

Cervical spondylotic myelopathy in elderly people: a high incidence of conduction block at C3-4 or C4-5

T Tani, H Yamamoto, J Kimura

Abstract

Objectives—To precisely localise the site of conduction block in elderly patients with cervical spondylotic myelopathy in the presence of multilevel compression shown by MRI.

Methods—A total of 44 patients aged 65 and older underwent serial intervertebral recording of spinal somatosensory evoked potentials (SSEPs) from either the intervertebral disc or the ligamentum flavum after epidural stimulation. The site of conduction block identified by abrupt reduction in size of the negative peak was designated as the 0 level with the other levels numbered in order of distance assigning a minus sign caudally.

Results—A single site of focal conduction block was disclosed in 42 patients, 23 (55%) at C3-4, 17 (40%) at C4-5, and two (5%) at C5-6. At these levels (0), the amplitude of the negative component was reduced ($p < 0.0001$) to 29% and the area to 22%, with a concomitant increase ($p < 0.0001$) of the initial positive component to 150% in amplitude and 293% in area as compared to the -2 level which was taken as the baseline (100%).

Conclusions—A high incidence (95%) of focal conduction block at C3-4 or C4-5 with normal conduction at C5-6 and C6-7 characterises cervical spondylotic myelopathy in elderly people. Incremental SSEP studies documenting the site of conduction block will help exclude clinically silent cord compression, directing the surgical intervention to the appropriate level of concern.

(J Neurol Neurosurg Psychiatry 1999;66:456-464)

Keywords: spinal somatosensory evoked potential, conduction block, cervical spondylotic myelopathy, elderly people

Cervical spondylotic myelopathy, the commonest non-traumatic spinal cord disorder in Japan, has been extensively investigated electrophysiologically using surface recording. Studies of somatosensory evoked potentials (SEPs), disclosing more abnormalities with stimulation of the lower than upper limb nerves, suggest a greater susceptibility of the longer axons to compression.^{1,2} Multisegmental upper limb nerve stimulation shows a high incidence of abnormalities of N13 cervical potential and P14 far field potential, indicating segmental involvement of the dorsal column.³ Transcortical magnetic stimulation⁴⁻⁶ also

helps to localise the lesion to the upper or lower cervical cord.⁶

To further delineate the conduction abnormalities⁷ for surgical intervention, pickup electrodes must be placed in the structures adjacent to the spinal cord itself. The ascending axonal volleys recorded at multiple sites along the spinal cord usually disclose an abrupt reduction in size of the negative wave as strong evidence for a focal conduction block.^{8,9} Establishing a functional change is of particular value in elderly patients who are known to have a high incidence of clinically silent compression at multiple levels on MRI.¹⁰⁻¹² We report our experience on correlation between intraoperatively recorded spinal somatosensory evoked potentials (SSEPs) and MRI abnormalities of multilevel cord compression seen in elderly patients.

Materials and methods

PATIENTS

We operated on 51 patients with cervical spondylotic myelopathy aged 65 and older for moderate to severe spastic limb paresis during a 5 year period from July 1991 to November 1996. Of these, 44 patients (17 men) aged from 65 to 86 years (mean 75 years) underwent intraoperative SSEP recording after epidural stimulation. Informed consent was obtained. Presurgical symptoms had lasted 12 months or less in 29 patients and more than 12 months in the remainder. The patients with a longer history had surgical decompression with the advent of new functional deterioration. We excluded the patients with myelopathy from other causes such as compression from the ossified posterior longitudinal ligament or trauma causing cord damage immediately after injury.

CLINICAL PICTURE

Based on the functional grading of Nurick,¹³ one patient walked normally despite signs of spinal cord compromise (grade 1), 12 had some difficulty, although ambulatory on their own (grade 2), 18 required walking aids (grades 3 and 4), and 13 were chairbound or bedridden (grade 5). Most patients had lost fine finger movement in doing up buttons and opening and closing the fists.^{14,15} All but four showed sensory impairment for light touch, pinprick, or vibration in the upper limb and all but six in the lower limb. Sensory symptoms were seen in every patient. Of the 28 patients with bladder symptoms, 25 had hesitancy and urgency and three retention and incontinence. Stretch reflexes were generally hyperactive except for diminished response for biceps reflex in nine patients, triceps reflex in two, the knee reflex in

Department of
Orthopaedic Surgery,
Kochi Medical School,
Japan

T Tani
H Yamamoto

Department of
Neurology, Faculty of
Medicine, Kyoto
University, Japan
J Kimura

Correspondence to:
Dr T Tani, Department of
Orthopaedic Surgery, Kochi
Medical School, Kohasu
Okochi-cho, Nankoku City,
Kochi 783, Japan. Telephone
0081 888 80 2387; fax 0081
888 80 2388; email
tanit@kochi-ms.ac.jp

Received 5 May 1998 and in
revised form
28 August 1998
Accepted 14 October 1998

one, and the ankle reflex in nine Extensor plantar responses were elicited in 23 patients.

ELECTRODIAGNOSIS

All recordings were made in the operating theatres of Kochi Medical School after preoperative general anaesthesia. A pair of stimulating electrodes (UKG-100-2PM, Unique Medical Corp, Tokyo, Japan), with two platinum tips at the end of an 18 gauge polyethylene tube, were introduced percutaneously into the dorsal epidural space at the lumbar or lower thoracic level via a Tuohy needle.¹⁶⁻¹⁸ With radiographic confirmation the cathode location varied from the L4 to the T10, lumbar spondylosis precluded the consistency of epidural puncture site and the distance the catheter tip ascended beyond the end of the needle. Electrical stimulation, 0.1 ms in duration and 20-40 mA in intensity, was delivered at a rate of 3-20/s. The recorded potentials were virtually unaltered when varying rates of stimulation were applied (3-20/s). Similar results have been reported for upper and mid-thoracic SSEPs after tibial nerve stimulation.¹⁹⁻²¹

