

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Influence of Higher Respiratory Rate on SDNN Heart Rate Variability and Heart Rate in Healthy Subjects.

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ABSTRACT

Reduced standard deviation of R-R intervals (SDNN), elevated heart rate (HR) and respiratory rate (RR) are noted in poor prognosis of cardiovascular disease. But is there association between these three parameters is not clear. To investigate the influence of RR on HR and SDNN in healthy subjects. In 10 healthy subjects, SDNN and HR was calculated from electrocardiogram tracing recorded from lead II in supine position while breathing at 12, 15 and 20 respiratory cycles/per minute for one minute duration. Statistical analysis was done by ANOVA/ Kruskal –Wallis test followed by multiple comparisons. Association between two continues variables were measured by Pearson Correlation Coefficient test. p value <0.05 considered significant. HR was higher while breathing at 20 cycles compared to 12 cycles/minute ($p < 0.05$). There was no difference in HR while breathing at 15 cycles compared to 12 and 20 cycles/ minute. There was no significant difference SDNN while breathing at 12, 15 and 20 cycles / minute. There was no correlation between HR and SDNN while breathing at 12, 15 and 20 cycles/minute. Higher respiratory rate may influence heart rate but not SDNN

Keywords: Respiratory rate, heart rate, SDNN

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INTRODUCTION

Autonomic nervous system plays a crucial role in maintaining homeostasis and normal functioning of the viscera [1]. Normal functioning of the autonomic nervous system in turn is assessed by indices of autonomic function namely heart rate variability [2]. Among the various heart rate variability parameters, reduced standard deviation of normal R-R intervals (SDNN) either derived from 24 hour ambulatory electrocardiogram [3] or derived from as short as 10 seconds electrocardiogram [4] is reported to predict all-cause mortality. SDNN is also reported to predict survival in post myocardial infarction patients [5]. Higher heart rate and respiratory rate are commonly observed in patients with acute and post-myocardial infarction patients [6- 7]. However it is not clear that weather SDNN is influenced by higher respiratory and heart rate. Finding the association of SDNN with heart rate and respiratory rate may provide better insight into autonomic control of heart in health and disease. Therefore this study was undertaken to assess the influence of higher range of respiratory rate on SDNN and heart rate.

MATERIALS AND METHODS

Subjects

Ten healthy young adults aged between 22-24 years of either sex (5 males and 5 females) who volunteered to take part in the study were included.

In them heart rate and standard deviation of R-R intervals were quantified during controlled breathing at the rate of 12, 15 and 20 breaths per minute. Parameters considered were standard deviation of R-R intervals (SDNN) and heart rate.

Procedures

All the study procedures were done in the morning between 10-11 AM in all the subjects. Before beginning the test procedure subjects were made clear about the study protocol. ECG was recorded in recumbent position for one minute during controlled breathing in the following rate of respiration. Recording of ECG was initiated for each respiratory rate after providing minimum of 5 minutes rest (that is after each respiratory maneuver).

- Breathing at 12 respiratory cycles/ minute
- Breathing at 15 respiratory cycles/minute
- Breathing at 20 respiratory cycles/ minute

Assessment of SDNN and heart rate

SDNN was estimated from one minute resting lead II ECG. All of the R–R intervals were measured from one minute resting electrocardiogram was computed. SDNN was estimated with suitable statistical functions using Microsoft Windows XP Professional (Microsoft Corporation, Redmond, WA, USA) [8] . All the R-R intervals counted during one minute was considered as heart rate

Statistical Analysis

As data on standard deviation of R-R intervals was highly skewed it was log transformed. Data was then analyzed by ANOVA followed by Tukey -Kramer multiple comparison test. When data was not uniformly distributed, non-parametric equivalent of ANOVA namely Kruskal –Wallis test was performed. Level of significance was tested by two tailed test. Statistical significance taken to be at $p < 0.05$.

RESULTS

The average age of the study subjects was 20.07 ± 0.70 years. In all the study subjects systolic and diastolic blood pressure was within physiological limits. The systolic blood pressure ranged from 110 mmHg to 130mmHg and the diastolic blood pressure ranged from 70 mmHg to 80 mmHg. The mean systolic blood pressure was 119.09 ± 7 mm Hg and the mean diastolic blood pressure was 77.27 ± 4.67 mmHg.

