## ORIGINAL ARTICLE

# A COMPARISON OF 12 LEAD ECG STATUS OF TOBACCO SMOKERS, TOBACCO CHEWERS AND NONTOBACCO USERS

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## ABSTRACT

**Introduction:** According to World Health Report (2002)[1] tobacco is the most preventable cause of overall mortality as well as cardiovascular mortality worldwide. Cigarette smoking has long been proved as a causative factor for CAD (coronary artery disease) but a very little work has been done on smokeless tobacco so present work done in Muzaffarnagar a city of Western Uttar Pradesh aims to evaluate the Electrocardiographic status of habitual smokers and chewers of tobacco,to have a comparative analysis of their ECG status with non tobacco users not smoking or chewing.

**Objective:** The objective is to find if chronic smoking or chewing tobacco produce any ECG changes.

**Methodology:** Three groups each having 30 human subjects were taken and 12 lead ECG record was taken and measured for various intervals, segment and amplitudes.

**Results:** Heart rate is increased significantly in smokers and chewers as compared to control subjects .There is significant shortening of duration of QRS complex in smokers and chewers as compared to control. There is significant shortening of TP interval in tobacco smokers and chewers as compared to control.

**Conclusion:** ECG demonstrates only significant increase in resting heart rate, with subsequent shortening in TP interval and QRS duration in tobacco smokers and tobacco chewers as compared to non tobacco users.Otherwise ECG status of chronic smokers and chewers is normal with no significant abnormality.

**Keywords:** ECG, Smoker, Tobacco chewer, cardiac disease

## INTRODUCTION

According to World Health Report (2002)[1] tobacco is the most preventable cause of overall mortality as well as cardiovascular mortality worldwide. Smoking has resulted in two fold increase in the risk of Coronary Artery Diseases. The consumption of nicotine is the single biggest avoidable cause of death and disability. The World Health Organization (WHO) predicts that 70% of the deaths from smoking-related illnesses will occur in low- and middle-income countries by 2020. Uttar Pradesh is the third largest cultivator of tobacco leaf in India. Tobacco is produced mainly in Mainpuri, Muradabad, Farrukhabad and Etah districts in UP. There are many Cigarette and gutka factories in the state[2,3]. MMWR, US [4], Price JF et al [5], Black HR et al [6], Jonas MA et al [7], Willete et al [8]have conducted epidemiologic studies which strongly support the assertion that cigarette smoking (CS) in both men and women increases the incidence of myocardial infarction (MI) and fatal coronary artery disease. Smokers are found worldwide while tobacco –chewers are restricted to South East Asia.

A number of studies have strongly correlated the role of smoking in causative factor for CAD but a very little work has been done on tobacco chewers prevalent in Uttar Pradesh where it is chewed in the form of paan, gutka, khaini, misri etc.

## OBJECTIVE OF THE STUDY

To do a comparative analysis of cardiovascular status of any form of exclusive tobacco smoker (cigarette, bidi etc) and any form of exclusive tobacco chewer (paan, gutka, khaini, misri) with that of nontobacco users (neither smoking nor chewing) by taking a 12 lead ECG record of these group of people.

## MATERIAL AND METHOD

The present study was conducted in Muzaffarnagar Medical College Hospital and Campus Muzaffarnagar from a period of July 2012 to June 2013 with permission of ethical committee on general population residing in and around campus.The subjects are working as ward boys-girls , sweepwers, peons ,hostel attendents

and also the patient attendents.The subjects were of both sexes and were of age group 20 -60 years with atleast 5 years of tobacco use in any form smoked or chewed .The mean age of subjects for study was 40 yrs. The subjects were included in the study after proper consent .The subjects were divided in three groups.

**Group A** Normal Population (Neither smoked nor chewed)

**Group B** Exclusive Smokers (Only smoked any form of tobacco cigarette, bidi etc and not chewing)

**Group C** Exclusive Chewers (Only chewed any form of tobacco paan, khaini, etc and not smoking)

The subjects having both the habits, any history of cardiovascular illness ,renal disease, diabetes, obstructive or restrictive lung disease were excluded from the study.