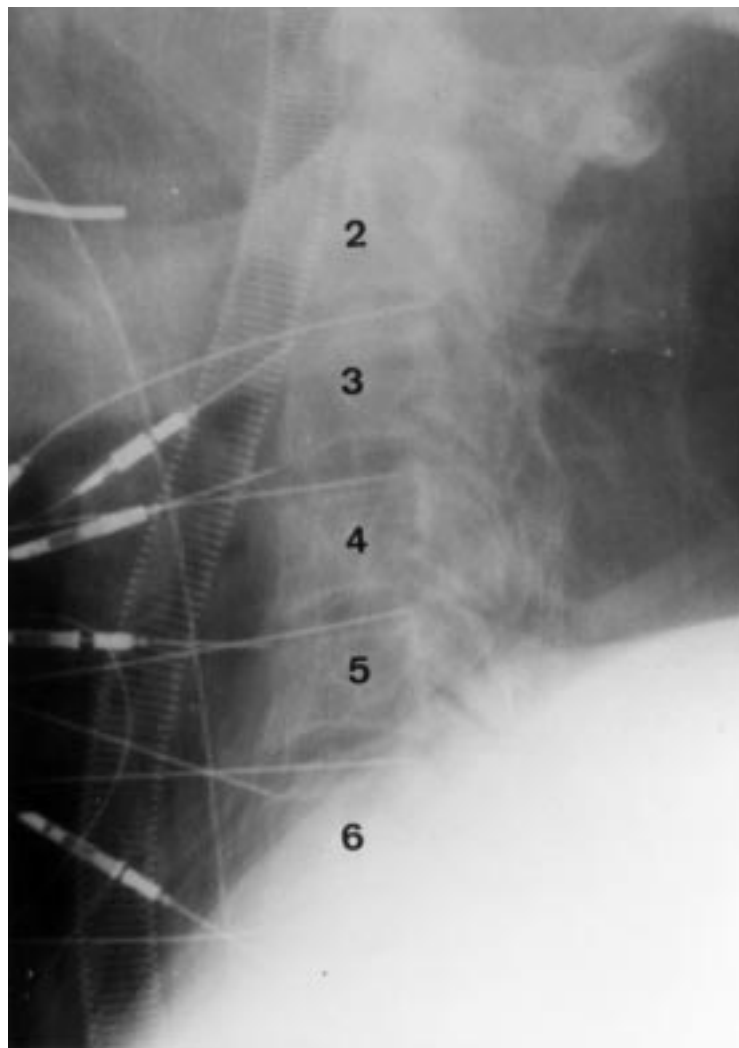


Figure 1 Lateral radiograph of the cervical spine with five active needle electrodes (G1) inserted into serial intervertebral discs from C2-3 to C6-7. The needles were advanced posteriorly enough to cover the disc diameter. Five reference electrodes (G2) inserted into the longus colli muscles at the same level as G1 can be seen.

In 33 patients with anterior operation, a series of needle electrodes (G1) (Dantec 13R23, Dantec Medical, Skovlunde, Denmark) were inserted into the intervertebral discs²⁰⁻²²⁻²⁵ in the midline after exposure of the anterior aspect of the vertebral bodies, and were advanced posteriorly enough to cover the disc diameter calculated from measurement on the plain lateral radiograph (fig 1).²⁵ In two patients the electrode placement was unsuccessful, at C4-5 in one, and at C5-6 in the other, because of a narrow space. In 11 patients with posterior operation, the needle electrodes (G1) were inserted into the ligamentum flavum²¹⁻²⁴⁻²⁶ in the midline at serial intervertebral spaces after exposure of the posterior aspect of the vertebrae.

As the reference, a series of needle electrodes (G2) (Dantec 13R21) were inserted into the longus colli muscles for anterior recordings and the erector spinae muscles for posterior recordings at the same level as G1. A pair of alligator clips was attached to the skin at the operative site as the ground electrode. The recordings were obtained anteriorly from three to five serial vertebral levels between C2-3 and C7-T1 and posteriorly from five to seven serial levels between C1-2 and C7-T1. Each test set comprised an average of 50-200 summated potentials with the frequency response of 20 Hz-5 kHz (3 dB down). An eight channel averager (Dantec Evomatic 8000) allowed simultaneous recording of SSEPs from all sets of electrodes. Two tracings obtained for each electrode derivation confirmed consistency.

As shown in figure 2B, 0 represented the site of conduction block identified by abrupt reduction in amplitude, with the other levels numbered in order of increasing distance from the 0 level, assigning a minus sign caudally. Measurements of SSEPs included: (1) latencies from the stimulus artifact to the initial positive peaks, (2) amplitudes from the baseline to the initial positive and the major negative peaks, and (3) areas (voltage-time integral) of the initial positive and the negative phases (bottom tracing in fig 2B).

MRI EVALUATION

All patients underwent surface coil MR examination of cervical spinal cord preoperatively with one of the three superconducting systems (0.5 T MRT-50 and 1.5 T MRT-200; Toshiba Corp, Tokyo, Japan, 1.5 T Signa; GE Corp, Milwaukee, USA). The MRI protocol included sagittal and axial T1 weighted images, and sagittal T2 weighted images, with slice thickness of 5 mm. The spin echo pulse sequences were 400-600/13-40 (TR ms /TE ms) for T1 and 1600-3000/40-100 for T2 weighted images.

Cord measurements at each intervertebral level from C2-3 to C6-7 included: (1) anteroposterior diameter by a vernier caliper to the nearest 0.1 mm on midsagittal T1 weighted images, and (2) cross sectional area on axial T1 weighted images using the MOP digital image analyser (Mitablet-II KD 4030 A; Graphtec Corp, Japan). The values were converted to the actual diameter and area with a magnification factor. Attention was also directed, on sagittal

T2 weighted images, to increased signal intensity resulting from cord compression.^{27 28}

STATISTICAL ANALYSIS

We used Wilcoxon's signed rank test, considering two tailed tests significant when $p < 0.05$.

Results

SSEPS

Whether recorded from the intervertebral disc (figs 2-4) or from the ligamentum flavum (fig 5), epidurally applied shock to the cauda equina or to the spinal cord consistently

