The Royal Society of Medicine

Journal of the Royal Society of Medicine Open;
5(6) 1–7
DOI: 10.1177/2054270414531127

Measuring the acute cardiovascular effects of shisha smoking: a cross-sectional study

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Summary

Objectives: To investigate the acute cardiovascular effects of smoking shisha.

Design: A cross-sectional study was carried out in six shisha cafes. Participants smoked shisha for a period between 45 min (minimum) and 90 min (maximum). The same brand of tobacco and coal was used.

Setting: London, UK.

Participants: Participants were those who had ordered a shisha to smoke and consented to have their blood pressure, heart rate and carbon monoxide levels measured. Excluded subjects were those who had smoked shisha in the previous 24 h, who smoke cigarettes or who suffered from cardiorespiratory problems.

Main outcome measures: Blood pressure was measured using a sphygmomanometer. Pulse was measured by palpation of the radial artery. Carbon monoxide levels were obtained via a carbon monoxide monitor. These indices were measured before the participants began to smoke shisha and after they finished or when the maximum 90 min time period was reached.

Results: Mean arterial blood pressure increased from 96 mmHg to 108 mmHg ($p < 0.001$). Heart rate increased from 77 to 91 bpm ($p < 0.001$). Carbon monoxide increased from an average of 3 to 35 ppm ($p < 0.001$). A correlation analysis showed no relationship between carbon monoxide and the other indices measured.

Conclusion: The acute heart rate, blood pressure and carbon monoxide levels were seen to rise significantly after smoking shisha. The weak correlation between carbon monoxide levels and the other variables suggests that carbon monoxide levels had not contributed to their significant increase.

Keywords

shisha smoking, hookah smoking, cardiovascular, respiratory

Introduction

Shisha (also called 'sheesha', 'hookah' or 'hubble bubble') smoking has been a favoured pass time activity for millions of people in the Middle East and the Asian subcontinent. The device is easily available and its structure, which includes a base, a shaft and the head, makes it very easy to assemble and smoke.

Since the mid-1990s, it has gained popularity in many Western countries, including the United Kingdom and the United States. After the smoking ban was enforced in the UK in 2007, there has been a 210% increase in the number of shisha cafes, rising from 179 shisha cafes to 556 in 2012.[1] In areas where there is a high density of immigrant Middle Eastern or South Asian population, specific 'shisha streets' have developed, where almost all shops will serve shisha to customers, often for a very affordable price. Such examples are Edgware Road in London and Wilmslow Road in Manchester.

Unfortunately, the potential health effects of shisha are not understood as well as cigarette smoking by both users and in the scientific literature. There is a common misconception that passing the smoke through water removes it of all harmful substances; a concept that entices the younger population to take up smoking shisha, especially at a time where there is an ongoing campaign to stop cigarette smoking.

Numerous studies have been conducted in the Middle East assessing the contents within shisha smoke compared to cigarette smoke. A study published in 2006 compared the cotinine levels of shisha smokers and cigarette smokers. They found that the mean cotinine levels in cigarette smokers (1487.3 ng/mL) were significantly higher than in shisha smokers (440.5 ng/mL), although the study suggested that chronic respiratory symptoms seemed to develop quicker in shisha smokers than cigarette smokers.[2] Other studies have shown that the amount of tar inhaled in a single shisha smoking session (ranges between 20 and 40 min) is significantly higher (242 mg) compared to a single cigarette (between 1 and 27 mg).[3] In addition, various research has reported the production of polycyclic aromatic hydrocarbons in shisha smoking, including one study which measured phenanthrene (0.748 µg), fluoranthene (0.221 µg) and chrysene (0.112), all of which have a wide range of potential health complications, including mouth and lung cancers, liver

derangements and nephropathies.[4] Heavy metals such as lead have also been reported in shisha smoke, levels of which are higher (7.92 μg/dL) than cigarette smoking (2.47 μg/dL).[5]

Research has primarily been based on the contents of shisha smoking and respiratory implications of shisha. However, to the best of our knowledge no study has reviewed the acute cardiovascular effects of shisha smoking. This study therefore aims to assess the effects that shisha smoking has on blood pressure, heart rate and carbon monoxide levels acutely. These measurements have been recognised in earlier studies to establish the acute effects on the cardiovascular and respiratory system in cigarette smokers.[6]

