CARDIOVASCULAR RESPONSE TO CHRONIC TOBACCO SMOKING WITH REFERENCE TO ELECTROCARDIOGRAM AND BLOOD PRESSURE

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ABSTRACT

In India smoking is a common habit prevalent in both urban and rural areas. Cigarette smoking has extensive effect on cardio-vascular function and is clearly implicated in the etiology of number of cardio-vascular diseases particularly atherosclerosis, myocardial infarction and hypertension. All attempts have been made to study cardio-vascular functions among smokers and non-smokers populations in a rural area at Rural Medical College Loni (Ahmednagar, Maharashtra). Blood pressure and electro-cardiogram were recorded in 100 healthy male subjects comprising of 50 healthy smokers and 50 non-smokers. Systolic blood pressure and heart rate are increased immediately after smoking.

Keywords: Smokers; Cardio-vascular response; Electro-cardiogram; Blood pressure

1. INTRODUCTION:

Cigarette kills an estimated 5 million peoples annually world-wide 1. Smoking is leading cause of lung cancer and of pulmonary and cardio-vascular diseases 2. Cardio-vascular and cerebro-vascular diseases are caused by many smoke constituents that pass through the lungs and dissolve in the blood affecting haemoglobin, platelets and vascular tissues 3. Tobacco smoke contains 4,000 chemicals out of them 60 are non-carcinogens 4. In India, tobacco is consumed mainly in the form of bidi (54 %), followed by smokeless tobacco (27 %) and cigarettes (9 %) 5. first cigarette of the day produces much greater cardio-vascular and subjective response than do those that follow. It is observed that the last puff of cigarette contain 2-3 times as much as nicotin as the first puff and this nicotin first stimulates autonomic ganglia and then paralyses it so first there is cardiac slowing followed by acceleration of heart rate6.

SMOKING INDEX

Smoking Index = Number of cigarettes smoked per day × Duration of smoking in years

Here, number of cigarettes smoked per day means average number of cigarettes smoked per day in last 7 days 7. Coronary heart diseases are now leading cause of death in many developed countries. In U.K. 30 % of these deaths are due to smoking. Smoking causes chest pain on exertion. Atherosclerotic narrowing of smallest coronary arteries is increased in heavy and moderate smokers. Carboxyhaemoglobinemia of habitual smokers is enough to interfere with the cardio-vascular system function 8.

2. MATERIALS AND METHODS:

The present study was carried out at Rural Medical College Loni, Maharashtra from may 2005 to April, 2008. Study population includes 100 healthy male subjects belonging to age group 20-45 years divided into experimental group and control group (50 each). Experimental groups were chronic tobacco smokers with history of tobacco smoking more than 6 months and still smokers with no history of any major illness at present and in the past. Smokers less than 6 months duration, quitters and ex-smokers were excluded from study. The control group was
healthy, non-smoker male of 20-45 years without history of any major illness in the past. Subjects of study groups were informed about the study and harmful effects of smoking and written consent was taken from them. Both systolic and diastolic blood pressure recorded in lying down position with the help of sphygmomanometer, 3 successive readings were taken and average was considered. Electrocardiogram of the same person was recorded by using 12 leads ECG machine. And from lead 2 duration of P wave, PR interval, QRS complex, T wave and RR interval was recorded and heart rate was found out. The amplitude of P, QRS and T wave was recorded. All the above procedure was carried out before smoking, and then subjects were directed to smoke and inhale 2 standard size cigarettes. Immediately after last puff, blood pressure and electro-cardiogram were recorded. In control group, blood pressure and ECG were recorded after having rest for 10 minutes. Obtained data is tabulated and statistically analysed by using various standard statistical methods like calculation of mean, standard error of mean, z-test, p-value equal or less than 0.05 were considered as significant.

3. OBSERVATION AND RESULTS

| TABLE – 1 Recording of blood pressure before and after smoking in smokers |
|---------------------------------------------------------------|
| Mean values |
| Parameters | Before smoking | After smoking | p-value |
| SBP mm Hg  | 120.88 ± 8.65 | 126.64 ± 9.34 | p < 0.05* |
| DBP mm Hg  | 77.84 ± 6.41 | 80.04 ± 7.83 | p < 0.05* |
| HR/min     | 73.68 ± 10.50 | 80.26 ± 12.37 | p < 0.05* |

SBP = Systolic blood pressure
DBP = Diastolic blood pressure
HR = Heart rate
* = Statistically significant

| TABLE – 2 Electro-cardiographic interpretations before smoking and after smoking in smokers (Durations in seconds) |
|---------------------------------------------------------------|
| Parameters | Before smoking | After smoking | p-value |
| PR interval | 0.262 ± 0.18 | 0.110 ± 0.016 | p < 0.05* |
| QRS complex | 0.077 ± 0.0068 | 0.078 ± 0.0083 | p > 0.05 |
| T wave | 0.177 ± 0.026 | 0.170 ± 0.0276 | p > 0.05 |

*= Statistically significant

| TABLE – 3 Electro-cardiographic interpretations before smoking and after smoking in smokers (Amplitude) |
|---------------------------------------------------------------|
| Parameters | Before smoking | After smoking | p-value |
| QRS complex (mv) | 0.944 ± 0.264 | 0.932 ± 0.297 | p > 0.05 |
| T wave (mv) | 0.256 ± 0.0822 | 0.259 ± 0.154 | p > 0.05 |

In smokers, after smoking 2 standard size cigarettes there was significant increase in systolic blood pressure, diastolic blood pressure and heart rate (p < 0.05; table no.1). Interpretation of electrocardiogram of all subjects showed a significant decrease in PR interval (p < 0.05; table no.2). Therefore this indicates that heart rate also increases significantly after smoking. There was an apparent decrease in duration of QRS complex and T wave, hence the electrocardiographic alterations were less prominent.
and even absent in some subjects. As subsequent cigarettes were consumed, less alteration in the reading are because blood pressure and electrocardiogram were measured in the afternoon. And the first cigarette of the day produces many cardiovascular and subjective responses than do those that follow.

