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Acute effect of cigarette smoking on peak expiratory flow rate and ventilation in resting Nigerian subjects

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Summary

The effects of cigarette smoking on peak expiratory flow rate (PEFR) and ventilation (V) in resting Nigerian subjects had been investigated in healthy male subjects (10 smokers, 8 non-smokers). The PEFR, V and tidal volume (V_T) decreased significantly after smoking two medium-tar cigarettes. The action of smoke particles (carbon and tar) on the irritant receptor reflex of the lung, thus producing bronchoconstriction, and the depressant action of nicotine on the peripheral chemoreceptors, central chemoreceptors and respiratory centre may be the causal factors.

Résumé

Les effets de fumer la cigarette sur la visière du taux de la circulation expiratoire (PEFR) et la ventilation (V) des sujets nigérians reposants, ont été étudié chez les mâles sujets sains (10 fumeurs, 8 non-fumeurs). La PEFR, la V et le volume de force de la marée ont diminué significativement après deux moyengoudron cigarettes fumées. L'action des particules de la fumée (la carbone et la goudron) sur le réflexe du récepteur irritant du poumon, qui produite la bronchoconstriction, et l'action dépressive de la nicotine sur les chimiorécepteurs périphériques, les chimiorécepteurs centraux et le centre respiratoire sont, peut-être, les facteurs causatifs.

Introduction

Comparatively little data on changes in ventilation before and after smoking are available in the literature, but it has been demonstrated that the acute inhalation of cigarette smoke is associated with an increase in airway resistance, i.e. is a decrease in the airway conductance/thoracic volume ratio (Gaw/TGV) [1–3].

This decrease in Gaw/TGV is indicative of a decrease in cross-sectional area of the airways after cigarette smoking. With such alterations in lung mechanics, the minute ventilation, peak expiratory flow rate and breathing pattern might be affected in a way similar to that observed by Du Bois *et al.* [4] in obstructive respiratory disease. Absorbed components of the smoke may also have an effect on the respiratory control mechanisms.

Subjects and methods

The subjects, aged 20–45 years, sat quietly and relaxed in a chair in the laboratory, and the transthoracic impedance monitor (TTIM) electrodes were applied between the fourth rib and the sixth rib on the mid-axillary line on each side of the thorax, and the earth electrodes were placed on the scapula. The leads from the electrodes were connected to the impedance pneumograph. Changes in impedance accompanying respiration were recorded as changes in voltage on a physiograph recorder. Prior to the above procedure the subjects were made to take a deep breath and expire into a Wright peak flow meter for control measurements of the peak expiratory flow rate (PEFR).

The peak flow meter was checked for its reliability by using readings from three healthy technicians as standards. Reproducibility of three readings from each of the technicians signals the reliability in the functioning of the peak flow meter before the experimental session.

The subjects sat quietly and relaxed for 10 min, then they inhaled smoke from two

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medium tar cigarettes, smoked one after the other over a period of 5 min. Continuous respiratory recordings were made before, during and for a further 10 min after the second cigarette.

Eighteen normal male adults (10 smokers, 8 non-smokers), with no known history of cardiopulmonary disease, were studied. All subjects were advised to restrain from smoking for at least 1 h before the beginning of the experiment, since Nadel and Comroe [1] indicated that the mild bronchoconstriction caused by the inhalation of cigarette smoke lasted for 10–80 min.

The TTIM measures changes in the respired volume and changes in resistance across the thorax. A simple calibration of the TTIM against spirometer volumes was done before and after every experimental session [5,6].

The last 5 min recorded before smoking and the last 5 min recorded after smoking were used for tidal volume (V_T), respiratory frequency (F) and ventilation (V) measurements.

The subjects also expired into the Wright peak flow meter after smoking, in order to measure peak expiratory flow rate. The 18 subjects used for the experiments were studied individually on three occasions, and the results were similar and comparative.