Despite the continued research into both the short- and long-term effects of smoking, people continue to smoke.[7] The effect on heart rate after smoking cigarettes is well documented in the literature, where a significant increase in the heart rate occurs and can require more than 80 min to return to normal.[8,9] Another measure which has been investigated is heart rate variability, which is calculated based on the difference between two heart beats, as a measure of the autonomic control of heart rate.[7] It has been shown that heart rate variability is significantly reduced, acutely and chronically, after smoking cigarettes.[7] There have been two mechanisms proposed to explain this either based on the action of nicotine to increase catecholamine release or action of suspended particles produced by incomplete combustion of cigarettes.[7] This reduction is correlated epidemiologically with cardiovascular diseases such as coronary heart disease.[7,10,11] Blood pressure is another important variable when measuring the acute effect of smoking on the cardiovascular system. Systolic and diastolic blood pressures have been shown to significantly increase acutely as well as chronically after smoking cigarettes.[9,12] Finally, carbon monoxide levels after smoking cigarettes are significantly higher, as determined by various measurements, such as the amount in exhaled air.[13] Significantly elevated levels of carbon monoxide in the body can lead to many detrimental effects such as nausea, vomiting, confusion, shortness of breath and memory loss.

Objectives

To investigate the acute effects that shisha smoking has on blood pressure, heart rate and carbon monoxide levels.

Methods

A cross-sectional study was carried out between the months of August and December 2012 in six London shisha cafes. Observers visited these cafés for 2 h between 8 pm to 10 pm, on a Saturday. Café managers were approached before observers engaged customers, to obtain verbal consent to measure indices on customers. Written consent was then obtained from the participants. Participants smoked shisha for a period between 45 and 90 min, which is routinely defined by the shisha cafes. The length of smoking was determined by the participant independent of the study. If a participant left prior to the defined 45- to 90-minute period, observers measured the required indices at that point. If the participants reached a total of 90 min of smoking, measurements were obtained.

All cafes used the same brand of tobacco and coal when serving participants. Blood pressure was measured using a manual sphygmomanometer. Pulse was measured manually by palpation over 30 s. Carbon monoxide levels were obtained via a portable carbon monoxide monitor.

This study included participants who came into the shisha café independently in the designated time frame. The observers excluded customers who were cigarette smokers, non-shisha tobacco consumers, individuals with past or present respiratory and cardiovascular problems and customers who had been at the café before the observers arrived (see Figure 1). This was to ensure that the magnitude of any changes seen were due to shisha alone and not due to any underlying illness or recent exposure.

Data analysis of the changes observed in the study was performed using a paired *t*-test using Prism software. The results were then verified manually by calculation of a random selection of results. A Pearson correlation analysis using Prism software was also performed to test for any relationship that may exist between the changes in carbon monoxide levels and levels of the other variables measured.

Results

Eighty-five potential participants were approached after they had ordered a shisha in the designated time frame. Nine of these did not consent to have their variables measured and used in the study, while 15 were excluded by the defined criteria set out initially. A total of 61 shisha smokers took part in this study, 49 males and 12 females. All subjects were between the ages of 18–25. Twenty-three of the 61 subjects were of Asian-Arabian origin, 19 of Asian-Pakistani origin, 12 of Asian-Indian origin, four of Black-African origin and three of White-British origin.

The results of average blood pressure, mean arterial pressure, heart rate and carbon monoxide with

Figure 1. Flowchart of study participants.

