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A COMPARISON OF HEART RATE VARIABILITY IN SMOKERS AND NON SMOKERS

Physiology

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ABSTRACT

Smoking is an escalating public health problem especially in developing countries like India.[1] Studies have shown that smoking increases the risk of myocardial infarction and sudden cardiac death. One of the mechanism by which smoking impairs the cardiovascular function, is its effect on autonomic nervous System control. There are several tests to determine the autonomic activity. Recently the most accepted tool is determining Heart Rate Variability. It measures the variation in the sinoatrial node due to sympathovagal change[2].This study intends to evaluate the autonomic functions in smokers and non smokers. This is a case control study having a total of 140 male subjects – 70 smokers (cases) and 70 non smokers (controls) in the age group of 20-50 years. Electrocardiography (ECG) was recorded in a quiet room with the subject in supine position after 10 minutes of rest. Analog ECG signal was converted to digital. Following parameters were taken – LF(nu), HF(nu), LF/HF. Results were compared with the help of Student t test and the level of significance was fixed at $p < 0.05$. There was significant decrease in HF ($p<0.05$) and significant increase in LF ($p<0.05$), LF/HF ($p<0.05$) in cases when compared to controls. This signifies higher sympathetic activity and decreased parasympathetic activity in smokers when compared to non smokers. The present study showed that smoking in males may affect the cardiac autonomic activity. There was a shift in the sympatho vagal balance towards sympathetic predominance among smokers when compared to non-smokers.

KEYWORDS

Heart rate variability, LH nu, Hf nu.,sympathovagal balance

Introduction:

Smoking is a major risk factor for coronary artery diseases, cerebro vascular stroke, peripheral vascular diseases [3] and there is a general agreement that smoking increases the risk of myocardial infarction and sudden cardiac death [4]. Harmful effects of smoking appear at an early age, seriously affecting the brain, cardiovascular system, gastrointestinal system, immune functioning, and the respiratory systems [5].Cessation of smoking is associated with reduced cardiovascular mortality and morbidity [6]. In India, smoking is less prevalent among females and for majority of whom it is also a taboo. So in the present study, we have included only male smokers.

Imbalance in cardiac autonomic activity might be a predisposing factor for arrhythmogenesis and subsequently sudden cardiac deaths [7]. Cigarette smoking increases the risk of cardiovascular events related with several mechanisms. One of the mechanism by which smoking impairs the cardiovascular function is its effect on Autonomic Nervous System [8]. There are several tests to determine the cardiac autonomic activity. Recently the most accepted tool is determining Heart Rate Variability (HRV) [9]. Therefore the present study was undertaken mainly to assess the effect of smoking on cardiac autonomic function using HRV.

Heart Rate Variability is a specific and sensitive non-invasive tool to evaluate cardiac autonomic activity. HRV is the degree of variation of the heart rate during the day under the balanced influence of sympathetic and parasympathetic component of the cardiac autonomic nervous system. It expresses the total amount of variation of both instantaneous heart rate and RR intervals. HRV also indicates the extent of neuronal damage to autonomic nervous system.

HRV has been shown to be a good tool to quantify the tone of autonomic nervous system to the myocardium. It has also been associated with high predictive value in many diseases where a disturbance in the autonomic activity is likely.[10-12]

This study is an effort to assess the cardiac autonomic activity using Heart Rate Variability in smokers.

Materials and methods

This study was conducted in SMS Medical College and Hospital, Jaipur. This is a comparative study having 70 male smokers in the age group of 20-50 yrs, smoking for at least 5 years as cases . BMI and sex matched non smoker subjects as controls (table 2). Clearance for the study protocol was obtained from institutional ethical committee. Subject's clinical history and details were taken according to the standard Performa. Informed written consent was taken from all the study subjects. The study involved non invasive procedures and was performed at room temperature.

Subjects with history of major illness like diabetes mellitus, hypertension, chronic respiratory illness, cardiac diseases and endocrinal disorders, subjects on any drugs affecting the functioning of Autonomic Nervous System- like alpha blockers , beta blockers and others which might have an effect on the Autonomic Nervous System and with BMI >25 kg/m2 were excluded from the study.

