Study of ECG Effects in Smokers and Normals

Swathi K[1], Sumanth Garimella[2]

[1]*Tutor, Department of physiology, Sri Padmavathi Medical College for Women,*
Tirupati, Andhrapradesh, India.
[2]*Post Graduate, Department of Pharmacology, ASRAM Medical College,*
Elur, Andhrapradesh, India.

Abstract

Background: More than one billion people worldwide are still addicted to cigarettes. Every year, 3 million die from smoking related causes. Cigarette smoking is a major risk factor for cardiovascular disease and epidemiological studies have established a gradient of risk, the highest for containing smokers, intermediate for reformed smokers and the lowest for non-smokers.

Methods: The 200 smoking people age above 20 years selected and same age of non alcoholic people of same age. The ECG were recorded in lying down and resting position. The ECG results were analysed for Heart rate, P wave, PR interval, QRS duration, QTC interval, ST segment, T wave, TP interval and frontal axis.

Results: The heart rate beets were in non-smokers was 74.86±7.98 and smokers was 80.28±9.12, duration was 0.092±0.009 and smokers was 0.087±0.020. P wave amplitude was 1.060.16 in non-smokers and 1.220.16 in smokers. P-R interval in seconds it was 0.1470.05 in non-smokers and 0.1560.03 in smokers. QRS in seconds it was 0.780.02 in non-smokers and 0.0620.03 in smokers. Qtc interval in seconds was 0.4140.08 in non-smokers and 0.4190.08in smokers. T wave was 100% normal in non-smokers and 91% normal in smokers, T wave was abnormal in 8% of smokers. ORS frontal axis in degrees was 53.024.7 in non-alcoholic and 44.623.6in smokers.

Conclusion: The present study shows smokers are more porn to cardio vascular diseases, the early finding of ECG abnormalities helpful to prevent cardiac diseases.

Key Words:
ECG, Cardio Vascular diseases, Smokers and Non-smokers.

INTRODUCTION

One billion people worldwide are addicted to cigarettes. Every year 3million die from smoking related causes[1]. Cigarette smoking is a major risk factor for cardiovascular disease and epidemiological studies have established a gradient of risk, the risk is more in smokers, moderate for reformed smokers and the lowest in non smokers[2]. 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. Tobacco consumption is the cause of the preventable deaths globally. Tobacco is consumed in the form of cigarettes. It contains nicotine which causes physical and psychological dependencies. Nicotine facilitates conduction block, re-entry and it increases the vulnerability to ventricular fibrillation. Nicotine and other components of cigarette can produce profound changes in the heart, which can be assessed by doing an ECG, which is cheapest and most reliable method for assessing cardiovascular abnormalities. It was estimated that there were 100 million deaths worldwide in the 20th century and currently, there are 5.4 million deaths every year. It has also been estimated that by 2030, there will be more than 8 million deaths every year. Nicotine, which is the main component of tobacco, causes sudden coronary death[3]. Nicotine also causes cardiac death by provoking ventricular arrhythmias. The cardiac effects of nicotine are attributed to the release of catecholamine , which are released due to the binding of nicotine to the nicotinic cholinergic gate on the cation channels in receptors throughout the body. A longer retention of nicotine occurs in the blood and in other specific tissues such as the oesophagus, fundus ,antrum, spleen, caecum, pancreas, testes, heart and the muscle via a constant exposure. Nicotine facilitates a conduction block and a re-entry and it increases the vulnerability to a ventricular fibrillation. Nicotine is a potent inhibitor of the cardiac A type potassium channels, which contributes to the changes in the electrophysiology and it also induces arrhythmias [4,5]. There has been a growing recognition of the importance of the autonomic nervous system in cardiovascular disease. Various measures of heart rate variability evaluate changes in beat-to-beat interval durations using ambulatory ECG. Various measures of heart rate variability provide quantitative indicators of cardiac autonomic function. Studies have documented the increase in heart rate variability shortly after smoking cessation. Changes in heart rate and heart rate variability are also described in association with acute passive smoking or exposure to respirable suspended particles[6,7]. Cigarette smoking contributes to the loss of over 5 million lifeyears annually in the United States. Nearly 20% of all coronary heart disease deaths can be attributed to smoking. Smoking, therefore, is one of the most important modifiable risk factors for cardiovascular disease and myocardial infarction [8,9]. Cigarette smoking leads to hypoxemia and endothelial dysfunction which accelerate atherosclerotic changes, increasing smokers' risk for cardiovascular disease. The 12-lead electrocardiogram is a routine, inexpensive tool for assessment of cardiovascular disease and cardiovascular disease risk in both clinical and research settings, and ECG changes powerfully predict future cardiovascular disease events. Large, population-based studies have described a higher prevalence of smoking in subjects with ECG abnormalities and demonstrated that ECG abnormalities are associated with increased cardiovascular disease events and death.

