

Nerve Conduction Studies of Upper Limbs in Rheumatoid Arthritis Patients

Rangrao M. Bhise¹, Kiran H. Buge², Siva Kumar U.³, Sunita G. Nighute⁴

Assistant Professor, Dept. of Physiology, DVVPPF'S Medical College, Ahmednagar, Maharashtra, India¹

Associate Professor, Dept. of Physiology, DVVPPF'S Medical College, Ahmednagar, Maharashtra, India²

Assistant Professor, Dept. of Physiology, DVVPPF'S Medical College, Ahmednagar, Maharashtra, India³

Professor, Dept. of Physiology, DVVPPF'S Medical College, Ahmednagar, Maharashtra, India⁴

ABSTRACT: Rheumatoid arthritis (RA) is a chronic inflammatory disorder which affects the joints and is associated with swelling, stiffness and pain. Advanced disease stages can lead to substantial loss of functioning and mobility. As RA causes functional limitations in the joints, this might affect the movements or the movement patterns of the damaged and inflamed joints. The prevalence of rheumatoid arthritis in India is about 1 to 1.5 %. The primary targets of inflammation are synovial membranes and articular structures but other organs also affected. Nerve conduction studies is most commonly used test, it is the main laboratory technique for the study of peripheral nerve function and it involve the surface stimulation of motor & sensory nerves recording of the elicited action potential in muscle(CMAP) and the sensory nerve action potential (SNAP). The result of these motor & sensory nerve conduction studies is expressed as amplitudes, latencies & conduction velocities. NCS was done by using a NEURO – MEP – NET (NEUROSOFT) Machine according to standard protocol and settings. Response for motor nerve conduction studies were recorded from distal muscles by using the surface electrodes.

Result shows that both Motor and sensory nerve conduction studies in Median and ulnar nerve was prolonged distal latency, low amplitude and decreased conduction velocities in the RA patients than the controls subjects. This could be due to peripheral neuropathy in RA patients.

KEYWORDS: Rheumatoid arthritis (RA), Nerve Conduction Velocity (NCV), Nerve Conduction Studies (NCS), Conduction Velocity (CV), Latency (L), Amplitude (A), Action Potential (AP) RA (Rheumatoid Arthritis).

I. INTRODUCTION

Rheumatoid Arthritis is a chronic systemic disease of unknown etiology. It is characterized by peripheral symmetrical polyarthritis. It has a progressive course with exacerbation and remissions being part of its natural history. Its onset could be at any age, although it usually starts in the fourth decade of life. Overall, there is a 3:1 female preponderance, but this excess is greater in young people and the age related incidence is approximately equal in elderly people. The prevalence of rheumatoid arthritis in India is about 1 to 1.5 % [1,2]

It begins with pain, stiffness and swelling of specific joints such as proximal interphalangeal, metacarpophalangeal, wrist and knee joints. The diagnosis is based routinely on the persistence of arthritic symptoms over a time. Important factors associated with RA are the possibility of infectious triggers, genetic predisposition and autoimmune responses. The primary targets of inflammation are synovial membranes and articular structures but other organs are affected as well [3]. Rheumatoid arthritis (RA) is a chronic inflammatory disorder which affects the joints and is associated with swelling, stiffness and pain. Advanced disease stages can lead to substantial loss of functioning and mobility.

RA is diagnosed on clinical, serological and radiological grounds. The American Rheumatism Association (ARA) first proposed classification criteria for RA in 1956 and then revised them in 1958 [4,5]. Although, these criteria were widely used to diagnose RA for many years, they were heavily criticized for their lack of sensitivity and specificity. The ARA published revised classification criteria for RA in 1988, based on cross-sectional data from a large group of patients with rheumatoid and other types of inflammatory arthritis [6]

As RA causes functional limitations in the joints, this might affect the movements or the movement patterns of the damaged and inflamed joints [7,8,9,10,11,12,13].

