Original Paper

Water-pipe Smoking among young healthy smokers:

Immediate effects on breathing pattern, respiratory drive and mechanics of tidal breathing

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Key Words:
- Carbon Monoxide
- Respiratory Center
- Respiratory Mechanics
- Respiratory Rate
- Smoking Water Pipes

Abbreviation List
CO: Carbon monoxide
COHb: Carboxyhaemoglobin
CoB: Control of Breathing
T_I/T_{tot}: Duty cycle
eCO: Exhaled carbon monoxide
V_T/T_I: Mean inspiratory flow
ANOVA: Repeated measurements analysis of variance
f: Respiratory frequency
V'_E: Minute Ventilation
WPS: Water-pipe smoking

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SUMMARY
BACKGROUND: The present study assessed the immediate respiratory effects of water-pipe smoking (WPS) specifically focusing on tidal breathing examining Impulse Oscillometry (IOS), Control of Breathing (CoB) and exhaled CO (eCO) among young healthy adults. **METHODS:** A cross-over study design with sample size of 50 young healthy smokers was used. All measurements were taken immediately pre and post a Control and Experimental session. Repeated analysis of variance (ANOVA) and log-transformations were used for comparisons between pre-post and sessions. Significance was set to $p<0.05$. **RESULTS:** During the Experimental session, T_I/T_E, T_I/T_{tot} ($p<0.001$), $P_{0.1}$ ($p=0.005$) and $P_{0.1}/(V_T/T_I)$ ($p=0.021$) increased significantly while T_E/T_{tot} decreased ($p=0.003$) post WPS. IOS parameters Z5, R5, R10, R20 and fdr all increased significantly immediately post WPS ($p<0.001$) as did eCO and COHb ($p<0.001$). **CONCLUSION:** A 30-minute session of WPS altered respiratory mechanics expressed by the increased large and peripheral airways resistance, control of breathing expressed by increased $P_{0.1}$ and modified the tidal breathing pattern.
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INTRODUCTION

Water-pipe smoking (WPS), typically practiced in Middle Eastern countries and perceived as less harmful than other tobacco products,[1,2] has become a global trend. Increased popularity and use of the water-pipe, has been associated with increased reported cases of Carbon Monoxide (CO) poisoning,[3] especially among adolescents and young adults.[4] During the sessions of WPS, the high quantities of CO produced by the burning charcoal are inhaled by the user and consequently lead to increased exhaled CO.

Previous studies on WPS have shown it is associated with adverse respiratory and cardio-vascular outcomes.[2] More specifically, studies on lung function, using the forced respiratory maneuvers of Spirometry and flow-volume loops, have shown respiratory mechanics alterations expressed as decreased forced expiratory flows and volumes (FEV1, FVC, PEF, FEF25-75), while WPS studies on cardiopulmonary exercise testing have shown decreased exercise capacity.[5–10] Furthermore, a consistent finding among the majority of studies conducted on WPS has been increased respiratory rate(f)[4,6,8,11] and increased exhaled CO (eCO).[3,5]

Respiratory rate and tidal volume are determinants of minute ventilation (V'E= VT x f), while increased CO inhalation and the consequent Carboxyhaemoglobin (COHb) formation leads to hypoxia and compensatory increase in ventilation.[5] Therefore, the above-mentioned findings are indicative of an immediate effect of WPS on the control and pattern of tidal breathing. This effect however, has not yet been systematically explored by the classical non-invasive techniques available for the study of control of breathing (CoB).[12] While the study of tidal breathing can provide more insight into respiratory pathophysiology and clinical symptomatology by detecting subtle alterations which are not evident by conventional forced techniques[13] the possible impact of WPS on the mechanics of tidal breathing has not yet been addressed.

Considering the existing gap, the current study aimed for the first time, to assess the immediate effect of WPS on CoB and respiratory mechanics. Specifically targeting tidal breathing the present study examined breathing pattern analysis,mouth occlusion pressure, and Impulse Oscillometry (IOS) in young healthy smokers.

METHODS

Participants

50 young adult smokers living in Athens, Greece voluntarily participated in the study. All participants reported no significant medical history or medications. Exclusion criteria included ages under 18 or over 35 years old, any acute or chronic disease, recent infection (<4 weeks prior to study), any use of medication (<2 weeks prior to study), pregnancy/lactation, or body mass index >30kg/m^2.

