Original Article

Six-year longitudinal prospective comparative study between preoperative and postoperative heart rate variability indices in congenital craniovertebral junction anomalies

ABSTRACT

Background: Craniovertebral junction (CVJ) anomalies involve mosaic interaction of multifaceted neurovascular and bony elements. Most of them present late in the course of illness usually as acute presentations following trivial trauma. Knowing subclinical autonomic dysfunction in such anomalies when managed medically can not only indicate progression but also provide en route to early intervention for better outcomes, especially in relatively asymptomatic patients.

Materials and Methods: We conducted a 6-year longitudinal prospective study including 40 consecutive patients of CVJ anomalies with clinical, radiological, and heart rate variability (HRV) parameters and found their correlation in preoperative and follow-up period.

Results: Twenty-eight patients were male and the rest were female. The mean age was 32 years with the least age being 8 years and maximum age being 75 years old. Mean Nurick's grade and Barthel's index were 1.8 and 83.75, respectively. 38% had severe-to-moderate compression. The mean follow-up was 17.4 months. Both sympathetic and parasympathetic oscillator HRV indices were significantly affected in the preoperative period ($P \leq 0.001$) with no association with Nurick's grade or degree of compression although there was association with grade of Barthel's index. Poincare plots showed "fan," "complex," or "torpedo" patterns in 36 patients. Forty patients had both preoperative and follow-up clinical grade whereas 22 patients HRV tests in the above periods. None of the HRV indices showed significant improvement at follow-up. Nonetheless both sympathetic and parasympathetic did improve at follow-up with sympathetic tone registering better scores. Poincare plots showed improvement toward "comet" patterns in all patients.

Conclusion: HRV indices not only help in prognosticating but may also help in predicting outcomes.

Keywords: Congenital craniovertebral junction anomalies, heart rate variability, subclinical autonomic dysfunction

INTRODUCTION

Craniovertebral junction (CVJ) anomalies are a spectrum of developmental anomalies that encircle and enclose compact neurological structures of complex neurophysiology. The clinical manifestations are attributable to manifold interaction between the solid bony anomaly, physiological cerebrospinal fluid flow, and blood flow along with some intrinsic white matter tract malfunction or abnormalities secondary to chronic compression.[1] Death in such cases may be either due to cardiovascular collapse following loss of proper coordination of autonomic functions or due to lung atelectasis secondary to compromised pulmonary

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functions. Overt autonomic dysfunction as seen in acute SCI[2] is not found in CVJ anomalies and hence laboratory assessment of subclinical autonomic nervous system (ANS) is warranted. Heart rate variability (HRV) in this regard can pick subtle changes in cardiovascular (vasomotor tone) and respiratory (parasympathetic) rhythms. Indications for HRV in overt autonomic dysfunctions for classification, diagnosis, and prognosis find sumptuous share in literature in which they have proved their utility. Applying these indices to pick subtle changes during preoperative conservative management for helping decide and prioritizing surgical decompression in such anomalies finds no supporting study in literature. It is well known that craniovertebral anomalies are associated with many bedside clinical changes in autonomic functions of the body. We report 1st study in this regard for testing preclinical or subclinical autonomic involvement in relatively asymptomatic CVJ patients and demonstrate their clinical utility in such cases followed up in postoperative period for minimum 3 months. We hypothesize that improvement in clinical grade should commensurate with comparable improvement in HRV indices. The possible improvement of the autonomic functions after corrective surgery too, has not been mentioned in literature.

MATERIALS AND METHODS

Span of study – October 2015 to June 2020 – 6 years; number of cases recruited – 40; study type – observational prospective study.

Participants

Inclusion criteria – All consecutive patients presenting to NIMHANS hospital in two units of neurosurgery with history, clinical findings, and imaging suggestive of CVJ anomaly were included in the study. Exclusion criteria – Patients on medical treatment that alter autonomic functions such as sympathomimetic drugs/B blockers. Patients with co-existing chronic disease like chronic kidney disease/chronic diabetes/cardiac disease that can alter autonomic functions. Patients who had worse clinical grades and required immediate traction were excluded from the study due to technical difficulties in performing bedside autonomic function tests (AFT). After excluding patients with the above factors, 40 patients diagnosed with congenital CVJ anomalies and treated at NIMHANS were evaluated. A detailed questionnaire [Appendix 1] was used. Postoperative evaluation included only clinical examination in immediate postoperative period ranging from 1 to 7 days and delayed assessment minimum at 3 months and during subsequent follow-ups.

Heart rate variability indices included for this study

Frequency domain measurement of HRV [Figure 1]:[3-7] Heart rate signal was decomposed into frequency components and quantified in terms of their relative intensity termed as power by power spectral density (PSD). PSD analysis is one of the spectral methods, which provides the basic information as to how power (i.e., variance) distributes as a function of frequency. Lomb–Scargle periodogram was employed to provide frequency-specific information of heart rate behavior. Spectral analysis for frequency domain parameters was done using parametric spectrum (autoregressive modeling).

Total power

It represents sum of the constituent frequencies (area under PSD curve) – Sensitive to all sources of variation reflecting overall HRV. Normal values are 3466 ± 1018. It represents total modulation or sinus oscillator rhythm signifying sympathovagal balance.

High-frequency power

Frequency band ranges from 0.15 to 0.4 Hz-Parasympathetic tone. Normal values are 975 ± 150. It signifies rapid respiratory modulator activity or parasympathetic oscillator.

Low-frequency power

Frequency band ranges from 0.04 to 0.15 Hz-sympathetic and parasympathetic tone but predominantly sympathetic tone for practical purposes. Normal values are 1170 ± 200. It represents slow vasomotor modulation of heart rate or sympathetic/vasomotor oscillator.

Very low-frequency power

Frequency band ranging from 0.0033 to 0.04 Hz-VLF is more reliable in long-term HRV than short-term HRV.

High-frequency normalized units

Relative value of high-frequency (HF) in proportion to the total power (TP) minus VLF component – Parasympathetic tone in normalized units. Normal values are 29 ± 3.

Low-frequency normalized units

The relative value of low frequency (LF) in proportion to the TP minus VLF component – Sympathetic and Parasympathetic tone but predominantly sympathetic tone for practical purposes in normalized units. Normal values are 54 ± 4.

Ratio of the power of low-frequency component to power of high-frequency component

LF/HF ratio – Sympatho-vagal balance. Normal values are 1.75 ± 0.25.

