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# Assessment of Autonomic Nervous System Functions in Patients with Chronic Inflammatory Demyelinating Polyradiculoneuropathy: An Electrodiagnostic Study

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# ASSESSMENT OF AUTONOMIC NERVOUS SYSTEM FUNCTIONS IN PATIENTS WITH CHRONIC INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY: AN ELECTRODIAGNOSTIC STUDY

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## ABSTRACT

### Background and Objective:

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a chronic immune-mediated neuropathy predominantly affecting the peripheral nervous system including the nerve roots. The objectives of this study were to assess autonomic dysfunction in patients with CIDP using electrodiagnostic techniques and to examine its relationship with clinical and demographic characteristics, COMPASS-31 scores, and the Hughes disability scale.

### Methods:

This case–control observational study included 25 patients with CIDP and 26 age- and sex-matched healthy controls and was conducted at the Neurophysiology Unit of Baghdad Teaching Hospital, Baghdad from February to November, 2024. All participants underwent autonomic function assessment, including Ewing's cardiovascular tests and sympathetic skin response (SSR). COMPASS-31 and Hughes disability scores were recorded for clinical correlation. The independent Student's t-test was applied to normally distributed quantitative variables, while non-normally distributed variables were assessed using the Mann–Whitney U test. Categorical variables were analyzed with the chi-square test. Associations between variables were examined using Pearson's correlation coefficient for parametric data and Spearman's rank correlation coefficient for non-parametric data.

### Results:

CIDP patients exhibited significantly reduced heart rate variability and exaggerated blood pressure fluctuations, along with prolonged SSR latencies and decreased amplitudes compared to controls. Autonomic dysfunction involved both parasympathetic and sympathetic systems. Significant correlations were found between autonomic parameters and age, disease duration, COMPASS-31 score, Hughes score, and sex.

### Conclusion:

Autonomic dysfunction in CIDP is common, typically mild to moderate, and affects both divisions of the ANS. Clinical and demographic variables may influence the pattern and severity of dysfunction.

**Keywords:** Autonomic dysfunction, Chronic inflammatory demyelinating polyradiculoneuropathy, Electrodiagnostic testing, Sympathetic skin response, Vagal function.

## INTRODUCTION

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated disorder affecting the peripheral nervous system, including the nerve roots characterized by symmetric weakness in both proximal and distal muscles.[1,2] The disease course can be monophasic, relapsing, or chronically progressive, and diagnosis requires persistence of symptoms for a minimum of eight weeks, corroborated by electrodiagnostic evidence of peripheral nerve demyelination and demonstrable response to immunotherapy.[3-5] Epidemiologically, CIDP exhibits a male predominance with a 2:1 sex ratio. A recent meta-analysis estimated its incidence at 0.3 per 100,000 population, with prevalence varying from 0.8 to 8.9 per 100,000 and rising

function.[6-11] Dysfunction of the ANS, or dysautonomia, may arise from lesions within the brainstem nuclei, intermediolateral spinal columns, or peripheral autonomic fibers.[12] Although AD is well-characterized in small-fiber neuropathies such as autoimmune autonomic ganglionopathy, its presence in CIDP has historically been under-recognized and poorly characterized.[13-15] Given the potential for confounding by comorbidities and pharmacologic influences, careful exclusion of these factors is essential before attributing autonomic symptoms to CIDP itself. Assessment of ANS involvement may include both laboratory-based measures and structured patient-reported tools. Among these, the Composite Autonomic Symptom Score (COMPASS-31) is the most widely validated instrument.[16-19]

Despite increasing awareness of the broader systemic manifestations of CIDP, severity, and clinical correlates of autonomic dysfunction in this disorder remain inadequately explored. This study aims to address this critical knowledge gap by systematically evaluating autonomic involvement in patients with typical CIDP using electrodiagnostic testing. Additionally, it investigates associations between autonomic findings and demographic or clinical variables, and assesses correlations with standardized functional scores including COMPASS-31 and the Hughes grading scale.

## METHODS

This Case-Control observational study was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines to ensure transparent and comprehensive reporting.[20] This study was conducted between February and November 2024 at the Neurophysiology Unit of Baghdad Teaching Hospital, Iraq.

