# Chronic Effect of Smoking on the Electrocardiogram

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SUMMARY

The electrocardiograms (ECG) of 232 male non-smokers and 224 male smokers, aged 20–60 years, devoid of cardiovascular diseases, were studied. Among these subjects 5.2% of non-smokers and 6.7% of smokers had pathological ECGs. Non-pathological ECGs of 220 non-smokers and 209 smokers were analyzed for R, S and T-wave amplitudes, P and QRS axes and P-R, QRS and Q-Tc intervals. The 2 groups did not differ significantly from each other for R and T-wave amplitudes in any of the age groups except for 40–60 years, in which R-amplitude obtained from standard limb leads was significantly lower in smokers. S-amplitude recorded from standard limb leads was significantly lower in smokers of all ages combined. The reverse phenomenon was noted for S-amplitude obtained from precordial leads. R, S and T-amplitudes decreased with the advancement of age at a relatively higher rate in smokers. These waves had significant negative correlation with pack years of smoking habit. QRS and P axes differed significantly between smokers and non-smokers. The rate of shifting of these axes to the left with increasing age was relatively higher in non-smokers. Lung function did not show any relation to electrocardiogram in normal healthy subjects. These results indicate that aging affects electrocardiographic wave patterns and that this aging effect is modified by long term smoking.

**Additional Indexing Words:**

Smoking Electrocardiogram Aging Body mass index Lung function

IDENTIFICATION of cigarette smoking as one of the main causative factors of coronary heart disease and chronic obstructive pulmonary disease[1),2)] has increased interest in the study of cardiopulmonary function of healthy smokers. The deleterious effects of long term cigarette smoking

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on maximal aerobic capacity and pulmonary function of healthy asymptomatic subjects were demonstrated in our earlier reports.[3),4)] The acute effects of smoking on electrocardiogram (ECG) and blood pressure were also reported.[5)] The present study has evaluated the difference in resting electrocardiogram between healthy smokers and non-smokers and the effect of aging on it in these 2 groups.

Electrocardiograms were reported to have excellent correlations with chronic obstructive pulmonary disease.[6)-8)] But there has been no systematic attempt to investigate the interrelationship of lung function and electrocardiogram in healthy adult subjects. The need for such investigations was also realized by Rose et al.[9)] Therefore, this study also evaluated the interrelationships between lung function and ECG in healthy subjects.

## Materials and Methods

A sample of 232 healthy male non-smokers and 224 healthy male smokers of the same age range was randomly selected from the population of university employees and students. The subjects were carefully screened at entry according to health criteria.[10),11)] Those individuals with a history of diabetes mellitus, hypertension (supine systolic blood pressure greater than 150 and diastolic blood pressure greater than 90 mmHg), coronary heart disease, chronic obstructive pulmonary disease, rheumatic heart disease, chest deformities, metabolic disorders, renal disease, collagenosis and anemia were not included.

The electrocardiograms were recorded between 10 a.m. and 12 noon. The 12-lead electrocardiograms were recorded from each subject after an overnight abstinence from smoking on a BPL-Cardiart 108T model electrocardiograph. The ECG recorder used in this study met the performance requirements of single channel ECG recorders for conventional ECG recording recommended by the American Heart Association.[12)] The ECGs thus obtained were first classified into categories: (a) pathological ECG (ECG positive) and (b) non-pathological ECG (ECG negative). In interpreting the present ECGs for pathological ECG, the WHO version of the Minnesota Code[9)] was used. Electrocardiograms with one or several of the following changes were designated as ECG positive:

i) Minnesota Code 1.1 (Major 'Q')
ii) ,, ,, 4.1, 5.1, 5.2 (Major ST/T depression)
iii) ,, ,, 6.1 (Complete AV block)
7.1 (Complete LBBB) and
8.3 (Arterial fibrillation)

The others were considered to be ECG negative.

