Kawasaki Med. J. Vol. 1: 133-143, 1975

SENSORY NERVE CONDUCTION VELOCITIES IN PATIENTS WITH VARIOUS NEUROLOGICAL DISORDERS

Akira TERAO, Nobuzugu NOMURA, Satoru TAWARA, Kenji MORIMOTO, and Shukuro ARAKI

Division of Neurology, Department of Medicine, Kawasaki Medical School, Kurashiki, 701-01, Japan.

Accepted for publication on Dec. 2, 1974

Abstract

Median and posterior tibial nerve sensory conduction velocities (SCVs) were measured orthodromically through surface electrodes. In 21 normal controls, the median nerve SCV from wrist to elbow was 67.7 m/sec (±1SD 4.7) and from finger to wrist was 45.4 m/sec (±1SD 5.6), and the posterior tibial nerve SCV from ankle to knee was 55.7 m/sec (±1SD 6.3) and from toe to ankle was 40.2 m/sec (±1SD 4.5).

In patients with various neurological disorders, the median nevre SCVs were recorded in 70 cases and posterior tibial nerve SCVs in 42 cases. SCVs were markedly reduced or not recorded in cases with severe neuropathies. In the neuropathies with relatively milder clinical symptoms, some cases revealed abnormally reduced SCVs and others SCVs of within normal limit. The similar discordances between clinical manifestations and SCVs were found in a case of Charcot-Marie-Tooth disease and in some cases with uremia or pernicious anemia. The results suggested there was a discrepancy between clinical manifestations and SCVs in milder neuropathies. To evaluate SCVs, several factors, viz., states of patients, clinical findings, and nature of disorders must be considered.

INTRODUCTION

The measurement of the human peripheral nerve conduction velocities is one of the most important clinical examinations in evaluating peripheral nerve dysfunction. Motor nerve conduction studies are now well established, sensory nerve conduction measurements, however, have been less frequently reported. Gilliatt[1] and Buchthal[2] reported sensory nerve fibers were often affected earlier than motor ones in mixed type polyneuropathy or sensory neuropathy, and then, studies of sensory nerve conduction velocities (SCVs) are indispensable to clarify the peripheral nerve dysfunction of various polyneuropathies.

In the present study, SCVs were measured in the distal and proximal portion of each median and posterior tibial nerves in patients

with various neurological disorders, and correlative studies between the electrophysiological data and clinical findings were presented. Measurement of SCV have been simplified by a digital computer, and orthodromic SCVs were recorded using superficial skin electrodes without pain or hazardous infections.

METHODS

Patients of various neurological disorders with and without clinical evidence of neuropathies were investigated. Median nerve SCVs were measured in 70 cases and posterior tibial nerve SCVs in 42 cases. A group of 21 normal subjects were used as control. Sixteen were male and five female. Age ranged from second to third decade.

Sensory nerve potentials were evoked by stimuli through surface electrodes on middle finger in median nerve and on great toe in posterior tibial nerve, and were recorded orthodromically through a pair of surface electrodes consisting of 4×25 mm silver strips with an interelectrode distance of 30 mm placed on the nerve at the wrist and elbow joint in median nerve and at the ankle and knee joint in posterior tibial nerve. The stimulus was provided by a SANEI 3F-31 stimulator using a voltage of 70 volts and a stimulus duration ranging from 0.3 to 1.0 msec.

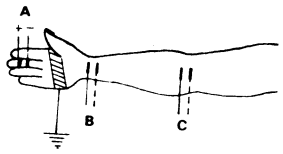
The responses were fed into an oscilloscope triggered by each stimulation and 20-500 responses were averaged with a digital computor 7T06. The interval between the stimulus artifact and the initial negative peak was taken as the latency.

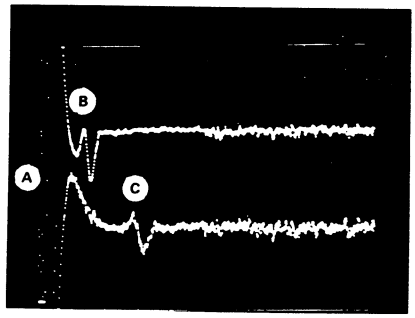
The temperature of the skin over the nerves was 31.0-34.4°C on the forearmes and 29.8-35.1°C on the legs in normal controls. A significant fall of the temperature was observed in no case with neurological disorders.

