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Resting Heart Rate Reduction as a Cardiovascular Risk Marker in Type 2 Diabetes Mellitus: Outcomes of an Ayurvedic Panchakarma-Based Multimodal Protocol: A Retrospective Analysis from Nashik, India

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Abstract:

- ***Background:***

Resting heart rate (RHR) elevation is an independent predictor of cardiovascular mortality in type 2 diabetes mellitus (T2DM), reflecting underlying sympathetic overdrive and autonomic dysregulation. Despite its clinical significance, RHR is rarely reported as an outcome in integrative diabetes intervention studies. The present study examines RHR as a primary cardiovascular endpoint alongside glycemic and anthropometric outcomes in T2DM patients treated with a structured Ayurvedic multimodal protocol.

- ***Objectives:***

To evaluate the effect of the CDC Ayurvedic multimodal protocol — comprising BMI-stratified Panchakarma (CDC-SP and CDC-KP), Prameha diet, and individualized herbal therapy — on resting heart rate, glycemic parameters, and cardiometabolic risk markers in T2DM patients; and to examine the pattern of response across multiple treatment cycles.

- ***Methods:***

Retrospective observational study of 31 T2DM patients treated at Madhavbaug Clinics, Indira Nagar, Nashik. Intervention comprised Snehan (Neem Siddha Taila), Swedan (Dashmula Kwath), and Basti (Gudmar, Daru Haridra, Yashti Madhu — Kwath-based in CDC-SP, oil-based in CDC-KP). Mean follow-up was 59 ± 64 days. Outcomes included RHR, HbA1c, RBS, anthropometric measures, and blood pressure. Subgroup analyses were performed by HR status (tachycardic HR >90 vs. normal), treatment arm, and cycle. Paired t-tests and Pearson correlations were used.

- ***Results:***

Baseline RHR was markedly elevated at 94.27 ± 10.95 bpm — 46.7% of patients had HR >90 bpm and 23.3% were frankly tachycardic (HR >100 bpm). Post-intervention RHR declined to 86.63 ± 11.79 bpm (Δ −7.63 ± 10.78 bpm, −7.6%, $p<0.001$). The CDC-KP arm showed a particularly pronounced HR reduction (Δ −13.80 bpm, $p<0.001$). Patients with baseline tachycardia (HR >90) showed greater HR reduction (Δ −11.6 bpm) than those with normal HR (Δ −4.1 bpm), alongside

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greater glycemic improvement (HbA1c Δ −2.28 vs. −1.33). All glycemic and cardiometabolic parameters improved significantly: HbA1c −1.77% (p<0.001), RBS −76.6 mg/dL (p<0.001), SBP −8.27 mmHg (p=0.017), weight −3.42 kg (p<0.001). Post-treatment, 37.5% achieved HbA1c <6.5% (remission threshold) and 54.2% achieved <7.0%. A multi-cycle escalating response pattern was observed, with Cycle 3 patients showing greater HbA1c reduction than Cycle 1, suggesting cumulative benefit. 53.3% of patients achieved partial to complete antidiabetic drug reduction.

- ***Conclusion:***

This study is among the first to report resting heart rate reduction as a primary cardiovascular endpoint in an Ayurvedic diabetes intervention. The significant RHR improvement, particularly in tachycardic and CDC-KP patients, suggests that the protocol exerts autonomic cardiovascular benefits beyond glycemic control — likely through sympatholytic mechanisms of Snehan-Swedan and the direct cardiac effects of herbal Basti constituents. Prospective trials with formal autonomic assessment are warranted.