Time domain parameters [Figure 2]: With time-domain measurement of HRV, either the heart rate at any point of time or the intervals between successive normal complexes were determined. In the electrocardiogram (ECG), each QRS
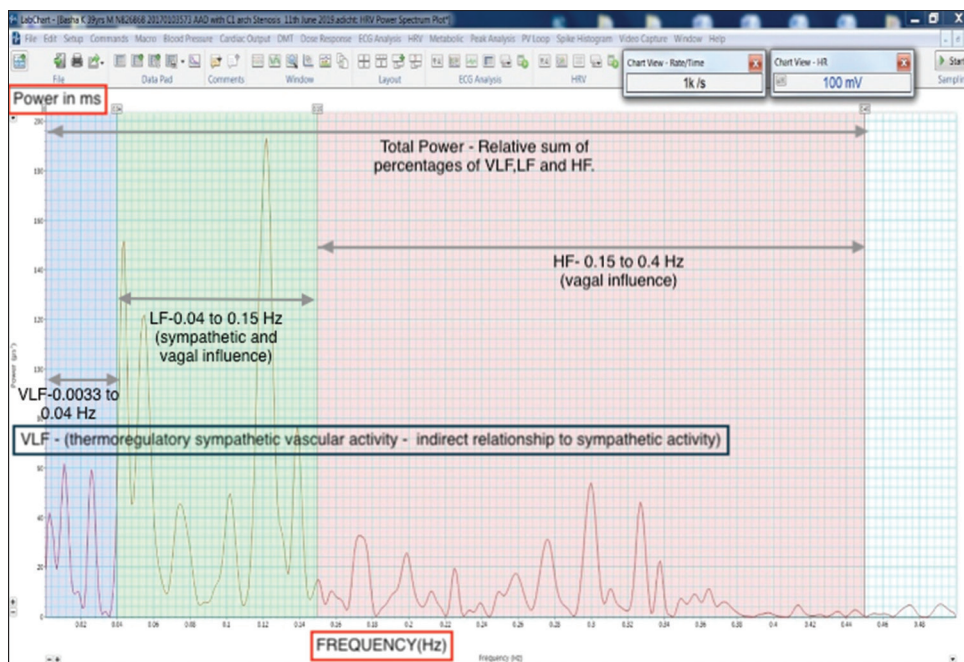
Figure 1: Frequency domain measurement of HRV of a patient in a supine resting position represented in a power spectrum plot. The frequency band boundaries at 0.04, 0.15, and 0.4 Hz are indicated in the spectral representation (very low frequency, low frequency, high frequency). This was done at our institute. HRV - Heart rate variability
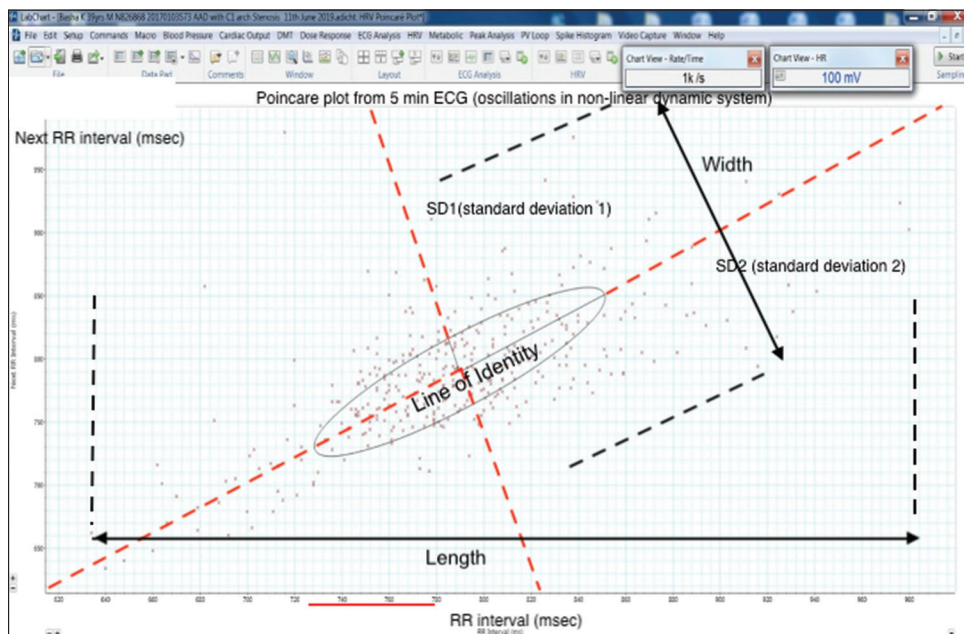

Figure 2: Representative heart rate changes in nonlinear dynamics in poincare plot (RR interval to calculate standard deviation and variance for calculation of heart rate variability.) SD1 – Minor axis of the ellipse, SD2 -major axis of the ellipse. Length (suggesting total power or total modulation) and width are used in calculating SD2 and SD1 used for analyzing RMSSD and SDNN respectively. For length and breadth widest or maximal points are taken. SD1 - Standard deviation 1; SD2 - Standard deviation 2

complex was detected and then all intervals between adjacent QRS complexes (NN/normal to normal intervals) resulting from sinus node depolarization and instantaneous heart rate were determined.[3-7]

1. Poincare plot [Figure 2]: It is a scatter plot of the current RR interval plotted against the preceding RR interval.[8]

It is a quantitative visual analysis of nonlinear dynamics plotting points of HRV over a 5-min ECG recording. Only sinus RR intervals were used whereas RR intervals due to ectopic beats and artifacts were excluded. The geometrical fitting of an ellipse to the shape of the Poincare plot were used for geometric HRV analysis.[9] Dispersion of points

perpendicular to line of identity (width) usually signifies parasympathetic influence and could be quantified into standard deviation 1 (SD1) of the successive differences of the RR intervals (SDSD) or root-mean-square differences of successive intervals (RMSSD). Points along line of identity were signaled as SD2, i.e., SD of the RR intervals (SDNN) signifying sympathovagal balance. The graphical presentation of all these points over 5 min ECG was classified into "torpedo," "fan," "complex," and "comet" variant as per the classification given by Woo *et al*.[10] The advantage of non-linear dynamics was coverage of complex cardiac autonomic phenomenon that could be missed by linear HRV analysis[11]

2. SDNN (SDANN) (ms) – SD of all NN intervals (SDANN as it was done for 5 min) – Sensitive to all sources of heart rate variation. Normal values are 141 ± 40.

3. RMSSD (root-mean-square differences of successive intervals in millisecond). RMSSD is a measure of parasympathetic activity/vagal tone. Normal values are 27 ± 12.

Statistical methods

Descriptive statistical analyses were carried out in the present study. Results on continuous measurements were presented in mean SD and results of categorical measurements were presented in number (%). Significance was assessed at 5% level and 95% confidence intervals. Mean, median, and interquartile range all were used wherever appropriate to find association between variables. Nonparametric tests (Kruskal–Wallis Test and Wilcoxon-Mann–Whitney *U* test) were used to make group comparisons and in data that were not normally distributed. Chi-squared test was used to explore the association between various clinical and autonomic subgroups versus age and versus gender. Parametric tests *t*-test and Paired sample *t*-test were used to make group comparisons and difference at the two-time points in normally distributed data. Paired Wilcoxon test was used to explore the difference at two-time points. Stuart-Maxwell test was used to assess the change in Nurick's Grade between two time points. Fisher's exact test (Cramer's V and Bias Corrected Cramer's V) was used to explore the association in parametric qualitative data whereas Kruskal–Wallis test (Kendall's Tau) was used to compare nonparametric data. Statistical software namely SPSS 26.0 (IBM Corp. Released 2019, Armonk, NY: IBM Corp), Stata 15.0 (StataCorp LLC, 4905, Texas , USA), MedCalc 19.1.6 (MedCalc Software Ltd, Acacialaan, Ostend, Belgium), were used for the analysis of this data.

RESULTS

A total of 70 patients were screened for the study. Out of which 20 patients had definite history of trauma, hence were excluded. Out of 50 patients, 10 were not included as they were lost to follow-up. Forty patients with congenital CVJ anomalies and relatively asymptomatic or mildly symptomatic were selected for the study. Only 22 patients were available for follow-up in person. Rest were present for follow only on telephone. Hence, preoperative autonomic functions were compared with 40 age-matched normal controls; whereas pre- and post-operative autonomic functions were compared for 22 patients available for follow-up. Clinical comparison between preoperative and postoperative follow-ups was done for 40 patients either in person or telephonically in consultation with local physician.

Demographic characteristics of patients

Twenty-eight patients were male. The mean age for the group was 32 years with least age being 8 years and maximum age being 75 years old. Only three patients were below the age of 16 years (2 were 11 years old and one was 8 years old). 45% of the patients had a more sedentary lifestyle whereas 30% were manual laborers; clinical profile of the patients [Table 1]: neck pain, weakness of limbs causing difficulty walking were the most common complaints. Ninety-five percent patients had increased deep tendon reflexes. Positive signs on examination [Table 1]: short neck, increased tone, increased DTRs, and up going plantars were the most common clinical findings

Nurick's grade and Barthel's index

Clinical improvement was seen in almost 98% (39 patients) at follow-up whereas only one patient showed deterioration [Table 1 and Figure 3]. At follow-up of 3–6 months, 75% (30 patients) had improved in both Nurick's and Barthel's Index [Table 1 and Figure 3]. 17.5% (7 patients) showed immediate improvement in the postoperative period. As shown in Table 2, degree of Nurick's grade had no relation with the severity of ANS or HRV dysfunction. In fact, apart from Grade 0, all were significantly affected. In addition, follow-up improvement was noted maximum in Grade 1 and Grade 2 as compared to Grade 3 and Grade 4 [Tables 2 and 3]. HRV – There was a significant difference between the five groups in terms of low frequency (Normalized Units) (χ^2 = 11.873, *P* = 0.018), with the median values being highest in the Nurick's Grade (preoperative): Grade 1 group (Kendall's Tau = 0.28) [Table 2]. There was a significant difference in terms of HF (Normalized Units) (χ^2 = 14.744, *P* = 0.005), with the median values being highest in Grade 0 group (Kendall's Tau = 0.35) [Table 2]. There was a significant difference between the five groups in terms of LF/HF Ratio (χ^2 = 12.027, *P* = 0.017), with the median values being highest in the Nurick's Grade (preoperative): Grade 1 group (Kendall's Tau = 0.28) [Table 2]. In postoperative period, no association was found between Nurick's grade improvement in any of the HRV functions.