RESULTS

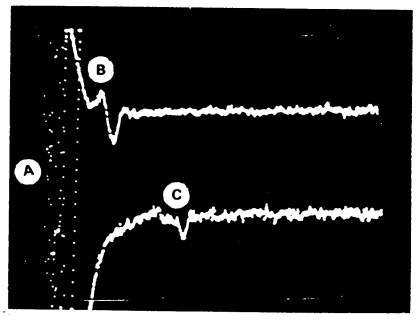
Most of the nerve action potentials (NAPs) of normal median nerves were biphasic and revealed negative sharp peaks followed by gradually descending deep positive ones (Fig. 1). The positive peaks became more marked than negative ones at the elbow joints. Posterior tibial NAPs revealed also similar wave form as median ones, though they were less distinct despite of 500 averaging (Fig. 2).

In the normal controls, SCVs ranged from 61.7 to 77.8 m/sec (mean 67.7, SD 4.7 m/sec) in the proximal and from 36.4 to 54.0 m/sec (mean 45.4, SD 5.6 m/sec) in the distal segments of the median nerves and from 40.9 to 70.7 m/sec (mean 55.7, SD 6.3 m/sec) in the proximal and from 30.0 to 46.5 m/sed (mean 40.2, SD 4.5 m/sec) in the distal segments of





26 yrs. Normal male, SCV : 70.0 m/sec (W - E)

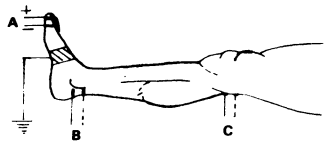


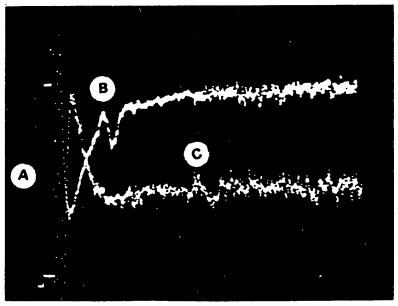
16 yrs. CMT Disease female, SCV : 43.3 m/sec (W - E)

Fig. 1. Nerve action potentials (NAPs) of median nerve in normal person and in patient with Charcot-Marie-Tooth disease.

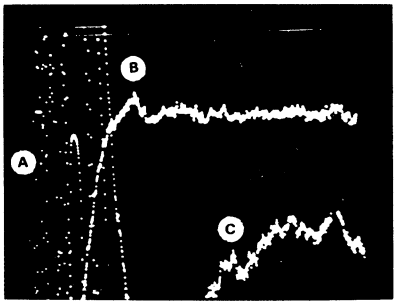
the posterior tibial nerves. The distal latencies ranged from 2.4 to 3.5 m/sec (mean 2.8, SD 0.3 m/sec) in the median nerves and from 3.7 to 6.0 m/sec (mean 4.7, SD 0.6 m/sec) in the posterior tibial nerves.

In a group of patients with neuropathies, motor neuron disease, progressive muscular dystrophy (DMP), uremia, pernicious anemia, and other neurological diseases, median nerve SCVs were measured in 70 cases and posterior tibial nerve SCVs in 42 cases. The distal latencies of





26 yrs. Normal male, SCV : 59.2 m/sec (A-K)



16 yrs. CMT disease. SCV : 45.0 m/sec (A-K)

Fig. 2. NAPs of posterior tibial nerve in normal person and in patient with Charcot-Marie-Tooth disease.

NAPs were also measured in both nerves. Posterior tibial NAPs were not evoked in 15 cases in the proximal and in four cases in both proximal and distal segments. They included one case of toxic polyneuropathy due to glue sniffing and three cases of SMON, viz., subacute myelo-optico-neuropathy.

For the convenience of evaluation of SCVs, normal limit SCVs were assumed tentatively above the values of mean SCV minus the standard

deviation (SD), borderline ones below normal limit to mean SCV minus twofold SD, and abnormal ones below borderline. Fig. 3 and Fig. 4 revealed the distribution of SCVs and distal latencies in median and in posterior tibial nerves in the group of patients with various disorders. Then, the all cases were divided into three groups by the gradings of SCVs above mentioned in median nerve (Table 1-3) and in posterior tibial nerve (Table 4).

