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Original Research Article

Heart Rate Variation (HRV) & Impaired Hearing with Emitting Fumes in New Youth Comparison

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Abstract:

Background: Heart rate variability, baroreceptor sensitivity, muscle sympathetic nerve activity, and sympathetic skin response are some of the parameters that can be measured in the temporal and frequency domains, respectively. Cardiovascular rate variability (HRV), which is defined as the variability in time and/or frequency between successive R waves of the heartbeats, is a measure of how well the cardiovascular system and the mechanisms it governs are integrated. Furthermore, heart rate variability (HRV) has been widely acknowledged as a metric for measuring autonomic function and quantifying sympathovagal balance for several decades. By leveraging a multidimensional approach encompassing clinical, biochemical, and audiometric assessments, we seek to elucidate the specific pathways through which emitting fumes may compromise auditory function and HRV. The findings of this research endeavour are anticipated to advance our understanding of the nuanced relationship between smoking and hearing health, potentially paving the way for targeted interventions, public health initiatives, and the development of early detection strategies for this consequential health concern.

Materials and Methods: The heart rate variability analysis study was done using VarioWin_HR, apc-based HRV analysis systems. This HRV analysis equipment was validated using the GOLD standards published by the American Heart Association and the European Society of Cardiology. The North American Society of Pacing and Electrophysiology and the Board of the European Society of Cardiology adopted the Task Force 1996 recommendations for HRV recording. The hearing tests were conducted on the outpatient department of the ENT department at SGRRIM & HS Patel Nagar Dehradun. General examination, ear examination, screening tuning fork test, and pure tone air-conduction and bone conduction tests using AD 226 Intra-acoustic audiometry. The study included 220 subjects, including 110 smokers and 110 age-matched non-smokers.

Results: Smokers had significantly elevated resting HR values than non-smokers. Heart rate increased acutely during smoking. Mean heart rate increased and returned almost to baseline after 30 min. HRV index significantly decreased in smokers as compared to non-smoking controls participants but the other spectral HRV parameters were comparable. Impaired hearing was found significant associated with smoking. Furthermore, the hearing loss was primarily sensor neural, with the mild type (26–40 dB) being the most usual in smokers.

Conclusions: Smoking was found to affect young smokers Heart Rate, increasing HR at rest. Spectral parameters of HRV analysis of smokers remained in normal limits, but more importantly geometrical parameter HRV index, showed significant abnormality.

Keywords: HRV, Impaired hearing, Emitting fumes, Audiometery.

Introduction

The World Health Organization (WHO) 2023 has documented an annual toll of 8 million deaths every year don't be one of them. The benefits of quitting tobacco are almost immediate. After just 20 minutes of quitting smoking, your heart rate improves. Within 1-9 months, coughing and shortness of breath decrease. Within 5-15 years, your stroke risk is reduced to that of a non-smoker. Within 10 years, your lung cancer death rate is about half that of a smoker. Within 15 years, your risk of heart disease is that of a non-smoker. If that's not enough here are a few more reasons. Tobacco use is responsible for 25% of all cancer deaths globally. Over 1 million people die every year from exposure to second-hand smoke. Non-smokers exposed to second-hand smoke are at risk of developing lung cancer. Cigarettes remain an important cause of accidental fires and resulting deaths. E-cigarettes also expose non-smokers and bystanders to nicotine and other harmful chemicals. Being exposed to second-hand smoke may increase the risk of progression from tuberculosis infection

to active disease. Being exposed to second-hand smoke is associated with type 2 diabetes. Everything stinks, from your skin, to your whole house, your clothes, and your fingers and breath. Tobacco causes teeth to yellow and creates excess dental plaque.

Smoking and chewing tobacco causes bad breath. Tobacco makes your skin wrinkly, making you look older faster. Smoking prematurely ages the skin by wearing away proteins that give the skin elasticity, depleting it of vitamin A and restricting blood flow. These wrinkles are more apparent around the lips and eyes and tobacco also makes skin leathery and dry. Tobacco smoking increases the risk of developing psoriasis, a noncontiguous inflammatory skin condition that leaves itchy, oozing red patches all over the body. Smoker's children suffer reduced lung function, which continues to affect them in the form of chronic respiratory disorders in adulthood. Exposure of children to e-cigarette liquid continues to pose serious risks. There is a risk of the devices leaking, or of children swallowing the liquid. E-cigarettes have been known to cause serious injuries, including burns, through fires and explosions. School-aged children exposed to the harmful effects of second-hand smoke are also at risk for asthma through inflammation of the airways to the lungs. Children under 2 years of age who are exposed to second-hand smoke in the home could get middle-ear disease possibly leading to hearing loss and deafness. Quitting smoking decreases the risk of many diseases related to second-hand smoke in children, such as respiratory diseases (e.g., asthma) and ear infections.

