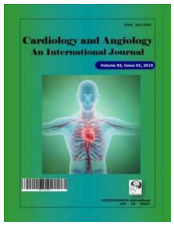

Cardiology and Angiology: An International Journal
4(2): 80-89, 2015, Article no.CA.2015.030
ISSN: 2347-520X

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Effects of Smoking on Arterial Stiffness in Male Adolescents in Lusaka, Zambia

Chikopela Theresa[1*] and M. Goma Fastone[2]

[1]Lusaka Apex Medical University, Basic Sciences, Zambia.
[2]The University of Zambia, School of Medicine, Zambia.

Authors' contributions

The study was a collaborative effort from the Cardiovascular Sciences Laboratory at University of Zambia School of Medicine. Author CT designed the experimental protocol, conducted the experiments and data analysis, and drafted the manuscript. Author FMG supervised the study protocol, data collection and management, and the finalization of the manuscript. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/CA/2015/19292
Editor(s):
(1) Wilbert S. Aronow, University of California, College of Medicine, Irvine, USA.
Reviewers:
(1) Damyanti Agrawal, Department of Cardiovascular and Thoracic Surgery, Banaras Hindu University, India.
(2) Tomohiro Osanai, Hirosaki University, Japan.
(3) Anonymous, Turkey.
(4) Anonymous, Safarik University, Slovak Republic.
Complete Peer review History: http://sciencedomain.org/review-history/10291

Original Research Article

Received 1st June 2015
Accepted 15th July 2015
Published 24th July 2015

ABSTRACT

Background: Tobacco smoke is harmful to health. In the acute phase it causes changes in the cardiovascular system that result in increase in blood pressure (BP). An increase in arterial stiffness due to arteriolar endothelial dysfunction has been cited among the causes. Pulse Wave Velocity (PWV) and Arterial Stiffness Index (ASI) are used as measures of arterial stiffness in the adult population.
Aim: To determine the acute effects of tobacco smoke on arterial stiffness in black male adolescents in Lusaka, Zambia.
Study Design: This was an observational study done at the University of Zambia School of Medicine Cardiovascular Research Laboratory in the month of December 2014.
Methodology: Twenty-two (22) black, male-adolescent (age range 19-25 years), active-smokers, consented to participate in the study. The Complior Analyse Unit (V1.9 Beta Version 2013; ALAM-Medical, France) protocol was used to obtain the carotid-femoral PWV (cfPWV) and carotid-femoral

**Corresponding author: E-mail: theresachikopela@yahoo.com, gomafm@unza.zm;*

ASI (cfASI) starting 15 minutes before smoking, on immediate cessation of smoking and thereafter every 15 minutes up to an hour after smoking. ASI was a surrogate measure of the loss of elasticity in the arteries.

Results: The mean baseline cfPWV was 7.9±1.94 m/s and cfASI was 26.1±6.0 m/s. Smoking two tobacco cigarettes (2.8 mg Nicotine) in 15 minutes caused an increase in mean cfPWV and cfASI from their baseline values to cfPWV of 8.5±1.87 m/s and cfASI of 28.6±6.19 m/s respectively. These values reverted to baseline within 15 minutes post-smoking cessation. There was further reduction in both cfPWV and cfASI to more stable values at 45th and 60th minutes which were statistically significantly lower than the peak values recorded.

Conclusion: The mean baseline cfPWV and cfASI in these late adolescents were comparatively higher than those recorded in non-smoking black adolescents and smoking white men and women (see Lemogoum, 2006). These recordings were also much higher than the values recorded 60 minutes after cessation of smoking. Compared to these values, we conclude that tobacco smoke may be the cause of the significant acute increase in cfPWV and cfASI in African male adolescents presumably signifying an increase in arterial stiffness probably due to endothelial dysfunction in elastic arteries. These alterations in vascular compliance may predispose these individuals to developing hypertension and other cardiovascular complications. There is need for further investigation of this phenomenon.

Keywords: Pulse Wave Velocity (PWV); Arterial Stiffness Index (ASI); arterial stiffness; adolescents.