Nerve Conduction Studies: Nerve conduction studies is most commonly used test, it is the main laboratory technique for the study of peripheral nerve function and it involve the surface stimulation of motor & sensory nerves recording of

the elicited action potential in muscle (CMAP) and the sensory nerve action potential (SNAP). The result of these motor & sensory nerve conduction studies is expressed as amplitudes, latencies & conduction velocities [14,15,16,17,18,19,20,21,]

Standardization and Quantification: Reliable electro-diagnostic evaluation requires standardization, with meticulous attention to details. Filter setting, type and size of electrode, location & recording of stimulating electrode, and measurement method should be defined. It is recommended generally that surface temperature should be maintained at >31 degrees for leg and foot and >32 degree Celsius for hand and forearm. The general agreement for optimal range between 32 to 36 degree Celsius. The recommended filter settings are 20 – 2000 Hz pass bands for sensory nerve conduction studies and 20 – 10000 Hz for motor conduction studies. Other important factors that can be standardized include qualification of the electro-myographer, method of data analysis and description and population based reference values.

II. AIM

Nerve Conduction Studies of Upper Limbs in Rheumatoid Arthritis Patients.

III. MATERIAL AND METHODS

After clinical evaluation and laboratory investigation, those patients satisfying the Modified American Rheumatology Classification Criteria (1987) were included in the study. 50 Rheumatoid Arthritis patients and 50 controlled subjects were randomly selected from the Rheumatology OPD. Age group of the subject from 25 to 65 years and it includes both male & female.

Inclusion criteria: Diagnosed Rheumatoid Arthritis Patients

Exclusion Criteria:

- Hemoglobin <10 gm/dl
- Pregnant Women
- Suffering from Diseases like diabetes mellitus, Parkinson's disease, cardiovascular diseases like hypertension, ischaemic heart disease, congestive heart failure, valvular heart disease, cardio-myopathy and cardiac arrhythmia. Neurological diseases like multiple sclerosis, polyneuropathy or Guillain-Barre Syndrome.
- Drugs that interfered with the ANS including antihypertensive, diuretic, adrenergic inhibitor, anti-arrhythmic, sedative, hypnotic, epileptic drugs were also excluded from the study.

Detailed History: All patients were evaluated with detailed history including Age, sex, duration of Rheumatoid Arthritis, duration of morning stiffness, list of painful joints, other systemic disease, history of extra-articular manifestations and treatment history of RA were documented.

Examinations: Blood pressure, pulse, heart rate and respiratory rate were recorded. Systemic examination of all joints was done for feature of activity, Tender and swollen joints count estimation was done. Cardiovascular, respiratory, abdominal and neurological examination was done, extra-articular manifestations were documented.

Investigations: Hemoglobin, erythrocyte sedimentation rate, C-reactive protein, RH factor and X-ray of the joints were done on OPD basis.

Procedure:

An accessible nerve is stimulated through the skin by surface electrode, using a stimulus that is large enough to recruit all the nerve fiber. The resulting action potential is recorded by electrode on skin:

1. Over the muscle (belly-tendon electrode) in case of motor fibers stimulated in a mixed or motor nerve (CMAP).
2. In orthodromic conduction: a distal portion of nerve e.g. digital nerve is stimulated and sensory nerve action potential (SNAP) is recorded at a proximal point along the nerve.
3. In antidromic conduction: the nerve is stimulated at proximal point, and action potential is recorded distally.

Instruments Used: NEURO – MEP – NET (NEUROSOFT) (8 channel)

IV. RESULT

Figure 1: Recording of NCV by Neuro-Mep- Net Machine (8 channel)



A complete NCS was done by using a NEURO – MEP – NET (NEUROSOFT) Machine according to standard protocol and settings. Response for motor nerve conduction studies were recorded from distal muscles by using the surface electrodes. Sensory nerve conduction studies were performed antidromically with the using of surface electrode.

Table 1: RA Patient Details

	N	Mean ± SD
Age (yr)	50	47.12 ± 8.62
Height (cm)	50	155.12 ± 7.39
Weight (kg)	50	59.66 ± 8.77
Duration of Disease(yrs)	50	3.09 ± 1.70
Hb gm/dl	50	12.44 ± 1.52
ESR	50	15.00 ± 11.95

The present study had patient with an age range of 25 – 75 years. The mean age at presentation was 47.12 ± 8.62 years, mean height was 155.12 ± 7.39 cm, mean weight was 59.66 kg ± 8.77, mean duration of disease was 3.09 ± 1.70 years, mean hemoglobin 12.44 ± 1.523 gm/dl and mean ESR was 15 ± 11.959 mm at the end of one hr.

