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REVIEW ARTICLE

Electrodiagnostic Study of Cranial Nerves in Patients with Chronic Inflammatory Demyelinating Polyneuropathy

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Abstract:

Background:Nearly 50% of patients frequently misdiagnose chronic inflammatory demyelinating polyneuropathy (CIDP). Despite a lack of consensus, the diagnosis relies on clinical characteristics and then electrophysiological criteria.

Objectives: to test for cranial nerve involvement in typical CIDP and correlate the findings with clinical characteristics of CIDP patients. Additionally, the goal is to evaluate the subclinical involvement of the cranial nerves, even when the neurological examination remains normal.

Methods: A case-control study of 26 patients with CIDP with a duration of illness of 2-300 months and 26 age- and sex-matched healthy controls. Clinical examination includes the House-Brackmann Facial Nerve Grading System and Medical Research Council (MRC) scale for trapezius and masseter muscles. Electrophysiological study of facial, spinal accessory, and blink reflex (BR) was done.

Results: The majority of patients (80.77%) had a typical CIDP diagnosis. The motor latencies of facial and spinal accessory nerves in the patient group were noticeably longer than the controls. Similarly, the patients have significantly longer BR R1, iR2, and cR2 latencies. The disease duration and cR2, as well as spinal accessory motor latencies, were found to be positively correlated. The spinal accessory and facial motor latencies were abnormal in 41 and 40 nerves, respectively, in patients with normal clinical examinations.

Conclusions: Patients with CIDP exhibited multiple cranial palsies. Subclinical involvement of cranial nerves was evident in 3 quarters of the patients. Disease duration had a favorable relationship with BR and spinal accessory parameters.

Keywords: CIDP, facial, spinal accessory, BR

Introduction

The most prevalent type of chronic immune-mediated inflammatory polyneuropathy that damages the myelin sheath in the nerve roots and peripheral nerves is known as chronic inflammatory demyelinating polyneuropathy (CIDP), and it has several subtypes that fall under the category of causally treatable neuropathies (1, 2).

The Peripheral Nerve Society (EFNS/PNS) and the European Federation of Neurological Societies (EFNS) have defined CIDP as being progressive or relapsing for more than two months, showing electrophysiological or pathological signs of peripheral nerve demyelination, and responding to immunosuppressive or immunomodulating therapies (3, 4).

Cranial nerve palsy associated with CIDP has been discussed in several case series or reports, but its prevalence, traits, prognosis, and associations with the CIDP subtypes have rarely been systematically reported (5, 6). In about 30% of cases, cranial neuropathies have been documented, including optic, oculomotor, trigeminal, and facial neuropathies in patients with multifocal acquired demyelinating sensory and motor (MADSAM) neuropathy (7); in contrast, involvement of cranial nerves in patients with distal acquired demyelinating symmetric (DADS) neuropathy would suggest an alternative diagnosis (8).

A review of the literature revealed a reported incidence of cranial nerve involvement in 5% to 20% of patients with CIDP. According to a retrospective study, cranial palsy is frequent in MADSAM (48%), but less frequent in typical CIDP (11%) and DADS (11%) (6). Facial paralysis is the most commonly involved cranial nerve condition, followed by bulbar involvement and

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oculomotor nerve paralysis (6, 9). Typical CIDP patients usually have bilateral cranial nerve involvement, while MADSAM patients frequently have unilateral paralysis (6). However, reports of hypoglossal neuropathy in CIDP are especially rare (10).

The objectives of our study are to study cranial nerve involvement in typical CIDP and its variant using neurophysiological tests. Correlate the results with demographic and clinical data for CIDP patients. Study the correlations between cranial and peripheral NCSs in patients with typical or atypical CIDP.

Methods

A case-control study was conducted at the Neurophysiology Unit at Baghdad Teaching Hospital/Medical City for the period from January 2023 to September 2023. The study was approved by the Iraqi Board for Medical Specialization (Order # 240: Date 22/1/2023). All the participants were informed about the technique and the aim of the study, and informed consent was obtained from them.

The Participants

Twenty-six patients with CIDP of either sex (17 males and 9 females) were recruited for the study. Their age ranges between 15 and 63 years. The disease duration ranges between 3 months and 25 years. Patients with diabetes, stroke, multiple sclerosis, sarcoidosis, amyloidosis, hereditary neuropathies, Bell's palsy, connective tissue disease, or those with HIV infection were excluded.