Table 1: Summary of preoperative assessment and follow-up

Preoperative assessment	Mean±SD||median (IQR)||minimum-maximum||frequency (%)
Neck pain (yes)	31 (77.5)
Neck tilt (yes)	8 (20.0)
Neck movement restriction (yes)	24 (60.0)
Transient attacks (yes)	12 (30.0)
Progressive weakness (yes)	24 (60.0)
Urinary sphincter disturbances (yes)	9 (22.5)
Short neck (yes)	32 (80.0)
Low hairline (yes)	26 (65.0)
Respiratory reserve	
Good	21 (52.5)
Affected yet adequate	16 (40.0)
Poor	3 (7.5)
Ataxia (yes)	16 (40.0)
Paresis	
None	10 (25.0)
Biparesis	3 (7.5)
Hemiparesis	5 (12.5)
Quadriparesis	22 (55.0)
Increased tone	
None	11 (27.5)
Unilateral	3 (7.5)
Lower limbs	5 (12.5)
All limbs	21 (52.5)
Plantar reflex	
Down going	3 (7.5)
Upgoing	35 (87.5)
Not elicitable	2 (5.0)
Nurick grade (baseline)	
Grade 0	1 (2.5)
Grade 1	18 (45.0)
Grade 2	12 (30.0)
Grade 3	5 (12.5)
Grade 4	4 (10.0)
Barthels index (baseline)	83.75±16.00||87.50 (75.00-100.00)||55.00-100.00
Degree of compression	
None	1 (2.5)
Mild	1 (2.5)
Moderate	8 (20.0)
Severe	30 (75.0)
Traction (yes)	11 (27.5)
Clinical improvement after cervical traction (yes)	2 (5.0)
Reduction after traction (yes)	9 (22.5)
Follow-up	**Mean±SD||median (IQR)||minimum-maximum||frequency (%)**
Duration of follow-up (months)	
3-6	9 (22.5)
6-12	15 (37.5)
12-24	3 (7.5)
>24	13 (32.5)
Clinical status (follow-up)	
Improved	30 (75.0)
Status Quo	5 (12.5)
Deteriorated	5 (12.5)
Urinary sphincter disturbances (follow-up)	
Improved	5 (12.5)

Contd...

Table 1: Contd...

Follow-up	Mean±SD||median (IQR)||minimum-maximum||frequency (%)
Status Quo	33 (82.5)
Deteriorated	2 (5.0)
Spasticity (follow-up)	
Improved	23 (57.5)
Status Quo	14 (35.0)
Deteriorated	3 (7.5)
Respiratory reserve (follow-up)	
Improved	9 (22.5)
Status quo	29 (72.5)
Deteriorated	2 (5.0)
Nurick grade (follow-up)	
Grade 0	15 (37.5)
Grade 1	12 (30.0)
Grade 2	8 (20.0)
Grade 3	4 (10.0)
Grade 5	1 (2.5)
Barthels index (follow-up)	89.88±13.23||95.00 (83.75-100.00)||55.00-100.00

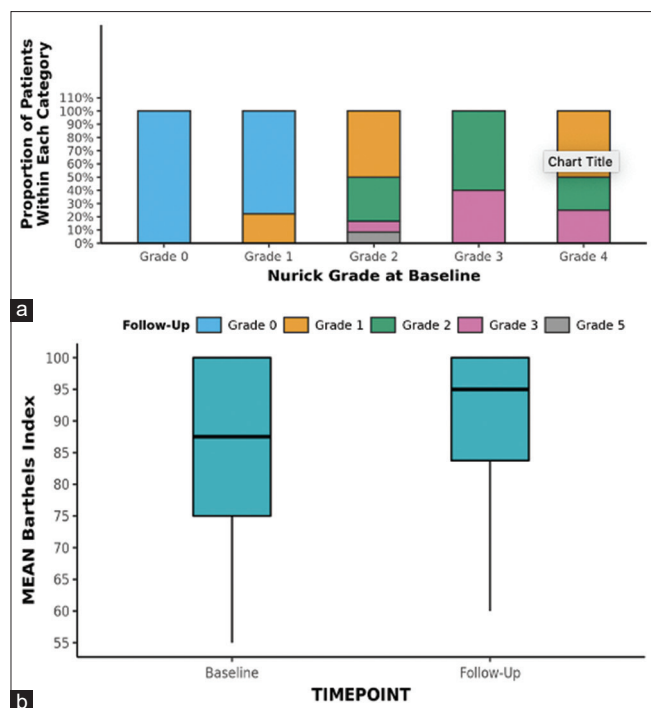DTR: Deep tendon reflexes, SD: Standard deviation, IQR: Interquartile range

Figure 3: The above box and whisker plot shows change in Nurick's grade (a) and Barthel's index (b) over time

Radiological features

All the patients in our group had atlantoaxial instability. Thirty-five patients had both anterior and posterior compression, four patients had nonreducible and 36 patients had reducible AAD. C1 assimilation and basilar invagination were most common CVJ anomalies associated with AAD.

Degree of compression

Most of the patients had severe decompression (80%) as per the classification given by Muhle *et al*.[12] and Kang *et al*.[13]

into none-normal, mild-partial obliteration of the anterior or posterior subarachnoid space or more than 50% obliteration, Moderate-central canal stenosis with cord deformity but without spinal cord signal change, severe-cervical cord compression or displacement or presence of spinal cord signal change near the compressed level on T2-weighted images. There was nil significant association between Nurick's grade and degree of compression as Grade 1 had maximum cases of severe compression [Table 3]. Nonetheless, there was a significant difference between the four groups in terms of Barthel's Index ($\chi^2 = 12.098$, $P = 0.007$), with mean values being highest in the degree of compression: None and Mild group (Kendall's Tau = 0.48). Nil significant association was found between any of the HRV indices and degree of compression. The degree of compression did not play a significant role in ANS signifying an early development of subclinical ANS dysfunction irrespective of grade or stage (Barthel's index) [Table 3]. Postoperative dislocation and neurological deterioration on follow-up: 90% of the patients had improved both radiologically and clinically on follow-up of minimum 3 months whereas 5 had deteriorated out of which 3 underwent Re-surgery and 2 refused for re-do surgery [Table 1]. Follow-up in months: All 40 patients had follow with mean follow-up being 17.4 months with 40% of patients having follow-up of more than 12 months and 32% having more than 2 years. One patient expired at a follow-up of more than 24 months due to cardiac failure.

Heart rate variability indices

For better comparison and understanding 4 peculiar representative HRV tachograms are analyzed here named (a, a', a", a''' a"" for 1st patient; b, b' b", b''', b"" for 2nd patient) and so on.

Table 2: Association of Nurick grade with clinical and autonomic heart rate variability indices