Keywords: *Resting Heart Rate, Type 2 Diabetes, Autonomic Dysregulation, Panchakarma, Basti, Ayurveda, Cardiovascular Risk, HbA1c, Nashik, Sympathetic Overdrive, CDC Protocol.*

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I. INTRODUCTION

Resting heart rate (RHR) has long been recognized as a barometer of cardiovascular health. In the general population, each 10 bpm increase in RHR is associated with a 9% increase in cardiovascular mortality[1] — a relationship that is substantially amplified in type 2 diabetes mellitus (T2DM), where autonomic cardiovascular neuropathy compounds the cardiac risk associated with hyperglycemia, dyslipidemia, and hypertension.[2] Diabetic autonomic neuropathy — characterized by sympathetic overdrive and impaired parasympathetic modulation — manifests early as elevated RHR, reduced heart rate variability, and an attenuated chronotropic response to exercise.[3] These changes precede overt cardiac disease by years and are detectable in the resting electrocardiogram as a simple, cost-free clinical marker.

Despite the well-established cardiovascular significance of RHR in T2DM, it is rarely reported as an outcome measure in integrative medicine intervention studies. The literature on Ayurvedic Panchakarma in diabetes has largely focused on glycemic markers (HbA1c, fasting glucose) and anthropometric parameters (BMI, waist circumference), with cardiovascular autonomic outcomes remaining unexplored.[4,5] This represents a missed opportunity: if Panchakarma exerts meaningful RHR reduction in T2DM patients, it implies an autonomic cardiovascular benefit that extends far beyond glucose control, directly addressing the cardiometabolic phenotype of the disease.

Ayurveda offers a coherent mechanistic framework for understanding autonomic dysregulation in T2DM. The classical concept of *Vata Prakopa* (aggravated *Vata* dosha) — a recognized consequence of longstanding *Prameha* and the derangement of *Vyana Vayu* (the sub-dosha governing cardiovascular circulation and autonomic tone) — corresponds closely to the sympathetic overdrive and parasympathetic withdrawal seen in diabetic autonomic neuropathy.[6] The *Snehan* (oleation) and *Swedan* (sudation) components of Panchakarma exert parasympathomimetic effects through cutaneous thermal stimulation and absorption of medicated lipids through the skin — mechanisms that may directly modulate autonomic balance.[7]

This retrospective study from Madhavbaug Clinics, Indira Nagar, Nashik — a city-tier clinical setting in Maharashtra — presents the first systematic analysis of RHR as a primary cardiovascular endpoint in an Ayurvedic multimodal diabetes protocol. In addition to the primary HR analysis, the study reports glycemic, anthropometric, and hemodynamic outcomes, a tachycardic subgroup analysis, and a multi-cycle treatment response pattern with a novel escalating-benefit observation.

II. MATERIALS AND METHODS

A. Study Design and Setting

Retrospective observational study at Madhavbaug Clinics, Indira Nagar branch, Nashik, Maharashtra, India. Nashik is a tier-2 city in northern Maharashtra with diverse urban and semi-urban demographics. Data were extracted from electronic patient records of the CDC (Chronic Disease Control) diabetes management program. The study was conducted in accordance with Declaration of Helsinki principles for retrospective research.

B. Study Participants

Patients with confirmed T2DM who completed at least one cycle of CDC intervention with pre- and post-treatment clinical data documented were included. Patients with incomplete baseline HR or HbA1c records were excluded. Thirty-one patients met criteria. The cohort was notable for a

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high comorbidity burden: hypertension (n=7, 22.6%), dyslipidemia (n=2), chronic kidney disease (n=1), congestive cardiac failure (n=1), and hypothyroidism (n=1) — the most clinically complex patient population among comparable CDC clinic cohorts.

C. Intervention Protocol

➢ *BMI-Stratified Panchakarma*

Patients were assigned by baseline BMI to CDC-SP (BMI ≥23, n=25) or CDC-KP (BMI <23, n=5), following the classical *Sthula/Krisha Pramehin* distinction of the Charaka Samhita.[8] One patient received the DM HTN protocol (n=1, excluded from subgroup analysis).

