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Research Article

Comparative Evaluation of Baroreflex Sensitivity and Cardiovascular Autonomic Regulation in Type 2 Diabetes Mellitus and Healthy Individuals: A Cross-Sectional Study

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Abstract: Type 2 Diabetes Mellitus (T2DM) may impair cardiovascular autonomic regulation, including arterial baroreflex function, cardiovagal responses and orthostatic blood-pressure control. Objective: To compare Baroreflex Sensitivity (BRS), cardiovascular autonomic reflex tests, resting hemodynamics and frequency-domain Heart Rate Variability (HRV) in individuals with T2DM and healthy non-diabetic controls. **Methods**: This comparative cross-sectional study included 120 adults aged 45-55 years (60 with T2DM and 60 non-diabetic controls). The assessed outcomes comprised resting, sequence-up and sequence-down BRS, baroreflex sequence count, cardiovagal and vasomotor reflex tests, resting cardiovascular indices and spectral HRV. Associations between BRS and clinical variables were examined within the diabetic group. **Results:** Resting BRS was lower in participants with T2DM than in controls (7.1±1.9 versus 14.2±2.8 ms/mmHg; $p<0.0001$). Sequence-up BRS (7.8±2.2 versus 15.8±3.1) and sequence-down BRS (6.5±1.8 versus 13.1±2.6 ms/mmHg) were similarly reduced. Deep-breathing E:I ratio, standing 30:15 ratio, Valsalva ratio and respiratory heart-rate variation were lower in T2DM, whereas postural systolic blood-pressure fall was greater (14.8±4.6 versus 4.2±2.1 mmHg). Total HRV power and high-frequency power were reduced. In T2DM, BRS correlated inversely with HbA1c ($r = -0.724$) and diabetes duration ($r = -0.681$), both $p<0.0001$. **Conclusion:** T2DM was associated with diminished baroreflex responsiveness, attenuated cardiovagal reflexes, impaired orthostatic compensation and reduced HRV. Higher HbA1c and longer diabetes duration were associated with lower BRS.

Keywords: Baroreflex Sensitivity, Type 2 Diabetes Mellitus, Cardiovascular Autonomic Function, Cardiac Autonomic Neuropathy, Heart Rate Variability, Active Standing

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is associated with progressive metabolic, neural and vascular abnormalities that extend beyond disturbances in glucose homeostasis. Cardiovascular Autonomic Neuropathy (CAN) is a clinically important complication because autonomic control contributes to moment-to-moment regulation of cardiac interval, vascular resistance and arterial pressure. Impaired autonomic regulation may remain asymptomatic initially but can manifest as resting tachycardia, diminished heart-rate responses to physiological challenges, exercise intolerance and orthostatic blood-pressure instability [1-3].

The arterial baroreflex provides rapid negative-feedback buffering of arterial-pressure fluctuations. Stretch-sensitive receptors in the carotid sinus and aortic arch transmit afferent signals to medullary cardiovascular centers, which modulate vagal and sympathetic efferent activity. Cardiac Baroreflex Sensitivity (BRS) is commonly expressed as the change in R-R interval per unit change in systolic blood pressure (ms/mmHg). Lower BRS is compatible with reduced reflex cardiac responsiveness; the magnitude of BRS also depends on vascular mechanics, baseline pressure, breathing, age and the method of estimation [4].

Chronic hyperglycemia, oxidative stress, microvascular injury, altered arterial compliance and autonomic nerve damage are biologically plausible contributors to abnormal cardiovascular reflexes in diabetes. Standard cardiovascular autonomic reflex tests-heart-rate variation during deep breathing, the 30:15 ratio after standing and the Valsalva ratio-assess cardiovagal responses. Postural blood-pressure measurements and the pressor response to sustained handgrip complement these tests by characterizing integrated vasomotor regulation [2,5].

Frequency-domain Heart-Rate Variability (HRV) provides an additional description of cardiac interval oscillations. Total power summarizes overall variability; High-Frequency (HF) power is strongly related to respiration-linked vagal modulation, whereas Low-Frequency (LF) power reflects mixed regulatory influences. The LF/HF ratio should not be regarded as a direct quantitative measure of sympathetic-to-parasympathetic neural activity [6].

