

# Sensory Action Potential Measurements From Sural Nerve Of Healthy Population

A M U D I N H Z B A Q A I M I R S H A D I K H A W A J A M T A R I Q M H I K R A M

Department of Physiology, King Edward Medical college, Lahore

Correspondence to : Arif Mohy Ud Din

The action potential studies of peripheral nerves are widely used as a tool for investigation and evaluation of neuropathies. These studies called as nerve conduction studies (NCS), include motor and sensory conduction velocities, duration, amplitude, latency and motor action potentials. The sensory action potential measurement is a fairly reproducible parameter. This study was designed to find out sural nerve sensory action potentials in normal individuals. We carried out this study at the Pakistan Institute of Medical Sciences, Islamabad from June 1993 to May 1995. Eighty one normal individuals were selected who fulfilled the inclusion and exclusion criteria. Age ranged from 17 to 75 years with a mean age of  $39.63 \pm 13.69$  years. Sex distribution was 62% males and 38% females. The Body Mass Index was  $23.21 \pm 4.52$  (KG/M<sup>2</sup>). Mean sural nerve sensory action potentials (SNSAP) in normal individuals in European population is  $13.6 \pm 7.5$   $\mu$ V (Aminoff 1987). Sural nerve sensory action potentials in our study were  $8.89 \pm 5.39$   $\mu$ V.

**Conclusion:** We conclude that our population has lower sural nerve sensory action potentials as compared to the Western population. The sex of the individual does not influence whereas the age does affect these potentials.

**Key words:** Sural Nerve Sensory Action Potentials, Conduction Velocity;

The action potential studies of peripheral nerves are widely used as a tool for investigation and evaluation of neuropathies. These studies called as nerve conduction studies (NCS), include motor and sensory conduction velocities, duration, amplitude, latency and motor action potentials. The sensory action potential (SAP) measurement is a fairly reproducible parameter. The SAPs have become the quantitative hallmark of the extent and progression of impairment in peripheral neuropathies. Such a study is usually done by the EMG machine using surface electrodes on any one side of the body. A supramaximal stimulus of 100 to 300 volts of 0.1 to 0.5 millisecond duration is applied at a rate of 1 per second. The action potentials from nerves like median, ulnar, tibial and peroneal are calculated by measuring the peak to peak response of the action potentials in micro volts ( $\mu$ V). Results of ten or more tracings are averaged. The sensory action potentials (SAP) of median and sural nerve are measured orthodromically or antidromically. Skin temperatures are maintained between 33° to 35° C<sup>1</sup>. The conduction velocity both motor and sensory which is dependent upon fiber diameter and integrity of myelin sheath is reduced in demyelinating diseases e.g. diphtheric or post infective polyneuritis of Guillain Barre type. It is also reduced in segmental demyelination e.g. diabetes mellitus, nerve injuries or compression leading to conduction block. In contrast axonal polyneuropathies result is decreased amplitude of response associated with alcoholism, uraemia, diabetes, malnutrition (thiamine), heavy metals e.g. lead, toxic chemical e.g. acrylamide and drugs e.g. isoniazid. Axonal neuropathy is similar to Wallerian degeneration, begins in the distal portions of axons and progresses slowly proximally i.e. distal axonopathy of dying back type. In diabetes mellitus a mixture of segmental demyelination,

axonal degeneration and regeneration is seen<sup>2</sup>.

These, however, assess only the largest, most rapidly conducting myelinated nerve component of the peripheral nerve which forms 25% of its fibers. Conduction velocity in diabetic patient is related to glycaemic control and improves with improved glycaemic control<sup>3</sup>. Conduction velocity is reduced both due to axonal degeneration as well as segmental demyelination<sup>4</sup>. There is a clear correlation in those having neuropathy with nerve conduction studies. Fluid and electrolyte imbalance is partially responsible for impaired conduction velocity since an improvement of 3 meters/second is observed in newly diagnosed NIDDM patients as well as in those with chronic established neuropathy once the glycaemic control is achieved<sup>3</sup>. The sensory action potentials and velocity are more consistently reduced in peripheral neuropathy. Subclinical neuropathy i.e. abnormal conduction studies are more common in sensory fibers. Diagnosis must be based on overall clinical and electrophysiological context<sup>4,5,6</sup>. Nerve conduction studies are most sensitive in detecting abnormalities of peripheral nerves<sup>7</sup>. Distribution of conduction velocities and compound action potential may be used to assess fiber diameters distribution in a nerve. There is 4% alteration in conduction velocity with each degree centigrade change in temperature of the limb. In normal or mild neuropathy, there is increased probability of getting slower conduction velocities<sup>8,9</sup>. Table I gives age adjusted values of sural nerve sensory action potentials (SNSAP)<sup>1</sup>. No studies have been carried out in Pakistan establishing the normal values in different ages and sexes.