Study Design

An experimental, cross-over study design was applied on the aforementioned population. Each participant underwent three sessions that took place over three consecutive days in a lung function laboratory. Participants underwent each session one at a time. Participants were instructed to avoid consuming food, drinks and beverages for four hours prior to the sessions[14] and smoking for 12 hours prior[15] to sessions, which was confirmed with eCO measurement <7ppm.[16] During the first visit, initial assessment including medical history and flow-volume loop were measured for inclusion. During the second day (Control) and the third day (Experimental) sessions, measurements of IOS, CoB and eCO were performed in the above-mentioned sequence, before (pre) and immediately after (post) 30 minutes of WPS.

During the Experimental session, all subjects were instructed to remain in a sitting position and smoke ad libitum for 30 minutes inside a devoted smoking area ~30m^3 using the same type of water-pipe device (16 inches), 10g of peach-flavored moasel of the same brand and the same instant-light charcoal disks (diameter=3.8cm; width=1.5cm).

For the Control session,all subjects mimicked smoking water-pipe for 30 minutes without it being lit under the above-mentioned conditions. Since there was no smoke production by the use of the water-pipe without tobacco and charcoal disks, blind control was not possible.

The ethics committee of the Hellenic Cancer Society in Athens, Greece provided ethics approval (protocol number: 561/28-1-14) for the current study. Each subject read and signed a consent form prior to study enrollment.

Measurements

Flow-volume loop

Flow-volume loop was obtained during initial assessment for inclusion criteria only and was performed in a sitting position with a nose-clip applied using a Jaeger Master Screen Spirometry system (Franklin Lake, NJ, USA) according to the American Thoracic Society/European Respiratory Society guidelines.[17] Forced vital capacity (FVC), forced expiratory volume in the first second (FEV1), Tiffeneau Index (FEV1/FVC%), peak expiratory flow (PEF) and mid-expiratory flows (FEF at 25%, 50% and 75% of exhaled FVC) were recorded.

IOS measurements

Total Impedance (Z5), Resistance at 5, 10 and 20 Hz (R5, R10, and R20, respectively), Reactance at 5, 10 and 20 Hz (X5, X10 and X20, respectively),Frequency Dependence

of Resistance (fdr=R5-R20),Resonant Frequency (fres) and Reactance Area (AX) were measured using the Viasys Jaeger Master Screen IOS system(Franklin Lake, NJ, USA). IOS was performed according to guidelines and included three reproducible trials with an intra-assay coefficient of variation <10%.[18,19]

CoB measurements: P0.1 and Analysis of Tidal Breathing

P0.1, f, V·E, inspiratory time (TI), expiratory time (TE), period of the respiratory cycle (Ttot), duty cycle (TI/Ttot), tidal volume (VT), mean inspiratory and expiratory flow (VT/TI and VT/TE) were measured, while TI/TE, TE/TTOT were calculated using a Care Fusion (formerly VIASYS and Cardinal Health) body-box system (Yorba Linda, CA, USA) and according to manufacturer guidelines. In addition, respiratory impedance [P0.1/(VT/TI)] was subsequently calculated during analysis. Each participant, in a sitting position with a nose-clip applied, was instructed to breathe quietly for ~30 seconds and then were instructed to inspire to total lung capacity. Mean values of four efforts were recorded for all above-mentioned measured parameters.[20]

Exhaled Carbon Monoxide measurements

eCO (ppm)measurements were performed using the Bedfont® Scientific Ltd. Micro+™ Smokerlyzer® (Harrietsham, Maidstone, Kent) equipment and according to manufacturer guidelines. The same device, also estimated-COHb levels using the Jarvis equation[21]. With a nose-clip applied, participants were instructed to quietly inhale and hold their breath for approximately 15 seconds and consecutively quietly exhale for approximately 10 seconds.[22]

Statistical analysis

Continuous variables were presented with means and standard deviations (SD). Differences in the changes between pre and post in Experimental and Control sessions for all measured parameters, were evaluated using repeated measurements analysis of variance (ANOVA). Log-transformations were made for the analysis of variance in case of skewed distribution. All p-values reported were two-tailed. Statistical significance was set at 0.05 and analyses were conducted using SPSS statistical software (version 20.0)

RESULTS

The current sample consisted of 50 young smokers with a mean age of 23±4.2 years old, an average of 3.6 pack-year history(average smoking of 7 cigarettes per day) and had normal flow-volume loop (Table 1).