Another 26 healthy, symptom-free, age- and sex-matched individuals comprised the control group.

Methods

History and Clinical examinations

The patients were referred by a senior neurologist and diagnosed with definite CIDP based on a thorough electrophysiological study and clinical manifestations, including abnormal increases in cerebrospinal fluid proteins of 2 months' duration.

The House-Brackmann Facial Nerve Grading System

The House-Brackmann (H-B) Facial Nerve Grading System is employed to assess the severity of symptoms in patients with facial paralysis. The system operates on a six-point scale, where grade I represents normal function, and grade VI indicates complete flaccid paralysis (11).

Medical Research Council (MRC) scale

The muscle strength of the trapezius and masseter muscles was assessed using the Medical Research Council (MRC) scale, where the examiner applied resistance to gauge the patient's strength on a scale ranging from 0 to 5. On this scale, a score of 0 indicates no muscle activation, and a score of 5 represents muscle activation against the examiner's full resistance and full range of motion (12).

Electrophysiological Assessments

Electrophysiologic testing was done by using Nihon Kohden, Japan. The room temperature was roughly maintained at 25°C-28°C during the test procedures, and skin temperature between 32°C and 34°C was ensured using a skin thermometer (13).

Nerve Conduction Study

The sensory conduction studies of median, ulnar, and sural nerves and motor conduction studies of median, ulnar, peroneal, tibial, facial, and spinal accessory nerves bilaterally, as well as the BR, were adopted according to the method of Preston and Shapiro (13). The parameters that were studied were the sensory latency (SL), sensory nerve action potential (SNAP) amplitude, sensory nerve conduction velocity (SNCV), distal motor latency (DML), compound muscle AP (CMAP), and motor NCV.

The examination of the facial nerve involved the application of stimulation directly over the anterior tragus, located in front of the lower ear, with subsequent recording of the nasalis muscle activity. In the study of the spinal accessory nerve, stimulation was applied posterior to the middle of the sternocleidomastoid muscle. Recording involved placing the active electrode over the muscle belly of the upper trapezius, with the reference electrode positioned distally over the shoulder joint (13).

For the motor nerves, the intensity of the stimulating current was adjusted manually to evoke a maximal muscle response, after which it was increased by 20–30% to ensure supramaximal stimulation. The electromyographic setting was a band-pass filter of 10 Hz-10 kHz, a sweep speed of 5 msec/division, and a sensitivity of 200 µV/division.

F-responses were recorded from the abductor pollicis brevis, abductor digiti minimi, and extensor hallucis brevis of both sides by stimulating the median at the wrist, ulnar at the wrist, and tibial at the ankle. Slight muscle contraction can enhance F-waves, and this can be advantageous in their identification. To assess F-wave latencies, 10 trials were automatically displayed on a storage oscilloscope as consecutive vertical sweeps. The distinctly identified F-waves were recorded and subsequently analyzed automatically by the program, which detected both the minimal and mean F-wave latencies (13).

To elicit the BR, the patient should be in a relaxed supine position on the examination table, with the eyes either open or gently closed. For recording the orbicularis oculi muscle, the active recording electrodes (G1) are positioned below the eye, just lateral and inferior to the pupil at mid-position. Place the corresponding reference electrodes (G2) just lateral to the lateral canthus on both sides. The ground electrode is situated on the mid-forehead or chin.

The supraorbital nerve was subjected to ipsilateral electrical stimulation. The current was gradually increased, typically 3-5 mA, starting from a baseline of 0 mA until achieving supramaximal stimulation, which produced the shortest latency and highest amplitude potentials. Stimulation of the nerve occurred effortlessly with low currents, usually requiring no more than 15-25 mA.

Once supramaximal stimulation was achieved, four to six responses were obtained on a rastered tracing and superimposed to determine the shortest response latencies. Next, we applied the same maneuver to the contralateral nerve. To prevent habituation, several seconds were ensured between successive stimulations.

The recorded BR components were R1, iR2, and cR2. Because the R2 potential varies in latency and morphology from stimulation to stimulation, four to six traces were registered and superimposed, with the shortest R2 latency selected.

The device must be set to the sweep speed at 5 or 10 ms/division and the initial sensitivity at 100 or 200 µV/division because the amplitudes of both R1 and R2 are quite small. The filter setting was 10 Hz-10 kHz.

