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ORIGINAL ARTICLE

A Comparative Study of ECG and Blood Pressure among Smokers and Non-Smokers

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ABSTRACT

Background: Smoking is one of the most significant risk factors for a wide range of illnesses, including lung and heart disease, cancer, and many others. There are currently 1.2 billion smokers around the globe, and that figure is expected to climb by 1.6 billion by the year 2020. Tobacco use kills about 3.5 to 4 million people worldwide every year, and this number is expected to rise to 10 million by 2020.

Mostly, the acute consequences of smoking are well-documented. These contain an increase in heart rate and blood pressure, as well as a decrease in cardiac output and vasoconstriction. Smoking is the subject of the current investigation. Smokers vs non-smokers on ECG and blood pressure.

Objectives: To examine the differences in blood pressure between people who smoke and those who don't and to examine the differences in ECG waveforms between smokers and nonsmokers

Methods: May 2020 through April 2021 was when the research was carried out at PUMHS, There were fifty smokers and fifty non-smokers aged 20 to 35 years old in each group. After a thorough description of the study's goals and objectives, only those individuals who agreed to take part were included. Participants who met certain criteria were discarded. Blood pressure (sitting position) and an electrocardiogram (supine posture) were got while the individuals were in a resting condition.

Results: The research had 100 participants, 50 of them were smokers and the other 50 were non-smokers. Nonsmokers and smokers had similar age distributions, and the disparity was not statistically significant (p-value- 0.97). In Lead III, 54% had an inverted wave compared to 40% normal and this difference found to be statistically significant, i.e. highest percentage of flat and inverted waves found in smokers. The difference in presence of abnormal waves among smokers and non-smokers in Lead V1 to V4 was found to be statistically significant. When a comparison of vitals and ECG between smokers based on pack-years (1- 3 pack- year, 4-6pack year, and 7-9pack year) was done there was no statistical significance attributed to various other parameters like pulse, heart rate, respiratory rate, systolic BP, diastolic BP, P wave amplitude, P wave duration, PR interval, QRS duration, QT interval, and QRS axis, except QTc.

Conclusion: Researchers found that in smokers, there were a variety of ECG alterations and an increase in blood pressure despite the lack of any heart disease.

Keywords: Smokers; Non-smokers; Blood Pressure; ECG.

INTRODUCTION

Tobacco smoking is "the most widely documented cause of illness ever explored in the history of biological study," says the US Surgeon General.[1] Since the dawn of time, people over the world have been smoking. Cigarette smoking is more dangerous than previously thought, according to new studies. Smoking has been proven to be a leading cause of a wide range of health issues, including respiratory disorders, heart disease, cancer, and many more. There are currently 1.2 billion smokers in the world, and that figure is expected to climb to 1.6 billion by the year 2020. Tobacco usage kills about 3.5 to 4 million people worldwide each year, and this number is expected to rise to 10 million by 2020.[2] There are around 22 million persons in Pakistan over the age of 18 who smoke cigarettes, water pipes, or some other tobacco product. The daily smoking rate in Pakistan is 15.9%, with 32.4% of Pakistani males and 5.7% of Pakistani women being regular users of tobacco..[3] Tobacco-related diseases cost the country over Rs.2.5 million in direct medical expenditures, absenteeism, treatment, and lost income because of early mortality for every patient.[4] At a rate of 41% among males and 14.9% among women, tobacco usage is on the rise in Karnataka as well. Cigarette smoking, high blood pressure, and excessive cholesterol levels are all three major risk factors for coronary artery disease. Tobacco use is a risk factor as well as when paired with other risk factors. Tobacco quitting had a 36% lower risk of death from coronary artery disease compared to those who continue smoking.[5] Smokers' lungs contain tar, which can harm DNA in certain ways.[6] Cigarette smoke contains nicotine, which raises blood pressure by 10 to 20 mm Hg each cigarette, resulting in a higher average daily BP for frequent smokers. Norepinephrine from adrenergic neurons is likely responsible for the elevation in blood pressure that lasts for 15 to 30 minutes after a nicotine spike.[7] Smokeless tobacco usage is associated with much longer-lasting increases in blood pressure.[8] Smokers' increased risk of strokes and heart disease,[9] as well as their resistance to antihypertensive medication,[10] must be because of this pressor impact.[11] A rise in blood pressure, heart rate, cardiac output, and carotid artery blockage are all well-known side effects of smoking, as is the occurrence of tachycardia, MI, and other symptoms of acute smoking effects.[12,13] As a result, the present research will look at the effects of smoking on the ECG and blood pressure to improve public awareness of the dangers of smoking.

