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ASSESSMENT OF CARDIAC AUTONOMIC DERANGEMENT IN SMOKERS BY COUNTING RESTING HEART RATE AND COUNTING HEART RATE AFTER INDUCING STRESS

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Abstract

Introduction

There is an established link between Heart Rate (HR) and cardiovascular health. HR is a very important vital parameter and it is an index of myocardial activity. According to the previous studies, smokers resting HR will be more due to autonomic dysfunction. The body releases a surge of hormones when under stress. These hormones cause the heart to beat faster and the blood vessels to narrow. Reacting to stress in unhealthy ways can raise blood pressure and increase the risk of heart attack and stroke.

So counting resting HR and HR after inducing stress in the form of left arm local ischemia may help in assessing cardiovascular health.

Objectives

To assess the effect of smoking on resting heart rate

To assess cardiac compliance by counting HR after inducing stress by left arm local ischemia

Methodology

After getting clearance from ethics committee, informed consent was taken from all the participants and different parameters were recorded as per study protocol.

Data collection was done in the Department of Physiology, Narayana Medical College, Nellore

Results

In our study, we found that the Resting HR was statistically significant in smokers when compared to non smokers with $P< 0.001$. There was no significant increase in HR after inducing local ischemia in smokers ($P = 0.45$). Where as in non-smokers HR was significantly increased ($P = 0.00094$)

Conclusion

Resting HR will be more in smokers because smoking increases circulating levels of catecholamine, augments sympathetic outflow, and causes a long-term reduction in vagal drive. Assessment of HR after inducing stress by left arm local ischemia may help in assessing cardiac compliance and help in early identification of cardiac complications.

Key words - Autonomic dysfunctions, Ischemia, Resting heart rate, Smokers & Non smokers

INTRODUCTION

The American Heart Association states the normal resting adult human heart rate is 60–100 bpm. An ultra-trained athlete would have a resting heart rate of 37–38 bpm. Increase in heart rate above 100 bpm is called tachycardia and decrease in heart rate below 60 bpm is called bradycardia. Heart rate during sleep will be around 40–50 bpm is common and considered normal. Abnormalities of heart rate sometimes indicate disease. Heart rate is an important vital sign and it is an index of myocardial activity.

Smoking is one of the primary cause of preventable illness. Smoking harms nearly every organ of the body and reduces both quality of life and life expectancy.

Most smoking-related deaths are from one of three types of disease: lung cancer, chronic obstructive pulmonary disease (COPD) & Coronary Heart Diseases (CHD).

According to WHO estimation, tobacco continues to kill - 6 million people each year, more than 600,000 passive smokers (heart disease, lung cancer, and other illnesses). If current trends continue, the death toll may reach more than 8 million per year by 2030.

Smoking disturbs vascular endothelial homeostasis by reducing the bioavailability of vascular nitric oxide (NO), mainly via the formation of oxidative stress and inflammatory processes . Since the formation of NO plays a key role for the endothelium to perpetuate its vasodilatory, antithrombotic, anti-inflammatory, and antioxidant functions, this pathological state promotes endothelial dysfunction and atherosclerotic plaque formation, ultimately leading to structural changes in the arterial wall and arterial stiffening . Importantly, arterial stiffness has been shown to be an independent risk factor for CVD and all-cause mortality after adjustment for traditional cardiovascular risk factors.

Cardiac sympathetic hyperactivity is a dominant feature in cigarette smokers.
The body releases a surge of hormones when under stress. These hormones cause the heart to beat faster and the blood vessels to narrow. These actions increase blood pressure for a time. Reacting to stress in unhealthy ways can raise blood pressure and increase the risk of heart attack and stroke.

Stress hormones are produced by activation of SNS and hypothalamic-pituitary adrenocortical axis. The SNS stimulates the adrenal medulla to produce catecholamines (e.g., epinephrine). In parallel, the paraventricular nucleus of the hypothalamus produces corticotropin releasing factor, which in turn stimulates the pituitary to produce adrenocorticotropin. Adrenocorticotropin then stimulates the adrenal cortex to secrete cortisol. Together, catecholamines and cortisol increase available sources of energy by promoting lipolysis and the conversion of glycogen into glucose (i.e., blood sugar). Lipolysis is the process of breaking down fats into usable sources of energy (i.e., fatty acids and glycerol).

Energy is then distributed to the organs that need it most by increasing blood pressure levels and contracting certain blood vessels while dilating others. Blood pressure is increased with one or two hemodynamic mechanisms.

The myocardial mechanism increases blood pressure by increasing cardiac output. So increase in heart rate and stroke volume.

The purpose of this study is to assess the effects of smoking on resting HR and to assess HR responses after inducing stress by left arm local ischemia in smokers might help in assessing cardiovascular compliance and also early identification of persons at high risk.

Objectives

- To assess the effect of smoking on resting heart rate
- To assess cardiac compliance by counting HR after inducing stress by left arm local ischemia in smokers

Materials

- Case control study
- After getting clearance from ethics committee, informed consent was taken from all the participants and different parameters were recorded as per study protocol.
- Data collection was done in the Department of Physiology, Narayana Medical College, Nellore.