Electromyography

Electromyographic activity of the first dorsal interosseous, brachioradialis, deltoid, tibialis anterior, gastrocnemius, and

vastus medialis or lateralis was analyzed using concentric needle electrodes (Micromed, code DIN42802, Italy). MUAPs were analyzed for duration and amplitude during rest, minimal, and maximal volitional effort. The setup used in this test was as follows: gain at 200 µV/division, sweep speed of 20 msec/division, and band-pass filter of 20 Hz to 10 kHz.

Statistical analysis

Statistical analyses were performed by using SPSS software version 26.0 (SPSS, Chicago). Continuous data were subjected to a normality test (Shapiro-Wilk test). Data with a normal distribution were presented as mean and standard deviation and analyzed with a Student's t-test. Data with non-normal distribution were presented as median and range and analyzed with the Mann-Whitney U test.

Categorical variables were expressed as number and percentage and analyzed with the chi-square test. Spearman's correlation test was used to explore the possible correlation of the non-normally distributed variables, and Pearson's correlation test was used to explore the possible correlation of the normally distributed variables. The mean and +3 SD were used to calculate the abnormal motor latencies and amplitude of facial and spinal accessory nerves.

A p-value less than 0.05 was considered to indicate a statistically significant difference.

Results

Demographic data

Age and sex did not significantly differ between the patients and controls. Disease duration ranges from 2 to 300 months. The majority of patients (80.77%) were diagnosed as having CIDP, four with multifocal acquired demyelinating sensory and motor neuropathy, while only one was diagnosed with distal acquired demyelinating symmetric neuropathy. The H-B grading system was used to assess the severity of facial weakness accordingly; the majority (80.77%) was scored as grade 1 bilaterally, and very few patients had grade 2 and 3.

Out of the total number of patients, eighteen (69.23%) showed grade 5 on the MRC scale of the trapezius muscle, whereas all the patients showed grade 5 on the MRC scale of the masseter muscle, as indicated in Table 1.

Electrophysiological data

No significant difference was demonstrated with respect to BR components, facial nerve latency and CMAP amplitude, and spinal accessory nerve latency and CMAP amplitude between the two sides for both control and patient groups, as shown in Tables 2 and 3. Therefore, we treated them as a single group for subsequent analysis.

The latencies of BR components and facial and spinal accessory motor latencies were significantly prolonged ($p < 0.001$) in the patient group versus the controls. On the contrary, the facial and spinal accessory CMAP amplitudes were significantly reduced ($p<0.001$) in the patient group relative to that of the controls, as shown in Table 4.

Correlation analysis

The data from the peripheral sensory and motor nerve conduction study of the upper and lower nerves showed no correlation with either disease duration or age. On the other hand, disease duration was positively correlated with right BR cR2 latency ($r = 0.433$, $p = 0.039$) and spinal accessory motor latency ($r = 0.574$, $p = 0.002$), as shown in Figure 1.

Figure 2 indicates a significant negative relationship between the MRC scale of the trapezius muscle and spinal accessory motor latency ($r = -0.481$, $p = 0.001$) and a positive correlation with spinal accessory CMAP amplitude ($r = 0.366$, $p = 0.008$).

Discussion

Regarding BR of the controls, the available data were consistent with numerous national studies that present nearly identical data (14, 15). The results of this study also agreed with numerous international studies conducted in Sudan (16) and India (17), as well as Senegal (18). The study's data on the facial nerve aligns with the findings of a national study (19) as well as international studies conducted in Sudan (20) and India (21).

Additionally, the spinal accessory nerve data were consistent with findings from numerous international studies conducted in Korea (22) and Egypt (23); however, the CMAP amplitude of the current study was higher than that of the latter study, which may be due to the latter's small sample size, which examined only 10 controls.

The R1, iR2, and cR2 latencies of the BR are prolonged in patients of this study, which makes them useful markers for identifying demyelination in CIDP patients. Kimura (24) observed that in 14 patients with chronic inflammatory polyneuropathy, there was a significant prolongation of the latencies of the direct R1 responses. This conclusion is further supported by a study by Kokubun et al. (25), which found that in 4 patients, the responses were not evoked bilaterally and the R1 and R2 latencies were prolonged.