All participants were diagnosed with typical CIDP according to the EFNS/PNS criteria and underwent systematic clinical and autonomic evaluations. Thirteen patients were administered immunomodulatory drugs: two patients underwent plasma exchange 10 days prior to testing, three patients received IVIg two days to one week before testing, two patients on azathioprine discontinued treatment on the day of testing, and six patients on prednisone ceased treatment the night before testing. A senior neurologist conducted detailed history taking and neurological examinations.

The functional status of patients was assessed using the Hughes Functional Grading Scale (F-score), which evaluates motor disability on a scale from 0 to 6. A score of 0 denotes normal motor function; 1 indicates the ability to run with minimal symptoms or signs; 2 signifies the ability to walk 5 meters independently; 3 implies walking 5 meters with aids; 4 corresponds to being chair- or bed-bound; 5 represents the need for assisted ventilation; and 6 indicates death.

Autonomic symptom burden was quantified using the Composite Autonomic Symptom Score-31 (COMPASS-31).[21] This validated instrument consists of 31 patient-reported items divided into six domains: orthostatic intolerance (10 points), vasomotor (6 points), secretomotor (7 points), gastrointestinal (28 points), bladder (9 points), and pupillomotor (15 points). The total score ranges from 0 to 100, with higher scores reflecting more severe autonomic dysfunction.

Autonomic testing was performed using Keypoint (Medtronic, Denmark) and Micromed (Italy) electromyography systems. All assessments were conducted in a temperature- and light-controlled environment during morning hours. The room temperature was maintained between 22°C and 24°C, and patients' skin temperature was ensured to be at least 35°C using a skin thermometer. Patients were instructed to abstain from caffeine, nicotine, heavy meals, and medications with autonomic effects for at least 24 to 48 hours prior to testing, if medically permissible. They were also asked to avoid applying lotions or powders below the neck and to remain well hydrated.

For parasympathetic testing, R–R interval variability was measured using surface electrodes placed on the left anterior chest and axillary line, with a ground electrode at the sternal midline. Recordings were taken at rest, during deep breathing, the Valsalva maneuver, and active standing. R peaks were monitored via oscilloscope for interval changes, using a 200 $\mu$V sensitivity, 1–20 Hz band-pass filter, and 0.5 s/div sweep. For deep breathing, subjects followed a 6-breaths-per-minute pattern, and respiratory sinus arrhythmia was assessed via the percentage change in R–R intervals. In the Valsalva maneuver, patients exhaled at 40 mmHg for 15 seconds, and the Valsalva ratio [the longest R–R interval after strain (phase IV) divided by the shortest during strain (phase II)] was calculated—values below 1.21 were abnormal. For standing, HR was recorded after 20 minutes of supine rest, followed by upright posture. The 30:15 ratio (R–R interval at the 30th vs. 15th beat post-standing) was used, with a ratio $\geq$1.04 considered normal.

For sympathetic nervous system testing, Blood pressure

response to standing was assessed after 20 minutes of rest using a mercury sphygmomanometer. BP was measured at 1–3 minutes post-standing. A systolic drop >20 mmHg or diastolic drop >10 mmHg indicated orthostatic hypotension. Sympathetic skin response (SSR) was tested by placing the active electrode on the palm or sole, with reference and ground electrodes on the dorsum and ankle, respectively. A 12–20 mA current with 0.1 msec pulse width was applied at the contralateral wrist or ankle. Latency and amplitude were recorded, with a sweep speed of 500 msec/div and sensitivity of 200–1000 $\mu$V/div. Normal SSR values: upper limbs—latency 1.5 ± 0.1 s, amplitude 0.5 ± 0.1 mV; lower limbs—latency 2.1 ± 0.2 s, amplitude 0.1 ± 0.04 mV.

Patients were classified based on the number of abnormal autonomic test results. Those with one abnormal test were considered to have early autonomic involvement, two abnormal tests indicated definitive involvement, and three or more abnormal tests suggested severe autonomic dysfunction. A total of six autonomic parameters were used to determine the severity classification.

All statistical analyses were performed using SPSS version 25 (IBM Corporation, USA). Data were initially assessed for normality. Normally distributed quantitative variables were expressed as mean ± standard deviation and compared using the independent Student's t-test. Non-normally distributed variables were expressed as median and range and analyzed using the Mann–Whitney U test. Categorical variables were presented as counts and percentages and evaluated using the chi-square test. Correlations between variables were analyzed using Pearson's correlation coefficient for parametric data and Spearman's rank correlation for non-parametric data.

