2, 5, 6, 8, 10, 13, 16–19], but also after anterior surgery [3–5, 7, 14, 15, 18, 19]. However, the number of studies analyzing C5 palsy after anterior cervical surgery is smaller than that for after posterior cervical surgery. Thus, the incidence, prognosis, and etiology of the C5 palsy after anterior cervical surgery have not yet been fully established. The aim of the present study was to investigate the incidence and prognosis of C5 palsy after anterior cervical surgery and discuss the mechanism of development of this disorder.

Materials and methods

Patient population

Between 1996 and 2004, 199 patients underwent anterior decompression surgery and spinal fusion (ASF) for cervical degenerative diseases in our institute (Table 1). Their

Table 1 Cervical lesions and incidence of postoperative C5 palsy

Cervical lesions	No. of cases	Cases of palsy (%)	Patients with MMT grade ≤2 (%)
Cervical spondylotic myelopathy	113	9 (7.9)	5 (4.4)
OPLL	62	6 (9.7)	5 (8.1)
Disc herniation	2	0	0
Cervical spondylotic amyotrophy	16	2 (12.5)	0
Cervical spondylotic radiculopathy	6	0	0
Total	199	17 (8.5)	10 (5.0)

MMT Manual muscle test, *OPLL* ossification of posterior longitudinal ligament

cervical lesions were cervical spondylotic myelopathy (CSM) in 113 cases, cervical ossification of the posterior longitudinal ligament (OPLL) in 62 cases, cervical spondylotic amyotrophy (CSAM) in 16 cases, cervical spondylotic radiculopathy in 6 cases, and disc herniation in 2 cases. The number of fused levels was 1 for 33 cases, 2 for 40 cases, 3 for 46 cases, 4 for 71 cases, and 5 for 9 cases (Table 2). One level fusion surgery was performed by anterior cervical discectomy and fusion, and two or more levels of fusion surgery were performed by anterior cervical corpectomy and arthrodesis. Autologous iliac bone was grafted for one- or two-level fusions and an autologous fibula strut was grafted for fusion surgery of three or more levels [11]. In cases of one or two level fusion, a cervical collar was used postoperatively. In cases where three or more levels were fused, patients were immobilized with a halo vest for 8 weeks after surgery. No spinal instrumentation such as an anterior cervical plate system was used in the present series.

Clinical assessment

We defined C5 palsy as when patients showed a deterioration in muscle power of the deltoid or biceps brachii by at least one grade in the manual muscle test (MMT) without aggravation of lower extremity function. We evaluated the laterality of C5 palsy, onset of pain, onset of weakness, and time course of any MMT grade change. The Japanese Orthopaedic Association (JOA) score was used to evaluate the severity of myelopathy [9].

Table 2 Fused levels and C5 palsy incidence

No. of levels fused	Fused level	No. of patients	Cases of palsy (%)	Patients with MMT grade ≤2 (%)
1	C3–C4	13	0	0
	C4–C5	5	0	0
	C5–C6	11	0	0
	C6–C7	3	0	0
	C7–T1	1	0	0
	Subtotal	33	0	0
2	C3–C5	14	0	0
	C4–C6	14	2 (14.3)	1 (7.1)
	C5–C7	12	0	0
	Subtotal	40	2 (5.0)	1 (2.5)
3	C2–C5	1	0	0
	C3–C6	27	4 (14.8)	2 (7.4)
	C4–C7	15	0	0
	C5–T1	3	0	0
	Subtotal	46	4 (8.7)	2 (4.3)
4	C2–C6	6	2 (33.3)	2 (33.3)
	C3–C7	63	9 (14.3)	5 (7.9)
	C4–T1	2	0	0
	Subtotal	71	11 (15.5)	7 (9.9)
5	C2–C7	9	0	0

Radiological assessment

Using lateral cervical radiographs and images from computed tomography (CT), CT myelography, and magnetic resonance (MR), we identified the most stenotic level of the spinal column. High signal changes (HSCs) of spinal cord were assessed on preoperative and postoperative T2-weighted MR images. Anterior shift of the spinal cord after surgery at the most stenotic level of the spinal column was calculated on axial images from preoperative and postoperative CT myelography (Fig. 1a). Lateral tilting of grafted bone was assessed on postoperative cervical anterior–posterior radiographs (Fig. 1b).

Statistical analysis

Fisher's exact probability test was applied for statistical analysis. $P < 0.05$ was considered significant.

Results

Postoperative C5 palsy was found in 17 of 199 cases (8.5%). In CSM cases, 9 of 113 patients (7.9%) developed C5 palsy. Similarly, 6 of 62 OPLL patients (9.7%) and 2 of 16 CSAM patients (12.5%) developed C5 palsy. No patients with cervical spondylotic radiculopathy and disc herniation developed C5 palsy (Table 1). When the degree of paralysis was restricted to MMT grade ≤2, ten patients (5.0%) showed paralysis. They included five CSM cases (4.4%) and five OPLL cases (8.1%) (Table 1).

No patient showed an incidence of C5 palsy where there was only one level fusion. C5 palsy was found in 2 (5.0%) of 40 patients who underwent 2 level fusion, 4 (8.7%) of 46 patients with 3 level fusion, and 11 (15.5%) of 71 patients with 4 level fusion. No case of C5 palsy was found in nine patients with 5 level fusion (Table 2). To summarize, 2 of 73 patients (2.7%) developed C5 palsy after fusion at 1 or 2 levels, while 15 of 126 patients (11.9%) developed the palsy after 3 or more levels of fusion. The incidence of the palsy after fusion at three or more levels was significantly higher than that when only one or two levels were fused.

No additional surgery was performed for the postoperative C5 palsy cases. When C5 palsy developed, we stimulated the patient's deltoid muscle using low-frequency sound waves and performed both active and passive shoulder range of motion (ROM) exercises to prevent contracture of the patient's shoulder.

JOA scores of 17 patients who developed C5 palsy are shown in Table 3. All of the 17 patients showed a recovery from their myelopathy. Their extent of recovery ranged from 27.6 to 100% (average 71.2%) (Table 3).

The onset and prognosis of C5 palsy in the 17 patients are summarized in Table 4. Sixteen of 17 patients had radiating neck and shoulder pain prior to their muscle weakness. Pain was recognized 1–7 days (average 3.6 days) after surgery. Muscle weakness developed 2–23 days (average 7.2 days) after surgery. Twelve patients completely recovered from their C5 palsy. However, the recovery was incomplete in three patients, and two patients with OPLL showed no recovery. No patient showed preoperative weakness of the deltoid or biceps brachii. All seven patients who had an MMT grade ≥3 at the onset of their C5 palsy showed full recovery. Among ten patients with MMT grade ≤2 at the onset, five showed recovery to MMT grade 5, and three to MMT grade 4; two patients did not show any recovery and still had an MMT grade ≤2 at their follow-up. There was 0.8–15 months to maximum recovery (average 2.8 months). No patient with C5 palsy developed a new sensory deficit at the C5 dermatome area.

The radiological characteristics of the 17 patients are summarized in Table 5. In all of the 17 patients, the most stenotic level of their spinal column included the C3–C4 or C4–C5 levels. On MR images, HSCs were found at C3–C4 or C4–C5 levels in 12 patients. When restricted to the ten patients who had an MMT grade ≤2 at the onset, nine of the ten had such HSCs (Table 5). Anterior shift of the spinal cord ranged 0.8–5.2 mm. Tilting of the grafted bone ranged from 0° to 14° (Table 5).