1. INTRODUCTION

Tobacco use, including cigarette smoking, is said to be the single most common cause of preventable morbidity and mortality globally. At present, tobacco use is responsible for 10% of all global deaths from cardiovascular diseases and is the second leading cause of cardiovascular diseases (CVD), after high blood pressure [1]. According to the 2014 Zambia Demographic and Health Survey, 19.8% of men age 15-49 years reported smoking cigarettes, a pipe, or using other tobacco products [2]. The number of adolescents that have ever-smoked cigarettes in Lusaka were reported as 19.3% overall among 3,377 respondents aged 13-15 years. This comprised 20.2% of the boys and 17.7% of the girls. ZDHS (2) also recorded an increase in smoking prevalence among late adolescents (age 20 – 24 years) of over three percentage-points from 2007.

Adolescents are said to be an emerging priority population for the tobacco industry which seems to be oblivious to the harms caused by promoting tobacco addiction. This age has been defined as the period in human growth and development that occurs after childhood and before adulthood (3). This 'habit forming' period is the stage when the smoking habit most easily gets initiated. Considering the importance of the late adolescence stage in human growth and development, this study focused on individuals aged 18 to 25 years. There are still very significant gaps in data pertaining to the harms of tobacco smoking in this age group.

There is paucity of data on the effects of smoking in adolescents with respect to haemodynamic variations and arterial stiffness. While there is documentation of these in experimental settings elsewhere, there is no study that has been done on Zambian adolescents. Cigarette smoking is known to cause changes in the cardiovascular system including changes resulting from alterations in peripheral resistance and cardiac output resulting in an increase in blood pressure [4]. Various mechanisms have been proposed for the hazardous effects of smoking that lead to cardiovascular morbidity and related mortality. These include changes in the haemostatic factors, the endothelial function and the blood lipids, as well as alterations in the dynamic properties of the arterial wall [5,6].

An increased arterial stiffness has been demonstrated by an increased Pulse Wave Velocity (PWV). This alteration in PWV is said to be due to changes in vascular compliance resulting from endothelial dysfunction. An increase in plasma catecholamines and an impaired nitric oxide (NO) production are the likely cause of the endothelial dysfunction. The catecholamines have vasoconstrictor influences while the impaired NO production will cause vasodilatory dysfunction. NO is an endothelial derived free radical that is primarily responsible for the vasodilatory function of the endothelium and also helps regulate inflammation, leukocyte

adhesion, platelet activation, and thrombosis [7] Therefore, an alteration in NO biosynthesis could have both primary and secondary effects on the initiation of stiffening of arteries. Indeed chemicals noted in cigarette smoke include highly toxic/heavy metals that even in very small amounts may be potentially harmful.

Arterial stiffness describes the reduced capability of an artery to expand and contract in response to pressure changes. Increased arterial stiffness is a determinant of cardiovascular mortality contributing to cardiovascular risk [8,4]. PWV is said to be the gold standard for measuring arterial stiffness. During systole, the contraction of the left ventricle and the ejection of blood into the ascending aorta acutely dilates the aortic wall and generates a pressure wave (pulse wave) that moves along the arterial tree. The velocity of this movement is a factor of arterial compliance. Indeed PWV is noted to increase when arteries are stiffened [9,10].

Acute cigarette smoking has been shown to increase the PWV, suggesting that there may be increased arterial stiffness in adult smokers [11]. Cryer et al. [12] described a corresponding increase in plasma catecholamines that peaked at the end of a 10-min period of smoking and returned to baseline levels 30 minutes after the end of smoking.

Another index used in this study was the Arterial Stiffness Index (ASI). The Pulse Wave Velocity is known to be influenced by several factors such as age, sex and body height. The ASI corrects for a subjects height and can therefore better determine levels of arterial stiffness [13]. ASI is therefore another measure of the loss of elasticity in the arteries that occurs with onset of vascular disease. There is said to be pronounced increase in ASI in cases of damaged vascular endothelium due to arterial stiffening [14]. This study aimed at determining the acute effects of smoking on arterial stiffness in black male adolescents using Pulse Wave Velocity and Arterial Stiffness Index and characterising the time course of the change in arterial stiffness to return to baseline after stopping to smoke.

