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# Muscle Sympathetic Nerve Activity in Patients with Lumbar Spinal Canal Stenosis

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**Abstract**

The present study aimed to measure sensory nerve conduction velocity (SNCV) and muscle sympathetic nerve activity (MSA) in both normal subjects and patients with lumbar spinal canal stenosis (LSCS), and to determine what sensory and sympathetic nerve systems relate to the development of abnormal sensation in the lower limbs of the patients. The study population was 12 patients and 10 age-matched healthy control subjects.

A statistical difference in the mean MSA intervals was found between the LSCS patients and the normal subjects. There was a fairly large difference between them in the values of the standard deviations as one of the parameters to determine the degree of fluctuation of MSA. These results suggest the LSCS patients have shorter MSA intervals and narrower fluctuations of MSA than normal subjects.

As for the range of fluctuation of the MSA intervals and SNCV, the faster the SNCV, the wider the range of fluctuation of MSA intervals in the normal subjects. Many patients with LSCS seem to maintain a correlation between SNCV and MSA intervals. This suggests that even in cases of LSCS, human homeostasis works to keep the relationship between sympathetic nerve function and somato sensory nerve function to some extent. A few LSCS patients showed no correlation between MSA and SNCV. These patients were rather old, suffered spinal stenosis in the relatively higher levels of the spinal canal, and had suffered from the disease for longer than the mean period of all the patients. When the peripheral nerves or cauda epuina are chronically compressed, the nerve systems can not maintain the relationship between them, which finally results in failure. It is suggested that the disrupted coordination between sympathetic nerve function and somato sensory nerve function is one of the reasons why abnormal sensations occur in the lower extremities of LSCS patients. (J Nippon Med Sch 2001; 68: 376—383)

**Key words**: microneurography, sympathetic nerves, abnormal sensations, sensory nerve conduction velocity and aging

## Introduction

It is well known that many patients with lumbar spinal canal stenosis (LSCS) complain of abnormal sensations such as tingling, tickling, psychroesthesia and so on. Furthermore these patients usually have a hypothermal cutaneous area in their lower extremities. Peripheral cutaneous blood flow is basically determined by sympathetic nerve activities, so these facts suggest that facilitation of sympathetic nerve activities have a considerable relationship with the occur-

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rence of such abnormal sensations[1,2].

Microneurography allows researchers to record sympathetic nerve action potentials directly from the peripheral nerve[3-6]. Muscle sympathetic nerve activity (MSA) and a sympathetic nerve discharge have a direct relationship with active vasoconstriction in the skeletal muscles[7-9]. There are a few articles that discuss the facilitation of MSA in patients with lumbar spine disease, particularly in those with chronic nerve compression of the cauda equina[10,11]. However, the mechanism of MSA changes in LSCS has not been clearly identified, and their clinical symptoms lead to the possibility of sympathetic nerve impairment. Few human studies have been reported concerning any interaction between the somatic and sympathetic nerve systems[12-14].

The aim of this study is to record sensory nerve conduction velocity (SNCV) and MSA in both normal subjects and LSCS patients, and to determine how the two nerve systems relate to the development of abnormal sensation in LSCS patients.

## Materials and Methods

Patient population and clinical findings;

Fifty-three LSCS patients were admitted to Nippon Medical School Hospital from 1998 to 1999, and 12 of these 53 were selected for this study. Dysesthesia is a continuous abnormal sensation that is considered to be uncomfortable, such as tingling, tickling or psychroesthesia; paresthesia is not a spontaneous sensation, but is also unpleasant. Both of them can be considered to be abnormal sensations resulting from sympathetic nerve dysfunction. As we have been pursuing studies involving dysfunction of the sympathetic nerve, all of the selected patients had definite symptoms of dysesthesia and/or paresthesia in their lower extremities, which was not caused by hypertension, diabetes mellitus or other diseases.

All patients and volunteers as controls were informed about the purpose of this study and risks involved and gave this consent before entering this study. This study was carried out with the approval of the clinical studying ethical review committee of Nippon Medical School Hospital.

