Central and peripheral cardiovascular changes immediately after waterpipe smoking

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Abstract

Background: Tobacco cigarette smoking is a global health problem that kills millions each year. Recently, tobacco smoking using a waterpipe (WP) has become popular worldwide. However, unlike cigarettes, the cardiovascular (CV) risks associated with WP smoking are uncertain. In this study, the immediate effects of WP smoking on central and peripheral CV indices were evaluated in 53 young healthy smokers.

Materials and methods: Strain-gauge plethysmography was used to measure forearm blood flow (Bf), vascular resistance (Vr), and venous capacitance (Vc) and outflow (Vf) at rest (R) and after occlusion (Oc), whereas heart rate (HR) and blood pressure (BP) were measured using standard automated auscultatory methods immediately before and after a 30-min WP smoking session.

Results: Smoking resulted in HR, diastolic BP, mean arterial BP, rate pressure product and OcVr increases (p < 0.05) 6.6, 3.6, 2.5, 8.0 and 16%, respectively, whereas OcBf and OcVf decreased (p < 0.05) 8.8 and 14.3%, respectively. Additionally, smoking-induced changes in the central CV components correlated (p < 0.05) with changes in the periphery.

Conclusion: These results demonstrated changes in the CV central and peripheral components immediately after WP smoking. The correlations between the changes in these components suggest that the periphery is controlled, at least partially, by the same mechanism(s) affecting the central CV components during WP smoking.

Keywords: Blood flow, blood pressure, cardiovascular function, heart rate, immediate CV changes, waterpipe smoking

Introduction

Tobacco cigarette smoking increases cardiovascular (CV)-related morbidity and mortality. This increased risk has been attributed primarily to vascular dysfunction and subsequent atherosclerosis [Office of the Surgeon General (US), 2004; Powell, 1998]. Tobacco smoking using a waterpipe (WP) is a different form of tobacco use in which smoke passes through water before inhaled by the smoker (Maziak, 2008). Though WP use is most common in the Arab world (Maziak, 2008), it is spreading rapidly elsewhere including USA and Europe (Maziak, 2008). The spread is observed mainly in youth (Azab, 2010; Dar-Odeh, 2011; Mirahmadizadeh & Nakhaee, 2008; Ward, 2006) and is driven in part by underestimation of WP smoke health risks and limited WP-specific tobacco control regulations (Cobb et al., 2010; Maziak, 2008, 2011).

Many of the CV changes that occur immediately after cigarette smoking are known (Barutcu et al., 2005; Failla et al., 1997; Giannattasio et al., 1994; Karatzi, 2007a,b; Kool, 1993; Lemogoum, 2006; Mahmud & Feely, 2003; Narkiewicz et al., 1998; Niedermaier et al., 1993; Nuttall et al., 2002; Powell, 1998; Tsuchiya et al., 2002; Winniford et al., 1986). However, the CV effects of tobacco smoking using WP are uncertain, with only a few studies evaluating CV function immediately before and after WP tobacco smoking. These studies reported central CV (i.e. cardiac function) changes, including increases in heart rate (HR) and blood pressure (BP) (Al-Kubati et al., 2006; Blank et al., 2011; Eissenberg & Shihadeh, 2009; Hakim et al., 2011; Shaikh et al., 2008). We are aware of no studies that have examined the immediate effects of WP smoking on peripheral CV components (i.e. the vasculature). Therefore, the current study examined cardiac and vascular function indices before and after a single WP smoking session. Given previous observations after cigarette smoking (Barutcu et al., 2005; Karatzi et al., 2007a,b; Kool et al., 1993; Mahmud & Feely, 2003; Narkiewicz et al., 1998; Niedermaier et al., 1993; Nuttall et al., 2002; Tsuchiya et al., 2002; Winniford et al., 1986), increase in HR and BP are expected to be coupled with diminished vascular function including increased vascular resistance (Vr) and decreased blood flow (Bf), and venous capacitance (Vc) and outflow (Vf). The results should further clarify the effects of WP smoking, especially on the CV health.

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Materials and methods

Participants and design

Individuals between 18 and 35 years old who smoked WP at least three times a week during the past year were recruited in the study. Volunteers with acute medical conditions, CV, kidney, or metabolic diseases, or who were using medications that might affect the CV system were excluded. After a comprehensive review of the study procedures, each participant signed an informed consent form approved by the Institutional Review Board of Jordan University of Science and Technology. Subsequently, the hemodynamics of the CV (i.e. cardiac and vessels) system were measured immediately before and after a WP smoking session.