MATERIALS AND METHODS

The research was carried out at Peoples University of Medical and Health Sciences for Women Shaheed Benazir Abad, Sindh Pakistan. When it began in May 2020 and ended in April 2021. Fifty smokers and 50 non-smokers, each between the ages of 20 and 35, were recruited from the student population at PUMHS, and the Medicine outpatient department at PUMHS, respectively. Subjects who agreed to take part in the study after hearing about its scope and goals were enrolled with their informed permission. The individuals were interviewed extensively before undergoing a thorough physical examination. Recording important data from each participant was done with the use of a pre-tested structured form. Besides assessing height and weight in kilos, the physical examination also involved palpating the radial artery to determine resting pulse rate and using a mercury sphygmomanometer to measure blood pressure. All of these measurements were

recorded in the patient's medical record. A thorough evaluation of the cardiovascular and respiratory systems was carried out by medical personnel. Inclusion and exclusion criteria were used to choose participants for the research.

Inclusion criteria:

1 Young adult smokers of age group ranging from 20 to 35 years as cases and

2 Young adult non-smokers of the same age group as controls

Exclusion criteria:

1 Persons with a history of cardiac diseases.

2 Persons with a history of respiratory diseases.

3 Persons with abuse of psychoactive substances.

4 Persons with alcohol abuse.

Recording Paper: Because of the usage of temperature-sensitive paper, Horizontal and vertical lines are spaced by 1mm on the paper used to record an ECG's readings. There is a noticeable difference in the weight of each 5th line in either direction. Voltage is shown on the vertical axis (amplitude). Each 0.1mV corresponds to 0.1mm. Regarding this graph, the arrows on the horizontal axis point toward (duration). ECG paper travels through 300 thick lines or 1500mm in a minute, which is 0.04 seconds per millimeter (25mm per second).

Recording of Electrocardiogram: A 12-lead electrocardiogram was collected while the individual was at rest after a thorough evaluation. On a bed, the individuals were placed in a supine posture. Chest, forearms, and legs were exposed. Objects such as electrical gadgets, metallic ornaments, and so on were taken out of the way to prevent interference. The patient was kept away from current-carrying cables to avoid interference from AC-powered devices. The subject was made to relax. The electrodes were placed on the arms and legs at a location with the least amount of mobility. Surface hair was eliminated from the electrode region. A mild redness was achieved by applying spirit to the skin and then allowing it to dry. Enough ECG gel was placed on the skin at the selected spot to achieve excellent electrical connections, around 2cm in length. The skin of the individual was clipped to the electrodes to ensure adequate contact. Six chest leads were positioned in various parts of the body. A lot of care was taken to avoid gel smearing between the electrodes on a chest. Various characteristics of the ECG were measured and analyzed, including heart rate, the P-wave, the QTc interval, the QRS complex, the QRS axis, the QT interval, and the QRS axis, as well as the ST-segment.

Statistical Analysis: SPSS (Statistical Package for the Social Sciences) version 23 was used to assemble and analyze the data. We compared quantitative variables by student-t between the two groups. ANOVA test: to compare quantitative variables in over two groups. The Chi-square test compared categorical variables between two or more groups.

Fischer's exact test compared categorical variables between two groups when any of the cell values is less than 5

RESULTS

The research had 100 participants, 50 of them were smokers and the other 50 were non-smokers. Smokers accounted for most of the study's participants, with 34% of them falling into the 26-30-year-old and 31-35-year-old age brackets, respectively, while non-smokers accounted for 34% of the total. Nonsmokers and smokers had similar age distributions, and the disparity was not statistically significant (p-value- 0.97) Table-1.

Weight (mean) was more among Non- smokers (57.74 kgs) compared to smokers (53.51 kgs) but this was an insignificant difference.

