

## EVALUATION OF SENSORY NERVE CONDUCTION IN IRON DEFICIENCY ANAEMIA PATIENTS



### Physiology

**KEYWORDS:** : Sensory Nerve Conduction, Iron Deficiency, Action Potential.

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### ABSTRACT

**Abstract:** Background: Iron is very essential and has many important functions in energy metabolism, neurotransmission and myelin formation. Iron deficiency anemia is the most common type of anemia, and it occurs when your body doesn't have enough of the mineral iron. **Aims and Objectives:** The aim of the present study was to evaluate the effects of Iron deficiency on sensory nerve conduction. **Materials and Methods:** A total number of 60 individuals (40 females and 20 males) with iron deficiency in the age range of 30-60 years from the Department of Medicine, Mahavir Institute of Medical Sciences, as the study group and 40 participants (30 females and 10 males) without anemia as the control group were enrolled into this cross-sectional study. The sensory nerve conduction parameters, viz., distal latency, amplitude of compound muscle action potential (CMAP), and motor nerve conduction velocity (MNCV) were recorded bilaterally in median nerves using standard protocols and settings. **Results:** Values from patients were compared with those of controls by unpaired student's t-test and one-way ANOVA. **Conclusion:** The results indicated that the alteration in sensory conduction might be due to various functional and structural changes in peripheral nerves associated with iron deficiency.

### INTRODUCTION:

Iron deficiency is the most common nutritional deficiency in the world. Iron is present in all cells in the human body and has several vital functions, such as: carrying oxygen to the tissues from the lungs as a key component of the hemoglobin protein; acting as a transport medium for electrons within the cells in the form of cytochromes facilitating oxygen use and storage in the muscles as a component of myoglobin and as an integral part of enzyme reactions in various tissues. Too little iron can interfere with these vital functions and lead to morbidity and death. Anemia caused peripheral neuropathy (Nerve Damage) A diet that lacks iron, folic acid (folate), or vitamin B12 can prevent your body from making enough red blood cells. The first signs of developing anemia maybe loss of appetite, constipation, headaches, irritability, and or difficulty in concentrating. Established anemia can produce such symptoms as weakness, fatigue, coldness of extremities, depression, dizziness, pallor (most notable pale and brittle nails, lips, and eyelids), soreness in the mouth, and in women, cessation of menstruation.

Anemia can be a symptom of an underlying disease such as arthritis, infection and other major illness. It should therefore be investigated to see find what inflammation is lurking in the body. A deficiency of iron can effect nerve conduction. A lack of B12 damages the myelin sheath that surrounds and protect nerves. Without this protection, nerves cease to function properly and conditions such as peripheral neuropathy occur. Even B12 deficiency that is relatively mild may affect the nervous system and the proper functioning of the brain. The nerve damage caused by a lack of B12 may become permanently debilitating, if the underlying condition is not treated. A nerve conduction study (NCS), also called a nerve conduction velocity (NCV) test is a measurement of the speed of conduction of an electrical impulse through a nerve. NCS can determine nerve damage and destruction. Nerve conduction studies may also be performed to identify the cause of symptoms such as numbness, tingling, and continuous pain. While iron is an essential element that plays an important role in neurotransmitter metabolism, cerebral energy metabolism, and myelin formation, IDA can cause dysfunctions of the central nervous system such as decline and slowing of cognitive functions, delayed growth and motor development, especially in childhood, and learning and memory disorders. IDA can also cause dysfunctions in the peripheral nervous system, such as paresthetic complaints due to generalized neuropathic processes caused by the effect of iron on myelin formation. The main aim of this study was to investigate electrophysiologically the interaction of IDA with the peripheral nervous system, and to investigate whether or not the possible

electrophysiological abnormalities are reversible with the appropriate doses and duration of iron therapy.

### MATERIALS AND METHODS:

This cross-sectional study included A total number of 60 individuals (40 females and 20 males) with iron deficiency in the age range of 25-60 years from the Department of Medicine, Mahavir Institute of Medical Sciences, as the study group and 40 participants (30 females and 10 males) without anemia as the control group were enrolled into this cross-sectional study. The study was approval by the institutional ethical committee. The procedure was clearly explained to the patients recruited for the study, and written informed consent was obtained from the patients. Patients with history of cardiac, renal, hepatic, endocrine and mental illness, any acute illness, diabetes mellitus, alcohol addiction, leprosy, neuromuscular disorders, drug-induced neuropathy, family H/o neuropathy, malignancy, HIV, myopathy, those received blood transfusion, and those under treatment for anemia, etc., were excluded. A detailed history taking and clinical examination were performed (Fig.1). Nerve conduction study was performed using the Neurostimulation machine. Three disc surface electrodes were used with surface stimulators - Recording electrode, reference electrode, and ground electrode. The electrodes were placed after applying jelly to reduce resistance in air between electrode and skin surface. The nerve is stimulated, usually with surface electrode patches attached to the skin. Two electrodes are placed on the skin over the nerve. One electrode stimulates the nerve with a very mild electrical impulse and the other electrode records it. The resulting electrical activity is recorded by another electrode. This is repeated for each nerve being tested. The nerve conduction velocity (speed) is then calculated by measuring the distance between electrodes and the time it takes for electrical impulses to travel between electrodes. A related procedure that may be performed is electromyography (EMG). An EMG measures the electrical activity in muscles and is often performed at the same time as NCS.