TABLE 1.

Sensory Conduction Velocities of Median Nerve
(Between wrist and elbow)

Diseases	Age	SCV (m/sec)	
A L S	47	67.2	
〃	41	63.0	
〃	45	64.3	
〃	40	63.2	
〃	49	68.4	
S P M A	52	66.9	
〃	61	67.9	
D M P	31	68.8	
〃	20	70.0	
Periodic Paralysis	26	63.8	
〃 〃	22	65.0	
Cerebellar Ataxia	56	62.7	
Epilepsy	22	65.7	
Parkinsonism	62	66.6	Normal
〃	53	64.5	
Tabes dorsalis	37	63.1	
C V D	61	67.1	
〃	43	64.2	
Myelopathy	44	65.7	
S M O N	59	70.6	
Diabetic Polyneuropathy	44	67.1	
Polyneuropathy (unknown cause)	68	66.1	
〃	41	63.9	
〃	56	63.9	
Polyneuritis	21	65.8	
〃	25	70.9	
Brain tumor	57	66.0	

ALS: amyotrophic lateral sclerosis, SPMA: spinal progressive muscular atrophy, DMP: prog. muscular dystrophy, CVD: cerebro-vascular diseases, SMON: subacute myelooptico-neuropathy, OPCA: olivopontocerebellar atrophy.

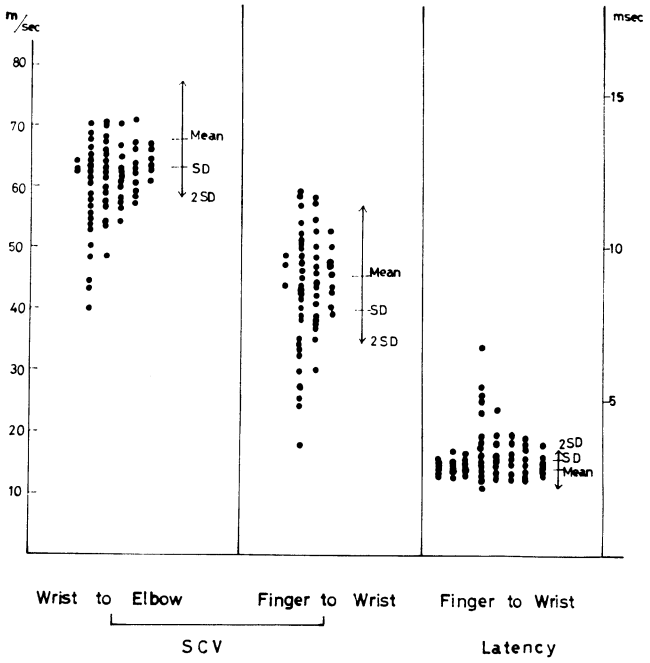


Fig. 3. Distribution of median nerve sensory conduction velocities (SCVs) and distal latencies in patients with various neurological disorders.

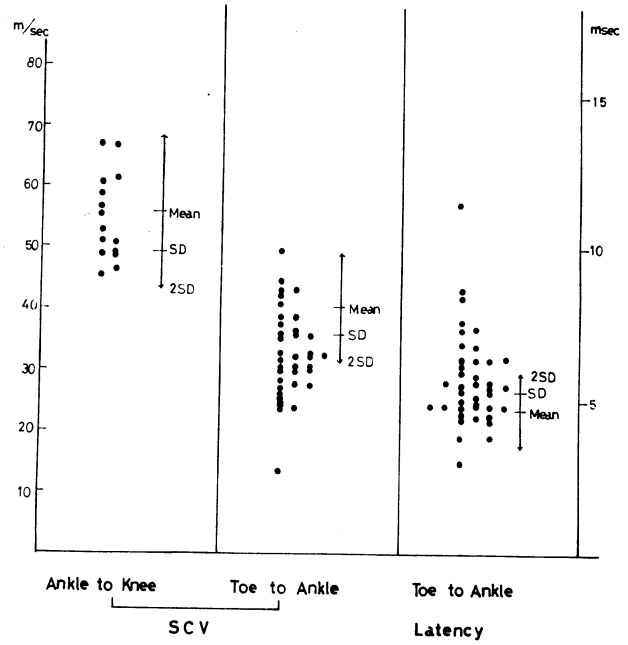


Fig. 4. Distribution of posterior tibial nerve SCVs and distal latencies in patients with various neurological disorders.