Participants

- 30 Smokers - aged between 20 - 40yrs, with duration of smoking more than 1yrs.
- 30 Non smokers – aged between 20 – 40yrs

Inclusion criteria

- Patient aged 20 – 40 yrs
- Males
- Smokers using beedi/cigarette (more than 1yrs)
- No history of diabetes mellitus / Hypertension or other systemic disorders

Exclusion criteria

- Patient aged < 20 and > 40 yrs
- History of diabetes mellitus / Hypertension or other systemic disorders
- Females
- Smokers (less than 1yrs)

Parameters

- Height - Stadiometer
- Weight - Digital weighing balance
- Body Mass Index (using formula weight in Kg/ height in meter square)
- Heart rate - calculated by recording finger pulse waves using finger pulse sensor connected to digital PPG (In house built) during rest as well as after inducing stress

METHODOLOGY

The subject was asked to come to the laboratory between 9 to 11am, After instrumentation 10mts rest is given.

- Height , Weight & Body mass index were recorded
- BP cuff was tied on left arm
- Finger pulse sensor was placed on left index finger it was connected to digital PPG and to computer.
- Finger pulse was recorded continuously for 1 mt
- Pressure in the BP cuff is raised 30mmHg above the systolic pressure and maintained it for 3 mts to induce ischemia and suddenly cuff pressure is released .
- Finger pulse recording was carried out throughout the procedure continuously.

RESULTS

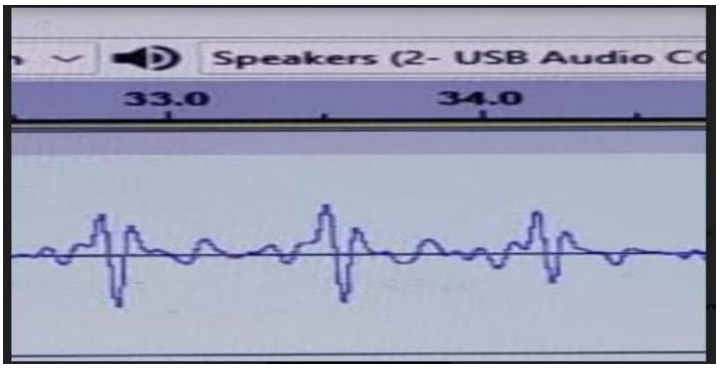
Table - 1 General characteristics of the subjects

Parameters	Smokers Mean ± SD	Non smokers Mean ± SD
Age (yrs)	25.2 ± 8.5	18.6 ± 0.9
Height (cms)	169.4 ± 7.1	174.9 ± 6.3
Weight (Kgs)	66.1 ± 15.5	69.4 ± 12.6
BMI	23.1 ± 5.7	22.7 ± 3.9

Table - 2 Cardiovascular parameters of smokers

Subjects	HR Before inducing ischemia	HR after inducing ischemia	P Value
Smokers Mean ± SD	86.2 ± 12.8	87.3 ± 10.6	0.456
Non smokers Mean ± SD	70 ± 10.5	74.9 ± 11.3	0.0009

Finger -1 Pulse recording device



Finger- 2 Digital finger pulse recording

- In our study , we found that,
  - The resting HR was statistically significant in smokers when compared to non smokers with $P < 0.001$.
  - There was no significant increase in HR after inducing stress by left arm local ischemia in smokers which says no sympathetic hyperactivity ($P = 0.45$) Where as in non-smokers HR was significantly increased ($P = 0.00094$).

DISCUSSION

- Normal resting heart rate will be 60 – 80 beats/mt because SA node will be under vagal tone i.e Parasympathetic nervous system activation releases acetylcholine , decrease Ca influx and increase K efflux which results in less HR.

According to our study,

- In smokers , Resting HR was more than non smokers
- Because in smokers nicotine increases the cardiac output by increasing both the heart rate and the myocardial contractility.
- Smoking increases in circulating levels of catecholamine, which augments sympathetic outflow and causes reduction in vagal drive.(norepinephrine – B1 recptor – increase Ca influx into SA node – increase in HR)

- In smokers, There was no significant increase in HR after inducing stress by left arm local ischemia (Normally HR should increased after inducing ischemia) which shows no sympathetic hyperactivity ($P = 0.45$) Where as in non-smokers HR was significantly increased ($P = 0.00094$).
- Nicotine besides being addictive, also causes narrowing and hardening of arterial walls all over the body making it difficult for the heart to pump blood through the constricted arteries.

Conclusion

- So assessment of resting HR in smokers, which is a simple one can be a screening tool for identification of person who is at high risk and it indicates prompt cessation of the smoking habit, especially in young people.
- As we all know that there are smoking cessation programs like COTPA Act 2003 (cigarette other products act) – according to this 1) Prohibition of public smoking 2) prohibition of advertisement of tobacco products, 3) Restriction of sales to minors 4) warning symbol on front of the pack 5) GST increased to 18 % should be implemented strictly.

- We feel that further research should be focused in this field to prevent complications of tobacco

Ethical clearance - Taken
Conflict of Interest – Nil
Source of Funding – Self

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