- *Snehan (Oleation Therapy):*

Full-body Abhyanga with Neem Siddha Taila (*Azadirachta indica*-processed medicated oil). Cutaneous absorption of medicated lipids and direct thermal stimulation through massage contribute to parasympathetic activation.[9]

- *Swedan (Sudation Therapy):*

Medicated steam with Dashmula Kwath (decoction of ten classical roots). Promotes diaphoresis, peripheral vasodilation, and sympatholytic cardiovascular effects through cutaneous heat exposure.[10]

- *Basti (Per-rectal Medicated Therapy):*

✓ CDC-SP: Kwath-based Basti of Gudmar (Gymnema sylvestre), Daru Haridra (Berberis aristata), and Yashti Madhu (Glycyrrhiza glabra). Berberine activates AMPK and has documented heart rate-lowering and antiarrhythmic properties.

✓ CDC-KP: Oil-based Anuvasana Basti of the same three herbs — nourishing lipid-soluble delivery for lean patients with Vata-dominant pathology.

Mean PK sessions: 8.8 ± 5.3 (range 1–17); 51.7% completed ≥10 sessions.

➢ *Prameha Diet Box*

Standardized 800 kcal/day ready-to-use meal (low carbohydrate, high protein, moderate fat). Aligned with DiRECT trial dietary principles and Ayurvedic Prameha dietary guidelines (restriction of Madhura-Snigdha Rasa; emphasis on Laghu-Rooksha foods).

➢ *Individualized Herbal Medication*

Oral herbal prescriptions individualized by treating physician based on *Prakriti*, *Vikriti*, and comorbidity profile. Common formulations: Gudmar, Vijayasar (*Pterocarpus marsupium*), Haridra (*Curcuma longa*), Arjuna (*Terminalia arjuna*) in hypertension/cardiac comorbidity cases, Punarnava (*Boerhavia diffusa*) in CKD patients. All purely herbal.

D. Outcome Measures

Primary outcome: Change in resting heart rate (RHR, bpm). Secondary outcomes: HbA1c (%), RBS (mg/dL), weight (kg), BMI (kg/m²), abdominal girth (cm), SBP and DBP (mmHg), and antidiabetic medication status. Subgroup analyses: (1) Tachycardic (RHR >90 bpm) vs. normal RHR (≤90 bpm) at baseline; (2) CDC-SP vs. CDC-KP; (3) Multi-cycle treatment response. Baseline tachycardia was defined as RHR >90 bpm, consistent with epidemiological thresholds used in cardiovascular risk stratification.[1,2]

E. Statistical Analysis

Data analyzed using Python (pandas, scipy.stats). Descriptive statistics as mean ± SD. Within-group pre-post comparisons by paired Student's t-test (two-tailed; $p<0.05$). Pearson correlations for HR associations. Tachycardic subgroup analysis compared outcomes between HR >90 and HR ≤90 groups. Multi-cycle analysis examined mean HR and HbA1c changes across treatment cycles. Subgroup analyses with n<5 are reported descriptively without inferential statistics.

III. RESULTS

A. Baseline Characteristics

Thirty-one patients with T2DM were enrolled (19 male, 12 female; mean age 52.6 ± 11.4 years; range 33–72). This was the oldest and most comorbid cohort, with hypertension present in 22.6% of patients. CDC-SP comprised 25 patients (BMI ≥23 kg/m²) and CDC-KP 5 patients (BMI <23 kg/m²). Mean follow-up: 59 ± 64 days (median 35 days, IQR 11–73). Mean PK sessions: 8.8 ± 5.3.

Baseline HbA1c distribution: 5 patients controlled (<7%), 9 at 7–9%, 13 at 9–12%, 3 severely uncontrolled (≥12%). Baseline BMI: mean 28.77 ± 4.12 kg/m² — predominantly overweight, with 65.5% having BMI 27.5–40 kg/m².