Previous work in Indian adults has demonstrated attenuated BRS in dysglycemia, supporting the importance of assessing autonomic regulation across the diabetes spectrum [7]. The present study compared quantitative baroreflex indices alongside resting cardiovascular measurements, cardiovagal and vasomotor reflex tests and spectral HRV in age-matched participants with T2DM and non-diabetic controls. Within the diabetic group, associations with glycemic control and disease duration were also examined.

MATERIALS AND METHODS

Study Design and Participants

An analytical cross-sectional comparative study was undertaken in the Department of Physiology, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh, India. The study population comprised 120 adults aged 45-55 years: 60 patients with established T2DM and 60 apparently healthy non-diabetic controls. Participants were enrolled using consecutive sampling, with controls selected from hospital staff, patient attendants and community volunteers. The two groups were matched for age.

Eligibility Criteria

The diabetic group included clinically stable adults with an established diagnosis of T2DM of at least one year of duration who could perform active standing independently. The control group included adults without diabetes, with normal clinical and biochemical glycemic assessment and no recognized cardiovascular autonomic disorder. Major cardiac disease, clinically important arrhythmia, uncontrolled hypertension, advanced kidney or major systemic disease, neurological disorders affecting autonomic function and medications with substantial autonomic or chronotropic effects were exclusion criteria. Conventional diabetes treatments were permitted.

Clinical and Biochemical Measurements

Anthropometric assessment included height, weight, Body Mass Index (BMI), waist circumference, hip circumference and waist-hip ratio. Glycemic assessment included fasting plasma glucose, postprandial plasma glucose and glycated Hemoglobin (HbA1c). Following supine rest, resting Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and heart rate were assessed. Mean Arterial Pressure (MAP) and Rate-Pressure Product (RPP) were derived from cardiovascular measurements.

Active-Standing and Cardiovascular Reflex Assessment

Cardiovascular responses to postural change were assessed during transition from supine rest to active standing. Resting cardiovascular measurements were obtained after approximately 10 minutes of supine rest; blood pressure was assessed during standing, including at three minutes. The 30:15 R-R interval ratio characterized the early cardiac response to standing. Other cardiovagal outcomes comprised the Expiration-to-Inspiration (E:I) ratio during deep breathing, the Valsalva ratio and deep-breathing heart-rate variation. Vasomotor outcomes comprised the postural change in SBP and DBP and the DBP rise during sustained isometric handgrip.

Baroreflex Sensitivity and Frequency-Domain HRV

Cardiac baroreflex regulation was characterized by resting BRS, sequence-up BRS, sequence-down BRS and the number of identified baroreflex sequences. BRS was expressed in ms/mmHg. The HRV frequency-domain panel comprised total power, absolute LF and HF power, LF and HF normalized units and the LF/HF ratio. These measurements were evaluated as complementary indices of cardiac autonomic modulation rather than interchangeable physiological endpoints.

Statistical Analysis

Continuous variables were summarized as mean ± Standard Deviation (SD). Between-group comparisons were evaluated using independent-samples Student t-tests. Pearson correlation coefficients quantified associations between BRS and clinical variables within the diabetic group. Multiple linear regression was used to evaluate the independent associations of HbA1c, diabetes duration, fasting glucose, age and BMI with BRS. Two-tailed p values <0.05 were considered statistically significant. Group differences and 95% Confidence Intervals (CIs) are presented where available.

RESULTS

Baseline Characteristics

All 120 participants were included in the comparative analysis (60 per group). Mean age was identical in the two groups (49.75±3.01 years). Compared with controls, participants with T2DM had higher body weight, BMI, waist circumference, waist-hip ratio, fasting and postprandial plasma glucose and HbA1c. Height and hip circumference were comparable between groups (Table 1).

Table 1: Baseline Anthropometric and Glycemic Characteristics

Parameter	Healthy controls (n = 60)	T2DM (n = 60)	p value
Age (years)	49.75±3.01	49.75±3.01	1.0000
Height (cm)	159.46±6.46	159.28±7.14	0.8873
Weight (kg)	64.94±8.11	68.01±7.31	0.0309
BMI (kg/m²)	25.59±3.35	26.90±3.22	0.0312
Waist circumference (cm)	88.91±5.91	95.46±6.18	<0.0001
Hip circumference (cm)	98.91±5.92	99.79±5.15	0.3879
Waist-hip ratio	0.90±0.09	0.96±0.08	0.0004
Fasting plasma glucose (mg/dL)	90.87±7.78	148.73±20.35	<0.0001
Postprandial glucose (mg/dL)	124.77±11.11	207.51±30.24	<0.0001
HbA1c (%)	5.38±0.28	7.34±0.95	<0.0001