2. METHODOLOGY

This observational study involved healthy young men who were active tobacco smokers. These were recruited by advertisements placed on the student notice boards within the University of Zambia (Ridgeway campus) and also by the use of the chain–referral (snowball) sampling. The sample population included all tobacco smoking, male adolescents between ages 18 and 25 years who were daily smokers (someone who has smoked daily during the past month). Excluded were adolescents with known hypertension, diabetes mellitus, high levels of cholesterol, and respiratory diseases such as bronchitis or asthma.

The participants were required to abstain from smoking prior to the protocol and to abstain from taking alcohol and coffee beverages for at least 12 hours before the study period. Written consent was obtained from each one of the participants and they were assured that denial of participation was not consequential on their academic progression and they were free to withdraw from the study at any time. All measurements taken were non-invasive and were taken in a research unit specifically designed for participant's privacy and comfort. All participants recruited were smokers and none of them were subjected to abnormal levels of smoking (more than what the participant usually smokes). Any abnormalities in the results were highlighted and the participants were advised to consult a clinician as appropriate. All data was stored on a trusted, password protected computer. Identification of participants was by unique research laboratory numbers and was stored as such. The research ethics clearance was obtained from the University of Zambia Biomedical Research Ethics Committee (UNZABREC).

2.2 Baseline Information

All consenting participants were invited to the Cardiovascular Laboratory at the University of Zambia, School of Medicine. They were interviewed to note socio-demographic data and health information such as age, marital status, current medications, existing pathological conditions, age at which participants started smoking, duration of smoking to date, amount of cigarettes smoked in a day, type of cigarettes smoked, alcohol consumption and time of last meal and beverage consumption.

2.3 Anthropometric Measurements

Height and weight were measured using a Micro T3 PW-BMI Digital Physician Scale with a height measure. Height was measured in meters (m) while weight was measure in kilograms (Kg). Blood pressure was measured by OMRON HEM-757 (Omron, Kyoto, Japan) digital blood pressure

measuring machine in both sitting and lying down positions (mmHg).

2.4 Pulse Wave Velocity Data Collection

The participants were instructed to lie down comfortably on a surface as flat as possible with no pillow or reclining seatback. The length from the carotid artery in the neck to the femoral artery at the groin was measured using the Finger-Finder measuring tape. The PWV was determined by the Complior Analyse Unit (V1.9 Beta Version 2013; ALAM-Medical, France) which has predefined sensors/probes for the carotid, femoral and radial pulses. In this procedure, the carotid-femoral PWV (cfPWV) was measured. The carotid probe was gently placed on the carotid artery on the neck at the point where the strongest carotid pulse was palpable.

The participant's groin was then exposed for access to the femoral artery. The probe was applied on the femoral artery and adjusted to obtain the best signal. In cases where the femoral artery was deep, some pressure was applied and once the probes were correctly in place, pressure signals from the sensors were displayed on the screen of the Complior Analyse Unit. The values for PWV, heart rate and transit time were displayed on the screen and were then transferred on to data collection sheets.

Baseline measurements were obtained for 15 minutes at 5-minute intervals and the average reading obtained for each variable was the baseline value. Following baseline measurements, the participant was requested to smoke two cigarettes (1.4 mg nicotine content each) in fifteen minutes. The PWV was recorded immediately following cessation of smoking and thereafter for an hour at 15-minute intervals into the recovery period.

2.5 Data Management and Analysis

The means and ranges for baseline data were obtained using STATISTIX statistical package for Windows Version 10, 2013. Using the Complior Analyse Unit, the pulse transit time was determined as recorded during PWV measurement. To correct for the size of the participant, the transit time was divided by the height of the participant. The resultant value was noted as the Arterial Stiffness Index (ASI), which was expressed in meters/second as was done by Binder and the group [15].

The mean for each variable before smoking was noted as the baseline value. The means of ASI and PWV obtained from the Complior Analyse Unit before and after smoking were compared to note for significant differences. All data was expressed as the mean ± standard deviation from mean. Analysis of Variance (ANOVA) of repeated measures was used for the comparison of participant's parameters before and after smoking. To determine the exact points of significant difference, the Bonferroni all pairwise comparison test was used.

