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ASSESSMENT OF NERVE CONDUCTION IN EVALUATION OF RADICULOPATHY AMONG CHRONIC LOW BACK PAIN PATIENTS WITHOUT CLINICAL NEURODEFICIT

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Abstract : The diagnostic evaluation of chronic low back pain (CLBP) is difficult, as its primary causes are multiple. Clinical, radiological and electrophysiological findings are of limited value in diagnosing radiculopathy as the cause of CLBP in early cases. Current study was undertaken on 50 controls and 50 CLBP patients without clinical neurological deficit to evaluate the potential of nerve conduction studies, particularly H-reflex study for diagnosis of radiculopathy in these cases. We observed that routine nerve conductions in CLBP without clinical neurodeficit showed no significant differences; whereas all the H-reflex parameters, H-threshold, H latency, H amplitude and H/M ratio were significantly different when compared with that of control (P value <0.0001 in each case). We concluded that subclinical cases might not have only partial conduction block but also secondary axonal loss due to compression of nerve roots. We further suggest inclusion of Soleus H-reflex study in evaluation of radiculopathy among early CLBP cases without clinical neurodeficit.

Key words : soleus H-reflex study radiculopathy chronic low back pain

INTRODUCTION

Low back pain is a disabling musculoskeletal disorder with an overall lifetime prevalence of 60% to 90%. Almost 95% of low back pain complaints resolve after 3 months, leaving 5% with persistence of symptoms developing into chronic low back pain (CLBP) (1). The diagnostic evaluation of CLBP is difficult as its primary causes are different affecting intervertebral discs, ligaments, facet joints, muscles etc. Also clinical signs and symptoms, radiological and electrophysiological evaluation can't exactly provide the source of pain responsible for patient's symptoms in most cases (2). Needle EMG (Electromyography) is of limited value as there is no motor root involvement in CLBP (3).

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Soleus H-reflex study though is considered as sensitive marker of sensory root dysfunction, information about its role in evaluation of chronic low back pain is quite insufficient. In this study attempt was made to evaluate the potential of nerve conduction studies, particularly H-reflex study for diagnosis of radiculopathy in early cases without clinical neurodeficit.

MATERIALS AND METHODS

The study was conducted during January 2007 to March 2008 in Clinical Neurophysiology Laboratory, Department of Physiology, MGIMS Sevagram, Wardha. An approval from Institutional Ethics Committee was taken for conducting the research before start of this study. Fifty cases were selected from patients attending orthopedics OPD with supportive inclusion and exclusion criteria under supervision of orthopedic surgeon. Inclusion criteria consisted of patients with age ranging from 25 to 60 years with history of CLBP more than three months duration not radiating below buttocks and clinical examination revealed no neurodeficit. Patients with local injuries/lesions that may interfere with the electrophysiological study, known cause of neuropathy both vascular and systemic like diabetes mellitus and clinical signs suggesting myelopathy, myopathy, polyneuropathy and neuromuscular transmission disorders like myasthenia gravis were excluded from the study.

The control group included fifty healthy subjects, mostly recruited from hospital staff and their relatives with no previous or current history of significant disease, in particular back pain and radiculopathy. They were selected on age and height basis so as to be matched with patient population after thorough physical and neurogical examination by consultant physician. Electrophysiological studies similar to cases were performed and data was obtained for comparison. EMG was not done in control group and study group.

Electrophysiological study

A written informed consent was taken from all the subjects screened under above inclusion and exclusion criteria who underwent the study. A detailed bilateral lower limb nerve conduction studies comprising of tibial and peroneal motor nerve conductions, sural sensory nerve conductions were done. F waves were recorded from extensor digitorum brevis muscle and flexor hallusis brevis muscle ("marker" muscles for L_5 and S_1 nerve roots respectively) after supramaximal stimulation of peroneal and tibial nerve at the ankle. H-reflex study was recorded from soleus muscle by subminimally stimulating tibial nerve in popleteal fossa. Settings were kept at sweep speed 10 ms/D, intensity 2 mV, frequency 2 Hz and stimulus strength duration was 1ms. By adjusting the position of stimulating electrode (cathode being proximal), the site with the lowest threshold for H-reflex was identified. Stimulus intensities were increased in steps of 1-2 mA until the maximum H wave amplitude was obtained and further by 2-5mA until maximum M wave amplitude was obtained. Three stimuli were live averaged for single response. Downward deflections were marked as latencies of waveforms. Minimum stimulus strength required for obtaining an H wave and M wave of 0.4 mV amplitude was considered H and M threshold respectively (4, 5).

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The data of motor, sensory conduction study, F wave minimum latency and H-reflex study parameters (H-threshold, H latency, H amplitude and H/M ratio) was collected. All tests were performed by same investigator and under constant room temperature on RMS EMG EP Mark-II machine manufactured by Recorders and Medicare systems Chandigarh, India. The age and height of each patient were recorded.

