Study of Effects of Smoking on Electrocardiography in Smokers Compared to Non-smokers

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ABSTRACT

Introduction: Cigarette smoking is one of the major risk factor for cardiovascular diseases and epidemiological studies have established a gradient of risk; the highest for containing smokers, intermediate for reformed smokers and the lowest for non-smokers. Materials & Methodology: The present cross sectional study conducted in Department of physiology, D.Y.Patil Medical College, Kolhapur in male individuals above 18 years up to 70 years of age group selected from the general population. The aim of the study was to know electrocardiographic pattern in non-smokers, to study electrocardiographic changes in different group of smokers, to compare the electrocardiographic changes in non-smoker to smokers. After consent detailed history from subjects, electrocardiogram was recorded during resting state in supine position. The ECG results are evaluated for different parameters. Results: The smokers & heavy smokers showed decrease in P-R interval. There was decrease in QRS complex in smokers and heavy smokers. There was difference in R wave voltage in smokers compared to non-smokers. S-waves voltage in smokers and non-smokers showed difference. ST segment shows difference in smoker and heavy smoker group. T-P interval has decrease in heavy smokers to non-smokers which may alter the heart rate. There was highly significant difference showed in T-wave abnormality in smoker & heavy smoker compared to non-smoker. Conclusion: Smokers and heavy smokers are prone for cardiovascular risk. Social climate conducive for non-smoking may be helpful to combat the epidemic of tobacco smoking.

KEYWORDS: Cigarette smoking, Non-smokers, Smokers, Heavy smokers, ECG changes.

INTRODUCTION

Cigarette smoking is one of the major risk factor for cardiovascular diseases and epidemiological studies have established a gradient of risk; the highest for containing smokers, intermediate for reformed smokers and the lowest for non-smokers [1]. Men under 65 years of age smoking 25 or more cigarettes a day had a relative risk of developing coronary heart disease of 2.6 times that of non-smokers [2,3]. Tobacco smoking is well recognized risk factor for sudden cardiac death as well as other manifestations of coronary artery diseases [4,5,6].Smoking now is recognized as foremost environmental hazard to health [7,8].Cigarette smoking is one of the most harmful and addictive habits which is widespread all over the world [9].World Health Organization (WHO) estimate that about 1.3 billion people smoke globally. Most of these smokers are in developing countries like India. Smoking causes 4.9 million deaths in a year globally. Nearly 13,000 people die due to smoking every day [10].Cigarette smoking and other tobacco use currently accounts for one of every ten adults deaths. By 2030, the tobacco toll will reach 10 million human lives [11].It is estimated that 20% of the total deaths due to cardiovascular diseases is mainly due to smoking [9]. Smokers have a two fold increase in having myocardial infarction (MI) compared with non-smokers [12].Cigarette smokers are likely to develop IHD at a younger age and having it are most likely to die suddenly compared to non-smokers. Cigarette smoking is a common problem in India and also a major public health problem associated with morbidity and mortality. The prevalence of cigarette smoking has peaked among the adults. Tobacco smoking appears to have an independent impact on sudden death occurrence over and above its contribution to the development of coronary artery disease [4,5,13,14].
Realizing the importance of adverse effects of smoking, the present study has been undertaken to detect the electrocardiographic changes due to smoking at an earlier date and thereby assessing the cardiovascular status in healthy adult male smokers.

MATERIALS AND METHODS

The present cross sectional study has been conducted in the Department of Physiology, D.Y.Patil Medical College, Kolhapur. Subjects were male individuals above 18 years up to 70 years of age group selected from the general population. The subjects were grouped as follows: Group A (Non-smoker): consisted of 50 apparently healthy adult non-smokers. Group B (All Smokers): consisted 70 apparently healthy adult smokers. sub divided into following groups: Group B1 (smokers): 40 smokers consuming 1-19 cigarettes / day for >5 years. Group B2 (heavy smokers): 30 smokers consuming ≥ 20 cigarettes / day for > 5 years.

