Patterns of smoking: measurement and variability in asymptomatic smokers

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Summary

1. Measurements of patterns of puffing (cigarette-holder pneumotachograph) and ventilation (plethysmography) were made in ten asymptomatic smokers during the smoking of a cigarette, on four separate occasions.

2. There were marked individual differences and these were consistent over 3-5 weeks. In itself, the pattern of smoking could be responsible for a threefold variation in smoke intake.

3. Puffing but not inhalation became less intense as a cigarette was smoked.

4. It was not possible to predict indices of absorption from smoking patterns.

5. Certain smoking patterns, e.g. small puff volume, low puff frequency, short duration of inhalation and expulsion of volume between puff and inhalation, may be less harmful than others and this may explain why some individuals remain healthy despite a lifetime of smoking.

Key words: breathing patterns, cigarette smoking.

Introduction

There is little doubt that cigarette smoking is associated with an increased incidence of respiratory and cardiovascular disease [1, 2]. However, clinical experience reveals that many life-long smokers suffer no such impairment of health. Some of these individuals attribute their freedom from smoking-related disease to an absence of inhaling.

The objective of the present study was to ascertain to what extent a group of healthy smokers adopted a reproducible pattern of smoking that might contribute to long-term risk over and above that due to the total number of cigarettes smoked per day and the number of years of smoking. The genuine non-inhaler clearly should be less at risk than his inhaling counterpart.

This study has therefore attempted to measure the ventilatory events associated with smoking and their reproducibility on different occasions. The objective has been to look at all the ventilatory factors that might contribute to deposition and absorption of tobacco smoke constituents within the lung.

The results of this study have been presented in a preliminary form [3].

Methods

Subjects

Ten asymptomatic smokers (age 18-30 years) took part in this study. All smoked filter cigarettes; two of the seven males and one of the three females smoked low tar brands; the remainder were 'middle tar' smokers. After a 'trial' visit each subject came to the laboratory on four occasions, with at least 1 week intervals between visits, and as far as possible at the same time of day. On the first visit a short smoking history was taken and a forced expiratory manoeuvre performed; all subjects had a normal FEV1.0 and flow-volume curve.

Protocol: measurement of smoking patterns

Each subject provided one of their own cigarettes, and this was tightly fitted into a specially designed cigarette-holder pneumotachograph (Fig. 1). The pressure drop across a centrally positioned filter insert was measured with a
FIG. 1. Arrangement for smoking studies showing: (1) Krogh spirometer; (2) headset for supporting nasal probe; (3) airtight rubber seals at arms and neck. Subject enters the box through detachable sealed door at rear and is made comfortable on adjustable chair. Cigarette-holder pneumotachograph is held by the subject, who is able to listen to music or read a magazine; a screen prevents him seeing either spirometer or recorder. The insert shows a cut-away diagram of cigarette-holder pneumotachograph showing: (1) stop ring for cigarette; (2) cellulose acetate filter insert (6 mm); (3) detachable mouthpiece. Differential pressure was measured across A–B. Total dead space volume = 2.5 ml.

Validyne MP45 differential transducer. The device plus cigarette was calibrated with air for both flow (1.92 litres/min) and, separately, volume (50 ml). Changes in lung volume during smoking were measured by means of a head and arms-out volume displacement plethysmograph (Fig. 1), connected by rigid plastic tubing (4 cm i.d.) to a 2 litres Krogh spirometer (Emerson Inc.). Before each study, the system was sealed and separately calibrated with syringe-delivered volumes of 1 and 0.05 litre. The subject was then connected to a three-lead electrocardiogram and entered the plethysmograph via an air-tight rear door. The head and arm ports were sealed by using soft tightly fitting rubber sleeves; by providing arm supports and instructing the subject to avoid excessive head movement the possibility of errors due to leaks was minimized. A nasal temperature thermistor probe (Elab DU-3 with D-F6 thermocouple) was fitted in one of the nares to record adequate temperature fluctuations without discomfort. At least 5 min was allowed to lapse for temperature equilibration of the volume-measuring system, during which time an attempt was made to put the subject at ease; reading material was offered and background music was played continuously.

After the recording of resting ventilation and heart rate for several minutes, the subject was asked to take the cigarette-holder and to commence smoking the cigarette as normally as possible; an ash-tray was provided. Throughout this period, a continuous recording of the following signals was obtained. (1) Plethysmographic volume at low gain (1 litre/cm) and separately at high gain (20 ml/cm). (2) Puff flow from the pneumotachograph, which was integrated to give puff volume. (3) Temperature in the external nares. (4) The electrocardiogram.

Puffs taken within an estimated 8 mm of the cigarette filter were not analysed because of calibration errors caused by heated smoke (see below). On completion of the study the subject was asked for comments in terms of comfort.
Patterns of smoking

during the study, and how the study related to the everyday experience of smoking.