Table 2: Gender distribution of RA patients

Gender	No	%
Male	16	32
Female	34	68
Total	50	100

The present study had 16 males accounting for 32 % and 34 females accounting for 68 %.



||Volume 9, Issue 4, April 2020||

Table 3: Control Details

	N	Mean ± SD
Age (yr)	50	35.44 ± 7.62
Height (cm)	50	154.62 ± 6.09
Weight (kg)	50	62 ± 10.70

The present study had controls with an age range of 25 – 75 years. The mean age at presentation was 35.34 ± 7.789 years, mean height was 154.62 ± 6.090 cm and mean weight was 62 ± 10.701 kg.

Table 4: Gender distribution of Controls

Gender	No	%
Male	33	66
Female	17	34
Total	50	100

The present study had 33 males accounting for 66 % and 17 females accounting for 34 %.

Table 5: Distribution of age: RA patient

Age (yr.)	No.	Percentage	No. of NCS Abnormality	Percentage
25 - 35	2	4 %	2	4 %
35 - 45	19	38 %	9	18 %
45 - 55	17	34 %	6	12 %
55 - 65	10	20%	8	16 %
>65	2	4%	1	2 %
Total	50	100 %	26	52 %

The maximum number of RA patients 19 (38%) was present in the age group of 35 – 45 years. The same age group had maximally affected patients accounting for 9 (18%).

Table 6: Distribution of age: Control subjects

Age (yr.)	No.	Percentage	No. of NCS Abnormality	Percentage
25 - 35	23	46 %	8	16 %
35 - 45	21	42 %	7	14 %
45 - 55	5	10%	1	2 %
55 - 65	1	2%	1	2 %
65 - 75	00	00%	0	0 %
Total	50	100 %	17	34 %

The maximum number of controls 23 (46%) was present in the age group of 25 – 35 years. The same age group had maximally affected patients accounting for 8 (16%).



Table 7: Finding of motor nerves:

		Group	N	Mean ± SD	Unpaired t test		
		Median nerve			T	DF	Sign. (2-tailed)
L	RA	50	7.42 ± 1.345	2.693	98	0.008	
	Control	50	6.79 ± 0.961				
A	RA	50	6.01 ± 2.520	-2.723	98	0.008	
	Control	50	7.17 ± 1.642				
CV	RA	50	56.76 ± 8.625	-2.524	98	0.013	
	Control	50	60.86 ± 7.600				
		Ulnar nerve					
L	RA	50	6.30 ± 1.218	2.179	98	0.032	
	Control	50	5.75 ± 1.272				
A	RA	50	5.63 ± 1.774	-1.206	98	0.231	
	Control	50	6.07 ± 1.873				
CV	RA	50	59.06 ± 9.236	-1.971	98	0.052	
	Control	50	62.26 ± 6.816				

P < 0.001 = highly significant, P > 0.001 to <0.05 = significant, P > 0.05 = not significant.

Mean latency of motor median nerve in RA patients was 7.42 ± 1.345 ms and 6.79 ± 0.961 ms for controls. There was statistically significant difference between the latency of RA patients and controls (p=0.008).

Mean amplitude of motor median nerve in RA patients was 6.01 ± 2.52 mV and 7.17 ± 1.642 mV in controls. There was statistically significant difference between the amplitude of RA patients and controls (p=0.008).

Mean conduction velocity of motor median nerve in RA patients was 56.76 ± 8.62 m/s and 60.86 ± 7.60 m/s in controls. There was statistically significant difference between the conduction velocity of RA patients and controls (p=0.013).

Mean latency of motor ulnar nerve in RA patients was 6.30 ± 1.21 ms and 5.75 ± 1.27 ms for controls. There was statistically significant difference between the latency of RA patients and controls (p=0.032).

Mean amplitude of motor ulnar nerve in RA patients was 5.63 ± 1.77 mV and 6.07 ± 1.87 mV for controls. There was not statistically significant difference between the amplitude of RA patients and controls (p=0.231).

Mean Conduction velocity of motor ulnar nerve in RA patients was 59.06 ± 9.23 m/s and 62.26 ± 6.81 m/s for controls. There was not statistically significant difference between the Conduction velocity of RA patients and controls (p=0.052).