Although the strong relation between smoking and is well-established, there are relatively few data that prospectively described ECG changes following a smoking cessation attempt. Furthermore, most of the existing data about CVD and smoking is from older cohorts that are not representative of today's smokers, who tend to be more overweight, are more likely to be female, and to have lower socioeconomic status[10,11].

MATERIALS AND METHODS

The 200 smoking people age above 20 years selected and same age of non alcoholic people of same age. The ECG were recorded in lying down and resting position. The ECG results were analysed for Heart rate, P wave, PR interval, QRS duration, QTC interval, ST segment, T wave, TP interval and frontal axis.

RESULTS:

The heart rate beets were in non-smokers was 74.867.98 and smokers was 80.289.12, duration was 0.0920.009 and smokers was 0.0870.020. P wave amplitude was 1.060.16 in non-smokers and 1.220.16 in smokers. P-R interval in seconds it was 0.1470.05 in non-smokers and 0.1560.03 in smokers. QRS in seconds it was 0.780.02 in non-smokers and 0.0620.03 in smokers. Qtc interval in seconds was 0.4140.08 in non-smokers and 0.4190.08in smokers. T wave was 100% normal in non-smokers and 91% normal in smokers, T wave was abnormal in 8% of smokers. ORS frontal axis in degrees was 53.024.7 in non-alcoholic and 44.623.6in smokers. The present study is concentrated on ECG changes in smokers and nonsmokers(Table 1).

ECG Report		Normal Individuals	Smokers
Heart Rate	Beets	74.867.98	80.289.12
	Duration	0.0920.009	0.0870.020
P – Wave	Amplitude	1.060.16	1.220.16
P – R intervals	In seconds	0.1470.05	0.1560.03
QRS complex	In seconds	0.780.02	0.0620.03
	IE	200(100%)	184(92%)
ST Segment	NSST	0	16(8%)
QTc Interval	In seconds	0.4140.08	0.4190.08
T wave	Normal	200(100%)	182(91%)
	Abnormal	0	18(9%)
QRS Frontal Axis	In degrees	53.024.7	44.623.6

Table 1. ECG report of Normal individuals and Smokers

Discussion

Smoking is the most important risk factor for the coronary artery disease. Smoking is known to have multiple ECG effects; however, the temporal relationship of the ECG changes to an act of smoking is not known. Ahn Von found that cigarette smoking during hypoxia increased the amplitude of the P waves. This might be due to development of corpulmonale subsequently producing right atria hypertrophy as a result of chronic smoking[12].

The study of Renukadevi et al widening of the QRS complex and shortening of the QTc interval in smokers and non smokers[13]. The similar results shown by study done by Karjalainen et al, which explained that a shortened QT interval was as a risk factor for smokers, which could cause death. The shortened QT and ST segments warn that there may be Shortening in the ventricular filling phase, during which the coronary supply occurs. This may lead to an insufficient myocardial perfusion, which may invite ischaemic episodes. Such altered ventricular electrical activities, like a widened QRS complex and a shortened ST segment also predisposes smokers to episodes of arrhythmia[14].

In the general population, major and minor ECG changes predict increased mortality [15]. Individuals who smoke are more likely to have ECG findings consistent with ischemic heart disease, structural heart disease and cardiac rhythm disorders [16]. In study of Adam D. Gepner[17] on average, being nearly a decade younger than participants in population-based studies, the smokers in our study had notably more major ECG abnormalities than has been observed in the general population[18,19].

Autonomic nervous system may play an important role in cardiovascular diseases. HRV is useful in non- invasive quantification of autonomic nervous system[20]. Large epidemiological studies have also linked an increased risk of coronary artery disease, death and cardiac mortality with decreased HRV in general populations. Although it is clear that low HRV has a negative prognostic impact, it is important to point out that causality and mechanisms have not been established. Decreased HRV among smokers is also shown in a few studies[21,22]. A study of acute tobacco smoke exposure to small numbers of volunteers has shown significant HRV changes[23]. Studies have also documented increase in HRV immediately after smoking cessation[24,25]. The component of tobacco smoke that is responsible for autonomic dysfunction is not known. Carbon monoxide was implemented in some studies. Role of nicotine is still not clear in the causation of autonomic dysfunction, since nicotine patches that release high level of nicotine are shown to have minimal effect on HRV. RSP may be the main culprit. A study of healthy volunteer had shown that 100 μg/m3 increase in 4 h RSP exposure was associated with approximately a 25 ms decline in both SDNN and r-MSSD[23].

