

# ANALYSIS OF AUTONOMIC REGULATIONS IN YOUNG ADULTS WITH TACHYCARDIA: UNRAVELING THE PARADOX OF VAGAL DOMINANCE

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**Resume.** The human heart is not a metronome; its rhythm is a dynamic conversation between the brain, nervous system and the heart itself. This conversation is translated by heart rate variability (HRV), the subtle fluctuations in the time of its intervals between consecutive heartbeats. Tachycardia is typically associated with sympathetic overactivity part of “fight or flight” system in resting heart rate above normal, yet in real practice we occasionally meet paradoxical patterns with fast heart rate despite clear markers of vagal predominance which is rolled by parasympathetic system. such mismatches represent a challenge for understanding of autonomic regulation and a call for a deeper analysis, that is why we need (HRV) as a window to overlook into the dynamicity of sympathetic and parasympathetic branches. The higher variability the better flexibility and regularity. However reduced variability reflects rigidity and stress control. This study seeks to explore the paradox of these changes.

**Keywords:** heart rate variability, cardiovascular diseases, tachycardia, vagal dominance, ans regulation.

**Relevance.** Heart rate variability quantifies the beat to beat variations in R-R intervals and represent a window that overlooking into the functional status of the cardiac Autonomic Nervous system (ANS). The interpretation of specific HRV metrics is critical for clinical applications. Time domain indices, specifically the root square of successive differences (RMSSD) and the percentage of the normal to normal intervals (pNN50) are a quantitative markers of the rapid vagal modulation. However, in the frequency domain a density power analysis delineates the total variance into frequency components. The High-Frequency (HF) components (0.15 – 0.40 Hz) is almost entirely vagal reflecting respiratory sinus rhythm. The opposite component is the Low-Frequency (LF) parameter (0.04-0.15 Hz) contains mixed

sympathetic and vagal input [1-3].

Tachycardia is a condition where an elevated resting heart rate associated with lack of vagal tone and an augmented sympathetic drive. The state most oven resulted in high (HL, LF) ratio and causing overall HRV depression which measured by parameters like (SDNN) the standard deviation of NN intervals. The diminished HRV clarify the low physiological flexibility and a prognostic marker for various cardiovascular diseases, and give us an early hint for the development of heart failure. A cardiologic assumption that a sustained high heart rate is primarily driven by hyper-activation of sympathetic system or generalized ANS impairment reducing vagal efference [8].

The fundamental challenge that is investigated in this study aimed to find

the conventional inverse relationship between heart rate and vagal activity. The group we call Tachycardia + Vagotonia has a high heart rate ( $80 >$ ) along with strong signs of vagal activity which is unusual. Normally, high vagal activity helps slow the heart, but the opposite here the heart stays fast referring to something else is driving the tachycardia [7]. What necessitates us to explore this profile is the mechanism that is similar to those implicated syndromes like Inappropriate Sinus tachycardia (IST) which has characteristics like unexplainable fast sinus heart rate, often due to elevated intrinsic pacemaker or Beta-adrenergic receptors hypersensitivity. Therefore, the quantitative vagal dominance of this group describes a high regulatory effort and yet not enough to maintain optimal cardiac control [5-6].

In order to distinguish between a healthy vagal state and a compensated pathological state, we must assess autonomic dynamics during physiological stress. Different types of functional tests specifically the Hyperventilation Test (HV) and the Orthostatic Test (OT), permit the observation of dynamics responsiveness and maximum ANS reserve capacity. Furthermore, the cardiovagal baroreflex integrity can be precisely measured by 30/15 ratio that is derived from R-R intervals sequences. This ratio reflects recovery reserves after stress, representing a pathway integral to hemodynamic stability. Analyzing this divergence alongside with baroreflex integrity provide necessary frame work to interpret the mechanism in both sympathicotonic and the paradoxical vagotonic tachycardia phenotypes.

**Materials and methods.** Design

and dataset; The study enrolled 38 young participants based on their LF/HF ratios who were allocated into two equally sized groups ( $n = 19$  each):

Group 1: Tachycardia + sympathetic dominance ( $n=19$ );

Group 2: Tachycardia + vagal dominance ( $n=19$ ).