The Control session revealed no significant changes between pre and post measurements for all IOS parameters (Table 2). In the Experimental session, Z5, R5, R10, R20 and fdr increased significantly immediately post WPS ($p<0.001$). These mean changes were also significantly different between Experimental and Control sessions ($p<0.001$) (Table 2).

Mean changes of eCO during the Experimental session increased significantly from 3.54±2.29 ppm to 27.16±12.32 ppm ($p<0.001$) pre to post WPS and COHb also increased from 1.22±0.35 % to 4.98±1.97 % ($p<0.001$) (Table 3).

In the Control session, no significant differences were found between pre and post measurements for all CoB parameters (Table 4). In the Experimental session, TI/TE, TI/Ttot increased significantly from 0.71±0.16 to 0.77±0.17 ($p<0.001$) and 0.39±0.05 to 0.40±0.05 ($p<0.001$),respectively, and TE/Ttot decreased from 0.56±0.05 to 0.54±0.05 ($p<0.05$) post WPS. P0.1 and P0.1/(VT/TI) also increased significantly ($p<0.05$) from 0.35±0.01 to 0.39±0.15kPa and 0.54±0.18 to 0.59±0.23kPa s-1l-1, respectively, in the Experimental session. The pre to post changes for TI/TE, TI/Ttot, TE/Ttot and P0.1/(VT/TI) in the Experimental session were found to be significantly different from those in the Control session ($p<0.05$) (Table 4).

TABLE 1. Mean and Standard Deviation (SD) for Demographics and Flow-volume Loop

	Mean (SD)
Males/Females	32/18*
Age (years)	23.4 (±4.2)
BMI (kg/m^2)	23.4 (±3.2)
Pack-years	3.6 (±2.8)
Flow-volume loop	
FVC	103.3 (±10.6)
FEV1	102 (±11.9)
FEV1/FVC%	84.5 (±7.5)
PEF	97.5 (±12.9)
FEF 25%-75%	93.7 (±27.5)
FEF 25%	96.6 (±38.3)
FEF 50%	95.3 (±25.5)
FEF75%	99.3 (±21.8)

Note: * – absolute frequency, BMI: body mass index, FVC: Forced vital capacity, FEV1: forced expiratory volume in the first second, FEV1/FVC%: Tiffeneau Index, PEF: peak expiratory flow, FEF at 25%, 50% and 75% of exhaled, FVC: mid-expiratory flows.
All Spirometry parameters were against their % predicted values.

TABLE 2. Mean and Standard Deviation (SD) of changes pre to post water-pipe smoking for Impulse Oscillometry parameters in Control and Experimental sessions

	Pre Mean (SD)	Post Mean (SD)	Change Mean (SD)	P*	P**
Z5 [kPa/(L/s)]					
Control	0.36 (0.12)	0.37 (0.13)	0.01 (0.05)	0.949‡	<0.001‡
Experimental	0.34 (0.1)	0.38 (0.11)	0.04 (0.05)	<0.001‡	
R5 [kPa/(L/s)]					
Control	0.35 (0.12)	0.35 (0.12)	0.00 (0.05)	0.932	<0.001
Experimental	0.32 (0.09)	0.36 (0.11)	0.04 (0.05)	<0.001	
R10 [kPa/(L/s)]					
Control	0.32 (0.11)	0.31 (0.11)	-0.01 (0.05)	0.699	<0.001
Experimental	0.29 (0.08)	0.33 (0.09)	0.04 (0.04)	<0.001	
R20 [kPa/(L/s)]					
Control	0.32 (0.1)	0.31 (0.09)	-0.01 (0.05)	0.612	<0.001
Experimental	0.29 (0.07)	0.32 (0.08)	0.03 (0.04)	<0.001	
fdr (R5-R20) [kPa/(L/s)]					
Control	0.33 (0.11)	0.33 (0.1)	0.00 (0.05)	0.877	<0.001
Experimental	0.31 (0.08)	0.34 (0.09)	0.03 (0.04)	<0.001	
X5 [kPa/(L/s)]					
Control	-0.11 (0.04)	-0.11 (0.04)	0.00 (0.02)	0.852	0.483
Experimental	-0.1 (0.04)	-0.1 (0.04)	0.00 (0.02)	0.421	
X10[kPa/(L/s)]					
Control	-0.01 (0.04)	-0.01 (0.05)	0.00 (0.02)	0.507	0.742
Experimental	-0.01 (0.04)	-0.01 (0.03)	0.00 (0.02)	0.260	
X20[kPa/(L/s)]					
Control	0.09 (0.07)	0.08 (0.05)	-0.01 (0.06)	0.095	0.210
Experimental	0.08 (0.05)	0.08 (0.04)	0.00 (0.02)	0.921	
*f*res(Hz)					
Control	11.5 (3.87)	11.62 (4.1)	0.12 (2.12)	0.660	0.973
Experimental	11.45 (3.58)	11.56 (3.52)	0.11 (1.85)	0.695	
AX (kPa/L)					
Control	0.35 (0.39)	0.38 (0.46)	0.03 (0.23)	0.608‡	0.640‡
Experimental	0.31 (0.3)	0.32 (0.28)	0.01 (0.19)	0.238‡	

