HEART RATE VARIABILITY FROM 24 HOURS VERSUS 72 HOURS HOLTER MONITORING IN PATIENTS WITH MITRAL VALVE PROLAPSE

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ABSTRACT

Objective: To compare recorded heart rate variability from 24 hours with that recorded from 72 hours holter monitoring in patients with mitral valve prolapse.

Study Design: Cross sectional study.

Place and Duration of Study: Department of Clinical Cardiac Electrophysiology Armed Forces Institute of Cardiology/National Institute of Heart Diseases, Rawalpindi from May 2007 to March 2008.

Patients and Methods: Patients from 15 to 38 years of age with confirmed diagnosis of mitral valve prolapse on 2 dimensional echocardiography were included. Patients with acute myocardial infarction (MI), Ischemic heart disease, diabetes mellitus or hypertension were excluded. Total 37 patients were included in the study through non-probability consecutive sampling. All these patients underwent 72 hours holter monitoring using Reynolds medical holter monitors 'life card CF'. Statistical time domain measures of heart rate variability i.e. standard deviation of all NN intervals (SDNN), standard deviation of the averages of NN intervals (SDANN) and square root of the mean of the squares of differences between adjacent NN intervals (RMSSD).

Results: Mean values of SDNN, SDANN and RMSSD from 24 hours holter monitoring were 141.62 ms, 125.16 ms and 28.40 ms whereas those recorded from 48 hours of holter monitoring were 136.94 ms, 122.37 ms and 26.46 ms respectively. Difference between none of the variables from the two recordings was significant.

Conclusion: Heart rate variability remains the same irrespectively of the length of holter monitoring

Keywords: Mitral valve prolapse, Heart rate variability, Ambulatory ECG recording, Holter monitoring

INTRODUCTION

Mitral valve prolapse is the most common valvular heart disease[1]. It refers to systolic displacement of an abnormally thickened, redundant mitral leaflet into the left atrium during systole[2]. With strict diagnostic criteria, prevalence of mitral valve prolapse has been decreased to about 2.4% in general population[3]. It has been associated with cardiac arrhythmias and sudden cardiac death along with other complications like mitral regurgitation, heart failure and bacterial endocarditis[4]. There is a large body of research based evidence suggesting sympatho vagal imbalance in these patients leading to fatal ventricular arrhythmias and sudden cardiac death[5]. Enhanced sympathetic and reduced parasympathetic activity is the hallmark of autonomic imbalance in these patients[6]. Identification of patients at high risk of sudden arrhythmic death remains a challenge for the current cardiovascular research. However, various noninvasive quantitative markers of autonomic activity have been developed for risk stratification[7].

Among the different techniques, heart rate variability (HRV) has emerged as an effective method to assess the sympatho-vagal balance at sinoatrial level[8]. It is an electrocardiographic marker of autonomic imbalance and can be used in such patients to isolate the high risk group[9]. This simple, noninvasive and cost effective method evaluates autonomic nervous system using holter ECG recordings. Heart rate variability is the temporal variation between sequences of consecutive heart beats[10]. Frequent small adjustments in heart rate are made by cardiovascular control mechanisms. This results

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in periodic fluctuations in heart rate. The main periodic fluctuations found are respiratory, baroreflex and thermoregulation bio feedbacks. Respiratory fluctuations, mediated by vagus nerve are the major contributions towards heart rate variability (sinus arrhythmia)[11]. Continuous and reciprocal changes in parasympathetic and sympathetic nervous system lead to fluctuations around mean heart rate. Heart rate variability thus corresponds to oscillations in intervals between heart beats, represented as variable RR intervals on standard ECG[12].

Heart rate variability within normal range represents sympatho-vagal balance with vagal preponderance. Sympatho-vagal imbalance due to decreased vagal and reciprocally increased sympathetic activity leads to reduced heart rate variability[13]. This autonomic imbalance as represented by reduced heart rate variability favors arrhythmogenesis[14]. There is substantial evidence that patients with mitral valve prolapse have higher sympathetic and lower vagal activity leading to reduced heart rate variability. This puts the patients with mitral valve prolapse at high risk of lethal ventricular arrhythmias and sudden cardiac death. These high risk patients can be screened out on the basis of reduced heart rate variability to be put under medical surveillance[5].