TABLE 2.

Sensory Conduction Velocities of Median Nerve
(Between wrist and elbow)

Diseases	Age	SCV (m/sec)	
Sylingomyelia	31	61.3	
A L S	58	60.6	
S P M A	58	62.8	
O P C A	61	62.9	
D M P	38	60.0	
〃	28	61.5	
Mytochondrial myopathy	63	62.5	
Myotonic Dystrophy	53	62.0	
〃 〃	43	60.2	
Scleroderma	44	59.1	
Parkinsonism	53	57.8	Borderline
〃	63	60.7	
Myxedematous Polyneuropathy	47	58.3	
Polyneuropathy (unknown cause)	41	63.9	
Brachial plexus injury	41	61.8	
Myelopathy	44	60.6	
Dermatomyositis	40	60.0	
Brain tumor	47	61.9	
Uremia	41	62.4	
〃	43	62.5	
Pernicious Anemia	69	59.3	

TABLE 3.

Sensory Conduction Velocities of Median Nerve
(Between wrist and elbow)

Diseases	Age	SCV (m/sec)	
C M T Disease	16	43.3	
D M P	49	53.9	
S P M A	48	56.0	
Parkinsonism	41	57.7	
Epilepsy	45	53.6	
C V D	52	56.8	
Myxedematous Polyneuropathy	67	57.2	
〃 〃	62	54.5	
Bond Polyneuropathy	19	39.8	
Diabetic Polynenropathy	65	53.7	
Polyneuropathy (unknown cause)	79	56.8	Abnormal
〃	61	48.1	
Cerebellar ataxia	48	57.1	
Uremia	30	55.4	
〃	64	56.8	
〃	54	52.9	
〃	29	50.0	
〃	24	57.8	
Pernicious Anemia	73	43.9	
〃 〃	59	52.7	
〃 〃	73	48.2	

TABLE 4.

Sensory Conduction Velocities of Tibial Nerve
(Between toe and ankle)

Diseases	Age	SCV (m/sec)	Latency (msec)	
D M P	31	44.7	3.8	Normal
Mytochondrial myopathy	63	36.4	4.5	
Periodic paralysis	26	40.6	4.8	
Cerebellar Ataxia	56	49.5	3.8	
Parkinsonism	53	43.1	2.9	
〃	53	42.4	4.2	
Epilepsy	22	43.0	4.3	
S M O N	68	38.8	4.8	
Diabetic Polyneuropathy	48	38.5	4.6	
Polyneuropathy (unknown cause)	62	37.3	4.8	
Pernicious Anemia	69	36.0	5.0	
Parkinsonism	53	32.9	6.3	Borderline
S M O N	68	32.4	4.5	
〃	67	32.1	5.6	
〃	59	35.3	4.9	
Diabetic Polyneuropathy	64	32.0	6.0	
〃 〃	65	31.3	4.8	
〃 〃	65	35.1	4.8	
Polyneuropathy (unknown cause)	31	32.1	5.6	
〃	60	35.4	4.8	
Uremia	41	32.2	5.4	
C M T Disease	16	24.2	6.2	Abnomal
D M P	28	30.7	5.2	
M S	44	29.7	6.4	
Epilepsy	45	27.1	5.8	
S M O N	55	23.1	7.6	
〃	47	28.2	5.5	
Diabetic Polyneuropathy	61	26.2	7.4	
〃 〃	68	27.2	6.8	
〃 〃	64	23.8	5.6	
Myxedematous Polyneuropathy	67	13.5	11.5	
〃 〃	62	30.0	5.5	
Polyneuropathy (unknown cause)	56	30.2	5.9	
Polyneuritis	16	30.7	4.4	
〃	37	24.9	6.9	
〃	11	27.7	5.6	
C V D	50	25.3	6.4	
Uremia	30	29.4	5.1	

SCVs were markedly reduced or were not able to measure in cases with severe neuropathies. Among the various neuropathies nine of 15 cases (between wrist and elbow) and 11 of 22 cases (between toe and ankle) revealed SCVs of borderline or within normal limit. Such cases had subjective sensory complaints but impairments of superficial or deep sensations were mild. One case of Charcot-Marie-Tooth disease, and several cases of pernicious anemia or uremia revealed abnormally reduced SCVs without sensory disturbances clinically. Mild reduction of SCVs were observed in some cases with various neurological disorders, viz., DMP, parkinsonism, epilepsy, etc.