Notes: * p-value for smoking effect,
** Effects reported include differences between the groups in the degree of change (repeated measurements ANOVA),
Z5: Total Impedance, R5: Resistance at 5Hz, R10: Resistance at 10Hz, R20: Resistance at 20Hz, X5: Reactance at 5Hz, X10: Reactance at 10Hz, X20: Reactance at 20Hz, fdr: Frequency Dependence of Resistance, fres: Resonant Frequency, AX: Reactance Area.
‡ p-value based on logarithmic transformations, significant p-values indicated in bold.

DISCUSSION

The current study examined for the first time, the immediate effects of a 30 minute WPS session on parameters of tidal breathing, among young healthy smokers.

Evaluation of respiratory mechanics, control of breathing and eCO showed increased respiratory impedance and resistance, increased mouth occlusion pressure, modification of the tidal breathing pattern and increased exhaled CO.

WPS exhibits some particularities and differences from other tobacco products, as the water-pipe is a complex device consisting of a vase and a tubing circuit that adds an exogenous resistance to breathing. Furthermore, the charcoal, used for burning the tobacco, produces high concentrations of CO in addition to the conventional tobacco smoke constituents.[23]

Current IOS findings showed increased Total Impedance and Resistance post WPS and are in line with previous stud-

TABLE 3. Mean and Standard Deviation (SD) of changespre to post water-pipe smoking forExhaled Carbon Monoxide (eCO)and Carboxyhaemoglobin (COHb) in Control and Experimental sessions

	Pre Mean (SD)	Post Mean (SD)	Change Mean (SD)	P*	P**
eCO (ppm)					
Control	4.32 (2.41)	4.18 (2.38)	-0.14 (1.54)	0.909	<0.001
Experimental	3.54 (2.29)	27.16 (12.32)	23.62 (12.17)	<0.001	
COHb (%Hb)					
Control	1.50 (0.67)	1.43 (0.55)	-0.07 (0.58)	0.716	<0.001
Experimental	1.22 (0.35)	4.98 (1.97)	3.76 (1.94)	<0.001	

Notes: SD –Standard deviation, * p-value for difference between pre and post; **p-value for differences between sessions in the degree of change;All p-values reported are derived from repeated measurements ANOVA. Significant p-values indicated in bold.

ies that have examined the health effects of active and passive smoking of various other tobacco products.[15,16,24–27] IOS, using an effort independent, tidal breathing manouever, is considered more sensitive for the evaluation of lung mechanics compared to Spirometry.[28] An increase in Z5, R5, R20 and fdriscorrelated with acute bronchoconstriction and reduction of airway calibre in healthy and individuals with asthma.[29–31] Therefore, current IOS findings of increased total impedance and resistance, indicate that WPS irritated both large(R20)and peripheral airways (R5-R20)and led to a degree of bronchoconstriction.[19]

The current study found that P0.1 was significantly increased post the Experimental session, as was the effective Inspiratory Impedance (P0.1/VT/TI), denoting an increased inspiratory load, which is in agreement with the current IOS findings of increased total respiratory impedance and resistance. The addition of inspiratory resistance to normal subjects leads to the increase of their P.01, denoting an increased respiratory drive[32] as was the case in the current study.

V·E, *f* and VT/TI remained unchanged after the Experimental session. However, an acute, mild modification of the breathing pattern was observed, expressed by a tendency for TE and Ttot to decrease, that became more obvious in the significantly decreased ratio TE/Ttot and the increased ratio VT/TE. Further analysis of the breathing cycle structure, showed an effect of WPS on the Timing component, expressed by the significantly increased inspiratory duty cycle (TI/Ttot), while the mean inspiratory flow (VT/TI) did not change.

