International Journal of Science and Healthcare Research
Vol. 8; Issue: 3; July-Sept. 2023
Website: ijshr.com
Original Research Article *ISSN: 2455-7587*

Effect of Smoking on Heart Rate at Rest, During Exercise, and on Heart Rate Recovery in Young Males

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DOI: https://doi.org/10.52403/ijshr.20230341

ABSTRACT

Background and need of research: Smoking contributes to several respiratory diseases due to its hazardous effect on respiratory and circulatory system which ultimately affects cardiovascular system. HR is very important, non-invasive and easy-to-measure index of myocardial work. From this study, difference in heart rate can be identified at an early stage among smokers to prevent effects of smoking on cardiac system.

Method: An Observational study was conducted in individuals of age between 18 to 45 years. 50 Male subjects who were smoking since more than 2 years, having normal BMI (18.5-22.9Kg/m2) according to Asian classification were included. Person with Severe Cardio respiratory disorder, neurological conditions, any fracture or musculoskeletal disorder which may hamper in step test were excluded. Subjects were divided into 2 groups: 1] Smokers and 2] Non-smokers.HR was measured at rest, during and post 5 minutes of Queens College Step Test to compare between both groups.

Result: Mean age, BMI, HR rest, HR during exercise and post HR of smokers were (36.24±6.65), (22.09±0.7), (77.2±4.89), (120.96±5.54) and (90.16±5.72) and in non-smokers were (35.76±7.03), (21.78±1.1), (46.88±6.46), (139.16±8.17) and (73.92±5.9) respectively. Comparison was done using unpaired-t test, Smokers had significantly higher HR at rest, slower HR increase during exercise compared to non-smokers. During recovery, the HR decline was significantly attenuated in smokers.

Conclusion and clinical implication: Smoking is found to affect young male smokers' Heart Rate. Therefore, more actions should be taken to spread awareness about its harmful effects and smoking cessation, especially in young adults.

Keywords: Smoking, Queens College Step Test, Heart Rate

INTRODUCTION

The number of deaths caused by smoking has increasing day by day. [1] Smoking contributes to the development of cancer and several respiratory diseases due to its effect on respiratory and circulatory system which ultimately affect cardiorespiratory endurance. [2]

Smoking is a major risk factor for cardiovascular morbidity and mortality, and is considered to be the leading preventable cause of death worldwide. [3] There is an established link between heart rate (HR) and cardiovascular health. [4] HR is very important, non-invasive and easy-to-measure index of myocardial work. [5] Overall, HR responses to cigarette smoking may be implicated in the link between smoking and cardiovascular diseases. [6]

Among various indirect protocols, Queen's College Step Test or QCT is the simplest one. The step test is performed on a stool of 16.25 inches (or 41.3 cm) height for a total

duration of 3 min at the rate of 24 cycles/min, which is set by a metronome.[7]
Queens college step test is a valid method to evaluate cardiorespiratory fitness and is considered gold standard in determining cardiorespiratory fitness.[8]
It is well-known that smoking is hazardous for health which is depicted by changes in heart rate of the smokers. From this study, difference in heart rate can be identified at an early stage among smokers to prevent effects of smoking on cardiac and other body systems.
With this background, this study aimed to find out effect of smoking on heart rate in smokers compared to non-smokers.

METHODOLOGY

Study Design: Cross-sectional, Observation study
Study Setting: Ahmedabad
Participants: young smokers and non-smokers
Sample Size: 50
Duration of study: One month

INCLUSION CRITERIA

- Gender: Male
- Age: 18- 45years
- Body Mass Index: Normal range: 18.5-22.9 kg/m^2 ***(****According to ASIAN BMI Classification*.)
- In smoker group, subjects who are smoking since past 2 years.
- Subjects who are fit to perform step test according to PAR-Q scale.
- Subjects willing to participate.
- Subjects who are able to follow the commands.