3. RESULTS

3.1 Anthropometric and Baseline Data for Smokers

Twenty-two (22) male participants were recruited for the study. The participants were all active-smokers aged between 19 and 25 years old. The anthropometric data of the study population is shown in Table 1. They had an average weight of 61.5±11 Kg which ranged from 49 kg to 92.2 kg. With a maximum body mass index (BMI) of 25.5 kg/m^2 none of them could be described as overweight or obese.

As noted in Table 2, among the 22 smokers, the mean number of cigarettes smoked per day was 6.9±5.05 cigarettes. The group was characterised by smokers who had a total duration of smoking ranging from 12 months to 192 months. The youngest age at which individuals initiated the habit of smoking was 7 years and the latest age was noted as 19 years.

3.2 Acute effects of smoking on the cfPWV and cfASI

The mean baseline cfPWV was 7.9±1.94 m/s. The values were noted to increase after smoking to 8.5±1.87 m/s. The cfPWV rapidly decreased over time with values recorded lower than the recordings obtained at baseline. There were statistical differences in cfPWV observed at the 45^{th} minute (7.7±1.49 m/s) and that observed immediately after smoking (8.5±1.87 m/s) (P =.04) as shown in Fig. 1. There was also a significant difference noted between the peak cfPWV value noted immediately after smoking and that observed at the 60^{th} minute (7.7±1.56 m/s). It is notable that in this study, PWV had reverted to baseline readings by the 15^{th} minute after cessation of smoking.

Table 1. Anthropometric and baseline data for smokers

Variable	Mean	SD	Minimum	Maximum
Age (years)	20.636	2.0827	19.000	25.000
Height (m)	1.688	0.0519	1.6000	1.8100
Weight (kilograms)	61.509	10.999	49.000	92.200
BMI (kg/m^2)	18.166	2.8103	14.244	25.470
cfPWV (m/s)	7.872	1.9423	4.5000	12.200
cfASI (m/s)	26.090	5.9796	17.174	39.765
SBP* (mmHg)	113.485	13.1525	88.500	156.00
DBP** (mmHg)	79.468	8.7860	60.000	116.00
Heart Rate (bpm)	74.25	13.7525	50.000	133.50

**SBP – Systolic blood pressure*
***DBP – Diastolic blood pressure*

Table 2. Smoking parameters in study population

Variable	Mean	SD	Minimum	Maximum
No. of cigarettes smoked per day	6.864	5.0455	1	20
Duration of smoking (months)	71.455	44.9730	12	192
Starting age of smoking (years)	14.681	3.0140	7	19

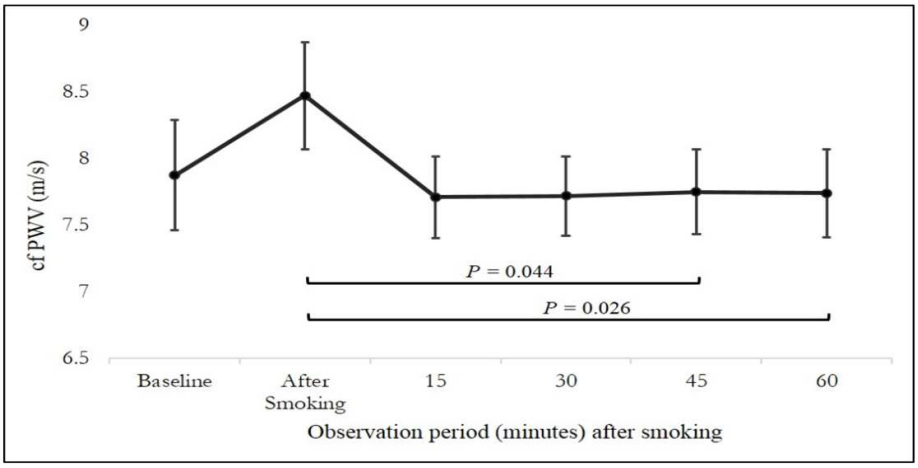

Fig. 1. Mean carotid-femoral Pulse Wave Velocity (cfPWV) (m/s) before and after smoking showing statistical significance of mean differences (*P*< .05). Bars represent the standard deviation

The mean baseline cfASI was 26.1±6.0 m/s. This increased to 28.6±6.19 m/s immediately after smoking (*P*=.08). The cfASI decreased significantly over time to values lower than those noted before smoking. The lowest cfASI was noted at the 45th minute (26.1±3.90 m/s) post-smoking. The cfASI value had actually returned to baseline value by the 15th minute.