Varela and Rubin examined an eighty-three-year-old woman who had facial numbness and weakness. They discovered bilaterally prolonged blink and jaw-jerk reflex latencies (5). Kokubun and Hirata (25) discovered in their study that 12 patients (60%) had an increased R1 response latency, and 17 patients (85%) had an abnormal BR.

In another study conducted on 58 CIDP patients, Wang et al. identified R1 prolongation (>13 ms) occurring in 62.1% of patients (94). A recent study showed prolonged R1 latencies identified in 23 of 104 patients (22.1%) with CIDP who had more severe functional impairments according to the ALSFRS-R (26).

The present study showed a positive correlation between cR2 latency and disease duration, which contradicts the findings of Kokubun and Hirata (25), probably because of the small sample size of the latter study. However, the absent correlation between clinical grading and latencies of the present study was consistent with the latter study.

The current study's findings indicated lower CMAP amplitudes and longer latencies about the facial nerve. Research on this nerve is scarce and has mostly focused on clinical assessment. Kokubun and Hirata (25) discovered that patients with CIDP had significantly longer facial nerve latency ($P < 0.001$). Based on clinical and electrophysiological features, the first sign of CIDP in a 40-year-old patient is unilateral facial weakness (27).

The most common comorbidity in two recent studies was facial paralysis, which was followed by bulbar involvement and oculomotor nerve paralysis (28). In contrast, typical CIDP was associated with multiple cranial neuropathies involving the bilateral trigeminal and facial nerves, as well as the right optic and lower cranial nerves (29).

Concerning the spinal accessory nerve, the study's CIDP patients

also displayed longer latencies and reduced CMAP amplitudes. The majority of the literature on this subject focused on case study reports. The 9th, 10th, 11th, and 12th cranial nerves were paralyzed in a 14-year-old boy who had been diagnosed with CIDP after a year of progressive quadriparesis (30).

Electromyography and nerve conduction studies of an adult male showed evidence for a multifocal, mainly motor neuropathy involving the left spinal accessory and hypoglossal nerves, combined with the presence of median and ulnar proximal conduction blocks bilaterally (31).

A recent study discovered an autoimmune nodo-paranodopathy (CIDP variant) in a 46-year-old previously healthy woman. Except for a right accessory spinal nerve that has a low amplitude CMAP of 2.5 mV and a prolonged distal motor latency of 8.28 ms, the MNCs are not responding (32). In a different recent study, an 18-year-old patient with MADSAM (CIDP variant) was examined using sonography. The results showed multifocal nerve enlargement and a change in the normal fascicular pattern within clinically affected cranial nerves, including the optic, spinal accessory, and hypoglossal nerves (33). This finding supports cranial nerve ultrasonography's potential uses in CIDP. Lastly, a recent study examined the clinical data of 132 consecutive CIDP patients, including those with distal acquired demyelinating symmetric (DADS) (n = 9), typical CIDP (n = 89), MADSAM (n = 31), and other conditions (n = 3). The study links cranial nerve impairment, a clinical feature of CIDP, to more severe phenotypes. Furthermore, CIDP implicates all cranial nerves except the olfactory, trochlear, and hypoglossal nerves. Additionally, there are differences in the frequency and occurrence of clinically noticeable involvement of the cranial nerves among the CIDP subtypes; however, facial and bulbar palsy is the most common among the typical CIDP and its variants. They conclude that cranial palsy is more common and unilateral in MADSAM among the CIDP subtypes, whereas it is less prevalent and bilateral in typical CIDP and DADS. Facial and bulbar palsy are indicative of more severe and widespread inflammation in typical CIDP (6).

The results of this study show a strong correlation between the MRC scale of the trapezius muscle and the motor NC data of the spinal accessory nerve. To the best of our knowledge, no literature contained any comparable data. Table 3-12 indicates multiple relationships between the peripheral nerves and cranial nerves data; among them was the proximal conduction block of all tested peripheral motor nerves. This was also reported by Körner et al. (26).

In conclusion, patients with CIDP had abnormal cranial nerves. The disease duration had a favorable relationship with BR and spinal accessory parameters. There was a significant correlation between the cranial and peripheral nerves.

