# Immediate effects of waterpipe smoking on control of breathing among young smokers

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## Background

Waterpipe smoking rates differ worldwide (from 8% in the UK to 33% in Pakistan)[1]. Often it is considered to be less harmful than cigarette smoking, it is of relatively low cost, and also, frequently involves a state of social interaction[2].

Cigarette smoke can have an acute impact on hypoxic chemosensitivity[3], while an increase in respiratory rate has been detected by researchers in controlled environments immediately after water-pipe smoking[4]. However, the immediate effects of waterpipe on respiratory neuromuscular response and respiratory timing have not been minutely investigated yet. Furthermore, smokers include people with and without respiratory symptoms and thus, all smokers cannot be studied as one homogenous group. This issue has been addressed by other studies comparing lung function among young subjects with and without mild respiratory symptoms[5,6], but it has never been applied in studies investigating the immediate response of the respiratory system after smoking.

The purpose of our study was to detect the immediate effects of waterpipe smoking on control of breathing, but also to investigate the possibility of a different acute response of the respiratory system between young smokers with and without mild respiratory symptoms.

## Design/Methods

50 young smokers (average age=23±4, 32 males, mean pack-years=3.6; average consumption=7 cigarettes/day) were recruited from a community setting in Athens, Greece, and voluntarily participated in this study. 25 reported a combination of mild respiratory symptoms (allergic rhinitis, mild cough and mild chest tightness) which were rare and sporadic, elicited by exposure to certain substances, exercise or following respiratory infections (MRS-subgroup). The other 25 had no respiratory symptoms (NRS-subgroup). All subjects were free of any acute/chronic disease (even a common cold during the previous 2 weeks) or current use of any medication.

***Table 1: Demographics and baseline spirometric data of total, mild respiratory symptoms (MRS) and non respiratory symptoms (NRS) subgroup participants***

| Characteristic | All 50 individuals | MRS subgroup (N=25) | NRS subgroup (N=25) | p-value |
|---|---|---|---|---|
| Males/Females | 32/18 | 15/10 | 17/8 | 0.554 |
| Age (years) | 23.4 ± 4.2 | 23.6 ± 3.9 | 23.2 ± 4.5 | 0.525 |
| BMI (kg/cm$^2$) | 23.4 ± 3.2 | 23.9 ± 2.9 | 23 ± 3.4 | 0.404 |
| Pack-years | 3.6 ± 2.8 | 3.6 ± 2.6 | 3.6 ± 2.9 | 0.747 |
| FVC (% pred.) | 101.9 ± 12 | 99.3 ± 10.6 | 105.5 ± 10.8 | 0.112 |
| FEV1 (% pred.) | 101.2 ± 12.4 | 93 ± 9.4 | 109.5 ± 9.2 | <0.0001* |
| FEV1/FVC (% pred.) | 84.5 ± 6.9 | 80.1 ± 5.6 | 88.8 ± 5.3 | <0.0001* |
| PEF (% pred.) | 96 ± 12.88 | 93.19 ± 12.34 | 99 ± 13.09 | 0.114 |
| $FEF_{25\%-75\%}$ (% pred.) | 93.4 ± 26.9 | 74.2 ± 13.9 | 112.7 ± 22.3 | <0.0001* |
| $FEF_{25\%}$ (% pred.) | 98 ± 21.25 | 88.43 ± 16.59 | 111 ± 16.61 | <0.0001* |
| $FEF_{50\%}$ (% pred.) | 95 ± 24.96 | 78.6 ± 14.63 | 114 ± 19.93 | <0.0001* |
| $FEF_{75\%}$ (% pred.) | 94 ± 38.43 | 72.79 ± 20 | 118 ± 39.6 | <0.0001* |

Using the Viasys, Vmax series body-box system, mouth occlusion pressure at 0.1 s ($P_{0.1}$), tidal volume($V_T$), inspiratory time(Ti), expiratory time($T_E$), period of the respiratory cycle($T_{TOT}$), duty cycle($Ti/T_{TOT}$), mean inspiratory and expiratory flows ($V_T/Ti$ and $V_T/T_E$) and ventilation per minute($V'_E$) were measured, before and immediately after 30 minutes of waterpipe smoking or equal session in the smoking area using the waterpipe without smoking (control group, blind control was impossible). Spirometry (Jaeger MasterScreen spirometry system) was performed at the end of the control session. Wilcoxon signed rank and U-Mann-Whitney rank sum tests were performed for the statistical analysis.

## Results

Immediately after 30 minutes of water-pipe smoking, $T_E$ and $T_E/T_{TOT}$ were significantly reduced (-5.2%, p=0.041 and -2.84%, p=0.0003 respectively) whereas $Ti/T_E$, $Ti/T_{TOT}$, $V_T/T_E$, $P_{0.1}$ and $P_{0.1}/(V_T/Ti)$ increased significantly (+7.67%, p=0.001, +4.12%, p=0.002, +7.12%, p=0.014, +10.6%, p=0.041 and +9.71%, p=0.022 respectively) in the total population studied (n=50). In both subgroups (MRS, n=25 and NRS, n=25), only $Ti/T_E$ and $Ti/T_{TOT}$ were significantly increased whereas $T_E/T_{TOT}$ was significantly reduced. No differences in the above changes identified between the two subgroups.

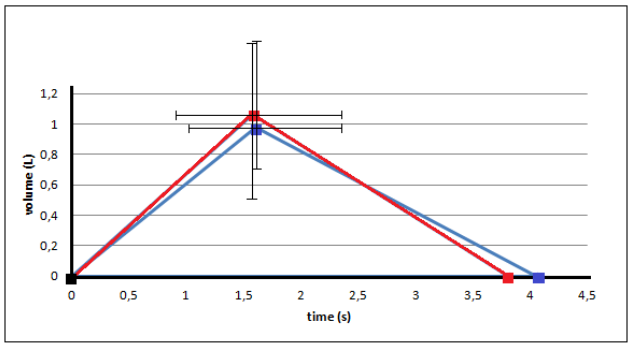


***Figure 1: Schematic representation of the volume-time curves, based on the mean values of tidal volume (y-axis), inspiratory time and period of the respiratory cycle (x-axis), before (blue curves) and immediately after (red curves) 30 minutes of waterpipe smoking, in the total population under study***

## Conclusion

We confirmed the finding of a previous study that waterpipe smoking causes immediate increase of the respiratory rate ($RR=1/T_{TOT}$)[4]. We found though that ventilation per minute was not significantly increased. Thus, all our 50 subjects maintained their $V'_E$ by increasing duty cycle ($Ti/T_{TOT}$) against $T_E$ and $T_{TOT}$, with consequent increase of breathing frequency. In regard to the neuromuscular inspiratory drive, we found that $P_{0.1}$ and inspiratory impedance [$P_{0.1}/(V_T/Ti)$], expressing the load surmounted at inspiration, increased significantly. Furthermore, we showed that waterpipe smoking has immediate effects on $P_{0.1}$ and respiratory timing, regardless of atopic history, respiratory symptoms and mild peripheral airways obstruction presence.

We hypothesize that, as it happens with cigarette smoke inhalation, identified modifications were attributable mainly to the stimulation of peripheral (carotid and lung) chemoreceptors, as a response to increased nicotine levels and inhaled dust[3], since carbon monoxide, even if it is increased, does not stimulates the carotid chemoreceptors, which mainly contribute to the control of breathing configuration in human[7].

## References

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