Table 8: Finding of sensory nerves:

		Group	N	Mean ± SD	Unpaired t test		
		Median nerve			T	DF	Sign. (2-tailed)
L	RA	50	2.19 ± 0.827	-0.759	98	0.450	
	Control	50	2.29 ± 0.414				
A	RA	50	31.13 ± 11.44	0.388	98	0.699	
	Control	50	31.59 ± 9.914				
CV	RA	50	63.03 ± 20.13	1.211	98	0.229	
	Control	50	59.06 ± 11.56				
		Ulnar nerve					



L	RA	49	1.91 ± 0.822	-0.470	97	0.640
	Control	50	1.99 ± 0.838			
A	RA	50	30.45 ± 8.286	1.204	98	0.231
	Control	50	28.89 ± 9.976			
V	RA	50	61.64 ± 14.01	-0.375	98	0.709
	Control	50	62.57 ± 10.62			

P < 0.001 = highly significant, P > 0.001 to <0.05 = significant. P > 0.05 = not significant.

Mean latency of sensory Median Nerve in RA patients was 2.19 ± 0.82 ms and 2.29 ± 0.41 ms for controls. There was not statistically significant difference between the latency of RA patients and controls (p=0.450).

Mean Amplitude of sensory Median Nerve in RA patients was 31.13 ± 11.44 µV and 31.59 ± 9.914 µV for controls. There was not statistically significant difference between the Amplitude of RA patients and controls (p=0.699).

Mean Conduction velocity of sensory Median Nerve in RA patients was 63.03 ± 20.13 m/s and 59.06 ± 11.56 m/s for controls. There was not statistically significant difference between the Conduction velocity of RA patients and controls (p=0.229).

Mean latency of sensory ulnar Nerve in RA patients was 1.91 ± 0.82 ms and 1.99 ± 0.83 ms for controls. There was not statistically significant difference between the Latency of RA patients and controls (p=0.640).

Mean Amplitude of sensory ulnar Nerve in RA patients was 30.45 ± 8.28 µV and 28.898 ± 9.976 µV for controls. There was not statistically significant difference between the Amplitude of RA patients and controls (p=0.231).

Mean Conduction velocity of sensory ulnar Nerve in RA patients was 61.64 ± 14.01 µV and 62.57 ± 10.62 µV for controls. There was not statistically significant difference between the Conduction velocity of RA patients and controls (p=0.709).

Table 9: Median Nerve (Motor) – Latency:

			Group		Total
			RA	Control	
L	N	Count	39	48	87
		% within Group	78.0%	96.0%	87.0%
	AN	Count	11	2	13
		% within Group	22.0%	4.0%	13.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig (2- sided)	Exact sig. (2 – sided)
Pearson chi-square	7.162 ^a	1	0.007	
Fishers exact test				0.015

Latency of the motor median nerve was abnormal in 11 (22%) of RA patients and 2 (4%) in controls and it was normal in 39 (78%) of RA patients and 48 (96%) of controls. There was statistically significant difference between the abnormal latency of RA patients and controls (p=0.007).



Table 10: Median Nerve (Motor) – Amplitude:

			Group		Total
			RA	Control	
A	AN	Count	9	1	10
		% within Group	18.0%	2.0%	10.0%
	N	Count	41	49	90
		% within Group	82.0%	98.0%	90.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2- sided)	Exact sig. (2 – sided)
Pearson chi-square	7.111 ^a	1	0.008	
Fishers exact test				0.016

Amplitude of the motor median nerve was abnormal in 9 (18%) of RA patients and 1 (2%) in controls and it was normal in 41 (81%) of RA patients and 49 (98%) of controls. There was statistically significant difference between the abnormal amplitude of RA patients and controls (p=0.008).