Height (mean) was more among Non-smokers (1.64 mts) compared to smokers (1.52 mts) and this difference was statistically significant. BMI (mean) was comparatively less among smokers (20.57) than non-smokers (21.34) and this difference was statistically significant. Table-2

Non-smokers (78.36 vs. 87.20 bpm) had a statistically significantly lower pulse (mean pulse rate) than smokers. Smokers' heart rates (mean) were higher than nonsmokers' (mean: 77.94 bpm), which was statistically significant. Smokers (15.80 pm) and non-smokers (15.96 pm) had similar respiratory rates, and the difference was statistically insignificant. Smokers had higher systolic blood pressure (mean, 121.56 mm Hg) than nonsmokers (mean, 118.68 mm Hg), and this was statistically significant. For the same reason, smokers had significantly higher (statistically significant) diastolic blood pressure (80.60 mm Hg) than non-smokers (79.32 mm Hg). Table-2

P wave duration (mean) was not significantly different between smokers (0.082 sec) and non-smokers (0.080 sec), and the amplitude (mean) was somewhat higher among smokers (1.02) than non-smokers (1.00), both of which were not significantly different. Table-2

Smokers' PR intervals were 0.012 seconds shorter than nonsmokers' on average. The difference was statistically significant. But. The QRS complex (mean) did not differ statistically between smokers and nonsmokers (0.081sec) (0.079 sec). Table-2

There was a statistically significant difference in the QT interval (mean) between smokers and nonsmokers (0.34 sec versus 0.36 sec). It was discovered, however, that the QTC interval was somewhat higher among smokers (0.39 sec) than nonsmokers (0.38 sec). The QRS axis (mean) was significantly higher in nonsmokers (65.440) than in smokers (42.620). Table--2.

Both smokers and nonsmokers had normal T waves in Lead I and Lead II whereas, in Lead III, 26% of smokers had a normal wave, 20% had a flat wave and 54% had an inverted wave compared to 40% normal, 16% fat and 44% inverted wave among non-smokers and this difference found to be statistically significant, i.e. highest percentage of flat and inverted waves found in smokers. Table-3

Table 1: Age-wise distribution of study subjects

Age group	Smokers	Non-smokers	Total
20–25 years	15 (30%)	16 (32%)	31 (31%)
26 – 30 years	18 (36%)	17 (34%)	35 (35%)
31–35 years	17 (34%)	17 (34%)	34 (34%)
Total	50 (100%)	50 (100%)	100 (100%)

Mean age of study subjects: 27.94 ± 4.25
Mean age of smokers: 28.06 ± 4.01
Mean age of study nonsmokers: 27.82 ± 4.52

Table 2: Comparison of anthropometric measurements, vitals, P wave, and PR and QR intervals b/w smokers and non-smokers

Measurements	Smokers (n = 50) Mean ± Sd	Non-Smokers (n = 50) Mean ± Sd	P-value
Weight (Kgs)	53.51 ±3.12	57.74 ± 5.71	0.074
Height (mts)	1.52 ± 0.046	1.64 ± 0.061	0.001
BMI	20.57 ± 1.04	21.34 ± 1.47	0.004
Vitals (BP, HR, RR, Pulse)			
Pulse	87.20 ± 5.66	78.36 ± 4.22	0.000
Heart rate	87.16 ± 5.69	77.94 ± 3.44	0.000
Respiratory rate	15.80 ± 1.01	15.96 ± 0.45	0.310
Systolic BP	121.56 ± 2.47	118.68 ± 3.01	0.000
Diastolic BP	80.60 ±1.29	79.32 ± 2.04	0.000
P wave			
Duration	0.082 ± 0.0049	0.080 ± 0.0044	0.09
Amplitude	1.02 ± 0.06	1.00 ± 0.10	0.23
PR and QRS interval			
PR interval	0.143±0.006	0.155 ± 0.01	0.00
QRS interval	0.081 ± 0.007	0.079 ± 0.004	0.26
QT, QT_C, and QRS axis			
QT	0.34 ± 0.03	0.36 ± 0.02	0.04
QT_C	0.39 ± 0.016	0.38 ± 0.012	0.00
QRS axis	42.62 ± 12.16	65.44 ± 7.22	0.00