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Table 1. Demographic data of the study population

Parameter	Patients (n=26)	Controls (n=26)	p-value
Age, years			
Mean±SD	40.23±14.94	40.08±14.92	0.946
Range	15-63	15-63	
Sex			
Males	17(65.38%)	12(46.15%)	0.132
Females	9(34.62%)	14(53.85%)	
Disease duration, months			
Mean±SD	40.96±60.120		
Range	2-300		
Disease type			
CIDP	21(80.77%)		
MADSAM	4(15.9%)		
DADS	1(3.85%)		
H-B for Right Facial Nerve			
Grade 1	21(80.77%)		
Grade 2	4(15.9%)		
Grade 3	1(3.85%)		
H-B for Left Facial Nerve			
Grade 1	21(80.77%)		
Grade 2	2(7.69%)		
Grade 3	3(11.54%)		
MRC scale of both trapezii			
Grade 4	8(30.77%)		
Grade 5	18(69.23%)		
MRC scale of both masseters			
Grade 5	26(100%)		

CIDP = chronic inflammatory demyelinating polyneuropathy; MADSAM = multifocal acquired demyelinating sensory and motor neuropathy; DADS = Distal acquired demyelinating symmetric

H-B = House-Brackmann Facial Nerve Grading System; MRC = Medical Research Council.

Table 2. Cranial nerve conduction study of the controls

Nerve	Right side (n=26)	Left side (n=26)	p-value
Blink Reflex			
R1 latency, ms	10.34±1.07	10.59±0.84	0.178
iR2 latency, ms	31.09±3.16	31.29±3.21	0.821
cR2 latency, ms	31.82±2.51	32.88±2.78	0.977
Facial Nerve			
Motor Latency, ms	2.6±0.51	2.57±0.43	0.380
CMAP amplitude, mV	2.9±0.75	2.88±0.59	0.158
Spinal Accessory Nerve			
Motor Latency, ms	2.06±0.38	2.09±0.37	0.725
CMAP amplitude, mV	6.31±1.91	5.89±1.82	0.713

Table 3. Cranial nerve conduction study of the patients

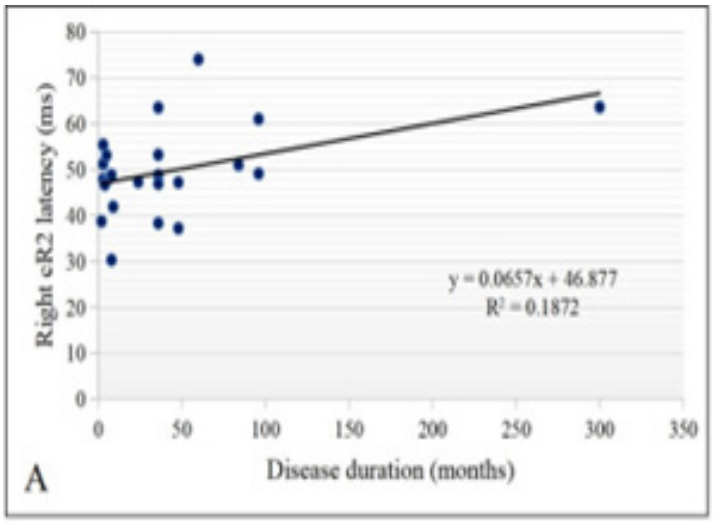
Nerve	Right side (n=26)	Left side (n=26)	p-value
Blink Reflex			
R1 latency, ms	17.04±5.04	16.16±3.82	0.185
iR2 latency, ms	47.14±9.92	48.64±10.7	0.989
cR2 latency, ms	49.7±9.64	52.87±12.52	0.199
Facial Nerve			
Motor Latency, ms	5.52±2.44	5.39±2.26	0.593
CMAP amplitude, mV	1.47±0.62	1.41±0.58	0.830
Spinal Accessory Nerve			
Motor Latency, ms	5.19±3.14	5.29±2.87	0.694
CMAP amplitude, mV	3.62±2.01	3.65±1.74	0.688