Heart rate variability is measured from ambulatory holter ECG recordings. Traditionally,[24] hours holter monitoring had been used to measure heart rate variability[16]. With the advent of digital holter recorders, it has now become possible to get prolonged ambulatory ECG recordings. Holter monitoring for 72 hours can easily be performed with these holters. Some of the digital holters can even record up till seven days in extended mode[17]. This set off a debate in the research arenas of cardiac electrophysiology about the length of holter monitoring to record heart rate variability. Effects of prolonged holter monitoring on heart rate variability in patients with mitral valve prolapse have not yet been studied. Therefore, whether holter monitoring for duration greater than 24 hours will yield better results, remains unanswered. We planned this study to assess the effects of prolonged holter monitoring on heart rate variability in patients with mitral valve prolapse.

The aim of this study was to compare heart rate variability recorded from 24 hours with that recorded from 72 hours holter monitoring in patients with mitral valve prolapse. The study will help in determining the optimal length of holter monitoring to measure heart rate variability.

METHODOLOGY

This cross-sectional study was conducted at Armed Forces Institute of Cardiology/National Institute of Heart Diseases, Rawalpindi from May 2007 to March 2008. Study commenced after obtaining formal approval from medical ethics committee. Written and informed consent was also taken from all the patients. Patients from 15 to 38 years of age with confirmed diagnosis of mitral valve prolapse on 2 dimensional echocardiography were included. Patients with acute MI, ischemic heart disease, diabetes mellitus or hypertension were excluded. Total 37 patients were included in the study through non-probability consecutive sampling. After history and clinical examination,[12] lead ECG was recorded followed by exercise tolerance test to rule out ischemia.Complete blood glucose profile was also obtained to rule out diabetes mellitus. These patients were subjected to ambulatory ECG recording for 72 hours using holter monitors. The holters 'Life Card CF' from Del Mar Reynolds Medical limited was used in this study. After 72 hours of recording, the digital ECG data were transferred from holter recorder to a computer having Pathfinder 700 series software installed. Out of three channels, the one which displayed best ECG recording and with least artifacts was selected. The whole data were edited manually with extreme care using visual checks and manual correction of all QRS complexes. All the erroneous beats were identified and edited from data. After editing, the time domain analysis of heart rate variability was carried out form 24

hours and 72 hours of holter recordings separately. Statistical time domain measures of heart rate variability i.e. SDNN (Standard deviation of all NN intervals), SDANN (Standard deviation of the averages of NN intervals in all 5 minutes segments of the entire recording) and RMSSD (The square root of the mean of the sum of the squares of differences between adjacent NN intervals) were calculated. Statistical analysis was done by using IBM SPSS Statistics version[21]. Descriptive statistics were used to describe the results. Paired sample 't' test was used to compare heart rate variability indices recorded from 24 and 72 hours. A p-value < 0.05 was considered as significant.

RESULTS

Thirty seven patients were included in the study with mean age of 26.27 ± 6.18 years and male to female ratio of 1.6 : 1. The mean values of heart rate variability indices, SDNN, SDANN and RMSSD, recorded from 24 hours and 72 hours holter monitoring are shown in clustered column chart (figure-1). Difference between means of HRV indices recorded from 24 and 72 hours were compared but the difference between the variables from the two recordings was found to be insignificant (Table-1).

DISCUSSION

Results of our study showed that there was no statistically significant difference between HRV recorded from 24 hours and that recorded from 48 hours. Goya-Esteban R et al carried out a study in 22 patients with heart failure whereby they compared heart rate variability indices recorded from 7 days with those recorded from 24 hours[18]. They evaluated various time and frequency domain variables of HRV. Results of their study for time domain parameters of heart rate variability were similar to ours. The heart rate variability index that both the studies shared was SDNN which showed no significant difference between 7 days and 24 hours recording.