There is an established link between heart rate (HR) and cardiovascular health. HR is a very important, non-invasive and easy-to-measure index of myocardial work. There is a plethora of studies suggesting that the findings of HR elevation in smokers are important surrogates for an underlying autonomic dysfunction associated with increased cardiovascular morbidity and mortality. Smoking has been associated with higher HRrest and chronotropic incompetence.

Overall, HR responses to cigarette smoking may be implicated in the link between smoking and cardiovascular disease. However, although the effects of smoking on HR seem to be age-dependent, relatively little is known about how smoking affects HR in young adults. The purpose of this study was to examine the effects of smoking on resting HR.

Cigarette smoking has become a common tendency worldwide. In general, tobacco is consumed by approximately 1.3 billion of the world's population [1]. While tobacco use is rising globally, the epidemic of tobacco related diseases has just begun. Most of tobacco's damage to the human health does not become evident until years or even decades after the onset of its use. While tobacco use is the leading cause of preventable death in the world [2] but this epidemic can be stopped by proper measures.

Tobacco, especially its content of nicotine, is a drug that has various deleterious effects besides having seductive effects and a dangerous risk of dependence. Many of the health effects of smoking depend on the exposure history, which includes the age at which the smoking began, the number of cigarettes which were smoked per day, the degree of inhalation, and the cigarette characteristics such as the tar and the nicotine content [3–5].

Besides the various tobacco related diseases, smoking has been associated with its effect on the senses, which includes the sense of hearing. The current smokers were 1.69 times as likely to have a hearing loss as the non-smokers. This relationship remained for those without a history of occupational noise exposure and in analyses which excluded those with non-age-related hearing loss [6]. Meta-analysis studies which were done in Japan and Korea also came with similar evidences of a positive association between smoking and hearing loss [7,8]. The Framingham 33 and Baltimore studies 34 didn't find any connection between hearing loss and smoking. Smoking may accelerate the age related hearing loss (presbycusis), as was demonstrated in various studies [7,9].An interaction between smoking and occupational noise was reported, whereby the deleterious effect of the noise exposure was exacerbated by smoking [10], while the recent publications reported no interaction, thus indicating that smoking affected the hearing loss independently [11].

Material and Methods

The present study was conducted at SGRRIM & HS Patel Nagar Dehradun. Participants were categorized into either the smoker group (individuals with a history of smoking or current smokers) or a control group of the non-smoker group (individuals who had never smoked). The study group consisted of male smokers (n=110) aged between 20 and 70 years, while the age-matched control group (n=110) comprised subjects with no history of smoking (control group).Participants in both groups were randomly selected based on inclusion and exclusion criteria, and informed consent was obtained from each participant.

The heart rate variability analysis study was done using VarioWin_HR, apc-based HRV analysis system. This HRV analysis equipment was validated using the GOLD standards published by the American Heart Association and the European

Society of Cardiology. The North American Society of Pacing and Electrophysiology and the Board of the European Society of Cardiology adopted the Task Force 1996 recommendations for HRV recording.

To minimise the impact of circadian rhythm, all participants' room temperatures were only recorded between 8 and 11 a.m, between 20 and 26 degrees Celsius. It was suggested that participants leave their telephones, jewellery, watches, and coins at home. The participant was told to pee and unwind in a supine posture for 10 minutes before to recording. ECG electrodes were attached to the participants and subsequently linked to acquisition equipment. The positive electrode was connected to the left arm, the negative electrode to the right arm, the reference electrode to the left foot, and the ground electrode to the right foot using a four-lead ECG cable. An extra 10 minutes of relaxation were requested of the participants. In a silent environment, a 5-minute lead II ECG was captured. Data was gathered using the built-in software at a sample rate of 500 cycles/sec from the standard lead II recording. Using analysis software, the ECG signal was digitalized and stored on the computer. Using the VarioWin-HR analysis programme, HRV analysis of the acquired data was performed.The final results were shown on an A4 sheet with explanation; the programme recognises all R waves and computes the R-R interval from the collected data. R-peak detection and RR calculation were carried out automatically and precisely by the Vario Win HR programme.