Both smokers and non- smokers had normal T waves in Lead aVR and Lead aVL whereas, in Lead aVF, 92% of smokers had a normal wave and 8% had inverted waves compared to 100% normal waves among non- smokers, and

this difference was found to be statistically not significant i.e. only smokers had 4% of the inverted wave. Table-3

Non-smokers had a 52% of the inverted T wave in Lead V1 only and 100% of a normal wave in remaining Lead V2 to V6. Whereas among smokers, 100% normal wave was present in Lead V5 and Lead V6. 68%, 24%, 24%, and 4% of non-smokers had inverted T wave in Lead V1, V2, V3, and V4 respectively. A flat wave was present in 4% and 4% of smokers in Lead V1 and V4, respectively. The difference in presence of abnormal waves among smokers and non-smokers in Lead V1 to V4 was found to be statistically significant. Table-3.

Q wave was present among 12%, 44%, and 52% of smokers compared with 8%,26%, and 44% of non-smokers in Lead I, II, and III respectively. This difference was not statistically significant. Table-4.

None of the nonsmokers exhibited Q wave in Lead aVR, while 8% of smokers had, and the difference was not statistically significant. In Lead aVL, 24% of smokers exhibited Q wave, compared to just 10% of non-smokers, and this huge disparity was determined to be statistically significant. In Lead aVF, smokers had the highest percentage of Q waves (36%) compared to non-smokers (32%), although this was not statistically significant. Table-4.

There was no statistically significant difference in the occurrence of Q waves between smokers and non-smokers in Lead V1, V4, V5, and V6. No Q wave was seen among both smokers and non-smokers in Lead V2 and Lead V3. Table-4.

When a comparison of vitals and ECG between smokers based on pack-years (1- 3 pack- year,4-6pack year, and 7-9pack year) was done there was no statistical significance attributed to various other parameters like pulse, heart rate, respiratory rate, systolic BP, diastolic BP, P wave amplitude, P wave duration, PR interval, QRS duration, QT interval, and QRS axis, except QTc. Table-5.

Table 3: Comparison of 'T' wave different leads, b/w smokers and nonsmokers

Limb leads	Smokers			Non-smokers			P-value
	N	F	I	N	F	I	
I	100%, n = 50	00	00	100%, n = 50	00	00	------
II	100%, n = 50	00	00	100%, n=50	00	00	------
III	13 (26%)	10 (20%)	27 (54%)	20 (40%)	8 (16%)	22 (44%)	0.03
'T' wave in augmented							
aVR	100%, n = 50	00	00	100%, n = 50	00	00	------
aVL	100%, n = 50	00	00	100%, n = 50	00	00	------
aVF	46 (92%)	00	4 (8%)	50 (100%)	00	00	0.48
'T' wave in the chest leads							
V1	13 (26%)	6 (4%)	34 (68%)	24 (48%)	00	26 (52%)	0.04
V2	38 (76%)	00	12 (24%)	100%, n = 50	00	00	0.00
V3	38 (76%)	00	12 (24%)	100%, n = 50	00	00	0.00
V4	43 (86%)	2 (4%)	5 (10%)	100%, n = 50	00	00	0.02
V5	100%, n = 50	00	00	100%, n = 50	00	00	-----
V6	100%, n = 50	00	00	100%, n = 50	00	00	-----

Table 4: Comparison of different 'Q' waves in limb leads b/w smokers and non-smokers

Limb leads	Smokers		Non-smokers		P-value
	Present	Absent	Present	Absent	
I	6 (12%)	44 (88%)	4 (8%)	46 (92%)	0.08
II	22 (44%)	28 (56%)	13 (26%)	37 (74%)	0.05
III	26 (52%)	24 (48%)	22 (44%)	28 (56%)	0.24
'Q' wave in augmented					
aVR	4 (8%)	46 (92%)	00	50 (100%)	0.26
aVL	12 (24%)	38 (76%)	5 (10%)	45 (90%)	0.03
aVF	18 (36%)	32 (64%)	16 (32%)	34(68%)	0.38
'Q' wave in the chest leads					
V1	00	50 (100%)	4 (8%)	46 (92%)	0.13
V2	00	50 (100%)	00	50 (100%)	-----
V3	00	50 (100%)	00	50 (100%)	-----
V4	8 (16%)	42 (84%)	8 (16%)	42 (84%)	0.75
V5	26 (52%)	24 (48%)	27 (54%)	23 (46%)	0.85
V6	31 (62%)	19 (38%)	27 (54%)	23 (46%)	0.43

