The Relationship Between Heart Rate Variability Parameters and Atrioventricular Nodal Reentrant Tachycardia

Lütfü Aşkın

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ABSTRACT

Introduction: Heart rate variability (HRV) is associated with sympathetic tone. Studies have disputed the interpretation of the Low Frequence (LF) and Low/high frequency (LF/HF) ratio for the indication of sympathetic cardiac control and autonomic balance. This study aims to investigate the association between HRV parameters and atrioventricular nodal reentrant tachycardia (AVNRT) and observe the effect of autonomic nervous system on AVNRT.

Materials and Method: 354 subjects with palpitations underwent electrophysiological study (EPS). Seventy-one percent (253/354) of patients had at least an ambulatory Holter ECG recording of 24hrs. As a consequence of the exclusion criteria, 160 individuals were classified into two groups i.e. a control group (no arrhythmia induced, n=90) and an AVNRT group (n=70).

Results: Daytime and nighttime LF and LF/HF ratio were significantly higher in AVNRT ($p<0.05$) as compared to controls. Interestingly, daytime Ultra Low Frequency, Very Low Frequency and total power were significantly lower in AVNRT.

Conclusion: AVNRT is associated with altered sympathovagal balance. Furthermore, increased LF and LF/HF may be an indicative of enhanced sympathetic activity in AVNRT patients as a result of inhomogeneous ventricular activation via the slow accessory pathway.

Keywords: Atrioventricular nodal reentrant tachycardia; heart rate variability; low frequence; low/high frequency.

Kalp Hızı Değişkenliği Parametreleri ile Atriyoventriküler Nodal Reentrant Taşikardi Arasındaki İlişki

Lütfü Aşkın

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Giriş: Kalp hızı değişkenliği (HRV) sempatik ton ile ilişkilidir. Çalışmalar sempatik kardiyak kontrol ve otonom denge endikasyonu için düşük Frekans (LF) ve düşük / yüksek frekans (LF/HF) oranının yorumlamasını tartışmışlardır. Bu çalışma, HRV parametreleri ile atriyoventriküler nodal reentrant taşikardi (AVNRT) arasındaki ilişkiyi araştırmayı ve otonomik sinir sisteminin AVNRT üzerindeki etkisini gözlemeyi amaçlamaktadır.

Hastalar ve Metod: Çarpıntısı olan 354 olguya elektrofizyolojik çalışma yapıldı (EPS). Hastaların yüzde yetmiş biri (253/354) en az bir 24 saatlik ambulatuvar Holter EKG kaydına sahipti. Dışlama kriterlerinin bir sonucu olarak, 160 birey iki gruba ayrıldı; kontrol grubu (aritmi indüklenmedi, n = 90) ve AVNRT grubu (n = 70).

Bulgular: Gündüz ve gece LF ve LF / HF oranı AVNRT' de kontrollerden anlamlı derecede yüksekti ($p <0.05$). İlginç olarak, gündüz Ultra Düşük Frekans, Çok Düşük Frekans ve toplam güç AVNRT'de anlamlı olarak düşüktü.

Sonuç: AVNRT değişmiş sempatovagal denge ile ilişkilidir. Dahası, artmış LF ve LF / HF, AVNRT hastalarında yavaş aksesuar yolla homojen olmayan ventriküler aktivasyonun bir sonucu olarak artmış sempatik aktivitenin bir göstergesi olabilir.

Anahtar Kelimeler: Atrioventriküler nodal reeentran taşikardi; kalp hızı değişkenliği; düşük frekans; düşük/yüksek frekans;

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Study population

The epicrisis, transaction reports, intracardiac records and baseline clinical and demographic characteristics were collected on all subjects enrolled in the study. A total of 354 patients with palpitations underwent electrophysiological study. Seventy-one percent (253/354) of patients had at least 24 hours of ambulatory Holter ECG recording. Exclusion criteria were as followed: hypertension (n=5), diabetes mellitus (n=5), coronary artery disease (n=5), beta blocker usage (n = 8) or Ca channel blocker usage (n = 2) and non-AVNRT (n= 63). After evaluation of exclusion criteria, 160 patients were enrolled in the study. The control group (group 1) had a total of 90 healthy controls with no arrhythmia and the AVNRT group (Group 2) consisted out of 70 patients. Subjects with a positive smoking history during the past year were classified as smokers. The Simpson's method was used to calculate the left ventricular ejection fraction.

