International Journal of Medical and Health Research
ISSN: 2454-9142
Received: 10-01-2019; Accepted: 12-02-2019
www.medicalsciencejournal.com
Volume 5; Issue 3; March 2019; Page No. 35-38

Effects of packs per year of smoking on different cardiovascular variables during stress testing

Dr. Abhilasha Singh[1], Dr. Abhishek Kamendu[2*]
[1] Assistant Professor, Department of Physiology, Narayan Medical College and Hospital, Jamuhar, Bihar, India
[2] Assistant Professor, Department of General Medicine, Narayan Medical College and Hospital, Jamuhar, Bihar, India
[*] Corresponding Author: Dr Abhishek Kamendu

Abstract
Smoking is the most important modifiable risk factor for cardiovascular disease, a major risk factor for cardiovascular morbidity and mortality, and is considered to be the leading preventable cause of death in the world. Hemodynamic parameters assessed in the smokers observed on higher side compared with the non-smoker patients. The present study concluded that the smokers are having more risk of Coronary Heart Diseases as compared to non-smokers. Exercise electrocardiographic testing is one of the most important and valuable noninvasive diagnostic tests in the clinical evaluation and management of patients with suspected or known cardiovascular disease particularly coronary artery disease. The exercise stress ECG test is also very useful tool as a screening procedure for healthy individuals who are considered to be at possible risk of coronary artery disease.
The present study was planned in the Narayan Medical College and Hospital, Jamuhar, Bihar. Total 60 patients were enrolled in the study. 30 patients were enrolled in study group and 30 patients were enrolled in control group. All patients were informed consents. The aim and objective of the study were clearly conveyed to all patients. Approval of the institutional ethical committee was taken prior to conduct of the study.
Hence based on the vast literature findings this study was planned to assess effects of quantity of smoking on cardiovascular function, providing essential information that could contribute to reducing the smoking epidemic and its consequences for cardiovascular health.

Keywords: cardiovascular disease, circulatory system, smoking

Introduction
Smoking ranks among the top causes of cardiovascular disease, including coronary heart disease, ischemic stroke, peripheral artery disease and abdominal aortic aneurysm. It is also associated with an increased risk of certain types of cancer, and is a major cause of chronic obstructive pulmonary disease [1]. Smoking, either active or passive, can cause cardiovascular disease via a series of interdependent processes, such as enhanced oxidative stress, haemodynamic and autonomic alterations, endothelial dysfunction, thrombosis, inflammation, hyperlipidaemia, or other effects [2]. Even exposure to small quantities—e.g. occasional smoking, passive smoking, a few cigarettes per day—is sufficient to have deleterious consequences. Cigarette smoke contains more than 4000 chemical substances that have harmful effects on cardiovascular function [3]. These include nicotine, carbon monoxide (CO), oxidative gases, polycyclic aromatic hydrocarbons, carbonyls, butadiene, minerals, carbon disulphide, and benzene. Although many of the toxic substances contained in tobacco smoke are generic products of the combustion of organic materials, exposure to smoking involves contact with two substances that are specific to tobacco smoke and are known to be damaging to the health: nicotine and CO [4].
Chemicals in cigarette smoke cause the cells that line blood vessels to become swollen and inflamed. This can narrow the blood vessels and can lead to many cardiovascular conditions.
Atherosclerosis, in which arteries narrow and become less flexible, occur when fat, cholesterol, and other substances in the blood form plaque that builds up in the walls of arteries. The opening inside the arteries narrows as plaque builds up, and blood can no longer flow properly to various parts of the body. Smoking increases the formation of plaque in blood vessels.
Coronary Heart Disease occurs when arteries that carry blood to the heart muscle are narrowed by plaque or blocked by clots. Chemicals in cigarette smoke cause the blood to thicken and form clots inside veins and arteries. Blockage from a clot can lead to a heart attack and sudden death.
Stroke is a loss of brain function caused when blood flow within the brain is interrupted. Strokes can cause permanent brain damage and death. Smoking increases the risk for strokes. Deaths from strokes are more likely among smokers than among former smokers or people who have never smoked.
Peripheral Arterial Disease (PAD) and peripheral vascular disease occur when blood vessels become narrower and the flow of blood to arms, legs, hands and feet is reduced. Cells and tissue are deprived of needed oxygen when blood flow is reduced. In extreme cases, an infected limb must be removed. Smoking is the most common preventable cause of PAD.
Abdominal Aortic Aneurysm is a bulge or weakened area that occurs in the portion of the aorta that is in the abdomen. The aorta is the main artery that carries oxygen-rich blood throughout the body. Smoking is a known cause of early damage to the abdominal aorta, which can lead to an aneurysm. A ruptured abdominal aortic aneurysm is life-threatening; almost all deaths from abdominal aortic

aneurysms are caused by smoking. Women smokers have a higher risk of dying from an aortic aneurysm than men who smoke. Autopsies have shown early narrowing of the abdominal aorta in young adults who smoked as adolescents [5].