Table 11: Median Nerve (Motor) – Conduction velocity:

			Group		Total
			RA	Control	
CV	AN	Count	13	5	18
		% within Group	26.0%	10.0%	18.0%
	N	Count	37	45	82
		% within Group	74.0%	90.0%	82.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2- sided)	Exact Sig. (2- sided)
Pearson Chi-Square	4.336 ^a	1	0.037	
Fisher's Exact Test				0.066

Conduction velocity of the motor median nerve was abnormal in 13 (26%) of RA patients and 5 (10%) in controls and it was normal in 37 (74%) of RA patients and 45 (90%) of controls. There was a statistically significant difference between the abnormal conduction velocity of RA patients and controls (p=0.037).



||Volume 9, Issue 4, April 2020||

Table 12: Ulnar Nerve (Motor) - Latency:

			Group		Total
			RA	Control	
L	N	Count	40	44	84
		% within Group	80.0%	88.0%	84.0%
	AN	Count	10	6	16
		% within Group	20.0%	12.0%	16.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	1.190 ^a	1	0.275	
Fisher's Exact Test				0.414

Latency of the motor Ulnar nerve was abnormal in 10 (20%) of RA patients and 6 (12%) in controls and it was normal in 40 (80%) of RA patients and 44 (88%) of controls. There was not statistically significant difference between the abnormal latency of RA patients and controls (p=0.275).

Table 13: Ulnar Nerve (Motor) – Amplitude

			Group		Total
			RA	Control	
A	AN	Count	4	2	6
		% within Group	8.0%	4.0%	6.0%
	N	Count	46	48	94
		% within Group	92.0%	96.0%	94.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	0.709 ^a	1	0.400	
Fisher's Exact Test				0.678

Amplitude of the motor Ulnar nerve was abnormal in 4 (8%) of RA patients and 2 (4%) in controls and it was normal in 46 (92%) of RA patients and 48 (96%) of controls. There was not statistically significant difference between the abnormal amplitude of RA patients and controls (p=0.400).

Table 14: Ulnar Nerve (Motor) - Conduction velocity

			Group		Total
			RA	Control	
CV	AN	Count	11	3	14
		% within Group	22.0%	6.0%	14.0%
	N	Count	39	47	86
		% within Group	78.0%	94.0%	86.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

AN: Abnormal, N: Normal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	5.316 ^a	1	0.021	
Fisher's Exact Test				0.041

Conduction velocity of the motor Ulnar nerve was abnormal in 11 (22%) of RA patients and 3 (6%) in controls and it was normal in 39 (78%) of RA patients and 47 (94%) of controls. There was statistically significant difference between the abnormal Conduction velocity of RA patients and controls (p=0.021).

Table 15: Median Nerve (Sensory) – Latency

			Group		Total
			RA	Control	
L	N	Count	43	49	92
		% within Group	86.0%	98.0%	92.0%
	AN	Count	7	1	8
		% within Group	14.0%	2.0%	8.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	4.891 ^a	1	0.027	
Fisher's Exact Test				0.059

Latency of the sensory Median Nerve was abnormal in 7 (14%) of RA patients and 1 (2%) in controls and it was normal in 43 (86%) of RA patients and 49 (98%) of controls. There was statistically significant difference between the abnormal latency of RA patients and controls (p=0.027).



Table 16: Median Nerve (Sensory) – Amplitude

			Group		Total
			RA	Control	
A	AN	Count	8	5	13
		% within Group	16.0%	10.0%	13.0%
	N	Count	42	45	87
		% within Group	84.0%	90.0%	87.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	1.507 ^a	1	0.220	
Fisher's Exact Test				0.326

Amplitude of the sensory Median Nerve was abnormal in 8 (16%) of RA patients and 5 (10%) in controls and it was normal in 42 (84%) of RA patients and 45 (90%) of controls. There was not statistically significant difference between the abnormal Amplitude of RA patients and controls (p=0.220).

Table 17: Median Nerve (Sensory) - Conduction velocity

			Group		Total
			RA	Control	
CV	AN	Count	9	2	11
		% within Group	18.0%	4.0%	11.0%
	N	Count	41	48	89
		% within Group	82.0%	96.0%	89.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	5.005 ^a	1	0.025	
Fisher's Exact Test				0.051

Conduction velocity of the sensory Median Nerve was abnormal in 9 (18%) of RA patients and 2 (4%) in controls and it was normal in 41 (82%) of RA patients and 48 (96%) of controls. There was statistically significant difference between the Conduction velocity of RA patients and controls (p=0.025).

Table 18: Ulnar Nerve (Sensory) – Latency

			Group		Total
			RA	Control	
L	N	Count	44	41	85
		% within Group	88.0%	82.0%	85.0%
	AN	Count	6	9	15
		% within Group	12.0%	18.0%	15.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	0.706 ^a	1	0.401	
Fisher's Exact Test				0.577

Latency of the sensory ulnar Nerve was abnormal in 6 (12%) of RA patients and 9 (18%) in controls and it was normal in 44 (88%) of RA patients and 41 (82%) of controls. There was not statistically significant difference between the abnormal Latency of RA patients and controls (p=0.401).