HRV data were collected using methodology compliant with established standards for short term recordings. The protocol included that all participants underwent a comprehensive standard ECG for 5 minutes at rest, followed by hyperventilation and orthostatic test each for 3 minutes.

HRV was analyzed by time and frequency domain (RRNN, SDNN, RMSSD, pNN50(%), TP, VLF, LF, HF, LF\HF). The parameters (RRNN, SDNN, RMSSD, pNN50(%) are the main representatives of time domain, therefore they are the baseline that reflecting variability, while (LF, HF, LF\HF) representing frequency domain, so they are reflecting the autonomic components contribution to heart rate regulation. The ratio 30/15 ratio was calculated too through RR intervals to the 15/30 beats after standing. Statistical data processing was carried out using Stat-Tech v 4.8.0 software. Normality was assessed by the Shapiro-Wilk test. Depending on data distribution, Students t-test or Mann-Whitney U test was applied. A p-value  $< 0.05$  was considered significant.

### **Results and their discussion.**

Baseline autonomic profile at rest: The analysis revealed that at rest heart rate (RRNN) did not significantly differ between both groups, they exhibited equal level of tachycardia. The (group 1) was 88.80 bpm, and the second group 88.00

( $p = 0.640$ ), meaning that R-R intervals (RRNN) were also statistically similar. However, a lot of parasympathetic markers were clearly higher in the tachycardia +vagotonia (group 2) including: SDNN +28% ( $P = 0.050$ ), RMSSD +57% ( $P = 0.004$ ), pNN50 is higher ( $p = 0.012$ ).

These results reflected higher vagal activity despite the presence of tachycardia. In addition that frequency domain HF power, HF norm, % HF were elevated noticeably.

**Tabl. 1.** HRV Parameters of Tachycardia During Rest

| Parameter              | Group 1                   | Group 2                    | p-value (Z) |
|------------------------|---------------------------|----------------------------|-------------|
| HR, BPM                | 88.80 (83.40 – 95.50)     | 88.00 (82.50 – 94.05)      | 0,640       |
| CV                     | 4.24 ± 1.19               | 5.45 ± 1.98                | 0,030*      |
| RRNN (ms)              | 667.53 ± 62.93            | 678.11 ± 56.26             | 0,588       |
| SDNN (ms)              | 28.79 ± 9.63              | 36.95 ± 14.60              | 0,050*      |
| RMSSD (ms)             | 19.79 ± 8.90              | 31.11 ± 13.04              | 0,004*      |
| pNN50, %               | 0.90 (0.20 – 3.45)        | 7.10 (2.15 – 10.50)        | 0,012*      |
| TP (ms <sup>2</sup> )  | 752.00 (499.50 – 1074.50) | 1138.00 (755.00 – 1748.50) | 0,062       |
| VLF (ms <sup>2</sup> ) | 331.00 (216.50 – 450.00)  | 383.00 (271.00 – 685.50)   | 0,300       |
| LF (ms <sup>2</sup> )  | 308.00                    | 241.00                     | 0,804       |
| HF (ms <sup>2</sup> )  | 146.00 (77.50 – 268.00)   | 515.00 (242.00 – 699.00)   | < 0,001*    |
| LF/HF                  | 1.78 (1.39 – 2.15)        | 0.62 (0.53 – 0.79)         | < 0,001*    |
| %VLF                   | 44.82 ± 10.53             | 37.97 ± 13.10              | 0,084       |
| %LF                    | 35.34 ± 7.60              | 23.75 ± 5.43               | < 0,001*    |
| %HF                    | 19.85 ± 6.65              | 38.29 ± 11.49              | < 0,001*    |
| LF norm                | 64.38 ± 9.20              | 39.09 ± 8.84               | < 0,001*    |
| HF norm                | 35.62 ± 9.20              | 60.91 ± 8.84               | < 0,001*    |

During hyperventilation neither group were in a state of well demonstrated patterns and the differences almost disappeared completely. Although the vagotonia possessed higher HF but no parameter reached a statistical significance.

