

## Comparative study of parameters of Heart Rate variability in smoker and non-smoker

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**Abstract-**In this article, a comparative study is presented to observe how smoking affects the heart of a human being in adolescence. To conduct this experiment, 30 healthy smokers and non-smokers between the ages of 19 to 23 had undergone an electrocardiograph (ECG) followed by a Heart Rate Variability (HRV) analysis of the ECG reports. It was found that many HRV time domain parameter along with the successive heartbeat intervals differed by at least 50ms (NN50) values of smokers and non-smokers vary distinctively. Also the mean of the RR distance of ECG signals (Mean RR) exhibits clear variation for smokers and nonsmokers.

**Keywords** -Smokers; Non Smokers; ECG; HRV; Mean RR; NN50

### 1. INTRODUCTION

Smoking is one of the most vital causes for cerebrovascular stroke, coronary artery diseases, and peripheral vascular diseases, furthermore a general relation has been found that smoking elevates the chance of myocardial infarction.[1-6] Smoking can also bring about serious damaging effects in the brain, immune functioning, gastrointestinal system, cardiovascular system, and the respiratory systems.[7-12] The World Health Organization estimates that tobacco kills nearly 6 million people each year, with more than 600,000 passive smokers, via heart disease, lung cancer, and other illnesses and the number can increase up to 8 million by 2030 if the current trends continue.[13-19] Unfortunately, even after raising extensive public awareness of the harmful effect of smoking, the number of smokers is increasing day by day and enormous number of young students can be seen in that list.

Majority of the organs of the human body, including the heart, are under the orchestration of the Autonomic Nervous System (ANS). There are numbers of tests to ascertain the autonomic activities of heart and among them the HRV analysis is widely accepted. Heart Rate Variability (HRV) is commonly defined as the phenomenon of variation in time interval of consecutive heartbeats which is evaluated by noting the variation in beat-to-beat interval. It is the level change of the heart rate under the balanced standing of parasympathetic and sympathetic

component of the cardiac ANS and it also manifests the stage of neuronal detrimental effects on ANS.[20,21] Diagnostic tools such as blood pressure, electrocardiogram (ECG) and also ballistocardiogram are often employed to carry out HRV analysis.

Sinnreich et al. (1998), Jáuregui-Renaud et al. (2001), Carrasco et al. (2003), Sandercock et al. (2005), Tannus et al. (2013) and others calculated HRV indices on the basis of short-term (5–7 min) stable ECG recordings in healthy young adults. However, the authors of these studies failed to consider an interaction between HRV and average heart rate which in turn was performed by Sacha et al. (2013), Monfredi et al. (2014), Billman et al. (2015) etc. HRV being primarily dependent on heart rate, different heart rates may exert various impact on HRV values- as evaluated by Sacha and Grzeszczak (2001), Monfredi et al. (2014), Stauss (2014), Gąsior et al. (2015), among others. So, an adequate correction designed to remove the HR influence on HRV should be performed before drawing ultimate conclusions about HRV corresponding to different heart rates (Sacha and Pluta (2008), Monfredi et al. (2014), Billman et al. (2015) [22, 23]. Also, Bruce (1996), Billman (2011), Quintana and Heathers (2014), and Quintana et al. (2016) suggested that breathing [24] or more precisely the respiratory frequency might significantly modify the HRV [25]. Now the Heart Rate Variability (HRV) gives an indication of the variation in the sinoatrial node as a result of the changes in the interaction between the vagus nerve and the sympathetic nervous

system [26]. Moreover the effects of secondhand smoke on the autonomic nervous system of non-smokers can be measured using microneurography and HRV and both of these have confirmed elevated sympathetic nerve activities during secondhand smoke [27, 28].

The aim of this research paper is to analyze the HRV parameters for smokers and nonsmokers, also to find out a how these parameters vary for smoker and non-smokers.