Assessment of absorption

Measurement of mixed expired carbon monoxide concentration was carried out before and 5 min after smoking the cigarette [4] and the 'CO boost' (increase in alveolar CO concentration from one cigarette) was derived and used as an index of absorption of the gaseous phase of tobacco smoke. The results were normalized to take into account the variable CO yields from different brands; the figures were supplied by the Tobacco Advisory Council and relate to 'machine-smoking' conditions. The range of normalization factors was from 0.79 to 1.16.

Measurement of the change in heart rate was obtained from the electrocardiogram before and immediately after smoking the cigarette; increase in heart rate was considered to be an index of nicotine absorption.

Subjective assessment of inhalation

The subject was asked to assess the extent of inhalation by marking a point on a Visual Analogue Scale (100 mm line) with extremes labelled 'no inhalation' and 'maximal inhalation'.

Validation of techniques

Puff flow

Linearity of the cigarette-holder pneumotachograph was assessed by using simulated puffs of known flow rates produced by a syringe-withdrawing motor. For each of nine flow rates (range 0.07-4.35 litres/min) a filter cigarette was fitted into the holder and the voltage output from the pneumotachograph was recorded during withdrawal of air. After lighting the cigarette, measurements were made for the first three 'puffs' of tobacco smoke (excluding the lighting puff) taken at 1 min intervals. This procedure was repeated four times; on each occasion the filter insert was renewed and the flow rate sequence changed.

Allowing for differences in insert and cigarette resistance, it was found that at any given flow rate with smoke both intra- and inter-cigarette variability was less than 1%. There were no discernible differences in measured flow rate when the cigarette was unlit as opposed to lit, except at the two highest levels of flow (3.45 and 4.35 litres/min), where smoke caused a higher reading of between 2 and 4%. In the presence of smoke the pneumotachograph response was linearly related to the actual flow rate below 3 litres/min but above this puff flow rate was overestimated. At the upper range of peak puff flows seen in this study (mean ± 2 SD = 2.5 ± 1.6 litres/min) this amounted to an error of approximately 7%. When such flows occurred during smoking they did so only transiently and it can be estimated that the error in puff volume measurement would be less than 4% in all cases.

The filter insert was always renewed after each cigarette had been smoked; the calibration for flow did not change with smoking.

The resistance to airflow of the pneumotachograph was 1 cm water min⁻¹ l⁻¹; that offered by a standard filter cigarette was 12 cm water min⁻¹ l⁻¹.

Puff volume

Puff volume was obtained by analogue integration of the puff flow signal. In practice any error due to nonlinearity of response was minimized by calibrating the integrated flow output by using a profile similar to an individual's characteristic puff profile (see below).

With simulated puffs it was found that a fixed volume of smoke drawn through the cigarette-holder pneumotachograph with a consistent flow pattern produced a constant integrated output. Puff volume could be quantified to an accuracy of 0.5 ml.

Effect of temperature on flow measurement

A needle probe thermistor was introduced into the filter insert of the pneumotachograph and a filter cigarette attached. On five occasions successive simulated square-wave puffs of cigarette smoke (35 ml) were taken at 1 min intervals until the burning end of the cigarette reached the filter. While most of a cigarette was 'smoked' only small increases (<2°C) in insert temperature occurred and there were no detectable changes in the recorded puff profiles. However, as the burning end came within 8 mm of the filter, large transient increases in the insert temperature (8-10°C) were associated with disturbances superimposed on the normal square-wave profile.

Lung volume

The linearity of the box-spirometer system was assessed over a range of volume changes normally seen during smoking studies. Two subjects were separately enclosed in the box and connected to a wedge spirometer at the mouth. They were instructed to breathe at different tidal volumes whilst keeping the respiratory frequency between
10 and 12 breaths/min. A linear regression analysis of 45 paired comparisons yielded the following relationship: box volume = (0.98 x mouth volume) + 0.01 litres (r = 0.99). The 1% underestimate at tidal volumes of 1 litre by the box-spirometer system could be due either to unavoidable leaks, or to the fact that the wedge spirometer becomes warmer than the box-spirometer during rebreathing.

The frequency response of the box-spirometer was investigated with a subject positioned in the box and breath-holding. A 1 litre syringe was used to simulate breathing, with a normal tidal pattern, over a wide range of frequencies (5-80 breaths/min). Up to 15 breaths/min, the overestimation error in volume was less than 2%; up to 30 breaths/min, this increased to around 5%. Above 30 breaths/min there was a marked and progressive decrease in measured volume. The entire system could discriminate changes of ± 5 ml.