The electrocardiographic variables on non-pathological ECGs were analyzed. Those examined in this investigation included R, S and T-wave amplitude, QRS axis, P-axis and P-R, QRS and Q-$T_c$ interval durations. Both R and S wave amplitudes were measured according to Baden et al.[13]

1) R and S amplitudes were measured in leads I, II and III; the largest R and S amplitudes in these leads were utilized. R-amplitudes and S-amplitudes thus obtained were designated as ' R-amplitude A' and ' S-amplitude A', respectively.

2) The largest R-amplitude from $V_4$, $V_5$ and $V_6$ and S-amplitude from $V_1$, $V_2$ and $V_3$ were utilized. R- and S-amplitudes thus obtained were designated as ' R-amplitude B ' and ' S-amplitude B ', respectively.

Amplitude of the T-wave was measured in lead $V_5$. P-R, QRS and Q-T intervals were obtained from lead II. Wave amplitudes were measured to the nearest millimeter and durations were measured to the hundredth of a second. QRS and P-axes were calculated by Einthoven's triangle method. Q-T intervals were corrected (Q-$T_c$) for heart rate, and were obtained by dividing the measured Q-T interval by the square root of the R-R duration.

Spirometric lung functions were measured from each subject according to the method described elsewhere.[4] From each spirogram forced vital capacity (FVC), forced expiratory volume in one second ($FEV_1$) and mid expiratory flow rate (FEF 25–75%) were calculated and were expressed in body temperature, pressure saturated with water vapor (BTPS).

Body mass index (BMI) was calculated from the following formula[14]:

$$\text{BMI} = \frac{\text{weight in kg}}{(\text{height in meters})^2}$$

## Results

Table I represents the prevalence of pathological ECG in different age groups of smokers and non-smokers. In the youngest age group pathological

Table I. Pathological ECG in Different Age Groups of Smokers and Non-smokers

| Age group | Non-smoker n=232 | Smoker n=224 | Level of significance |
|---|---|---|---|
| 20-29 | 0 ( 0/65) | 0 ( 0/60) | NS |
| 30-39 | 2.9 ( 2/68) | 3.6 ( 2/56) | NS |
| 40-60 | 9.9 (10/99) | 12.0 (13/108) | NS |
| 20-60 | 5.2 (12/232) | 6.7 (15/224) | NS |

Values in parentheses indicate number of subjects with pathological ECG out of total subjects.

Table II. Physical Characteristics and Lung Functions of

| Age group | Category | n | Age (yrs) | Height (cm) | Weight (kg) |
|---|---|---|---|---|---|
| 20-29 | Non-smoker | 65 | 24.5± 2.94 | 165.9±6.21 | 55.5±7.66 |
| | Smoker | 60 | 25.7± 2.64 | 166.5±5.98 | 54.8±6.36 |
| | Level of p | | NS | NS | NS |
| 30-39 | Non-smoker | 66 | 36.1± 2.77 | 166.0±6.66 | 56.5±7.77 |
| | Smoker | 54 | 35.0± 2.87 | 166.2±6.32 | 55.3±6.98 |
| | Level of p | | NS | NS | NS |
| 40-60 | Non-smoker | 89 | 48.5± 6.57 | 166.2±6.36 | 57.7±8.51 |
| | Smoker | 95 | 49.1± 6.46 | 165.3±6.08 | 56.2±7.06 |
| | Level of p | | NS | NS | NS |
| 20-60 | Non-smoker | 220 | 37.7±11.36 | 165.9±6.16 | 56.7±8.02 |
| | Smoker | 209 | 38.7±10.92 | 165.8±6.23 | 55.6±7.25 |
| | Level of p | | NS | NS | NS |

Values are mean±SD. NS=non-significant; † BMI=body mass index = $\frac{\text{weight in gm}}{(\text{height in cm})^2}$;