Parameters	Nurick Grade (baseline)					P
	Grade 0 (n=1)	Grade 1 (n=18)	Grade 2 (n=12)	Grade 3 (n=5)	Grade 4 (n=4)	
Age (years)	15.00±0	38.72±15.94	37.92±19.12	22.00±9.14	34.75±14.50	0.163[a]
Gender						
Male	1 (100.0)	11 (61.1)	7 (58.3)	5 (100.0)	4 (100.0)	0.255[b]
Female	0	7 (38.9)	5 (41.7)	0	0	
Barthels Index (baseline)***	100.00±0	95.83±6.91	77.92±13.05	74.00±7.42	55.00±0.00	<0.001[a]
Degree of compression						
None	1 (100.0)	0	0	0	0	0.189[b]
Mild	0	1 (5.6)	0	0	0	
Moderate	0	5 (27.8)	3 (25.0)	0	0	
Severe	0	12 (66.7)	9 (75.0)	5 (100.0)	4 (100.0)	
Barthels index (follow-up)***	100.00±0	99.17±2.57	84.58±15.14	75.00±9.35	80.00±10.80	<0.001[a]
SDNN (ms) (baseline)	104.19±0	34.93±14.68	42.17±26.71	52.45±22.16	42.94±40.64	0.280[a]
RMSSD (ms) (baseline)	120.81±0	22.43±10.69	39.39±34.07	57.05±31.27	43.04±53.56	0.153[a]
Total power (ms^2) (baseline)	10083.62±0	1250.62±994.05	2120.44±2843.70	2645.15±2047.64	2426.55±3922.92	0.241[a]
LF power (ms^2) (baseline)	1825.78±0	336.06±277.78	406.03±571.85	396.73±236.73	615.75±994.85	0.399[a]
HF power (ms^2) (baseline)	5048.01±0	268.16±223.02	903.05±1246.82	1368.20±1050.75	1242.30±2210.98	0.125[a]
LF (normalized units) (baseline)***	23.33±0	53.87±18.05	39.00±16.46	25.53±11.92	43.30±14.40	0.018[a]
HF (normalized units) (baseline)***	64.51±0	37.12±14.58	54.06±15.45	66.00±11.24	47.51±22.86	0.005[a]
LF/HF ratio (baseline)***	0.36±0	1.96±1.76	0.88±0.69	0.41±0.25	1.65±2.02	0.017[a]
SDNN (ms) (postoperative)	12.56±0	40.89±25.84	29.96±9.69	55.24±0.34	52.02±44.93	0.265[a]
RMSSD (ms) (postoperative)	6.55±0	35.53±31.14	24.72±7.86	58.97±0.60	64.09±54.08	0.220[a]
Total power (ms^2) (postoperative)	169.07±0	2168.27±3058.66	1033.59±643.74	3069.86±1189.15	3263.83±4379.51	0.348[a]
LF power (ms^2) (postoperative)	46.91±0	657.35±1116.08	288.83±185.00	927.87±392.97	780.29±1149.24	0.298[a]
HF power (ms^2) (postoperative)	17.10±0	856.14±1594.36	250.58±195.39	1333.40±345.91	1627.42±2536.45	0.299[a]
LF (normalized units) (postoperative)	65.80±0	55.56±17.03	49.34±17.05	39.83±0.24	26.65±14.63	0.130[a]
HF (normalized units) (postoperative)	24.01±0	44.13±15.88	41.88±20.17	42.91±10.03	43.05±23.14	0.850[a]
LF/HF ratio (postoperative)	2.74±0	1.61±1.07	1.78±1.73	0.80±0.42	0.65±0.16	0.230[a]

***Significant at P<0.05, [a]Kruskal-Wallis test, [b]Fisher's exact test. SDNN: Standard deviation of the RR intervals, RMSSD: Root-mean-square differences of successive intervals, LF: Low frequency, HF: High frequency

Time domain components of heart rate variability
Poincare plot
We used a graphical Poincare plot to label patterns. Thirty-six patients in the preoperative period had disturbed pattern of either torpedo, fan, or complex [Figure 4]. In the follow-up period, all patients had change in patterns toward comet types reflecting return of sympathovagal balance [Figure 5]. We did not conduct tests of significance for this change due to inter-observer variability for graphical representative labeling. SD1, SD2, or SD1/SD2 was not included in the study.

Standard deviation of the RR intervals
Sensitive to all sources of heart rate variation (sympathovagal).[3,4] Preoperative SDNN (SDANN) was significantly affected in all (100%) patients [Table 4]. This indicates 100% disturbance of sympathovagal balance. There was a significant decrease in mean SDNN in preoperative values as compared to control values (W = 537.000, P = 0.011, Point-Biserial Correlation = 0.23). SDNN improved in all patients in postoperative follow-up at 3 months [Table 4] but was still significantly lower than control values (W = 280.000, P = 0.019, Point-Biserial Correlation = 0.23).

RMSSD
Most sensitive to parasympathetic activity/vagal tone.[3,4] Median RMSSD was significantly decreased in preoperative period as compared to controls (W = 483.000, P = 0.002, Point-Biserial Correlation = 0.21). Overall 36 patients showed abnormal variance, with 21 patients showing a significant decrease, which again highlights abnormal parasympathetic functioning. Mean and Median RMSSD improved in follow-up period as compared with comet patterns in most of the cases. There was nil significant difference between the cases versus controls in terms of RMSSD (ms^2) (postoperative)([12] W = 313.000, P = 0.063, Point-Biserial Correlation = 0.17). This signifies improvement in parasympathetic autonomic functions.

Frequency domain components of heart rate variability
Normalized low frequency and low frequency in ms^2
Normalized LF power and LF in ms^2 gives the assessment of both parasympathetic and sympathetic (mainly) activities.[3,4] For all practical purposes low frequency mainly signifies sympathetic tone. Median low frequency in ms^2 was significantly decreased as compared to controls [Figure 6].

Table 3: Association of degree of compression with clinical and autonomic heart rate variability indices

Parameters	Degree of compression				P
	None (n=1)	Mild (partial obliteration of the anterior or posterior subarachnoid space or >50% obliteration) (n=1)	Moderate (central canal stenosis with cord deformity but without spinal cord signal change) (n=8)	Severe (cervical cord compression or displacement or presence of spinal cord signal change near the compressed level on T2-weighted images) (n=30)	
Age (years)	15.00±0	11.00±0	36.50±14.00	36.60±17.08	0.203[a]
Gender					
Male	1 (100.0)	0	5 (62.5)	22 (73.3)	0.411[b]
Female	0	1 (100.0)	3 (37.5)	8 (26.7)	
Barthels Index (baseline)***	100.00±0	100.00±0	96.88±5.94	79.17±15.76	0.007[a]
Barthels Index (follow-up)	100.00±0	100.00±0	98.12±3.72	87.00±14.06	0.075[a]
SDNN (ms) (baseline)	104.19±0	17.00±0	38.86±20.09	41.36±23.74	0.233[a]
RMSSD (ms) (baseline)	120.81±0	12.06±0	30.67±22.12	35.88±31.55	0.300[a]
Total power (ms^2) (baseline)	10083.62±0	169.00±0	1872.53±1665.07	1857.97±2336.27	0.175[a]
LF power (ms^2) (baseline)	1825.78±0	41.00±0	507.15±401.10	375.66±491.23	0.100[a]
HF power (ms^2) (baseline)	5048.01±0	29.00±0	671.48±1114.84	735.76±1100.04	0.170[a]
LF (normalized units) (baseline)	23.33±0	45.00±0	50.68±20.46	42.93±18.71	0.487[a]
HF (normalized units) (baseline)	64.51±0	29.00±0	39.56±19.51	49.72±17.44	0.213[a]
LF/HF ratio (baseline)	0.36±0	1.42±0	1.80±2.11	1.29±1.33	0.474[a]
SDNN (ms) (postoperative)	12.56±0	20.64±0	51.80±32.79	39.83±23.30	0.252[a]
RMSSD (ms) (postoperative)	6.55±0	6.87±0	47.73±37.82	39.33±30.72	0.200[a]
Total power (ms^2) (postoperative)	169.07±0	533.65±0	3454.67±4357.32	1885.18±2174.70	0.278[a]
LF power (ms^2) (postoperative)	46.91±0	92.60±0	1117.60±1662.88	503.96±569.71	0.311[a]
HF power (ms^2) (postoperative)	17.10±0	22.73±0	1389.30±2324.84	790.39±1223.87	0.180[a]
LF (normalized units) (postoperative)	65.80±0	82.76±0	50.83±11.46	45.79±18.59	0.311[a]
HF (normalized units) (postoperative)	24.01±0	20.31±0	47.53±12.99	43.60±16.66	0.290[a]
LF/HF ratio (postoperative)	2.74±0	4.07±0	1.27±0.79	1.30±1.05	0.256[a]

***Significant at *P*<0.05, [a]Kruskal-Wallis Test, [b]Fisher's exact test. SDNN: Standard deviation of the RR intervals, RMSSD: Root-mean-square differences of successive intervals, LF: Low frequency, HF: High frequency

LF in ms^2 was significantly low (W = 452.000, *P* = 0.001, Point-Biserial Correlation = 0.27). Twenty-seven patients had a significant decrease. Totally, 35 patients were affected. Increased in eight patients suggesting 35 patients to be totally affected [Figure 6]. This feature suggests gross affection of sympathetic parameters. There was a definite improvement in both modalities in follow-up period, but the difference was not significant [Table 4]. The improvement suggests partial return of sympathetic tone.