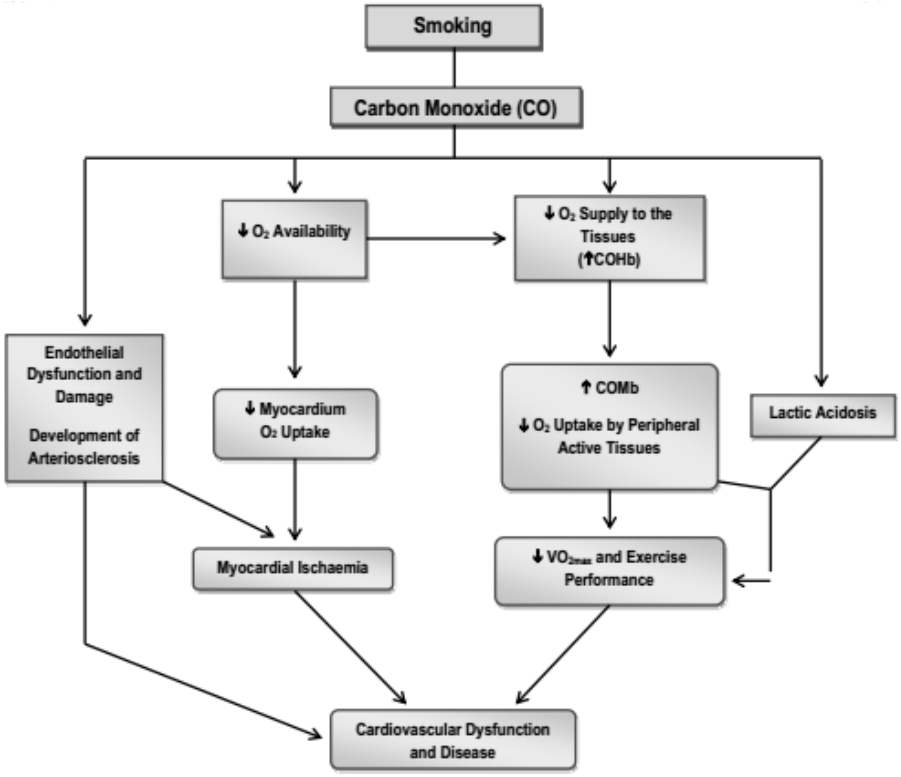

Fig 1

Exercise electrocardiographic testing or TMT is one of the most important and valuable noninvasive diagnostic tests in the clinical evaluation and management of patients with suspected or known cardiovascular disease particularly coronary artery disease. The exercise stress ECG test is also very useful tool as a screening procedure for healthy individuals who are considered to be at possible risk of coronary artery disease. The cardiovascular variables observed during test are resting heart rate, chronotropic response, heart rate recovery, exercise duration and ventricular ectopy. These variables have independent CVD risk prediction than the ST-T changes during Variables measured during exercise treadmill testing that predict outcome are actually indicators of general fitness and function of the autonomic nervous system. Variables measured during exercise treadmill testing that predict outcome are actually indicators of general fitness and function of the autonomic nervous system

Hence based on the vast literature findings this study was planned to assess effects of packs per year of smoking on cardiovascular function, providing essential information that could contribute to reducing the smoking epidemic and its consequences for cardiovascular health.

Methodology

The present study was planned in the Narayan Medical College and Hospital, Jamuhar, Bihar. Total 60 patients were enrolled in the study. 30 smokers patients were enrolled in study group and 30 non-smokers patients were enrolled in control group. All patients were informed consents. The aim and objective of the study were clearly conveyed to all patients. Approval of the institutional ethical committee was taken prior to conduct of the study.

Following was the Inclusion Criteria and Exclusion criteria for the present study.

Inclusion Criteria

- Age less than 45 years
- Smoking for more than 3 years

Exclusion Criteria

- Patients having history of Hypertension, dyslipidaemia and diabetics

All the Participants underwent a maximal symptom limited TMT according to Bruce protocol. Heart rate, blood pressure, and 12-lead ECG was recorded before exercise, at the end of each exercise stage and at 1-minute intervals during recovery till maximum of 4 minutes of recovery phase. The test was stopped for limiting symptoms like angina, dyspnea, fatigue or abnormalities of rhythm or abnormal blood pressure response, or marked and progressive ST-segment deviation or attainment of target heart rate.