The study was approved by the institutional ethics committee, and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

## RESULTS

### *Demographic and Clinical Characteristics*

The study population comprised 25 patients with typical CIDP and 26 age- and sex-matched healthy controls. There was no statistically significant difference in age between the two groups (41.40±11.24 years in patients versus 39.85±10.38 years in controls; p=0.610). Similarly, sex distribution showed no significant variation, with males constituting 56% of the patient group and 53.85% of the control group (p=0.550). The average disease duration among CIDP patients was 3.75 ± 3.88 years, with a wide range spanning from one to 20 years. Regarding clinical evaluation scores, the mean COMPASS-31 score in the CIDP group was 8.80±7.42 versus 2.46 ± 3.06 of the controls (p = 0.001), while the mean Hughes disability score was 2.24±0.93. At the time of assessment, 52% of the patients were receiving immunomodulatory therapy.

### *Autonomic Function Test Results*

A comparative analysis of autonomic function revealed significant differences between CIDP patients and healthy controls across both parasympathetic and sympathetic domains (Table 1). Parasympathetic parameters, including heart rate responses to normal breathing (HRNB), deep breathing (HRDB), Valsalva maneuver (HRVals), and standing (HRS), were all significantly reduced in patients (p-values ranging from 0.032 to <0.001), indicating impaired vagal activity. Similarly, sympathetic dysfunction was evident, with patients exhibiting significantly greater systolic and diastolic blood pressure drops (p = 0.009 and 0.005, respectively), prolonged latencies in both palmar and planter sympathetic skin responses (p = 0.008 for both), and reduced SSR amplitudes (p = 0.014 and <0.001, respectively).

Table 1 shows a summary of autonomic function test results in the study population (parasympathetic and sympathetic domains).

**Table 1: Summary of Autonomic Function Test Results in the Study Population (Parasympathetic and Sympathetic Domains)**

| Autonomic Domains | Test Parameter | Controls (n=26) | Patients (n=25) | p-value |
|---|---|---|---|---|
| Parasympathetic Function | HRNB (beat/min) | 26.0 ± 4.59 | 18.01 ± 7.39 | <0.001 |
| | HRDB (beat/min) | 31.50 ± 7.08 | 25.76 ± 11.08 | 0.032 |
| | HRVals | 1.93 ± 0.32 | 1.60 ± 0.41 | 0.002 |
| Sympathetic Function | HRS | 1.72 ± 0.44 | 1.12 ± 0.57 | <0.001 |
| | SBP Drop (mmHg) | -1.54 ± 5.25 | 3.4 ± 8.86 | 0.009 |
| | DBP Drop (mmHg) | -2.31 ± 5.33 | 2.65 ± 5.97 | 0.005 |
| | Palmar SSR Latency (s) | 1.31 ± 0.21 | 1.63 ± 0.54 | 0.008 |
| | Palmar SSR Amplitude (mV) | 3.58 ± 1.93 | 2.44 ± 1.09 | 0.014 |
| | Planter SSR Latency (s) | 1.98 ± 0.36 | 2.39 ± 0.64 | 0.008 |
| | Planter SSR Amplitude (mV) | 3.01 ± 1.69 | 1.58 ± 0.46 | <0.001 |

HRNB = heart rate during normal breathing; HRDB = heart rate during deep breathing; HRVals = heart rate during Valsalva; HRS = heart rate during standing; SSR = sympathetic skin response; SBP = systolic blood pressure; DBP = diastolic blood pressure.

***Distribution Based on the Number of Abnormal Autonomic Tests***

Among the patients, three (12%) exhibited normal autonomic function across all tests. Eight patients (32%) showed one abnormal test result, while 10 (40%) had abnormalities in two tests. Three patients (12%) demonstrated abnormalities in three autonomic tests, and one patient (4%) had all four autonomic tests affected. These data reflect the heterogeneous extent of autonomic impairment in CIDP.

***Distribution Based on the Severity of Autonomic Dysfunction***

When categorized according to severity, three patients (12%) were found to have no autonomic dysfunction, while eight (32%) demonstrated early dysfunction. Ten patients (40%) were classified as having definite autonomic dysfunction, and four (16%) showed evidence of severe dysfunction. The type of dysfunction varied: four patients (16%) had isolated parasympathetic involvement, six (24%) had purely sympathetic dysfunction, and 12 (48%) had combined sympathetic and parasympathetic abnormalities. Notably, among the ten patients with definite dysfunction, eight (32%) exhibited combined impairment, and all four patients with severe dysfunction had both systems involved.