Values are mean ± SD. BMI, Body Mass Index; T2DM, Type 2 Diabetes Mellitus

Table 2: Resting Hemodynamic and Cardiac Parameters

Parameter	Healthy controls	T2DM	p value
Resting SBP (mmHg)	118.4±8.2	126.8±10.5	<0.0001
Resting DBP (mmHg)	76.2±5.4	81.4±6.8	<0.0001
Mean arterial pressure (mmHg)	90.27±5.8	96.53±7.4	<0.0001
Resting heart rate (beats/min)	72.4±5.8	81.6±7.2	<0.0001
Rate-pressure product	8571.8±820	10347.7±1150	<0.0001

Values are mean ± SD. SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure

Table 3: Comparison of Baroreflex Sensitivity Parameters

Parameter	Healthy controls	T2DM	Difference (95% CI)	p value
Resting BRS (ms/mmHg)	14.2±2.8	7.1±1.9	7.1 (6.2-8.0)	<0.0001
Sequence-up BRS (ms/mmHg)	15.8±3.1	7.8±2.2	8.0 (7.0-9.0)	<0.0001
Sequence-down BRS (ms/mmHg)	13.1±2.6	6.5±1.8	6.6 (5.8-7.4)	<0.0001
Total baroreflex sequences (n)	142±28	68±19	74 (65-83)	<0.0001

Values are mean ± SD. Difference = Healthy-Control Mean minus T2DM mean, CI, Confidence Interval, BRS, Baroreflex Sensitivity

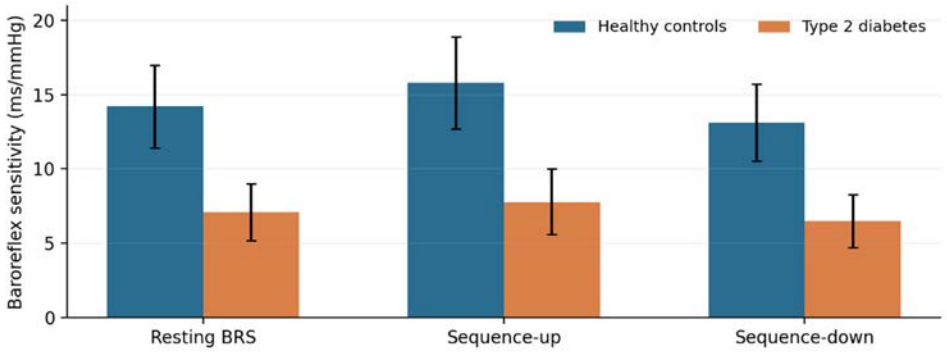


Figure 1: Resting, Sequence-Up and Sequence-Down BRS in Healthy Controls and T2DM (mean ± SD)

Resting Cardiovascular Parameters

Resting SBP, DBP, MAP, heart rate and RPP were higher in participants with T2DM (all p<0.0001; Table 2). Mean resting heart rate was 81.6±7.2 beats/min in T2DM and 72.4±5.8 beats/min in controls. Resting SBP was 126.8±10.5 and 118.4±8.2 mmHg, respectively. The elevated RPP in the diabetic group reflected a higher resting hemodynamic load.

Baroreflex Sensitivity and Sequence Parameters

All four baroreflex indices differed between groups (Table 3). Resting BRS was 14.2±2.8 ms/mmHg in healthy controls and 7.1±1.9 ms/mmHg in T2DM, representing a difference of 7.1 ms/mmHg (95% CI 6.2-8.0; p<0.0001). Sequence-up BRS and sequence-down BRS were reduced by approximately half in T2DM. The mean number of identified baroreflex sequences was also lower (68±19 Vs 142±28; p<0.0001). The pattern was consistent across pressure-rise and pressure-fall sequences (Figure 1).

Cardiovagal and vasomotor reflex responses

Cardiovagal responses were consistently attenuated in the diabetic group (Table 4). The E:I ratio was 1.18±0.10 in T2DM versus 1.38±0.12 in controls, while the 30:15 ratio was 1.09±0.07 versus 1.24±0.09. The Valsalva ratio and respiratory heart-rate variation were likewise reduced (all p<0.0001).