DISCUSSION

In 1966, Buchthal and Rosenfalck[3] described a precise method to measure SCV by using needle electrodes. In this study, we adopted surface electrodes that were fairly useful clinically to record NAPs in patients with various neurological disorders as well as normal individuals. Though NAPs were hardly evoked to assess SCVs in some patients with severe neuropathies, NAPs in the distal segments of median and posterior tibial nerves were usually accessible to evaluate SCVs. The latencies in distal segments were also found to be beneficial as an indicator of peripheral nerve function.

There have been several reports about SCV in median nerve of normal persons: between finger and wrist 50.2±4.8 m/sec (Preswiek and Jeremy[4], 1964), 52.3±4.1 m/sec (Loong and Seah[5], 1971), 58.7±3.7 m/sec (Sato[6], 1972), 48.2±2.8 m/sec (Yoshida et al.[7], 1973), between wirst and elbow 64.5±4.8 m/sec (Buchthal and Rosenfalck[3], 1966), 69.1±2.7 m/sec[5] and 66.7±4.5 m/sec[7].

About SCVs in sciatic or sural nerve of normal persons, a few papers has been published: in sciatic nerve, between ankle and popliteal fossa (popl. foss.) 53.9±1.1 m/sec, between popl. foss. and buttock 62.3± 1.7 m/sec, and between ankle and buttock 56.8±1.1 m/sec (Buchthal and Rosenfalck[3], 1966), in sural nerve, between foot and high ankle, 44.0±4.7 m/sec and between foot and popl. foss. 50.0±3.9 m/sec (Shimozawa and Mavor[8], 1969), between ankle and popl. foss. 54.0±3.3 m/sec (Murai and Kuroiwa[9], 1973).

It has been noted that SCV is influenced by several factors, especially by skin temperature and the age of the subject. Buchthal and Rosenfalck[3] reported that between 36 and 21°C the conduction velocity decreased by 2 m/sec/°C, and that the SCV in the median and ulnar nerve was almost 15% slower in subjects 70-80 years than in subjects

18-25 years old. Asai et al.[10] reported the SCV in the median and ulnar nerve was 14 % in the former, 11 % in the latter slower in the subjects 50-83 years than in subjects 20-30 years old. From these results the standard SCV in our normal young adult may be shifted to a little faster side to evaluate SCVs in the aged subjects.

As already mentioned, SCVs were markedly reduced or were immeasurable in cases with severe neuropathies. In a group of patients with relatively milder clinical symptomes, however, some cases revealed abnormally reduced SCVs and others normal limit ones. Reviewing the relevant literatures about SCV, Murai and Kuroiwa[9] in SMON and peripheral neuropathies, Yoshida et al.[7] in various neurological disorders, Nöel[11] in diabetic neuropathy and Dyck et al.[12] in Charcot-Marie-Tooth disease, described the reduction of SCVs were not always parallel with the clinical sensory disturbances. These results including our findings suggest there is a discrepancy between subjective complaints or neurological findings and conductive function of sensory nerve fibers. There may be a clinical limit to the nerve conduction studies, because the presence of a cutaneous nerve action potential of normal latency and amplitude gives information only about A alpha fibers[13].

In a case with Charcot-Marie-Tooth disease, and in some case with uremia and pernicious anemia, as well, reduced SCVs were noted without clinical evidence of neuropathies. It is suggested that a state of subclinical neuropathy exists in renal insufficiency, similar to that described in diabetes mellitus and alcoholism[4]. In Charcot-Marie-Tooth disease, abnormal motor conduction was usually associated with clinical wasting but sensory nerve conduction abnormalities occured in the absence of clinical sensory loss in most patients studied by Humberstone[14]. In experimental neuropathies in which segmental demyelination is the major histological feature, conduction velocities in the peripheral nerves may be markedly reduced in contrast to those conditions in which axonal degeneration is the predominant pathological abnormality[15]. Thus, as segmental demyelination is a feature of Charcot-Marie-Tooth disease, the finding of severe abnormalities of nerve conduction is not surprising[16]. According to the report of Humberstone[14] it is possible that, although conduction will be slowed because of the loss of alpha fibers, lack of clinical sensory loss can be accounted for the presence of beta and gamma fibers in normal numbers or to excess, the excess of which may be due to regenerating alpha fibers which are smaller in diameter in the early stages of regeneration.