Exclusion criteria were as follows: patient age <20; history of mental illness, systemic, cardiac problems, renal failure, hepatic syndromes, endocrine disorders, peripheral nervous system or infectious disease; nutritional deficiency; family history of peripheral nerve disease; drug and alcohol addiction (alcohol consumption more than 15 units/ week); uncooperative patients; patients having taken any drug for any reason in the previous four weeks; and IDA patients on iron treatment.

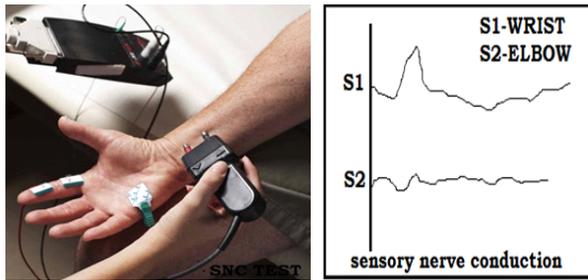


Figure.1.Sensory Nerve Conduction Test

**RESULTS AND DISCUSSION:** The procedures help to detect the presence, location, and extent of diseases that damage the nerves and muscles. For all the patients, we recorded parameters such as distal latency (DL), amplitude of compound muscle action potential (CMAP), and sensory nerve conduction velocity (SNCV) after stimulation of the median nerves on both sides. Table 1 compares the motor nerve conduction parameters between patients with mild, moderate, and severe anemia, which shows that in the sensory nerves. Nerve conduction was performed bilaterally on all limbs and as left side data were similar to that of the right side; only right sided values were depicted (Table.1). The sensory nerve action potential is normal on wrist stimulation but absent on elbow stimulation, indicating conduction block or severe temporal dispersion in sensory nerve fibers. Motor conduction and sensory conduction in the right median nerve were normal. Sensory nerve conduction studies included antidromic de-termination of conduction velocity, latencies and amplitude of the sensory nerve action potential of the median, ulnar, radial and sural nerves. Ke F responses of the median and peroneal nerves were also studied. Carpal tunnel syndrome (CTS) was diagnosed when the median nerve distal motor and/or sensory latencies exceeded 4.4 and 3.5 ms, respectively. Our results revealed a predominantly sensory neuropathic process particularly in lower extremities. In support to our results that IDA leads to neuropathic processes, several previous studies have re-ported that iron has an important role in myelino-genesis via two possible mechanisms as a direct and an indirect pathway All the data were entered in MS Excel spreadsheet, and the statistical analysis was done using SPSS. The data were expressed as mean  $\pm$  standard deviation. Descriptive tables were generated, and Student t-test and one-way ANOVA were used to demonstrate the findings.  $P < 0.05$  was considered to be statistically significant.

Table 1: Comparison of sensory nerve conduction

Nerve	Parameters	Mean SD			F value	P value
		Mild Anemia	Moderate Anemia	Severe Anemia		
Sensory Nerve	DL (msec)	2.650.34	3.46	6.230.68	16.08	0.05
	CMAP amplitude	14.060.65	11.850.23	07.451.29	13.67	0.05
	MNCV (m/sec)	57.141.34	59.080.86	62.541.20	04.98	0.05

#### CONCLUSION:

This study reveals the involvement of sensory nerves in iron deficiency. In adults, as the iron deficiency has insidious onset, diagnosis can be delayed by months or years and the neuropathic manifestations can go unrecognized for a longer period. Therefore, performing electrophysiological studies in anemic patients are recommended, early in the course of the disease to detect nervous system involvement. . Detection of iron responsive neuropathic processes (PNP and CTS) in IDA patients suggested that IDA may cause peripheral nervous system involvement. It is important to emphasize the examinations for IDA as an etiologic factor on planning treatment for neuropathy patients Further research is needed to see whether the neurological dysfunction in iron

deficiency is reversible with the appropriate iron replacement therapy.

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