Table 4: Cardiovascular Autonomic Reflex Test Parameters

Reflex parameter	Healthy controls	T2DM	p value
E:I ratio (deep breathing)	1.38±0.12	1.18±0.10	<0.0001
30:15 ratio (standing)	1.24±0.09	1.09±0.07	<0.0001
Valsalva ratio	1.62±0.18	1.31±0.15	<0.0001
Deep-breathing HR variation (beats/min)	22.4±4.1	12.8±3.6	<0.0001
Postural SBP fall (mmHg)	4.2±2.1	14.8±4.6	<0.0001
Postural DBP fall (mmHg)	2.1±1.4	7.6±2.8	<0.0001
Handgrip DBP rise (mmHg)	18.6±3.4	10.2±2.9	<0.0001

Values are mean ± SD. E:I, Expiration-to-Inspiration; HR, Heart Rate

Table 5: Frequency-Domain Heart-Rate Variability Indices

Parameter	Healthy controls	T2DM	p value
Total power (ms^2)	2145.6±412.3	1120.4±285.6	<0.0001
LF power (ms^2)	648.2±124.5	385.1±92.4	<0.0001
HF power (ms^2)	482.5±98.4	194.2±54.8	<0.0001
LF (normalized units)	57.3±6.2	66.5±7.1	<0.0001
HF (normalized units)	42.7±6.2	33.5±7.1	<0.0001
LF/HF ratio	1.34±0.28	1.98±0.42	<0.0001

Values are mean ± SD. LF, Low Frequency; HF, High Frequency; HRV, Heart-Rate Variability

Table 6: Correlations between BRS and Clinical Factors in T2DM (n = 60)

Clinical variable	Pearson r	95% CI	p value
HbA1c (%)	−0.724	−0.812 to −0.608	<0.0001
Diabetes duration (years)	−0.681	−0.780 to −0.553	<0.0001
Fasting plasma glucose (mg/dL)	−0.542	−0.677 to −0.378	<0.0001
Age (years)	−0.315	−0.518 to −0.076	0.0142
BMI (kg/m^2)	−0.184	−0.412 to +0.062	>0.05

Pearson correlation coefficients; CI, Confidence Interval

Table 7: Multiple Linear Regression Predictors of BRS in T2DM

Predictor	B coefficient	Standardized β	95% CI for B	p value
HbA1c (%)	−0.482	−0.458	−0.712 to −0.252	0.0001
Duration of diabetes (years)	−0.314	−0.362	−0.492 to −0.136	0.0009
Fasting glucose (mg/dL)	−0.012	−0.135	−0.028 to 0.004	0.1394
Age (years)	−0.028	−0.098	−0.070 to 0.014	0.1881
BMI (kg/m^2)	−0.015	−0.034	−0.079 to 0.049	0.6410

B, Unstandardized Coefficient; β, Standardized Coefficient; CI, Confidence Interval

Greater orthostatic BP falls were evident in T2DM: mean SBP fall was 14.8±4.6 versus 4.2±2.1 mmHg and DBP fall was 7.6±2.8 versus 2.1±1.4 mmHg (both p<0.0001). The handgrip-associated DBP rise was smaller in T2DM (10.2±2.9 versus 18.6±3.4 mmHg; p<0.0001). These differences indicate diminished cardiovagal reactivity and altered orthostatic vasomotor compensation.

Frequency-Domain Heart-Rate Variability

Total HRV power was reduced in T2DM (1120.4±285.6 ms^2) compared with controls (2145.6±412.3 ms^2; p<0.0001). LF and HF absolute power were also lower, with a particularly pronounced reduction in HF power (194.2±54.8 Vs 482.5±98.4 ms^2). LF normalized units and the LF/HF ratio were higher, whereas HF normalized units were lower, in participants with T2DM (Table 5).

Clinical Correlates of BRS in T2DM

Within the diabetic group, BRS showed strong negative correlations with HbA1c (r = −0.724, p<0.0001) and duration of diabetes (r = −0.681, p<0.0001). Fasting blood glucose showed a moderate inverse correlation and age had a weaker inverse association. The correlation with BMI was not statistically significant (Table 6).

Multiple Linear Regression

Multivariable regression showed that HbA1c (B = −0.482, standardized β = −0.458; p = 0.0001) and diabetes duration (B = −0.314, β = −0.362; p = 0.0009) were independently associated with lower BRS. Fasting plasma glucose, age and BMI were not significant after simultaneous adjustment (Table 7).