### Measurements

The subjects in the prone position were instructed to relax and not to make any active motions. Both heartbeat and respiration were continuously monitored during the examination.

Sensory nerve activities were recorded with a silver disc electrode of 8 mm in diameter from the intermediate dorsal cutaneous branch of the superficial peroneal nerve at room temperature (22～25℃) according to Jabre's method[15] (**Fig. 1**). A pick-up electrode was fixed at 2 cm medial to the lateral malleolus, where the nerve runs underneath the skin, and another one on the lateral as a reference electrode. A stimulating bipolar electrode was positioned 12 cm proximal from the recording electrode. A ground electrode was also positioned between the recording and stimulating electrodes on the anterior aspect of the fibula. An electrical stimulation was applied antidromically, at the intensity of supramaximum, rectangular pulse of 0.5 msec and 1 Hz of frequency. Then, the latency of the averaged potentials was determined and SNCV was calculated. As the obtained potentials were very small in amplitude, ten acquired discharges were averaged.

MSA was induced directly from the common peroneal nerve at the popliteal fossa of the knee using a mono-polar microelectrode made of tungsten and 43～53 μm in diameter with an uninsulated top (2～5 MΩ impedance). The electrode was inserted percutaneously into the nerve manually, and a reference silver disc electrode was positioned 1.5～2.0 cm proximally to the detection microelectrode (**Fig. 2**). When the

Fig. 1 A silver disc electrode of 8 mm in diameter for the measurement of sensory nerve activities

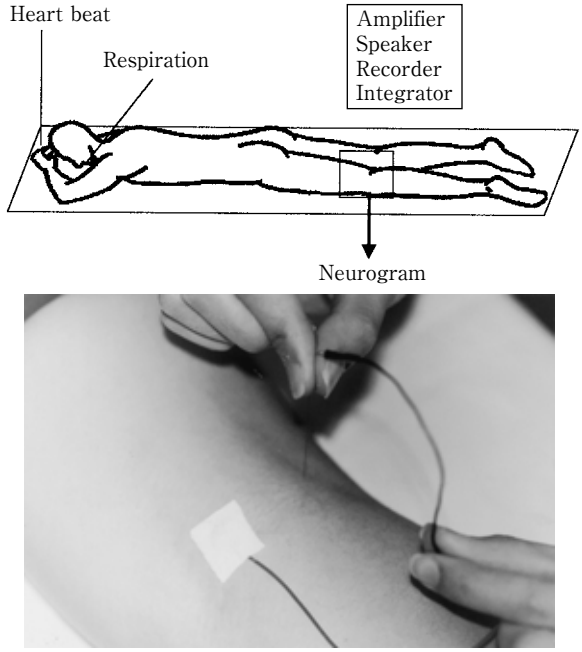


Fig. 2 Schematic diagram of the test equipment for the measurement of muscle sympathetic nerve activity

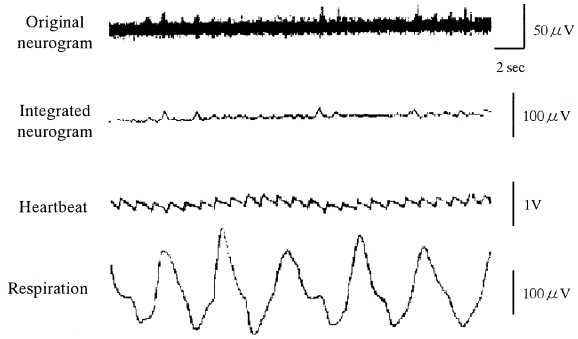


Fig. 3 Muscle sympathetic nerve activity in a normal subject

Table 1 Characteristics of patients and controls

| | LSCS patients | controls |
|---|---|---|
| Number | 12 | 10 |
| Male : female | 8 : 4 | 6 : 4 |
| Age (years) (mean ± S.D.) | 66.7 ± 9.0 | 58.9 ± 6.2 |

LSCS, lumbar spinal canal stenosis
Age expressed as mean ± S.D. The ages of patients and controls were very similar, and did not have any statistical significance.

microelectrode was inserted into the nerve bundle correctly, spontaneous nerve action potentials were acquired and local dysesthesia appeared simultaneously in the lower limbs. Throughout the examination, all procedures were performed on the patients while they were awake and without any anesthesia.