Measures of chronotropic response included the following:

- Peak HR achieved with maximal exercise stress testing expressed as a percentage of age predicted maximum heart rate
- HR reserve HRR= HRpeak–HRrest
- Ability to achieve >85% age-predicted HR

Chronotropic index takes into account age, physical fitness (exercise capacity), and resting HR. The chronotropic index is the ratio of the heart rate reserve (HRR) to the metabolic reserve (MR) used at peak exercise For the usual application, the following formula defines the chronotropic index: [(HRmax −HR rest)×100] [(220 − Age)−HR rest]
Categorical variables were expressed as numbers and percentages. Statistical analysis was done using $\chi 2$ test and 't' test and ―p‖ value<0.05 was accepted as indicating statistical significance, p<.001 as highly significant. Variables were compared between smokers and non-smokers, and among smokers based on pack years Light smokers<9 pack years, moderate 10-19 pack years and heavy ≥20 pack years

Results & Discussion

The data from the 30 patients doing smoking from more than 3 years and 30 non-smoker patients were collected. Both the study group patients had undergone the maximal treadmill exercise operation.

Table 1: No. of Cigarettes per day in Study Group: Smokers

No. of Cigarettes	No. of Cases
1-3	8
3-5	12
More than 5	10
Total	30

Table 2: Comparison of exercise testing variables between smokers and non-smokers

Smokers	Study Group: Smokers	Control Group: Non Smokers	P Value
Resting Heart Rate (bpm)	80-94	84-101	<0.05
Percentage of maximum heart rate	76-94	82-92	<0.05
Heart Rate reserve	48-86	67-81	<0.05
Heart rate recovery in 1 minute	22-37	24-35	<0.05
Exercise duration	8-13	9-14	<0.05

Table 3: Comparison of smokers and non smokers that attained different stages of Treadmill Test (TMT)

Stages of Treadmill Test (TMT)	Study Group: Smokers	Control Group: Non Smokers
1	3	0
2	2	1
3	10	6
4	6	8
5	5	10
6	4	5
7	0	0
Total	30	30

The resting heart rate is more in smokers. This finding is in line with data reported by Papathanasiou *et al.* [6] & Minami *et al.* [7].The effect of nicotine probably contributed to the significantly higher resting HR in smokers. Smoking is associated with selective alterations in cardiac autonomic control. Smoking, acting at peripheral sympathetic sites, increases circulating levels of catecholamines, augments sympathetic outflow, and causes a long-term reduction in vagal drive [8, 9]. This sympathetic predominance, seen even in young heavy smokers is also associated with impaired baro reflex function leading to a marked increase in heart at rest. Elevated resting heart rate is an independent risk factor for cardiovascular disease in healthy men and women [10].
Claire M. Bernaards *et al.* [11] stated that a negative longitudinal relationship was found between moderate to heavy smoking and HR max. Asha Asthana *et al.* [12] had also found that heavier smokers had lower peak HR increase (p< .05), and HR reserve (p< .01). The CARDIA [13] study reported that the mean maximum heart rate was lower in smokers than in non-smokers although maximum rating of perceived exertion was nearly identical in smokers and non-smokers. Chronic smoking appears to blunt the heart rate response to exercise, so that exercise duration to submaximal heart rates is increased even though maximal performance is impaired.
In this study no significant difference in HR recovery was observed between smokers and non-smokers. In many epidemiological HR-related studies in healthy middle-aged populations, smoking was inversely associated with HR decline during recovery. Kobayashi *et al* had found that return of heart rate to resting levels after exercise was slower in chronic smokers. Asha Asthana *et al.* [12] reported that HR recovery was correlated inversely with smoking burden. But in multivariate analysis no significant independent association has been found. Attenuated HR decline during recovery is an important surrogate for underlying autonomic dysfunction that is associated with increased cardiovascular morbidity and mortality as was reported in many studies like that done by Morshedi-Meibodi A *et al.* [14].
The mean exercise duration of smokers was shorter than that of non-smokers. Exercise duration is progressively decreasing with increasing burden of smoking. Exercise duration is the strongest prognostic variable measured during exercise test. Its prognostic value has been demonstrated in healthy subjects being screened for coronary artery disease Ekelund LG *et al.* [15] and Blair SN *et al.* [16], and in patients being evaluated for suspected or known coronary artery disease as by Roger VL [17] *et al* and Goraya TY *et al.* [18].