This study includes normal healthy individual smokers as per WHO's 10th revision of International Statistical Classification of Diseases and related health problems (ICD-10) criteria of harmful use [15]. Subjects below 18 years of age and smokers associated with systemic illness like diabetes mellitus, hypertension, bronchial asthma and others. Female population & subjects with other forms of smoking except cigarette are excluded.

Recording of Electrocardiogram:

Following detailed assessment of subject, a 12 lead electrocardiogram was recorded during the resting state. The subjects are made to lie in supine position on a bed of adequate size. The ECG was recorded using BPL 108 ECG machine. Then ECG was evaluated for different parameters and results were drawn.

RESULTS

Smoking has varied effects on the cardiovascular system. The quality, quantity, duration and frequency of smoking play an important role in determining whether it is beneficial or harmful to the cardiovascular system. Present study shows the more number of subjects from low & middle income group i.e. 41.67% & 37.50% respectively as compared to high income group (20.83%).

Heart Rate:

Table-1 shows, heart rates were 76.78 ± 5.97, 79.45 ± 11.64 & 80.97 ± 10.91 among non-smokers, smokers and heavy smokers respectively. Heart rates showed significant increase in heavy smokers to non-smokers (p < 0.05).

P-Wave:

P-wave duration (in seconds) in non-smokers, smokers and heavy smokers were 0.08 ±0.013, 0.07 ± 0.014 and 0.08 ± 0.016 respectively. P-wave amplitude (in milli volts) was 1.02 ± 0.25, 0.81 ± 0.29 and 0.88± 0.31 in non-smokers, smokers and heavy smokers respectively (Table-1) In this study there is change in duration and amplitude of P-wave and there was statistically significant difference seen in non-smoker to smokers, and smokers to heavy smokers (p < 0.05).

Multiple group comparisons: One way ANOVA, F-test, Pair wise comparisons : Student's 't' test, p < 0.05, p< 0.01 : Significant, p< 0.001: Highly significant, p>0.05 : Not significant (NS)

Table 1: Heart Rate & P- Wave in different groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Particulars</th>
<th>Non-smokers (A)</th>
<th>Smokers (B1=1-19 cigarettes/day)</th>
<th>Heavy smokers (B2=≥20 cigarettes/day)</th>
<th>Difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>F ratio</td>
</tr>
<tr>
<td>H.R. (beats/min)</td>
<td>Mean ± SD</td>
<td>76.78 ±5.97</td>
<td>79.45 ± 11.64</td>
<td>80.97 ± 10.91</td>
<td>2.013</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td>68 – 100</td>
<td>60 - 110</td>
<td>70 – 110</td>
<td>NS</td>
</tr>
<tr>
<td>P-wave</td>
<td>Mean ± SD</td>
<td>0.08 ±0.013</td>
<td>0.07 ± 0.014</td>
<td>0.08 ± 0.016</td>
<td>9.063</td>
</tr>
<tr>
<td>Duration (in sec)</td>
<td>Range</td>
<td>0.04 - 0.1</td>
<td>0.06 - 0.12</td>
<td>0.06 - 0.12</td>
<td>0.0002,</td>
</tr>
<tr>
<td>P-wave</td>
<td>Mean ± SD</td>
<td>1.02 ± 0.25</td>
<td>0.81 ± 0.29</td>
<td>0.88 ± 0.3130</td>
<td>6.3906</td>
</tr>
<tr>
<td>Amplitude (in sec)</td>
<td>Range</td>
<td>0.5 - 1.5</td>
<td>0.5 - 1.5</td>
<td>0.5 - 1.5</td>
<td>0.002,</td>
</tr>
</tbody>
</table>

Multiple group comparisons: One way ANOVA, F-test, Pair wise comparisons : Student's 't' test, p < 0.05, p< 0.01 : Significant, p< 0.001: Highly significant, p>0.05 : Not significant (NS)
P-R Interval:
P-R interval measurements (in seconds) in non-smokers, smokers and heavy smokers were 0.14 ± 0.02, 0.13 ± 0.02 & 0.12 ± 0.02 respectively (Table 2). Smokers & heavy smokers showed decrease in P-R interval and there was difference found in P-R interval of both smokers & heavy smokers as compared to non-smokers. (P < 0.05)

QRS Complex: QRS complex measurements (in seconds) were 0.08 ± 0.02, 0.06 ± 0.01, 0.05 ± 0.01 in non-smokers, smokers and heavy smokers respectively (Table 2). Our study showed that there was decrease in QRS complex in smokers as well as in heavy smokers (p < 0.001) compared to non-smoker group. When smokers and heavy smokers groups were compared there was also difference (p < 0.001).