Weight and height were measured using standard calibrated instruments. Procedure was done between 9:00 AM to 12:00PM. All the subjects abstained from meals, alcohol or caffeine-containing beverages& any energetic physical activity for 4 hours prior to the examination, while smokers were asked not to smoke for 8 hours at least prior to the examination. Patient was explained in detail about the ongoing procedure and ECG was digitally recorded Heart Rate Variability software (version 1.1), was used in the computer, to detect the peak to peak intervals and further mathematical and analytical calculations in order to get the values of the parameters.

The patient was made to lie down for 10 minutes and allowed to relax and then the limb electrodes were placed on to the respective limbs along with ECG gel. The chest leads were not used & ECG was recorded in lead II only, as the requirement for the procedure was only peak detection for the determination of RR intervals. First, a 5 minutes ECG in supine position was recorded, with subject respiring normally], LF nu (Low Frequency component, where nu means statistically normalized units. This mainly signify sympathetic component), HF nu (High Frequency component This signify parasympathetic component) and LF/HF (Ratio of Low Frequency component to High Frequency component which signify the sympathovagal balance.). Then, the person was periodically instructed to take alternating 5 seconds of deep inspiration and 5 seconds of deep expiration for a period of 2 minutes and simultaneous ECG was recorded. This data was used to measure the E/I ratio (Expiratory RR interval to Inspiratory RR interval ratio which mainly signify parasympathetic component.) [9].

Statistical analysis

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements were presented on Mean SD (Min-Max) and results on categorical measurements were presented. Significance was assessed at 5 % level of significance (p value <0.05). Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups Inter group analysis) on metric parameters in Number (%).

Significant figures
+ Suggestive significance (p value< 0.05)
* Moderately significant (p value:0)
** Strongly significant (p value <0.001)

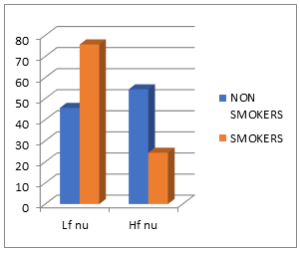
Results and discusion The major findings of this study there was a significant decrease Hfnu and increase in the values of LF nu and LF/HF ratio in smokers (p< 0.05) when compared to non smokers.

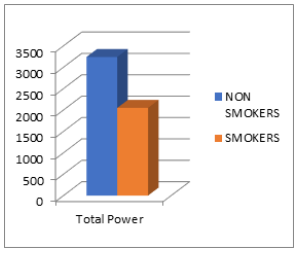
TABLE -1

	NON SMOKERS		SMOKERS		
	Mean	S D	Mean	SD	P Value
Total Power	3240	489	2050.9	435	<0.05
Lf nu	45.6	.98	75.68	3.7	<0.05
Hf nu	54.4	1.2	24.32	3.67	<0.05
Lf/Hf ratio	.84	.05	3.22	.82	<0.05

TABLE-2

	Non smokers		Smokers		
	Mean	SD	Mean	SD	P Value
Age(yrs)	33.4	8.79	33	8.6	>.05
Height(cms)	166.94	7.15	168.3	6.04	>.05
Weight(kg)	63.99	11.22	65.89	8.67	>.05
BMI(kg/cm2)	22.85	2.93	23.31	3.39	>.05





This study was designed to evaluate the autonomic function in cigarette smokers and non smokers using HRV. All the study subjects were age, BMI matched.

In the present study the following HRV parameters were estimated frequency domain analysis parameters like LF nu, HF nu and LF/HF in smokers and non smokers in the age group of 20-50 years.

The major findings of this study were, smokers showed significant reduction HF nu which are considered as a measure of parasympathetic activity and showed a significant increase in the values of LF nu (which is considered as a measure of sympathetic activity) and LF/HF ratio (which signifies the sympathovagal balance)when compared to non-smokers.

This observation is in agreement with experimental data which showed that smoking resulted in acute modifications in the HRV indices, characterized by a decrease in the parasympathetic activity and increase in the sympathetic activity [13].

The acute effect of smoking is mainly due to nicotine while the reduction in the cardiac vagal tone is responsible for chronic effects.(14)

Furthermore, it has been shown that Smoking impairs the sympathovagal balance which would probably contribute to a higher rate of cardiovascular events [15]

As for as the mechanisms responsible for the sympatho excitatory effects are concerned, Shinozaki N, et al. showed that in healthy subjects, cigarette smoking has been found to increase HR, BP, and the plasma concentration of nor epinephrine. These changes are attenuated markedly by alpha-adrenergic and beta-adrenergic blockade, indicating that these hemodynamic effects of cigarette smoking are derived from sympathetic activation. However, the mechanisms of cigarette smoking-mediated sympatho excitation are unclear.