Normalized high frequency and high frequency in ms^2

Normalized HF power and HF in ms^2 gives the assessment of vagal predominance (parasympathetic tone).[3,4] Median HF in ms^2 was significantly increased as compared to controls [Figure 6] (W = 495.000, *P* = 0.003) strength of association (Point-Biserial Correlation) = 0.17 (Small Effect Size). It was significantly affected in 17 patients whereas 36 patients had decreased parasympathetic tone. Even though HF ms^2 did improve in follow-up period it remained significantly lower than controls (W = 274.000, *P* = 0.015, Point-Biserial Correlation = 0.14). This reduction in follow-up period suggests partial return of parasympathetic autonomic functions [Figure 7].

Low-frequency/high-frequency ratio

LF/HF ratio gives assessment of Sympatho-vagal activity.[3,4] Its values are usually averaged between 0.5 and 1.5. If the values are more toward 0.5 it suggests parasympathetic predominance whereas sympathetic dominance if it is at other end of the spectrum. The median LF/HF ratio was not significantly affected in the preoperative period as compared to controls [Table 4] (W = 880.000, *P* = 0.444). Nonetheless, it increased significantly in nine patients (suggesting parasympathetic involvement) whereas in eight patients (suggesting sympathetic involvement), it decreased significantly. In other patients, the LF/HF ratio was more or less in normal spectrum. The nine patients with parasympathetic involvement showed much lesser improvement as compared to patients in the sympathetic arm persisting even after 3 months of surgery as compared to normal controls [Figure 8]. This suggests that the sympathetic pathway recovered faster as compared to parasympathetic pathway which did show early signs of recovery.

Total power

TP signifies HRV. It is sensitive to all sources of variation reflecting overall HRV.[3,4] Median TP showed a significant

Table 4: Comparison between controls, preoperative and postoperative values of cases for various heart rate variability indices

HRV	Control values, mean±SD	Median (IQR)	Minimum-maximum	Patients (preoperative n=40), mean±SD	Median (IQR)
SDNN (ms)	54.04±28.36	48.43 (41.11-60.51)	11.56-177.48	41.82±24.68	34.65 (24.57-52.93)
RMSSD (ms)	52.27±42.66	26.4 (14.47-38.51)	8.83-250.94	36.37±32.14	26.40 (14.47-38.51)
Total power (ms²)	3845.58±4947.28	2294.72 (1647.03-4195.9)	120.6-29,322.02	2024.30±2517.14	1067.53 (617.80-2336.24)
LF power (ms²)	776.20±736.70	530.05 (351.82-812.55)	17.98-3240.37	429.85±515.40	272.30 (151.82-396.13)
HF power (ms²)	1738.45±3726.07	693.05 (387.04-1432.58)	29.06-22,631.01	813.04±1267.76	286.62 (96.44-532.36)
LF (normalized units)	39.81±14.36	40.75 (28.54-52.06)	11.17-62.4	44.04±18.88	41.87 (32.24-53.01)
HF (normalized units)	51.55±15.16	51.25 (41.12-63.16)	4.46-80.09	47.54±18.09	51.33 (38.52-59.49)
LF/HF ratio	0.87±0.50	0.82 (0.48-1.2)	0.14-1.83	1.37±1.48	0.78 (0.56-1.40)

HRV	Minimum-maximum	P	Postoperative mean follow up at 3 months (n=22) (P value as compared to controls), mean±SD	Median (IQR)	Minimum-maximum	P value for statistical improvement
SDNN (ms)	11.2-104.3	0.011[a]	40.44±25.56 (P=0.019[a])	34.86 (21.04-48.33)	12.6-105.7	0.732[a]
RMSSD (ms)	5.7-123.1	0.002[a]	38.27±31.92 (P=0.063[a])	28.62 (17.08-54.29)	6.6-123.1	0.813[a]
Total power (ms²)	102.1-10,083.6	0.001[a]	2102.45±2744.74 (P=0.008[a])	1099.21 (495.91-2150.49)	169.1-11,136.1	0.240[a]
LF power (ms²)	16.4-2107.3	0.001[a]	603.95±916.66 (P=0.048[a])	273.90 (95.95-637.50)	46.9-4070.1	0.191[a]
HF power (ms²)	7.9-5048.0	0.003[a]	856.46±1471.54 (P=0.015[a])	217.52 (77.62-729.89)	17.1-5534.0	0.813[a]
LF (normalized units)	10.9-85.9	0.263[b]	49.52±18.19 (P=0.037[b])	51.21 (39.72-63.49)	11.0-83.3	0.197[b]
HF (normalized units)	12.8-81.1	0.285[b]	42.55±16.24 (P=0.038[b])	45.55 (27.11-54.58)	15.4-68.0	0.216[b]
LF/HF ratio	0.1-6.7	0.444[a]	1.49±1.13 (P=0.030[a])	1.11 (0.70-1.94)	0.4-4.3	0.295[a]

***Significant at $P<0.05$, [a]Wilcoxon-Mann-Whitney *U*-test, [b]*t*-test. HRV: Heart rate variability, SDNN: Standard deviation of the RR intervals, RMSSD: Root-mean-square differences of successive intervals, LF: Low frequency, HF: High frequency, SD: Standard deviation, IQR: Interquartile range
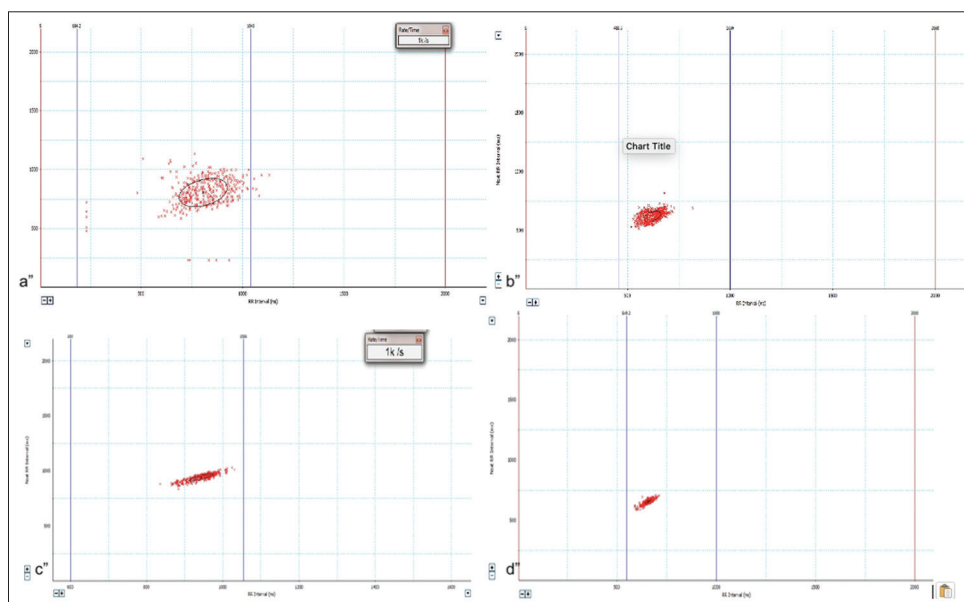

Figure 4: Represents Poincare plotting of HRV frequencies of illustrative patients. (a") Denotes gross sympathovagal disturbance with frequency plotting widely distributed between 500 ms and 1000 ms of RR interval representing "fan" or "complex" pattern. (b") shows a narrow spectrum of frequencies between 900 ms to 1000 ms with wider SD1 signifying parasympathetic predominance "torpedo pattern," (c" and d") show "torpedo" patterns with elongated SD2 suggestive of sympathetic predominance. HRV - Heart rate variability

decrease in all patients as compared to controls (W = 452.000, $P = 0.001$, Point-Biserial Correlation = 0.27). TP is a sum of various frequencies and hence can little predict any particular modality involvement, although it does suggest overall autonomic functionality. TP showed some improvement in postoperative period [Figure 8], ($P = 0.240$) but remained significantly lower than control values (W = 258.000,

$P = 0.008$, Point-Biserial Correlation = 0.19). This signifies persistent sympathovagal imbalance.