In orthostatic test also showed no significant statistical differences but noticeably a marked rise in HR, shift towards sympathetic dominance. However, the recovery ratio 30/15 revealed a

notable pattern in group 1 (1.18) and group 2 (1.15). Both values below normal threshold indicating insufficient cardiovascular recovery after standing.

The results that founded confirms the existence of two distinct autonomic phenotypes in young adults with tachycardia. Despite having nearly the same resting heart rate and both groups exhibited different autonomic signature when evaluated through HRV analysis.

The (group 2) showed all classic

HRV signs of strong dominance of parasympathetic system activity – higher (RMSSD), higher HF power, and high pNN50% values. Normally, this should result in a lower heart rate creating paradox where the the active “braking” system fails to maintain its optimal physiological work.

The first group fits the traditional expectations, yet the the second group showed different phenomena which urges us to wonder why the heart does not slow down despite the vagal activation which reflect for us a deeper imbalance like the vagal signal reaches the heart but does not exert the expected results or perhaps the heart have become less responsive. This abnormality could be early marker of autonomic strains. The most interesting finding that during the stress test. When both groups were put under physical and physiological challenge these differences vanished and both groups responded similarly showing that high vagal tone does not provide better adaptability [10].

Furthermore, the poor 30/15 ratio in both groups shared a weakness in heart rate recovery. It reinforced this message through this simple reflex test, where it should show a quick adaptability in healthy young people, instead it revealed slow or insufficient heart rate recovery in

both groups which suggests something more important to investigate [11-13].

Whether during sympathetic dominance or paradoxically high vagal tone the presence of tachycardia is telling us that the autonomic system is less flexible than it should be. This indicate that tachycardia caused by any underlying cause can be early sign of reduced resilience in young adults.

### Conclusions:

1. Subjects with baseline tachycardia present with two different resting autonomic profiles: an expected sympathicotonia-low vagal tone-and an unexpected pattern of vagotonia-characterized by markedly higher resting parasympathetic indices, such as RMSSD and HF.

2. Significant differences in resting HRV measures (SDNN, RMSSD, and HF power) disappeared after exposing participants to functional tests of hyperventilation and orthostatic challenge.

3. The recovery of the cardiovascular system from stress-as reflected by the 30/15 ratio-already appears to be compromised in both groups of tachycardic individuals, with the lowest reserve found in the tachycardia + vagotonia group. This finding suggests that the unusual autonomic combination may be a marker of reduced adaptive capacity under stress.

### Literature

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## АНАЛИЗ ВЕГЕТАТИВНОЙ РЕГУЛЯЦИИ У МОЛОДЫХ ЛЮДЕЙ С ТАХИКАРДИЕЙ: РАЗГАДЫВАНИЕ ПАРАДОКСА ДОМИНИРОВАНИЯ БЛУЖДАЮЩЕГО НЕРВА

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**Резюме.** Человеческое сердце не метроном; его ритм – это динамический диалог между мозгом, нервной системой и самим сердцем. Этот диалог передается через вариабельность сердечного ритма (ВСР) – едва заметные колебания интервалов между последовательными сердечными сокращениями. Тахикардия обычно связана с симпатической гиперактивностью, частью системы «бей или беги», при которой частота сердечных сокращений в состоянии покоя превышает норму. Однако в реальной практике мы иногда сталкиваемся с парадоксальными паттернами с учащенным сердечным ритмом, несмотря на явные маркеры преобладания вагуса, контролируемого парасимпатической системой. Такие несоответствия представляют собой проблему для понимания автономной регуляции и призывают к более глубокому анализу, поэтому нам нужна ВСР как окно, позволяющее увидеть динамику симпатической и парасимпатической ветвей. Чем выше вариабельность, тем выше гибкость и регулярность. Однако сниженная вариабельность отражает ригидность и контроль над стрессом. Данное исследование направлено на изучение парадокса этих изменений.

**Ключевые слова:** вариабельность сердечного ритма, сердечно-сосудистые заболевания, тахикардия, вагальное доминирование, регуляция ВНС.