J Anesth (1994) 8:32–35

Journal of
Anesthesia

Effects of continuous epidural block on motor nerve conduction velocity in patients with lower spine disorders

SUMIHISA AIDA, TOSHIYUKI TOBITA, and KOKI SHIMOJI

Department of Anesthesiology, Niigata University School of Medicine, 1-757 Asahimachi, Niigata, 951 Japan

Abstract: Thirty-one patients with severe low back pain were treated by continuous epidural block for 18 ± 3 (mean ± SEM) days. Motor nerve conduction velocity (MCV) of the common peroneal nerve was measured before and after the treatment. After the treatment, the visual analogue scale score (VAS) and straight leg-raising (SLR) test were markedly reduced ($P < 0.01$), and MCV was increased significantly ($P < 0.001$). A significant correlation ($P < 0.01$) between the SLR test and MCV was found before the treatment. A significant correlation ($P < 0.001$) between VAS and MCV was demonstrated after treatment. However, in three patients who showed no reduction in VAS even after the treatment, MCV became significantly ($P < 0.05$) slower in spite of nearly normal SLR test results. These results suggest that epidural block treatment improves not only pain but also MCV, and that two parameters, SLR test and pain intensity, are related closely to the MCV.

Key words: Motor nerve conduction velocity, Low back pain, Sciatica, Straight leg-raising test, Lower spine disorder

Introduction

In patients with low back pain, sensory and/or motor nerve dysfunction of the lower limbs, such as sciatica, paresthesia, muscle spasm (or spasticity), and gait disturbance are frequently observed. These symptoms are considered to originate directly or indirectly from local damage to the nerve tissue, such as compression, decreased local blood flow, adhesion, edema, and/or inflammation [1,2]. Furthermore, a decrease in the conduction velocity in peripheral nerves has been recorded in patients with sciatica [3,4] and cauda equina lesions [5]. Therefore, a relationship between nerve conduction velocity and severity of signs and symptoms is thought to exist, and it is expected that slowed nerve conduction velocity may recover after epidural block therapy. However, there are no reports in the literature which address this problem. The present study, therefore, was undertaken to test the relationship between motor nerve conduction velocity (MCV) and severity of signs and symptoms in patients with low spinal disorders before and after the treatment.

Address correspondence to: S. Aida

Received for publication on March 1, 1993; accepted on April 30, 1993

Materials and methods

After receiving approval from the institutional ethics committee, 31 patients aged 65 ± 6 (mean ± SEM, range 33–85), 17 women and 14 men, hospitalized with severe low back pain, were included in this study. All procedures, risks and benefits pertaining to this study were explained to the patients, and each of them signed a written informed consent form. Diseases causing low back pain were lumbar disc herniation ($n = 11$, group A), other low spinal disorders including facet syndrome, spondylolisthesis, spondylosis, or spinal canal stenosis ($n = 11$, group B), and a combination of the disorders in groups A and B ($n = 9$, group C). Patients with low back pain due to tumors were excluded. The patients were diagnosed by analysis of plain radiography, myelography, computed tomography (CT) and/or magnetic resonance imaging (MRI). An epidural catheter made of polyvinyl chloride (1.0 mm in external diameter, Hakko, Tokyo, Japan) was introduced into the epidural space between the second and third lumbar vertebrae and advanced 100 mm from the tip of the Tuohy needle in a cranial direction. Procaine hydrochloride (1.0%, 10 ml) was administered through the catheter into the epidural space once a day, and the catheter was kept in place for 18 ± 3 (range, 12–24) days.

Pain was assessed using a visual analogue scale score (VAS). Although the VAS of all the patients was high (more than 7) before the treatment, the VAS varied and ranged from 0 to 10 after the treatment. The patients

were then divided retrospectively into 4 classes according to the intensity of pain in terms of VAS: I, 0–2.5; II, 2.5–5; III, 5–7.5; and IV, 7.5–10. A straight leg-raising (SLR) test was also performed on both sides.

MCV of the bilateral common peroneal nerve was measured under a controlled room temperature of 23°–25°C. Through two pairs of nonpolarizable surface electrodes 10 mm in diameter (one on the popliteal space and the other on the fibular neck), the nerves were stimulated supramaximally and orthodromically by a square pulse, 0.1 ms in dration, delivered from a stimulator (SMP-3100, Nihon Kohden, Tokyo, Japan) once per second. Evoked potentials from the extensor digitorum brevis muscle were amplified and recorded by a computer (MEB-3102, Nihon Kohden). MCV was calculated from differences in the latencies of the onset time of the evoked potentials and the distance between the two sets of stimulating electrodes (Fig. 1) [6].