Statistical analysis

Analysis was done using STATA-10 and Graph pad Prism 4.1 software. Student's t test (Unpaired) was applied to obtain difference of statistical significance between control and study group. P value < 0.05 was considered to be significant.

RESULTS

Control group consisted of fifty volunteers with mean age 44.14 ± 7.783 , age range 32 to

60 years, mean height 166.04 ± 4.8 , height range 158 to 178 cms, three females and 47 males and study group consisted of fifty patients with mean age 41.52 ± 10.2 , age range 25 to 60 years, mean height 161.5 ± 8.214 , height range 148 to 180 cms, 16 females and 34 males (Table I).

Distal motor latencies (DML), Compound Muscle Action Potential (CMAP) amplitude and conduction velocity (CV), F minimum latencies of tibial, peroneal motor nerves and onset latency, Sensory Nerve Action Potential (SNAP) amplitude and conduction velocity of sural nerve showed no significant

TABLE I: Comparison of physiological parameters of subjects of control and study groups.

Parameters	Control Group	Study Group	P-
	(n=50)	(n=50)	value
Age (years)	44.14 ± 7.783	41.52±10.2	0.1637
Height (cm)	166.0 ± 4.886	161.5±8.214	0.0010*
Weight (kg)	58.04 ± 8.741	56.92+10.05	0.5534

*Statistically significant.

TABLE II:	Comparison	of	Motor	and	sensory	ner	ve co	nduction
	parameters of	of	subjects	of	control	and	study	groups.

Parameters	Side	Control group $(n=50)$	Study group $(n=50)$	P-value
Tibial Distal Motor Latency (ms)	Right Left	3.678 ± 0.5772 3.860 ± 0.7389	3.694 ± 0.7608 3.875 ± 0.8753	$0.9048 \\ 0.9254$
Tibial CMAP amplitude (mV)	Right Left	$\frac{18.02 \pm 6.557}{17.98 \pm 7.120}$	16.99 ± 6.349 16.69 ± 5.652	$0.4277 \\ 0.3204$
Tibial conduction velocity (m/s)	Right Left	$\begin{array}{c} 48.88 {\pm} 4.269 \\ 49.06 {\pm} 5.601 \end{array}$	48.75 ± 4.342 47.91 ± 3.866	$0.8794 \\ 0.2600$
Tibial F minimum Latency (ms)	Right Left	46.63 ± 3.446 46.59 ± 3.380	46.80 ± 3.936 47.04 ± 3.977	$0.8129 \\ 0.5431$
Peroneal Distal Motor Latency (ms)	Right Left	$\begin{array}{c} 4.122 {\pm} 0.5857 \\ 3.995 {\pm} 0.5721 \end{array}$	4.037 ± 0.7058 3.932 ± 0.6504	$0.5128 \\ 0.6059$
Peroneal CMAP amplitude (mV)	Right Left	$\begin{array}{c} 8.492 {\pm} 2.711 \\ 8.703 {\pm} 2.733 \end{array}$	8.574 ± 3.133 7.650 ± 2.395	0.8900 0.0433*
Peroneal conduction velocity (m/s)	Right Left	$52.86{\pm}4.860\\50.67{\pm}4.101$	50.25 ± 4.406 50.36 ± 3.982	0.005* 0.7083
Peroneal F minimum Latency (ms)	Right Left	$\begin{array}{c} 44.46{\pm}3.321\\ 45.32{\pm}3.355\end{array}$	44.95 ± 3.904 45.39 ± 4.285	$0.4978 \\ 0.9731$
Sural SNAP amplitude (μV)	Right Left	$\begin{array}{c} 15.29 {\pm} 8.909 \\ 16.32 {\pm} 7.406 \end{array}$	$\begin{array}{c} 15.44{\pm}8.034 \\ 15.19{\pm}8.169 \end{array}$	$0.9307 \\ 0.4704$
Sural conduction velocity (m/s)	Right Left	$\begin{array}{c} 50.86 {\pm} 5.130 \\ 51.47 {\pm} 5.373 \end{array}$	50.42 ± 5.393 50.31 ± 5.190	$0.6799 \\ 0.2766$

*Statistically significant; CMAP: Compound muscle action potential; SNAP: Sensory nerve action potential.

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TABLE III:	Comparison	of H	reflex	parameters	of	subjects	of	control	and	study	groups.

Parameters	Side	Control group $(n=50)$	Study group $(n=50)$	P-value
H-Threshold (mA)	Right Left	3.98 ± 1.42 3.74 ± 1.671	7.1 ± 3.1 7.76 ± 3.28	<0.0001* <0.0001*
H latency (ms)	Right Left	$\begin{array}{c} 28.15{\pm}2.008\\ 28.15{\pm}1.991 \end{array}$	$\begin{array}{c} 29.3 {\pm} 1.95 \\ 29.2 {\pm} 2.06 \end{array}$	<0.0001* <0.0001*
H amplitude (mV)	Right Left	5.49 ± 3.25 5.354 ± 3.131	2.9 ± 2.3 3.2 ± 2.6	<0.0001* <0.0001*
H/M ratio (%)	Right Left	33.97 ± 19.36 31.21 ± 15.701	$19.8 {\pm} 12.8 \\ 22 {\pm} 15$	<0.0001* <0.0001*

*Statistically significant.

difference except in left Peroneal CMAP amplitude and right peroneal CV (P-value <0.05) (Table II).