MSA consisted of grouped spontaneous discharges, repeated either at irregular intervals or in a rhythmical fashion. According to the characteristics of MSA firing patterns, it shows spontaneous, regular pulse-synchronous burst impulses, and increases as blood pressure decreases or on application of Valsalva's maneuver. MSA was integrated to identify it clearly for practical use (Mano's[6] and Delius's[3] criteria) (**Fig. 3**).

**Data analysis**

Each original MSA was rectified and integrated through a filter with a time constant of 0.1 sec. on a Neuropack 8 (MEM-4200, Nihon Kohden).

The data were analyzed statistically by Student's t-test, Mann-Whitney's U test and simple regression. Statistical significance was fixed at a level of 0.05%.

## Results

The examined patients consisted of 8 males and 4 females with an average age of 66.7 (ranging from 51 to 77). Ten age-matched healthy volunteers (ranging from 49 to 71 years old, mean age 58.9) were also used in this study. It takes a long time to examine MSA, and it is very difficult to detect clear MSA during the examination. Furthermore, the examination is uncomfortable for the patients. For these reasons, only 12 of the 53 patients consented to be involved in this study (**Table 1**). The examination was performed on the patients an average of 14.2 months (ranging from 1 to 36 months) after their symptoms initially occurred (**Table 2**).

**1. Sensory nerve conduction velocity:**

The mean and standard deviation values of SNCV were 46.4 ± 10.3 m/sec in the LSCS patients, and 52.0 ± 4.6 m/sec in the normal subjects. The difference in the mean values of SNCV was statistically significant by Mann-Whitney's U test ($P<0.05$) (**Table 3**).

Table 2 Detailed characteristics in the LSCS patients

| No. | age | sex | disease period (month) | level of stenosis |
|---|---|---|---|---|
| 1 | 77 | M | 24 | L2, 3, 4 |
| 2 | 54 | F | 12 | L3, 4, 5, S1 |
| 3 | 74 | F | 36 | L3, 4, 5 |
| 4 | 77 | M | 3 | L3, 4, 5, S1 |
| 5 | 75 | F | 24 | L3, 4, 5 |
| 6 | 73 | M | 3 | L4, 5 |
| 7 | 68 | M | 24 | L4, 5 |
| 8 | 67 | F | 2 | L4, 5 |
| 9 | 65 | M | 1 | L4, 5 |
| 10 | 61 | M | 36 | L4, 5 |
| 11 | 58 | M | 3 | L4, 5 |
| 12 | 51 | M | 2 | L4, 5, S1 |
| mean | 66.7 | | 14.2 | |

LSCS, lumbar spinal canal stenosis ; M, male ; F, female ; L, lumbar ; S, sacral

There were a few patients (No. 1, 2, 3) whose correlation of SNCV and the range of fluctuation of MSA intervals seemed to be disrupted. These patients were relatively old in the patients group, suffered spinal stenosis in the relatively higher levels of the spinal canal, and had suffered from the disease for longer than the mean period of all the patients.

Table 3 Data of sensory nerve conduction velocity

| SNCV (m/sec) | | | |
|---|---|---|---|
| No. | LSCS patients | No. | Controls |
| 1 | 34.1 | 1 | 50.8 |
| 2 | 28.8 | 2 | 51.7 |
| 3 | 69.0 | 3 | 53.1 |
| 4 | 41.4 | 4 | 61.2 |
| 5 | 48.4 | 5 | 52.2 |
| 6 | 46.9 | 6 | 48.0 |
| 7 | 46.9 | 7 | 56.6 |
| 8 | 49.6 | 8 | 45.5 |
| 9 | 45.1 | 9 | 53.6 |
| 10 | 40.7 | 10 | 47.2 |
| 11 | 56.1 | | |
| 12 | 50.3 | | |
| (Mean ± S.D.) | 46.4 ± 10.25* | | 52.0 ± 4.62* |

SNCV, sensory nerve conduction velocity ; LSCS, lumbar spinal canal stenosis

There was a statistically significant difference between the LSCS patients and the controls (*$p < 0.05$).