CV hemodynamics measurements

Central components

Indices of blood pressure (BP) including diastolic (DBP) and systolic (SBP) were obtained using standard automated auscultatory method (Omron HEM-907XL; Omron Healthcare, Inc., Bannockburn, IL) from supine position at rest, and after 4 and 5 min of arterial occlusion. Mean arterial (MAP) and pulse (PP) pressures as well as rate pressure product (RPP) were then calculated (Alomari et al., 2004).

Peripheral components

A strain-gauge plethysmography (model: EC5R, D.E. Hokanson, Inc., Bellevue, WA) was used to measure vascular indices including Bf at rest (RBf) and after 5 min of arterial occlusion (OcBf) in the dominant forearm. Additionally, resting Vc (RVc) and Vf (RVf) as well as occlusion Vc (OcVc) and Vf (OcVf) were also obtained from the same arm. Subsequently, Vr at rest (RVr) and after occlusion (OcVr) were calculated using MAP/Bf. The measurements were obtained with a strain-gauge wrapped around the forearm ~10 cm below the olecranon process. Two pressure cuffs were placed, above the elbow and at the wrist, to manipulate arterial and venous flows.

Forearm RBf was measured instantly after increasing a venous pressure of 7 mmHg above DBP from the upper cuff while inflating the wrist cuff to 240 mmHg 1 min before the measurement. Forearm RVc was measured after maintaining the upper arm venous occlusion for 6 min, whereas RVf was measured after releasing the venous pressure from the upper cuff. Five minutes later, the upper cuff was inflated for 5 min; subsequently, OcBf was measured as in RBf. Similarly, OcVc and OcVf were measured as described in the resting condition.

Forearm RBf and OcBf were recorded on a graph paper with a speed of 5 and 25 m/s, respectively. The slope drawn at first 2–3 beats of the volume graph was used to calculate Bfs. Measurement of RVc and OcVc were

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Table 1. Subject characteristics (n=53).

|                          | Age (years) | Weight (kg) | Height (cm) | BMI  | Body fat (%) |
|--------------------------|------------|-------------|-------------|------|--------------|
|                          | 22.7 ± 4.8 | 166.9 ± 9.5 | 56.2 ± 12.2 | 23.4 ± 3.6 | 23.9 ± 6.5 |

Table 2. Changes in the central components of the CV system.

|                  | HR          | SBP         | DBP         | MAP     | PP          | RPP          |
|------------------|-------------|-------------|-------------|---------|-------------|--------------|
| Before smoking   | 78.7 ± 13.8 | 111.2 ± 10.7| 65.7 ± 7.9 | 80.9 ± 7.6| 45.6 ± 10.0 | 8767.7 ± 1814.8|
| After smoking    | 83.9 ± 12.3 | 112.9 ± 9.9 | 68.1 ± 7.9 | 83.0 ± 6.9| 44.8 ± 11.0 | 9468.3 ± 1603.8|

Table 3. Changes in the peripheral components of the CV system.

|                  | Arterial     | Occlusion   | Venous capacitance | Venous outflow |
|------------------|--------------|-------------|--------------------|----------------|
| Blood flow       | -4.3 ± 1.8   | 21.1 ± 6.5  | 18.1 ± 8.7         | 5.6 ± 3.1      |
| Vascular resistance | 22.0 ± 7.5 | 16.5 ± 8.5  | -8.8               | 6.5 ± 3.8      |
| Percentage of differences | -2.3      | 4.2         | -8.8               | 16.0           |
| p Value          | 0.724        | 0.387       | 0.035              | 0.003          |
| Venous capacitance | 2.9 ± 1.0   | 21.4 ± 10.2 | 1.75 ± 0.86        | 24.4 ± 12.9    |
| Venous outflow   | 2.7 ± 1.0    | 19.9 ± 11.2 | 1.84 ± 0.9         | 20.9 ± 10.0    |
| Percentage of differences | -6.9     | -7.0        | -5.1               | -14.3          |
| p Value          | 0.149        | 0.220       | 0.539              | 0.012          |

Values are expressed in mean ± standard deviation. HR: heart rate; SBP (mmHg): systolic blood pressure; DBP (mmHg): diastolic blood pressure; MAP (mmHg): mean arterial pressure; PP (mmHg): puls pressure; RPP: rate pressure product.
calculated as the change in volume graph after 6 min of venous occlusion where as RVf and OcVf were recorded on a graph paper with a speed of 25 m/s and calculated as change in the volume graph after the release of venous occlusion (Alomari et al., 2004).