Since smokers already smoke one or two cigarettes, there are less cardiovascular changes.

Paradoxically in epidemiologic studies, smokers have been found to have lower blood pressures than non-smokers.

4. DISCUSSION

Tobacco smoking is well recognized risk factor for sudden cardiac death as well as other manifestations of coronary artery diseases. Smoking now is recognized as foremost environmental hazard to health.

The most interesting finding in our study was that the subjects (healthy smokers) showed marked and sustained increase in systolic blood pressure when compared with non-smokers suggesting of increased arterial stiffness. Our results are supported from the statements made by Azara Mahmud and John Feely.

We also found a significant increase in heart rate among smokers when compared to non-smokers. Our results are supported by Grabial et al who found increase in heart rate in their environment.

Armitage A K concluded that reflex stimulation of vaso-motor centres via carotid body is important mechanism in response to smallest dose of nicotine.

Components of cigarette smoke damages vascular endothelium which leads to atherosclerosis.

Fewer coronary thrombi were present in the more than one pack per day cigarette smokers.

Grasssi et al proposed that because of smoking there is reduction in arterial compliance due to the reduction in sensitivity of stretch ‘sensors’ such as baro-receptors.

Thromboxane biosynthesis is increased in healthy individuals who are chronic smokers which lead to arterial thrombi.

Cigarette smoking is an independent risk factor of coronary artery diseases cause of occult dyslipidemia.

There is shortening of platelets survival in smokers which enhances platelets aggregation and atherosclerosis.

Large dose of nicotin affects cardio-vascular system via autonomic nervous system.

Nicotin reduces oxygen carrying capacity of blood through increase serum carboxy-hemoglobin level because its affinity to hemoglobin is greater than oxygen. Therefore diminishes oxygen carrying capacity and damage mitochondria and vascular endothelium that results in thrombo-embolic phenomenon or arterial stiffness.

CONCLUSION

With the study we conclude that

- Chronic tobacco smoking leads to systolic hypertension.
- Leads to coronary occlusion and myocardial infarction.
- It may lead to thrombo-embolism
- Tachycardia.

Hence the risk of cardio-vascular morbidity and mortality is high with chronic tobacco smoking.

REFERENCES

1. Bulletin of WHO, International Journal of Public Health, June 2006; 84 (6):495.
2. Ruth Roemer. World Tobacco Epidemic. 2nd Edition; 1993: 47.
3. WHO: Women and Tobacco. Geneva, 1992.
4. Judich Mackay and Eriksen. Tobacco Atlas; 2002: 26.
5. Annonymous. IUALTD: The world tobacco situation. IUALTD news bulletin. Tobacco health, 1998; 11: 19-21.
6. Greenspan K, Robert E, Edemands et al. Some effects of nicotine on Cardiac Automaticity, Conduction and Introphy. Archintern Med. 1969; 123: 707-712.
7. Sharma B K et al. Hypertension among the industrial workers and professional
classes in Ludhiana. Punjab I H J, 1985; 37 (6): 382-85.
8. Jaffe J H. Drug addiction and drug abuse, the pharmacological basis of therapeutics. Goodman and Gillman’s, 8th Edition: 545-49.
9. Kanel W B, Doyle J T et al. Precursors of sudden coronary death, factors related to the incidence of sudden death. Circulation, 1975; 51: 606-613.
10. Haustrom A P, Cobb L A and Ray R. Smoking as a risk factor for recurrence of sudden arrest. N England J Medicine, 1986; 314: 271-275.
11. Barry J, Mead K, Nabel E G et al. Effect of smoking on the activity of ischaemic heart diseases. JAMA, 1989; 261: 298-402.
12. Reimert Raven Holt. Measurement of smoking experiences. N England J Medicine; 275: 15.
13. Article, Azra Mahmud, John Feely from Department of Pharmacology and Therapeutics. Trinity College, Dublin. American Heart Association, 2003; 41: 183.
14. Armitage A K. Effect of nicotine and tobacco smoke on blood pressure and release of catecholamines from adrenal glands. British J Pharmacology, 1965; 25: 515-526.
15. Phillips A N, Neaten J D, Cook D G, Grimm R H and Shaper A G. The leucocyte count and lung cancer. 1992; 69 (3): 680-84.
16. Sowinski P. The enzymatic deficiencies in lymphocytes of smokers. Med interne. 1989; 27 (1): 41-46.
17. Grassi et al. Cigarette smoking and sympathetic activity. Circulation, 1994; 90: 248-253.
18. Gillium R F and Ingram D D. White blood cell count, Coronary heart diseases and Death. Am Heart J, 1993; 125 (3): 855-863.
19. Tollerud D J, Clerk J W. The effect of cigarette smoking on T cell subsets. Am Rev Respi Diseases, 1989; 139 (6): 1446-1451.
20. Ensurd K and Grimm R H. The white blood cell count and risk factor for coronary heart diseases. Am Heart, 1992; 124 (1): 207-213.
21. Winifred G N. Effect of nicotine on cardiac muscle contraction and radio-calcium movement. Am J Physiology, 1963; 205 (5): 800-896.
22. Kenne W J M C Cyc Chew et al. Myocardial infarction with normal coronary angiogram: possible mechanism of smoking risk in coronary artery diseases. Brit Heart J, 1980; 43: 493-498.