## Materials And Methods

**Subjects:** Eighty one healthy individuals from both sexes were studied to determine the sural nerve sensory action

potentials. A strict exclusion criteria was maintained and any subject giving a positive history of any of the following condition was excluded from the study.

#### Exclusion Criteria:

Patients with diabetes mellitus, marked paralysis after stroke, having known neuropathy, signs and symptoms of cervical or lumbar spondylosis or myelopathy, thyroid disease, nerve compression or irritation, occupational, therapeutic or otherwise exposure to neurotoxins e.g. Lead, arsenic, alcohol, drugs, history of trauma significantly damaging sural nerve, patients with serum creatinine greater than 2.0 mg/dl, symptoms or signs of vitamin b1, b6, nicotinamide or folate deficiency, hypoxic conditions e.g. Chronic airway disease, cardiac decompensation and peripheral vascular diseases were excluded from the study.

#### Clinical Evaluation

All subjects were given a thorough physical examination including neurological assessment. Presence of symptoms of peripheral sensory neuropathy i.e. paraesthesias, numbness or unsteady gait were specifically asked and if present, the subject was excluded from the study. Questions pertaining to exclusion criteria were asked from all subjects. Presence of generalised or focal muscular weakness were inquired. Symptoms of autonomic neuropathy including postural dizziness, fainting, abdominal distention, flatulence nocturnal diarrhea, impotence (males only), urinary and sweat disturbances were asked about.

#### Sural Nerve Conduction Studies (Methodology)

The sural nerve was selected for conduction studies because this has been found to be the most useful method for screening patients suspected of having a sensory polyneuropathy. The earliest degenerative changes of polyneuropathy arise in the distal sensory fibers in the leg and sural nerve is the most accessible of peripheral sensory nerves<sup>4</sup>.

The sural nerve conduction studies were done with Racia EMG 21P electromyographic system of French design and manufacture (Users Manual Racia EMG ZIP). The sural nerve sensory action potentials (SNSAP) were recorded antidromically, being more convenient. Bipolar surface stimulating electrodes with cathode distal to the anode, were placed slightly lateral to the midline on the posterior aspect of the calf at the junction of the middle and lower thirds of the leg where the sural nerve runs a superficial course.

The response was recorded by surface electrodes distal to the cathode of stimulating electrode the active one was placed just posterior to the lateral malleolus and the reference one was positioned below and behind the lateral malleolus some 3 cm more distally. The ground lead was attached on the lower lateral aspect of the calf between the stimulating and recording electrode. A supramaximal stimulus of 100 to 300 volts of 0.1 millisecond duration was applied at a frequency of 1/second.

Measurements were made from the peak to peak amplitude of the action potential in  $\mu$ V. An average of ten recordings was taken. Table I shows sural nerve sensory action potentials (SNSAP) obtained in normal individuals varying with age<sup>4</sup>. The sural nerve conduction studies were carried out at the Neurophysiology laboratory (EMG room) of the department of neurology, Pakistan Institute of Medical Sciences (P.I.M.S), Islamabad. Fig II shows the placement of electrodes on the surface of the body for the action potential measurements.

Table-I Age Adjusted Values Of Normal Sural Nerve Sensory Action Potentials (Snsap) (Aminoff, 1987)

Age	Sural nerve sensory action potential
1-15	23.1.(SD 4.4)
0-15	43.7.(SD 3.8)
0-20	18.4.(SD 6.4)
21-20	16.4.(SD 5.5)
41-60	13.6.(SD 7.5)
61-80	9.8.(SD 3.6)

#### Results

Eighty one subjects fulfilling the criteria laid down in material and methods were examined in the present study. The summary of result is shown in Table 2.