These three groups containing at least 30 human subjects were evaluated for Standard 12 lead Electrocardiogram after proper consent in the deptt. of Physioology.ECG strips were taken and analysed for amplitudes and intervals.

**Statistical Evaluation:** It was done by. Mean**,** Standard Deviation(S.D.) and T-Test using SPSS 17 software.

## OBSERVATIONS

ECG observations among all three study groups were given in table 1.

**Table 1: ECG parameters among Smoker, Chewer and Study group**

| Parameters | Control | Smoker | Chewer |
|---|---|---|---|
| P amp. | 1.02±0.20 | 1.01±0.30 | 0.99±0.37 |
| P durat. | 0.08±0.02 | 0.09±0.01 | 0.08±0.01 |
| PR interval | 0.14±0.02 | 0.16±0.02 | 0.16±0.01 |
| QRS complex (sec.) | 0.08±0.01 | 0.05±0.01 | 0.06±0.02 |
| QT interval | 0.40±0.06 | 0.38±0.04 | 0.41±0.04 |
| T amp. | 0.552±1.02 | 0.56±1.06 | 0.57±1.04 |
| TP interval | 0.29±0.04 | 0.24±0.07 | 0.25±0.06 |
| ST mv | 0.115±0.033 | 0.122±0.040 | 0.118±0.038 |
| MEA | 51±23.7 | 42±22.7 | 44±20.7 |
| Heart Rate | 72.24±8.4 | 88.56±5.06 | 85.29±4.7 |

Pamp, Tamp, STmv are in milivolts; durat., PRinterval, QRScomplex, QTinterval, TPinterval are in seconds; MEA mean electric axis in degrees; Heart rate per minute.

As shown in table I and table II resting heart rate of tobacco smokers and chewers was increased significantly ($p<0.001$) * as compare to non tobacco users population but when tobacco smokers and chewers were compared for any significant change p was found to be >.05 i.e. non significant.

Similarly, duration of QRS complex and TP interval were decreased significantly in tobacco users (both smokers and chewers) as compared to non tobacco users, no significant change in this parameter when compared with each other, i.e. between smokers and chewers. Rest all the parameters in any group does not show any significant change.

**Table 2: Comparison of ECG Parameters among study groups**

| Parameters | A vs B | B vs C | C vs A |
|---|---|---|---|
| P amp. | >0.05 | >0.05 | >0.05 |
| P durat. | >0.05 | >0.05 | >0.05 |
| PR interval | >0.05 | >0.05 | >0.05 |
| QRS complex (sec.) | <0.001* | >0.05 | <0.001* |
| Qt interval | >0.05 | >0.05 | >0.05 |
| T amp. | >0.05 | >0.05 | >0.05 |
| TP interval | <0.01# | >0.05 | <0.01# |
| ST mv | >0.05 | >0.05 | >0.05 |
| MEA | >0.05 | >0.05 | >0.05 |
| Heart Rate | <0.001* | >0.05 | <0.001* |

$p < 0.05$ significant, #$p < 0.01$ very significant; *$p < 0.001$ highly significant

## DISCUSSIONS

In present study ECG results of smokers and chewers show statistically significant increase in heart rate and significant decrease in TP interval and duration of QRS complex as compared to control. Other parameters such as P wave amplitude (mv), P wave duration (sec), QT interval (sec),T wav amp (mv), ST wave amp.(mv), MEA do not show any change when compared by unpaired t test. These changes in heart rate, QRS duration and TP interval are in accordance with results of Venkatesh G et al [9] who did a study in 2010 to compare the ECG changes in smokers to normal human beings. They found statistically significant increase in the heart rate and decrease in QRS complex and T-P interval in smokers compared to controls. P-wave, P-R interval, $QT_C$ interval, QRS frontal axis, ST segment and T-wave were not showing statistically significant results. The increase in heart rate and in smokers in the present study is also supported by Moliterno D Jet al[10], Mujataba et al[11] and Singh K[12].