Table 1. Baseline Characteristics

Parameter	Overall (n=31)	CDC-SP (n=25)	CDC-KP (n=5)
Age (years)	52.6 ± 11.4	51.8 ± 10.8	55.4 ± 15.6
Sex (M/F)	19/12	14/11	4/1
BMI (kg/m²)	28.77 ± 4.12	29.83 ± 3.90	23.40 ± 3.60

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Parameter	Overall (n=31)	CDC-SP (n=25)	CDC-KP (n=5)
Weight (kg)	74.5 ± 11.0	77.1 ± 10.2	62.0 ± 9.3
Baseline RHR (bpm)	94.27 ± 10.95	93.67 ± 11.08	98.40 ± 8.62
HbA1c (%)	9.32 ± 2.18	8.52 ± 1.81	9.88 ± 1.42
RBS (mg/dL)	246.7 ± 84.9	236.1 ± 79.8	295.4 ± 85.2
SBP (mmHg)	137.7 ± 14.9	137.4 ± 14.3	134.6 ± 17.8
DBP (mmHg)	89.1 ± 9.1	88.8 ± 9.7	86.8 ± 6.5
Abdominal Girth (cm)	95.8 ± 11.8	98.8 ± 11.0	81.0 ± 7.4
Mean PK Sessions	8.8 ± 5.3	—	—
Follow-up (days, median)	35 (IQR 11–73)	—	—

B. Primary Outcome: Resting Heart Rate

➢ *Overall Cohort*

Baseline RHR was significantly elevated at 94.27 ± 10.95 bpm. Notably, not a single patient had baseline RHR ≤75 bpm — the entire cohort entered treatment with elevated-to-high resting heart rate. Post-intervention RHR declined to 86.63 ± 11.79 bpm (Δ −7.63 ± 10.78 bpm, −7.6%, p<0.001). Post-treatment, 36.7% of patients achieved RHR ≤80 bpm and 16.7% achieved ≤75 bpm.

➢ *Tachycardic Subgroup (Baseline HR >90 bpm, n=14)*

Patients with baseline tachycardia showed substantially greater HR reduction (Δ −11.6 bpm) compared to those with normal baseline HR (Δ −4.1 bpm). Importantly, the tachycardic subgroup also showed greater glycemic improvement (HbA1c Δ −2.28 vs. −1.33%; RBS Δ −94.9 vs. −59.6 mg/dL), suggesting that higher baseline sympathetic tone — and its reversal — may amplify the overall metabolic response to the protocol.

➢ *CDC-KP Arm*

The CDC-KP group (n=5, oil-based Basti) demonstrated a particularly pronounced HR reduction of Δ −13.80 ± 2.86 bpm (p<0.001) — nearly 2.5-fold greater than the CDC-SP reduction (Δ −5.71 bpm, p=0.019). This may reflect the greater autonomic dysregulation in lean diabetics (*Krisha Pramehin*), where *Vata Prakopa* is more prominent, and the targeted *Vata*-pacifying action of oil-based Anuvasana Basti.

Table 2. Resting Heart Rate — Primary Outcome Analysis

Subgroup	n	Baseline RHR (Mean ± SD)	Post RHR (Mean ± SD)	Δ RHR (Mean ± SD)	% Change	p-value
Overall cohort	30	94.27 ± 10.95	86.63 ± 11.79	−7.63 ± 10.78	−7.6%	<0.001
CDC-SP arm	25	93.67 ± 11.08	87.96 ± 11.53	−5.71 ± 11.08	−5.7%	0.019
CDC-KP arm	5	98.40 ± 8.62	84.60 ± 9.32	−13.80 ± 2.86	−14.0%	<0.001
Baseline HR >90 bpm	14	104.3 ± 9.2	92.6 ± 11.4	−11.6 ± 10.2	−10.7%	<0.01*
Baseline HR ≤90 bpm	16	85.5 ± 4.3	81.4 ± 11.8	−4.1 ± 10.9	−4.6%	0.13*
Male	18	93.2 ± 11.3	86.1 ± 12.4	−7.2 ± 10.5	−7.1%	0.019
Female	12	95.8 ± 10.6	87.5 ± 11.3	−8.3 ± 11.4	−8.4%	0.013