Table-2 Summary of data of our study and standards

Parameter	Our Study		Standard (Aminoff, 1987)
	Mean	± SD	Mean ± SD
Age (Year)	39.63	13.69	-
BMI (Kg/M <sup>2</sup> )	23.21	4.52	-
SNSAP(m/s)	8.89	5.39	13.6,7.5
FBS (mg/dl)	99.00	8.68	-

Key: SD= Standard Deviation, BMI= Body Mass Index, SNSAP= Sural Nerve Sensory Action Potential, FBS= Mean Fasting Stood Sugar.

#### Gender:

There were 50 (62%) males and 31 (38%) females:

#### Age:

The mean age was  $39.63 \pm 13.69$ , range being 17 to 75 years.

#### Anthropometry:

The mean height of the controls was  $164.55 \pm 9.9$  cm, range 126 to 198 cm. The mean weight was  $62.43 \pm 12.77$ , range 38 to 100 Kg. The mean BMI (Body Mass Index) in the control group was  $23.21 \pm 4.52$  Kg/M<sup>2</sup>, range 13.77 to 35.71. The BMI was normal (< 25 Kg/M<sup>2</sup>) in 57 (70%) and greater than normal in 24 (30%) persons

#### Sural Nerve Conduction Studies:

The mean SNSAP (Sural Nerve Sensory Action Potential) among the controls was  $8.89 \pm 5.39$ , range 1.21 to 25.93  $\mu$ V as compared to the normal values quoted by Amiroff in 1987 which were 13.6 (SD 7.5)  $\mu$ V.

It was observed that the age of the subject seems to have a bearing on his SNSAP.

#### Discussion

No studies have been carried out to assess the normal range of Sural Nerve conduction studies in Pakistan. In

our study the mean SNSAP is 8.89 (SD 5.39)  $\mu$ V which is LOWER than the quoted values by <sup>1</sup>, i e; 13.6 (SD 7.5)  $\mu$ V. The sex of the individuals does not seem to influence sural nerve conduction velocities. The age of the individual has an inverse relationship with sural nerve Sensory Action Potentials.

#### Conclusion:

More studies must be carried out in different regions of Pakistan with uniform methodology. This will establish any factual difference between our and western population. Similarly sural nerve conduction velocities should be measured and standardised, with respect to our population.

#### References

1. Rendell M, Katims JJ, Richters R, Reowland F. A comparison of nerve conduction velocities and current perception thresholds as correlates of clinical severity of diabetic sensory neuropathy. *J Neurol Neurosurg Psychiatry* 1989; 52: 502-11.
2. Shahani BT, Sumner AJ. Electrophysiological studies in peripheral neuropathy; early detection and monitoring. In: *Neurology - Clinical Neurophysiology*. Stalbery E, Young RR, Eds. Butterworth 1981: 117-44.
3. Word JP. Clinical Aspects of Diabetic Somatic Neuropathy. In: *Text book of Diabetes*. Pickug J, William G, Eds. Blackwell Scientific Publications 1991: 623-34.
4. Ammoff MJ. *Electromyography in clinical practice*: Churchill Livingstone 2nd Edition 1987: 215-232.
5. Kayser-Gatahalian MC, Neundorfer B. Sural nerve conduction in Mild polyneuropathy. *Neurol*. 1984; 231(3): 122-5.
6. Dyck PJ, Zimmerman BR, Vilen TH, Minnenath SR, Karner JL, Yao KJ, et al. Nerve glucose, fructose, sorbitol, myoinositol and fiber degeneration and regeneration in diabetic neuropathy. *N Engl J Med*: 1988; 319(9): 542-8.
7. Ohnishi A, Yamamoto T, Murai Y, Ikeda M, Sugimoto H, Miyoshi T. Correlation between vibratory detection threshold and conduction study of sural nerve in diabetic patients. *Sangyo Ika Daigaku Zasshi* 1989; 11(4): 425-8.
8. Cummins KL, Dorfman JJ. Nerve fiber conduction velocity distributions: Study of normal and diabetic human nerves. *Am. J. Neurol*. 1981; 9(1): 67-74.
9. Lehtinea JM, Niskanen L, Hyvoanen K, Sitenen O, Uvstupa M. Nerve function and its determinants in patients with newly diagnosed type II (NIDDM) and in control subjects - a 5 years follow-up. *Diabetologia* 1993; 36(1): 68-72.