Levels of maximum end-expiratory carbon monoxide and certain cardiovascular parameters following hubble-bubble smoking

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ABSTRACT

Objective: The physiological effects of cigarette smoking have been widely studied, however, little is known regarding the effects of smoking hubble-bubble. We examined the acute effects of hubble-bubble smoking on heart rate, systolic, diastolic, and mean arterial blood pressure and maximum end-expiratory carbon monoxide.

Methods: This study was carried out in the student laboratory, School of Medicine, Department of Physiology, University of Jordan, Amman, Jordan, during the summer of 1999. In 18 healthy habitual hubble-bubble smokers, heart rate, blood pressure, and maximum end-expiratory carbon monoxide was measured before, during and post smoking of one hubble-bubble run (45 minutes).

Results: Compared to base line (time zero), at the end of smoking heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, and maximum end-expiratory carbon monoxide were increased 16 ± 2.4 beats per minute, 6.7 ± 2.5 mm Hg, 4.4 ± 1.6 mm Hg, 5.2 ± 1.7 mm Hg, and 14.2 ± 1.8 ppm, (mean ± standard error of mean, P<.05).

Conclusions: Acute short-term active hubble-bubble smoking elicits a modest increase in heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and maximum end-expiratory carbon monoxide in healthy hubble-bubble smokers.

Keywords: Hubble-bubble, heart rate, blood pressure, carbon monoxide.

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(HB); known under different names namely oriental pipe, water pipe, Arghileh, Narghileh, Narguila, Hookah, Sheesha, Chicha and Gozah for example is widely used in Jordan and in other countries. Briefly, in HB smoking, with each puff, mainstream smoke is drawn through the burning tobacco and passes into water via a stem, then enters the rubber tube and is finally delivered to the smoker mouth via a mouth piece for more detailed description see reference. This traditional type of smoking is traced back to ancient India. Nevertheless, in 1980 an article was written in one of the most famous Arab journals the author deplored the continuous shrinkage in HB smokers, and he predicted that within few years HB would be placed in Museums only. He urged that HB, as part of the Arab culture, should be resurrected again. Unfortunately, this urge became true as early as 1990s. Since then, HB smoking gained a great acceptance among all age groups in most Middle Eastern countries. In contrast to cigarette smoking, in HB smoking everyone can participate including women and teenagers. For instance, in Egypt HB may be smoked in groups. Teenagers by imitating adults, smoke HB to enter the wonderland!

Even though there are no epidemiological studies regarding its prevalence in Jordan, it is however, observed that HB popularity is increasing. In contrast to cigarette smoking, HB smoking became a normal social behavior and public toleration to this habit is becoming wider. People think of HB as an innocent habit and therefore, its consequent increase in Jordan is approaching epidemic proportions.

Reviewing the literature (from 1970-2001), did not reveal any studies relating HB smoking to HR, BP, and maximum EECO. In general, research in the hazards of HB smoking is very primitive and only a few papers have been published in this area. A few studies measured COHb concentrations in the blood of sheesha smokers while others measured CO fractions in hookah smoke. Hubble-bubble smoking can cause oral, esophageal, and lung cancer, abnormal pulmonary function tests and low-birth-weight infants born to HB smoking mothers. Recently, we have reported nicotine and cotinine levels in different biological fluids in HB smokers.

We report our observations of a number of cardiovascular parameters measured before, during, and following HB smoking. We were particularly interested in the acute effect of HB smoking on the above parameters. Obviously, this study aims to correlate HB smoking with possible health hazards.