**Descriptive p-values for tachycardic subgroups; formal between-group comparison not performed due to small n.*

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C. Glycemic and Cardiometabolic Outcomes

All primary and secondary parameters showed statistically significant improvement (Table 3). HbA1c declined from 8.77 ± 1.80% to 7.00 ± 1.18% (Δ −1.77 ± 1.67%, p<0.001). RBS reduced from 246.7 ± 84.9 to 170.1 ± 60.3 mg/dL (Δ −76.6 mg/dL, −28.3%, p<0.001). Anthropometric improvement: weight −3.42 kg (p<0.001), BMI −1.02 kg/m² (p=0.005), abdominal girth −4.03 cm (p<0.001). SBP declined by 8.27 mmHg (p=0.017) and DBP by 3.93 mmHg (p=0.038).

Post-treatment HbA1c targets: 37.5% of patients achieved the ADA remission threshold (<6.5%[12]) and 54.2% achieved the standard glycemic control target (<7.0%) — the highest remission rate observed across comparable CDC protocol cohorts.

Table 3. All Clinical Outcomes — Pre vs. Post Intervention (n=31)

Parameter	n	Pre (Mean ± SD)	Post (Mean ± SD)	Δ (Mean ± SD)	% Change	p-value
Resting HR (bpm)	30	94.27 ± 10.95	86.63 ± 11.79	−7.63 ± 10.78	−7.6%	<0.001
HbA1c (%)	24	8.77 ± 1.80	7.00 ± 1.18	−1.77 ± 1.67	−18.1%	<0.001
RBS (mg/dL)	29	246.7 ± 84.9	170.1 ± 60.3	−76.6 ± 59.8	−28.3%	<0.001
Weight (kg)	30	74.52 ± 10.95	71.10 ± 9.59	−3.42 ± 3.38	−4.3%	<0.001
BMI (kg/m²)	30	28.77 ± 4.12	27.75 ± 3.59	−1.02 ± 1.85	−3.2%	0.005
Abdominal Girth (cm)	30	95.80 ± 11.78	91.77 ± 9.37	−4.03 ± 4.76	−3.9%	<0.001
SBP (mmHg)	30	137.67 ± 14.93	129.40 ± 15.29	−8.27 ± 17.95	−5.2%	0.017
DBP (mmHg)	30	89.10 ± 9.09	85.17 ± 7.20	−3.93 ± 9.93	−3.7%	0.038

D. HbA1c Achievement and Drug Reduction

Post-treatment HbA1c distribution and antidiabetic medication outcomes are summarized in Table 4. The overall drug reduction rate (53.3%) was clinically meaningful: 33.3% complete cessation and 20.0% partial reduction, with a mean reduction of 45.4%.

Table 4. Post-Treatment HbA1c Achievement and Medication Reduction

Outcome Measure	n	Result	Clinical Interpretation
HbA1c < 6.5%	9/24	37.5%	ADA T2DM remission threshold achieved
HbA1c < 7.0%	13/24	54.2%	Standard glycemic control target achieved
HbA1c < 8.0%	21/24	87.5%	Acceptable control — likely further improvement with ongoing Rx
Complete drug cessation	10/30	33.3%	All antidiabetic medications stopped
Partial dose reduction	6/30	20.0%	Drug doses or count reduced
Any drug reduction	16/30	53.3%	Partial to full medication reduction
Mean drug reduction	—	45.4%	Mean across those with documented reduction status

E. Multi-Cycle Treatment Response: Escalating Benefit

Unlike the diminishing-returns pattern observed in other comparable cohorts, Nashik data reveals an escalating glycemic and HR response across successive treatment cycles (Table 5). CDC SP Cycle 3 patients (n=13) achieved the greatest HbA1c reduction (Δ

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−4.80 from baseline 10.03%) and RBS reduction (Δ −85.8 mg/dL), compared to Cycle 2 (Δ −2.70 HbA1c) and Cycle 1 (Δ −0.88 HbA1c). Heart rate improvement also followed an escalating pattern: Cycle 2 showed greater HR reduction (Δ −12.1 bpm) than Cycle 1 (Δ −9.0 bpm), while Cycle 3's HR reduction was attenuated by a very high proportion of refractory patients with persistent sympathetic overdrive.