**2. MSA discharge intervals:**

The bursts of MSA were often "pulse-synchronous", and they tended to appear periodically during certain phases of the respiratory cycle and heartbeat. As the intervals of MSA fluctuated during the experiments, the obtained MSA series was divided into 3 to 4 sections in each time unit of 30 seconds along the time course to gain a better understanding of the function of sympathetic nerve activity.

The mean interval of the MSA in each 30-second unit was 36.9 ± 18.4 msec in the LSCS patients, and 60.3 ± 71.6 msec in the normal subjects. We took the standard deviation of MSA intervals as one of the parameters to determine the degree of fluctuation of MSA. The value of the standard deviation was 29.2 ± 20.2 msec in the LSCS patients, and 45.9 ± 54.2 msec in the normal subjects.

A statistical difference in the mean of the MSA intervals was found between the LSCS patients and the normal subjects by Student's t-test (**Table 4**): the intervals in the LSCS patients were clearly shorter than those in the normal subjects. There was a fairly large difference in the values of the standard deviations in the LSCS patients and control subjects, but no considerable difference was acquired statistically. To sum up, LSCS patients have shorter MSA intervals and narrower fluctuations of MSA than normal subjects.

**3. Sensory nerve conduction velocity and the range of fluctuation of MSA intervals in individual cases:**

The range of SNCV and standard deviations of MSA in the LSCS patients were 28.8～69.0 m/sec and 6.8～65.6 msec, respectively. On the other hand, in the normal subjects, they were 45.5～61.2 m/sec and 12.6～139.8 msec, respectively (**Table 5**). The only statistically positive correlation was found in the normal subjects ($r=0.684$) (**Fig. 4**). It is suggested that the faster the SNCV, the wider the range of fluctuation of MSA intervals in normal subjects. There was no significant correlation in LSCS patients ($r=0.031$).

## Discussion

MSA is one of the impulses running through the sympathetic nerves and has the effect of a vasoconstrictor. Senma[11] reported that MSA recorded from patients with low back pain was considerably increased in comparison with other subjects. Iwase et

Table 4 Mean interval and the standard deviation of the MSA intervals of each 30-second unit and SNCV in the LSCS patients and the normal subjects