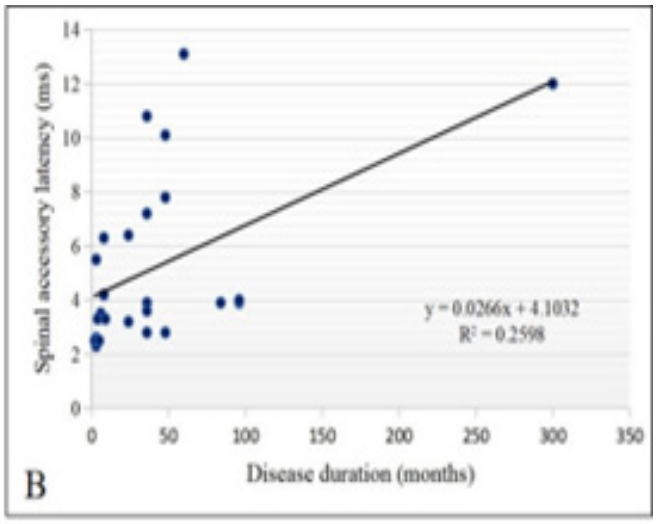
CMAP = compound muscle action potential

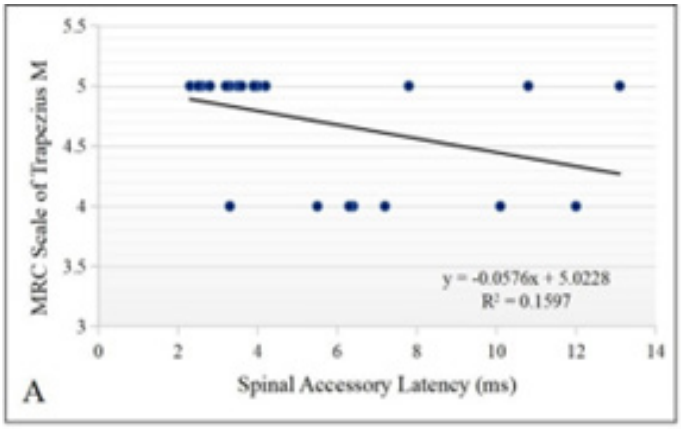
Table 4. Cranial motor nerve conduction study of the patients and controls

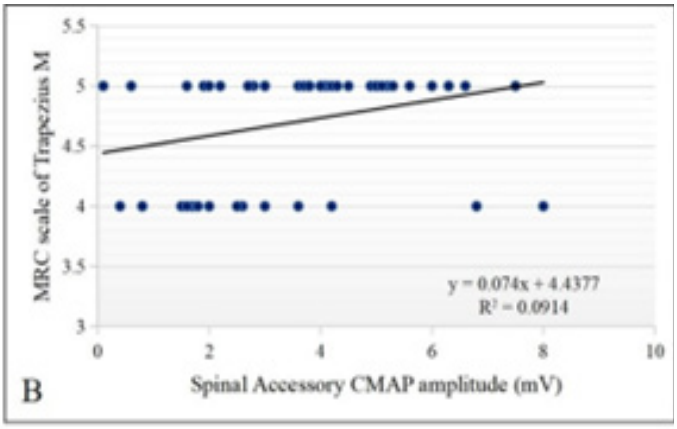
Parameter	Patients (n=52)	Controls (n=52)	p-value
Blink Reflex			
R1 latency, ms	16.58±4.42	10.46±0.96	0.001
iR2 latency, ms	47.92±10.25	31.19±2.70	0.001
cR2 latency, ms	51.35±11.23	32.35±2.68	0.001
Facial Latency, ms			
Mean±SD	5.46±2.33	2.59±0.47	0.001
Median	4.9	2.6	
Range	2.2-10.6	1.7-3.5	
Facial CMAP amplitude, mV			
Mean±SD	1.44±0.59	2.89±0.67	0.001
Median	1.5	2.9	
Range	0.3-2.9	1.9-436	
SA Motor Latency, ms			
Mean±SD	5.24±2.98	2.07±0.37	0.001
Median	4.1	2.0	
Range	2.1-13.1	1.4-3.0	
SA CMAP amplitude, mV			
Mean±SD	3.64±1.86	6.1±1.86	0.001
Median	3.8	5.95	
Range	0.1-8.0	3.9-11.1	

CMAP = compound muscle action potential; SA = spinal accessory


Right cR2 latency (ms)
y = 0.0657x + 46.877
R² = 0.1872
Disease duration (months)
A


Spinal accessory latency (ms)
y = 0.0266x + 4.1032
R² = 0.2598
Disease duration (months)
B


MRC Scale of Trapezius M
y = -0.0576x + 5.0228
R² = 0.1597
Spinal Accessory Latency (ms)
A


MRC scale of Trapezius M
y = 0.074x + 4.4377
R² = 0.0914
Spinal Accessory CMAP amplitude (mV)
B