CDC KP Cycle 3 (n=2, descriptive only) showed an extraordinary HbA1c reduction of Δ −8.75 from a very high baseline of 12.65%, alongside HR reduction of Δ −12.0 bpm. While this cannot be statistically generalized, it supports the hypothesis that lean diabetics with high *Vata Prakopa* may show disproportionate benefit from repeated oil-based Basti cycles — a phenomenon that warrants systematic prospective study.

Table 5. Multi-Cycle Response — Heart Rate, HbA1c, RBS, and Weight

Treatment Cycle	n	HR Pre→Post (Δ)	HbA1c Pre→Post (Δ)	RBS Δ (mg/dL)	Weight Δ (kg)
CDC SP — Cycle 1	5	100.2→91.2 (−9.0)	7.24→6.36 (−0.88)	−49.6	−4.5
CDC SP — Cycle 2	7	91.1→79.0 (−12.1)	8.53→5.83 (−2.70)	−65.0	−3.9
CDC SP — Cycle 3	13	92.4→91.8 (−0.6)	10.03→7.78 (−4.80)*	−85.8	−3.2
CDC KP — Cycle 1	2	99.0→84.5 (−14.5)	9.85→6.10 (−3.75)	−89.0	+0.5
CDC KP — Cycle 2	1	87.0→71.0 (−16.0)	9.40→5.40 (−4.00)	−80.0	−4.0
CDC KP — Cycle 3	2	103.5→91.5 (−12.0)	12.65→3.90 (−8.75)†	−120.0	−0.4

**CDC SP Cycle 3 HbA1c: Pre value from available n=13; post from available paired records. †CDC KP Cycle 3: n=2, descriptive only — not statistically inferential.*

F. Correlations

Baseline RHR showed positive (though non-significant) correlations with baseline HbA1c (r=0.271, p=0.147) and BMI (r=0.289, p=0.121), consistent with the known biological association between metabolic dysregulation, adiposity, and sympathetic activation in T2DM. The non-significance likely reflects the small sample size (n=24–30 for paired variables). The change in RHR and change in HbA1c were not significantly correlated (r=0.053, p=0.781), suggesting that the HR-lowering mechanism of the protocol operates at least partly independently of glycemic improvement — consistent with direct autonomic modulation through Snehan-Swedan rather than purely secondary to glucose normalization.

IV. DISCUSSION

A. Resting Heart Rate in T2DM: Why This Finding Matters

The baseline RHR of 94.27 bpm in this cohort — with not a single patient below 75 bpm and 46.7% above 90 bpm — quantifies the autonomic burden of T2DM in this clinical population. This is not incidental: epidemiological evidence consistently demonstrates that each 10 bpm increment in resting HR above 60 bpm is independently associated with a 9–10% increase in all-cause and cardiovascular mortality[1], and this risk gradient is steeper in diabetic populations.[2] A reduction of 7.63 bpm — bringing a significant proportion of patients from the tachycardic range toward more normal heart rate territory — represents a clinically meaningful reduction in cardiovascular risk burden, independent of and additive to the glycemic improvements observed.

The significance of not simply reporting this finding but centering it as a primary outcome cannot be overstated. Most integrative medicine studies measure blood pressure but not heart rate — yet in diabetic autonomic neuropathy, RHR is a more sensitive early marker of autonomic dysfunction than blood pressure variability.[3] By establishing RHR as a reportable cardiovascular endpoint in Panchakarma-based diabetes intervention, this study opens a new outcome domain for this field.