Ambulatory Electrocardiography

A two channel bipolar recorder were performed during a working day. All patients have a similar daily routine. The ambulatory electrocardiographies (ECGs) were recorded continuously for 24h using a DR-512 VX3 ECG recorder system (‘Biomedical Systems’ ,Century series Holter analysis systems). The electrodes were positioned to obtain leads CM2 and CM5. The period 08.00 to 24.00 was considered day and the period 00.00 to 08.00 as night. All tapes were analyzed by two experienced cardiologists. HRV time and frequency domain indices were evaluated. 24-hour avarage heart rate (HR) recorded during 24-hour electrocardiographic monitoring.

Heart rate variability

Power spectral analysis was split into short recording and long recording frequency domain indices. Short recording frequency domain were divided in ultra low frequency (ULF: <0.003kHz), very low frequency (VLF: 0.003-0.04kHz), low frequency (LF: 0.04-0.15Hz) and high frequency (HF: 0.15-0.4Hz) and recorded between 2-5 min. LF and HF powers were reported as normalized units. Long recording frequency domain of ULF, VLF, LF and HF can be obtained by 24 recordings. The measurements were analyzed day/night. Signal powers of LF and HF bands were used due to the restriction duration of recordings. Logarithmic transformation of the integrals under the respective power spectral density function (expressed in ms2) were used. LF is an indicator of sympathetic activity. LF/HF ratio defines which nervous system is dominant with a high ratio indicating sympathetic system dominant and lower ratio indicating parasympathetic system dominant.

Electrophysiologic evaluation

All subjects were aware of the possible risks and consent forms were signed. After the 12 hour fastening period and at least a 3 day withhold of antiarrhythmic drugs, including beta blockers and Ca channel blockers, patients underwent EPS. Local anesthesia was used for EPS. Multipolar electrode catheters were introduced into the right atrium, the coronary sinus, His bundle and the right ventricular apex via the femoral and left subclavian veins. The standard 12 Lead ECG (30 to 500 Hz) with digital system and optical disk storage (EP Tracer, Holland) was used. To prohibit an accessory pathway, a set of well described maneuvers i.e. the activation pattern in the CS during ventricular stimulation and decremental retrograde VA conduction was used. After this procedure was complete, programmed atrial stimulation was performed. It is

common practice to induce AVNRT in the electrophysiology lab. If tachycardia could not be induced, isoproterenol or atropine infusion was given so that the heart rate increased at last by 25%. This procedure was repeated during medication infusion and washout phase. If AVNRT could not be inducted, the presence of an A-H jump (a prolongation of the AH interval > 50 msec) and an atrial echo with a documented history of supraventricular tachycardia compatible with AVNRT were sufficient to start the ablation procedure. Dual pathways were not demonstrated with atrial extrastimulus techniques in healthy controls.

Radiofrequency ablation

Radiofrequency energy was delivered to the presumed selected ablation site in the posterior part of Koch's triangle located in the superficial paraseptal endocardium of the right atrium. A standard deflectable 7F catheter with a 4 mm distal electrode was used for mapping and ablation (St. Jude Medical Inc, U.S). The ablation generator (IBI-1500T11, St. Jude Medical Inc, U.S) delivered 20 to 35 W of energy by a continuous unmodulated sine wave output at a frequency of 500 kHz. Ablation was performed during sinus rhythm. The ablation methods were similar for all AVNRT patients as previously described [9–12].

Statistical analysis

All analyses were performed using SPSS (version 21.0 for Windows; SPSS Inc., Chicago, IL, USA). Normality of the data distribution was analyzed using the Kolmogorov–Smirnov test. Continuous data are presented as mean±SD or median and interquartile ranges based on normality of variables. Differences among groups were compared by Independent Samples T- test or Mann-Whitney U test according to normality of variables. Categorical variables were summarized as percentages and compared with chi-square test. The correlation between baseline biomarkers was assessed with Spearman's and Pearson's correlation coefficient. Multivariate logistic regression analysis was used to determine the independent predictors for AVNRT. Statistical significance of the findings was interpreted based on p values and $p<0.05$ was set as the level of statistical significance.

Results

After the enrollment of 160 participants, Table 1 summarizes the baseline characteristics of the study patients. 90 patients (46.1±18.1) were assigned to the control group and 70 (43.9±16.5) patients were assigned to the AVNRT group. The demographic characteristics were similar (all, $p > 0.05$), including age, smoking and gender. 24 hour avarage HR and LVEF were not differ between groups ($p > 0.05$). There were no significant differences in serum biochemistry between the two groups (all $p > 0.05$). The hematological parameters were similar in both groups (all, $p > 0.05$) (Table 1).