***Correlation Between Autonomic Measures and Demographics***

Correlation of age, disease duration, COMPASS-31 score Hughes score are presented in Table 2.

| Table 2: Correlation of Age, Disease Duration, COMPASS-31 Score and Hughes Score with Autonomic Function TestSympathetic Domains) | | | | |
|---|---|---|---|---|
| **Variable** | **Age, years** | **DD, years** | **COMPASS-31 score** | **Hughes score** |
| **HRNB** | r = -0.386 | r = -0.010 | r = -0.480 | r = -0.188 |
| | p = 0.057 | p = 0.961 | p = 0.015 | p = 0.368 |
| **HRDB** | r = -0.346 | r = 0.057 | r = -0.522 | r = -0.157 |
| | p = 0.091 | p = 0.785 | p = 0.007 | p = 0.455 |
| **SBP drop** | r = 0.444 | r = 0.462 | r = 0.142 | r = 0.242 |
| | p = 0.026 | p = 0.020 | p = 0.497 | p = 0.244 |
| **DBP drop** | r = 0.462 | r = 0.264 | r = 0.081 | r = 0.317 |
| | p = 0.020 | p = 0.203 | p = 0.699 | p = 0.123 |
| **Palmar SSR amplitude** | r = -0.397 | r = -0.191 | r = -0.449 | r = -0.404 |
| | p = 0.004 | p = 0.407 | p = 0.041 | p = 0.050 |
| **Planter SSR latency** | r = 0.311 | r = 0.541 | r = 0.260 | r = 0.189 |
| | p = 0.030 | p = 0.037 | p = 0.260 | p = 0.364 |
| **Planter SSR amplitude** | r = -0.164 | r = -0.198 | r = -0.143 | r = -0.551 |
| | p = 0.259 | p = 0.366 | p = 0.514 | p = 0.006 |

DD= disease duration; HRNB = heart rate during normal breathing; HRDB = heart rate during deep breathing; SSR = sympathetic skin response; SBP = systolic blood pressure; DBP = diastolic blood pressure.

***Correlation Between Age, Disease Duration, and Autonomic Function Parameters***

The analysis showed significant links between age and autonomic functions. Age positively correlated with systolic (r=0.444, p=0.026) and diastolic (r=0.462, p=0.020) blood pressure drops, indicating older patients experience greater orthostatic BP decline. It also linked age with longer plantar SSR latency (r=0.541, p=0.037), hinting at delayed sympathetic conduction, while negatively correlating with palmar SSR amplitude (r=-0.397, p=0.004), reflecting reduced sympathetic output. Disease duration was related to increased systolic BP drop and plantar SSR latency.

***Correlation of COMPASS-31 Scores with Autonomic Dysfunction***

The COMPASS-31 score, a subjective measure of autonomic symptoms, demonstrated significant negative correlations with multiple objective autonomic function parameters (Figure 1). Specifically, HRNB was negatively correlated with COMPASS-31 score (r = -0.480, p = 0.015), as was HRDB (r = -0.522, p = 0.007). Moreover, a negative correlation was observed between COMPASS-31 score and palmar SSR amplitude (r = -0.449, p = 0.041).