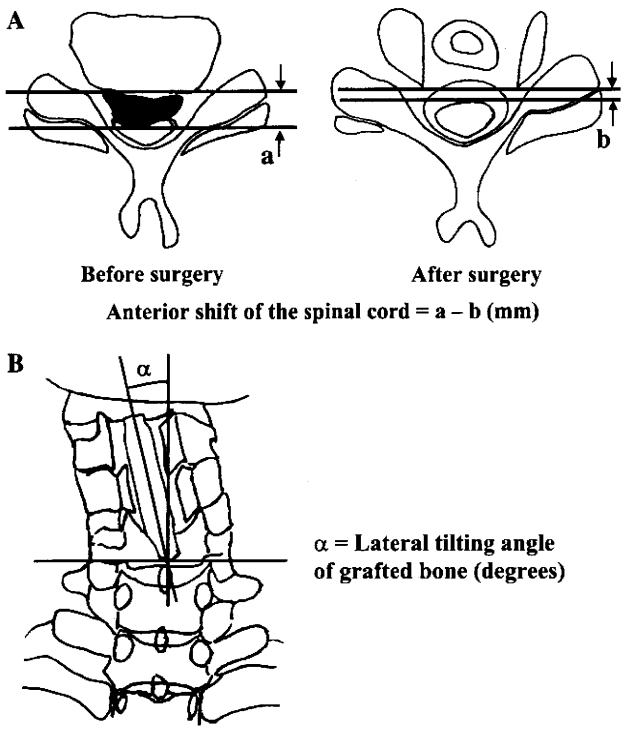

Fig. 1 Schematic drawings of anterior shift of the spinal cord (**a**) and lateral tilting of the grafted bone (**b**)

Table 3 JOA score of 17 patients who developed C5 palsy

Patient no.	Age (years)/sex	Lesion	Levels fused (*n*)	FU period	JOA score						Recovery rate (%)
					Pre-op			At FU			
					Total	M-UE	M-LE	Total	M-UE	M-LE	
MMT grade ≤2											
1	37/F	CSM	C4–C6 (2)	9y 3m	16	4	4	17	4	4	100
2	26/M	CSM	C3–C6 (3)	10y 10m	13	3	3	17	4	4	100
3	50/M	CSM	C3–C6 (3)	1y	13	3	3	15	3	4	50
4	73/F	CSM	C2–C6 (4)	9y 10m	8	1	1	15	4	3	77.8
5	62/M	OPLL	C3–C7 (4)	4y 4m	7	1	1	16	4	3	90
6	52/M	CSM	C3–C7 (4)	5y 1m	10	2	3	15	3	4	71.4
7	61/M	OPLL	C3–C7 (4)	1y	2.5	0	0	6.5	1	1	27.6
8	70/F	OPLL	C3–C7 (4)	5y 6m	10	3	2	12.5	3.5	2.5	35.7
9	71/M	OPLL	C2–C6 (4)	5y 6m	8.5	2	1.5	13	4 (−2)	2	64.7
10	64/M	OPLL	C3–C7 (4)	10y	10.5	2.5	2	13	4 (−2)	3	38.5
MMT grade ≥3											
11	73/M	CSAM	C4–C6 (2)	9y 10m	15	4 (−2)	2	17	4	4	100
12	70/M	CSAM	C3–C6 (3)	5y 2m	16	4 (−1)	4	17	4	4	100
13	65/F	CSM	C3–C7 (4)	10y 3m	12	4	4	17	4	4	100
14	44/F	CSM	C3–C7 (4)	10y	13	3	3	15	4	4	50
15	64/M	CSM	C3–C7 (4)	5y 5m	10.5	2	2	13	3	2	38.5
16	48/M	CSM	C3–C7 (4)	4y 6m	14	3	3	16	4	4	66.7
17	65/M	OPLL	C3–C7 (4)	4y 2m	13.5	3	3	17	4	4	100
Mean ± SD					11.3 ± 3.5	2.4 ± 1.1	2.5 ± 1.1	14.8 ± 2.7	3.4 ± 0.9	3.3 ± 1.0	71.2 ± 27.0

JOA score Japanese Orthopaedic Association score for cervical myelopathy, *FU* follow-up, *M-UE* motor function score of upper extremity, *M-LE* motor function score of lower extremity, *CSM* cervical spondylotic myelopathy, *CSAM* cervical spondylotic amyotrophy

Case presentation

Case 2

A 26-year-old man presented with myelopathy due to cervical spondylosis and a preoperative JOA score of 13 points. A preoperative roentgenogram showed kyphotic alignment of his cervical spine (Fig. 2a). Anterior corpectomy of C4 and C5 and arthrodesis at C3–C6 were performed using an autologous fibula strut. After surgery, the fibula strut tilted toward his right side. The lateral tilt angle of the grafted fibula was 5° immediately after surgery (Fig. 2b), and 14° on the seventh day (Fig. 2c). On the seventh day, subluxation of the right uncovertebral joint of C4–C5 was shown on an anterior–posterior roentgenogram (Fig. 2c, arrow). Postoperative CT 8 weeks after surgery showed foraminal stenosis of C4–C5 on the right side (Fig. 2d, e, arrows). On the third day after surgery, he had neck and right-shoulder pain. On the fourth day, his right deltoid muscle began to deteriorate to MMT grade 2. On the 70th postoperative day, the palsy naturally recovered to MMT grade 5. In this case, we speculated that the cause of the C5 palsy was foraminal stenosis at the right C4–C5.

Case 9

A 71-year-old man presented with cervical myelopathy caused by OPLL. A preoperative roentgenogram showed mixed type OPLL from C1 to C6 (Fig. 3a). His preoperative JOA score was 8.5 points. Preoperative T2-weighted MR images showed compression of the spinal cord and HSCs at C3–C4 and C4–C5 levels (Fig. 3b). He underwent anterior corpectomy of C3, C4, and C5 and arthrodesis using an autologous fibula strut from C2 to C6 (Fig. 3c). On the first day after surgery, he complained of left shoulder pain. On the fifth day, his left deltoid muscle power had deteriorated to MMT grade 2. On the 22nd postoperative day, his right deltoid muscle power had deteriorated to MMT grade 2. Eight months later, muscle power in his right upper extremities recovered to MMT grade 5. However, his left deltoid did not show any recovery, and was still MMT grade 2 at a final follow-up of 5 years and 6 months after surgery. Postoperative T2-weighted MR images showed HSCs at C3–C4 and C4–C5 levels (Fig. 3d, e). High intensity signal changes were detected in the gray matter of the spinal cord. Axial views of a CT myelogram revealed an excessive 5.2 mm

Table 4 Onset and prognosis of C5 palsy

Patient no.	Impaired muscle	Laterality	Onset of pain (days)	Onset of weakness (days)	MMT grade			Months to recovery	Degree of recovery
					Pre-op	At onset	At FU		
MMT grade ≤2									
1	D, B	Rt	2	13	5	2	5	3	Complete
2	D, B	Rt	3	4	5	2	5	1.8	Complete
3	D, B	Blt	3	8	5	2	5	0.8	Complete
4	D, B	Rt	3	6	5	2	5	2	Complete
5	D, B	Lt	3	4	5	2	5	1	Complete
6	D, B	Lt	7	12	5	2	4	6	Incomplete
7	D, B	Rt	3	4	5	2	4	1.8	Incomplete
8	D, B	Lt	–	2	5	2	4	15	Incomplete
9	D, B	Blt	1	5	5	2	2	NR	No
10	D, B	Lt	5	5	5	1	1	NR	No
MMT grade ≥3									
11	D, B	Rt	3	7	5	4	5	1	Complete
12	D, B	Lt	2	3	5	3	5	1.3	Complete
13	D, B	Rt	7	23	5	4	5	2	Complete
14	D, B	Lt	1	2	5	4	5	1	Complete
15	D, B	Lt	3	10	5	3	5	0.8	Complete
16	D, B	Lt	5	5	5	4	5	1	Complete
17	D, B	Rt	7	10	5	3	5	3	Complete
Mean ± SD			3.6 ± 2.0	7.2 ± 5.3	5.0 ± 0	2.6 ± 0.9	4.4 ± 1.2	2.8 ± 3.6	