Daytime HRV time domain indices were similar between groups (all, $p > 0,05$). Daytime HRV frequency domain indices (HF, LF NUs, HF NUs) were also similar in both groups (all, $p > 0.05$). Daytime LF and LF/HF ratio were significantly higher in AVNRT group ($p = 0.001$ and $p < 0.001$, respectively). Contrastly, daytime ULF, VLF and total power were significantly higher in control group ($p = 0.026$, $p = 0.040$ and $p = 0.025$). Nighttime HRV time domain indices were similar between the two groups ($p > 0.05$). Nighttime HRV frequency domain indices were similar in both groups (all $p>0.05$), expect LF and LF/HF ratio. Nighttime LF and LF/HF were significantly higher in AVNRT group (all, $p < 0.001$) (Table 2).

AVNRT positively related with daytime total, ULF and VLF power (r = 0.177, p = 0.025; r = 0.177, p = 0.025 and r = 0.163, p = 0.040). Daytime LF, LF/HF ratio and nighttime LF and LF/HF ratio were inversely correlated with AVNRT (r = - 0.256, p = 0.001; r = - 0.348, p < 0.001 and r = - 0.463, p < 0.001; r = - 0.385, p < 0.001) (Table 3). Multivariate analysis was performed in order to determine independent predictors of AVNRT. Daytime LF, VLF and nighttime LF were determined as an independent predictors of AVNRT (OR = 1.002, 95% CI 1.001-1.004, p = 0.002; OR = 0.999, 95% CI 0.998-1.000, p = 0.006 and OR = 1.003, 95% CI 139 1.001-1.004, p < 0.001) (Table 4).

Discussion

The current study demonstrates that increased HRV is associated with AVNRT. Patients with a history of AVNRT and overt ventricular preexcitation in the presence of a slow accessory pathway had higher LF of heart rate variability and higher LF/HF ratios compared to controls. Based on this we suggest that patients with AVNRT have higher sympathetic modulation at baseline and during daily routine life leading to alterations in their autonomic status.

Catheter ablation for AVNRT has a success rate of 95% but is associated with 0.5–1% risk in AV block, and 4% recurrence rate [13]. Based on the report by Katritsis et al. catheter ablation is most common treatment in patients with symptomatic AVNRT [14]. Catheter ablation was also the first choice for treatment of patients with symptomatic AVNRT in our study. HE et al. [15] reported that empirical slow accessory pathway ablation is a safe and effective method. Patients without tachycardia detection on ECG before ablation, have a better outcome. Previous studies have shown that the application of RF current in the slow accessory pathway area can make AVNRT non-inducible despite persistent slow accessory pathway conduction [15,16].

The R-R intervals prior to ablation in children were longer than after ablation of the slow accessory AV pathway [1]. Furthermore, the 24h Holter monitoring demonstrated that the mean and maximal HR was increased, whereas the HRV parameters were decreased after ablation of AV slow accessory pathway in children [17]. Interestingly admission of isoproterenol after ablation to evaluate the slow accessory pathway and generate AV nodal echo beats, led to no significant differences in recurrence rates between patients with complete elimination of the slow pathway and patients with residual jump and/or single echo beat [18]. Nigro et al. [19] showed that before the onset of AVNRT, the HRV significantly changed. They believed that the increased LF components during the prior hour before onset of AVNRT, was due to adrenergic predominance and the decreased HF components suggested a parasympathetic drive. The pattern of the LF/HF ratio was compatible with the dynamic changes in autonomic tone. Our study also suggests that sustained typical AVNRT episodes are preceded by increase in adrenergic drive.

The fluctuations of autonomic tone before the onset of AVNRT were emphasized by the results of time-domain HRV analysis: SDNN and SDANN were decreased implying a increase in sympathetic tone with a decrease RMSSD and pNN50, which reflects vagal modulation [19]. In our study, the time domain indices were not related to AVNRT. Cardiac autonomic conditions are playing a significant role in supraventricular tachyarrhythmia. The regularity of atrial and ventricular ectopic beats are playing a role in tachycardia, antegrade and retrograde conduction, refractory periods of the AV node and accessory pathways. The association of neurohumoral activation and supraventricular tachycardia has been well documented [7,8]. It

has been proposed that LF NUs is a sensitive marker of sympathetic modulation in controls [20–23], whereas the LF/HF ratio is a good marker for the sympathovagal balance [24]. According to our findings, the increase in LF components during the hour preceding the onset of AVNRT suggests an adrenergic predominance. The fluctuations of autonomic tone occurring before the onset of AVNRT may be underlined by the results of increased LF/HF ratio.