Table 19: Ulnar Nerve (Sensory) – Amplitude

			Group		Total
			RA	Control	
A	AN	Count	1	5	6
		% within Group	2.0%	10.0%	12.0%
	N	Count	49	45	94
		% within Group	98.0%	90.0%	94.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	11.960 ^a	1	0.253	
Fisher's Exact Test				0.403

Amplitude of the sensory ulnar Nerve was abnormal in 1 (2%) of RA patients and 5 (10%) in controls and it was normal in 49 (98%) of RA patients and 45 (90%) of controls. There was not statistically significant difference between the Amplitude of RA patients and controls (p=0.253).



Table 20: Ulnar Nerve (Sensory) - Conduction velocity

			Group		Total
			RA	Control	
CV	AN	Count	4	3	7
		% within Group	8.0%	6.0%	7.0%
	N	Count	46	47	93
		% within Group	92.0%	94.0%	93.0%
Total		Count	50	50	100
		% within Group	100.0%	100.0%	100.0%

N= Normal, AN= Abnormal

	Value	DF	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	0.154 ^a	1	0.695	
Fisher's Exact Test				1.000

Conduction velocity of the sensory ulnar Nerve was abnormal in 4 (8%) of RA patients and 3 (6%) in controls and it was normal in 46 (97%) of RA patients and 47 (94%) of controls. There was not statistically significant difference between the Conduction velocity of RA patients and controls (p=0.695).

Table 21: Abnormality in nerve conduction velocity of Motor nerves

Nerve	RA patients		Control	
	No.	Percentage	No.	Percentage
Median	13	26 %	5	10 %
Ulnar	11	22 %	3	6 %
Total	24	48 %	8	16 %

In rheumatoid arthritis, the most commonly affected motor nerve was median nerve (26%), then ulnar nerve (22%) and deep peroneal nerve (4%). In controls commonly affected nerve was median (10%) and ulnar (6%).

Table 22: Abnormality in nerve conduction velocity of Sensory nerves

Nerve	RA patients		Control	
	No.	Percentage	No.	Percentage
Median	9	18 %	2	4 %
Ulnar	4	8 %	3	6 %
Total	13	26 %	5	10 %

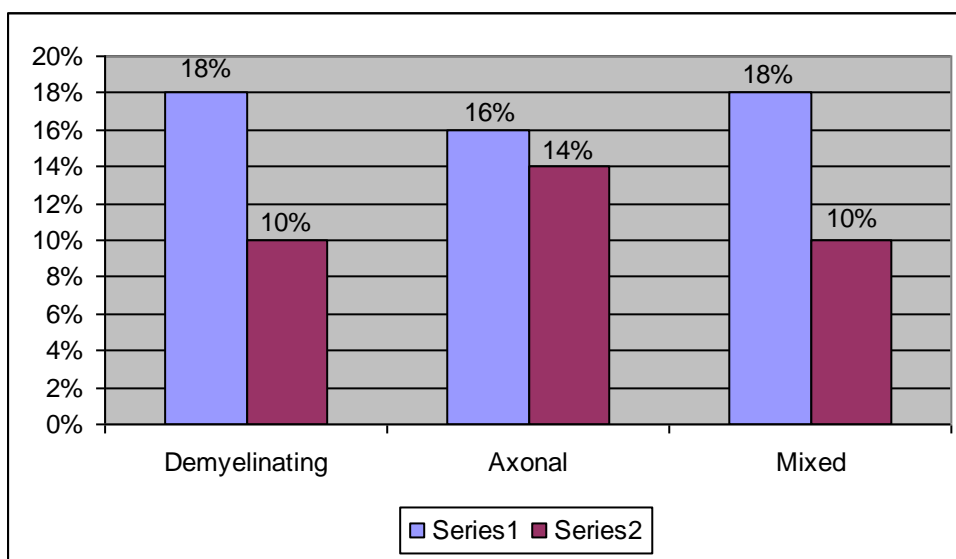
In rheumatoid arthritis, the most commonly affected sensory nerve was median nerve (18%), ulnar nerve (8%), then deep peroneal nerve and sural nerve (6%) each. In controls commonly affected nerve was ulnar (6%), then median (4%).