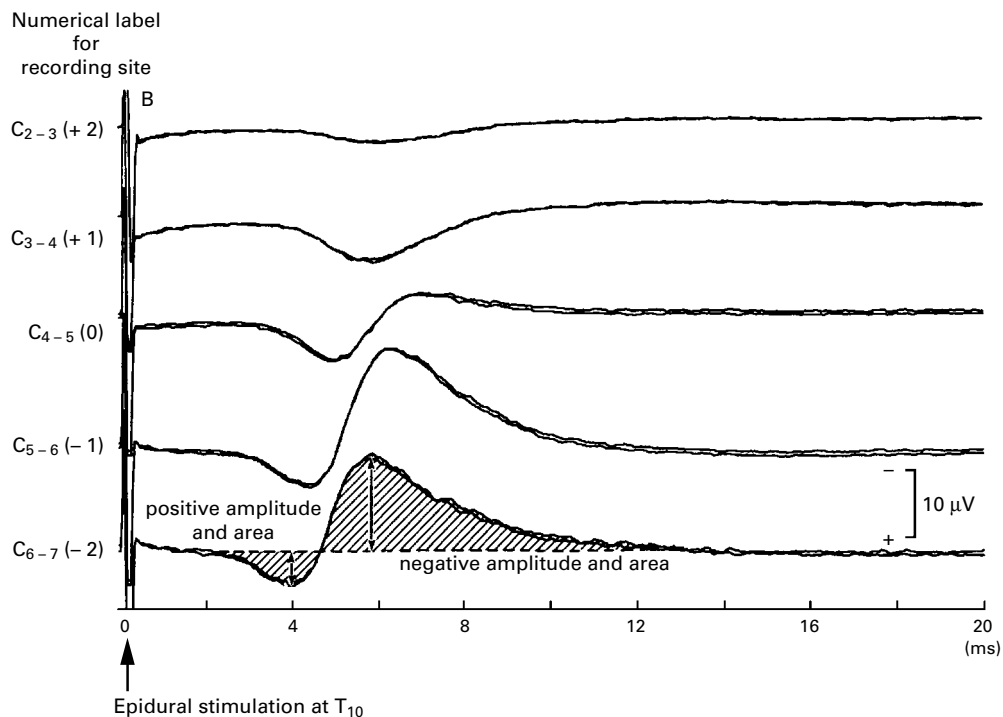
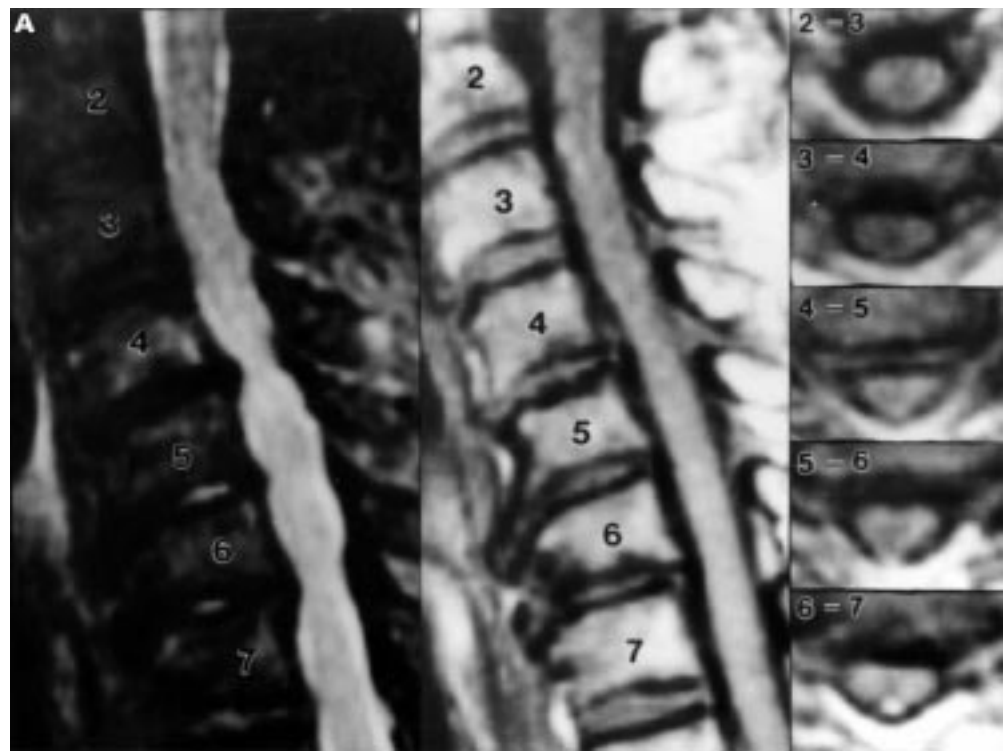


Figure 2 (A) Sagittal T2 weighted MR image (TR 1957 ms; TE 80 ms) (left), and sagittal (middle) and axial (right) T1 weighted images (TR 600 ms; TE 20 ms) in a 71 year old man with cervical spondylotic myelopathy. Cord compression is at C4-5, 5-6, and 6-7, maximal at C4-5. (B) A recording of SSEPs obtained from the same patient as in (A). The SSEPs were recorded unipolarly from the intervertebral disc of C6-7 through C2-3 after epidural stimulation at T10. The method of measurement is illustrated in the bottom trace. The numerical label for each recording site is indicated on the left side. Note an abrupt reduction in size of the negative component and a concomitant augmentation of the initial positive component at C4-5 (0) followed by a monophasic positive wave at C3-4 (+1). A severe conduction block resulted from a moderate cord compression at C4-5.

yielded well defined SSEPs. With the cathode at the level of the spinal cord, the potentials tended to consist of two major negative components (fig 3 B). There was a considerable individual variability in the waveform and amplitude of the potential: in some patients, the first component was more prominent than the second or vice versa, reflecting