Summary of heart rate variability analysis
Although both sympathetic (LF) and parasympathetic (SDNN, RMSSD, HF in ms², LF in ms²) autonomic functions were significantly affected in the preoperative period, none
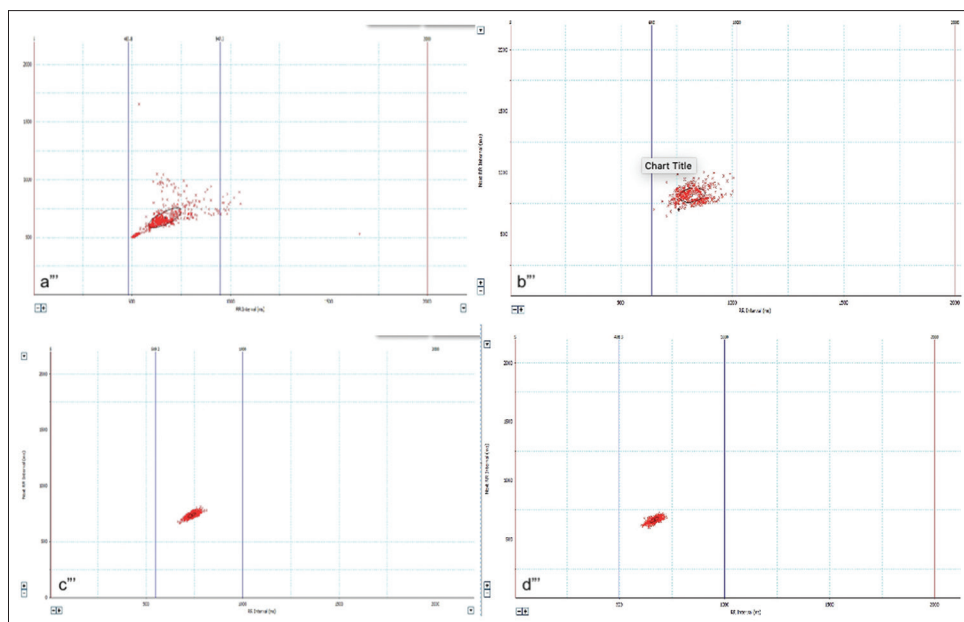
Figure 5: (a''' and c''') Illustrate significant improvement to "comet pattern" with a tail and wide head signifying return of sympathovagal balance (tail – increase in heart rate with decrease in RR interval whereas head signifies the opposite), (b''') – "fan" pattern suggesting persistent sympathovagal imbalance whereas (d''') shows some improvement as compared to preoperative Poincare progressing toward "comet" pattern suggesting a return of parasympathetic tone
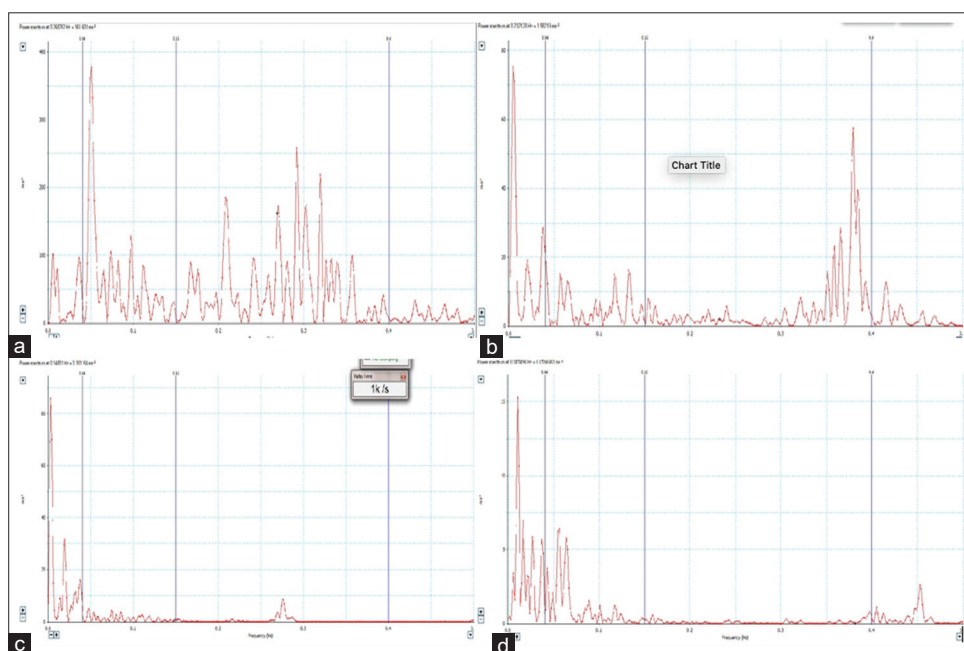

Figure 6: PSD of various patients (a) there is grossly disturbed vasomotor tone (LF) and respiratory oscillations (HF) both showing increased rate and variability, (b) Both HF and LF oscillations have decreased although sympathetic oscillations have decreased more suggesting parasympathetic overdrive, (c) grossly reduced total power showing decreasing in both LF and HF, whereas (d) shows purely affection of parasympathetic tone with a significant decrease in HF. PSD - Power spectral density; LF - Low frequency; HF - High frequency

showed significant improvement in follow-up period [Table 4]. Sympathetic functions showed better improvement as compared to vagal tone. Overall sympatho-parasympathetic balance (LF/HF, TP) showed improvement but was not significant [Figure 8]. This may be explained due to possible concurrent subclinical respiratory dysfunction in these patients.

DISCUSSION

The respiratory and cardiovascular dysfunctions are considered to be the result of either repeated trauma to CVJ or by the pincer action on the cord by the bony anomalies.[14,15] Autonomic testing hold prognostic, localization, verification, staging, and monitoring of treatment value in disorders of
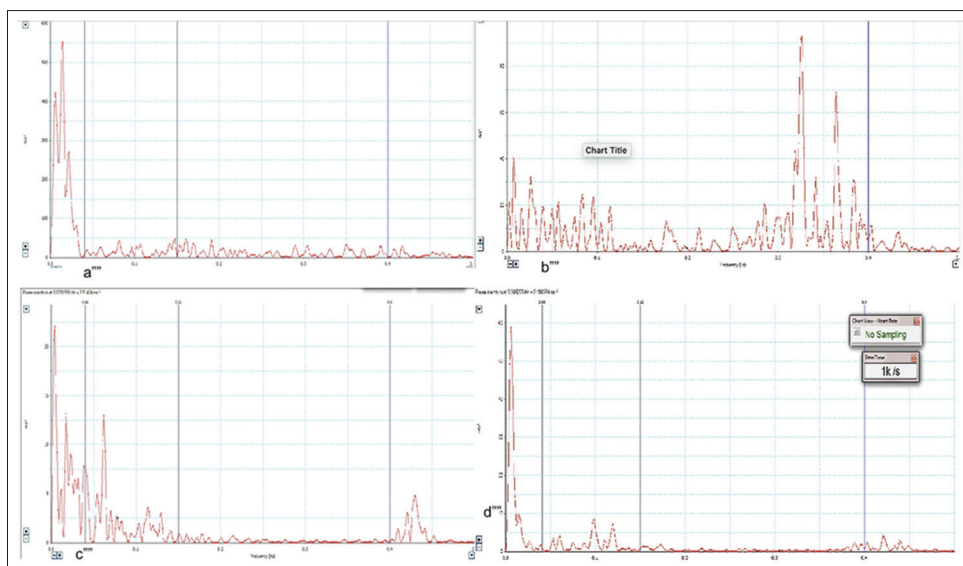
Figure 7: (a"") – Reduction in total power as compared to preoperative values although overall LF/HF ratio remains the same, (b"") – return of sympathetic oscillations with rise in vasomotor tone (LF), (c"") – although there is rise in vasomotor tone, respiratory oscillations (parasympathetic tone) remains affected, (d"") the total power has improved along with LF/HF ratio suggesting return of sympathovagal balance. LF - Low frequency; HF - High frequency
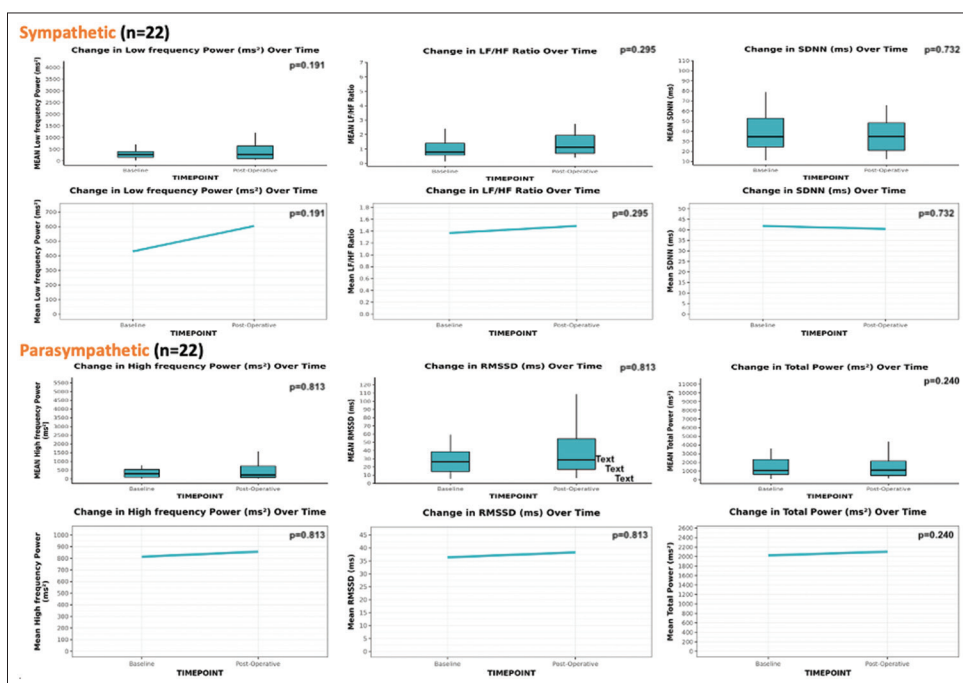