D Deltoid, *B* biceps, *Rt* right, *Lt* left, *Blt* bilateral, *NR* not recovered

Table 5 Radiological characteristics of 17 patients who developed the C5 palsy

Case no.	Most stenotic level	T2W HSC	Anterior shift of spinal cord (mm)	Lateral tilting of grafted bone (°)
MMT ≤2				
1	C4/5	–	NA	1
2	C4/5, C5/6	C4/5	1.9	14
3	C3/4	C3/4	3.4	2
4	C4/5	C4/5	NA	11
5	C4/5	C4/5	3.6	2
6	C3/4, C4/5	C3/4, 4/5	1.0	1
7	C4/5, C5/6	C4/5, 5/6	NA	NA
8	C3/4	C3/4	1.2	0
9	C3/4, C4/5	C3/4, 4/5	5.2	2
10	C3/4	C3/4, 4/5	1.0	6
MMT ≥3				
11	C4/5	–	2.0	1
12	C4/5	–	1.9	1
13	C4/5, C5/6	–	NA	6
14	C4/5, C5/6	C4/5, 5/6	0.8	0
15	C4/5	C4/5	1.9	1
16	C3/4	C3/4	4.3	1
17	C3/4, C4/5	–	3.6	2

T2W HSC High signal change in T2-weighted magnetic resonance images, *NA* not assessed

anterior shift of the spinal cord (Fig. 3f). In this case, we suggest that the cause of C5 palsy was an excessive anterior shift of the spinal cord and tethering of nerve roots. In addition, we speculate that there was asymptomatic damage of the anterior horn cells in the spinal cord, which had existed before surgery and may have also participated in the development of C5 palsy.

Discussion

Sakaura et al. [12] have reviewed the literature regarding postoperative C5 palsy published from 1986 to 2002. They reported that the incidence of C5 palsy after cervical posterior decompression surgery was 4.7% on average, ranging from 0 to 30.0%. On the other hand, its incidence after ASF for cervical lesions was 4.3% on average, ranging from 1.6 to 12.1%, although the number of reports regarding anterior surgery was smaller than that for posterior surgery. To the best of our knowledge, there have been only five large studies on C5 palsy that analyzed more than 100 cases of cervical anterior surgeries [4, 5, 7, 14, 19]. We have summarized their data in Table 6. According to these studies, the incidence of C5 palsy after anterior surgery differed considerably from 3.2 to 9.1%, depending on the institution where they were conducted. The major difference seems to be because a unified definition of C5 palsy has not yet been clearly made. In the present study, we defined C5 palsy as a deterioration of upper extremity motor function by at least one grade in a standard MMT without aggravation of lower extremity function. According to our criteria, the incidence of C5 palsy after the surgeries described was 8.5% in our institute. When the palsy was restricted to a MMT grade ≤ 2, the incidence was 5.0%. When reporting C5 palsy, therefore, it is essential to define it clearly.

Regarding the correlation between the fused levels and the incidence of C5 palsy, Ikenaga et al. [7] described that

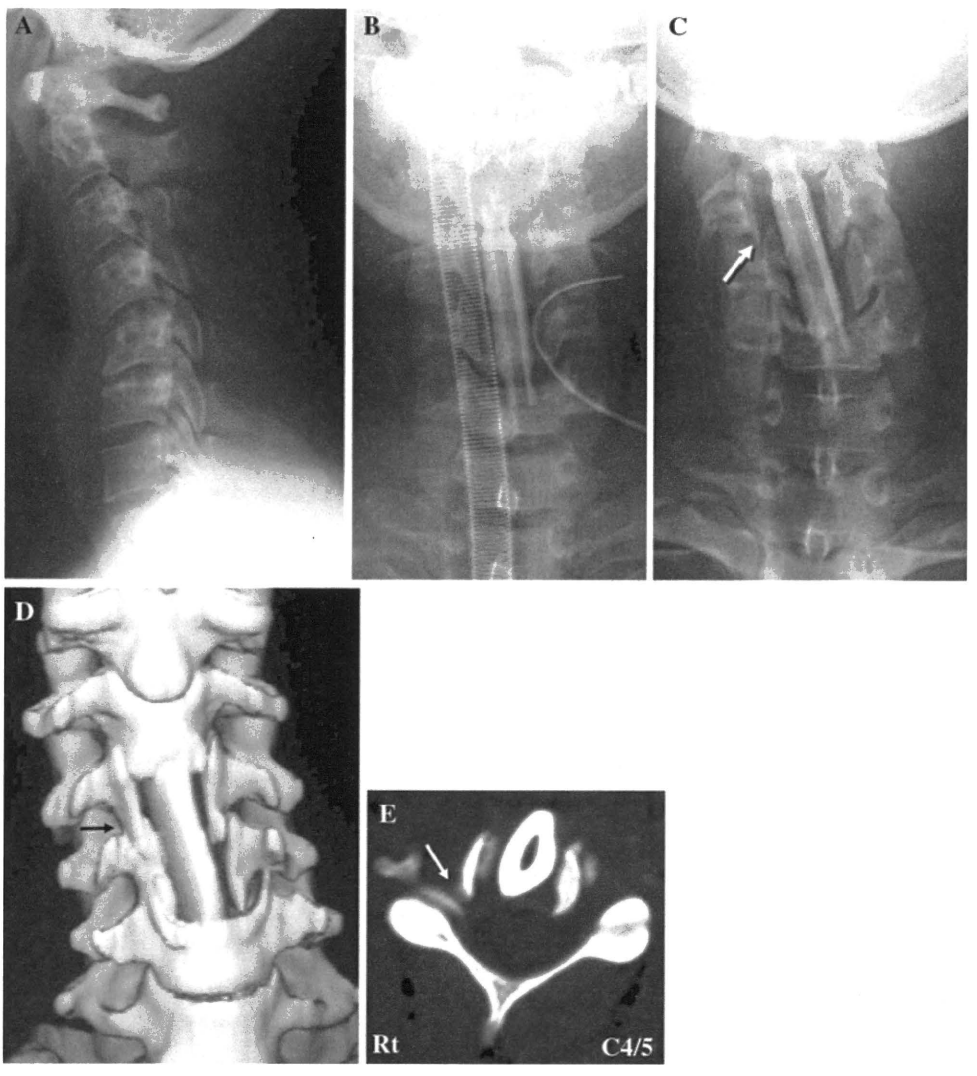

Fig. 2 Case 2. A preoperative lateral cervical radiograph showing kyphotic alignment of the cervical spine (**a**). Anterior–posterior views of cervical radiographs just after anterior corpectomy of C4 and C5 and arthrodesis at C3–6 (**b**) and on the seventh day after surgery (**c**). **c** The lateral tilting angle of the grafted fibula was 14° and the right C4–C5 uncovertebral joint was subluxed (*arrow*). Front view of three-dimensional CT (**d**) and axial CT images at the level of C4–C5 (**e**). CT 8 weeks postoperatively showing a subluxed right C4–C5 uncovertebral joint and stenosis of the right C4–C5 foramen (*arrows*)