Since the exogenous resistance added by the water-pipe tubing circuit did not account for any pre to post changes in the control session measurements, the increased P0.1 found in the current study could be interpreted as the respiratory system's response to either the direct effect of CO on CoB,[33] the inhalation of the mixture of irritative constituents of WPS, the direct nicotine effect on the Central Nervous System, or to a combination of the above-mentioned factors.

Regarding the eCO, the significant increase observed post WPS in the current study is in agreement with previous studies on WPS.[4,23,34] CO inhalation during WPS has been shown to be ten times higher as compared to cigarette smoking.[35] Moreover, the burning charcoal releases increased concentrations of CO into the environment to which both smokers and bystanders are exposed and consequently at risk for CO intoxication.[35]

The underlying mechanism for CO toxicity is based on the intense chemical affinity of CO to haemoglobin (240 times higher than that of oxygen),[36] that leads to the inability for cells to use oxygen, the end-result being tissue and cellular hypoxia.[33] The consequent brain hypoxia has been shown to activate the central chemoreceptors, initially leading to hyperventilation that is followed by delayed hypoventilation.[33] This response however, usually occurs at higher levels of COHb (>60%),[33] while at concentrations <20%, only the more sensitive higher centers of the central nervous system respond presenting as cognitive and psychomotor impairment.[37]

As mean eCO measurements found post WPS were 27ppm with the estimated COHb levels in the range of 3-7%,the present study results indicate that even low COHb concentrations in the range of 5-20%are possibly associated with detectable alterations of the respiratory center output and lung mechanics.

Furthermore, it highlights the advantage of examining tidal breathing and increases our understanding of how WPS affects the respiratory function,not only at the airway level by increasing airway resistance, but also and more importantly,at the level of its central regulation leading to a modification of the breathing pattern and increased respiratory drive.

TABLE 4. Mean and Standard Deviation (SD) of changes pre to post water-pipe smoking forControl of breathing parameters in Control and Experimental sessions

	Pre Mean (SD)	Post Mean (SD)	Change Mean (SD)	P*	P**
f (breaths/min)					
Control	15.30 (5.00)	15.48 (4.60)	0.18 (2.70)	0.665	0.564
Experimental	15.70 (4.30)	16.26 (4.67)	0.52 (3.10)	0.213	
V·E (1/min)					
Control	15.25 (4.19)	14.95 (3.90)	-0.30(2.90)	0.479	0.147
Experimental	15.57 (3.87)	16.15 (4.19)	0.58(3.10)	0.179	
TI (s)					
Control	1.66 (0.53)	1.67 (0.52)	0.01 (0.32)	0.839	0.707
Experimental	1.62 (0.53)	1.66 (0.55)	0.04 (0.40)	0.463	
TE (s)					
Control	2.40 (0.91)	2.43 (0.92)	0.03 (0.41)	0.654	0.109
Experimental	2.34 (0.80)	2.22 (0.72)	-0.12 (0.52)	0.069	
Ttot(s)					
Control	4.27 (1.41)	4.29 (1.36)	0.02 (0.66)	0.860	0.488
Experimental	4.17 (1.28)	4.08 (1.20)	-0.09 (0.88)	0.421	
TI/TE					
Control	0.72 (0.14)	0.72 (0.17)	0.00 (0.10)	0.888	0.017
Experimental	0.71 (0.16)	0.77 (0.17)	0.06 (0.11)	<0.001	
TI/Ttot					
Control	0.39 (0.04)	0.39 (0.05)	0.00 (0.03)	1.00	0.012
Experimental	0.39 (0.05)	0.40 (0.05)	0.01 (0.03)	<0.001	
TE/Ttot					
Control	0.55 (0.05)	0.56 (0.06)	0.01 (0.04)	0.525	0.011
Experimental	0.56 (0.05)	0.54 (0.05)	-0.02 (0.03)	0.003	
VT (l)					
Control	0.97 (0.28)	0.99 (0.26)	0.02 (0.15)	0.605	0.805
Experimental	1.03 (0.22)	1.05 (0.27)	0.02 (0.21)	0.387	
VT/TI(l/s)					
Control	0.61 (0.18)	0.62 (0.17)	0.01 (0.11)	0.756	0.715
Experimental	0.67 (0.17)	0.67 (0.17)	0.00 (0.13)	0.836	
VT/TE(l/s)					
Control	0.44 (0.16)	0.45 (0.17)	0.01 (0.12)	0.670	0.253
Experimental	0.47 (0.15)	0.5 (0.16)	0.03 (0.11)	0.043	
P0.1 (kPa)					
Control	0.32 (0.09)	0.33 (0.10)	0.01 (0.12)	0.763	0.153
Experimental	0.35 (0.1)	0.39 (0.15)	0.04 (0.10)	0.021	
P0.1/(VT/TI) (kPa s-1l-1)					
Control	0.66 (0.17)	0.64 (0.17)	-0.02 (0.11)	0.469	0.013
Experimental	0.54 (0.18)	0.59 (0.23)	0.05 (0.15)	0.005	