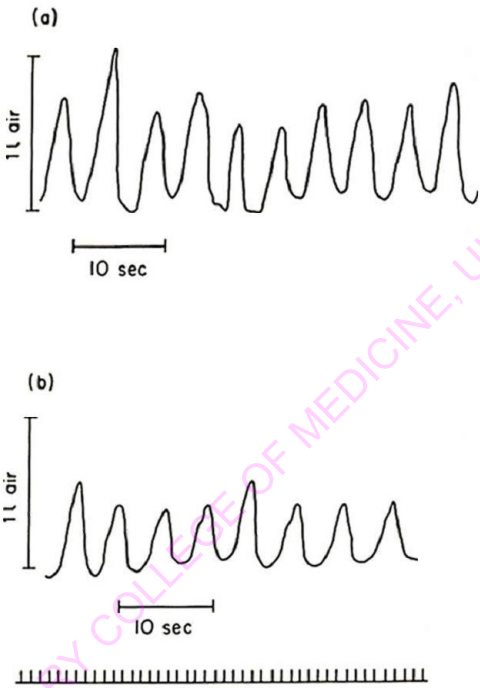

Fig. 1. (a) Represents control respiratory movements before smoking; (b) represents respiratory movements after smoking two cigarettes.

Results

Table 1 and Fig. 1 show typical results of the experiments. The V_T and V decreased significantly after smoking the two cigarettes by 25% and 20%, respectively ($P < 0.005$; Student's *t*-test). No significant change was found with the respiratory frequency.

We have observed that no significant changes occur in ventilation measurements from 0 to 30 min in seated subjects, using the TTIM (see Table 2), and so the changes observed in these

Table 1. Effect of smoking on tidal volume (V_T), respiratory frequency (F) and ventilation (V)

		Before smoking	After smoking
V_T	Mean	0.51	0.38*
	s.e. ±	± 0.05	± 0.04
F	Mean	16.7	17.8
	s.e. ±	± 1.3	± 1.4
V	Mean	8.31	6.64*
	s.e. ±	± 0.73	± 0.63
	$n = 18$		

Means and standard errors were calculated from the values obtained in 18 subjects.
*$P < 0.005$.

Table 2. TTIM measurements of ventilation (V) at 0 and 30 min in five subjects

	Ventilation (litres/min)	
	0	30
	6.64	6.60
	5.98	6.31
	5.50	5.60
	4.73	4.75
	4.50	4.45
Mean	5.47	5.54
s.e. ±	0.21	0.43

experiments were likely to be due to smoking. Table 3 summarizes the results from the peak expiratory flow rate (PEFR) measurements before and after smoking (n = 10). Using student's paired t-test, a significant difference was observed in the PEFR before and after smoking the two cigarettes ($0.01 > P > 0.001$).

Table 3. Peak expiratory flow rate (litres/min) before smoking (control) and after smoking two cigarettes

Subject no.	Before smoking (control)	After smoking two cigarettes
1	555	500
2	460	450
3	570	500
4	500	420
5	610	570
6	500	370
7	560	500
8	570	490
9	560	500
10	600	560
	Mean ± s.e. = 548.5 ± 14.8	Mean ± s.e. = 486 ± 18.7

Using Student's paired t-test there is a significant difference between the means, $0.01 > P > 0.001$.

Discussion

Possible explanation for these observations may be found in some of the properties of the major constituents of cigarette (carbon, tar and nicotine).

The classical ventilatory depression that might be caused by bronchoconstriction would be decreases in both tidal volume and respiratory frequency [4] but in this study only a decrease in tidal volume was observed.

Chiang and Wang [7] reported an insignificant decrease ($P > 0.1$) in ventilation in seven subjects (non-smokers), but in this study a significant fall in ventilation ($P < 0.05$) has been found. The dissimilarity in results may be due to the choice of subjects and the technique used in the measurements. The subjects in our study included both smokers and non-smokers, and the measurements were made by a method that was non-invasive, atraumatic and did not cause any obstruction to the airway. Chiang and Wang [7] used the spirometer for their measurements and their subjects were mainly non-smokers. The spirometer has been found to alter ventilation and its patterns [8] so the V_T, F and V measurements of Chiang and Wang [7] may be unreliable.

Nicotine in small doses is known to stimulate and in large doses depress respiration by its action on the chemoreceptors of the carotid and aortic bodies and of the respiratory centre, as first demonstrated by Nadel and Comroe [1]. Plasma nicotine levels after smoking were not measured in this study but might have contributed to the observations.

In conclusion, the effects of cigarette smoke in reducing peak expiratory flow rate, ventilation and tidal volume demonstrated here may be due to either or both of the following: (i) the action of smoke particles (carbon and tar) on the irritant receptor reflex of the lung, producing broncho-constriction; (ii) the depressant action of nicotine on the peripheral chemoreceptors, central chemoreceptors and respiratory centre.

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