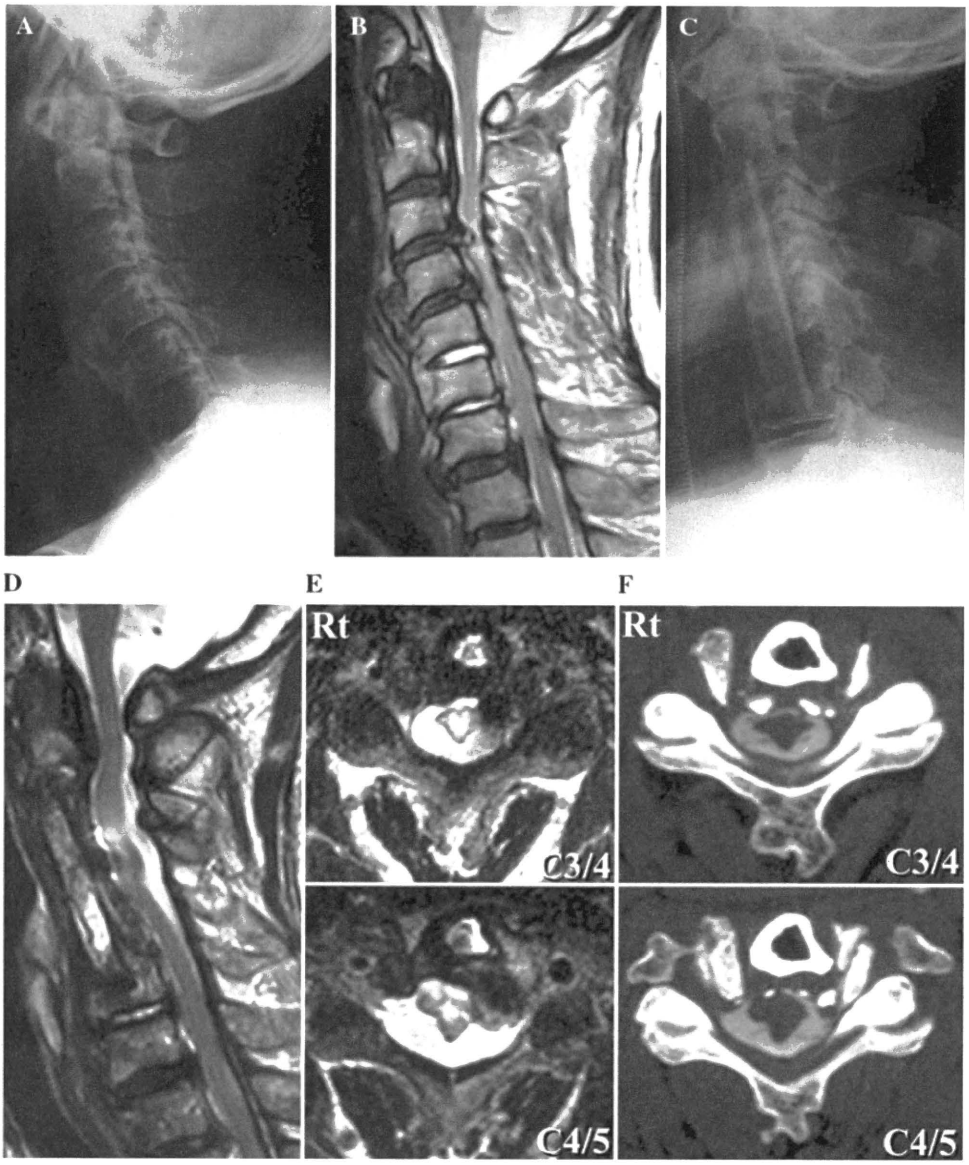

Fig. 3 Case 9. A preoperative lateral cervical radiograph (**a**) showing mixed type OPLL from C1 to C6. A midsagittal T2-weighted MR image (**b**) showing severe compression of the spinal cord and HSCs at C3–C4 and C4–C5 levels. A postoperative lateral cervical radiograph shows anterior corpectomy of C3, C4, and C5 and arthrodesis at C2–C6 (**c**). T2-weighted MR midsagittal (**d**) and axial views at C3–C4 and C4–C5 (**e**) and a CT myelogram (**f**) showing an excessive anterior shift of the spinal cord at C3–C5. **e** HSCs in the gray matter at the C3–C4 and C4–C5 levels

no patient developed C5 palsy after fusion of 1 or 2 levels, and 18 of 362 patients (5.0%) developed the palsy after fusion of 3 or more levels. Greiner-Perth et al. [4] described that 3 of 65 (4.6%) patients developed C5 palsy after fusion of 1 or 2 levels, and 7 of 56 patients (12.5%) developed the palsy after fusion of 3 or more levels. In the present study, we found that 2 of 73 patients (2.7%) developed C5 palsy after fusion of 1 or 2 levels, and 15 of 126 patients (11.9%) developed the palsy after fusion of 3 or more levels. The present results, taken together with those previous reports collectively indicate that the more levels involved in anterior cervical decompression, the more likely the occurrence of C5 palsy.

Previous reports showed that C5 palsy generally had a good prognosis for neurologic and functional recovery. However, irreversible cases of C5 palsy were also reported. Ikenaga et al. [7] reported 7 cases of partial recovery in 18 patients with cases of C5 palsy, and Greiner-Perth et al. [4] reported two cases of partial recovery in ten patients with cases of C5 palsy. In the present study, among 17 patients with cases of C5 palsy, all 7 cases with an MMT grade ≥ 3 at the onset showed complete recovery. However, only five out of ten patients with cases of C5 palsy and an MMT grade ≤ 2 palsy showed complete recovery, three patients recovered incompletely, and two patients remained unchanged at the latest follow-up. For the two palsy patients with no recovery, both had OPLL, and deteriorated to MMT grades 1 and 2 at the onset, and had HSCs on T2-weighted MR images at both C3–C4 and C4–C5 levels. This suggests that, when patients with compression myelopathy and an ossified mass at C3–C4 and C4–C5 levels develop C5 palsy with an MMT grade ≤ 2 after ASF,

Table 6 Incidence and prognosis of C5 palsy after anterior decompression surgery for cervical lesions

Report (year)	No. of patients	No. of cases of palsy (% of patients)	Cases of palsy with MMT grade ≤2 (% of all cases)	1 and 2 levels fusion		3 and more levels fusion		Recovery of the palsy (*n*)	
				No. of cases	Cases of palsy (%)	No. of cases	Cases of palsy (%)	Complete	Incomplete and none
Yonenobu (1991)	204	8 (3.9)	8 (3.9)	ND	0	ND	8	0	8
Saunders (1995)	176	16 (9.1)	ND	20	1 (5.0)	156	15 (9.6)	ND	ND
Greiner-Perth (2005)	121	10 (8.2)	3 (2.5)	65	3 (4.6)	56	7 (12.5)	8	2
Ikenaga (2005)	563	18 (3.2)	7 (1.2)	201	0 (0)	362	18 (5.0)	11	7
Hasegawa (2007)	424	22 (5.2)	ND	ND	ND	ND	ND	ND	ND
Present report	199	17 (8.5)	10 (5.0)	73	2 (2.7)	126	15 (11.9)	12	5

ND Not described

favorable recovery from their palsy may not necessarily be expected. We speculate that certain pre-existing asymptomatic damage to the anterior horn cells in the gray matter of the spinal cord at the C3–C4 and C4–C5 levels may participate in the poor neurological improvement.