**2. METHOD AND MATERIALS**

In this paper, HRV analysis was done of 30 healthy subjects who were grouped into two categories- smokers (Test group 1) and non-smokers (Test group 2). None of the subjects had any major illness like endocrinal disorders, chronic respiratory illness, diabetes mellitus, cardiac diseases etc. All the chosen subjects were in the age group 19-23 years. The 15 smokers were noted to have been smoking for a period of 3-10 years. For the HRV analysis, a 5 minute ECG was taken with the help of an ECG machine and LabVIEW software and analyzed via Kubios software which is a well-recognized tool for heart rate variability analysis in time domain and in frequency domain. For further investigation, 9 important parameters in time domain were considered to establish the variations that can be appeared, for smoking, in HRV report. The parameters are 1) Mean RR\* - it is the mean of successive R-R intervals in

QRS complex of the electrocardiography, 2) STD RR of SDNN- it is the standard deviation of processed normal beats (NN) which can be represented as the total variability of the heart rate, 3) Mean HR\*- this denotes the average heart rate, 4) STD HR- it is the standard deviation of the Heart Rate, 5) RMSSD- The square root of the average of the sum of the squares of differences between adjoining NN interim, 6) NN50- this means the number of pairs of the adjacent NN that differ by more than 50 ms, 7) pNN50- it is the percentage of the ratio of NN50 to the total number of NN intervals, 8) RR triangular index- this is a geometrical measures of total number of the RR intervals and divided by the height of the histogram of all RR intervals with the bins of 1/128s, and 9) TINN – it is another geometrical measurement where a triangular interpolation of the discrete distribution of RR intervals that is the histogram counts is used [29].

**3. RESULTS AND DISCUSSIONS**

Table 1a lists the values of 9 parameters including Mean HR, Mean RR, pNN50, TINN etc. as obtained from HRV analysis of the 15 smokers whose ECG was carried out. From Table 1a 9 parameters were observed and analyzed for each of the smokers to comprehend the variation. The average values of all 9 parameters in the table are marked and accentuated below.

Subject	Mean RR* (ms)	STD RR(SDNN) (ms)	Mean HR* (1/min)	STD HR (1/min)	RMSSD (ms)	NN50 (count)	pNN50 (%)	RR triangular index	TINN (ms)
Subject 1	787.9	72.2	76.82	7.46	61.9	115	47.3	14	340
Subject 2	730.7	49.6	82.5	5.72	35.8	27	10.3	11	245
Subject 3	781.7	52.2	77.1	5.26	38.9	77	20.1	16	270
Subject 4	660.9	60.3	91.52	8.09	35.9	62	13.6	11	330
Subject 5	677.4	56	84.3	7.1	31	23	8.9	13	245
Subject 6	678.2	84.2	89.64	9.87	73	67	23.7	18	495
Subject 7	698.1	58.5	86.53	6.95	32.5	25	9.1	13	240
Subject 8	695.7	34	86.45	4.16	23.9	9	2.1	9	155
Subject 9	729	87.3	83.46	9.87	58.3	97	23.4	20	480
Subject 10	659.7	28.3	91.11	3.93	21.4	6	1.3	9	130
Subject 11	761.6	54.2	79.16	5.36	38.3	43	17.1	12	255
Subject 12	611.6	35.6	98.42	5.58	40.3	135	27.1	8	165
Subject 13	683.3	44.6	88.19	5.8	21.4	4	0.9	10	205
Subject 14	810.9	57	74.35	5.09	51.4	98	26.4	13	260
Subject 15	603.3	33.4	99.76	5.44	23.7	14	4.4	9	170
<b>Average</b>	<b>706.6</b>	<b>53.67</b>	<b>86.07</b>	<b>6.33</b>	<b>39.18</b>	<b>55.6</b>	<b>17.4</b>	<b>12</b>	<b>265.67</b>

**Table 1a-HRV analysis parameters for Test group 1 (Smokers)**

Table 1b shows the values of the same 9 parameters calculated by HRV analysis for 15 non-smokers. Similarly the Mean RR\*, STD RR (SDNN), Mean HR\*, STD HR, RMSSD pNN50 etc. were scrutinized

for each of the 15 subjects and average values were obtained to find a general relationship of the HRV analysis.