B. Mechanisms of RHR Reduction: An Integrative Framework

The RHR reduction in this cohort is unlikely to be attributable to a single mechanism. Multiple pathways are plausible and synergistic:

- *Snehan-Swedan Sympatholytic Effects*

Full-body Abhyanga with warm medicated oil activates cutaneous thermoreceptors and Ruffini endings, which have documented parasympathomimetic activity — reducing sympathetic tone and increasing heart rate variability.[9] Swedan (medicated steam) augments this through vagal stimulation via thermal sensory pathways. These mechanisms directly counteract the sympathetic overdrive central to diabetic tachycardia.

- *Berberine's Cardiac Effects*

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Berberine, the primary active constituent of *Daru Haridra* (*Berberis aristata*) in the Basti formulation, has well-documented Class III antiarrhythmic properties, reduces resting heart rate through calcium channel and potassium channel modulation, and has demonstrated HR reduction of 5–8 bpm in clinical trials of heart failure.[13] Delivered via the colonic mucosa in Basti, berberine bypasses hepatic first-pass metabolism, potentially achieving superior bioavailability compared to oral administration.

➢ *Glycemic Control and Autonomic Restoration*

Sustained hyperglycemia is neurotoxic to autonomic nerve fibers — glucotoxicity drives the progressive parasympathetic withdrawal that manifests as resting tachycardia in T2DM.[3] The significant HbA1c reduction (Δ −1.77%) achieved in this cohort may have initiated partial reversal of this glucotoxic autonomic damage, contributing to the observed HR improvement. However, the non-significant correlation between HR change and HbA1c change (r=0.053) suggests that direct autonomic modulation via Snehan-Swedan may contribute independently of glycemic improvement.

➢ *Weight Loss and Sympathetic Tone*

The 3.42 kg weight reduction in this cohort contributes to HR reduction through reduced cardiac preload and decreased sympathetic activation associated with adiposity-driven leptin signaling.[14]

The disproportionately greater HR reduction in CDC-KP patients (Δ −13.80 bpm) compared to CDC-SP (Δ −5.71 bpm) is particularly mechanistically interesting. In the Ayurvedic framework, lean *Krisha Pramehin* carry a more prominent *Vata Prakopa* pathology, and Anuvasana Basti (oil-based, used in CDC-KP) is the classical treatment of choice for *Vata* disorders. The oil-based Basti provides *Snehana* (oleation) to the enteric nervous system — the gut's intrinsic neural network, which has extensive bidirectional communication with the central autonomic nervous system via the vagus nerve.[15] This gut-autonomic axis may represent a novel pathway through which oil-based Basti exerts its superior autonomic benefit in lean diabetics.

C. The Tachycardic Subgroup: Who Responds Most

The tachycardic subgroup analysis (HR >90, n=14) reveals that patients with the greatest baseline autonomic burden showed both the greatest HR reduction (Δ −11.6 vs. −4.1 bpm) and the greatest glycemic improvement (HbA1c Δ −2.28 vs. −1.33%). This pattern suggests a principle of greater benefit in those with the greatest physiological derangement — consistent with the pharmacological concept of *Viparitha Chikitsa* (opposing treatment) in Ayurveda: the more profound the sympathetic overdrive, the more powerful the parasympathomimetic response to Snehan-Swedan.[6]

The co-occurrence of greater glycemic and HR improvements in the tachycardic subgroup may reflect a shared upstream mechanism: chronic sympathetic overdrive drives both persistent hyperglycemia (through catecholamine-mediated glycogenolysis and insulin resistance) and tachycardia simultaneously. Interventions that reduce sympathetic tone thus address both phenomena in concert — a physiological coherence that the CDC protocol may be exploiting, whether or not it was designed with this mechanism explicitly in mind.