Over the past few years, hypotheses regarding the etiology of postoperative C5 palsy have been proposed by many authors. Most of the speculation regarding the etiology is roughly classified into two groups. One hypothesis is that C5 palsy is caused by nerve root damage. Some authors proposed that this might be caused by direct injury to the nerve root [6]. They proposed that surgical instruments could injure neural tissue. However, such a hypothesis cannot explain why many cases of C5 palsy develop several days after the surgery. Others have presumed that tethering of nerve roots might cause C5 palsy as the result of a shift of the spinal cord in association with anchoring of the nerve root [6, 16]. Saunders [14] hypothesized that narrowing of the width of anterior decompression decreased the anterior shift of the spinal cord and the traction of the roots, and consequently prevented the development of radiculopathy after corpectomy. Ikenaga et al. [7] reported the possibility that the extent of anterior dural expansion might have enhanced the incidence of C5 palsy after anterior surgery.

The other hypothesis is that C5 palsy might be caused by a spinal cord disorder. Chiba et al. [1] analyzed C5 palsy after laminoplasty, and proposed that postoperative upper extremity paresis might be caused by a deterioration of the gray matter and proposed that local reperfusion injury in the spinal cord could be the pathomechanism. Hasegawa et al. [5] analyzed C5 palsy after laminoplasty and anterior decompression surgery, and commented that postoperative upper extremity palsy following cervical decompression surgery might result from a transient and localized spinal cord lesion caused by the decompression of a chronic compressive cervical cord disorder. However, both theories regarding the nerve root and spinal cord remain hypothetical because of non-availability of reliable evidence for verifying their proposed hypothetical mechanism.

In the present study, 16 of 17 patients with cases of C5 palsy presented neck and shoulder pain prior to the onset of muscle weakness. This finding supports the theory that postoperative C5 palsy is caused by a certain nerve root lesion. In Case 9, excessive anterior shift of the spinal cord was observed. Because shoulder pain was detected prior to the muscle weakness in this case, it is possible that tethering of C5 nerve roots occurred bilaterally at the posterior edge of excavated vertebra, and that this caused the bilateral C5 palsy. However, the presence of HSCs on T2-weighted MR images at the C3–C4 and C4–C5 levels also provided the possibility of a spinal cord disorder as the etiology of the C5 palsy in this case.

In the present study, it is of particular interest that in all 17 patients with C5 palsy, the most stenotic level of their spinal canal included C3–C4 or C4–C5 levels. In addition, in 12 out of the 17 cases, HSCs were detected at C3–C4 or C4–C5 levels. When restricted to the ten cases of palsy with an MMT grade ≤2, nine cases showed such HSCs. Because all of the 17 patients with C5 palsy had normal muscle power of deltoid and biceps preoperatively, we suggest that asymptomatic damage of anterior horn cells at the gray matter of the spinal cord pre-existed in these cases. Taking all these findings into account, we propose a "double lesion" hypothesis for the development of C5 palsy after ASF for cervical lesion as follows: the pre-existing asymptomatic damage at the anterior horn cells may contribute to the development of postoperative C5 palsy, in combination with nerve root lesions caused by the foraminal stenosis or the excessive anterior shift of the spinal cord after the anterior decompression procedure. Further studies using preoperative and intraoperative electrodiagnostic techniques, such as preoperative EMG and intraoperative neuromonitoring, should provide us with useful information to support this hypothesis.

In conclusion, when patients having spinal cord lesions at C3–C4 and C4–C5 levels undergo multilevel ASF, we should be alert to the possible occurrence of postoperative C5 palsy. To avoid the development of C5 palsy, further improvement of surgical techniques is required to minimize the damage to the nerve roots that may occur subsequent to anterior decompression procedures.

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Clinical Study

Static versus dynamic factors for the development of myelopathy in patients with cervical ossification of the posterior longitudinal ligament

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ABSTRACT

We studied 27 patients with cervical ossification of the posterior longitudinal ligament (OPLL) but no clinical symptoms of myelopathy. We investigated the occupation ratio of the spinal canal by OPLL with cervical radiographs, assessed the morphological types of OPLL, and measured the segmental range of motion (ROM) at the level of maximum cord compression on flexion and extension radiographs. Patients were classified as having continuous-type OPLL (17 patients), mixed-type OPLL (seven patients), or segmental-type OPLL (three patients). The segmental ROM was negatively correlated with the OPLL occupation ratio ($r = -0.49$, $p < 0.01$). No patient developed myelopathy during the study period. Three patients with massive OPLL did not develop myelopathy and the mobility of their cervical spine was highly restricted, suggesting that dynamic factors such as the segmental ROM preferentially contribute to the development of myelopathy in patients with cervical OPLL. Thus, by controlling the dynamic factors (hypermobility), we might be able to reduce neurological deterioration in patients with cervical OPLL.

1. Introduction

Ossification of the posterior longitudinal ligament (OPLL) of the cervical spine results in static compression of the spinal cord that causes myelopathy.[1–3] Both anterior and posterior surgical approaches have been performed for cervical myelopathy due to OPLL,[1,2,4,5] and several studies have shown that the anterior surgical approach results in a better surgical outcome when the occupation ratio by OPLL is large,[2,4] indicating that complete removal of the static compression factor is important in the treatment of myelopathy.

Dynamic factors, such as the mobility of the spinal column, are also important in the development of myelopathy when a considerable degree of the canal is occupied by OPLL.[6–8] However, the contribution of dynamic factors to the development of myelopathy in cervical OPLL patients has not been fully determined. In the present study, we radiographically and clinically evaluated patients who had cervical OPLL but no symptoms of myelopathy, and analyzed the contribution of static and dynamic factors to the development of myelopathy.

2. Materials and methods

From April 2000 through March 2007, 27 patients with cervical OPLL (11 men, 16 women, mean age at first diagnosis 63.3 years [range, 37–78 years]) in whom the space available for the spinal cord (SAC) (Fig. 1A) at the cervical spine was ⩽ 12 mm visited our institute for initial consultation. When the patients were diagnosed with cervical OPLL, they had no clinical symptom of myelopathy. The mean follow-up period for all the patients was 59 months (range, 12–95 months).

Using cervical radiographs, we measured the occupation ratio of the spinal canal by OPLL [(thickness of OPLL/anteroposterior diameter of the bony spinal canal) × 100][8] (Fig. 1A). Morphologically, patients were classified as continuous, mixed, segmental, or localized OPLL types according to the criteria of the Japanese Investigation Committee on the Ossification of the Spinal Ligaments.[9] We also evaluated the segmental range of motion (ROM) at the maximum cord compression level based on flexion and extension radiographs[8] (Fig. 1B), and on T2-weighted MRI we examined the high signal change in the spinal cord. The patients' clinical course was assessed using the Japanese Orthopaedic Association (JOA) scoring system for cervical myelopathy.[8] On physical examination, we assessed the patients' deep tendon reflexes of the lower extremities and the Babinski reflex.