The hearing examination included a general examination, ear examination, screening tuning fork test, and pure tone air-conduction and bone conduction tests using AD 226 Intra-acoustic audiometry. The audiometry test, conducted & utilized pure tone audiometry (PTA) to assess hearing thresholds for different frequencies. Participants responded to tones, and the lowest volume level at which a response was recorded was considered the threshold.

The study differentiated between right and left ears and independently assessed the extent of sensorineural hearing loss.Statistical analysis, including unpaired Student's t-test and chi-square test using SPSS version 17, revealed that both smoking and non-smoking groups had Puretone average values below 25 dB HL.

Results:

The analysis demonstrated statistically significant impaired hearing in smokers compared to non-smokers, with a higher mean in the smoking group. The study classified hearing loss according to ASHA standards, revealing that the severity of hearing impairment in both groups indicated only mild hearing loss.

On analyzing the audiometric data for the severity of the hearing impairment, it was observed that the mild form (26-40 db loss). Smoking was found to be statistically associated with the hearing impairment, with 69.8% of the smokers and 29.93% of the non-smokers having hearing impairment. Also, as the age increased, the percentage of the affected individuals also increased, with greater percentages of the smokers being affected in comparison to the non-smokers.

The hearing loss in case of non-smokers was found almost 16.4 %, whereas 83.6 %was found without hearing loss. The severity of the hearing loss in the smokers increased with an increase in the number of bidis/cigarettes which were smoked and the duration of smoking and this association was found to be significant statistically.

These data suggest that environmental exposures may play a role in age-related hearing loss. If longitudinal studies confirm these findings, modification of smoking habits may prevent or delay age-related declines in hearing sensitivity.

Cigarette smoking may affect hearing through its effects on antioxidative mechanisms or on the vasculature supplying the auditory system [12] An association between cigarette smoking and hearing loss among adults has been found in some clinical studies. Weiss, found that men who smoked more than 1 pack per day had worse hearing thresholds at 250 to 1000 Hz than nonsmokers or "light" smokers, but there was no difference at higher frequencies. Siegelaub et alreported on a large study of 33146 men and women seen at Kaiser-Permanente, Oakland, Calif [13-14]. Among men without a history of noise exposure, current smokers were more likely than nonsmokers to have a hearing loss at 4000 Hz, but the size of the effect was small. There was no association among women. The Baltimore Longitudinal Study of Aging[15-16], found no association between cigarette smoking and the development of a hearing loss in 531 white, upper-middle-class men.

There have been few population-based studies of smoking and hearing. In the Health Interview Survey,[17], men who smoked 2 or more packs per day were more likely to report having a hearing loss than nonsmokers. In the Framingham Study [18] which tested hearing with audiometry, there was no association between cigarette smoking and hearing loss. The purpose of our article was to evaluate the association between cigarette smoking and hearing loss in a large population-based cohort of adults aged 20 to 70 years.

Table 1: Impaired hearing loss in smokers and non-smokers due to smoking:

Smoking status	No hearing loss		Affected Subjects		Degree of hearing loss	Total
	Number	%	Number	%	26-40db	
SMOKERS	**33**	**29.93**	**77**	**69.8**	**MILD**	**110**
NON-SMOKERS	**92**	**83.6**	**18**	**16.4**	**MILD**	**110**
GRAND TOTAL	**125**		**95**			**220**
p-value	< 0.001					

Table 2: Smokers and non-smokers as per age Comparison:

Smoking Status	Age (in years)	Affected Subjects		No Hearing loss		Severity 26-40db	Total
		No	%	No	%		
Smokers	20-30	52	81.7	11	18.3	Mild	61
	30-50	19	72.9	07	27.1	Mild	27
	50-70	18	85.3	03	14.7	Mild	22
Non-Smokers	20-30	12	18.8	53	81.2	Mild	23
	30-50	06	21.6	21	78.4	Mild	19
	50-70	08	43.4	10	56.6	Mild	13
Grand total		**115**		**105**			**220**

Discussion

Cigarette smoking was found to be statistically correlated with sensorineural hearing loss. The sensory component is due to damage of organ of Corti or to an insufficiency of hair cells to stimulate the auditory nerve. Sensorineural hearing impairment (HN) is caused by distress of the cochlear sensory epithelium or the peripheral auditory neurons. The neural element refers to when damage is proximal to the cochlea and auditory nerve (retrocochlear damage),[19-20] Conductive hearing loss (HT) is characterized by obstruction or illness in the outer or middle ear. Consequently, a mixed hearing loss (HM) implies a combination of conductive and sensorineural hearing damage, respectively the problem occurs in the outer or middle and the inner ear [21-22].