| LSCS patients | | | | normal subjects | | | |
|---|---|---|---|---|---|---|---|
| Mean interval (ms) | | S.D. (ms) | SNCV (m/s) | Mean interval (ms) | | S.D. (ms) | SNCV (m/s) |
| No. | | | | No. | | | |
| 1 | 41.14 | 43.88 | 34.1 | 1 | 32.44 | 21.93 | 50.8 |
| | 38 | 6.928 | 34.1 | | 36.75 | 27.42 | 50.8 |
| | 75 | 106 | 34.1 | | 30.4 | 32.87 | 50.8 |
| | 63.6 | 57.35 | 34.1 | | 45.14 | 39.98 | 50.8 |
| 2 | 22.55 | 6.138 | 28.8 | 2 | 157 | 190.9 | 51.7 |
| | 23.64 | 5.644 | 28.8 | | 69 | 69.6 | 51.7 |
| | 21.69 | 3.545 | 28.8 | | 63.33 | 35.13 | 51.7 |
| | 22.43 | 10.23 | 28.8 | | 122 | | 51.7 |
| 3 | 39 | 43.59 | 69 | 3 | 35.71 | 29.99 | 53.1 |
| | 25.5 | 22.9 | 69 | | 23 | 11.17 | 53.1 |
| | 14.53 | 7.23 | 69 | | 52.33 | 38.35 | 53.1 |
| | 18.2 | 9.644 | 69 | | 25.17 | 22.76 | 53.1 |
| 4 | 31.11 | 22.41 | 41.4 | 4 | 14.67 | 11.55 | 61.2 |
| | 23.14 | 21.75 | 41.4 | | 103.6 | 103.4 | 61.2 |
| | 17.29 | 11.73 | 41.4 | | 310 | | 61.2 |
| | 24 | 13.4 | 41.4 | | 42.5 | 46.2 | 61.2 |
| 5 | 14.6 | 15.88 | 48.4 | 5 | 32.86 | 34.06 | 52.2 |
| | 23.27 | 16.6 | 48.4 | | 46.57 | 26.07 | 52.2 |
| | 46.86 | 53.55 | 48.4 | | 65 | 64.63 | 52.2 |
| | 20 | 19.77 | 48.4 | | 57.67 | 38.25 | 52.2 |
| 6 | 32 | 24.76 | 46.9 | 6 | 37.5 | 41.28 | 48 |
| | 55 | 32.02 | 46.9 | | 22.83 | 22.47 | 48 |
| | 43.43 | 34.42 | 46.9 | | 32.6 | 14.08 | 48 |
| | 87.33 | 76.79 | 46.9 | | 24 | | 48 |
| 7 | 33.09 | 16.57 | 46.9 | 7 | 58.8 | 101.6 | 56.6 |
| | 47.6 | 45.48 | 46.9 | | 19.38 | 36.58 | 56.6 |
| | 46 | 21.66 | 46.9 | | 49 | 54.86 | 56.6 |
| | 56 | 49.3 | 46.9 | | 364 | | 56.6 |
| 8 | 19.71 | 21.64 | 49.6 | 8 | 49.6 | 25.35 | 45.5 |
| | 35.5 | 48.71 | 49.6 | | 74.5 | 39 | 45.5 |
| | 100 | 40.15 | 49.6 | | 53 | 41.81 | 45.5 |
| | 51.2 | 51.7 | 49.6 | | 33.56 | 13.33 | 45.5 |
| 9 | 31.67 | 31.1 | 45.1 | 9 | 22.75 | 15.45 | 53.6 |
| | 48 | 29.26 | 45.1 | | 29.08 | 48.07 | 53.6 |
| | 25.67 | 17.29 | 45.1 | | 15.4 | 12.87 | 53.6 |
| | 49.67 | 55.24 | 45.1 | | 101 | 289.7 | 53.6 |
| 10 | 41.14 | 32.27 | 40.7 | 10 | 12.89 | 9.29 | 47.2 |
| | 26 | 17.72 | 40.7 | | 11.75 | 8.32 | 47.2 |
| | 17.75 | 8.226 | 40.7 | | 15.5 | 11.28 | 47.2 |
| | 20.71 | 11.84 | 40.7 | | 19.88 | 20.88 | 47.2 |
| 11 | 39.67 | 34.63 | 56.1 | | | | |
| | 48 | 36.81 | 56.1 | | | | |
| | 29.11 | 17.35 | 56.1 | | | | |
| | 55.33 | 51.67 | 56.1 | | | | |
| 12 | 30.89 | 23.35 | 50.3 | | | | |
| | 22 | 23.25 | 50.3 | | | | |
| | 32 | 23.1 | 50.3 | | | | |
| | 40.67 | 25.88 | 50.3 | | | | |
| (Mean ± S.D.) | | | | | | | |
| 36.89 ± 18.37* | | 29.17 ± 20.17 | | 60.30 ± 71.62* | | 45.85 ± 54.18 | |

SNCV, sensory nerve conduction velocity ; MSA, muscle sympathetic nerve activity ; LSCS, lumbar spinal canal stenosis. There was a statistically significant difference in the mean intervals of the patients and the normal controls (*$p < 0.05$).

al.[8] reported that MSA obtained from the tibial nerve at rest had significantly higher frequency in elderly subjects (aged 65～75 years) who do not have any distinguishable diseases than in young subjects (aged 18～25 years). In this study, age-matched healthy volunteers were used as controls, because aging is a very important factor in analyzing MSA data from LSCS patients, who are usually from the older generation[16].