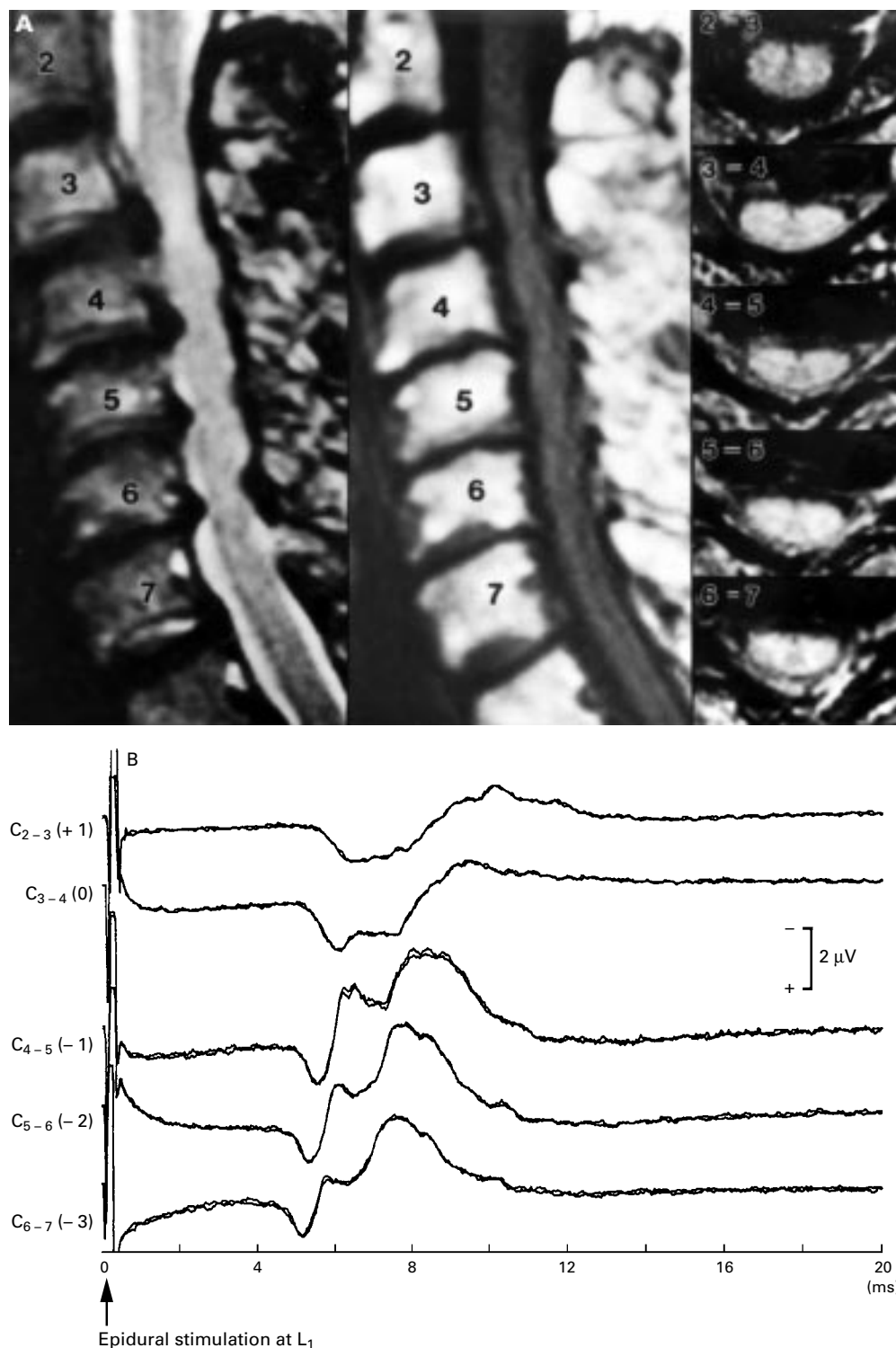


Figure 3 (A) Sagittal T2 weighted MR image (TR 2200 ms; TE 60 ms) (left), and sagittal (middle) and axial (right) T1 weighted images (TR 400 ms; TE 13 ms) in a 68 year old woman with cervical spondylotic myelopathy. A cord compression at C3-4 (0) is equivalent in anteroposterior diameter to those at C4-5 (-1) and C5-6 (-2) and exceeded in cross sectional area by those at C4-5 (-1), C5-6 (-2), and C6-7 (-3). High intensity cord signal on T2 weighted image is seen at the C3-4 (0). (B) Recording of SSEPs obtained from the same patient as in fig 3A. The SSEPs were recorded unipolarly from the intervertebral disc of C6-7 through C2-3 after epidural stimulation at L1. Note a progressive increase in size of the negative component from C6-7 (-3) to C4-5 (-1) followed by an abrupt reduction of the component with a concomitant augmentation of the initial positive component at C3-4 (0). A conduction block corresponded to the site of a intramedullary high signal on T2 weighted image. Spinal cord conduction was not affected by any cord compression at C6-7 (-3), C5-6 (-2), or C4-5 (-1).

inconsistency in the location of the stimulating electrodes. Incremental studies in 44 patients uncovered a single site of focal conduction

block with a marked reduction in size of the negative peak in 42 patients, 23 (55%) at C3-4 (figs 3 and 4), 17 (40%) at C4-5 (figs 2 and 5)

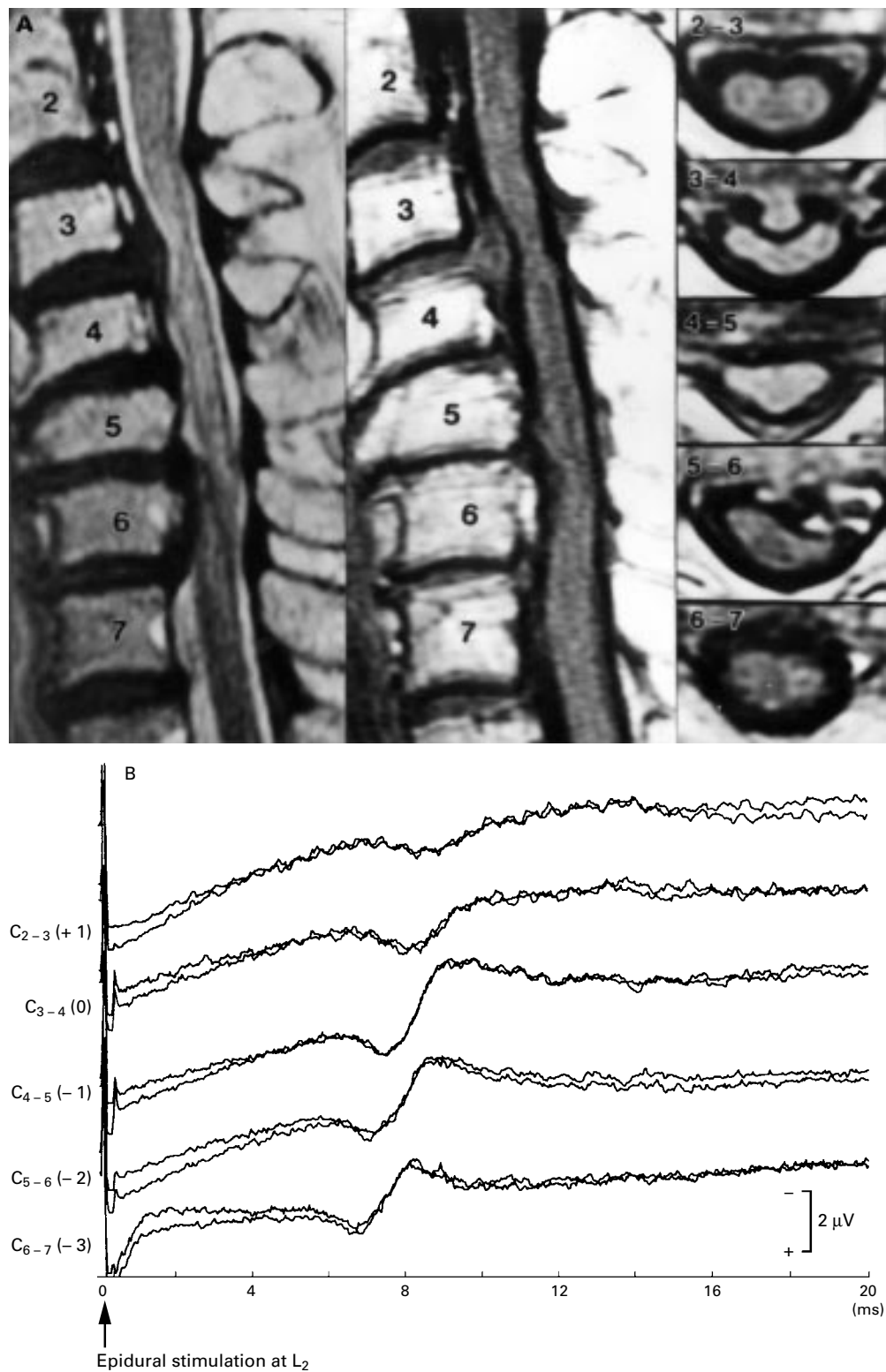


Figure 4 (A) Sagittal T2 weighted MR image (TR 3000 ms; TE 98 ms) (left), and sagittal (middle) and axial (right) T1 weighted images (TR 500 ms; TE 19 ms) in a 69 year old woman with cervical spondylotic myelopathy. A cord compression at C3-4 (0) is greatest in terms of anteroposterior diameter, and equivalent to or even exceeded by those at C4-5 (-1) and C5-6 (-2) in terms of cross sectional area. (B) Recording of SSEPs obtained from the same patient as in fig 4A. The SSEPs were recorded unipolarly from the intervertebral disc of C6-7 through C2-3 after epidural stimulation at L2. Note a progressive increase in size of the negative component from C6-7 (-3) to C4-5 (-1) followed by an abrupt reduction of the component with a concomitant augmentation of the initial positive component at C3-4 (0). Spinal cord conduction was not affected by cord compression either at C5-6 (-2) or C4-5 (-1).