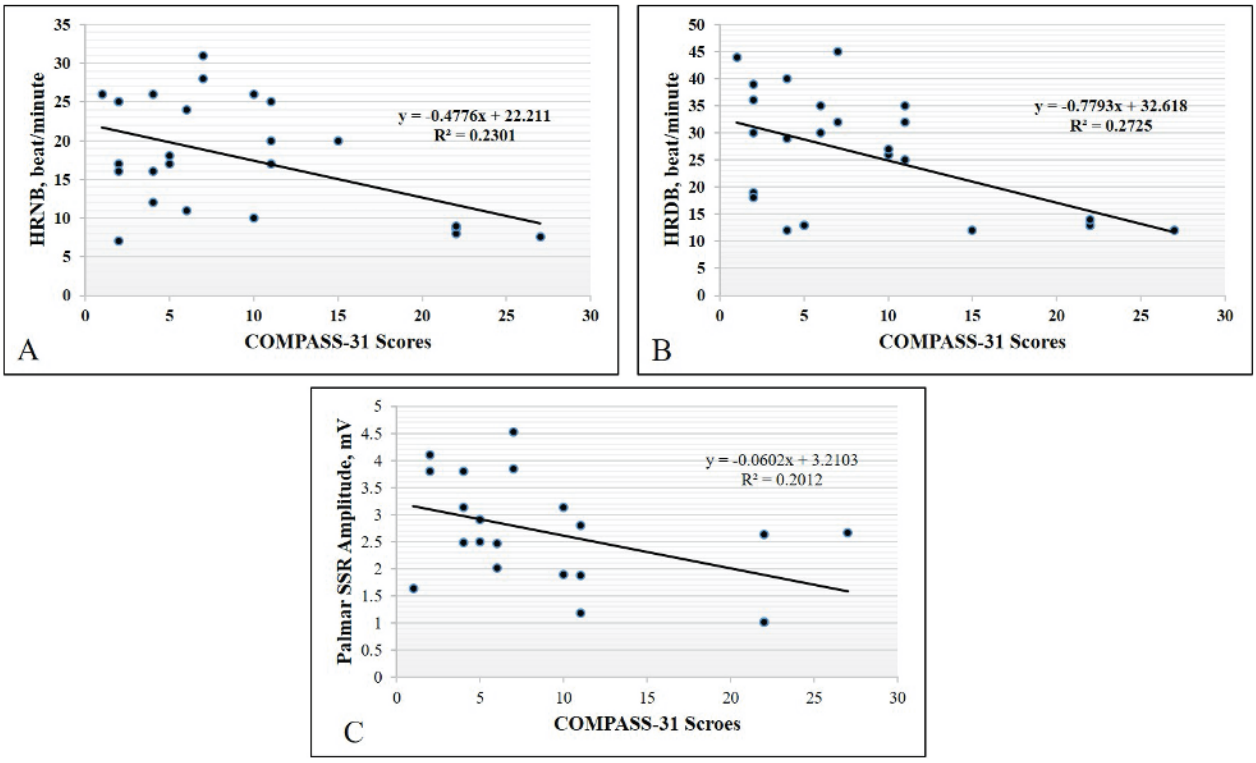


**Figure 1:** Scatter plots illustrating negative correlations between COMPASS-31 scores and selected autonomic function parameters in CIDP patients.
**A:** Scatter plot and regression line showing a negative correlation between COMPASS-31 score and HRNB, indicating reduced parasympathetic activity with higher symptom burden.
**B:** Scatter plot and regression line showing a negative correlation between COMPASS-31 score and HRDB, reflecting impaired vagal modulation in patients with higher autonomic symptom scores.
**C:** Scatter plot and regression line showing a negative correlation between COMPASS-31 score and palmar SSR amplitude, suggesting reduced sympathetic output in patients with more severe autonomic symptoms.

**Correlation of Hughes Functional Score with Autonomic Parameters**

The Hughes functional score exhibited a weaker but notable correlation with specific SSR measures (Figure 2). Palmar SSR amplitude showed a negative correlation (r = -0.404, p = 0.050), and planter SSR amplitude demonstrated an even stronger inverse correlation (r = -0.551, p=0.006)