The increase in heart rate in ECG in tobacco smokers and chewers in our study can be attributed to presence of nicotine in both smoke and smokeless tobacco as Benowitz et al [13] showed that infusion of nicotine in 14 healthy young men increases the heart rate.So nicotine in smoked and chewed tobacco leads to increase in heart rate which leads to decrease in QRS complex duration and TP interval. As in ECG strip if heart rate would increase in distance between T wave end of one cardiac cycle and P wave staring of new cardiac cycle would decrease and is cause of shortening of TP interval.

We found no change in significant change in the amplitude or duration of P wave as is supported by an early study of Karapinar H et al [14] **on** effect of smoking on P wave. Simiarly no significant change in QT interval in our study has been supported by similiar study by Karakaya O et al [15] .

## CONCLUSIONS

Nicotine is a sympathomimetic substance that promotes the release of catecholamines and other neurotransmitters, acting centrally and peripherally. This release of catecholamine is responsible for its cardiovascular effects such as increase in resting heart rate.

Resting heart rate is increased significantly in our sudy in smokers and chewers as compared to non tobacco users. There is significant shortening of duration of QRS complex in smokers and chewers as compared to control. There is significant shortening of TP interval in tobacco smokers and chewers as compared to control. This shortening of TP interval and QRS duration is subsequent to increase in resting heart rate.

So ECG status of chronic tobacco users(smoking or chewing) , as a parameter of cardiovascular morbidity in our study, show significant increased resting heart rate levels as compared to non taobacco users with subsequent decrease of QRS duration and TP interval.

## REFERENCES


1. World Health Report 2002.Reducing risks, promoting healthy life.Genva World Health Organization 2002, 47-98.
2. GYTS Report – Uttarpradesh 2002 by DN Sinha from internet site www.searo.who.int/LinkFiles/GYTS_Rep_UttarPradesh_2002
3. Agriculture, Centre for monitoring Indian Economy, September 1999 p396
4. MMWR-Centers for Disease Control and Prevention. Annual smoking attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999. MMWR Morb Mortal Wkly Rep 2002;5:300–3.
5. Price JF, Mowbray PI, Lee AJ, Rumley A, Lowe GD, Fowkes FG. Relationship between smoking and cardiovascular risk factors in the development of peripheral arterial disease and coronary artery disease: Edinburgh Artery study. Eur Heart J 1999;20:344–53.
6. Black HR, Laragh JH, Brenner BM. Smoking and cardiovascular diseases. Hypertension: pathophysiology, diagnosis and management. 2nd edition. New York, NY; Raven Press Ltd, 1995: 2621-47.
7. Jonas MA, Oates JA, Ockene JK, Hennekens CH. Statement on smoking and cardiovascular disease for health care professionals: American Heart Association. Circulation 1992;86:1664–9.
8. Willett WC, Green A, Stampfer MJ, et al. Relative and absolute excess risks of coronary heart disease among women who smoke cigarettes. N Engl J Med 1987; 317: 1303–9.
9. Venkatesh G, Swamy RM. A Study of Electrocardiographic changes in smokers compared to normal human beings. Biomedcal Research 2010; 21 (4): 389-392
10. Moliterno D J, Willard J, Lange R et al., "Coronary-artery vasoconstriction induced by cocaine, cigarette smoking, or both," New England Journal of Medicine, 1994. vol. 330, no. 7, pp. 454–459,
11. Mujtaba FA. Effect of smoking on Electrocardiogram and blood pressure IJPP 1977; 21 (4):393-395
12. Singh K Effect of Smoking on QT Interval, QT Dispersion and Rate Pressure Product. Indian Heart J 2004; 56: 140–142
13. Benowitz NL, Jacob P, Jones RT, Rosenberg J. Inter individual variability in the metabolism and cardiovas-cular effects of nicotine in man. J Pharm Exp Ther 1982; 221: 368-372.
14. Karapinar H , Esen O , Bulut M , Pala S , Akçakoyun M .Effect of Habitual Cigarette Smoking on P Wave Duration and Dispersion. Kofluyolu Kalp Dergisi . 2010;13(3):10-13
15. Karakaya O, Saglam M, Esen AM,Barutcu I. Acute effect of Cigarette Smoking on ventricular repolarization parameters. Kofluyolu Heart Journal 2005;9(1):1-7.