For statistical analysis we applied the Fisher's exact probability test and Pearson's correlation test. A $p < 0.05$ was considered sig-

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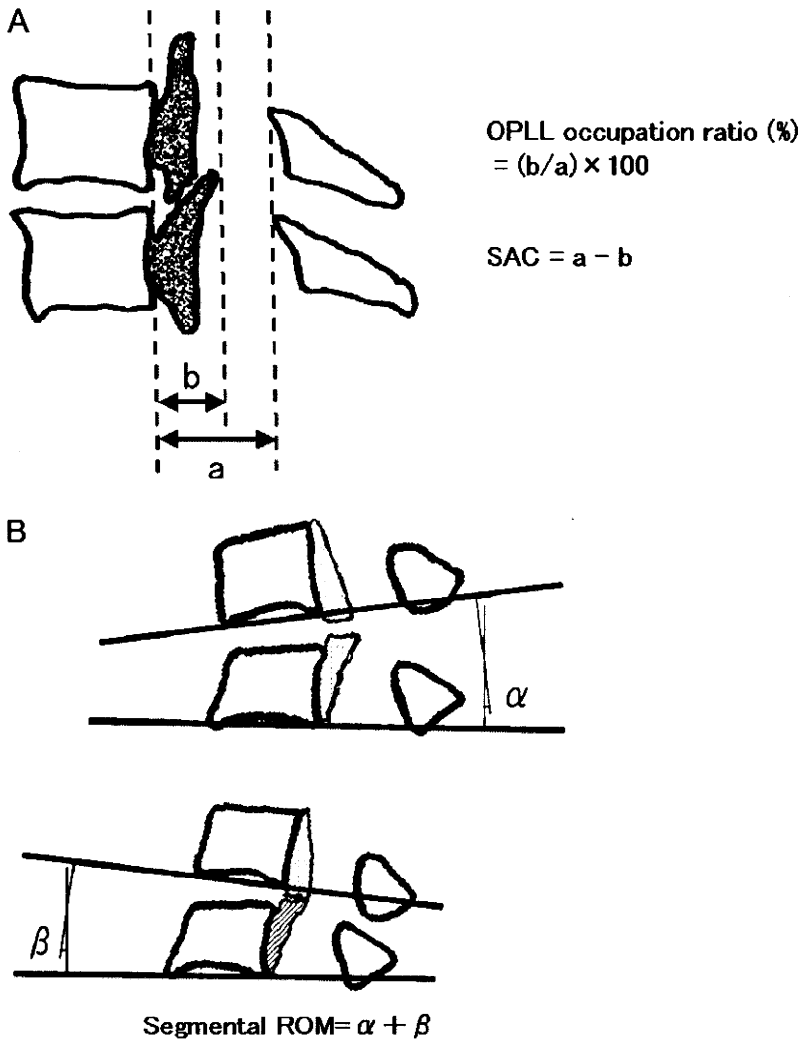

Fig. 1. Schematics showing the radiographic parameters assessed. (A) The ossification of the posterior longitudinal ligament (OPLL) occupation ratio and space available for the spinal cord (SAC) were measured from lateral cervical radiographs. (B) The segmental range of motion (ROM) at the maximum cord compression level was measured from flexion and extension radiographs.

Table 1
Radiographic findings for 27 patients with ossification of the posterior longitudinal ligament

	Type of OPLL[†]		
	Continuous	Mixed	Segmental
No. patients (*n*)	17[a]	7	3
SAC (mm)	7.6 ± 2.3 (3–12)	7.8 ± 1.0 (7–9)	9.4 ± 2.8 (7–12)
Occupation ratio (%)	42.5 ± 11.1 (25–64)	39.4 ± 7.6 (29–50)	27.7 ± 3.3 (25–31)
Segmental ROM (°)	2.4 ± 3.0 (0–9)	4.9 ± 2.9 (1–10)	9.7 ± 4.5 (5–14)

OPLL = ossification of the posterior longitudinal ligament, occupation ratio = occupation ratio of spinal canal by OPLL, ROM = range of motion (intervertebral disc mobility at the maximum cord compression level), SAC = space available for the spinal cord (see Fig. 1).
[†] Mean ± standard deviation (range).
[a] Statistically different from mixed-type OPLL and segmental-type OPLL ($p < 0.05$).

nificant. Data are presented as the mean ± standard deviation of the mean.

3. Results

3.1. Radiographic findings

Of the 27 patients analyzed, 17 (63%) were classified with the continuous type, 7 (26%) with the mixed type, and 3 (11%) with the segmental type of OPLL (Table 1), and the incidence of patients with continuous-type OPLL was significantly higher than that for the other types ($p < 0.05$).

The mean OPLL occupation ratio was 39.8% for all types. It was 42.5% in patients with continuous-type OPLL, 39.4% in patients with mixed-type OPLL, and 27.7% in patients with segmental-type OPLL (Table 1). The mean segmental ROM at the level of maximum cord compression was 3.8 degrees. It was 2.4 degrees in patients with continuous-type OPLL, 4.9 degrees in patients with

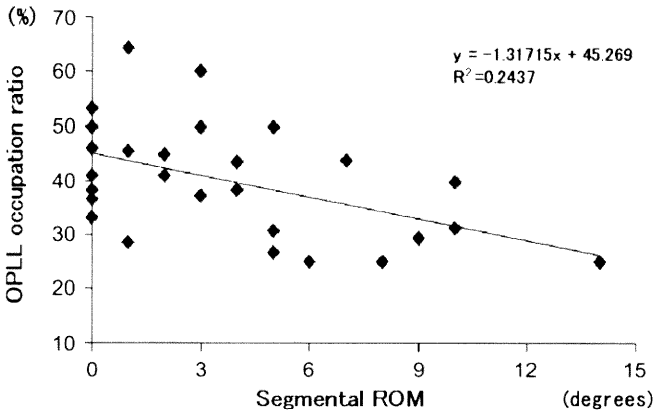

Fig. 2. The relationship between the ossification of the posterior longitudinal ligament (OPLL) occupation ratio and the segmental range of motion (ROM) in the cervical spine of 27 patients showing that as OPLL occupation ratio increased, the ROM decreased ($y = -131715x + 45.269$ [$r = -0.49$; $p < 0.01$]).

Table 2
Characteristics of three patients with an ossification of the posterior longitudinal ligament occupation ratio greater than 50%

	Patient 1	Patient 2	Patient 3
Age (yrs)/Gender	65/F	37/F	67/M
Sites of OPLL	C2-C5	C2-C7	C4-C6
Maximum cord compression level	C4-C5	C5-C6	C4-C5
Type of OPLL	Continuous	Continuous	Continuous
Occupation ratio (%)	64	60	53
SAC (mm)	3	3	6
Segmental ROM (degrees)	1	3	0
T2-high signal change	None	None	None
Deep tendon reflexes	Hyper	Hyper	Normal
Babinski reflex	+	+	+

OPLL = ossification of the posterior longitudinal ligament, occupation ratio = occupation ratio of spinal canal by OPLL, ROM = range of motion (intervertebral disc mobility at the maximum cord compression level), SAC = space available for the spinal cord, T2-high signal change = high signal change in T2-weighted MRI, yrs = years, Hyper = hyperactive (See Fig. 1).

mixed-type OPLL, and 9.7 degrees in a patient with segmental-type OPLL (Table 1). A significant negative correlation was detected between the segmental ROM and the OPLL occupation ratio by Pearson's correlation test ($r = -0.49$, $p < 0.01$) (Fig. 2). Three patients had an OPLL occupation ratio >50% and a SAC of ≤6 mm (Table 2). All three patients had continuous-type OPLL, and their segmental ROM ranged from 0 degrees to 3 degrees.

Using T2-weighted MRI, we were able to analyze the signal intensity of the spinal cord at the maximum compression level in 18 of the 27 patients. None of the patients showed high signal change in the spinal cord.