and two (5%) at C5–6. At this level, designated as 0, the amplitude of the negative component was reduced ($p < 0.0001$) to 29 (SD 17)% (range 0–66%) and the area to 22 (SD 15)%

(range 0–60%) compared with the –2 level which was taken as the baseline (table 1). In contrast, the initial positivity at this level was increased ($p < 0.0001$) to 150 (SD 73)% (range

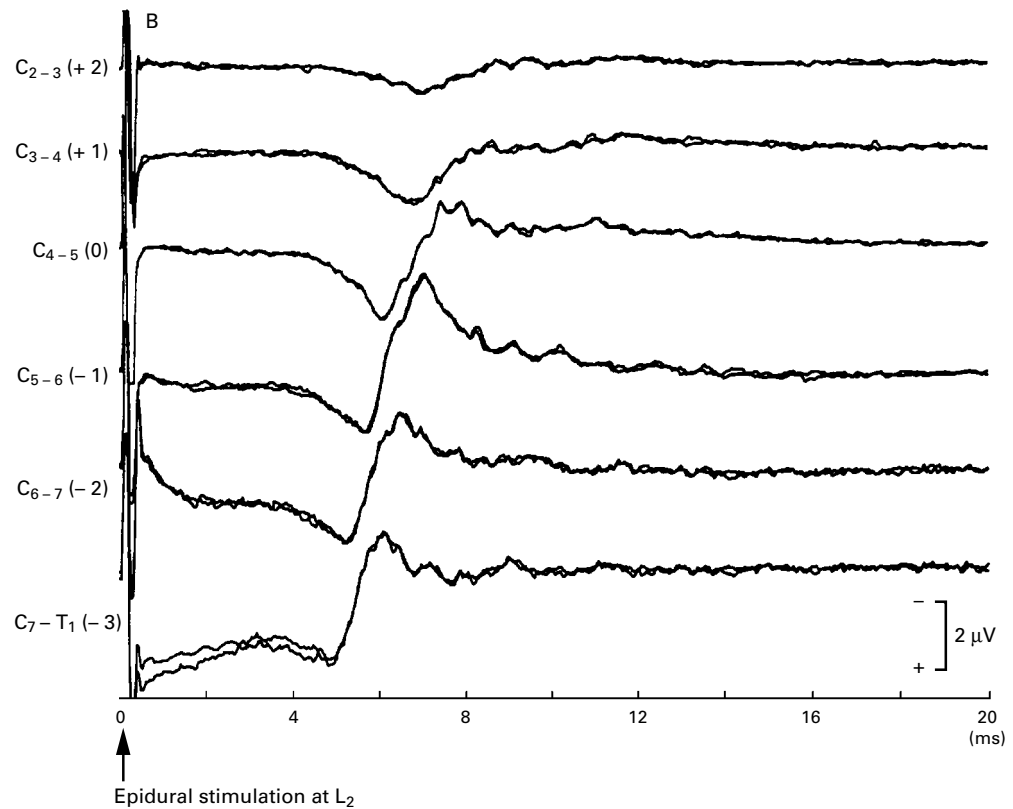
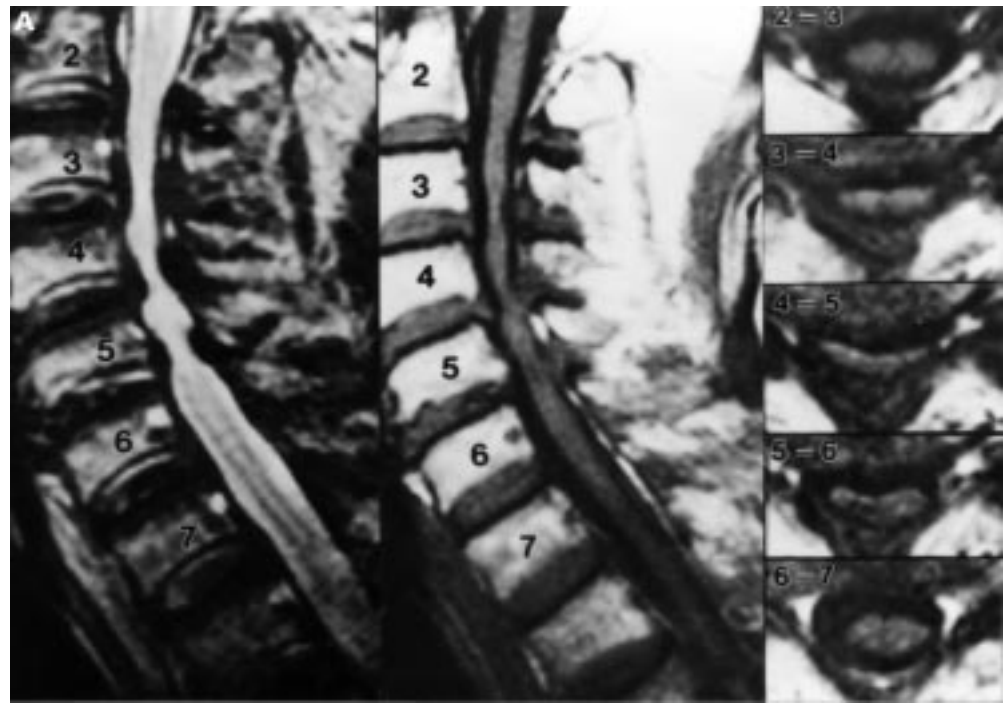


Figure 5 (A) Sagittal T2 weighted MR image (TR 1800 ms; TE 60 ms) (left), and sagittal (middle) and axial (right) T1 weighted images (TR 400 ms; TE 15 ms) in a 73 year old woman with cervical spondylotic myelopathy. Marked spinal cord compression with intramedullary high signal is seen at C3–4 (+1), 4–5 (0) and 5–6 (–1), the compression being maximal at C4–5 (0). (B) Recording of SSEPs obtained from the same patient as in fig 5A. The SSEPs were recorded unipolarly from the ligamentum flavum of C7–T1 through C2–3 after epidural stimulation at L2. Note the progressive increase in size of the negative component from C7–T1 (–3) to C5–6 (–1) followed by an abrupt reduction of the component with a concomitant augmentation of the initial positive component at C4–5 (0) and a monophasic positive wave at C3–4 (+1). Spinal cord conduction was not affected by marked cord compression at C5–6 (–1).