Table 5: Comparison of vitals and ECG among smokers based on pack-years

Parameters	1–3 pack years	4–6 years	7–9 years	P value
Pulse	87	87	85.72	0.73
Heart rate	88	88	86.65	0.72
Respiratory rate	16.33	15.69	15.74	0.39
Systolic BP	124.34	122.53	122.24	0.17
Diastolic BP	82.334	81.78	81.38	0.21
P wave amplitude	1.08	1.03	1.02	0.12
P wave duration	0.070	0.083	0.083	0.57
PR interval	0.164	0.164	0.163	0.71
QRS	0.075	0.080	0.080	0.24
QT	0.32	0.33	0.33	0.61
QT_C	0.37	0.38	0.41	0.00
QRS axis	41.68	42.47	41.27	0.94

* ANOVA test (above values are mean values)

DISCUSSION

We studied and analyzed the differences in blood pressure and ECG between smokers and non-smokers in this study.

Heart rate: In this study, smokers' heart rates (mean) were higher (87.16 bpm) compared to non-smokers (77.94 bpm) and it was statistically significant (P=0.000). Ramon C. Hermida et al[7] found a statistically significant increased heart rate among smokers (P<0.001), Zahi Khoury et al[8] found smokers had (74bpm) whereas nonsmokers(71.3bpm), Venkatesh G and Swamy RM[9] found smokers had (80.8+10.2) whereas nonsmokers (76+6.9), Osman karakaya et al[11] found smokers had (72+8) whereas nonsmokers (67+7) and were statistically significant (P<0.001). Catecholamines are released from the adrenal medulla and they stimulate the sympathetic ganglia, which might explain the elevated heart rate.[9]

Blood Pressure: It was discovered in this study that smokers had a significantly higher Systolic Blood Pressure (mean of 121.56) than non-smokers had (118.68 mm Hg). A statistically significant difference in the Diastolic BP (mean) between the smokers and the non-smokers was found. Smokers had higher systolic and diastolic blood pressure, according to Ramon C. Hermida et al[7], Zahi Khoury et al[8], and S. B. Sharma et al[12], all of whom observed statistically significant increases in diastolic pressure among smokers (P0.001).
Benowitz NL et al[13], Istvan JA et al[10], Tachmes L et al[15], Gropelli Antonella et al[2], James D Neaton et al[16], Nabipur et al[17] and Gupta et al[18] also found increased
In smokers, both systolic and diastolic blood pressure is higher than in non-smokers. The adrenal medulla produces catecholamines, which force blood vessels to contract, resulting in a rise in blood pressure.
P wave: Compared to non-smokers, smokers had a P wavelength (mean) of 0.082 seconds, which was statistically insignificant. Smokers had a somewhat higher amplitude (mean) than non-smokers (1.00), but the difference was not statistically significant. Smokers had somewhat higher amplitude than non-smokers, according to S B Sharma et al[12] and Khan IS et al.[19]
PR interval: An average difference of 0.012 seconds among those who smoke and those who do not smoke in the PR interval (mean), with the former being statistically significantly shorter. The findings of Baden L et al[20] and Khan IS et al[19] agreed with this conclusion. The AV node's effective refractory time is shortened and conduction velocity rises once in the length of the QRS complex (mean) (0.079 sec). It was found in Khan IS et al[19], as well as Chatterjee S et al.[21] The duration of the QRS complex has increased by milliseconds. Electrocardiographic wave patterns are affected by age and the aging impact is changed by long-term smoking.[21]

QRS Axis: When smokers (42.620) were compared to non-smokers in this research, the QRS axis (mean) exhibited a statistically highly significant decrease (65.440). Smokers (42.0+22.7) had a lower QRS axis than nonsmokers (51+23.7), although the difference was not significant, according to Venkatesh G and Swamy RM.[9] Chatterjee S et al[21] discovered that smokers and non-smokers have considerably different QRS and P axis. Nonsmokers had a faster rate of changing these axes to the left as they became older. In normal healthy participants, there was no correlation between lung function and ECG. These findings suggest that electrocardiographic wave patterns are affected by age and that this aging impact is influenced by long-term smoking.
QT interval: Smokers had a lower mean QT interval (0.34 sec) than non-smokers (0.36 sec), which was statistically significant (p 0.0001). Reports of a similar discovery were made by Dilaveris P et al.[14]