Table III. Mean±SD Values of Electrocardiographic Variables for Smokers and Non-smokers of Different Age Groups

| Age group | Category | Heart rate (beats/min) | P-R interval (1/100 sec) | Q-$T_c$ interval (1/100 sec) | QRS-duration (1/100 sec) | QRS-axis (degree) | P-axis (degree) |
|---|---|---|---|---|---|---|---|
| 20-29 | Non-smoker | 75±6.00 | 13.66±1.36 | 38.15±1.92 | 7.52±0.18 | 67.0±20.8 | 70.6±20.6 |
| | Smoker | 76±5.59 | 13.26±1.55 | 38.42±1.80 | 7.55±0.20 | 65.1±21.3 | 71.5±22.5 |
| | Level of p | NS | NS | NS | NS | NS | NS |
| 30-39 | Non-smoker | 74±6.27 | 14.92±1.45 | 37.98±1.85 | 7.76±0.24 | 48.7±25.3 | 55.0±18.2 |
| | Smoker | 79±6.27 | 14.32±1.51 | 38.36±1.92 | 7.56±0.18 | 55.2±27.6 | 64.7±19.6 |
| | Level of p | <0.01 | NS | NS | NS | NS | <0.01 |
| 40-60 | Non-smoker | 74±6.09 | 16.02±1.73 | 37.99±2.10 | 7.88±0.28 | 38.3±31.5 | 47.3±17.4 |
| | Smoker | 78±5.27 | 15.26±1.33 | 38.03±1.82 | 7.62±0.19 | 49.7±35.6 | 57.5±17.2 |
| | Level of p | <0.01 | NS | NS | NS | <0.06 | <0.001 |
| 20-60 | Non-smoker | 74±5.25 | 14.99±1.67 | 38.03±2.22 | 7.73±0.19 | 49.9±28.6 | 56.5±19.32 |
| | Smoker | 78±5.66 | 14.44±1.56 | 38.22±1.45 | 7.58±0.20 | 55.5±30.2 | 63.4±18.60 |
| | Level of p | <0.01 | NS | NS | NS | <0.02 | <0.001 |

ECGs were absent. The prevalence of pathological ECG was slightly higher among smokers (6.7%) than non-smokers (5.2%).

Table II describes the physical characteristics and pulmonary functions of smokers and non-smokers of different age groups. It was found that all the physical characteristics were about the same in smokers and non-

Smokers and Non-smokers in Different Age Groups

| BSA ($cm^2$) | †BMI ($gm/cm^2$) | *Pack-years | FVC (1) | $FEV_1$ (l) | FEF 25-75% (l/min) |
|---|---|---|---|---|---|
| 1.61±0.12 | 2.02±0.06 | — | 3.926±0.62 | 3.408±0.54 | 272.0±69.6 |
| 1.60±0.11 | 1.98±0.07 | 5.26± 3.35 | 3.865±0.58 | 3.194±0.52 | 238.2±62.8 |
| NS | NS | | NS | <0.05 | <0.01 |
| 1.62±0.12 | 2.05±0.06 | — | 3.707±0.66 | 3.095±0.57 | 240.7±53.3 |
| 1.61±0.10 | 2.00±0.07 | 12.06± 8.34 | 3.589±0.59 | 2.842±0.56 | 198.5±49.2 |
| NS | NS | | NS | <0.02 | <0.001 |
| 1.64±0.13 | 2.10±0.09 | — | 3.526±0.60 | 2.768±0.46 | 209.5±50.2 |
| 1.62±0.12 | 2.05±0.09 | 19.11±10.62 | 3.233±0.63 | 2.373±0.50 | 152.1±45.6 |
| NS | NS | | <0.01 | <0.001 | <0.001 |
| 1.62±0.11 | 2.05±0.08 | — | 3.698±0.61 | 3.058±0.54 | 237.3±68.7 |
| 1.61±0.10 | 2.01±0.09 | 13.31± 9.88 | 3.526±0.62 | 2.730±0.57 | 189.0±65.5 |
| NS | NS | | <0.01 | <0.001 | <0.001 |

* Pack-years = no. of packets of cigarettes consumed per day × no. of years of smoking.