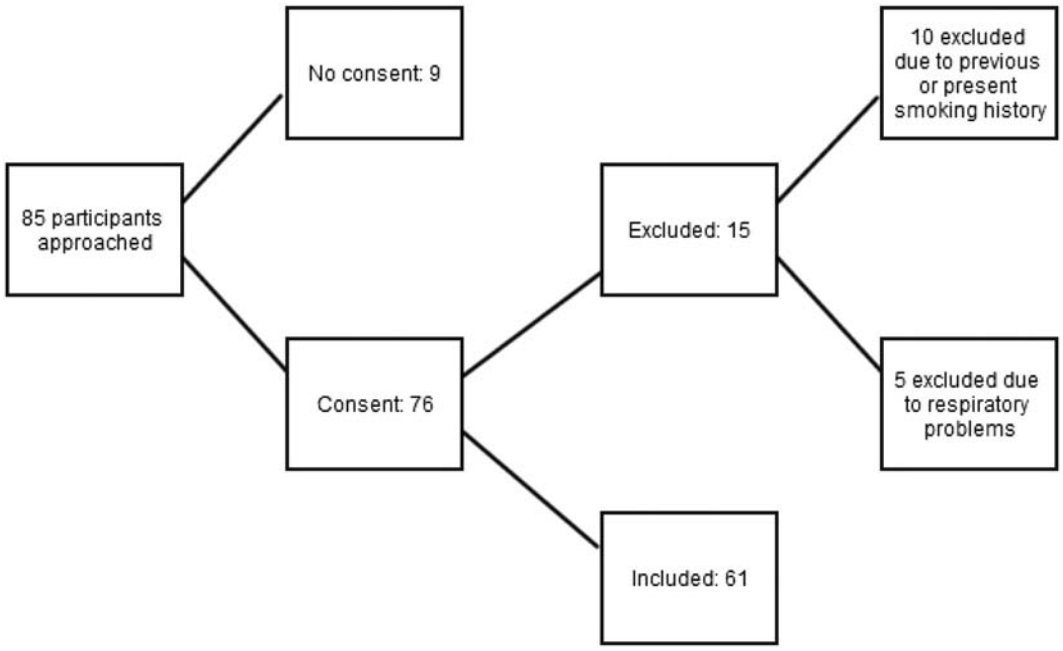


Table 1. Changes in indices before and after shisha smoking.

	Mean value before smoking shisha (standard deviation)	Mean value after smoking shisha (standard deviation)	Mean change (95% confidence interval)
Systolic blood pressure (mmHg)	129 (12)	144 (16)	15 (11–19)*
Diastolic blood pressure (mmHg)	80 (8)	90 (14)	10 (6–12)*
Mean arterial blood pressure (mmHg)	96 (8)	108 (13)	12 (8–14)*
Heart rate (bpm)	77 (8)	91 (11)	14 (12–16)*
Carbon monoxide (ppm)	3 (2)	35 (17)	32 (27–36)*

*$p < 0.0001$.

standard deviations are shown in Table 1. There was a statistically significant difference between indices measured before and after shisha exposure in heart rate, mean arterial pressure and carbon monoxide (Table 1).

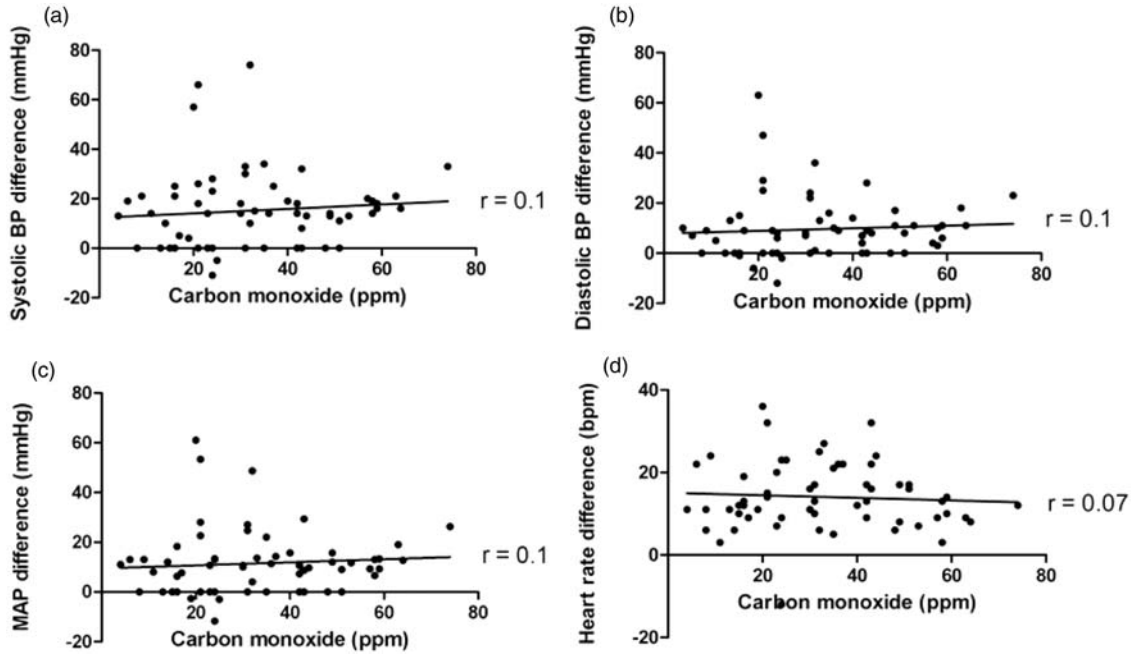
A correlation analysis was then performed in order to test whether the significant increase in carbon monoxide contributed to the significant rise of blood pressure and heart rate. No real correlation was found between the levels of carbon monoxide and the other indices measured (Figure 2).