QTc interval: When compared to non-smokers, the QTC interval was statistically significant for smokers at 0.39 seconds. According to Dilaveris P et al[14] a similar finding was seen. Tobacco use by young male smokers alters the repolarization of the heart. Because of changes in heart rate between the two research groups, smokers and nonsmokers have different ventricular repolarization heterogeneity.[14]

T wave: Both smokers and non-smokers had normal T waves in Lead I and Lead II, Whereas in Lead III, 26% of smokers had a normal wave, 20% had a flat wave and 54% had an inverted wave compared to 40% normal, 16% flat, and 44% inverted wave among non-smokers, and this difference was discovered to have statistical significance i.e. highest percentage of flat and inverted waves found in smokers. Both smokers and non-smokers had normal T waves in Lead aVR and Lead aVL whereas, in Lead aVF, 92% of smokers had a normal wave and 8% had an inverted wave compared to 100% normal wave among non-smokers, and this difference was found to be statistically not significant i.e only smokers had 4% of the inverted wave. Non-smokers had a 48% of the inverted T wave in Lead V1 only and 100% of a normal wave in remaining Lead V2 to V6. Whereas among smokers,100% normal wave was present in Lead V5 and Lead V6. 68%, 24%, 24%, and 10% of non-smokers had inverted T wave in Lead V1, V2, V3, and V4 respectively. A flat wave was present in 8% and 2% of smokers in Lead V1 and V4, respectively. The difference in presence of abnormal waves among smokers and non-smokers in Lead V1 to V4 was found to be statistically significant. In a study by Baden L et al.,[20] P. Dilaveris, and others,[14] Smokers had lower R, S, and T wave amplitudes than nonsmokers, according to studies by Chatterjee S et al.[21] and Khan IS et al.[19] R, S, and T wave amplitudes have decreased, however, the reason behind this has not been established. A likely explanation for this finding is that smoking alters ventricular electrical activity directly. Non-specific alterations in R, S, and T waves are attributed to smoking, which causes atherosclerosis to progress more rapidly.[20]

Q wave: Q wave was present among 12%, 44%, and 52% of smokers compared to 8%, 26%, and 44% of non-smokers in Lead I, II, and III respectively. This difference was not statistically significant. In Lead aVR, none of the non-smokers had a Q wave but 68% of smokers had a Q wave and this difference was not statistically significant. In Lead aVL, 24% of smokers had a Q wave whereas only 10% of non-smokers had a Q wave and this vast difference was found to be statistically significant. In Lead aVF, the highest percentage of Q wave was seen among smokers (36%) compared to non-smokers (32%) and this was not statistically significant. There was no statistically significant difference in the occurrence of Q wave among smokers and non-smokers in Lead V1, V4, V5, and V6. No Q wave was seen among both smokers and non-smokers in Lead V2 and Lead V3. Gupta R et al[18] found there was a higher prevalence of hypertension and the presence of ECG-Q waves (higher prevalence of CHD) in smokers.

CONCLUSION

We can infer the following from this research's findings. Smokers had a statistically significant rise in heart rate compared to non-smokers, according to the study.

Compared to non-smokers, smokers had a statistically significant rise in systolic and diastolic blood pressure. People who smoke had a lower PR interval than those who don't smoke, according to a study published in the journal Pediatrics.

QT interval was significantly reduced in smokers compared with those who were nonsmokers.

There was a statistically significant increase in QTc interval in smokers when compared to non-smokers. Comparing smokers to nonsmokers, we found a statistically significant drop in the QRS axis. Smokers' T wave amplitude decreased significantly compared to non-smokers. Showed Non-smokers have a much lower incidence of Q wave than smokers. Smokers' ECG alterations and elevated systolic and diastolic blood pressure, even in the absence of cardiac illness, are explored in our study, albeit it is not comprehensive. Although we have a basic understanding of these alterations and there have been very few studies on the subject, more research into the impact of smoking on electrocardiograms is needed.

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