Table 23: Electrophysiology – Types of Neuropathy

Types of Neuropathy	RA Patients		Control	
	Frequency	percentage	Frequency	percentage
Demyelinating	9	18 %	5	10 %
Axonal	8	16 %	7	14 %
Mixed	9	18 %	5	10 %
Total	26	52 %	17	34 %
Normal	24	48 %	33	66 %

Graph no. 10: Types of Neuropathy:



Series 1 = RA patients, Series 2 = Controls.

In present study, in Rheumatoid Arthritis, 9 (18%) patients had nerve conduction finding suggesting Demyelinating neuropathy, 8 (16%) patients had Axonal neuropathy and 9 (18%) patients had mixed (axonal and demyelinating) neuropathy. Remaining 24 (48%) patients had normal conduction studies.

In controls, 5 (10%) patients had nerve conduction finding suggesting Demyelinating neuropathy, 7 (14%) patients had Axonal neuropathy and 5 (10%) patients had mixed neuropathy. Remaining 33(66%) patients had normal conduction studies.

V. DISCUSSION

Present study had several practical limitations. The major weakness of the study was the lack of a pathological gold standard namely “nerve biopsy” studies to confirm our electrophysiological findings. As a matter of fact none of the patients consented to a nerve biopsy. Most of the patients had history of intake of DMARDs and this might have confounded the study results, as certain drugs are known to affect peripheral nerves. Our study shows, regular electrophysiological studies will be helpful for early detection of neuropathy in patients with Rheumatoid Arthritis.

Electrical studies have contributed to understanding nerve and muscle damage in Rheumatoid Arthritis by confirming the segmental nature and anatomical extent of involvement and serving as an index of response to treatment.

1. Motor Nerve Conduction Studies

In Rheumatoid arthritis patients, 11 (22%) had prolonged distal latency, 9 (18%) had low amplitude of compound muscle action potential (CMAP) and 13 (26%) had low conduction velocity of motor median nerve. In ulnar nerve, 10 (20%) had prolonged distal latency, 4 (8%) had low amplitude of CMAP and 11 (22%) had low conduction velocity.

In controls, 2 (4%) had prolonged distal latency, 1 (2%) had low amplitude of compound muscle action potential (CMAP) and 5 (10%) had low conduction velocity of median nerve. In ulnar nerve, 6 (12%) had prolonged distal latency, 2 (4%) had low amplitude of CMAP and 3 (6%) had low conduction velocity.

Based on conduction velocity, the most commonly involved motor nerve were the median nerve 13 (26%) followed by ulnar nerve 11 (22%). This indicate that the upper limb was earliest affected than lower limb. In controls, commonly involved motor nerve was median nerve 5 (10%) followed by ulnar nerve 3 (6%).

2. Sensory nerve conduction studies

In Rheumatoid arthritis patients, 7 (14%) had prolonged distal latency, 8 (16%) had low amplitude of Sensory nerve action potential (SNAP) and 9 (18%) had low conduction velocity of median sensory nerve. In ulnar nerve, 6 (12%) had prolonged distal latency, 1 (2%) had low amplitude of CMAP and 4 (8%) had low conduction velocity.

In controls, 1 (2%) had prolonged distal latency, 5 (10%) had low amplitude of compound muscle action potential (CMAP) and 2 (4%) had low conduction velocity of median nerve. In ulnar nerve, 9 (18%) had prolonged distal latency, 5 (10%) had low amplitude of CMAP and 3 (6%) had low conduction velocity.

Based on conduction velocity, the most commonly involved sensory nerve were the median nerve 9 (18%) followed by ulnar nerve 4 (8%)

In controls, commonly involved sensory nerve was ulnar nerve 3 (6%) followed by median nerve 2 (4%).