EXCLUSION CRITERIA

- Severe Cardio respiratory disorder
- Neurological conditions
- Any fracture or musculoskeletal disorder which may hamper in step test.
- History of drug abuse or alcohol
- Regular exercise person
- Uncontrolled Diabetes mellitus, Hypertension or Hyperlipidaemia

MATERIALS

1. Weight Machine
2. Stadiometer
3. PAR-Q form
4. Wooden stepper of height 16.25 inch (41.25 cm)
5. Stop watch
6. Pulse oximeter
7. Metronome
8. Calculator
9. Proforma
10. Consent form

PROCEDURE

Ethical committee approval was taken. Subject falling in inclusion criteria was invited for the study.
Written consent was obtained. Height was measured using a stadiometer. Weight was measured using portable weighing machine. BMI was calculated using the formula - Weight (kg)/ Height (m²) [will be classified according to ASIAN Classification]
Subjects were divided into 2 different groups:
1] Smokers
2] Non-smokers

PAR- Q (Physical Activity Readiness Questionnaire)

PAR-Q screening of the participant was done to evaluate readiness before engaging in fitness test.

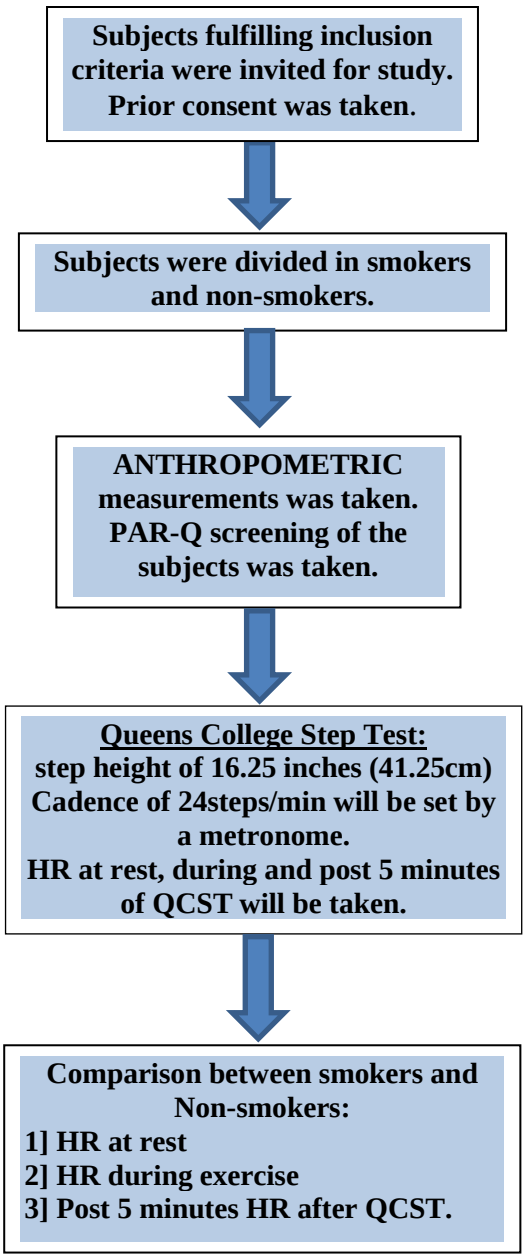
Queens College Step Test: [9]

Subjects falling in inclusion criteria were asked to perform Queens College Step Test. Participants were be required to step up and step down on a wooden stepper with standardized step height of 16.25 inches (41.25cm).

Cadence of 24steps/min was set by a metronome. Participants was instructed to perform the steps using a four-step cadence "up-up-down-down" continuously for 3 minutes. Continuous monitoring of BP, RPE, HR and SpO_2 was done during step test to prevent adverse effects.

HR was measured at rest, at peak and post 5 minutes of QCST. They have to maintain their determined stepping rhythm during the test and stop immediately on completion of test.

After taking values of heart rate, comparison of these values between smokers and non-smokers was done.



OUTCOME MEASURE:

Heart Rate at rest, during exercise and post 5 minutes of Queens College Step Test.

STATISTICAL ANALYSIS

Statistical analysis was done using SPSS version 20. Data was checked for normal distribution using Shapiro-Wilk test.