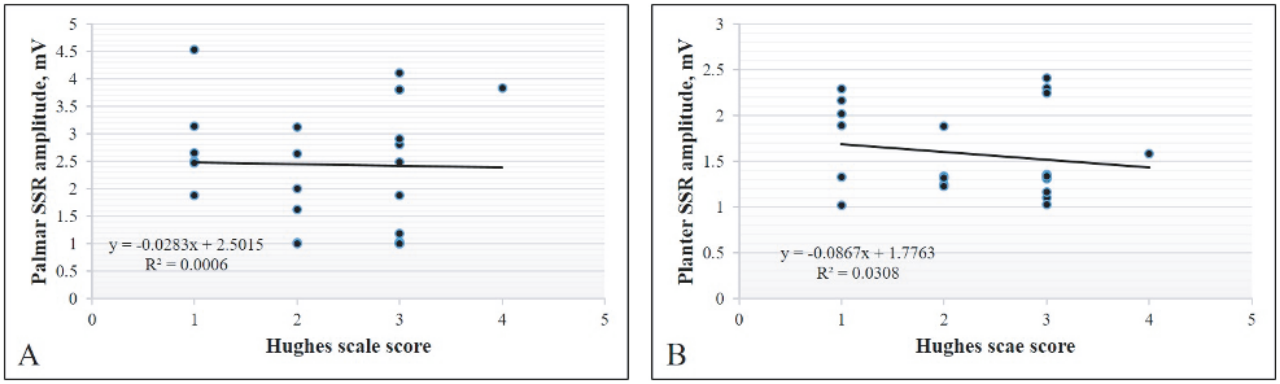

**Figure 2:** Scatter plots demonstrating the inverse relationship between Hughes functional disability scores and sympathetic sudomotor parameters in CIDP patients.
**A:** Scatter plot and regression line showing a negative correlation between Hughes score and palmar SSR amplitude, indicating reduced sympathetic activity with increased motor disability.
**B:** Scatter plot and regression line showing a stronger negative correlation between Hughes score and plantar SSR amplitude, suggesting that greater functional impairment is associated with diminished peripheral sympathetic output.

***Association of Autonomic Function with Sex and Treatment Status***

Analysis of parasympathetic and sympathetic autonomic function tests revealed no statistically significant differences between males and females across most parameters, except for palmar SSRL, which was significantly prolonged in males compared to females (1.88±0.62s vs. 1.34±0.19s, p=0.011), indicating potential sex-based variation in sympathetic reactivity. Regarding treatment status, none of the autonomic parameters showed significant differences between treated and untreated patients, though a borderline significance was observed in the HRS (p=0.052) (Table 3).

**Table 3: Association of Sex and Treatment with Parasympathetic and Sympathetic Autonomic Function Tests**

| Parameter | Sex Difference | | | Treatment | | |
|---|---|---|---|---|---|---|
| | Males (n=14) | Females (n=11) | p-value | Yes (n=13) | No (n=12) | p-value |
| **Parasympathetic Tests** | | | | | | |
| HRNB, b/m | 16.73±8.59 | 19.64±5.46 | 0.403 | 18.09±7.87 | 17.92±7.18 | 0.852 |
| HRDB, b/m | 23.71±10.89 | 28.38±11.28 | 0.244 | 25.77±11.77 | 25.75±10.81 | 0.979 |
| HRVals | 1.74±0.74 | 1.85±0.38 | 0.634 | 1.84±0.69 | 1.73±0.51 | 0.673 |
| HRS, b/m | 1.53±1.04 | 1.46±0.85 | 0.979 | 1.20±0.69 | 1.83±1.09 | 0.052 |
| **Sympathetic Tests** | | | | | | |
| Palmar SSRL, s | 1.88±0.62 | 1.34±0.19 | 0.011 | 1.70±0.61 | 1.56±0.48 | 0.522 |
| Palmar SSRA, mV | 2.12±1.01 | 2.23±0.79 | 0.129 | 2.23±0.79 | 2.64±1.33 | 0.371 |
| Planter SSRL, s | 2.46±0.77 | 2.31±0.50 | 0.593 | 2.18±0.84 | 2.57±0.34 | 0.153 |
| Planter SSRA, mV | 1.53±0.35 | 1.63±0.56 | 0.591 | 1.43±0.38 | 1.71±0.50 | 0.145 |
| SBP drop, mmHg | 4.29±10.89 | 2.27±5.64 | 1.000 | 1.54±5.16 | 5.83±11.84 | 0.320 |
| DBP drop, mmHg | 3.93±5.61 | 0.91±6.25 | 0.134 | 2.31±3.30 | 2.92±8.11 | 0.769 |

HRNB = heart rate during normal breathing; HRDB = heart rate during deep breathing; HRVals = heart rate during Valsalva; HRS = heart rate during standing; SSRL = sympathetic skin response latency; SSRA = sympathetic skin response amplitude; SBP = systolic blood pressure; DBP = diastolic blood pressure.