All parameters were measured before the introduction and after the extraction of the epidural catheter, when no direct effect of local anesthetic or epidural blockade was observed. Values were expressed as the mean and standard error of the mean (mean ± SEM). Data were analyzed statistically using Student's *t*-test, Mann-Whitney U-test, or Spearman's rank correlation coefficient (Rs). Differences at $P < 0.05$ were considered significant.

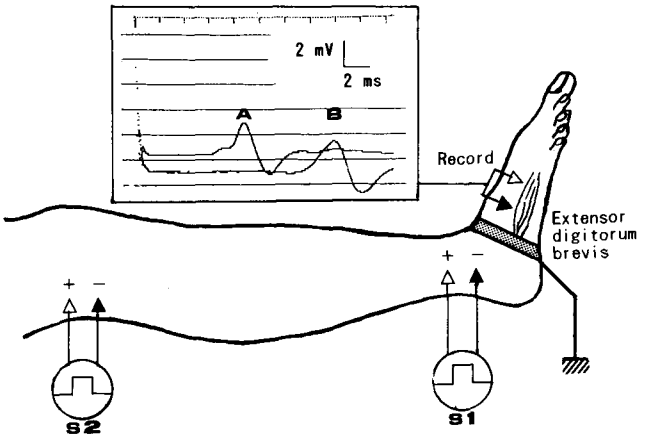

Fig. 1. Method used to stimulate the common peroneal nerve and record the response from the extensor digitorum brevis muscle. The nerve was stimulated supramaximally and orthodromically. A ground electrode was placed around the ankle. Evoked potential *A* was recorded in response to distal stimulation (*S1*) and *B* was recorded in response to proximal stimulation (*S2*)

Results

In all patients, MCV was increased significantly ($P < 0.01$) after the treatment. MCV in group A was increased significantly ($P < 0.001$), and MCV in class I ($P < 0.001$) and II ($P < 0.05$) patients was also increased significantly following the treatment. However, in class IV patients, MCV became significantly ($P < 0.05$) slower even after the treatment. On the other hand, the maximum amplitudes of evoked potentials in all patients as well as in the respective groups and classes were not significantly altered (Table 1, Fig. 2).

SLR tests in all patients ($P < 0.001$) as well as the groups A ($P < 0.001$) and C patients ($P < 0.01$) (these groups A and C showed SLR test positivity) showed significant recovery after the treatment. SLR tests of the class I ($P < 0.001$), II ($P < 0.05$) and III ($P < 0.05$) patients were improved significantly after the treatment, whereas that of the class IV patients was almost normal before and after the treatment. VAS for all

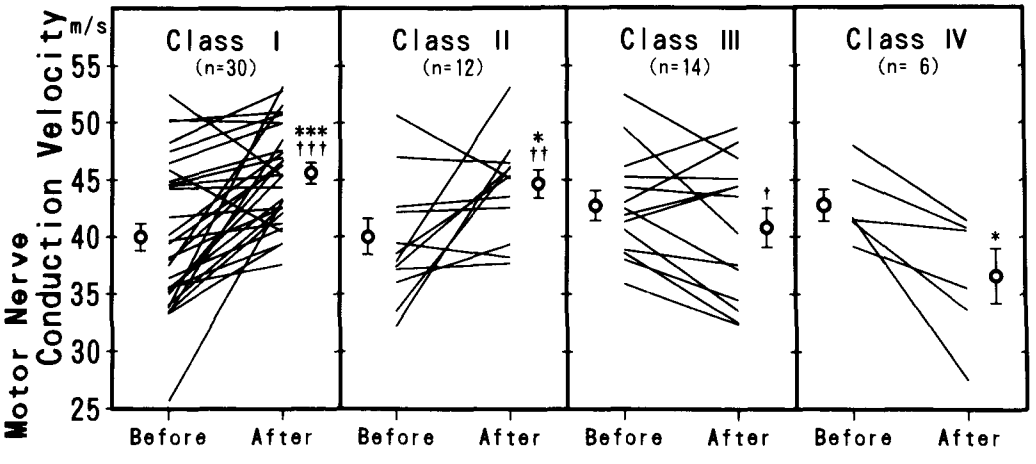

Fig. 2. Recovery of motor nerve conduction velocity (MCV) in each class. $^{*}P < 0.05$, $^{***}P < 0.001$ between before and after treatment. $^{\dagger}P < 0.05$, $^{\dagger\dagger}P < 0.01$ and $^{\dagger\dagger\dagger}P < 0.001$ compared with class IV after the treatment. *Lines* represent respective cases, and *circles* with *vertical bars* represent mean and standard error of the mean (SEM)