Table 5 Mean interval and the standard deviation of the MSA and SNCV in individual cases in the LSCS patients and the normal subjects

| LSCS patients | | | | normal subjects | | | |
|---|---|---|---|---|---|---|---|
| | Mean interval (ms) | S.D. (ms) | SNCV (m/s) | | Mean interval (ms) | S.D. (ms) | SNCV (m/s) |
| No. | | | | No. | | | |
| 1 | 55.71 | 65.57 | 34.1 | 1 | 37.31 | 29.29 | 50.8 |
| 2 | 22.45 | 6.61 | 28.8 | 2 | 86.67 | 83.95 | 51.7 |
| 3 | 21.17 | 19.97 | 69.0 | 3 | 32.11 | 25.80 | 53.1 |
| 4 | 22.81 | 17.43 | 41.4 | 4 | 80.15 | 101.04 | 61.2 |
| 5 | 22.11 | 26.00 | 48.4 | 5 | 48.42 | 38.57 | 52.2 |
| 6 | 46.70 | 39.39 | 46.9 | 6 | 29.85 | 33.81 | 48.0 |
| 7 | 43.57 | 32.34 | 46.9 | 7 | 35.68 | 58.56 | 56.6 |
| 8 | 37.20 | 43.04 | 49.6 | 8 | 49.18 | 32.25 | 45.5 |
| 9 | 36.13 | 32.72 | 45.1 | 9 | 22.15 | 29.33 | 53.6 |
| 10 | 22.69 | 16.48 | 40.7 | 10 | 29.94 | 25.50 | 47.2 |
| 11 | 41.48 | 34.72 | 56.1 | | | | |
| 12 | 30.63 | 23.95 | 50.3 | | | | |

SNCV, sensory nerve conduction velocity ; MSA, muscle sympathetic nerve activity ; LSCS, lumbar spinal canal stenosis.
The mean value of the MSA intervals throughout the individual experiments was proposed for regression analysis with the value of SNCV.

According to the individual discharges of MSA during this study, the intervals of MSA in the LSCS patients were shorter than those in the normal group. The shorter MSA intervals took the place of a much larger number of MSA discharges. Short-interval MSA is probably associated with simultaneous high activity of the cutaneous sympathetic nerve. Though there was no statistical difference in the standard deviations of MSA intervals, the LSCS patients had narrower standard deviations than the normal controls. Larger standard deviations suggest a large variety of sympathetic nerve functions. It seems that in the LSCS patients, who have narrow standard deviations, the regulatory mechanism for the nervous systems was unable to regulate their sympathetic nerve functions promptly to maintain a healthy condition.

Taylor[17] and Matsumoto et al.[10] proposed that SNCV in most peripheral nerves becomes markedly slower after the age of 50 years old. Robertson et al.[18] noted that the slowing of SNCV in the sciatic nerve of mice was associated with a loss of large myelinated fiber and atrophy of the axons. In contrast, the small myelinated and unmyelinated fibers showed no histopathological changes[19]. In our study, SNCV in the patient group was slower than in the controls, which would indicate that chronic compression of the nerve roots and cauda equina cause strong atrophy and degeneration of axons and demyelination of the nerves.

The correlation between SNCV and the standard deviation of MSA intervals reveals a relationship between the somatic and sympathetic nervous systems. There has not been any strong evidence to indicate a relationship between these nervous systems until now, but a positive correlation between them was clearly shown in the regression analysis of our study: the faster the SNCV, the wider the range of fluctuation of MSA intervals in normal subjects. This may be partly a reflection of central nervous regulatory mechanisms. In subjects with fast SNCV, more afferent impulses are conducted in a certain time to the spinal cord and the supraspinal center than in subjects with slow SNCV. It would not be efficient for the autonomic nervous system to respond to each impulse. And to decrease the vast amount of energy required to respond to them, the sympathetic nervous system may protect itself by maintaining a wide range of fluctuation. This means that it organizes a tolerance against each stimulus conducted through the peripheral nerves.