Data was normally distributed and are presented as mean ± standard deviation. Data analysis was performed using unpaired t-test for comparison between both groups. P< 0.05 was considered as statistically significant.

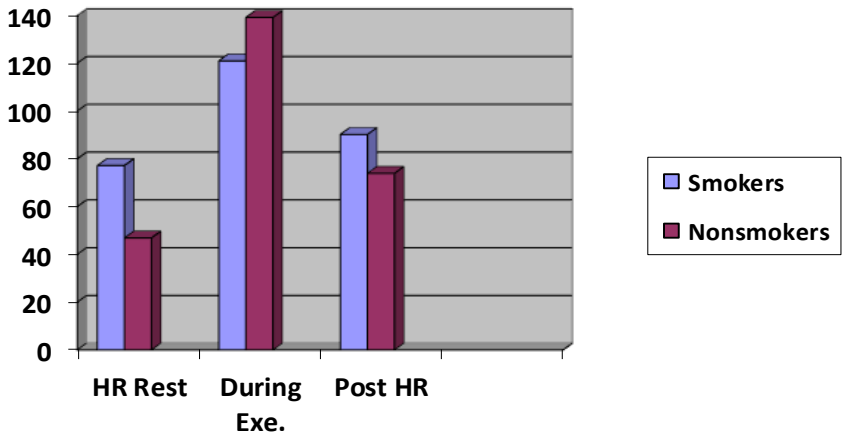
RESULT

Mean age, BMI, HR rest, HR during exercise and post HR of smokers were (36.24±6.65), (22.09±0.7), (77.2±4.89), (120.96±5.54) and (90.16±5.72) and in non-smokers were (35.76±7.03), (21.78±1.1), (46.88±6.46), (139.16±8.17) and (73.92±5.9) respectively.

Comparison was done using unpaired-t test, Smokers had significantly higher HR at rest, slower HR increase during exercise compared to non-smokers. During recovery, the HR decline was significantly attenuated in smokers.

Variables	Smokers	Non-smokers
Mean age	36.24±6.65	35.76±7.03
BMI	22.09±0.7	21.78±1.1
HR rest	77.2±4.89	46.88±6.46
HR during exercise	120.96±5.54	139.16±8.17
post HR	90.16±5.72	73.92±5.9

Table 1: Mean age, BMI, HR rest, during exercise & post HR



DISCUSSION

HR REST:

In the present study, smoking was found to affect the resting and exercise HR responses in young male smokers. Smoking contains nicotine which increases resting heart rate via increase in myocardial contractility. In addition, smoking impairs baro-reflex sensitivity which ultimately leads to rise in blood pressure and heart rate. These results are in line with previously published data from young populations [10,11,12,13] and they are also in agreement with many HR-related studies of healthy middle-aged populations, where smoking has been associated with increased resting HR values. [14,15]

More specifically, smoking, acting at peripheral sympathetic sites, increases circulating levels of catecholamines, [16] augments sympathetic outflow, [17,18] and causes a long-term reduction in vagal drive.[19] This sympathetic predominance, seen even in young heavy smokers[19] is also associated with impaired baro-reflex function [20] leading to a marked increase in HRrest.

HR DURING EXERCISE:

During exercise, the increased metabolic demands are met by an increased cardiac output, achieved through an augmentation in HR and stroke volume. The elevation of HR-confounded by age, HRrest and exercise capacity[5,21] is regulated by exercise induced autonomic control, where sympathetic activity increases and vagal tone is reduced. In the present study, male smokers showed a significantly slower HR increase during testing compared to non-smokers. Our results are also in line with many HR-related epidemiological studies in healthy middle-aged[14,22,23] or young populations, where smoking was always significantly associated with an impaired HR increase during exercise, a lower HRR or a lower HRmax achieved.[24] It has been reported that smoking blunts HR elevation during progressive exercise, posing an increased risk to smokers' health.[25,26] Adaptations to chronic smoking, such as down-regulation of β-adrenergic receptors, have been used in order to explain smokers' blunted HR response to exercise.[25,27] Long-term smoking has been found to decrease the density of lymphocyte or adipose tissue β-receptors, down-regulating the β-receptors of the cardiovascular system.[16] The down-regulation of β-adrenergicreceptors may explain why β-adrenergic blockers are not so effective in smoking cardiac patients who, despite β-adrenergic blockade, have higher HRsubmax compared with non-smoking cardiac patients.[28] However, it is questionable whether these adaptations, which usually refer to middle-aged or older-aged smokers, can explain the chronic effects of smoking on HR response during exercise in healthy young adults. In addition, it must be considered whether it may be the smokers' impaired exercise capacity that results in their inability to achieve a good exercise response and thus an adequate HR increase.[29]