Costa O et al compared heart rate variability recorded from short and long term holter ECG recordings but the length of maximum recording was not greater than 24 hours[19]. They reported that heart rate variability was affected

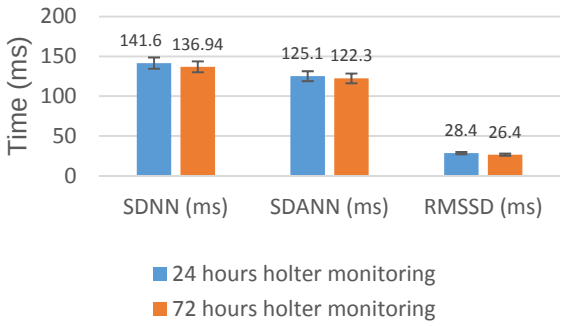

Figure-1: Heart rate variability (HRV) indices after 24 and 72 hours holter monitoring.

Table-1: Comparison of heart rate variability (HRV) recorded from 24 hours holter monitoring with that recorded from 72 hours.

HRV indices	Duration of holter monitoring		p-value
	24 hours recording	72 hours recording	
SDNN (ms)	141.62 ± 30.8	136.94 ± 29.2	0.093
SDANN (ms)	125.16 ± 25.58	122.37 ± 23.0	0.205
RMSSD (ms)	28.40 ± 8.06	26.46 ± 8.40	0.140

Standard deviation of all NN intervals (SDNN), standard deviation of the averages of NN intervals (SDANN) and square root of the mean of the squares of differences between adjacent NN intervals (RMSSD)

significantly by the duration of holter monitoring. Result of their study was contradictory to that of ours. The physiologic basis of the opposing results is that, a substantial part of the long term heart rate variability is contributed by the day and night differences[16]. Heart rate variability determined from holter ECG recordings obtained during day time and night would yield significantly different results. To obtain uniformity in long term time domain heart rate variability values, at least 18 hours of holter

monitoring that includes part of day and night should be carried out[16].

Studies mentioned above including ours provide an insight about the probable mechanism for no difference between heart rate variability recorded from 24 hours and that recorded form longer holter monitoring. One day night cycle is essential and enough to get maximum possible and attainable variability in heart rate. Holter monitoring beyond 24 hours cannot add any further variability to the already achieved heart rate variability[20].

Another explanation of the fact that there is no difference between holter recordings of 24 hours or longer for heart rate variability is the actual mechanism of beat to beat variability. Although cardiac automaticity is intrinsic to various pacemaker tissues, heart rate and rhythm are largely under the control of the autonomic nervous system[21]. Sympathetic nervous system increases heart rate whereas parasympathetic or the vagal system decreases. Under resting conditions, vagal tone prevails and variations in heart rate are largely dependent on vagal modulation[13]. Vagal afferent stimulation, primarily from lungs, leads to reflex excitation of vagal efferent activity and inhibition of sympathetic efferent activity. Efferent sympathetic and vagal activities directed to the sinus node are characterized by discharge largely synchronous with each cardiac cyclewhich can be modulated by central and peripheral oscillators. Central oscillators are vasomotor and respiratory centers and peripheral oscillators are oscillations in arterial blood pressure and respiratory movements. These oscillators generate rhythmic fluctuations in efferent neural discharge which manifest as heart rate variability. Within about 24 hours the unusual variations in heart rate including day/night cycle are averaged out and overall uniform heart rate variability is achieved[11]. Therefore, prolonging the duration of monitoring will only be an added burden for the patient and will not have any significant effect on HRV indices.

CONCLUSION

Heart rate variability recorded from 24 hours holter monitoring provides maximum possible information and longer recordings are not required. That mean heart rate variability remains the same irrespectively of the length of holter monitoring. This information will not only provide relief to the patients but will also remove extra and unnecessary burden from health care facilities.

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