3.2. Clinical findings

At first diagnosis, none of the 27 patients showed any motor, sensory, or bladder dysfunction, and their JOA scores were 17 points (full score). Regarding their physical examination, the deep tendon reflexes of the lower extremities were hyperactive in 10 of 27 patients (37%) and the Babinski reflex was positive in 6 of 27 patients (22%). During follow-up, none of the patients developed myelopathy, and the incidence of hyperactive deep tendon reflex and Babinski reflex remained unchanged.

3.3. Illustrative patient

3.3.1. Patient 1 (OPLL occupation ratio >50%)

A 65-year-old woman was diagnosed with continuous-type OPLL from C2 to C5, and the maximum cord compression was at C4/5. Her OPLL occupation ratio was 64% and the SAC was 3 mm. The patient's segmental ROM was 1 degree at the C4/5 level (Fig. 3A, B), indicating that she had massive OPLL, but no segmental hypermobility. Her deep tendon reflexes of the lower extremities were hyperactive, and her Babinski reflex was positive. T2-weighted MRI showed anterior compression of the spinal cord from C3/4 to 4/5, but no high signal change in the spinal cord (Fig. 3C, D). In spite of massive OPLL, the patient did not develop myelopathy, possibly due to the lack of dynamic factors (no hypermobility) at the level of cord compression.

4. Discussion

Static compression factors are important in the development of myelopathy in cervical OPLL patients, including the occupation ratio of the spinal canal by OPLL and the residual space available for the spinal cord.[2,4] Iwasaki et al. reported that the surgical outcome after laminoplasty was insufficient in patients with an OPLL occupation ratio >60% and hill-shaped ossification.[1] Tani et al. reported that anterior decompression surgery was superior to laminoplasty in cervical OPLL patients when their occupation ratio was more than 50%.[4]

Recent reports have described the importance of dynamic factors, including mobility of the spinal column, in the development of myelopathy in patients who have a considerable degree of canal occupation by OPLL.[7,8,10] Matsunaga et al. analyzed 247 patients with cervical OPLL, and reported that when the SAC was <6 mm, all patients developed myelopathy, but when the SAC was >14 mm, no patients developed myelopathy. This indicates that the static compression factor preferentially contributed to the development of myelopathy. However, they also reported that when the SAC was between 6 mm and 14 mm, patients with a larger C1–C7 ROM preferentially developed myelopathy, indicating that this dynamic factor also contributed to the development of myelopathy.[7] Ogawa et al. analyzed long-term results after laminoplasty for cervical OPLL patients, and reported that patients with segmental type OPLL and a larger C2–C7 ROM had poor surgical outcomes.[10] We also previously reported that a larger segmental ROM at the level of maximum cord compression was a risk factor leading to poor surgical outcome after laminoplasty for cervical OPLL patients.[8]

In the present study, we characterized patients who had cervical OPLL but no clinical symptoms of myelopathy. The results demonstrated that even in patients with massive OPLL, myelopathy may not develop when the mobility of the cervical spine is highly restricted. We paid particular attention to three patients who had OPLL occupation ratios of more than 50% (Table 2). In spite of the remarkable static compression in these patients, myelopathy did not develop, possibly because the mobility of their cervical spine was highly restricted. These findings suggest that segmental ROM (a dynamic factor) at the level of maximum cord compression preferentially contributes to the development of myelopathy rather than static compression factors. We speculate that if we control this dynamic factor, we will be able to reduce neurological deterioration in cervical OPLL patients.

Our findings highlight that when performing surgery on cervical OPLL patients with canal stenosis we should consider not only the need for decompression of the spinal cord but also the suppression of dynamic factors. We believe that complete excision of the ossified mass using an anterior approach together with stabilization of the spinal column by a strut bone graft is theoretically the best

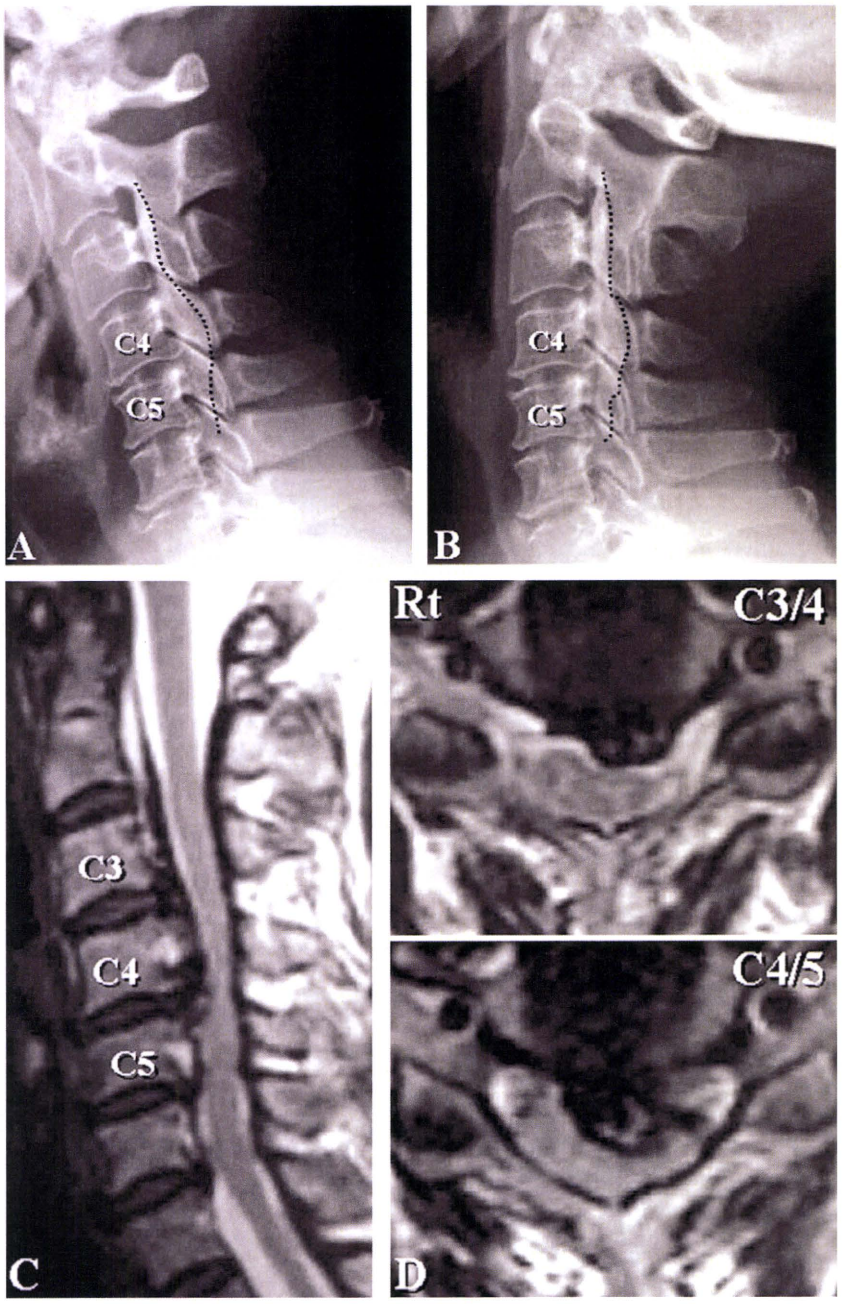

Fig. 3. (A) Flexion and (B) extension cervical radiographs at the initial visit of a 65-year-old woman with continuous type ossification of the posterior longitudinal ligament (OPLL) (patient 1) showing that the segmental range of motion was 1 degree at C4/5. T2-weighted (C) mid-sagittal and (D) axial MRI indicated anterior compression of the spinal cord at multiple levels, but high signal change was not apparent.

procedure. However, in spite of the superiority of anterior surgery, some patients, especially older patients, do not choose anterior surgery because the postoperative course is difficult to tolerate. Based on the present findings, when laminoplasty is selected for such patients, the addition of posterior instrumented fusion is desirable for stabilizing the spine and decreasing damage to the spinal cord.