Table 1. Effects of the epidural block on motor nerve conduction velocity (MCV), amplitude of evoked electromyogram, straight leg-raising (SLR) test and visual analogue scale score (VAS)

		MCV (m/s)		Amplitude (mV)		SLR Test (°)		VAS	
	n	Before	After	Before	After	Before	After	Before	After
Group									
A	22	40.6 ± 1.3	46.4 ± 2.1***	2.32 ± 0.23	2.44 ± 0.22	50 ± 8	89 ± 9***	8.5 ± 0.3	1.3 ± 0.4***
B	22	41.7 ± 1.1	43.2 ± 1.1	1.99 ± 0.18	2.24 ± 0.25	93 ± 1	92 ± 1	9.5 ± 0.2	3.0 ± 0.2**†
C	18	40.1 ± 1.5	40.5 ± 1.4††	2.04 ± 0.16	2.02 ± 0.12	70 ± 4	80 ± 3**	9.1 ± 0.4	5.8 ± 0.9**††
Class									
I	30	40.0 ± 1.2	45.7 ± 0.8***¶¶	2.07 ± 0.18	2.23 ± 0.17	67 ± 5	89 ± 1***	8.9 ± 0.2	0.6 ± 0.1***¶¶
II	12	40.0 ± 1.7	44.7 ± 1.2*¶	2.17 ± 0.24	2.44 ± 0.35	71 ± 6	91 ± 1*	9.1 ± 0.2	3.1 ± 0.2***¶¶
III	14	42.8 ± 0.7	40.8 ± 1.7	2.08 ± 0.22	2.15 ± 0.23	71 ± 5	79 ± 3*	9.1 ± 0.3	6.4 ± 0.2***¶¶
IV	6	42.8 ± 1.3	36.6 ± 2.2*	2.33 ± 0.42	2.16 ± 0.64	88 ± 4	88 ± 4	9.7 ± 0.2	9.4 ± 0.2
In All	62	40.9 ± 0.7	43.5 ± 0.7**	2.12 ± 0.11	2.25 ± 0.13	71 ± 3	89 ± 1***	9.0 ± 0.2	3.2 ± 0.6***

* $P < 0.01$, ** $P < 0.01$ and *** $P < 0.001$ within a group before and after treatment. † $P < 0.05$ and †† $P < 0.01$ compared with group A. ¶ $P < 0.05$ and ¶¶ $P < 0.001$ compared with group IV. Student's *t*-test was used. Values are given as mean ± SEM. Respective measurements, except for VAS (total $n = 31$), were from both sides.