**Methods.** Eighteen male volunteers participated in this study. This study was carried out in the student laboratory, School of Medicine, Department of Physiology, University of Jordan, Amman, Jordan, during the summer of 1999. Their ages varied from 20-45 years (27 ± 8, mean ± standard deviation), weight from 60 kgs to 110 kgs (80 ± 13), height from 165 cm to 188 cm (176 ± 6 cm), and body mass index (BMI) from 18.1 to 34.7 (25.7 ± 4 kg/m²). To overcome anxiety, the study was explained for all participants, and informed consent was obtained before participation. The study was approved by the University Ethics Committee. All volunteers fulfilled the following criteria before they were considered for the study. No history of recent acute illness, not taking any medication at the time of the study, no clinical evidence suggestive of cardio-pulmonary disease in particular hypertension, and no history of drug or alcohol dependence. Sitting systolic blood pressure (SBP) measured by a mercury sphygmomanometer was <140 mm Hg and the diastolic blood pressure (DBP) was <90 mm Hg at zero time (before smoking). Volunteers were habitual HB smokers for 3.8 years ± 2.3 years (mean ± SD), range one year - 10 years who smoked at least 3 runs per week with an average of 20 g Mu'asal per run. Mu’asal is made from tobacco leaves with additives to give different flavours such as honey, mint, fruits ± glycerin and so forth. All volunteers were considered primary HB smokers (had not previously smoked any other type of tobacco such as cigarette, cigar, pipe for example.)

**Blood pressure measurement.** Blood pressure (BP) measurements were made as precise as possible using a simple mercury sphygmomanometer. Volunteers were asked not to practice strenuous exercise during the study day. They were seated for 5 minutes and then base line measurements were taken. Consequently, volunteers remained seated throughout the smoking period and the observer was in a comfortable position in relation to the volunteer.

In brief, the cuff was placed on the volunteer’s right arm and was inflated to 30 mm Hg above the pulse obliteration pressure. The sphygmomanometer was placed on a horizontal surface with its mercury column being in an upright position. The systolic pressure was determined by the first perception of sound, and the diastolic pressure was determined by the perception of disappearance of sound. The cuff then deflated rapidly to zero pressure. Successive measurements were conducted exactly the same way that the first one was carried out. Volunteers were not informed regarding the blood pressure values at any point during the study.

**Maximum end-expiratory carbon monoxide measurement.** Maximum EECO level in parts per million (ppm) was measured by a trained person using Bedfont EC50-MICRO CO monitor smokerlyser (Bedfont Instruments; Kent, United Kingdom). This portable CO monitor has been shown to be accurate, and its mechanism of action was described elsewhere. It is quick and easy to use, cheap, and noninvasive. Basically, on breath holding, the blood CO equilibrates with the alveolar...
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CO. Therefore, maximum EECO levels following breath-holding correlate closely with COHb concentrations. Before maximum EECO measurements, volunteers were instructed to inhale and exhale deeply several times. Next, volunteers were instructed to expire to residual volume, take a vital capacity inspiration, breath-hold for 15 seconds and then exhale fully. Volunteers breathed out steadily and gently through the smokerlyser and then exhale fully. Volunteers breathed out as much of lung contents as possible gives the best sample of alveolar air. All volunteers were able to hold their breath for 15 seconds.

Procedure. Volunteers reported to the lab between 9:00 am and 10:00 am after having light breakfast. One-to-two volunteers were taken at a time and placed in the student laboratory, which was adequately ventilated and had dimensions of (10m x 7m x 4m). This large room was quiet and its temperature was comfortable. Volunteers were instructed to expire to residual volume, take a vital capacity inspiration, breath-hold for 15 seconds and then exhale deeply several times. Next, volunteers were instructed to inhale as much of lung contents as possible. Exhalation of as much of lung contents as possible gives the best sample of alveolar air. All volunteers were able to hold their breath for 15 seconds.

Physiological measures (HR, SBP, DBP, and maximum EECO) were taken before, during, and after HB smoking. Heart rate increased over the 45 minutes smoking period and declined after the end of smoking. Compared to pre-smoking values, HR was significantly increased 5 minutes following smoking (91.8 ± 2.5 versus 79.1 ± 2.7 bpm, P<0.001). At the end of the 45 minutes smoking period, HR reached its peak value 95 ± 2.4 bpm with a maximal change of +20%. At 25 minutes following the end of smoking period, HR fell to values significantly lower than the peak value (89.1 ± 2.3 bpm, P<0.001), but remained statistically higher than the base line values. At 60 minutes following the completion of smoking, HR remained higher than the base line (83.3 ± 2.4 bpm, P =0.14) (Figure 1 and Table 1).