In some cases with parkinonism, DMP, or epilepsy, mild reduction

of SCVs were observed. There might be a certain possibility of peripheral nerve dysfunction in these neurological disorders. Recently, Eisen[17] reported the mean peroneal nerve (motor) and sural never (sensory) conduction velocities in the lower extremities of 45 patients treated with diphenylhydantion for more than 10 years showed a small but significant reduction compaired with the velocities in a control group and in normal subjects.

ACKNOWLEDGEMENTS

The authors thank Miss H. Fukunaga and Miss E. Matsuda for skillful technical assistance.

REFERENCES

1. Gilliatt, R. W. and Sears, T.: Sensory nerve action potentials in patients with peripheral nerve lesions. J. Neurol. Neurourg. Psychiat. 21: 109-118, 1958
2. Buchthal, F. and Rosenfalck, A.: Sensory potentials in polyneuropathy. Brain 94: 241-262, 1971
3. Buchthal, F. and Rosenfalck, A.: Evoked action potentils and conduction velocity in human sensory nerve. Brain Res. 3: 1-122, 1966
4. Preswick, G. and Jeremy, D.: Subclinical neuropathy in renal insufficiency. Lancet II: 731-732, 1964
5. Loong, S. G. and Seah, C. S.: Comparison of median and ulnar sensory nerve action potentials in the diagnosis of the carpal tunnel syndrome. J. Neurol. Neurosurg. Psychiat. 34: 750-754, 1971
6. Sato, K.: Sensory conduction in the nerves of the arm and leg in man. Jap. J. EEG EMG 1: 44-49, 1972 (in Japanese)
7. Yoshida, T., Takeuchi, H., Ishibashi, H., Muneyuki, T. and Shigiya, R.: Sensory nerve action potential and conduction velocity of the median nerve. Clin. Neurol. 13: 347-354, 1973 (in Japanese)
8. Shimozawa, R. and Mavour, H.: In vivo human sural nerve action potentials. J. Appl. Physiol. 26: 623-629, 1969
9. Murai, Y. and Kuroiwa, Y.: Sural nerve conduction velocity in peripheral neuropathies and myelo-optico-neuropathies (SMON). J. Neurol. Sci. 20: 339-344, 1973
10. Asai, T., Sugiyama, S. and Sato, K.: Sensory action potentials in upper extremity of aged people. Clin. EEG 16: 111-116, 1974 (in Japanese)
11. Nöel, P.: Sensory nerve conduction in the upper limbs at various stages of diabetic neuropathy. J. Neurol. Neurosurg. Psychiat. 36: 786-796, 1973
12. Dyck, P. J., Lambert, E. H. and Mulder, D. W.: Charcot-Marie-Tooth disease: nerve conduction and clinical studies of a large kinship. Neurology 13: 1-11, 1963
13. Dyck, P. J. and Lambert, E. H.: Compound nerve action potential and morphometry. Electroenceph. clin. Neurophysiol. 36: 573-574, 1974
14. Humberstone, P. M.: Nerve conduction studies in Charcot-Marie-Tooth disease. Acta Neurol. Scandinav. 48: 176-190, 1972
15. Mcleod, J. G., Prineas, J. M. and Walsh, J. C.: The relationship of conduction velocity to pathology in peripheral nerves, in New development in electromyography and clinical neurophysiology, edited by J. D. Desmedt, Vol. 2, pp. 248-258, Karger, Basel, 1973
16. Dyck, P. J., Gutrecht, J. A., Bastron, J. A., Karnes, W. E. and Dale, A. J. D.: Histologic and teased-fiber measurements of sural nerve in disorders of lower motor and primary sensory neurons. Mayo CIin. Proc. 43: 81-123, 1968
17. Eisen, A. A., Woods. J. F. and Sherwin, A. L.: Peripheral nerve function in long-term therapy with diphenylhydantoin. Neurology 24: 411-417, 1974