REFERENCES

1. Doll R, Peto R. Mortality in relation to smoking 20 years observation on maleBritish Doctors. British Medical Journal 1996; 4:1525-36.
2. Kannel WB Schatzkin A. Risk factor analysis. Prog Cardiavase Dis 1983;26:309- 32.
3. Garden T, Kannel WB, Mcgee D et al. Death and coronary attacks in men aftergiving up cigarette smoking: a report from the Tramingham study, lancet 1974;2:1345- 8.
4. Yashima M, Ohara T, Cao JM , Kim YH, Fishbein MC, Mandel WJ, et al..Nicotine increases ventricular vulnerability to fibrillation in hearts with healed myocardial infarction. Am.J Physiol Heart Circ Physiol. 2000 Jun; 278(6): H2124-33.
5. Huizhenwang . Hang Shi MD, Baofeng Yang. Nicotine is a potent Blocker of the cardiac A- type KChannels. Circulation. 2000; 102: 1165-71.
6. Auer R, Bauer DC, Marques-Vidal P, Butler J, Min LJ, et al. (2012) Association of major and minor ECG abnormalities with coronary heart disease events. JAMA 307: 1497–1505.
7. Liao YL, Liu KA, Dyer A, Schoenberger JA, Shekelle RB, et al. (1988) Major and minor electrocardiographic abnormalities and risk of death from coronary heart disease, cardiovascular diseases and all causes in men and women. J Am Coll Cardiol 12: 1494–1500.

8. Smoking-attributable mortality, years of potential life lost, and productivity losses–United States, 2000–2004. MMWR Morb Mortal Wkly Rep.2008;57: 1226–1228.
9. Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson TB, et al. Heart disease and stroke statistics–2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2009;119: e21–181.
10. Ambrose JA, Barua RS (2004) The pathophysiology of cigarette smoking and cardiovascular disease: an update. J Am Coll Cardiol 43: 1731–1737.
11. Waters D, Lesperance J, Gladstone P, Boccuzzi SJ, Cook T, et al. (1996) Effects of cigarette smoking on the angiographic evolution of coronary atherosclerosis. A Canadian Coronary Atherosclerosis Intervention Trial (CCAIT) Sub study. CCAIT Study Group. Circulation 94: 614–621.
12. Ahn, Von. B. The acute effect of tobacco smoking and nicotine on the Electrocardiogram, especiall during induced hypoxia; A clinical and experimental investigation. Acta Medica Scandinavica. 1994; 292: 15.
13. M.R. Renuka devi, T. Arvind, Sai kumar. hanges in Smokers and Non Smokers-A Comparative Study. Journal of Clinical and Diagnostic Research. 2013 May, Vol-7(5): 824-826.
14. Karjalainen. J.Reunanen A, Ristola P, Vitasalo M. QT interval as a cardiac risk factor in a middle aged population. Heart. 1997Jun; 77 (6): 543-48.
15. De Bacquer D, De Backer G, Kornitzer M, Blackburn H (1998) Prognostic value of ECG findings for total, cardiovascular disease, and coronary heart disease death in men and women. Heart 80: 570–577.
16. Nabipour I, Amiri M, Imami SR, Jahfari SM, Nosrati A, et al. (2008) Unhealthy lifestyles and ischaemic electrocardiographic abnormalities: the Persian Gulf Healthy Heart Study. East Mediterr Health J 14: 858–868.
17. Adam D. Gepner, Megan E. Piper, Miguel A. Leal, Asha Asthana, Michael C. Fiore, Timothy B. Baker, James H. Stein. Electrocardiographic Changes Associated with Smoking and Smoking Cessation: Outcomes from a Randomized Controlled Trial. 2013 ;8: e62311.
18. De Bacquer D, De Backer G, Kornitzer M (2000) Prevalences of ECG findings in large population based samples of men and women. Heart 84: 625–633.
19. Cedres BL, Liu K, Stamler J, Dyer AR, Stamler R, et al. (1982) Independent contribution of electrocardiographic abnormalities to risk of death fromcoronary heart disease, cardiovascular diseases and all causes. Findings of threeChicago epidemiologic studies. Circulation 65: 146–153.
20. Lahiri MK, Kannankeril PJ, Goldberger JJ. Assessment of autonomic function in cardiovascular disease: physiological basis and prognostic implications. J Am Coll Cardiol 2008;51:1725–33.
21. Dekker JM, Crow RS, Folsom AR, et al. Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: the ARIC Study. Atherosclerosis risk in communities. Circulation 2000;102:1239–44.
22. De Bruyne MC, Kors JA, Hoes AW, et al. Both decreased and increased heart rate variability on the standard 10-second electrocardiogram predict cardiac mortality in the elderly: the Rotterdam Study. Am J Epidemiol 1999;150:1282–8.
23. Pope CA 3rd, Eatough DJ, Gold DR, et al. Acute exposure to environmental tobacco smoke and heart rate variability. Environ Health Perspect 2001;109:711–16.
24. Stein PK, Rottman JN, Kleiger RE. Effect of 21 mg transdermal nicotine patches and smoking cessation on heart rate variability. Am J Cardiol 1996;77:701–5.
25. Yotsukura M, Koide Y, Fujii K, et al. Heart rate variability during the first month of smoking cessation. Am Heart J 1998;135:1004–9.