Figure 8: Box and Whisker plots and Trend over time period at preoperative and 3-month follow-up showing better sympathetic tonal improvement as compared to parasympathetic tone

autonomic system and the predominant component involved, whether it is sympathetic, parasympathetic, or both.[4,16] CVJ anomalies are known to cause compression at the level of cervicomedullary junction both clinical and radiologically. However, many patients follow a relatively asymptomatic and chronic clinical picture and usually present later in the course of the disease.[17,18] Knowing amount ANS dysfunction in the preoperative period may help in predicting the prognosis in the long run. Furthermore, patients having progressive

ANS dysfunction even though subclinical may warrant early intervention for better long-term outcomes. It is well known that recovery in neurological grade of the patient is directly correlated with the preoperative clinical status, hence with improvement in the above, should commensurate with improvement in AFT.[5] The autonomic outflow has been studied in cervical spondylotic myelopathy before.[5] However, there are no studies of detailed evaluation of autonomic functions in CVJ anomalies. Even though Rath *et al*.[19] has

described a score for respiratory dysfunction in patients with cervical cord compromise in Congenital CVJ anomalies, there is no guideline on documentation of autonomic dysfunction in CVJ anomalies.

Pathophysiology

We propose a triple-hit hypothesis in this regard (1) relative deficient myelination and small diameter fibers, (2) anatomic vulnerability of these tracts, and (3) compromised blood supply due to chronic compression and watershed area. Autonomic tracts coming from hypothalamic parvocellular paraventricular neurons and other extra-hypothalamic neurons have intermediate myelination or belong to Type b and c myelination categories and are more prone to pressure effects as compared to thickly myelinated sensory and motor tracts. This parvocellular pathway mainly relay through dorsal longitudinal fasciculus and medial forebrain bundle. So also their position in the intermediate column of the white matter distribution places them more at risk. The CVJ bony anomaly is like a triple-hit due to compromised workspace and clustering of tracts leading to chronic compressive trauma over the cord. To compensate for the decreased space and chronic venous congestion, blood supply to this region is compromised. This can lead to chronic ischemia and a vicious cycle of blood compromise-ischemia events if superimposed with progressive instability. In this fight of space, some tracts have to give way, and Type b and c myelinated fibers due to their thin and deficient myelination and relatively less blood supply are the first to suffer. The relatively lesser blood supply is both due to their deficient myelination because they are smaller to intermediate fibers and also because they lie in the watershed area of the anterior and posterior spinal artery vascular supply which may add to the deleterious effect on these tracts and thus a delayed recovery or progressive atrophy. This cluttered space is a potential harbinger of sudden decompensation even if minor trauma ensues in view of highly compromised neurovascular relations. To support this hypothesis at a microvascular level, the concept of neurovascular unit by Muoio *et al.*[20] could help in understanding these recovery mechanisms to an extent. The extent of this balance between the destructive and reparative process actually manifests in the amount of subtle or clinical dysfunction that occurs due to chronic compression. Most of this reparative prowess is usually adept along with their medial placement making them inherently resistant. Most of this dysfunction usually remains subclinical for a long period unless surfaced with minor complaints such as neck pain, neck movement's restriction, torticollis, or minimal weakness demonstrated in the form of myelopathy symptoms.

In the searched English literature of PubMed, Google Scholar, Scopus, Crossref, and Publons database, we could not find any material regarding AFT in CVJ anomalies. Although there is a paper on AFT in compressive myelopathy from the same institute as authors by Srihari *et al.*[5] In order to find whether compressive myelopathy at two levels of cervical spine behave any differently, we have compared our study with the above.

Table 5 analysis and showcases differences and similarities between the two studies. The above comparison denotes the following: Our mean age group is younger than above study and other western literature. Most patients in our study had a better Nurick's grade of 1.8 than other study of 2.8 signifying that clinically they had fewer complaints. The postoperative improvement in our group in terms of Nurick's grade is better and significant than the other study signifying a better clinical outcome in patients with better Nurick's grade at presentation. In addition, it has been proven by same authors that conventional autonomic functions significantly affected in relatively asymptomatic patients with congenital CVJ anomalies.[21] Srihari *et al.*[5] showed nil significant preoperative involvement in HRV for compressive myelopathy patients whereas our study for CVJ anomalies demonstrated that the majority of HRV indices were significantly affected. Furthermore, their study showed a significant improvement in parasympathetic tone whereas in our study neither sympathetic nor parasympathetic tone improved significantly. Nonetheless, LF and SDNN had better improvement as compared to HRV indices signifying parasympathetic tone. We assume that there is an associated subclinical respiratory dysfunction which may alter the dynamics of respiratory effect on this parasympathetic tone leading to much slower and delayed improvement. Studies by Nomura *et al.*,[22,23] Reddy *et al.*[24] and Rosomoff[14] have shown that multiple respiratory functions are affected in the chronic higher cervical spine and CVJ compressive pathologies and are largely subclinical. Even though these abnormalities did improve with time, they had a slow tardive course. This may well explain the delay in parasympathetic tonal recovery which has a major respiratory influence. This may suggest that an AFT at a later follow-up or consecutive AFTs at every follow-up demonstrate progressive recoup of sympathovagal balance. We speculate that dysfunction of both respiratory and autonomic dysfunction is compromised in chronic compression. However, it remains subclinical due to either compensation because of its chronic course or its inherent resistance or due to its anatomical arrangement of fibers which is more medial than other major motor and sensory fibers. This theory is also supported by Toyoda *et al.*[25]

Table 5: Two comparative studies of autonomic dysfunction in cervical compressive myelopathy and craniovertebral junction anomalies

Studies	Nurick's grade (mean)		RMMSD (ms) (Mean)		SDNN (ms) (mean)		Total power (ms²) (mean)		Normalized (nu) LF (mean)		Normalized HF (nu) (Mean)		LF/HF ratio		Lf in ms² (mean)		HF in ms² (mean)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Srihari *et al*.[5]	2.8	2.7	57	28	-	-	8025	1934	47	47	43	57	-	-	-	-	-	-
Current study	1.8	1.1	36.37*	38.27*	42*	40.5*	2024*	2102*	44*	49.5*	47.5*	42.5*	1.37*	1.49*	430*	604*	813*	856*

*Significant at *P*<0.05 as compared to control values. SDNN: Standard deviation of the RR intervals, RMSSD: Root-mean-square differences of successive intervals, LF: Low frequency, HF: High frequency

CVJ anatomy and biomechanics are different as compared to cervical spine. Considering this anatomico-physio-biomechanical peculiarity, there is a dilemma regarding the possible interindividual feasibility of neurophysiological studies and standardization of the same. Hence, an integrated approach comprising medical history, the knowledge of the natural course of the disease, neurological examination, and neuroradiology along with electrophysiological tests can provide firm and convincing timeline for intervention and also prioritize the patients requiring early versus not-so-early surgery and the patients that may require ventilator support in immediate postoperative period based on pulmonary and AFT.