Note: SD: Standard Deviation,

* p-value for difference between pre and post;

** p-value for differences between sessions in the degree of change);f–Respiratory frequency,

V·E: Ventilation, TI: Inspiratory time, TE: Expiratory time, Ttot: Period of the respiratory cycle, TI/Ttot: Duty cycle, VT: Tidal volume, VT/TI: Mean inspiratory flow, VT/TE: Mean Expiratory flow, P0.1: Mouth occlusion pressure, P0.1/VT/TI: Inspiratory Impedance.
All p-values reported are derived from repeated measurements ANOVA. Significant p-values indicated in bold.

Study limitations included non-blind control, non-standardized puffing topography and not having addressed the possible role of nicotine on current findings.

CONCLUSION

A 30-minute session of WPS had significant immediate effects on tidal breathing mechanics and control of breathing expressed by increased respiratory impedance and resistance of central and peripheral airways, increased mouth occlusion pressure and a modification of the breathing pattern. Given the rising global trend of water-pipe smoking, these findings add to the growing amount of evidence on the harmful effects of water-pipe smoking.

ΠΕΡΙΛΗΨΗ

Κάπνισμα ναργιλέ: άμεσες επιδράσεις στον τύπο της αναπνοής, στην κεντρική αναπνευστική ώση και στη μηχανική της ήρεμης αναπνοήςσε υγιείς νεαρούς ενήλικες

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Εισαγωγή: *Η παρούσα έρευνα, μελέτησε τις άμεσες επιπτώσεις καπνίσματος ναργιλέ στο αναπνευστικό νεαρών υγιών ατόμων, με ιδιαίτερη έμφαση στην ήρεμη αναπνοή, εξετάζοντας την Παλμική Ταλαντωσιμετρία (IOS), τη Ρύθμιση της Αναπνοής(CoB) και το Εκπνεόμενο Μονοξείδιο του άνθρακα(e-CO).* ***Μεθοδολογία:*** *50 νεαροί υγιείς ενήλικες συμμετείχαν σε μελέτη διασταυρούμενης μετάβασης (cross over). Όλες οι μετρήσεις πραγματοποιήθηκαν πριν(pre) και αμέσως μετά (post) από μια συνεδρία ελέγχου (Control) και μια πειραματική (WPS). Η σύγκριση μεταξύ pre, post μετρήσεων και συνεδριών πραγματοποιήθηκε με χρήση Repeated Analysis of Variance (ANOVA) και λογαριθμικής μετατροπής (log transformations). Η σημαντικότητα ορίσθηκε ως p<0.05.* ***Αποτελέσματα:*** *Οι μεταβλητές T_I/T_E, T_I/T_{TOT}, (p<0.001), $P_{.01}$ (= 0.005) και $P_{.01}/(V_T/T_I)$ (p=0.021) αυξήθηκαν σημαντικά, ενώ η σχέση T_E/T_{TOT} μειώθηκε σημαντικά (p=0.003) μετά το κάπνισμα ναργιλέ (post WPS). Οι μεταβλητές IOS, Z5, R5, R10, R20 και fdr αυξήθηκαν σημαντικά (p<0.001) αμέσως post WPS, όπως και το e-CO και η COHb (p<0.001).* ***Συμπέρασμα:*** *Η τριαντάλεπτη συνεδρία WPS είχε επιπτώσεις στη μηχανική της αναπνοής που εκφράσθηκαν με αύξηση της αντίστασης κεντρικών και περιφερικών αεραγωγών, καθώς και στη Ρύθμιση της Αναπνοής που εκφράσθηκε με αύξηση της πίεσης Σύγκλεισης και μεταβολή του τύπου της ήρεμης αναπνοής.*

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Λέξεις - Κλειδιά: *Μονοξείδιο άνθρακα, Αναπνευστικό κέντρο, Μηχανική της Αναπνοής, Αναπνευστική Συχνότητα, Κάπνισμα ναργιλέ*

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