Table IV. Mean±SD Values of Electrocardiographic Variables for Smokers and Non-smokers of Different Age Groups

| Age group | Category | R amplitude A (mm) | S amplitude A (mm) | R amplitude B (mm) | S amplitude B (mm) | $T_{v5}$ amplitude (mm) |
|---|---|---|---|---|---|---|
| 20-29 | Non-smoker | 11.80±3.51 | 1.98±2.21 | 17.93±6.17 | 16.21±4.98 | 4.83±2.23 |
| | Smoker | 11.82±3.58 | 1.53±2.12 | 17.71±6.51 | 17.51±5.66 | 6.59±2.56 |
| | Level of p | NS | NS | NS | NS | NS |
| 30-39 | Non-smoker | 9.71±3.87 | 1.74±2.07 | 17.1 ±4.52 | 13.31±4.25 | 4.58±1.66 |
| | Smoker | 9.51±3.85 | 1.44±2.02 | 16.65±4.31 | 14.01±4.23 | 4.73±1.69 |
| | Level of p | NS | NS | NS | NS | NS |
| 40-60 | Non-smoker | 7.40±2.95 | 1.38±1.85 | 15.02±4.02 | 10.43±3.98 | 3.60±1.35 |
| | Smoker | 6.52±2.77 | 1.25±1.73 | 14.00±3.97 | 10.20±3.22 | 3.65±1.32 |
| | Level of p | <0.05 | NS | NS | NS | NS |
| 20-60 | Non-smoker | 9.39±3.47 | 1.66±1.55 | 16.50±4.55 | 13.00±4.60 | 4.25±1.86 |
| | Smoker | 8.81±3.37 | 1.38±1.32 | 15.75±4.22 | 14.08±5.05 | 4.77±2.15 |
| | Level of p | NS | <0.05 | NS | <0.01 | NS |

R amplitude A = the largest R wave amplitude from leads I, II and III; R amplitude B = the largest R wave amplitude from leads $V_4$, $V_5$ and $V_6$; S amplitude A = the largest S wave amplitude from leads I, II and III; S amplitude B = the largest S wave amplitude from leads $V_1$, $V_2$ and $V_3$; $T_{v5}$ amplitude = T wave amplitude from lead $V_5$.

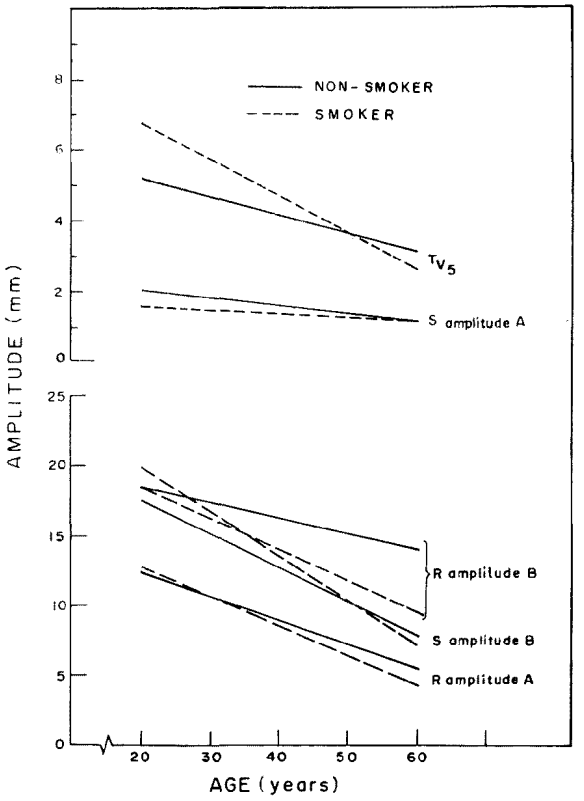


Fig. 1. Relationship of different electrocardiographic wave amplitudes to age in smokers and non-smokers. Regression lines for smokers and non-smokers are shown.

smokers. Lung function variables were significantly lower in smokers in respect to their non-smoking counterparts ($p<0.01$) in almost all age groups.