The present study revealed that cigarette smoking shows a significant association with visual and hearing impairment. Consistent with our study findings, other studies have reported a highly significant association between current smoking and sensorineural hearing loss and age-related macular degeneration. Cigarette smoking has pernicious effects on the retinal tissues and inner ear. This provides powerful support for a causal relation between cigarette smoking and the prevalence of sensorineural hearing loss and age-related macular degeneration [23-26]. Two phases have been recognized in cigarette smoke, gas and tar phases, which are abundant in free radicals and non-radical oxidants. Particularly, hydroxyl radical and peroxyl are implied in oxidative stress and tissue malady. We emphasized the hint of possible similar roles of melanin in progress of these two disorders in the retinal tissues and inner ear. Stria vascularis in the Corti structure of the cochlea is the basic structure including melanocytes. The number of Corti structures and melanocytes is lower at the base of the cochlea. Small melanin concentration makes the cochlear base more responsive to oxidative stress and lipofuscin aggregation. One way through which the chemicals found in cigarette smoke may produce an effect on hearing status, is by provoking injury to the anti-oxidative systems and to the ear vasculature [23-26] On the other hand, melanin in the RPE cells defends photoreceptors against oxidative stress[24] Therefore, melanin alteration caused by oxidative stress might account for important concomitant visual and hearing impairment[27,28,29]. Cigarette smoke is known to be rich in chemical compounds. The two most toxic constituents of cigarette smoke are nicotine and carbon monoxide, however, cigarette smoke contains about 4000 additional toxic components. Cigarette smoking decreases the blood supply due to the vasospasms induced, favors the atherosclerotic narrowing of the blood vessels and accelerates ischemic damage due to increased blood viscosity and by thrombotic occlusions with endothelial changes of the vessels [30-32]. One of the means through which the chemicals found in cigarette smoke may have an effect on cochlea is by causing vasculopathy. Besides this, chemicals found in cigarette smoke damage cochlear melanin. Cigarette smoke implies a great number of prooxidant compounds[33,34] and also consists of dioxins, which are moderated by aryl hydrocarbon receptor (AhR), which is a ligand-dependent transcriptional factor widely expressed among immune, epithelial, endothelial and stromal cells in barrier tissues. Dioxin affects ocular tissues through the AhR pathway, develops vascular endothelial growth factor (VEGF) production in human RPE cells which potentiates retinal exudation by neovascularization[35-36].

Our observations align with investigations by Uchida et al. (year), who reported statistical interactions between smoking and hearing loss in

adults, supporting the conclusion that smoking may indeed be a causative factor for hearing loss in the adult population. The nuanced differences in our study underscore the importance of considering population-specific factors and highlight the need for further research to deepen our understanding of the complex relationship between smoking and hearing impairment in the Indian context [37]. The correlation between smoking cigarettes and ageing was also accessed in our study. In which it was found that although not significantly, the prevalence of hearing loss increases with ageing. Our study's findings on the association between hearing loss and advancing age are consistent with earlier research that has shown a connection between smoking and hearing loss with age. The proportion of ears that could respond decreased for each age-decade as the frequency increased[38]. According to earlier research done by Cruickshanks KJ et aland Itoha et al, smoking increased the risk of hearing loss and was a contributing factor in age-related hearing loss. Smoking has also been linked to age-related hearing loss by a multipurpose study. Although the direct impairment of the auditory system and pathways was not investigated in the current analysis but some recent researches like research done by Rogha M et al [39] concluded that Cigarette smoking may affect the hearing through the direct ototoxic effect of nicotine on the cochlea. As smokers are constantly exposed to the levels of carbon monoxide in the range of 500 to 1,500 parts per million. They also concluded that the carbon monoxide in tobacco smoke causes a rise in the carboxyhemoglobin levels in smokers, which may in turn reduce the oxygen which is available for the organ of Corti[40], resulting in damage to the hair cells which are sensitive to oxygen, and leading to hearing loss.

Conclusion

The study included 220 subjects and on analyzing the audiometric results, it was noticed that cigarette smoking was found to be statistically associated with hearing impairment ($p<0.05$), especially sensorineural hearing impairment (HN), thus 69.8% of smokers and 16.4% of nonsmokers were identified with sensorineural hearing loss among Indian adult males. In this study, only sensoneural hearing loss impairment was seen among smokers as well as non-smokers. It was also found that although not significantly, the prevalence of hearing loss increases with ageing.

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