Table 1 Negative component of SSEPs

Recording level	Number of patients	Amplitude			Area		
		Mean (range) μV	Mean (SD) %	p Value	Mean (range) $\mu V \times ms$	Mean (SD) %	p Value
+2	17	1.6 (0-6.5)	15 (9)	< 0.03	3.9 (0-24.8)	12 (13)	NS
+1	35	2.4 (0-14.0)	22 (19)	< 0.003	3.8 (0-27.7)	14 (18)	< 0.003
0	42	3.1 (0-18.7)	29 (17)	< 0.0001	5.5 (0-21.6)	22 (15)	< 0.0001
-1	40*	8.7 (0.6-35.0)	108 (62)	NS	51.1 (0.5-124.0)	111 (41)	NS
-2	42	10.2 (0.6-60.0)	100	NS	40.8 (0.5-79.9)	100	< 0.007
-3	24	9.3 (1.0-58.1)	95 (27)		29.0 (1.0-33.9)	80 (30)	

*In two patients the electrode placement was unsuccessful at the -1 level.

42–424%) in amplitude and 293 (SD 226)% (range 55–988%) in area (table 2). In addition, the latency increase was greater ($p \leq 0.0002$) from -1 to 0 (0.63 (SD 0.34) ms) than from -2 to -1 (0.33 (SD 0.09) ms) or from -3 to -2 (0.34 (SD 0.13) ms) (table 2). Caudal to the site of conduction block, the negative component was greater in area at -1 (111 (SD 41)%, $p < 0.004$) and -2 (100%, $p < 0.007$) than at -3 (80 (SD 30)%) (table 1).

A complete focal conduction block was seen in 15 patients: nine (69%) of 13 in Nurick's grade 5 (fig 2 B); and six (33%) of 18 in grades 3 and 4 (fig 4 B). None of the patients in grades 1 and 2 showed a complete block.

The remaining two patients showing no abrupt reduction in amplitude had a localised conduction delay as evidenced by a disproportionate latency increase from C5–6 to C4–5 (0.76 ms and 1.20 ms, respectively) compared with the adjacent caudal or rostral segments.

MRI

In the 42 patients with a focal conduction block at single levels, sagittal T1 weighted MR images disclosed cord indentation at 92 intervertebral levels, one at C2–3, 28 at C3–4, 32 at C4–5, 25 at C5–6, and six at C6–7. The axial images disclosed a deformed cord at 144 levels, four at C2–3, 39 each at C3–4, C4–5, and C5–6, and 23 at C6–7. Table 3 summarises quantitative assessment of the cord compression in relation to the level of conduction block (0 level). The 0 level had significantly smaller anteroposterior diameter ($p \leq 0.0001$) and cross sectional area ($p \leq 0.0003$) than the remaining more caudal or rostral levels, although the degree of cord compression suffi-

cient to cause conduction block varied widely from one patient to another (figs 2–5). Further, anteroposterior diameters or cross sectional areas were equally reduced or smaller at more caudal levels in 10 patients, one at C4–5 (-1), seven at C5–6 (-1 or -2), and four at C6–7 (-2 or -3) (figs 3 and 4) and at a more rostral C3–4 level (+1) in one patient.

Sagittal T2 weighted images (figs 3 and 5) disclosed high intensity spinal cord signals at 29 intervertebral levels in 25 patients, 11 at C3–4, 12 at C4–5, four at C5–6, and two at C6–7. All matched the site of conduction block with the exception of four levels, located at +1, -1, -2, and -3.

Discussion

Among the physiological consequences of spinal cord compression, conduction block probably plays the most important part in producing potentially treatable clinical deficits.^{29 30} In fact, as shown in this study, there was a correlation between the presence of a complete conduction block and the severity of functional incapacity. In this series of patients with moderate to serious disabilities of subacute onset, SSEPs consistently disclosed a precise site of conduction block characterised by an abrupt reduction in size of the negative peak and a concomitant augmentation of the initial positive peak.^{8 26} The combination of the two opposite changes helps to document the site of abnormality, which may otherwise escape detection. The enhancement of the initial positive wave at the site of conduction block can be explained using the concept of phase cancellation.³¹ An impulse that approaches but does not reach a recording electrode produces

Table 2 Initial positive component of SSEPs

Recording level	No of patients	Amplitude			Area			Latency		
		Mean (range) μV	Mean (SD) %	p Value	Mean (range) $\mu V \times ms$	Mean (SD) %	p Value	Mean (SD) ms	Difference ms	p Value
+2	17	1.7 (0.1-5.9)	75 (30)	0.0003	11.0 (0.1-127)	183 (206)	0.0003	6.60 (1.89)	0.26 (0.19)	
+1	35	3.4 (0.2-14.4)	143 (76)	NS	17.6 (0.1-375)	446 (589)	< 0.04	6.28 (2.07)	0.61 (0.48)	< 0.02
0	42	4.5 (0.2-30.9)	150 (73)	0.0004	22.0 (0.1-683)	293 (226)	< 0.0001	5.55 (1.94)	0.63 (0.34)	NS
-1	40*	3.1 (0.2-16.8)	115 (71)	NS	4.9 (0.1-90.8)	123 (96)	NS	4.91 (1.81)	0.33 (0.09)	< 0.0001
-2	42	3.6 (0.1-23.1)	100	NS	4.7 (0.1-80.7)	100	NS	4.59 (1.77)	0.34 (0.13)	NS
-3	24	3.2 (0.3-20.6)	101 (29)		6.8 (0.1-103)	103 (35)		4.67 (1.55)		

*In two patients the electrode placement was unsuccessful at the -1 level.

Table 3 Cervical cord measurement

Intervertebral level	No of patients	APD† mean (SD) (range) mm	CA‡ mean (SD) (range) mm ²
+2	19	6.8 (1.3) (3.8-9.5)	70.7 (13.6) (46.5-97.8)
+1	42	6.4 (1.2) (2.6-8.5)	70.0 (16.1) (25.9-100)
0	42	3.8 (1.0)* (2.1-5.7)	49.4 (14.2)* (17.3-89.6)
-1	42	5.5 (1.0) (3.4-7.8)	63.7 (17.0) (35.5-111)
-2	40	6.0 (1.1) (3.5-8.5)	64.9 (16.5) (31.6-105)
-3	23	6.4 (0.8) (4.6-7.9)	67.5 (14.0) (38.2-106)

† APD=anteroposterior diameter measured on midsagittal T1weighted MRI. ‡ CA=cross sectional area measured on axial T1weighted MRI. * Significantly smaller APD ($p \leq 0.0001$) and CA ($p \leq 0.0002$) than those at the remaining more caudal and rostral levels.

the initial positivity and a subsequent low amplitude negativity.³²⁻³⁴ This diminution of the negativity tends to increase the size of the initial positive component. Other characteristic findings near the point of conduction block include an increased negativity caudally and an enhanced positivity rostrally (figs 2-5).^{26, 34} The first change is apparently paradoxical and therefore diagnostically useful because, were it not for a conduction block, the negative components would be progressively smaller with increasing distance of impulse propagation, as predicted from physiological temporal dispersion with phase cancellation.^{26, 31} All six patients without clinical sensory deficits also had evidence for a conduction block, indicating the usefulness of SSEPs for assessing subclinical sensory impairment.