Tables III and IV represent the electrocardiographic variables of smokers and non-smokers. Analysis of variance revealed that P-axis, QRS-axis and S-wave differed significantly between smokers and non-smokers. 'R-wave amplitude A' was significantly lower in smokers in the age group of 40–60 years ($p<0.05$). No significant difference was noted for 'R-amplitude B' between smokers and non-smokers. The S-wave amplitude obtained from limb leads (S-wave amplitude A) was significantly lower in smokers of all ages combined (20–60 years) while the S-wave amplitude obtained from precordial leads was significantly higher in the smokers aged 20–60 years. In other age groups, although R and S (S-amplitude A) waves were found to be lower in smokers, these differences did not achieve significance. T-wave amplitude was relatively higher in smokers, but with in-

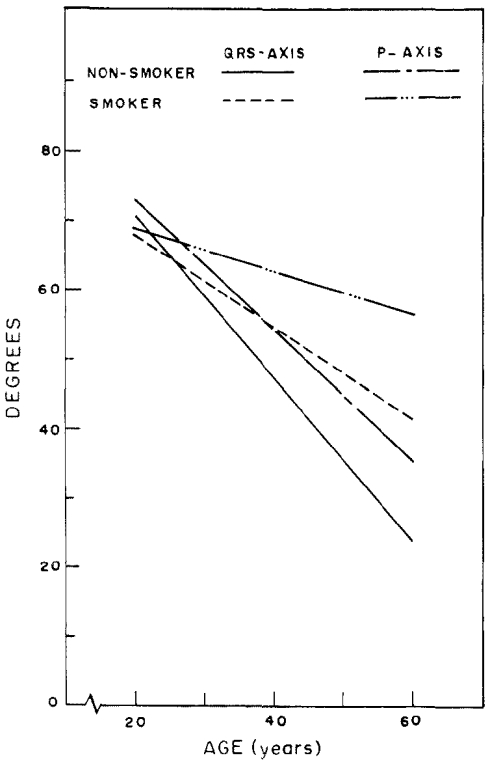


Fig. 2. Relationship of P- and QRS-axes to age in smokers and non-smokers. Regression lines of smokers and non-smokers are shown.

crease of age T-wave amplitude showed a greater fall in this group. Heart rates were significantly higher in smokers. P-R interval was relatively shorter in smokers. Q-$T_c$ interval and QRS duration did not show any significant difference.

Figures 1 and 2 represent the relationship of age to different electrocardiographic wave amplitudes, P-axis and QRS-axis. It was observed that with the increase of age, the wave amplitudes declined faster in smokers. Shifting of P and QRS axes to the left with increasing age was faster in non-smokers. The relationship of these waves to pack years of smoking (pack-years=no. of packets (20's) smoked a day×duration of smoking in years) is graphically shown in Fig. 3. It reveals that these measurements were decreased with the increase in amount of cigarettes consumed. P and QRS-axes were found not to be related to pack-years of smoking. The relationships of electrocardiographic variables to body mass index (BMI) are shown in Figs. 4 and 5, which demonstrate that with an increase of physical charac-

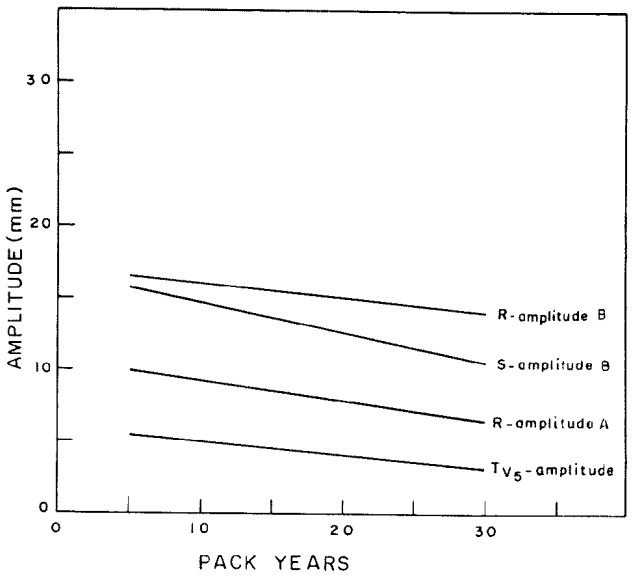


Fig. 3. Relationship of different electrocardiographic wave amplitudes to pack-years of smoking (pack-years=no. of packets (20's) smoked a day × duration of smoking in years).