The nerve conduction demonstrated decreased compound muscle action potential (CMAP) or sensory nerve action potential (SNAP) suggestive of Axonal changes while increased distal latency and delayed conduction velocities suggestive of Demyelinating changes in both motor and sensory nerves. Frequency of neuropathy was higher in the patients with RA when compared with the control group, and the difference was statistically significant ($p < 0.05$)

VI. CONCLUSION

Abnormal electrophysiological findings existed in more than 25% in rheumatoid arthritis and were independent of disease duration. According to this finding, we suggest that patients be investigated electro-physiologically despite the associated discomfort of testing. Clinical examination alone may fail to detect early peripheral neuropathy. Therefore, the inclusion of an electro-diagnostic examination for the RA patients is recommended in routine diagnostic evaluation.

REFERENCES

1. Young A, Dixey J, Cox N, Davies P, Devlin J, Emery P, et al. How does functional disability in early rheumatoid arthritis (RA) affect patients and their lives? Results of 5 years of follow-up in 732 patients from the Early RA Study (ERAS). *Rheumatology (Oxford)* 2000 Jun; 39 (6):603-11.
2. Symmons DP. Epidemiology of rheumatoid arthritis: determinants of onset, persistence and outcome. *Best Pract Res Clin Rheumatol* 2002 Dec; 16 (5):707-22.
3. Isaacs J, Moreland L. Pathogenesis. In: Isaacs J, Moreland L, editors. *Fast Facts - Rheumatoid Arthritis*. Health Press Limited, Oxford; 2002. p. 21-30.
4. Wordsworth P, Pile KD, Buckely JD, Lanchbury JS, Ollier B, Lathrop M, et al. HLA heterozygosity contributes to susceptibility to rheumatoid arthritis. *Am J Hum Genet* 1992 Sep; 51(3):585-91.
5. BJ Paul. History of rheumatology: what are the lessons for us? *Indian Journal of Rheumatology* 2006 December Volume 1, Number 3; pp. 123-128.
6. Bennett GA, Cobb S, Jacox R, Jessar RA, Ropes MW. Proposed diagnostic criteria for rheumatoid arthritis. *Bull Rheum Dis* 1956 Dec; 7(4):121-4.
7. Ropes MW, Bennett GA, Cobb S, Jacox R, Jessar RA. 1958 Revision of diagnostic criteria for rheumatoid arthritis. *Bull Rheum Dis* 1958 Dec; 9(4):175-6.
8. Arnett FC, Edworthy SM, Bloch DA, McShane DJ, Fries JF, Cooper NS, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 1988 Mar;31(3):315-24.
9. Wolfe F, Sharp JT. Radiographic outcome of recent-onset rheumatoid arthritis: a 19-year study of radiographic progression. *Arthritis Rheum* 1998; 41(9):1571-82.
10. Symmons DP. Epidemiology of rheumatoid arthritis: determinants of onset, persistence and outcome. *Best Pract Res Clin Rheumatol* 2002 Dec; 16 (5):707-22.
11. *Text Book Rheumatology*, Fourth Edition. William N. Kelley, Edward D. Harris, Shaun Ruddy, Clement B. Sledge.
12. *Harrisons Text Book of Principles of Internal Medicine*. Anthony S. Fauci, Eugene Braunwald, Dennis L. Kasper, Stephen L. Hauser, Dan L. Longo, J. Larry Jameson, Joseph Loscalzo. 17th Edition, Mc Graw Hil Medical Publication.
13. Gregersen PK, Silver J, Winchester RJ. The shared epitope hypothesis. An approach to understanding the molecular genetics of susceptibility to rheumatoid arthritis. *Arthritis Rheum* 1987 Nov; 30 (11):1205-13.
14. Kimura J. Principles and Pitfalls of Nerve Conduction Studies. *Ann Neurol* 16:415-429, 1984.
15. American Association of Electrodiagnostic Medicine. Recommended policy for electrodiagnostic studies. *Muscle Nerve* 1999; 22: S101-S104.
16. Ralph M. Buschbacher, Nathan D. Prahlow. Indiana University School of Medicine Indianapolis. *Manual of Nerve Conduction Studies* Second Edition. Demos Medical Publishing, LLC, 386 Park Avenue South, New York, New York 10016. RC77.B87 2006. 616.8'56—dc22.
17. Aminoff MJ. *Electromyography in Clinical Practice: Clinical and Electrodiagnostic Aspect of Neuromuscular Disease*. 3rd ed. New York: Churchill Livingstone, 1997.
18. Buschbacher RM. *Manual of Nerve Conduction Studies*. New York: Demos Medical, 1999.