BRS According to Glycemic Control and Disease Duration

A graded decline in BRS was evident across worsening HbA1c categories: 9.2±1.6 ms/mmHg in participants with HbA1c<7.0%, 7.4±1.4 in the 7.0-8.5% category and 5.2±1.1 in those with HbA1c > 8.5% (reported trend p<0.0001). Similar changes occurred with diabetes duration: BRS was 8.8±1.5 ms/mmHg at<5 years, 7.1±1.3 at 5-10 years and 4.9±1.0 at > 10 years (reported trend p<0.0001). Total HRV power and HF power declined, whereas the LF/HF ratio rose, across these strata (Table 8 and Figure 2).

Table 8: Autonomic Indices by Glycemic Control and Duration of T2DM

Stratum (n)	Resting BRS	Total power (ms^2)	HF power (ms^2)	LF/HF
HbA1c <7.0% (14)	9.2±1.6	1380.5±210.4	265.4±42.1	1.65±0.22
HbA1c 7.0-8.5% (26)	7.4±1.4	1125.8±185.2	198.6±35.4	1.92±0.31
HbA1c >8.5% (20)	5.2±1.1	840.2±150.6	138.2±28.5	2.28±0.45
Duration <5 years (18)	8.8±1.5	1320.4±195.8	248.5±38.6	1.72±0.25
Duration 5-10 years (27)	7.1±1.3	1105.6±172.4	189.2±32.1	1.98±0.35
Duration >10 years (15)	4.9±1.0	795.8±138.2	130.4±24.8	2.31±0.48

Values are mean ± SD. BRS is expressed as ms/mmHg. Reported p for the trend across each three-level classification was <0.0001

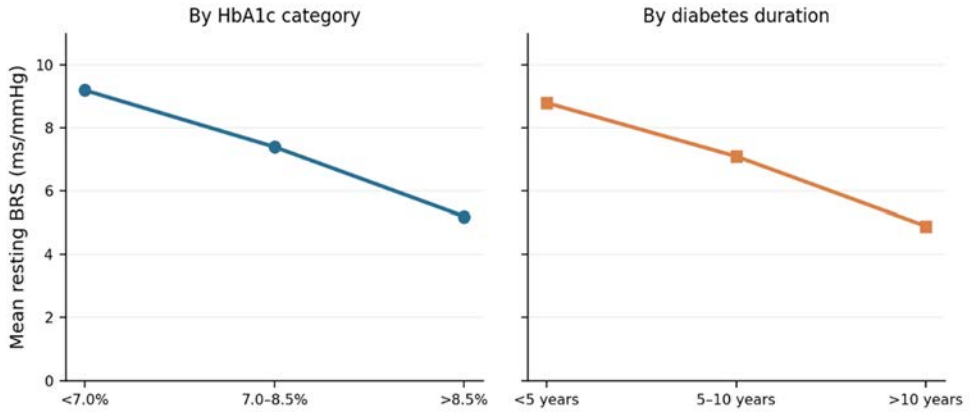


Figure 2: Mean Resting BRS across HbA1c Categories and Diabetes-Duration Strata among Participants with T2DM

DISCUSSION

This study identified a broad pattern of cardiovascular autonomic impairment in middle-aged adults with T2DM. The central observation was an approximately 50% lower resting BRS in T2DM than in age-matched non-diabetic controls. Reductions in sequence-up and sequence-down BRS, as well as fewer identified baroreflex sequences, were accompanied by weaker cardiovagal reflex responses, a greater postural BP fall and depressed HRV spectral power. These convergent observations are consistent with diminished cardiovascular reflex responsiveness in the setting of diabetes.

Reduced Baroreflex Sensitivity and Physiological Mechanisms

The baroreflex protects short-term cardiovascular stability by rapidly adjusting cardiac interval and vasomotor tone in response to arterial-pressure changes. A reduced BRS slope indicates a smaller cardiac interval change associated with a given pressure fluctuation. In diabetes, possible contributors include autonomic axonal injury, microvascular dysfunction, oxidative injury and reduced arterial wall distensibility, which may lessen baroreceptor engagement for a given pressure increment [1,4]. The consistent impairment of resting, sequence-up and sequence-down measures indicates that the difference is not restricted to a single directional component of the reflex response.

The findings align with the Indian study by Keerthi and colleagues, which described lower BRS and HRV in normotensive participants with prediabetes and diabetes compared with normoglycemic adults [7]. Although populations and measurement protocols are not identical, both studies support the presence of baroreflex impairment across dysglycemic states.