Discussion

The principle findings of this study were that shisha smoking led to a statistically significant acute increase in heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and carbon monoxide levels.

The mean blood pressure significantly rose from 129/81 mmHg to 144/90 mmHg showing an overall increase of 15/9 mmHg. In addition, mean arterial blood pressures significantly increased from an average of 97 mmHg to 108 mmHg after the period of

Figure 2. A correlation analysis was conducted between carbon monoxide levels and systolic blood pressure (a), diastolic blood pressure (b), mean arterial pressure (c) and heart rate (d). The Pearson correlation coefficient (*r*) is displayed.



shisha smoking. These results suggest that shisha smoking acutely alters the cardiovascular physiology to elevate blood pressure. Acute mean arterial blood pressure changes in subjects who smoke shisha regularly could predispose to the long-term development of hypertension. Further epidemiological studies are required to address this.

The results demonstrate a statistically significant acute increase in heart rate with an average change of 14 bpm. This is similar to many stimulants that induce tachycardia such as illicit drugs, nicotine and caffeine. The increase in heart rate observed over a short period suggests that shisha has instantaneous effect on the cardiovascular system.

Carbon monoxide values were significantly higher after smoking shisha with an average level of 36 ppm. These levels are higher than previously documented by studies regarding cigarette smoking.[14] Such high figures of carbon monoxide might explain why many subjects feel 'light headed' whilst smoking shisha. The affinity between haemoglobin and carbon monoxide is approximately 230 times stronger than the affinity between haemoglobin and oxygen. Therefore, haemoglobin binds to carbon monoxide in preference to oxygen, starving organs of oxygen, leading to an increased risk of ischaemic events. A weak correlation was seen between the rise in carbon monoxide, heart rate and blood pressure. This correlation did not reach statistical significance. This indicates that the cardiovascular changes seen in this study are unlikely to be driven by the increase in carbon monoxide. Alternatively, the various chemicals found within the shisha tobacco and coal may contribute to these observations. This trend has been documented in a previous study investigating the cardiovascular effects of cigarette smoking and carbon monoxide.[15]

The changes observed in heart rate and blood pressure followed a similar trend to those previously published in relation to cigarette smoking. Carbon monoxide changes, however, were comparably higher in shisha smokers in this study (Table 2). A study investigating the effects of cigarette smoking on sympathetic outflow revealed that both systolic and diastolic peripheral pressure increased by approximately 12 and 4 mmHg, respectively, and the heart rate rose by 23 bpm.[16] Cigarette usage has previously been documented to cause an increase in carbon monoxide level of 10 ppm whereas shisha smokers in our study showed a far greater change of 32 ppm, a finding that is similar to previous observational studies.[16] This discrepancy seen in carbon monoxide levels is likely to be due to the differences in time spent smoking and the amount of tobacco used.

This study has shown similarities between cigarette and shisha smoking with regard to changes in acute cardiovascular indices. This could suggest that the physiological changes that occur after smoking shisha are similar to the changes that occur after

Table 2. A comparison of the changes in heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and carbon monoxide levels after smoking shisha and cigarettes.[16]

	Mean heart rate change (bpm)	Mean systolic change (mmHg)	Mean diastolic change (mmHg)	MAP change (mmHg)	Mean carbon monoxide change (ppm)
Shisha	14*	15*	10*	12*	32*
Cigarettes	23*	12*	4*	7*	10*

*$p < 0.05$.
Smoking shisha shows a larger increase in all the indices observed, except for heart rate.
MAP: mean arterial pressure.