Subject	Mean RR* (ms)	STD RR(SDNN) (ms)	Mean HR* (1/min)	STD HR (1/min)	RMSSD (ms)	NN50 (count)	pNN50 (%)	RR triangular index	TINN (ms)
Subject 1	653.4	41	92.18	5.69	23.1	18	3.9	11	195
Subject 2	999.7	94.5	60.58	6.05	93.2	151	49.7	18	235
Subject 3	660.9	60.3	91.52	8.09	35.9	62	13.6	11	330
Subject 4	983.5	179.1	63.15	12.01	173.5	232	68.8	31	425
Subject 5	769	54.2	78.42	5.64	54.1	145	36.9	11	260
Subject 6	549.7	47.8	109.92	8.8	33	50	9.1	8	210
Subject 7	809.1	75.9	74.83	7.21	60.7	135	36.3	18	360
Subject 8	571	20.5	105.21	3.85	8	11	2.7	6	95
Subject 9	695.3	95	87.86	11.59	43.5	83	19.2	19	390
Subject 10	790.6	123.2	77.5	10.58	103.2	136	35.6	20	415
Subject 11	515.3	17.6	116.58	4.04	10.7	10	3.1	5	90
Subject 12	854.7	64.6	70.6	5.3	53.1	156	44.2	17	325
Subject 13	632.2	40.3	95.27	5.84	22.1	12	2.5	9	220
Subject 14	797.2	75.6	76.17	10.66	88.3	125	33	10	495
Subject 15	850.2	44.2	70.76	3.63	43.90	86	24.2	11	215
<b>Average</b>	<b>748.5</b>	<b>70.91</b>	<b>84.17</b>	<b>7.38</b>	<b>58.8</b>	<b>94.13</b>	<b>25.52</b>	<b>14</b>	<b>290.36</b>

Table 1b- HRV analysis parameters for Test group 2 (Non-smokers)

For graphical comparison the average values of all the above mentioned parameters of both Test group 1 and Test group 2 have been put into a bar plot labeled Fig: 1. From this graphical implementation it the variations of different parameters for smokers and non-smokers can be identified. Here it can be clearly

observed that time domain HRV parameters such as Mean RR\*, NN50, RMSSD, TINN etc. of smokers and non-smokers exhibit distinguishable variation whereas Mean HR, RR triangular index etc. do not vary much for smokers and non-smokers.