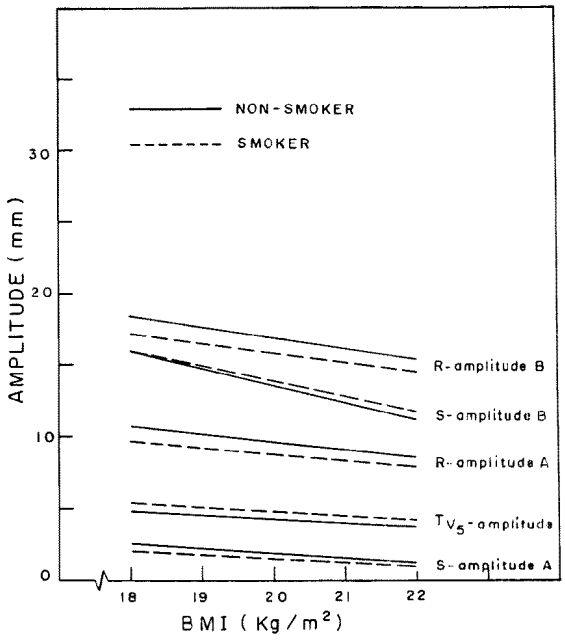


Fig. 4. Relationship of different wave amplitudes to BMI (regression lines are shown).

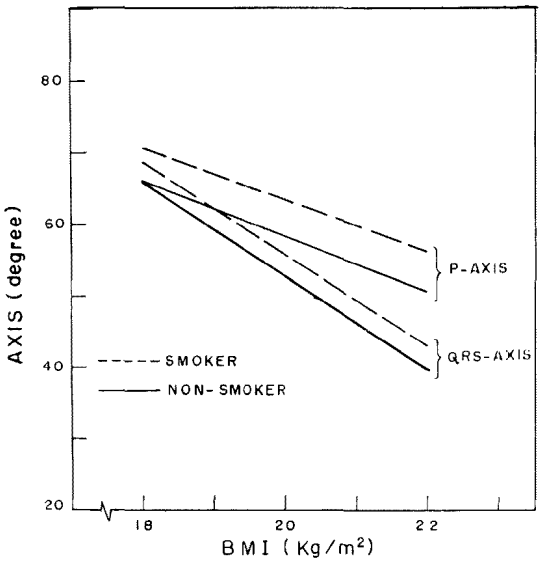


Fig. 5. Relationship of P- and QRS-axes to BMI (regression lines are shown).

teristics, these measures were decreased. The correlation coefficients of different wave variables with pulmonary function measurements were found to be insignificant and hence are not related to each other.

## Discussion

On the basis of data collected from 232 non-smokers and 224 smokers certain cross sectional smoking differences were observed. Among these subjects 5.2% of non-smokers and 6.7% of smokers had pathological ECGs. This difference between the 2 categories of subjects was not significant and in the youngest age group it was not present at all. Erikssen and Enger[15] reported a higher prevalence of pathological ECG in comparison to the present study. They reported the presence of pathological ECG in 10.4% and 13.3% of non-smokers and smokers, respectively. The comparatively lower prevalence of pathological ECG in our subjects might be due to the exclusion of subjects with a known history of cardiovascular diseases from the present study.

The non-pathological ECGs of healthy male smokers (n=209) and healthy male non-smokers (n=220) were analyzed for different electrocardiographic variables with the greatest care. Analysis of data revealed that smokers were comparable to non-smokers for their mean age, height, body

weight, body mass index (BMI) and body surface area.

It was observed that the P-R interval was shorter in smokers than in non-smokers. The interval duration measurements are representative of the conduction time of electrical currents in the heart. In the present study smokers had higher resting heart rates than non-smokers. Similar results were also reported by Goldbeust and Medalic.[16] This higher resting heart rate may account wholly or in part for the difference noted in P-R interval between smokers and non-smokers. P-R interval was found to be related to age. It increased with age and the rate of increase was higher in non-smokers than in smokers. This can be explained on the basis of an increase in resting heart rate in smokers with increasing age. It was not found to be correlated with body mass index, hence it was not related to obesity.