The WP smoking session

After completing the first CV measurement, a 30-min self-paced WP smoking session commenced in a well-vented and air-conditioned room. The tobacco (Two Red Apples; Nakhla, Egypt) was prepared for all the participants by the same investigator using the same amount (10 g), and WP apparatus and was heated with fast-let charcoal (Three Kings, Holland). The plastic mouthpiece and water content were replaced for each participant.

Statistics

All statistical analyses were performed using SPSS Statistics software for Windows (version 17.0; SPSS Inc., Chicago, IL). Group data are expressed as mean ± standard deviation, and α was set a priori at p < 0.05. A paired t-test was used to compare CV indices before and after WP smoking. The relationships between CV central and peripheral changes were examined with Pearson’s product correlations.
Results

Participants
As presented in Table 1, 53 WP young healthy smokers (19 women) agreed to participate in the study. The participants smoked WP regularly for at least 1 year. The average age, weight and height were 22.7 ± 4.8 years, 65.2 ± 12.2 kg and 166.9 ± 9.5 cm, respectively.

Immediate effects of smoking on CV hemodynamics
As in Table 2, the participant’s HR, DBP, MAP and RPP increased ($p<0.05$) 6.6, 3.6, 2.5 and 8.0%, respectively, after WP smoking. Additionally, Table 3 shows that WP smoking resulted in 8.8 and 14.3% decrease ($p<0.05$) in OcBf and OcVf, respectively, as well as 16% increase ($p<0.05$) in OcVr.
Relationships between changes in the central and peripheral components

Figure 1(A and B) shows correlations of changes in RBf with changes in resting HR and RPP. Additionally, Figure 2(A–C) shows changes in RVr correlations with changes in resting HR, DBP and MAP while changes in RVc correlations with changes in DBP and MAP are shown in Figure 3(A and B). Finally, Figure 4(A–C) shows correlations of changes in OcVc with changes in SBP, DBP and MAP.

Discussion

A single WP smoking session resulted in immediate changes in the central components of the CV system, confirming previous studies (Blank et al., 2011; Cobb et al., 2011; Eissenberg and Shihadeh, 2009; Hakim et al., 2011; Shaikh et al., 2008). Additionally, for the first time, this study revealed alterations in forearm vascular function. The central and peripheral changes, following WP smoking, were related suggesting mutually mediating mechanism(s) for these changes.

Average HR, SBP, DBP, MAP and RPP increased whereas PP decreased immediately following the WP smoking session. Previous work demonstrates that the central CV component response to WP tobacco smoking is mediated by changes in participants’ plasma nicotine concentration (Blank et al., 2011) and baroceptors function (Al-Kubati et al., 2006). Blank et al. (2011) reported an increase in BP and HR as well as plasma nicotine concentration. Additionally, changes following cigarette smoking indicated that the elevated nicotine mediates an increase of sympathetic nervous system activities and a release of epinephrine, norepinephrine (Cryer et al., 1976; Narkiewicz et al., 1998; Niedermaier et al., 1993) and vasopressin (Waeber et al., 1984) hormones. This sympathohormonal- excitatory response mediated the increase in the central components of the CV system. Others cited concurrent baroreceptor depression with the pressor and tachycardiac responses to WP smoking. The authors attributed these central changes to baroceptors-mediated sympathoexcitatory response (Al-Kubati et al., 2006). However, more studies are needed to clarify mechanism(s) for central changes following WP smoking.