The present study has disclosed a high incidence (95%) of focal conduction block at the upper cervical level (C3-4 or C4-5) in patients with cervical spondylotic myelopathy aged 65 and older. This is counter-intuitive because, in general, age related degenerative radiological changes of the cervical spine develop with the greatest frequency and extent at the lower cervical levels (C5-6 and C6-7).^{35, 36} In fact, in most previous reports³⁶⁻⁴³ which have included a significant proportion of middle aged patients, C5-6 was the commonest site of decompressive surgical treatment. In a radiographic study for cervical spondylotic myelopathy using myelography followed by CT, Hayashi *et al*⁴⁴ noted that cord compression in patients aged over 60 was more extensive, involving C3-4 and C4-5 more often than in patients below 60 years of age. As shown in the present study, MRI in elderly patients tends to show multilevel compression. In this age group, intense cord compression on T1 weighted images and high intensity spinal cord signals on T2 weighted images also abound at C3-4 and C4-5. Thus, with advancing age, the primary sites responsible for myelopathy shift from lower to upper cervical levels.

Our data suggest that cervical spondylotic myelopathy tends to functionally spare C5-6 and C6-7 levels despite the same degree of compression causing conduction block at more rostral levels in elderly patients. Teresi *et al*¹⁰ also found clinically silent concave defects of the spinal cord on T1 weighted MR images in as many as 23% of 42 asymptomatic subjects 65 and older, mostly at the C5-6 level. This dissociation may suggest the beneficial nature of a stable cervical segment,^{36, 45, 46} accounting for the lack of conduction block in the face of apparent compression. In elderly patients the

lower cervical segments show less mobility than the upper cervical segments reflecting the degree of bony degeneration. By contrast, an excessive compensatory movement imposed on the C3-4 and the C4-5 segments would cause instability,^{36, 45, 47} leading to spinal cord compromise.

Whereas cord compression may appear without functional change, the presence of conduction block implies a sufficient degree of cord compression, usually at the level of the smallest anteroposterior diameter and cross sectional area (table 3). This indicates the functional importance of direct mechanical pressure on the white matter rather than vascular factors involving the grey matter remote from the site of compression, thus causing false localising signs.⁴⁸⁻⁵⁰ Most high intensity spinal cord signals on sagittal T2 weighted images also corresponded to the site of conduction block. In the four exceptional cases, the discrepancy may have resulted from pathological changes of the cord segments with high intensity signals confined to the grey matter.⁵¹

Strictly speaking, the cord potentials recorded after epidurally applied cord stimulation consist not only of SSEPs but possibly also antidromic motor activity. Therefore, evoked potential studies in our series should be interpreted with caution, not implicating any specific pathways. However, the dorsal spinocerebellar tract and the dorsal columns primarily, though not exclusively, mediate the two major negative components with cord stimulation, as evidenced in previous studies.^{17, 52, 53} Besides, according to one study on cervical spondylotic myelopathy,²⁵ both sensory and motor pathways are usually, though not always, compromised at the same level. If so, incremental SSEP studies are useful additions to MRI in localising the level of maximal cord involvement. The technique can be carried out during surgery, before decompression procedures, to exclude clinically silent cord compression, rendering the surgical intervention at the appropriate level responsible for the main functional change.

- 1 Yu YL, Jones SJ. Somatosensory evoked potentials in cervical spondylosis. *Brain* 1985;108:273-300.
- 2 Yiannikas C, Shahani BT, Young RR. Short-latency somatosensory-evoked potentials from radial, median, ulnar, and peroneal nerve stimulation in the assessment of cervical spondylosis. *Arch Neurol* 1986;43:1264-71.
- 3 Restuccia D, Valeriani M, Lazzaro VD, *et al*. Somatosensory evoked potentials after multisegmental upper limb stimulation in diagnosis of cervical spondylotic myelopathy. *J Neurol Neurosurg Psychiatry* 1994;57:301-8.
- 4 Jaskolski DJ, Jarratt JA, Jakubowski J. Clinical evaluation of magnetic stimulation in cervical spondylosis. *Br J Neurosurg* 1989;3:541-8.
- 5 Maertens de Noordhout A, Remacle JM, Pepin JL, *et al*. Magnetic stimulation of the motor cortex in cervical spondylosis. *Neurology* 1991;41:75-80.
- 6 Lazzaro VD, Restuccia D, Colosimo C, *et al*. The contribution of magnetic stimulation of the motor cortex to the diagnosis of cervical spondylotic myelopathy. Correlation of central motor conduction to distal and proximal upper limb muscles with clinical and MRI findings. *Electroencephalogr Clin Neurophysiol* 1992;85:311-20.
- 7 Kimura J. The carpal tunnel syndrome: localization of conduction abnormalities within the distal segment of the median nerve. *Brain* 1979;102:619-35.
- 8 McDonald WI, Sears TA. The effects of experimental demyelination on conduction in the central nervous system. *Brain* 1970;93:583-98.
- 9 Cornblath DR, Sumner AJ, Daube J, *et al*. Conduction block in clinical practice. *Muscle Nerve* 1991;14:869-71.