Acknowledgement

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ORIGINAL ARTICLE

Posterior decompression with instrumented fusion for thoracic myelopathy caused by ossification of the posterior longitudinal ligament

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Abstract We evaluated the clinical results of posterior decompression with instrumented fusion (PDF) for thoracic myelopathy due to ossification of the posterior longitudinal ligament (OPLL). A total of 24 patients underwent PDF, and their surgical outcomes were evaluated by the Japanese Orthopaedic Association (JOA) scores (0–11 points) and by recovery rates calculated at 3, 6, 9 and 12 months after surgery and at a mean final follow-up of 4 years and 5 months. The mean JOA score before surgery was 3.7 points. Although transient paralysis occurred immediately after surgery in one patient (3.8%), all patients showed neurological recovery at the final follow-up with a mean JOA score of 8.0 points and a mean recovery rate of 58.1%. The mean recovery rate at 3, 6, 9 and 12 months after surgery was 36.7, 48.8, 54.0 and 56.8%, respectively. The median time point that the JOA score reached its peak value was 9 months after surgery. No patient chose additional anterior decompression surgery via thoracotomy. The present findings demonstrate that despite persistent anterior impingement of the spinal cord by residual OPLL, PDF can result in considerable neurological recovery with a low risk of postoperative paralysis. Since neurological recovery progresses slowly after PDF, we suggest that additional anterior decompression surgery is not desirable during the early stage of recovery.

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Keywords Thoracic myelopathy · Ossification of posterior longitudinal ligament · Kyphosis · Spinal mobility · Instrumented fusion

Introduction

Previous reports have shown that the results of surgery for thoracic myelopathy caused by ossification of the posterior longitudinal ligament (OPLL) unfavorably compare with results for cervical OPLL [12, 19]. Surgeons have employed a variety of surgical procedures to treat thoracic OPLL, including laminectomy [5], OPLL extirpation through thoracotomy [2, 4, 10], OPLL extirpation through a posterior approach [13], and circumspinal decompression [6, 15]. However, postoperative paraplegia remains a major risk [2, 4, 8–10, 13, 15]. At our institute, two patients experienced transient postoperative paraparesis after laminectomy, which resolved after the addition of posterior instrumented fusion without OPLL extirpation [16, 17]. On the basis of these two cases, we hypothesized that stabilizing the spine with instrumentation could yield a certain degree of neurological recovery even without complete OPLL extirpation. Based on this hypothesis, in 1989, we introduced the surgical procedure of posterior decompression with instrumented fusion (PDF) for patients with thoracic OPLL, in whom OPLL extirpation entailed a risk of neurological deterioration [18].

In an earlier series of ours, our patients enjoyed a considerable degree of neurological recovery following PDF despite persistent anterior impingement of the spinal cord by residual OPLL [18]. In addition, PDF was associated with an extremely low risk of postoperative paralysis and late neurological deterioration, compared with complication rates for laminectomy [5] and OPLL extirpation [2, 4].

However, the mechanisms by which PDF produced neurological recovery in thoracic OPLL patients have not yet been fully established. To better elucidate these recovery mechanisms, in the present study, we analyzed the process of neurological recovery after PDF in patients we have treated at our institute. In addition, we analyzed the contribution of thoracic kyphosis correction following PDF to neurological recovery.

Materials and methods

Patient population

From May 1989 through October 2004, a total of 24 patients (7 males and 17 females) with thoracic myelopathy due to OPLL underwent PDF at our institute. In the present study, we analyzed all 24 patients. The mean age at surgery was 54.8 years, ranging from 32 to 74 years. The mean follow-up period was 4 years and 5 months, ranging from 1 year and 2 months to 12 years and 9 months (Table 1).

Posterior decompression with instrumented fusion

Informed consent

Before performing PDF, we explained the surgical plan to the patients that laminectomy and posterior instrumented fusion would be performed as the first-step surgery and that if neurological recovery was insufficient after PDF, OPLL extirpation via thoracotomy could be performed as a second-step operation. The choice of adding anterior surgery was then left to the patient.

Surgical procedure

We principally performed laminectomy at sites where preoperative radiographic and magnetic resonance (MR) images showed disappearance of the subarachnoid space on the dorsal side of spinal cord. Regarding instrumented fusion anchors, we initially used hooks when we first introduced PDF, but more recently we have been using pedicle screws (PSs) in most cases (Table 1). After laminectomy, we used intraoperative spinal ultrasonography to assess whether the area of posterior decompression was adequate [3, 14]. After confirming the adequacy of the posterior decompression, we connected the rods to the anchors. We usually did not correct kyphosis at the rod setting but performed the fixation in situ. For bone grafting, we used spinous processes that we had extirpated before laminectomy and grafted them onto the facets and between the transverse processes.

Table 1 Key characteristics of the 24 patients who underwent posterior decompression with instrumented fusion

Case no.	Age (years)/sex	Most stenotic level	Instrumented fusion	
			Levels	Anchors
1	41/M	T9/10	T1–L2	Hooks
2	49/F	T9/10	T7–L2	Hooks
3	53/F	T6/7	T1–L1	Hooks
4	64/F	T4/5	T1–T11	Hooks
5	45/F	T8/9	T2–L2	Hooks
6	49/F	T9/10	T2–L2	Hooks
7	51/F	T9/10	T4–L2	Hooks, PSs
8	44/F	T9/10	T5–L2	Hooks, PSs
9	57/F	T11/12	T7–L4	Hooks, PSs
10	43/F	T9/10	T3–L1	Hooks, PSs
11	74/F	T6/7	T3–T10	Hooks
12	71/M	T10/11	T6–L2	Hooks, PSs
13	61/M	T4/5	T2–T8	PSs
14	52/F	T9/10	T3–T12	Hooks, PSs
15	52/F	T5/6	T2–T9	PSs
16	32/M	T10/11	T6–L1	PSs
17	66/F	T8/9	T5–T12	PSs
18	41/F	T6/7	T1–T10	Hooks, PSs
19	55/M	T9/10	T6–L2	PSs
20	55/F	T4/5	T1–T10	PSs
21	72/F	T9/10	T1–L1	PSs
22	65/F	T5/6	T1–T10	PSs
23	60/M	T4/5	T2–T10	PSs
24	64/M	T7/8	T1–T11	PSs

PS pedicle screw

Postoperative course

The patients were allowed to sit and walk with a soft orthosis 2 days after surgery. The patients generally wore the orthosis for at least 12 weeks to prevent PSs from pulling out and/or displacement of hooks.

Clinical assessment

Surgical outcomes were evaluated using the Japanese Orthopaedic Association (JOA) score (full score = 11 points) [7]. The JOA scoring system evaluates motor function of the lower extremity (0–4 points), sensory function of the trunk (0–2 points) and lower extremity (0–2 points), and bladder function (0–3 points). Recovery rates were calculated using the formula listed in Table 2 [7]. We assessed the JOA score before surgery, at 3, 6, 9, 12 months after surgery and at the final follow-up. In accordance with previous reports [18, 19], the results were ranked as either good (recovery rate $\geq$ 50%), fair