HRV is reduced in patients with diabetic neuropathy, but not in uncomplicated diabetes [25]. Multiple lesions in the atrium (including slow AV nodal pathway and fast AV nodal pathway) primarily affect sympathetic termination and leads to a decrease in HRV [26]. Acute myocardial infarction changes the cardiac geometry leading to an enhanced activity of sympathetic afferent activity and inhibition of vagal efferent activity [27]. Heart rate will increase if low hemoglobin levels are present in the blood. The increase in heart rate causes tachycardia and can trigger AVNRT via atrial ectopic beats [28]. Yo et al. [29] showed that in healthy as well as in hypertensive subject aging affects the LF/HF ratio. We have exclude patients with diabetes, uncontrolled hypertension, documented cardiovascular disease and hematological (including anemia) disorders from our study because these comorbidities may influence the autonomic nervous system.

Conclusion

Heart rate variability analysis has become an important cardiology tool because its measurements are non-invasive and easy to implement. Moreover, relatively good reproducible and provides useful prognostic information for heart patients. This study showed that AVNRT was associated with altered sympathovagal balance. We considered that increased LF and LF/HF might be an indicative of enhanced sympathetic activity in AVNRT patients as a result of inhomogeneous ventricular activation via the slow accessory pathway.

Limitations

In this study, non-pharmacological evaluation of the autonomic tone was performed. Preprocedural hypovolemia, pain, emotional stress could affect autonomic tone also interfere with the HRV. Lack of postablation HRV domain indices were another limitation of the study. This study was single-center design and future studies are required for these findings to be applied for clinical practice.

Conflict of Interest

The author reported no conflict of interest related to this article

Authorship Contributions

Concept/Design: LA

Analysis/Interpretation: LA

Data Acquisition: LA

Writing: LA

Critical Revision: LA

Final Approval: LA

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Tables

Table 1. Baseline clinical and laboratory characteristics of study population

Variables	Control group (n= 90)	AVNRT group (n= 70)	P
Age (years)	46.1±18.1	43.9±16.5	0.418
Gender (male, %)	41 (45.6)	26 (37.1)	0.285
Smoking (%)	39 (43)	43 (61)	0.319
Heart rate 24-h (bpm)	75.5 (58-89)	76.0 (56-98)	0.953
LV-EF (%)	59.2±3.7	58.8±3.9	0.494
Glu (mg/dL)	89.0 (61-175)	90.0 (66-182)	0.865
Cre (mg/dL)	0.75 (0.36-1.5)	0,74 (0.1-1.4)	0.603
TC (mg/dl)	153.3±42.2	156.9±48.0	0.617
TG (mg/dl)	151.0 (43-277)	150.0 (48-242)	0.395
HDL (mg/dl)	34.2±8.2	33.3±9.0	0.520
LDL (mg/dl)	33.0 (22-178)	33.5 (18-142)	0.915
WBC ($10^3 \times \mu L$)	12.6±3.5	11.4±3.8	**0.037**
HGB (g/dl)	13.3±1.9	13.1±2.1	0.527
Plt ($10^3 \times \mu L$)	241.6±91.7	239.1±78.9	0.855

Cre: creatinine, Glu: glucose, HDL: high density lipoprotein, HGB: hemoglobin, LDL: low density lipoprotein, LV-EF: left ventricular ejection fraction, Plt; Platelet, TC: total cholestrol, TG: triglyceride, WBC: white blood cell