4. DISCUSSION

4.1 Anthropometric and Baseline Data

A total of twenty-two (22) participants were recruited to participate in this study. All participants were active-smokers and eleven of them (50%) smoked 5 or more cigarettes per day. This high percentage of participants who smoked 5 or more cigarettes is unlike that reported by Siziya et al. [16] that showed only 11% of adolescents smoked more than five cigarettes daily. However, Siziya et al. [16] conducted their study on a larger sample than in this study. Aside the statistical bias that may arise from the smaller sample size in this study, the greater prevalence of adolescents smoking more cigarettes may be attributed to socio-demographic factors, such as socioeconomic status of the selected participants and developmental challenges associated with

adolescence. Environmental factors such as acceptability and availability of tobacco products, interpersonal variables and perceived environmental variables may also play a role in the increase in number of smokers who smoke more cigarettes daily.

The average age at which the participants started smoking was 14 years, with some reporting that they initiated the smoking at 7 years of age. They were all normotensive, with a mean blood pressure of 118/81 mmHg and none of the participants were overweight or obese. The highest blood pressure noted in this study was SBP of 156 mmHg and a DBP of 116mmHg. The relatively high blood pressure values could have been due to the white-coat effect. In this case the blood pressure observed is higher when it is taken in a medical or experimental setting than it is when taken at home. According to Mancia and colleagues [17] this white-coat effect can lead to falsely elevated recorded blood pressure values and mistaken diagnoses of hypertension, known as white-coat hypertension (WCH) [18]. WCH is thought to be present in 15% to 30% of people diagnosed with hypertension [19].

The mean baseline cfPWV in the study participants was 7.9±1.94 m/s. This was higher than the cfPWV noted by Lemogoum et al. [20] (6±1.6 m/s) in black adolescents who smoked. However, Lemogoum's study population comprised both males and females. This could have potentially contributed to the lower PWV values observed in Lemogoum's study as females have lower PWV compared to males [21]. This difference may also be a result of the increased frequency of smoking in this study population, causing a significant stiffening of the arteries.

Kingwell et al. [22] observed a lower mean PWV value (cfPWV of 6.2 m/s) in healthy, non-smoking, white young-adults compared to this study (7.9±1.94 m/s). The increased mean PWV noted in the current study could be due to the elastic arteries being stiffened as a result of smoking. Levenson et al. [23] and Jatoi et al. [24] observed that healthy non-smokers had significantly lower baseline mean PWV values compared to healthy smokers. They consequently postulated that chronic smoking increased arterial stiffness, and might be an underlying mechanism for the increased cardiovascular events observed in hypertensive patients. There is therefore an increased risk of hypertension and cardiovascular related diseases among the actively smoking adolescent population in Zambia.

Empirical evidence suggests that whites have a lower baseline PWV compared to blacks [20]. According to Schutte et al. [25] and Morris et al. [26], blacks have arterial stiffness that could already be elevated from earlier years of life, leaving them susceptible to an increased baseline PWV due to the influence of smoking. Lemogoum et al. [27] attributed these differences to a greater presence of free-radicals noted in blacks after cigarette smoking compared to whites. This may be a contributing factor to enhancing the PWV response to smoking in blacks.