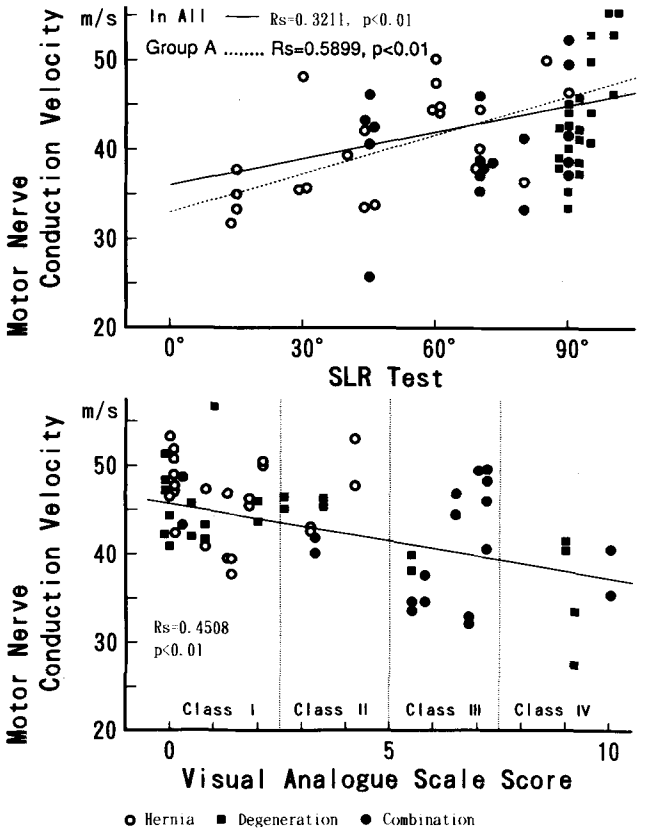

Fig. 3. Relationship between straight leg-raising (SLR) test and MCV before the treatment (*upper*), and between visual analogue scale score (VAS) and MCV after the treatment (*lower*). Group A, (*open circles*); group B, (*closed squares*) and group C, (*closed circles*). A significant Spearman's rank correlation (Rs) between SLR test and MCV was demonstrated in all patients (Rs = 0.3211, $P < 0.05$) as well as group A (Rs = 0.5899, $P < 0.01$) before the treatment. On the other hand, a significant Spearman's rank correlation (Rs = −0.4508, $P < 0.01$) between VAS and MCV was shown in all patients. Respective measurements were from both sides

groups as well as for the class I, II, and III patients were significantly (all $P < 0.001$) reduced after the treatment, although reduction of the VAS was not seen for the class IV patients (Table 1).

Significant correlations between SLR and MCV in all patients (Rs = 0.3211, $P < 0.05$) as well as in group A patients (Rs = 0.5899, $P < 0.01$) were demonstrated before the treatment. After the treatment, however, a significant correlation (Rs = −0.4508, $P < 0.01$) was demonstrated between VAS and MCV in all patients (Fig. 3).

Discussion

The present study as well as other reports [3–5] suggest that MCV is slowed in various spinal disorders, and that epidural block treatment not only improves pain and SLR test but also leads to recovery of MCV (Table 1, Fig. 2). MCV recovery is thought to parallel the improvement of pain and SLR (Fig. 3). Slowing of the nerve conduction velocity distal to tourniquet compression has been demonstrated experimentally [7], and disc herniation is known to compress the nerve root [1,2]. A significant correlation between SLR and MCV was found in all subjects including those in group A (Fig. 3). Therefore, it is suggested that slowing of MCV is caused by compression of nerve tissue, such as with disc herniation. However, MCV slowing did not seem to be attributable to disk herniation alone, since MCV became slowed even further even though SLR was nearly normal (mean 88°) in the class IV patients who showed no reduction in pain intensity even after the treatments (Table 1). Therefore, several other intraspinal factors, such as spinal canal atresia, a decrease in local blood flow, adhesion, edema, and/or inflammation [1,2], which might be manifested as pain, may contribute to the

slowing of MCV. This is supported by the significant correlation between VAS and MCV in the present study (Fig. 3).

Whereas the intensity of pain may represent the sum of intraspinal damage due to local pressure, a blood flow decrease, adhesion, edema and/or inflammation, abnormal SLR results may be more closely associated with root compression [1,2]. At the acute stage (before treatment), the intensity of pain was roughly equal among the patients and this was reflected in the narrow range of VAS scores (7–10), whereas after the treatment the SLR test recovered to nearly normal levels in most patients and the intensity of pain became wider and was separated into 4 classes. Therefore, a significant correlation was noted between SLR and MCV before the treatment, whereas the correlation between VAS and MCV became significant after the treatment. These phenomena support the existence of two independent factors which slow the MCV, i.e., root compression and intraspinal damage.

Epidural block is commonly applied for low back pain and sciatica [2]. The resulting pain relief is considered to be due mainly to increased local blood flow following sympathetic blockade [8–10]. An increase in local blood flow is thought to ameliorate local inflammation and edema [2], to decrease local pain-inducing substances [11–13], and to lower local pressure. The increase in the local blood flow is considered to break the vicious cycle of pain [14].