QTc Interval: QTc interval values (in seconds) were 0.41 ± 0.05, 0.42 ± 0.03 & 0.41 ± 0.04 among non-smokers, smokers and heavy smoker groups respectively (Table 2). There was no difference (p > 0.05) seen when values of non-smokers, smokers and heavy smokers were compared.

Table 2: P-R interval, QRS Complex & QTc interval in different groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Particulars</th>
<th>Non-smokers(A)</th>
<th>Smokers (B1=1-19 cigarettes/day)</th>
<th>Heavy smokers (B2=≥20 cigarettes/day)</th>
<th>Difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-R interval</td>
<td>Mean ± SD</td>
<td>0.14 ± 0.02</td>
<td>0.13 ± 0.02</td>
<td>0.12 ± 0.02</td>
<td>F ratio: 4.7418, t = 2.293, p &lt; 0.05</td>
</tr>
<tr>
<td>(in sec)</td>
<td>Range</td>
<td>0.08 - 0.16</td>
<td>0.1 - 0.16</td>
<td>0.1 - 0.16</td>
<td></td>
</tr>
<tr>
<td>QRS complex</td>
<td>Mean ± SD</td>
<td>0.08 ± 0.0189</td>
<td>0.06 ± 0.012</td>
<td>0.05 ± 0.02</td>
<td>F ratio: 38.061, t = 5.660, p &lt; 0.001</td>
</tr>
<tr>
<td>(in sec)</td>
<td>Range</td>
<td>0.04 - 0.12</td>
<td>0.04 - 0.08</td>
<td>0.04 - 0.08</td>
<td></td>
</tr>
<tr>
<td>QTc interval</td>
<td>Mean ± SD</td>
<td>0.4 ± 0.0518</td>
<td>0.42 ± 0.034</td>
<td>0.42 ± 0.04</td>
<td>F ratio: 0.0337, t = 0.194, p = 0.064</td>
</tr>
<tr>
<td>(in sec)</td>
<td>Range</td>
<td>0.27 - 0.5</td>
<td>0.33 - 0.472</td>
<td>0.333 - 0.468</td>
<td></td>
</tr>
</tbody>
</table>

QRS Frontal Axis:
Axis measurements (in degrees) were 48.0 ± 23.47, 47.25 ± 19.08 & 40.0 ± 21.33 among non-smokers, smokers and heavy smokers respectively (Table-3). There was no difference found when values of all groups were compared (p > 0.05).

Ventricular Activation Time (V.A.T.):
Ventricular activation duration (in seconds) was compared in different groups. The values were 0.020 ± 0.002, 0.031 ± 0.005 & 0.031 ± 0.006 among non-smokers, smokers and heavy smokers respectively (Table-3). In our study statistically significant increase in VAT is seen in smokers and heavy smokers compared to non-smoker group (p < 0.001). There is no statistically significant difference between smokers and heavy smoker groups. But values in all the three groups are within normal range.

Voltage of R-wave:
The R-wave measurements were (in mm) 17.62 ± 3.16, 16.1 ± 3.20 & 16.83 ± 3.00 among non-smokers, smokers and heavy smoker groups respectively (Table-3). There is slight decrease in values of smokers and heavy smoker groups compared to non-smokers, and there was difference in smokers compared to non-smokers (p < 0.05).

Voltage of S-wave:
S-wave measurements (in mm) were in non-smokers, smoker and heavy smokers groups were 15.78 ± 3.82, 14.12 ± 3.29 & 15.5 ± 4.19 respectively (Table-3). When smokers and non-smokers were compared, there was difference (p < 0.05). No difference seen between non-smoker & heavy smokers.