The data on comparison of heart rate and SDNN while breathing at 12, 15 and 20 respiratory cycles per minute is presented in table 1. The correlation between heart rate and SDNN while breathing at 12, 15 and 20 respiratory cycles per minute is presented in table 2.

The mean heart rate was significantly different while breathing at three different rates of respiration ($F = 3.711, p = 0.037$, table 1). There was significant increase in heart rate during breathing at 20 respiratory cycles per minute compared to breathing at 12 respiratory cycles per minute ($q = 3.853, p < 0.05$, table 1). There was no significant difference in heart rate while breathing at 20 respiratory cycles per minute compared to 15 respiratory cycles per minute ($q = 1.94, p > 0.05$). Heart rate was not significantly different while breathing at 15 respiratory cycles per minute compared to 12 respiratory cycles per minute ($q = 1.907, p > 0.05$, table 1). SDNN did not differ significantly while breathing at 12, 15 and 20 respiratory cycles per minute ($KW = 4.622, p = 0.099$, table 1).

There was no significant correlation between SDNN and heart rate during breathing at 12, 15 and 20 respiratory cycles per minute (table 2).

Table1. SDNN and heart rate during three different rates of respiration
(Values are mean \pm SD)

variables	12 cycles/minute	15 cycles /minute	20 cycles/minute
SDNN (milliseconds)	44.36 \pm 7.86 (1.64 \pm 0.08)	53.79 \pm 23.6 (1.69 \pm 0.18) ^{NS}	63.4 \pm 18.34 (1.78 \pm 0.13) ^{NS}
Heart rate (beats/min)	71.10 \pm 5.70	76.10 \pm 9.19 ^{NS}	81.2 \pm 9.43 ^{*NS}

* $P < 0.05$ compared to 12 respiratory cycles/minute; NS= non-significant compared to other groups; sample size (n) = 10; log transformed values of SDNN in parenthesis

Table 2. Correlation between SDNN and heart rate during three different rates of respiration.

variables	Correlation coefficient (r value)	P value
At 12 respiratory cycles / minute	- 0.4114	0.237
At 15 respiratory cycles / minute	0.28	0.42
At 20 respiratory cycles / minute	- 0.0913	0.801

DISCUSSION

Heart rate and SDNN a heart rate variability parameter were investigated in 10 volunteers while doing controlled respiration at three different respiratory rates for one minute. In the present study, respiration had a considerable effect on the heart rate but not on SDNN. And there was no correlation between heart rate and SDNN in all the three respiratory rates of controlled breathing. Thus SDNN values derived from one minute electrocardiogram may reflect mainly cardiac autonomic modulation guided by the underlying physiological or pathological condition independent of prevailing rate of respiration.

In the present study, it was observed that heart rate increases significantly while breathing at 20 respiratory cycles per minute compared to 12 respiratory cycles per minute (table 1). There are important effects of respiration on the heart and circulatory system. In the central nervous system there are interactions between “pacemakers” and efferent autonomic tone which directly affect heart rate. There are also variations in vagal tone that centrally mediated and related to respiration [9]. Altered respiratory pattern may also influence chemical composition of the blood and trigger chemoreceptor reflex leading to increase in heart rate [10]. Thus we speculate that increasing the respiratory rate from 12 cycles 20 cycles per minute must have reduced the vagal tone on heart either mediated through the respiratory center or chemoreceptor triggered reflex rise in heart rate.

In the present study, heart rate did not differ significantly while breathing at 15 cycles per minute compared to 12 cycles per minute. Our findings is in agreement with the findings of Maria Vittoria Pitzali et al who too had found no difference in heart rate while breathing at 14 cycles per minute compared to 10 beats per minute [11]. However their study did not investigate the influence of breathing at 20 cycles per minute. In the present study breathing at 20 cycles caused significantly higher heart rate compared to breathing at 12

cycles per minute. At rest, a normal human breathes 12-15 times a minute [12]. Nonetheless, breathing at 20 respiratory cycles per minute is considered as the upper limit of normal range of respiratory rate [13-14]. Thus it appears that in the upper range of respiratory rate, increased heart rate may be a cardiovascular adjustment required to meet the bodily demands.

CONCLUSION

Respiratory rate may influence heart rate but not standard deviation of RR intervals obtained during one minute respiratory maneuvers.

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