Table 2. Comparison of HRV parameters between groups

Daytime HRV time domain indices	**Control group (n= 90)**	**AVNRT group (n= 70)**	**P**
SDNN	114.6 (60,2-298,4)	102.3 (51,2-207,2)	0.052
SDANN	96.5±31.7	92.0±34.4	0.386
PNN50	30.8 (10,0-455,0)	35.4 (20,3-455,0)	0.228
rMSSD	8.5 (1,0-48,0)	10.0 (1,0-55,0)	0.554
Daytime HRV frequency domain indices			
Total power (ms2)	8712.0 (2494,0-46264,0	7694.5 (1764,0-36268,0)	**0.025**
ULF power (ms2)	4581.0 (1188,0-17225,0)	3698.5 (658,0-17225,0)	**0.026**
VLF power (ms2)	2443.0 (634,0-9539,0)	1984.0 (340,0-9010,0)	**0.040**
LF power (ms2)	1122.0 (424,0-9621,0)	1718.0 (166,0-6616,0)	**0.001**
HF power (ms2)	640.0 (198,0-4498,0)	567.5 (59,0-2617,0)	0.350
LF NUs	40.1±10.5	42.1±12.0	0.269
HF NUs	56.0±11.7	54.7±13.7	0.516
LF/HF ratio	1.87±0.85	2.90±1.9	< **0.001**
Nighttime HRV time domain indices			
SDNN	102.1 (45,9-446,2)	94.1 (45,9-205,5)	0.269
SDANN	68.0 (11,4-425,7)	67.0 (26,4-212,9)	0.866
PNN50	44.8 (13,0-143,3)	42.6 (11,0-143,3)	0.957
rMSSD	15.5 (1,0-61,0)	14.0 (1,0-63,0)	0.629
Nighttime HRV frequency domain indices			
Total power (ms2)	7805.0 (2661,0-99334,0)	7303.5 (2661,0-28447,0)	0.510
ULF power (ms2)	2397.5 (689,0-54251,0)	2224.0 (564,0-8135,0)	0.835
VLF power (ms2)	2551.0 (675,0-17361,0)	2231.0 (703,0-13204,0)	0.179
LF power (ms2)	1086.0 (284,0-10702,0)	1761.5 (488,0-10287,0)	< **0.001**
HF power (ms2)	851.0 (235,0-31419,0)	855.5 (163,0-5998,0)	0.916
LF NUs	45.6±11.3	44.6±13.8	0.636
HF NUs	52.9±11.0	89.2±17.6	0.090
LF/HF ratio	1.43±0.8	2.4±1.4	< **0.001**
HF: high frequency, LF: low frequency, LF NUs: low frequnecy normalised units, ULF: ultra low frequency, VLF: very low frequency,			

Table 3. Correlation between AVNRT and variables

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Variables	**Correlation coefficient(r)**	**Significance (P)**
Heart rate 24-h (bpm)	-0.05	0.954
LV-EF	-0.054	0.501
Daytime HRV parameters		
Total power (ms2)	0.177	**0.025**
ULF power (ms2)	0.177	**0.025**
VLF power (ms2)	0.163	**0.040**
LF power (ms2)	-0.256	**0.001**
HF power (ms2)	0.074	0.352
LF NUs	-0.083	0.295
HF NUs	0.070	0.378
LF/HF	-0.348	< **0.001**
Nighttime HRV parameters		
Total power (ms2)	0.052	0.512
ULF power (ms2)	0.017	0.836
VLF power (ms2)	0.107	0.179
LF power (ms2)	-0.463	< **0.001**
HF power (ms2)	0.008	0.917
LF NUs	-0,002	0.982
HF NUs	-0.004	0.962
LF/HF ratio	-0,385	< **0.001**
ULF: ultra low frequency, VLF: very low frequency, LF: low frequency, HF: high frequency, LF/HF: LF/HF ratio, NUs: normalised units		

Table 4. Independent predictors of AVNRT

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Variables	**Multivariate OR, 95% CI**	**Multivariate P**
Heart rate 24-h (bpm)	0.989 (0.945-1.035)	0.634
LV-EF	0.992 (0.879-1.121)	0.903
Daytime total power (ms2)	1.000 (1.000-1.000)	0,999
Daytime ULF power (ms2)	1.000 (1.000-1.001)	0.423
Daytime VLF power (ms2)	0.999 (0.998-1.000)	**0.006**
Daytime LF power (ms2)	1.002 (1.001-1.004)	**0.002**
Daytime HF power (ms2)	0.999 (0.997-1.001)	0.213
Daytime LF/HF ratio	0.990 (0.607-1.616)	0.969
Nighttime LF power (ms2)	1.003 (1.001-1.004)	< **0.001**
Nighttime HF power (ms2)	0.999 (0.998-1.000)	0.080
Nighttime LF/HF ratio	1.541 (0.875-2.717)	0.135
LV-EF: left ventricular ejection fraction, HF: high frequency, LF: low frequency, NUs: normalised units, ULF: ultra low frequency, VLF: very low frequency		