T-P Interval:
T-P interval values (in seconds) were 0.29 ± 0.04, 1.70 ± 5.27 & 3.02 ± 8.66 among non-smokers, smokers and heavy smoker groups respectively (Table-4). In our study, T-P interval has statistically decreased seen in heavy smokers when compared with non-smokers (p< 0.05).

ST Segment:
In our study, ST segment abnormality was not seen in non-smokers. In smoker group five subjects showed non-specific ST segment and heavy smokers nine subjects with non-
specific ST segment (Table-4). When Chi-square test is applied there was difference seen in non-smoker to smoker (p < 0.01) and highly difference in non-smoker to heavy smoker group (p < 0.001).

T-wave: There was no difference in T-wave abnormality in non-smoker to smokers. But in heavy smokers group one subject showed T-wave elevation and seven subject with flattening of T-wave, showed highly significant difference in non-smoker to heavy smoker (p <0.001) & significant difference in smoker to heavy smoker (p<0.05).

Table 3: QRS Frontal axis, V.A.T, Voltage of R wave & Voltage of S wave in different groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Particular</th>
<th>Non-smokers (A)</th>
<th>Smokers (B1=1-19 cigarettes/day)</th>
<th>Heavy smokers (B2=≥20 cigarettes/day)</th>
<th>Difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>F ratio</td>
</tr>
<tr>
<td>QRS frontal Axis (in degrees)</td>
<td>Mean ± SD</td>
<td>48 ± 23.473</td>
<td>47.25 ± 19.08</td>
<td>40 ± 21.334</td>
<td>1.4356</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>0 – 90</td>
<td>0 – 90</td>
<td>0 – 60</td>
<td>0.242, NS</td>
</tr>
<tr>
<td>V.A.T. (in sec)</td>
<td>Mean ± SD</td>
<td>0.02 ± 0.002</td>
<td>0.031 ± 0.005</td>
<td>0.03 ± 0.01</td>
<td>79.395</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>0.02 - 0.03</td>
<td>0.02 - 0.04</td>
<td>0.02 - 0.04</td>
<td>0.0000, p &lt; 0.001</td>
</tr>
<tr>
<td>Voltage of R-wave (in mm)</td>
<td>Mean ± SD</td>
<td>17.62 ± 3.161</td>
<td>16.1 ± 3.209</td>
<td>16.83 ± 3.01</td>
<td>2.622</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>10 - 23</td>
<td>10 - 22</td>
<td>12 - 22</td>
<td>0.077, NS</td>
</tr>
<tr>
<td>Voltage of S-wave (in mm)</td>
<td>Mean ± SD</td>
<td>15.78 ± 3.82</td>
<td>14.12 ± 3.2909</td>
<td>15.5 ± 4.19</td>
<td>2.323</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>08-026</td>
<td>09-020</td>
<td>9-025</td>
<td>0.102, NS</td>
</tr>
</tbody>
</table>

Table 4: T-P interval, ST Segment & T-Wave in different groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Particular</th>
<th>Non-smokers (A)</th>
<th>Smokers (B1=1-19 cigarettes/day)</th>
<th>Heavy smokers (B2=≥20 cigarettes/day)</th>
<th>Difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>F ratio</td>
</tr>
<tr>
<td>T-P interval (in sec)</td>
<td>Mean ± SD</td>
<td>0.29 ± 0.04</td>
<td>1.709 ± 5.2753</td>
<td>3.02 ± 8.66</td>
<td>2.582</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>0.12 - 0.34</td>
<td>0.1 – 20</td>
<td>0.1 - 32</td>
<td>0.080, NS</td>
</tr>
<tr>
<td>ST segment</td>
<td>IE</td>
<td>50 (100%)</td>
<td>35 (87.5%)</td>
<td>21 (70%)</td>
<td>( \chi^2 = 0.015, P &lt; 0.001 )</td>
</tr>
<tr>
<td></td>
<td>NSST</td>
<td>5 (12.5%)</td>
<td>9 (30%)</td>
<td>( \chi^2 = 0.146, NS )</td>
<td>0.001, p &lt; 0.05</td>
</tr>
<tr>
<td>T-Wave (in sec)</td>
<td>N</td>
<td>50 (100%)</td>
<td>37 (92.5%)</td>
<td>22 (73.3%)</td>
<td>( \chi^2 = 0.0146, NS )</td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>2 (5%)</td>
<td>1 (3.3%)</td>
<td>( \chi^2 = 0.0146, NS )</td>
<td>0.001, p &lt; 0.05</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1 (2.5%)</td>
<td>7 (23.3%)</td>
<td>( \chi^2 = 0.0146, NS )</td>
<td>0.001, p &lt; 0.05</td>
</tr>
</tbody>
</table>