In subacute and chronic myelopathies associated with CVJ anomalies, overt autonomic and pulmonary dysfunction is not seen hence laboratory assessment of ANS and pulmonary functions is mandatory.

CONCLUSION

CVJ anomalies definitely affect all HRV indices compared to age-matched controls. Most of this dysfunction persists even after surgery. We document subclinical autonomic impairment which significantly affects both sympathetic and parasympathetic autonomic functions in patients with CVJ anomalies. Sympathetic and parasympathetic functions showed only partial improvement which may be due to earlier follow-ups. Hence, consecutive follow-ups at 3–6 monthly intervals with HRV tests may expound to significant improvement. These tests can thus help in future to prognosticate CVJ anomalies and prioritize management based on the progression of subclinical deterioration.

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Nil.

Conflicts of interest
There are no conflicts of interest.

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PROFORMA FOR DATA COLLECTION

Name/Neuro No :

Age:

Sex:

UHID :

Occupation :

Address :/Contact phone no:

Email Id:

Presenting Symptoms :Duration : ....

A.Neck pain	1. Yes	2. No
B.Neck Tilt	1. Yes	2. No
C.Movement restriction	1. Yes	2. No
D.Difficulty in walking	1. Yes	2. No

E.Stiffness of Limbs — 1.None   2. Lower limbs   3. One side Limbs   4. All 4 limbs

F.Weakness of Limbs — 1.None   2. Lower limbs   3. One side Limbs   4. All 4 limbs   5. Upper limbs Only

G.Transitory Attacks   1. Yes   2. No
   1a Paresthesias   1b Weakness
   1c Flexing neck forward   1d Others

H.Progressive weakness	1. Yes	2. No
I.Sphincter Disturbances	1. Yes	2. No
J.Sensory Symptoms	1. Yes	2. No
K.Comorbidities	1. Yes	2. No

Findings :

A.Headish	1. Yes	2. No
B.Short Neck	1. Yes	2. No
C.Low Hairline	1. Yes	2. No
D.Neck movement restriction	1. Yes	2. No

E.Respiratory Reserve   1.Good   2.Affected yet Adequate   3. Poor

F.Spinal Deformity   1.Kyphosis   2.Scoliosis   3. None

G.Cranial nerves	1. Yes	2. No
H.Papilledema	1. Yes	2. No
I.Lower cranial nerves	1. Yes	2. No
J.Nystagmus	1. Yes	2. No

MOBILITY (ON LEVEL SURFACES)
0 = immobile or < 50 yards
5 = wheelchair independent, including corners, > 50 yards
10 = walks with help of one person (verbal or physical) > 50 yards
15 = independent (but may use any aid, for example, stick) > 50 yards _____

STAIRS
0 = unable
5 = needs help (verbal, physical, carrying aid)
10 = independent _____
TOTAL (0–100): _____

Radiology :
X ray

CT C spine

Ct angiogram

MRI

A.AAD	1.Reducible	2. Non-reducible
B.Rotary dislocation	1. Yes	2. No
C.Basilar Invagination	1. Yes	2. No
D.C1 Assimilation	1. Yes	2. No
E.Osodontoideum	1. Yes	2. No
F.Osterminale	1. Yes	2. No
G.Absent odontoid	1. Yes	2. No
H.Lateral mass anomaly	1. Yes	2. No
I.LowerClivus anomaly	1. Yes	2. No
J. Klippel-Feil anomaly	1. Yes	2. No

K. Others

L. Compression   1. Anterior 2.Posterior 3.Both 4. None

M. Degree of Compression   1.Mild 2.Moderate 3.Severe 4. None (After reduction)

N. Arnold Chiari Malformation   1. Yes   2. No

O.Syringomyelia	1. Yes	2. No
P. Traction	1. Yes	2. No
Q.Clinical improvement after traction	1. Yes	2. No
R.X ray Reduction after Traction	1. Yes	2. No

L.Posterior column Involvement   1. Yes   2. No

M.Ataxia   1. Yes   2. No

N.Pansels   1.Upper limbs 2. One side limbs   3. All limbs and trunk   4. No involvement

O.Head Muscle Involvement   1. Yes   2. No

P.Increased Tone   1.Absent 2. Lower Limbs   3. One side Limbs 4. All Limbs

Q.DTR 's increased   1. Absent 2. Lower Limbs   3. One side Limbs 4. All Limbs

R.Plantars   1.Upgoing   2. Downgoing 3. Not elicitable

S.C2 sensory impairment/anomalies   1. Yes   2. No

Nurick Grade/Social Functioning Before treatment
0 sign or symptoms of root involvement, but no evidence of cord disease
1 signs of cord disease but no difficulty walking
2 slight difficulty walking and preventing full-time employment
3 difficulty walking preventing full-time employment or ability to do all housework, but not so severe as to require someone's help to walk
4 able to walk with someone's help or with the aid of frame
5 chairbound or bedridden

THE BARTHEL INDEX
Activity Score
FEEDING
0 = unable
5 = needs help cutting, spreading butter, etc., or requires modified diet
10 = independent _____

BATHING
0 = dependent
5 = independent (or in shower) _____

GROOMING
0 = needs to help with personal care
5 = independent face/hair/teeth/shaving (implements provided) _____

DRESSING
0 = dependent
5 = needs help but can do about half unaided
10 = independent (including buttons, zips, laces, etc.) _____

BOWELS
0 = incontinent (or needs to be given enemas)
5 = occasional accident
10 = continent _____

BLADDER
0 = incontinent, or catheterized and unable to manage alone
5 = occasional accident
10 = continent _____

TOILET USE
0 = dependent
5 = needs some help, but can do something alone
10 = independent (on and off, dressing, wiping) _____

TRANSFERS (BED TO CHAIR AND BACK)
0 = unable, no sitting balance
5 = major help (one or two people, physical), can sit
10 = minor help (verbal or physical)
15 = independent

Management :

1.Anterior decompression   2. Anterior and posterior decompression with fusion
3. Posterior decompression and fusion   4. Posterior fusion

Operative complications:

A.Durabar	1. Yes	2. No
B.Cord trauma	1. Yes	2. No

Postoperative complications:
1.open wound infection
2.Closed wound infection
3.Dislocation
4.Neurological deterioration
5.Others
6. None

Immediate Results :
1.Improved   2.Status quo   3. Deteriorated   4. NA

A.Muscle Power	1.Improved 2.Status quo 3. Deteriorated
B.Sensation	1.Improved 2.Status quo 3. Deteriorated
C.Sphincter involvement	1. Improved 2.Status quo 3. Deteriorated
Spasticity	1.Improved 2.Status quo 3. Deteriorated
E.Gait	1.Improved 2.Status quo 3. Deteriorated
F.Respiration	1.Improved 2.Status quo 3. Deteriorated

Outcome :   1. Alive 2. Dead
Follow up :   1. Yes   2. No

Clinical Status at follow up :

Duration in months ....
1.Improved   2.Status quo   3. Deteriorated   4. NA

A. Muscle Power
Right upper limb	1.Improved 2.Status quo 3. Deteriorated
Right lower limb	1.Improved 2.Status quo 3. Deteriorated
Right hand Grip	1.Improved 2.Status quo 3. Deteriorated
Left upper limb	1.Improved 2.Status quo 3. Deteriorated
Left lower limb	1.Improved 2.Status quo 3. Deteriorated
Left hand Grip	1.Improved 2.Status quo 3. Deteriorated

Spasticity(Modified Ashworth scale)
Right upper limb	1.Improved 2.Status quo 3. Deteriorated
Right lower limb	1.Improved 2.Status quo 3. Deteriorated
Left upper limb	1.Improved 2.Status quo 3. Deteriorated
Left lower limb	1.Improved 2.Status quo 3. Deteriorated

B.Sensation   1.Improved   2.Status quo   3. Deteriorated
C.Sphincter Involvement   1.Improved   2.Status quo   3. Deteriorated
Spasticity   1.Improved   2.Status quo   3. Deteriorated
E.Gait   1.Improved   2.Status quo   3. Deteriorated
F.Respiration   1.Improved   2.Status quo   3. Deteriorated
Expired During Follow up :   1. Yes   2. No

Nurick's Grade/Social Functioning at last follow up ....

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Appendix 1: A detailed clinical questionnaire used for collecting data