Although neural damage may occur at the intervertebral foramen and/or spinal canal, slowing of MCV in the present study seemed to be induced closer to the periphery at the common peroneal nerve. Distal MCV slowing recovered after epidural block at the site of the injured nerve. These facts suggest that an axonal disorder can affect even remote peripheral sites [3–5]. To maintain normal electrical activity at the axonal membrane, some substances are transported from the soma to more peripheral portions in the axon, via axoplasmic transport [15,16]. Thus, the axoplasmic transport might be disturbed in lower spine disorders, leading to the dysfunction of the peripheral motor nerve and eventually resulting in slowed MCV. On the other hand, pain can be caused by decreased blood flow in the lower extremities mediated by sympathetic reflexes [14] in lower spine disorders [2], and insufficient blood flow is thought to slow MCV. The epidural block is considered to alleviate these conditions by its effects on pain and the sympathetic nerves [8–10].

In the present study, the amplitude of evoked potential recorded from the muscle was not significantly altered (Table 1). This seems to indicate that the effects of the neural disturbances hardly affected the muscle in our patients. On the other hand, recovery in both the VAS and MCV was delayed significantly in groups B and C as compared with that in group A (Table 1, Fig. 2). This suggests that improvements in MCV, SLR, or pain in group A can be achieved more easily than MCV and pain in groups B and C as well as in the class IV patients. The disorders in groups B and C might be associated with degenerative changes in spinal and/or neural tissues.

Thus, it can be assumed that patients with spine disorders who show a slower MCV even after epidural block have degenerative changes in the nervous tissue. In such patients, recovery of nerve function by epidural block is considered to be difficult, and the dysfunction may become chronic. These results may be of help in the evaluation of prognosis or application of the epidural block in spine disorders. Considering that MCV can be measured very easily [3,4], MCV is considered to be an important indicator of nerve tissue damage.

References

1. Austin G (1972) The spinal cord, 2nd edn. Thomas, Springfield
2. Loeser JD, Bigos SJ, Fordyce WE, et al. (1990) Low back pain, The Management of Pain, 2nd edn, Vol II. Bonica JJ (ed) Lea and Febiger, Philadelphia, pp 1448–1483
3. Ertekin C, Ertekin N, Karcioglu M (1975) Conduction velocity along human nociceptive reflex afferent nerve fibers. J Neurol Neurosurg Psychiat 38:959–965
4. Kaplan PE (1982) A motor nerve conduction velocity across the lumbosacral plexus. Electromyogr Clin Neurophysiol 22:527–530
5. Swash M, Snooks SJ (1986) Slowed motor conduction in lumbosacral nerve roots in cauda equina lesions: a new diagnostic technique. J Neurol Neurosurg Psychiat 49:808–816
6. McComas AJ (1977) Neuromuscular function and disorders. Butterworth, New York, pp 24–26
7. Fowler TJ, Danta G, Gilliat RW (1972) Recovery of nerve conduction after a pneumatic tourniquet: observations on the hindlimb of the baboon. J Neurol Neurosurg Psychiatry 35:638–647
8. Kennedy Jr WF, Sawyer TK, Gerbershagen HU, et al. (1969) Systemic cardiovascular and renal hemodynamic alteration during peridural anesthesia. Anesthesiology 31:414–421
9. Peters J, Kutkuhn B, Medert HA, et al. (1990) Sympathetic blockade by epidural anesthesia attenuates the cardiovascular response to severe hypoxia. Anesthesiology 72:134–144
10. Kerkkamp HEM, Gielen MJM (1991) Cardiovascular effects of epidural local anaesthetics. Anaesthesia 46:361–365
11. Fields HL (1987) Pain. McGraw-Hill, New York, p 36
12. Inoki R, Toyoda T, Yamamoto I (1973) Elaboration of a bradykinin-like substance in dog's canine pulp during electrical stimulation and its inhibition by narcotic and non-narcotic analgesics. Naunyn Schmiedeberg's Arch Pharmacol 279:387–398
13. Dubner R, Price DD, Beitel RE, Hu JW (1977) Peripheral neural correlates of behavior in monkey and human related to sensory-discriminative aspects of pain. In: Anderson DJ, Matthews BP (eds) Pain in the trigeminal region. Elsevier, Amsterdam, pp 57–66
14. Bonoca JJ (1990) General consideration of chronic pain. In: Bonica JJ (ed) The Management of Pain, 2nd edn, Vol I. Lea and Febiger, Philadelphia, pp 180–208
15. Ochs S (1974) Systems of material transport in nerve fibers (axoplasmic transport) related to nerve function and tropic control. Ann NY Acad Sci 228:202–223
16. Willis WD, Grossman RG (1981) Medical neurobiology. Mosby, St Louis, pp 25–26