Table 1 - Cardiovascular responses to smoking bubble bubble for 45 minutes (Data are mean ± standard error of mean).

<table>
<thead>
<tr>
<th>Variable</th>
<th>5 minutes</th>
<th>Difference (*) with respect to base line (time zero minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25 minutes</td>
<td>45 minutes</td>
</tr>
<tr>
<td></td>
<td>P-25 minutes</td>
<td>P-60 minutes</td>
</tr>
<tr>
<td>Heart rate</td>
<td>12.0 ± 2.4 16</td>
<td>14.1 ± 1.9 18</td>
</tr>
<tr>
<td>Systolic Blood pressure</td>
<td>6.9 ± 1.9 6</td>
<td>7.5 ± 2.4 6</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>4.2 ± 1.2 5</td>
<td>6.1 ± 1.2 8</td>
</tr>
<tr>
<td>Mean arterial blood pressure</td>
<td>5.1 ± 1.3 6</td>
<td>6.6 ± 1.4 7</td>
</tr>
</tbody>
</table>

* - changes from baseline for heart rate, systolic, diastolic and mean arterial blood pressure, numbers listed on the right hand side of the columns are percent change with respect to baseline values, P-25 and P-60 are values at 25 and 60 minutes following the end of smoking.

Results. The effect of smoking HB on different cardiovascular parameters was studied. Physiological measures (HR, SBP, DBP, and maximum EECO) were taken before, during, and after HB smoking. In published studies, some individuals needed longer time for cotinine to reach that value. Unfortunately, it was not feasible to ask our volunteers to stop smoking for longer period of time. In addition, maximum EECO level of 6 ppm was taken as the cut off between compliant and noncompliant.

After baseline measurements were taken (HR, SBP, DBP, and maximum EECO), volunteers were signaled readiness to start smoking. Next, volunteers started smoking 20 gm of HB Mu’asdl from the local market, the quantity approximately equal to that commonly used. Large-size Arghileh was used (for full description of the smoking instrument “Arghileh” see Reference 12). The single HB run usually lasts for 30-60 minutes. Therefore, we asked volunteers to smoke for 45 minutes. They smoked according to their own regular habit.

Statistics. Data is presented as mean ± SEM in the text and the figures unless indicated otherwise. Comparisons between data from presmoking, smoking, and postsmoking were performed with the paired Student’s t test and a P value of <0.05 was considered statistically significant.
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Figure 2 - Effects of 45 minutes smoking hubble-bubble on systolic, diastolic and mean arterial blood pressure, as a function of time. (Mean ± standard error of mean). *significant difference (P<0.05) from pre-smoking value, +significant difference (P<0.05) from end of smoking.

Table 2 - Correlation coefficient between nicotine and cardiovascular parameters.

<table>
<thead>
<tr>
<th>Variable</th>
<th>r</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>0.77</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>0.52</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>0.57</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean arterial blood pressure</td>
<td>0.56</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

The time course of the changes in BP are shown in Figure 2. During smoking, SBP was significantly higher than the presmoking value and it was obvious as early as 5 minutes following smoking (126.7 ± 3.1 versus 119.7 ± 2.3 mm Hg, P<0.005). It remained significantly elevated throughout the smoking period with a maximal change of +6%. At 25 minutes following the end of smoking, it had returned to its baseline level (120.8 ± 2.4 mm Hg).

Diastolic blood pressure in general, showed an almost similar pattern to that of SBP. Compared to baseline values, DBP rose significantly 5 min following smoking (83.1 ± 2.0 versus. 78.9 ± 2.0 mm Hg, P < 0.005). At 25 minutes following the end of smoking, DBP declined to values higher than the base line value 80.8 ± 2.2 mm Hg, but this was not significant. MBP rose from 92.5 ± 2.0 at baseline to 97.6 ± 2.2 mm Hg (P<0.001) after 5 minutes smoking and remained elevated until the end of smoking. Pre, during and post smoking mean arterial blood pressure values are shown in Figure 2. The BP increase was no longer present at 60 minutes following the end of smoking. This was true, despite maintenance of elevated plasma nicotine concentrations (data not shown).