HR DECLINE DURING RECOVERY:

After the termination of exercise, sympathetic activity is withdrawn and vagal reactivation mediates the rate at which HR declines. HR decline during recovery is a useful marker of cardiac autonomic control, being directly associated with the intensity of post-exercise parasympathetic activity.[30,31] Attenuated HR is defined as abnormal if it declines by ≤12 bpm in the first post-exercise minute for tests that use a cool-down protocol, or by ≤18 bpm for protocols that stop exercise abruptly.[30,31] Abnormal HR following exercise test is directly related with a higher risk of cardiovascular disease,[30,32] being also an independent predictor of mortality.[31] In this study, HR decline during recovery was attenuated in young male smokers. In many epidemiological HR-related studies in healthy middle-aged populations, smoking was inversely associated with HR decline during recovery.[32] There are very few studies that have examined the association between smoking and HR recovery in young adults. Kobayashi et al reported that young smokers had attenuated HR decline after sub-maximal exercise.[13] In contrast, in the CARDIA study the prevalence of smoking was significantly lower in the slower HR recovery quartiles.[33] The important differences in the research design of the studies just mentioned, such as the workload intensity at termination of exercise (maximal vs. sub-maximal) and the recovery protocol (cool down vs. abrupt cessation of exercise) may explain the discrepancy between the results. There is a direct correlation between the HRmax achieved at peak exercise test and the subsequent HR decline during

recovery.[31,32] This relationship may partially explain our results , where smokers had a significantly lower HRmax and blunted HR recovery comparing to non-smokers. In addition, it has been found that an attenuated HR recovery may also represent a marker of decreased exercise capacity, independently of health. [30,31] Indeed, the CARDIA study reported a significant direct p-trend towards a decreased exercise capacity as HR recovery slowed.[33]

However, as gender-specific effects are not fully explained, further studies are needed in the future to thoroughly examine and establish findings.

Limitations & future recommendation:

The main strength of this study was the strict selection criteria used for the enrolment of subjects from a well-defined and homogeneous target population. On the other hand, the size of the sample is the main limitation. In addition, it can be argued that it is preferable to use objective measures in order to define smoking selection criteria, such as serum nicotine or cotinine levels, rather than formal questions. Finally, our target population consisted of healthy young males but Socio-economic status, smoking years, dietary habits, as well as other factors might differ the results.

Given the grave consequences of smoking for cardiovascular health, the early detection of its effects on resting HR and on HR responses during exercise is of great importance and supports the need for prompt cessation of the smoking habit, especially in young male population. Although there appears to be a general agreement that smoking increases resting HR, further research is needed for a thorough evaluation of the effects of tobacco on HR changes during and after exercise, since abnormal HR responses may serve as an important prognostic factor for future cardiovascular morbidity.

CONCLUSION

In present study, Smoking is affecting young male smokers' HR deleteriously. There is increased in HR at rest, slowing HR increase during exercise, attenuated heart rate decline during recovery in smokers compare to non-smokers.

Therefore, more actions should be taken to spread awareness about its harmful effects and smoking cessation, especially in young males.

Declaration by Authors

Ethical Approval: Approved
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

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How to cite this article: Aarti Pandya, Sweety Shah. Effect of smoking on heart rate at rest, during exercise, and on heart rate recovery in young males. *International Journal of Science & Healthcare Research.* 2023; 8(3): 296-303. DOI: *https://doi.org/10.52403/ijshr.20230341*

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