The observed changes in the peripheral components of the CV system (i.e. Bf, Vr, Vc and Vf) immediately following WP smoking are consistent with the findings from cigarette smoking studies (Barutcu et al., 2005; Karatz et al., 2007a,b; Kool et al., 1993; Mahmud & Feely, 2003; Narkiewicz et al., 1998; Niedermaier et al., 1993; Nuttall et al., 2002; Tsuchiya et al., 2002; Winniford et al., 1986). Inhalation of tobacco cigarette smoke immediately increases arterial stiffness (Kim et al., 2005; Kubozono et al., 2011) and Vr (Klein et al., 1984; Winniford et al., 1986) subsequent to a decrease in plasma nitrate, nitrite and serum antioxidant (Tsuchiya et al., 2002). These changes were associated with accelerated HR, higher BP, and reduced coronary Bf and reserve, with possible coronary spasm in patients with coronary artery disease (Klein et al., 1984; Winniford et al., 1986). Local (i.e. endothelium) and systemic (i.e. autonomic) mechanisms were implicated in these vascular changes. Previous studies reported arterial rigidity (Kool et al., 1993; Mahmud & Feely, 2003) attributed to reduced nitric oxide bioavailability (Nuttall et al., 2002; Tsuchiya et al., 2002), endothelium dysfunction (Karatzi et al., 2007a) and compromised vasodilatory capacity (Karatzi et al., 2007b; Winniford et al., 1986) immediately after smoking a cigarette.
However, the systemic contribution to vascular changes should not be overlooked especially as the current study found relationships between the central and peripheral changes of the CV system. The changes in HR and BP associated with the changes in Bf, Vr and Vc suggesting that the periphery is controlled, at least partially, by the same mechanism(s) affecting the central CV components during WP smoking. This relationship might be expected because the heart and vessels are similarly connected to and controlled by the autonomic nervous system. The baroceptors/nicotine-mediated sympathohormonal-excitatory response noted during WP smoking (Al-Kubati et al., 2006; Blank et al., 2011; Cobb et al., 2012), might have also contributed to the modifications in the vasculature. This was confirmed as HR variability was reduced immediately after WP smoking suggesting changes in the autonomic nervous system balance (Cobb et al., 2012). However, these speculations need to be substantiated with appropriately controlled studies.

Regardless of the mechanism(s), these central and peripheral changes can certainly be harmful to the CV function and health (Ambrose & Barua, 2004; Jonas, 1992; Ockene, 1997). The increased Vr indicates diminished arterial vasodilatation which can induce multiple deleterious effects on the CV system. It can instantaneously increase BP and load on the
cardiac muscle. Vasoconstriction can be even more detrimental in small arteries or in vessels supplying vital organs, such as the coronary, carotid and pulmonary arteries (Powell, 1998).

As in Table 2, the increased RPP (product of HR and SBP) indicates increased cardiac metabolic taxing and exhaustion, which results in extra requirements for oxygen delivery during WP smoking. This increase is defied with a mismatched oxygen delivery resulting from coronary artery vasoconstriction, usually following nicotine administration. This mismatch between delivery and demand can affect left ventricular function (Lichodziejewska et al., 2007; Stork et al., 1992). In a recent study, smoking one cigarette resulted in impaired left and right ventricular filling (Lichodziejewska et al., 2007).

Figure 4. (A) Relationship between changes in post-occlusion Vc and SBP following with WP smoking. $r = 0.33; p = 0.015$. (B) Relationship between changes in post-occlusion Vc and DBP following WP smoking. $r = 0.45; p = 0.001$. (C) Relationship between changes in post-occlusion Vc and MAP following WP smoking. $r = 0.45; p = 0.001$. DOI: 10.3109/08958378.2014.936572
Additionally, the sympathoexcitatory response is an independent source of atherosclerosis and subsequent ischemic diseases (Al-Kubati et al., 2006; Blank et al., 2011). These central and peripheral changes are detrimental as found to be associated with increased risk for CV-related diseases, morbidity and possibly mortality (Ambrose & Barua, 2004; Jonas et al., 1992; Ockene & Miller, 1997). This increased risk is markedly profound as these changes were observed in relatively young individuals who may continue to smoke WP for years to come. Therefore, effective WP-specific smoking cessation strategies, including interventions focused on young people, are an important public health goal. Especially these efforts have been shown to decrease CV risks in cigarette smokers (Critchley & Capewell, 2003). Diet high in antioxidant has been shown to minimize oxidative stress in smokers (Haibach et al., 2013; Kelly, 2002). Furthermore, regular participation in physical activities is known to lower the risk of adverse consequences of smoking and improve various health aspects among smokers, let alone the importance of exercise for the treatment of smoking addiction and dependency (Bloomer & Fisher-Wellman, 2009; deRuiter & Faulkner, 2006; Korhonen et al., 2011; Roberts et al., 2012; Rooks et al., 2011; Ussher et al., 2012).

Conclusion

The study confirms central changes in the CV system immediately following WP smoking. Additionally, for the first time, WP smoking resulted in instantaneous changes of the peripheral circulation. Finally, WP-induced peripheral circulatory changes associated with changes in the CV central components. These changes are documented contributing factors to CV-related diseases, morbidity and mortality. Thus confirms the adverse health consequences of WP smoking, opposite to commonly believed (Cobb et al., 2010; Mazia, 2008, 2011). Additionally, the results provide further support for national and international programs designed to slow the global spread of this form of tobacco use.

Declaration of interest

Authors declare no conflicts of interest except Prof. Eissenberg reports grants from US National Institutes of Health during the conduct of the study. The authors alone are responsible for the content and writing of this article.

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