Cardiovagal Reflex Impairment

The lower E:I ratio, 30:15 ratio, Valsalva ratio and deep-breathing heart-rate variation indicate widespread attenuation of cardiovagal reflex responsiveness. The E:I ratio and respiratory heart-rate variation are particularly sensitive to respiratory-related vagal modulation, whereas the 30:15 ratio characterizes the rapid R-R interval adaptation that follows an active postural transition. The combined reduction in these measures is more informative than an isolated abnormal response. Established consensus guidance recommends several cardiovascular reflex tests when characterizing CAN [2,5].

Orthostatic and Resting Hemodynamic Differences

The greater average orthostatic decline in SBP and DBP suggests a reduced ability to maintain arterial pressure during standing. Effective adaptation requires rapid changes in cardiac autonomic output and resistance-vessel constriction after gravitational pooling. The smaller pressor response to sustained handgrip further supports impaired integrated vasomotor responsiveness. However, mean postural BP changes alone should not be equated with a participant-level diagnosis of orthostatic hypotension.

Participants with T2DM also had higher resting heart rate, arterial pressure and rate-pressure product. Resting tachycardia can occur with reduced cardiovagal restraint, although baseline BP, adiposity, physical fitness and other clinical influences can also contribute. The higher resting cardiovascular load provides additional context for the attenuated reflex responses observed during physiological maneuvers.

Heart-Rate Variability and Autonomic Regulation

The lower absolute total, LF and HF HRV power in the diabetic group indicates reduced cardiac interval variability across several frequency bands. The marked reduction in HF power is consistent with lower respiratory-linked vagal modulation. Although LF normalized units and the LF/HF ratio were increased, those changes should not be interpreted as direct proof of heightened sympathetic neural outflow; LF power arises from multiple physiological influences, including baroreflex-linked oscillations [6]. The HRV findings are best considered complementary evidence of altered cardiac autonomic modulation.

Relationships with HbA1c and Diabetes Duration

Within the diabetic group, higher HbA1c and longer duration of diabetes were associated with lower BRS. The strength of these associations exceeded those for age and BMI and both HbA1c and duration remained independently associated with BRS in the multivariable model. HbA1c reflects longer-term glycemic exposure, whereas disease duration may capture cumulative metabolic and vascular injury. The graded reductions across glycemic and duration categories reinforce the consistency of these associations, but a cross-sectional design cannot determine whether improved control would reverse established impairment.

Clinical Relevance

CAN has clinical significance because autonomic impairment is associated with adverse cardiovascular outcomes and mortality in diabetes [2,8]. Assessment of cardiovagal function together with orthostatic responses may help characterize physiological impairment that is not apparent from routine resting vital signs alone. Quantitative BRS and HRV may contribute additional physiological information when measured with standardized instrumentation and interpreted alongside established reflex testing. The present findings support further validation of integrated autonomic assessments in patients with persistent hyperglycemia or long-standing T2DM.

Strengths and Limitations

The study compared a broad set of baroreflex, cardiovagal, vasomotor and frequency-domain HRV measures in two equally sized groups within a restricted age range. Correlation, multivariable regression and clinical subgroup analyses added information regarding the relationship between BRS, glycemic status and disease duration.

The single-center cross-sectional design limits causal interpretation and generalizability. Differences in BMI, resting blood pressure, treatment exposure, physical fitness, vascular stiffness and unmeasured comorbidities may have contributed to group contrasts. Standardization and reporting of the original beat-to-beat blood-pressure acquisition, synchronized ECG recording, BRS sequence algorithm and spectral HRV processing are necessary for reproducibility and comparison with other studies. Participant-level diagnostic classification of CAN and orthostatic hypotension was not established by the group summary measures. Larger prospective studies are needed to determine predictive value and longitudinal changes in BRS.

CONCLUSION

Adults with T2DM demonstrated markedly lower resting, sequence-up and sequence-down baroreflex sensitivity and fewer baroreflex sequences than healthy non-diabetic controls. These differences occurred alongside weaker parasympathetic cardiovascular reflexes, greater postural blood-pressure reductions, impaired handgrip pressor responsiveness and reduced frequency-domain HRV. Higher HbA1c and longer diabetes duration were independently associated with lower BRS. The findings support comprehensive investigation of cardiovascular autonomic dysfunction in T2DM, with standardized physiological measurements and prospective validation.

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