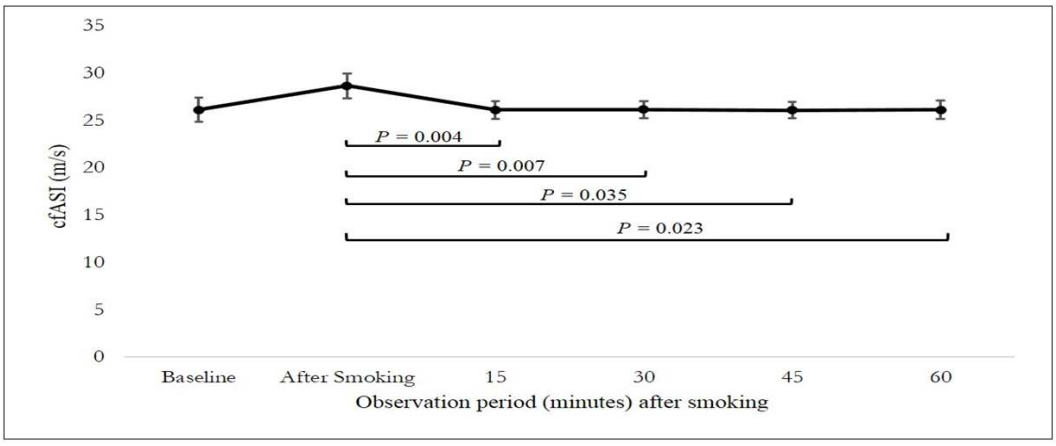

Fig. 2. Mean carotid-femoral Arterial Stiffness Index (cfASI) (m/s) before and after smoking with statistical significance of mean differences (*P*<.05) shown. Bars represent the standard deviation

4.2 Effects of Smoking on Arterial Stiffness

4.2.1 Effects of smoking on PWV

Arterial stiffness is measured by the rate of transmission of the arterial pulse pressure wave from the carotid artery, an upstream pressure point, to some defined downstream pressure points (Pulse Wave Velocity). The downstream pressure point used in this study was the femoral artery to obtain the cfPWV. The cfPWV offers the simplest reproducible and non-invasive evaluation of regional arterial stiffness as the aorta is the major component of arterial elasticity.

Pulse Wave Velocity (PWV) is defined as the speed at which the pulse wave travels on an arterial segment, (Complior Analyse Operator Manual, 2013) and is a measure of arterial stiffness; that is, the higher the PWV, the higher the arterial stiffness. Among other factors such as haemodynamic stress and lipid abnormalities, cigarette smoke has been noted to cause injury to the endothelium, significantly affecting the arterial compliance. Endothelial damage is a central feature in the evolution of vascular disease induced by smoking. Factors that continuously injure the intima may result in the formation of fibro-fatty plaques which cause endothelial dysfunction. Furthermore, the inhalation of cigarette smoke results in the activation of the adrenergic mechanism. The nicotine contained in tobacco stimulates the sympathetic nerve terminals, with consequent systemic release of epinephrine and norepinephrine. The released catecholamines bind to α_1-adrenergic receptors on the vascular smooth muscle to cause muscle contraction and a reduction in arterial distensibility exacerbating endothelial dysfunction and thus increasing the PWV [27,28].

In this study, smoking 2 cigarettes (2.8 g Nicotine) caused a rise in the cfPWV. The initial rise of 8% from 7.9±1.94 m/s to a peak of 8.5±1.87 m/s was statistically insignificant (*P*>.05). However, the values for cfPWV significantly reduced by the 45th minute to 7.7±1.49 m/s (*P*<.05) which persisted through the 60th minute 7.7±1.56 m/s (*P*<.05).The significant reduction in PWV noted during the recovery period could be due to these individuals having an exaggerated PWV in the 15 minutes before the experimentation ("baseline") as they anticipated the investigation and it may have returned to their "true baseline" after the experimentation. Indeed this "white coat" effect has been reported in several studies [29,30]. It would therefore be assumed that the true baseline cfPWV was around 7.7±1.56 m/s. This would then imply that there was indeed a statistically significant acute increase in PWV due to smoking in this group of 10.4% (*P*<.05).These findings are similar to the findings of Ozgur (27), who also showed that smoking 2 cigarettes (2.8 mg nicotine) caused significant changes in cfPWV in healthy smokers.

These observations were also similar to those noted by Lemogoum et al. [20], who had an increase in cfPWV in adolescent smokers (blacks and whites) after smoking. He noted a significant increase in cfPWV from 5.9±1.7 m/s to 6.5±1.5 m/s (39% increment, *P*<.05) after 5 minutes of smoking. The percentage increase in PWV was lower in this study compared to the increase noted by Lemogoum et al. [20]. This could have been due to the higher baseline PWV noted in the current study.