In the lower lumbar spine, the cauda equina and

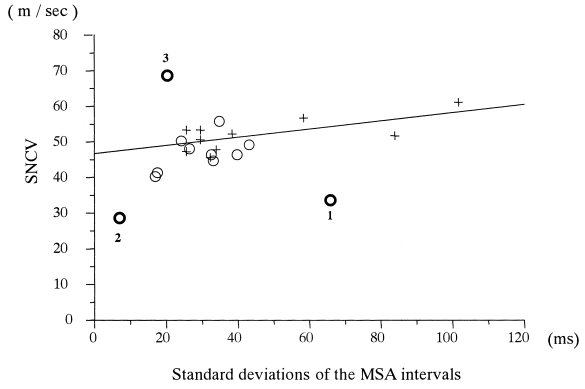


Fig. 4 Correlation between the SNCV and the standard deviations of MSA discharge intervals in individual LSCS patients and normal subjects

Open circle, LSCS (n=12); cross, control (n=10)
The solid line is the regression line of the normal subjects.
SNCV, sensory nerve conduction velocity; MSA, muscle sympathetic nerve activity; LSCS, lumbar spinal canal stenosis

A positive correlation was found in the normal subjects (r=0.684, p=0.029). There was no significant correlation in the LSCS patients (r=0.031). Patient No. 1, 2 and 3 were far out of the normal regression line.

nerve roots do not make a direct connection to sympathetic fibers[20]. Many patients with LSCS still maintain a correlation between SNCV and the standard deviation of MSA, but in a few patients with LSCS, the correlation between both parameters seemed to be disrupted. These patients were among the older of the patients, and their spinal stenoses were in the relatively higher levels of the spinal canal. Furthermore, their disease had generally lasted longer than the mean period of the disease (**Table 2**). Even though there are no direct transmissions to the sympathetic nerves from the cauda equina or nerve roots, when stenosis levels are very close to the level where there is direct connection between them, coordination between the two nervous systems is obstructed. Suffering from the disease for long periods could also have an unfavorable effect on the regularity of the two systems.

In animal models of chronic nerve compression, Wall et al.[14] noted that chronic dorsal root compression enhanced spontaneous excitability of dorsal root ganglion (DRG) cells in rats, resulting in the occurrence of ectopic firing. Howe et al.[7] found that rabbits with sciatic nerve injuries showed spontaneous ectopic firings and hypersensitization for mechanical stimuli. These findings lead to the hypothesis that when the cauda equina or peripheral nerves are chronically compressed in humans, the ectopic discharges generated impair these nerves. These ectopic discharges might reflect sympathetic nerve activity by increasing the discharges of MSA. Furthermore, nervous system dysfunctions such as increasing adrenosensitivity in impaired sensory nerve fibers have been reported[1,14]. This is probably due to the physiological changes in the nerve membrane. It has also been reported that in squid, ephaptic transmission is sometimes organized in the peripheral nerves[21]. We do not have any clear evidence in humans to describe these special connections between disordered peripheral nerves. However, it is still worthwhile to propose a hypothesis that in chronically compressed nerves the physiological and anatomical membrane condition changes gradually, and that finally ephaptic transmission or pseudosynapse is produced between the somatic and sympathetic nerves. Sympathetic innervation of DRG following nerve injury[2] has also been reported. These facts contribute to increased afferent impulses, disruption of the interaction between the sympathetic nervous system and the sensory nervous system, and finally to an increase in MSA.

We conclude that the high activity of these sympathetic nerve functions may result in the clinical symptoms in LSCS patients in terms of abnormal sensations (dysesthesia and paresthesia: tingling, tickling and psychroesthesia).

In summary, we examined SNCV and MSA in both LSCS patients and normal subjects. The LSCS patients had shorter MSA intervals and narrower fluctuations of MSA than the normal subjects. As for the range of fluctuation of the MSA intervals and SNCV, the faster the SNCV, the wider the range of fluctuation of MSA intervals in the normal subjects. However, many patients with LSCS seem to maintain a correlation, as among the normal subjects. It was suggested that in some LSCS patients who were rather old, whose spinal stenosis was in the relatively higher levels of the spinal canal, and whose disease periods

were longer, the correlation between the sympathetic nervous systems and somatic nervous systems was disrupted. If the nerve roots and cauda equina are chronically compressed, coordination between the sympathetic nerve and somatic nerve systems is disrupted. We believe that this disrupted coordination is one of the reasons why abnormal sensations occur in the lower extremities of LSCS patients.

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