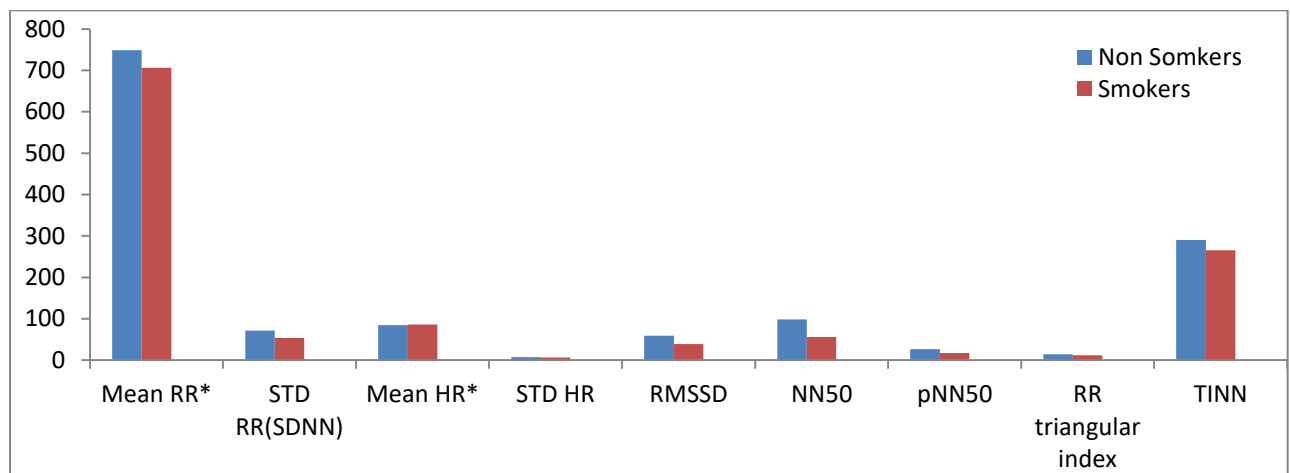


Fig 1: Graphical Representation of different statistical parameters

4. CONCLUSION

Thus it is evident that a close study of the HRV parameters will generate ample information about the normal or abnormal functioning of a subject's heart and also lungs. These parameters, particularly pNN50, TINN, Mean RR, and RMSSD etc. show considerable

variations in smokers and non-smokers and thus detailed analyses will help us to determine disease conditions and abnormalities in a patient.

## 5. FUTURE SCOPE

In future we plan to use our findings and draw a comparison between the Peak Expiratory Flow Rate (PEFR) of smokers and nonsmokers. We will then be able to draw more specific conclusions regarding the heart-lung condition of the patient.