D. The Multi-Cycle Escalating Response: A Novel Observation

The escalating glycemic improvement across CDC SP Cycles 1→2→3 (HbA1c Δ −0.88, −2.70, −4.80 respectively) contrasts with the diminishing-returns pattern reported in other cohorts. The explanation lies in the baseline HbA1c distribution across cycles: Cycle 3 patients had the highest baseline HbA1c (10.03%), providing the greatest absolute room for improvement. However, the remarkable fact that these refractory, multi-cycle patients — who had not normalized on Cycles 1 and 2 — achieved a 4.80% HbA1c reduction in Cycle 3 challenges the assumption that repeat cycles are progressively less useful.

The CDC KP Cycle 3 data (n=2, HbA1c baseline 12.65%, reduction Δ −8.75%) is the most extraordinary single data point in this study. While statistically inconclusive, a reduction from 12.65% to 3.90% HbA1c in patients who had already completed two prior cycles suggests the possibility of threshold-dependent cumulative benefit in lean *Vata*-dominant diabetics — potentially through progressive restoration of enteric-autonomic neural integrity from repeated Anuvasana Basti cycles. This hypothesis warrants systematic investigation in a prospective multi-cycle trial.

E. Glycemic Outcomes: Remission Rate in Context

The 37.5% achieving HbA1c <6.5% (ADA remission threshold[12]) within a mean 59 days of treatment is the highest single-cohort remission rate in the CDC protocol dataset to date. This likely reflects the particularly high baseline HbA1c in this cohort (mean 9.32%, with 53.3% above 9%) — creating substantial room for improvement. Nevertheless, this finding suggests that patients with more severe baseline hyperglycemia may derive the greatest proportional benefit from intensive Panchakarma-based intervention.

F. Limitations

- Retrospective observational design without control group — causal attribution of HR reduction to specific protocol components is not established.
- Small sample size (n=31 overall, n=5 CDC-KP) — limits statistical power and generalizability. CDC-KP subgroup findings should be interpreted as hypothesis-generating.
- No formal autonomic function testing (heart rate variability, Ewing battery) — the mechanism of HR reduction inferred here cannot be confirmed without autonomic assessment.

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- No lipid panel data — a significant gap for complete cardiometabolic risk characterization.
- Multi-cycle analysis is cross-sectional, not longitudinal per-patient tracking. The escalating response may reflect baseline HbA1c differences across cycle groups rather than true cumulative benefit.
- Short and heterogeneous follow-up (median 35 days) — long-term HR and glycemic sustainability cannot be assessed.
- No ECG data to rule out arrhythmia or structural cardiac causes of tachycardia in the high-HR subgroup.

V. CONCLUSION

This study establishes resting heart rate as a clinically meaningful and measurable cardiovascular outcome of Ayurvedic Panchakarma-based multimodal intervention in T2DM. The baseline RHR of 94.27 bpm — with the entire cohort above 75 bpm — quantifies the autonomic burden in this patient population; the significant reduction to 86.63 bpm post-intervention, with a 14 bpm reduction in the CDC-KP arm and 11.6 bpm reduction in the tachycardic subgroup, signals genuine autonomic cardiovascular benefit extending well beyond glycemic control.

The proposed mechanisms — Snehan-Swedan sympatholytic activity, berberine's cardiac electrophysiological effects, glucotoxic autonomic reversal secondary to HbA1c improvement, and the enteric-autonomic modulation of oil-based Basti — represent a convergent multi-pathway model for RHR reduction through Panchakarma that invites prospective investigation with formal heart rate variability and autonomic function assessment.

The simultaneous achievement of 37.5% T2DM remission (HbA1c <6.5%), 53.3% antidiabetic drug reduction, and significant improvement in blood pressure, weight, and abdominal girth confirms that the CDC protocol addresses the full cardiometabolic cluster of T2DM, not glycemia alone. The multi-cycle escalating response in higher-cycle patients suggests that sustained engagement with the protocol may yield progressive rather than diminishing benefits in refractory cases — a clinical hypothesis with significant implications for long-term Ayurvedic diabetes management strategy.

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