the inhalation of cigarette smoke. The elevated heart rate seen after smoking cigarettes is a marker for increased mortality from coronary artery disease and hypertension as shown by the progressive increase of cardiovascular mortality with heart rate.[13,17] Therefore, the elevation in heart rate witnessed after smoking shisha may also act as a marker of cardiovascular morbidity and mortality, in the same way as a cigarette-induced elevation. In addition, systolic, diastolic and mean arterial blood pressures are all significantly elevated in both cigarette smoking according to the literature and in shisha smoking.[18] Elevated blood pressure increases myocardial demand, leads to damage of the endothelium by shearing forces and endothelial dysfunction within blood vessels.[19,20] This accelerates atherosclerosis and development can lead to cardiovascular events.[19,20] In addition, the elevated levels of carbon monoxide after smoking shisha may result in carbon monoxide intoxication resulting in symptoms such as syncope, headache and shortness of breath.[21]

Other pathological changes known to occur by cigarette smoking include impaired vasodilation, enhanced thrombosis and oxidation of cholesterol in light density lipoprotein and it is possible that these may also be present after smoking shisha.[20] The long-term consequences of smoking cigarettes such as the impact on atherosclerosis and myocardial infarction may also be the long-term consequence of exposure to shisha.[20] However, these concepts must be further investigated.

To the best of our knowledge, this is the first study to investigate the acute effect of smoking shisha on the cardiovascular system in a young adult population. Due to the novelty of this study, the observed results cannot currently be compared to other studies with respect to shisha smoking and were only compared to cigarette smoking. Further research must be performed to verify these results and investigate the wider effects of smoking shisha.

Potential limitations to our study that may have had an impact on the observed results should be noted. Sixty-one participants were studied due to a predetermined time frame for data collection. It was noted that data collection beyond this time frame may have resulted in a saturation of available participants with the observed presence of regular customers who were already recruited. Increasing the number of participants by including more shisha cafes may have increased the strength of the results and narrowed the confidence interval further. However, the results observed in this study were all statistically significant. Also, the resting heart rate of the participants was 77 bpm. This is higher than predicted for the age range of 18–25 which may reflect other factors such as anxiety of having heart rate measured. Furthermore, the carbon monoxide levels before smoking shisha varied between subjects, which could be attributed to the subjects waiting for their shisha to arrive. This may have led to some second-hand smoke inhalation caused by smoke emitted from the surrounding shishas.

This study was an observational study and no causation can be determined between the exposure of smoking shisha and subsequent disease. Further studies are required to investigate the contents of the shisha smoke as well as the acute and chronic effects of smoking shisha. The chronic effects of shisha smoking need to be thoroughly investigated to identify any similarities or differences with cigarette smoking, particularly in disease pathogenesis. The risks of shisha may be similar to smoking cigarettes but the use of coal to burn the tobacco in shisha is considerably different to that of cigarettes and therefore may have significantly different results.

The culture of shisha smoking in the United Kingdom is increasing at an exponential rate, with minimum public awareness of its health risks and a lack of scientific interest. Our results suggest an acute physiological impact on the cardiovascular system after smoking shisha for the first time in the scientific literature. As a consequence, this study should encourage the scientific community to explore the wider effects of shisha smoking. Physicians should

also be aware that shisha smoking may potentially impact a patient's long-term health as a risk factor for cardiovascular disease. Furthermore, if future studies observe chronic and detrimental effects on the cardiorespiratory system, the authors would advise improvements in public education and awareness of the health effects of shisha smoking. For example, this would include the mandatory labelling of shisha tobacco packets and apparatus with 'smoking kills', which to date is not enforced at shisha cafés.

Conclusion

This study demonstrated the presence of acute measurable cardiovascular effects after smoking shisha. Carbon monoxide, heart rate, diastolic blood pressure, systolic blood pressure and mean arterial pressure levels were significantly higher after smoking shisha. The weak correlation between carbon monoxide levels and the other cardiovascular indices suggests that carbon monoxide levels did not significantly contribute to the acute increase in blood pressure and heart rate observed. The direct cause of the observed changes in this study remains unknown and further research is required to establish this.

Declarations

Competing interests: None declared

Funding: None declared

Ethical approval: Ethical approval was obtained by St. George's Hospital Medical School.

Guarantor: BM

Contributorship: AH and AJ conceived the idea for the study. MK, AJ and AH contributed equally to write the first draft. BM and JB revised the manuscript.

Acknowledgements: We would like to thank the shisha cafe owners around London for allowing us to carry out the measurements in their premises.

Provenance: Not commissioned; peer-reviewed by Mahesh Rao.

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