Other studies showing significant increase in PWV due to smoking include Vachapoulos et al. [28], Mahmud et al. [29] and Kool et al. [5]. The latter observed that smoking caused a short-term increase in arterial wall stiffness of both the elastic common carotid and the muscular brachial arteries. They postulated that such increase could be due to acute effects on the endothelial function [30]. Therefore, the increase in PWV observed in this study suggests that there is also an increase in arterial stiffness after acute smoking in the black, male adolescents.

The mean PWV was noted to return to baseline 15 minutes after the cessation of smoking. This is similar to the findings of Cryer et al. [12], who also reported a return to baseline of the PWV in 15 minutes. They also reported that plasma catecholamines were maximal at the end of a 10-minute period of smoking after which there was a decline. Catecholamines are said to cause impaired nitric oxide (NO) production and central nervous system sympathetic discharge, leading to endothelial dysfunction. PWV would therefore return to normal after the catecholamines have been cleared in the blood [20].

Other studies have shown a longer period of recovery of up to 45 minutes after smoking 2 cigarettes (nicotine content 3 mg) [31] and a recovery of 15 minutes after smoking 1 cigarette (nicotine content of 0.9 mg) [6]. Both authors observed the recovery period of PWV in

normotensive smokers. Their populations of Asians consisted of comparatively older males. In this study, nicotine content of 2.8 mg was used and it took 15 minutes for PWV in the elastic arteries (cfPWV) to return to baseline. The quick recovery noted in this study could be explained by the faster metabolism of nicotine noted in blacks compared to the Asians [32].

It has been observed that Asian male smokers have higher plasma nicotine levels after overnight tobacco abstinence compared with other males [33]. This could be the reason for the longer time taken for both Kim and Rhee's population to revert to baseline. Age is another factor that could have added to the difference noted in the recovery time. Clearance of nicotine decreases with age due to reduced liver blood metabolism [34].

4.2.2 Effects of smoking on ASI

In this study, cfASI increased by 9.6%,though statistically insignificant, after smoking from a mean baseline value of 26.1±6.0 m/s to 28.6±6.20 m/s. However, the record shows a significantly decreased cfASI at the 45th minute (26.1±3.90 m/s) when compared to the peak value noted immediately after smoking (P<.05). This persisted through the 60th minute as well. This also implies a possible exaggerated initial baseline value obtained which reverted to the actual normal baseline after the experimentation. Therefore, using the values obtained 45 minutes post-smoking as the probable true baseline value, it can be inferred that there was indeed a significant increase in cfASI due to smoking. This finding is similar to that of Prabha [14 who reported a significant increase in ASI in smokers of more than double that noted in non-smokers. The increase in ASI values noted after smoking are indicative of an increased large artery stiffness [35,13].

5. CONCLUSION

The present study demonstrates that smoking may cause an acute increase in arterial stiffness in smokers. Both cfPWV and cfASI increased acutely in the black male late adolescents following smoking 2 cigarettes. The increased effect of cfPWV and cfASI lasted for 15 minutes in the elastic arteries. Smoking may therefore be a risk factor for exacerbation of high blood pressure and cerebro-vascular pathology. This group of individuals, highly targeted by tobacco companies, is therefore at an increased CVD risk, including hypertension, myocardial infarction, and total mortality, as well as stroke, dementia, and renal disease in later life [36].

In order to reduce the risk of CVD related to increase in these parameters, awareness among this age group should be considered as a primary goal for both health practitioners and policy makers. As the methods used in this study are simple and reproducible, parameters such as cfPWV may be a useful tool to assess the effect of smoking and also for screening the risk of CVD. Considering the effects of certain confounders that might alter the blood nicotine concentration, like relation to puff volume and depth of inhalation, rate of puffing and type, a larger sample-sized study would increase the power of analysis giving more insight into the acute non-physiologic haemodynamic changes of smoking. There is need for further evaluation of this phenomenon which is an important finding for advocacy for smoke free environments.

ETHICAL APPROVAL

All authors hereby declare that the protocol was approved by University of Zambia Biomedical Research Ethics Committee (UNZABREC) and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

ACKNOWLEDGEMENT

This project was supported by grant # 5R24TW008873 administrated by the Fogarty International Center of the National Institutes of Health and funded by OGAC and OAR.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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