They assume 3 different mechanisms underlying activation of the sympathetic nervous system due to cigarette smoking. First, a direct effect on the central nervous system; second, a stimulatory effect on ganglionic sympathetic transmission that leads to a subsequent increase in postganglionic efferent sympathetic nerve activity; and third, a direct effect on peripheral sympathetic nerve endings. It has been shown that cigarette smoking stimulates the release of catecholamine directly from postganglionic peripheral sympathetic nerve endings. However, the mechanism of the effect of cigarette smoking on the central nervous system is still controversial [16]. A statically significant difference found in the R-R interval between test and control groups. Niedermaier et al.found a reduced heart rate in smokers compared to non-smokers(17)

Thus, our study proves that there is strong impairment of parasympathetic activity with elevated levels of sympathetic activity in healthy smokers when compared to non smokers. And this has led to definite shift in the sympatho vagal balance towards sympathetic component.

Therefore smokers are prone for cardiovascular risk. So encouraging abstinence from smoking, in early stages convert back the cardiovascular changes to nearly normal or in more severe cases to recovery with little residual damage to the heart.

Conclusion: In this study, HRV parameters i.e. Frequency domain were compared between 70 non smokers and 70 smokers. At the end of the study, the following conclusions can be drawn.

There was a demonstrable decrease in parasympathetic activity in smokers when compared with age and BMI

An increase in sympathetic activity and decrease in parasympathetic activity tilted the balance of autonomic functions to sympathetic predominance among the smokers.

REFERENCES

[1] Reddy KS, Gupta PC (eds). Report on tobacco control in India, New Delhi, Ministry of Health and Family Welfare, Government of India; 2004)
[2] Pride NB,Burrows B.Developmentof impaired lung:Natural history and risk factors. In: Calverley PM,Pride NB,editors.Chronic obstructive pulmonary diseae. London: champam Hall;1995.pp.69-92.
[3] Dwivedi S, Sanjeev Sharma. JIACM 2005; 5: 93-4.
[4] Ferhan ESEN, Fatma Y. Hamza ESEN. Turk J Med Sci 2001; 31: 317-22.
[5] Aoshiba K, Nagai A. Tobacco Induced Diseases 2003;3(1):219-26.
[6] Godtfredsen NS, Osler M, Vestbo J, Andersen I, Prescott E. J Epidemiol Community Health 2003; 57: 412–16
[7] Nageswari SK, Sharma R, Kohli DR. Indian J Physiol Pharmacol 2007;51(3):235-43.
[8] Acharya UR, Joseph KP, Kannathal N, Lim CM, Suri JS. Medical and Biological Engineering and Computing 2006; 44(12): 1031-1051.
[9] European Heart J 1996;17:354-81
[10] Sztajzel J. Swiss Med Wkly 2004;134:514-22.
[11] Low PA. Clinical autonomic disorders:Analysis of blood pressure and heart rate variability. 2nd ed. Philadelphia: Lippincott-Raven Publishers; 1997. 309-20.
[12] Katira T, Narain VS, Puri VK. JAPI 1997;45(1):49-51.
[13] Manzano BM, Vanderlei LC, Ramos EM, Ramos D. Arq Bras Cardiol 2011;96:154–60
[14] Khan A,Sabbir K,Ansari JK,Zia N.Comparison of forced expiratory volume in one second(FEV1)among asymptomatic smokersand non-smokers.J Pak Med Assoc.2010;60:209(PubMed)
[15] Narkiewicz K, van de Borne PJH, Hausberg M, Cooley PL, Winniford MD, Davison DE, et al. Circulation 1998;98(6):528-34.
[16] Shinozaki N, Yuasa T, Takata S. Int Heart J 2008;49(3):261-72.
(17) Niedermaier ON,Smith ML,Beightol LA,Zukowska-Grojec Z,Goldstein DS,eckberg DL.Influence of cigarette smoking on human autonomic function. Circulation. 1993; 88: 562-71.(PubMed)