## REFERENCES

- [1] Sandhu RK; Jimenez MC; Chiuvè SE; et al. (2012): Smoking, smoking cessation and risk of sudden cardiac death in women. *CircArrhythmElectrophysiol*, 5:1091-7.
- [2] Nikolozakes AW; Binkley PF; Leier CV (1988): Hemodynamic effects of smoking in congestive heart failure. *Am J Med Sci* 1988, 296: 377-80.
- [3] World Health Organization (WHO) (2012) Mortality attributable to tobacco.
- [4] Harte C; Meston C (2014): Effects of smoking cessation on heart rate variability among long-term male smokers. *Inter J Behav Med*, 21: 302-309.
- [5] Munjal S; Koval T; Muhammad R; Jin Y; Demmel V; et al. (2009): Heart rate variability increases with reductions in cigarette smoke exposure after 3 days. *J CardiovascPharmacolTher*, 14: 192-198.
- [6] Reddy KS; Gupta PC (eds) (2004): Report on tobacco control in India, New Delhi, Ministry of Health and Family Welfare, Government of India.
- [7] Lauer M; Pashkow FJ; Larson MG; Levy D (1997): Association of cigarette smoking with chronotropic incompetence and prognosis in the Framingham Heart Study. *Circulation*, 96: 897-903.
- [8] Sidney S; Sternfeld B; Gidding SS; et al. (1993): Cigarette smoking and submaximal exercise test duration in a biracial population of young adults: the CARDIA study. *Med Sci Sports Exerc.*, 25: 911-916.
- [9] Vriz O; Nesbitt S; Krause L; Majahalme S; Lu H; Julius S (1997): Smoking is associated with higher cardiovascular risk in young women than in men: the Tecumseh Blood Pressure Study. *J Hypertens.*, 15: 127-134.
- [10] Srivastava R; Blackstone EH; Lauer MS (2000): Association of smoking with abnormal exercise heart rate responses and long-term prognosis in a healthy, population-based cohort. *Am J Med.*, 109: 20-26.
- [11] Lauer MS (2004): Chronotropic incompetence: ready for prime time. *J Am CollCardiol.*, 44: 431-432.
- [12] Astrand PO; Rodahl K; Dahl HA; Stromme SB (2003): Textbook of work physiology-Physiological basis of Exercise. Champagne, IL: Human Kinetics, pp.134-176.
- [13] Shalnova S; Shestov DB; Ekelund LG; Abernathy JR; Plavinskaya S; Thomas RP; et al. (1996): Blood pressure response and heart rate response during exercise in men and women in the USA and Russia lipid research clinics prevalence study. *Atherosclerosis*, 122: 47-57.
- [14] Skinner JS; Gaskell SE; Rankinen T; et al. (2003): Heart rate versus %VO<sub>2</sub>max: age, sex, race, initial fitness, and training response—HERITAGE. *Med Sci Sports Exerc.*, 35: 1908-1913.
- [15] Ekelund LG; Haskell WL; Johnson JL; Whaley FS; Criqui MH; Sheps DS (1988): Physical fitness as a predictor of cardiovascular mortality in asymptomatic North American men. The Lipid Research Clinics Mortality Followup Study. *N Engl J Med*, 319: 1379-1384.
- [16] Savonen KP; Lakka TA; Laukkanen JA; Rauramaa TH; Salonen JT; Rauramaa R (2007): Effectiveness of workload at the heart rate of 100 beats/min in predicting cardiovascular mortality in men aged 42, 48, 54, or 60 years at baseline. *Am J Cardiol*, 100: 563-568.
- [17] Kinoshita M; Herges RM; Hodge DO; et al (2009): Role of smoking in the recurrence of atrial arrhythmias after cardioversion. *J Am CollCardiol*, 104: 678-82.
- [18] Sandhu RK; Jimenez MC; Chiuvè SE; et al. (2012): Smoking, smoking cessation and risk of sudden cardiac death in women. *CircArrhythmElectrophysiol*, 5:1091-7.
- [19] Nikolozakes AW; Binkley PF; Leier CV (1988): Hemodynamic effects of smoking in congestive heart failure. *Am J Med Sci*, 296: 377-380
- [20] Holly R. Middlekauff; Jeanie Park; Royaand S. Mohemani: "Adverse Effects of Cigarette and Non cigarette Smoke Exposure on the Autonomic Nervous System", journal of the American College of cardiology, issn: 0735-1097, vol. 64, no. 16, pp-1740-50.
- [21] Christopher B. Harte & Cindy M. Meston (2014): "Effects of Smoking Cessation on Heart Rate Variability among Long-Term Male Smokers", *Int.J. Behav.Med.*, 21:302-309.
- [22] Joshil Kumar Behera; Sushma Sood; Naresh Kumar; Kirti Sharma; Reshmi Mishra; Prasanta Saha Roy (2013): "Heart Rate Variability and its Correlation with Pulmonary Function Test of Smokers", Jan-Mar 13 Issue 1, Vol 14, pp-22-25.
- [23] Christopher B. Harte; Gabrielle I. Liverant; Denise M. Sloan; Barbara W. Kamholz; Laina E. Rosebrock; Maurizio Fava & Gary B. Kaplan (2013): "Association Between Smoking and Heart Rate Variability Among Individuals with Depression", *Ann. behav. Med.*, Vol-46, pp-73-80.
- [24] Pranay Swarnkar; Narendra Kumar; Kamy Verma and Sunny Goel (2013): "Effects of Active Smoking on Heart Rate Variability, Heart Rate & Various Other Cardiac Risk Events in Chronic

- Smokers”, *International Journal of Physiology*, Vol 1, No 2, pp-77-81.
- [25] Zdravkotaralov; Peter Dimov; Kirilterziyski; Ilchoilchev and Stefankostianev (2015): “The effect of smoking on the autonomic heart regulation in young “healthy” male smokers”, *Journal of IMAB - Annual Proceeding (Scientific Papers)*, vol. 21, issue 1, pp-718-721.
- [26] CosioPiqueras MG; Cosio MG (2001): Disease of the airways in chronic obstructive pulmonary disease. *EurRespir J Suppl*, 34:41s-49s.
- [27] Dinas PC; Koutedakis Y; Flouris AD (2013): Effects of active and passive tobacco cigarette smoking on heart rate variability. *Int J Cardiol*, 163: 109-15
- [28] Pope CA 3<sup>rd</sup>; Eatough DJ; Gold DR; et al. (2001): Acute exposure to environmental tobacco smoke and heart rate variability. *Environ Health Perspect*, 109: 711-6.
- [29] Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (March 1996): "Heart rate variability. Standards of measurement, physiological interpretation, and clinical use". *European Heart Journal*. 17 (3): 354–81.