## DISCUSSION

In the current study, quantitative AFTs revealed autonomic dysfunction in 88% of CIDP patients, predominantly mild to moderate (81.8%). Cardiovagal involvement was seen in 64%, sympathetic dysfunction in 72%, and both divisions of the ANS were affected in 48%, indicating a comparable prevalence across parasympathetic and sympathetic pathways. These findings align with earlier studies. Given the demyelinating nature of CIDP, greater vagal than sympathetic dysfunction is expected, as sympathetic fibers are mostly unmyelinated except for short preganglionic segments.[22] A prospective Indian study using COMPASS-31 and CASS found autonomic

symptoms in 78% of CIDP patients and objective autonomic dysfunction in 89%, with predominance of cholinergic (86%) over adrenergic (55%) deficits.[23] Similarly, a Greek study of 17 CIDP patients reported mild autonomic symptoms in 64.7% of cases. Parasympathetic and sympathetic involvement occurred equally, and 47% had abnormal tilt table responses. SSR was absent in 42% of lower limbs and 16% of both upper and lower limbs.[24] A United States retrospective study involving 47 CIDP patients found mild autonomic dysfunction, primarily sudomotor (34%) and cardiovagal (21%), with relatively spared adrenergic function (9%) based on CASS.[25]

An Australian cohort reported mild autonomic abnormalities in 57.1% of CIDP patients, characterized by notably reduced 30:15 ratios and abnormal sweating patterns, although testing was incomplete in all patients.[26] In Taiwan, Lyu et al. found no clinical signs of autonomic dysfunction; however, subclinical involvement was present in 25% of cases via AFTs, and 50% had abnormal SSRs.[27] Yamamoto et al., described a CIDP variant with predominant sensory and autonomic involvement, suggesting immune-mediated damage to both myelin and axonal components.[28]

Our study demonstrated significantly prolonged SSR latencies and reduced amplitudes in CIDP patients, supporting sympathetic sudomotor impairment. Similar SSR abnormalities have been reported by Lyu et al., Chiang et al., and Pasangulapati et al.[27,29,23.] Absence SSR in 9 patients should be interpreted cautiously, given the dependence on afferent integrity and possible trophic skin changes in chronic neuropathy.

The mean COMPASS-31 score (8.80±7.42) in this study closely matches that of Pasangulapati et al., (9±5.16), indicating mild autonomic dysfunction. [23] As a clinical screening tool, COMPASS-31 aids in identifying patients needing quantitative ANS testing and tracking longitudinal symptom changes.[30] Control group scores in validation studies ranged from 10.2±8.9 to 11.2±9.1, whereas higher scores are typical in conditions like multiple system atrophy (mean 32.1).[30-33] Our findings showed significant investigatory correlations between autonomic abnormalities and age, disease duration, COMPASS-31 score, Hughes score, and sex. Age showed a negative correlation with palmar SSR amplitude and a positive correlation with plantar SSR latency. Larger cohort studies were intended to validate these preliminary findings. Moreover, systolic and diastolic BP drops correlated positively with age, as reported in other studies, likely due to impaired baroreflex sensitivity and reduced sympathetic compensation during orthostatic stress. [34-,36] To our knowledge, no previous studies have assessed correlations between COMPASS-31 scores and AFTs or between Hughes scores and SSR amplitude in CIDP. However, similar correlations have been found in multiple sclerosis, type 2 diabetes, and fibromyalgia.[37-39]

Discrepancies in autonomic dysfunction prevalence across studies likely stem from variations in patient selection, AFT methodologies, disease chronicity, and lack of standardized test protocols and cutoff values.

This study has several limitations. First, the sample size was relatively small, which may limit the generalizability of the findings. Second, while efforts were made to exclude confounding variables, the potential influence of comorbid conditions and medications on autonomic function could not be entirely ruled out. Third, not all autonomic domains were evaluated with equal rigor; for example, gastrointestinal and urinary autonomic functions were assessed only through patient-reported outcomes, rather than with objective testing. Finally, this was a cross-sectional study, which precluded the assessment of temporal changes in autonomic function over the disease course or treatment response.

## CONCLUSION

This study highlights that autonomic dysfunction in CIDP may involve both the sympathetic and parasympathetic systems, with comparable prevalence across divisions. Significant associations were observed between autonomic abnormalities and various factors, including age, disease duration, COMPASS 31 score, Hughes score, and sex. These findings suggest that autonomic involvement, though often mild or subclinical, is not uncommon in CIDP. Early and repeated autonomic testing, including the use of screening tools like COMPASS 31, may help in better identifying and documenting autonomic dysfunction in CIDP patients.

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