Conclusion

Smoking is a major modifiable risk factor for cardiovascular morbidity and mortality, and is considered to be the leading preventable cause of death in the world. Hemodynamic parameters assessed in the smokers observed on higher side compared with the non-smoker patients. The present study concluded that the smokers are having more risk of Coronary Heart Diseases as compared to non-smokers. .Our study showed greater risk for CAD in smokers compared to non smokers as per changes in different cardiovascular parameters, But HR recovery in 1 minute showed no significance. Cardiovascular morbidity smoking increases with increased amount and duration of smoking.

References

1. World Health Organization. Report on the Global Tobacco Epidemic. Geneva, 2008; available at: http://www.who.int/tobacco/mpower/mpowerreport_full_2008.pdf (last day accessed 3 October 2013)
2. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: An update. J Am Coll Cardiol. 2004; 43:1731-1737.

3. Bullen CH. Impact of tobacco smoking and smoking cessation on cardiovascular risk and disease. Expert Rev Cardiovasc Ther. 2008; 6(6):883-895.
4. USA Institute of Medicine of the National Academies. Secondhand Smoke Exposure and Cardiovascular Effects: Making Sense of the Evidence. Washington DC: The National Academies Press, National Academy of Sciences, 2009; available at: http://www.nap.edu/catalog/12649.html (last day accessed 24 July 2013).
5. https://www.cdc.gov/tobacco/data_statistics/sgr/50th-anniversary/pdfs/fs_smoking_CVD_508.pdf
6. Papathanasiou G, Georgakopoulos D, Papageorgiou E, Zerva E, Michalis L, Kalfakou V, *et al*. Effects of smoking on heart rate at rest and during exercise, and on heart rate recovery in young adults. Hellenic J Cardiol. 2013; 54:168-177.
7. Minami J, Ishimitsu T, Matsuoka H. Effects of smoking cessation on blood pressure and heart rate variability in habitual smokers. Hypertension. 1999; 33:586-590.
8. Lucini D, Bertocchi F, Malliani A, Pagani M. A controlled study of the autonomic changes produced by habitual cigarette smoking in healthy subjects. Cardiovasc Res. 1996; 31:633-639.
9. Hayano J, Yamada M, Sakakibara Y, Fujinami T, Yokoyama K, Watanabe Y, *et al*. Short and long-term effects of cigarette smoking on heart rate variability. Am J Cardiol. 1990; 65:84-88.
10. Cooney MT, Vartiainen E, Laakitainen T, Juolevi A, Dudina A, Graham IM. Elevated resting heart rate is an independent risk factor for cardiovascular disease in healthy men and women. Am Heart J. 2010; 159(4):612-619.
11. Claire M Bernaards, Jos WR Twisk, Willem Van Mechelen, Jan Snel, Han CG Kemper. A Longitudinal Study on Smoking inRelationship to Fitness and Heart Rate Response.
12. Asha Asthana, Megan E Piper, Patrick E McBride, Ann Ward, Michael C Fiore, Timothy B Baker, *et al*. Long-term effects of smoking and smoking cessation on exercise stress testing: Three-year outcomes from a randomized clinical trial.
13. Sidney S, Sternfeld B, Gidding SS, Jacobs DR Jr, Bild DE, Oberman A, *et al*. Cigarette smoking and submaximal exercise test duration in a biracial population of young adults: the CARDIA study.
14. Morshedi-Meibodi A1, Larson MG, Levy D, O'Donnell CJ, Vasan RS. Heart rate recovery after treadmill exercise testing and risk of cardiovascular disease events (The Framingham Heart Study).
15. Ekelund LG, Haskell WL, Johnson JL, Whaley FS, Criqui MH, Sheps DS. Physical fitness as a predictor of cardiovascular mortality in asymptomatic North American men. The Lipid Research Clinics Mortality Follow-up Study. N Engl J Med. 1988; 319:1379-1384.
16. Blair SN, Kohl HW, Paffenbarger RS Jr, Clark DG, Cooper KH, Gibbons LW. Physical fitness and all-cause mortality. A prospective study of healthy men and women. JAMA. 1989; 262:2395-2401.
17. Roger VL, Jacobsen SJ, Pellikka PA, Miller TD, Bailey KR, Gersh BJ. Prognostic value of treadmill exercise testing: a population based study in Olmsted County, Minnesota. Circulation.
18. Goraya TY, Jacobsen SJ, Pellikka PA, *et al*. Prognostic value of treadmill exercise testing in elderly persons. Ann Intern Med. 2000; 132:862-870.