- 10 Teresi LM, Lufkin RB, Reicher MA, et al. Asymptomatic degenerative disc disease and spondylosis of the cervical spine: MR imaging. *Radiology* 1987;164:83-8.
- 11 Boden SD, McCowin PR, Davis DO, et al. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. *J Bone Joint Surg A* 1990;72:1178-84.
- 12 Rowland LP. Surgical treatment of cervical spondylotic myelopathy: time for a controlled trial. *Neurology* 1992;42:5-13.
- 13 Nurick S. The pathogenesis of the spinal cord disorder associated with cervical spondylosis. *Brain* 1972;95:87-100.
- 14 Crandall PH, Batzdorf U. Cervical spondylotic myelopathy. *J Neurosurg* 1966;25:57-66.
- 15 Ono K, Ebara S, Fujii T, et al. Myelopathy hand; new clinical signs of cervical cord damage. *J Bone Joint Surg B* 1987;69:215-19.
- 16 Shimoji K, Higashi H, Kano T. Epidural recording of spinal electrogram in man. *Electroenceph Clin Neurophysiol* 1971;30:236-9.
- 17 Tsuyama N, Tsuzuki N, Kurokawa T, et al. Clinical application of spinal cord action potential measurement. *Int Orthop* 1978;2:39-46.
- 18 Tamaki T, Tsuji H, Inoue S, et al. The prevention of iatrogenic spinal cord injury utilizing the evoked spinal cord potential. *Int Orthop* 1981;4:313-17.
- 19 Jones SJ, Edgar MA, Ransford AO. Sensory nerve conduction in the human spinal cord: epidural recordings made during scoliosis surgery. *J Neurol Neurosurg Psychiatry* 1982;45:446-51.
- 20 Halonen JP, Jones SJ, Edgar MA, et al. Conduction properties of epidurally recorded spinal cord potentials following lower limb stimulation in man. *Electroenceph Clin Neurophysiol* 1989;74:161-74.
- 21 Pelosi L, Caruso G, Cracco RQ, et al. Intraoperative recordings of spinal somatosensory evoked potentials to tibial nerve and sural nerve stimulation. *Muscle Nerve* 1991;14:253-8.
- 22 Matsuda H, Kondo M, Hashimoto T, et al. The prediction of the surgical prognosis of the compression myelopathy. *Osaka City Med J* 1984;30:91-112.
- 23 Satomi K, Okuma T, Kenmotsu K, et al. Level diagnosis of cervical myelopathy using evoked spinal cord potentials. *Spine* 1988;13:1217-24.
- 24 Matsuda H, Shimazu A. Intraoperative spinal cord monitoring using electric responses to stimulation of caudal spinal cord or motor cortex. In: Desmedt JE, ed. *Neuromonitoring in surgery*. New York: Elsevier, 1989:175-190.
- 25 Tani T, Ushida T, Yamamoto H. Surgical treatment guided by spinal cord evoked potentials for tetraparesis due to cervical spondylosis. *Paraplegia* 1995;33:354-8.
- 26 Tani T, Ushida T, Yamamoto H, et al. Waveform analysis of spinal somatosensory evoked potential: paradoxically enhanced negative peaks immediately caudal to the site of conduction block. *Electroencephalogr Clin Neurophysiol* 1998;108:325-30.
- 27 Takahashi M, Sakamoto Y, Miyawaki M, et al. Increased MR signal intensity secondary to chronic cervical cord compression. *Neuroradiology* 1987;29:550-6.
- 28 Takahashi M, Yamashita Y, Sakamoto Y, et al. Chronic cervical cord compression: clinical significance of increased signal intensity on MR images. *Radiology* 1989;173:219-24.
- 29 Kimura J, Kaji R. Comment from the editorial office. *Muscle Nerve* 1991;14:867-8.
- 30 Waxman SG, Kocsis JD, Black JA. Pathophysiology of demyelinated axons. In: Waxman SG, Kocsis JD, Stys PK, eds. *The axon*. New York: Oxford University Press, 1995:438-61.
- 31 Kimura J, Sakimura Y, Machida M, et al. Effect of desynchronized input on compound sensory and muscle action potentials. *Muscle Nerve* 1988;11:694-702.
- 32 Woodbury JW. Potentials in a volume conductor. In: Ruch TC, Patton HD, Woodbury JW, et al, eds. *Neurophysiology*. 2nd ed. Philadelphia: WB Saunders, 1965:85-91.
- 33 Kimura, J. *Electrodiagnosis in diseases of nerve and muscle: principles and practice*. 2nd ed. Philadelphia, FA Davis, 1989:30-34.
- 34 Tani T, Ushida T, Yamamoto H, et al. Waveform changes due to conduction block and their underlying mechanism in spinal somatosensory evoked potential: a computer simulation. *J Neurosurg* 1997;86:303-10.
- 35 Friedenber ZB, Miller WT. Degenerative disc disease of the cervical spine: a comparative study of asymptomatic and symptomatic patients. *J Bone Joint Surg A* 1963;45:1171-8.
- 36 Hayashi H, Okada K, Hamada M, et al. Etiologic factors of myelopathy, a radiographic evaluation of the aging changes in the cervical spine. *Clin Orthop* 1987;214:200-9.
- 37 White III AA, Southwick WO, Deponce RJ, et al. Relief of pain by anterior cervical fusion for spondylosis. *J Bone Joint Surg A* 1973;55:525-34.
- 38 Lunsford LD, Bissonette DJ, Zorub DS. Anterior surgery for cervical disc disease: part 2: Treatment of cervical spondylotic myelopathy in 32 cases. *J Neurosurg* 1980;53:12-19.
- 39 Hukuda S, Mochizuki T, Ogata M, et al. Operations for cervical spondylotic myelopathy: a comparison of the results of anterior and posterior procedures. *J Bone Joint Surg B* 1985;67:609-15.
- 40 Yonenobu K, Fujii T, Ono K, et al. Choice of surgical treatment for multisegmental cervical spondylotic myelopathy. *Spine* 1985;10:710-16.
- 41 Jeffreys RV. The surgical treatment of cervical myelopathy due to spondylosis and disc degeneration. *J Neurol Neurosurg Psychiatry* 1986;49:353-61.
- 42 Arnold H, Feldmann U, Miffler U. Chronic spondylogenic cervical myelopathy: a critical evaluation of surgical treatment after early and long-term follow-up. *Neurosurg Rev* 1993;16:105-9.
- 43 Wu W, Thuomas KA, Hedlund R, et al. Fast spin-echo MR assessment of patients with poor outcome following spinal cervical surgery. *Acta Radiol* 1996;37:153-61.
- 44 Hayashi H, Okada K, Hashimoto J, et al. Cervical spondylotic myelopathy in the aged patient, a radiographic evaluation of the aging changes in the cervical spine and etiologic factors of myelopathy. *Spine* 1988;13:618-25.
- 45 Bohlman HH. Cervical spondylosis with moderate to severe myelopathy: a report of 17 cases treated by Robinson anterior cervical discectomy and fusion. *Spine* 1977;2:151-62.
- 46 Barnes MP, Saunders M. The effect of cervical mobility on the natural history of cervical spondylotic myelopathy. *J Neurol Neurosurg Psychiatry* 1984;47:17-20.
- 47 Braakman R. Management of cervical spondylotic myelopathy and radiculopathy. *J Neurol Neurosurg Psychiatry* 1994;57:257-63.
- 48 Taylor AR, Byrnes DP. Foramen magnum and high cord compression. *Brain* 1974;97:473-80.
- 49 Stark RJ, Kennard C, Swash M. Hand wasting in spondylotic high cord compression: an electromyographic study. *Ann Neurol* 1981;9:58-62.
- 50 Goodridge AE, Feasby TE, Ebers GC, et al. Hand wasting due to mid-cervical spinal cord compression. *Can J Neurol Sci* 1987;14:309-11.
- 51 Al-Mefty O, Harkey LH, Middleton TH, et al. Myelopathic cervical spondylotic lesions demonstrated by magnetic resonance imaging. *J Neurosurg* 1988;68:217-22.
- 52 Imai T. Human electrospinoogram evoked by direct stimulation on the spinal cord through epidural space. *Nippon Seikeigeka Gakkai Zasshi* 1976;50:1037-56. (In Japanese.)
- 53 Machida M, Weinstein SL, Yamada T, et al. Spinal cord monitoring: electrophysiological measures of sensory and motor function during spinal surgery. *Spine* 1985;10:407-13.