Soleus H-reflex study showed bilateral significant differences as compared to the control values (P-value <0.0001) as, increased H-threshold, prolonged H latency, reduced H amplitude and reduced H/M ratio (Table III).

DISCUSSION

Presently, imaging studies are standard diagnostic aids in the common radiculopathies caused by structural lesions; they often are unrevealing in radiculopathy caused by infection, infiltration, demyelination, or infarction. Imaging studies do well in visualizing the spinal cord, nerve roots and their relationship to the vertebra and intervertebral discs; however they yield no information about nerve functioning. In this regard, EMG and MRI are complementary to each other as former asses the nerve functionally and later structurally (4).

In our study, we observed that routine nerve conductions in CLBP without clinical neurodeficit showed no significant difference except in left Peroneal CMAP amplitude and right peroneal CV (P-value <0.05). These findings are nonspecific and could not be explained as involvement is neither side specific nor variable specific. It will be too preliminary to conclude on above findings.

Whereas all the H- reflex parameters were found to be affected when compared with the normative data. H- threshold reflects activity in the most excitable Ia afferent fibers, which in turn, will influence the recruitment of the alpha-motoneurones and inducing excitatory post-synaptic potentials (EPSPs) in other ones, the so called subliminal fringe(6). The H wave amplitude and H/M ratio i.e. the ratio of peak to peak maximum H wave to that of maximum M wave amplitude provides an easy estimate of motoneurones pool activation, and therefore excitability (5).

In previous work, where 26 patients with CLBP of mechanical origin were examined (7), the only abnormality detected was significant increase in H-reflex threshold with normal routine nerve conductions and other parameters of H-reflex study. In present work, not only H-reflex threshold but also H wave latency, H wave amplitude and H/M ratio of study group were found to

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be significantly different from the control group. It was proposed from previous work that the increase in H-reflex threshold might be due to changes in the peripheral sensory input. Specifically, repeated mechanical or tensile stresses contributing to the type of pain associated with spondylolisthesis may lead to partial conduction block or temporal dispersion of the sensory volley, through compressive or ischemic mechanisms within the lumbar canal, resulting in subclinical sensory root dysfunction.

In present study all parameters involved in CLBP without clinical neurodeficit suggest that these subclinical cases might not have only partial conduction block but also secondary axonal loss due to compression and or change in the gain of the reflex due to modulation of central mechanisms (8). Chronic pain in any lumbar structure i.e. ligaments, joints or muscles may influence the pathways subserving soleus muscle either through a the common element in the central neuronal circuitry (9) or following a motor pattern of trunk musculature (10). Changes of Soleus H-reflex recruitment curve may thus reflect changes at pre- and/or post-synaptic level (6).

Although many causes that would otherwise affect the excitability of alpha motor neurons as a result of sensory-motor interaction at all levels of the central nervous system (CNS) following CLBP could not be excluded, a possibility that can be favored is, decrease in peripheral afferent feedback may explain not only the increased H-threshold but also other parameters of H-reflex recruitment curve pattern. Alteration in the recruitment property of the Ia afferent fibers or motor axons may derive from morphological and functional factors. Morphological factors include focal disturbance to myelin secondary to insufficient microcirculation and ischemia due to nerve root compression. Functional factors include membrane potential and ion channel changes. Both together in turn may affect the temporal dispersion of the compound sensory afferent volley and/or axonal conduction block.

Mechanical deformation of spinal nerve roots following vertebral instability due to CLBP is likely to affect both microvessels of nerve roots and nerve fibers (11) may explain rise in H-threshold whereas more severe compression of nerve roots along with central neuronal circuitry modifications may explain reduction in H amplitude, H/M ratio and prolonged H latency of Soleus H-reflex study.

Although physiological parameters like age, sex, height or limb length, body mass index (BMI) are known to affect H reflex study, only age and height or limb length are most commonly affecting the H latency (4, 5). It has been established that maximal H amplitude may essentially be related to activation of motor neuron pool by sensory Ia afferent volley and presynaptic inhibition as well (12). Race, gender and BMI differences are not associated with changes in H reflex (13, 14) therefore they are less likely to affect our findings. Age and height shows direct correlation with H latency (4, 5). In our study, significant difference (P-value <0.05) was noted in heights among control and study group but mean H latency is more in study group in spite of less mean height as compared to control group.

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We concluded that subclinical cases might not have only partial conduction block but also secondary axonal loss due to compression of nerve roots. We further suggest inclusion of Soleus H-reflex study in evaluation of radiculopathy in early CLBP cases. Indian J Physiol Pharmacol 2010; 54(1)

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