In most of the leads, the R-wave is the largest positive deflection reflecting the electrical activity of the heart that occurs during ventricular depolarization. The S-wave signals the end of depolarization. Two studies have reported lower R and S wave amplitudes in older individuals suggesting that there is less ventricular electrical force in the heart with increasing age.[2),11] The present findings for R and S waves are in agreement with these observations. There was a significant difference in S-amplitude between smokers and non-smokers in the 20–60 year age range. The higher rate of decline of R-wave and S-wave (S-amplitude B) with the increase of age in smokers (Fig. 1) and the significant negative correlation of these waves to pack years of smoking suggest that the effect of aging on these waves are modified by smoking. BMI was correlated positively to these two variables. This indicates that overweight persons would show low R and S amplitudes on their electrocardiogram.

From the previous studies it is known that T-wave amplitude diminishes with the advancement of age.[17),18] The present study also revealed a decrease in the T-wave amplitude with an increase in age, suggesting a diminution of repolarization current with age. It was observed that smoking modifies the effect of aging on the T-wave (Fig. 1). The higher rate of decline of T-wave amplitude with age in smokers and its significant negative correlation with pack-years of smoking suggest that smoking enhances the age-related change of this variable. Previous studies also reported that T-wave amplitude is affected by smoking.[11),13),19]

The mechanism by which R, S and T wave amplitudes decrease more in smokers with an increase in age is not clear. It is thought that smoking has a direct effect on ventricular electrical activity. An alternative explanation is that smoking results in an acceleration of atherosclerosis, which in turn leads to non-specific R, S and T-wave changes. One previous study has

reported an increase in chest circumference in normal smoking men with an increase in age.[20] This is thought to result from an increase in lung compliance. Increased chest size and hence increased distance of the heart from the chest wall and elongation of the heart within the chest, could potentially explain the decrease in ventricular electrical forces for both depolarization and repolarization. Though BMI was significantly associated with R, S and T-wave amplitudes, the differences in these waves between smokers and non-smokers cannot be attributed to BMI as both groups were matched for their BMI and body weight.

The QRS axis of the subjects shifted to the left with an increase of age (Fig. 2). This is consistent with the observations made by others.[13),21)-23)] The present study revealed a significant difference in QRS-axis between smokers and non-smokers ($p<0.02$). In non-smokers the frontal axis was more leftward than that in their smoker counterparts. The rate of shift of this axis towards the left with increasing age was found to be lower in the smokers than in the non-smokers (Fig. 2). The P-axis reflected a result similar to that of the QRS-axis. One previous study reported that the P-axis was shifted more towards the right in smokers than in non-smokers.[24)] Our results are in accordance with the above findings.

These findings indicate that the age-related change in the QRS-axis was affected by smoking which might have a tendency to shift the axis towards the right. Body mass index showed a negative correlation with the QRS and P axes. The greater the body weight the greater the shift of the axis to the left. The reason for this shift is probably as follows. The presence of obesity or a large gain in weight would lead to increased abdominal girth and the presence of a higher hemidiaphragm.[22)] This, in turn, would produce a mechanical effect on the heart resulting in a left axis shift. Our results as well as those of others seem to confirm the above view.[21)]

Although in smokers as a group, a change in electrocardiographic variables was noticed, it was not present in each individual. Some of them exhibited ECG patterns similar to those of non-smokers.

In the present study, an attempt was made to correlate pulmonary function measurements that reflect airway obstructions in the subjects to electrocardiographic variables. Forced vital capacity (FVC), forced expiratory volume in one second ($FEV_1$) and maximum mid expiratory flow rate (FEF 25–75%) did not show any relation to electrocardiographic variables. Electrocardiograms were reported to have excellent correlation with pulmonary functions in patients with obstructive pulmonary diseases.[6)-8)] But, from the present study it seems that normal or healthy individuals with normal lung function do not reveal a significant association between lung

function and electrocardiographic changes.

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