\( \chi^2 \): Chi square, IE: Isoelectric, N: Normal, NSST: Non-specific ST segment, F: Flattening, E: Elevated
DISCUSSION

Many workers have extensively studied the effects of smoking on the heart throughout the world over a long period of time.

Heart Rate: Benowitz et al [12] showed that infusion of nicotine in 14 healthy young men increases the heart rate within 5-10 minutes. Mujtaba et al [16] have shown that smoking increased the heart rate from 72 ± 3 to 96 ± 3. The increase in heart rate could be due to stimulation of sympathetic ganglia and discharge of catecholamines from adrenal medulla.

P-Wave: Our result is in agreement with Khan et al [17] showed 8% smokers with P pulmonale on their ECG & Sharma et al. Ahn Von found that Cigarette smoking during hypoxia increased the amplitude of the P waves. This might be due to development of cor pulmonale subsequently producing right atria hypertrophy as a result of chronic smoking[1].

P-R Interval:
Cigarette smoking increases the velocity of conduction and shortens the effective refractory period at the AV node [18]. Mujtaba et al [16] found that in smokers there was a little decrease in the duration of P-R interval. The probable cause of shorter P-R interval is due to faster heart beat among the smokers. But it was not statistically significant. The subjects in their study were habitual smokers between 20-45 years of age with more than 10 years of smoking habit.

QRS Complex:
Mujtaba et al [16] found a slight decrease in QRS duration in smokers, though it was not significant. Smoking produces ischemia by causing vasoconstriction of coronary arteries.

Ventricular Activation Time (V.A.T.):
Ventricular activation time is the time taken for impulse to traverse the myocardium from the endocardial to the epicardial surface. Goni M et al [19] studied voltage changes in 200 healthy asymptomatic males, based in Murphy's multiple electrocardiographic criteria. He observed abnormal voltage variation in same subjects. But they give no explanations for these changes.

Voltage of R-wave:
In Venkatesh et al [20] there is slight decrease in the voltage of R-wave in smokers and alcoholics compared to non-smokers, but is not statistically significant.

Voltage of S-wave:
Khan et al [17] showed, there are no significant change in the mean ±SD amplitude of S wave in smokers and non-smokers (p>0.05). But the result is significant between group A and group B2 (p<0.05) and very significant between group B1 and group B3 (p<0.01).

T-P Interval:
In Khan et al [17] the T-P interval showed statistically significant decrease in smokers and alcoholics compared to non-smokers. The ECG changes are T-P phenomenon i.e. close approximation of T and P wave.

ST Segment:
ST segment deviation from isoelectric line is a predictor of future coronary events in asymptomatic population [21].

T-wave:
Baden et al [22] Chatterjee et al also found that tobacco smoking or injection of nicotine during hypoxia increased heart rate and the decreased the amplitude of T or flattened the T wave mainly due to increased sympathetic tone and probably due to increased adrenal secretion.

CONCLUSION

Present study showed significant increase in Heart Rate, Ventricular Activation Time in smokers. There was significant decrease in P-R interval, QRS complex & T-P interval in smokers. There was difference in R wave voltage, S-wave voltage, ST segment & T-wave abnormality in smokers. Results showed that smokers are prone for cardiovascular risk. Smoking control measures viz. illustrate smoking control actions in a case, prevent onset of smoking habit, change smoking behavior & reduce smoking rate, and establish social climate conducive for non-smoking may be helpful to combat epidemic of tobacco smoking.

REFERENCES


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