The smoking induced increase in maximum EECO is shown in Figure 3. None of the volunteers had a pre-smoking maximum EECO greater than 6 ppm.
which was considered a cut off point for compliance. A maximum EECO at time zero ranged from 0 to 5 ppm. The maximum EECO level rose from 2.0 (± 0.34 ppm) at baseline to 6.8 (± 1.0) ppm 5 minutes following smoking (P<0.001). Maximum EECO levels rose steeply during smoking and reached a peak just after the last puff (16.2 ± 1.8 ppm) when its level varied between 10-39 ppm. Following the completion of smoking, maximum EECO declined significantly. At 25 minutes and 60 minutes following the end of smoking, maximum EECO values declined but remained significantly higher than those of the baseline values (14.9 ± 1.8 and 14.4 ± 1.6 ppm). Even after 24 hour, maximum EECO levels remained significantly higher than the baseline value (3.6 ± 0.5; P=0.034, data not shown). In contrast to BP and HR, maximum EECO remained significantly elevated throughout the 60 minutes follow-up.

Finally, a significant correlation was found between plasma nicotine and the above variables (Table 2).

Discussion. Unfortunately, HB smoking is almost entirely restricted to the Middle Eastern countries, which explains the primitive level of research in this area. Nevertheless, for the last twenty years data is accumulating regarding the health damage induced by HB smoking.

Hubble-bubble prevalence is increasing, especially among women, but public awareness of its risk factors is virtually non-existent or still premature. Health officials are not sounding any alarm and very little, if any, effort is being made to reduce HB smoking. It is worth mentioning that health statistics already indicate that among women lung cancer is more prevalent than breast cancer, reflecting the increase in female smoking rates.29,30 In general, there is misconception that HB smoking is a safe alternative to cigarette smoking. Since quitting smoking is a major goal of the majority of cigarette smokers, shifting to a less harmful smoking pattern is a more achievable goal. Surprisingly, HB smokers are willing to believe that as smoke goes through water, many of the harmful substances are filtered out. However, there is no evidence to support such assumption; in fact, less than 5% of the nicotine content is trapped in the water of the Arghileh.31 When considering many HB smokers smoke cigarettes too, it makes the risks even worse. It is therefore, important to investigate the health hazards HB smoking can induce. The HB smoking-induced cardiovascular effects seen here are in broad agreement with those observed following cigarette smoking32-33 or nicotine infusion.32,33 However, the maximal effects following HB smoking were less when compared to those following cigarette smoking.32 Such results are not unexpected. Usually, HB smokers do not inhale all the smoke puffed; some of the smoke is expelled from the oral cavity before inhalation. In addition, our volunteers were primary HB smokers (primary HB smokers do not inhale deeply). Therefore, comparing to cigarette smokers, maximum EECO levels in HB smokers were found to be lower. In agreement with our findings, light cigarette smokers maintain lower CO levels.35 Primary cigar and pipe smokers also maintain lower CO levels when compared to secondary smokers.36 It is worth comparing maximum EECO between primary and secondary HB smokers (those who had previously been regular cigarette smokers "switchers"). All the cardiovascular changes due to smoking are attributed to nicotine through stimulation of the sympathetic nervous system.37 Actually; we found a strong correlation between plasma nicotine levels and the above parameters (Table 2).

Limitations of this study include the intervals at which maximum EECO was measured. It has been shown that when maximum EECO is measured within the first 5 minutes after smoking, it may not be good indicator of smoke inhalation.32,33 However, in our study, we could not interrupt smoking period, since it would not resemble the normal smoking pattern.

In conclusion, the results of this study clearly indicate that smoking HB resulted in statistically significant increase in HR, BP, and maximum EECO levels. Those results compromise the concept of HB being a safe alternative to cigarette smoking.

Finally, much work remains to be carried out to adequately define the hazardous effects of HB smoking. Meanwhile, a massive educational campaign needs to be launched, and governments should start implementing restrictions on HB smoking.

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References