Nasal temperature during smoking

By studies on a model and by careful positioning of the nasal thermistor within the nares it was found possible to be certain of the existence and direction of volume flow through the nose, but only over the first 0.5 litre of an inspiration or expiration. Over this degree of volume change it was found that on average, 20 ml of nasal airflow produced a detectable change in temperature.

Measurement and statistics

Measurements were made from records on a Mingograph (Elema-Scholander) to an accuracy of 0.5 mm; this was equivalent, at worst, to 5% of the signal. Neither puff nor inhalation volumes were corrected to BTPS. Statistical analyses were performed by using an Analysis of Variance computer package, producing means, the F variance ratio statistic and Fisher's Least Significant Difference [5]. The conventional level of statistical significance has been taken as P<0.05.

Results

The subjects appeared to be comfortable during the study with a mean resting ventilation of 7.2 ± 1.8 litres/min (± SD) and a mean resting heart rate of 81 ± 13 beats/min.

Pattern of smoking

Typical records from two individuals, (i) and (ii), are shown in Fig. 2; one puff is recorded with corresponding changes in lung volume. Puff flow profiles produced by each subject were reproducible. Puff volume is similar in these two subjects despite markedly different profiles.

Both examples show that puffing of cigarette smoke occurs at a relatively constant level of lung volume, with the inhalation of tobacco smoke (inspiration and expiration) occurring after the puff is completed. The pattern of inhalation, however, is quite different. In (i), the subject inhales to a volume equivalent to tidal volume but this is maintained over a period of 8 s; inhalation is from, and returns to, the normal end-expiratory level. In (ii), the subject inhales a volume twice the size of a typical tidal breath but the duration remains similar; in this case inhalation is from, but goes back to below, the level of end-expiration.

Closer scrutiny of lung volume at a gain equivalent to puff volume is possible by examination of the expanded lung volume trace. In (i), the subject shows an expiration of approximately 200 ml during the 50 ml puff with little further change between the end of the puff and the beginning of the inhalation. On the other hand, in (ii), the subject shows relatively little change during the 55 ml puff but does expire a volume of 80 ml, before inhaling.

An indication of the partition of airflow between nose and mouth during smoking is given by the recording of nasal temperature. During normal respiration both subjects show evidence of nasal breathing. In (i), both inspiration and expiration after the puff occur in the absence of nasal temperature changes, indicating that this subject adopts mouth breathing for this manoeuvre. In (ii), there is evidence of nasal breathing after the puff, in conjunction with both the pre-inhalation expiration and the inhalation itself. However, for the inhalation the temperature fluctuations are less than those during tidal breathing, suggesting that some portion of the inhalation occurs through the mouth. Major inter-subject differences in partition of airflow during inhalation were found; four subjects inhaled almost exclusively via the mouth (despite regular nasal breathing), four inhaled predominantly through the nose, and the remaining two demonstrated variably both types of behaviour.

For purposes of analysis, the smoking patterns of the ten subjects in this study have been characterized in terms of the following variables: number of puffs, interval between puffs, puff volume, puff duration, change in lung volume during the puff (commonly expiration), change in lung volume immediately after the puff (commonly expiration), inhaled volume, duration of inhalation and exposure index (area under inhalation curve). Since changes in nasal temperature provided only
a qualitative index of the route of airflow during smoking, they have not been included in the analysis.

*Repeatability of smoking patterns*

For each variable, within-subject and within-cigarette consistency of smoking behaviour has been assessed by using an analysis of variance on the first five and last five puffs of each cigarette (excluding the lighting puff). Statistically significant differences were tested for, with respect to: visit number \((n = 4)\), stage (i.e. beginning/end) of cigarette \((n = 2)\) and number of puffs within each stage \((n = 5)\). The results are summarized in Table 1; for all variables the mean levels derived from all
TABLE 1. Group results

Group mean levels of smoking variables with SD in parentheses (n = 614), and subdivided by (a) occasion studied (1–4) and (b) beginning (1) and end (2) of cigarette. VEDP, Volume expired during the puff; VEAP, volume expired immediately after the puff. *Statistically significant differences according to Fisher's Least Significant Difference (LSD) at $P = 0.05$.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall mean (SD)</th>
<th>(a) Visit no.</th>
<th>(b) Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1  2  3  4</td>
<td>1  2</td>
</tr>
<tr>
<td>Puff interval (s)</td>
<td>25.9 (14.5)</td>
<td>25.1 28.5 25.5 27.5</td>
<td>23.1 * 30.2</td>
</tr>
<tr>
<td>Puff volume (ml)</td>
<td>44.2 (13.7)</td>
<td>47.9 39.7 43.9 46.1</td>
<td>46.7 * 42.0</td>
</tr>
<tr>
<td>Puff duration (s)</td>
<td>1.88 (0.91)</td>
<td>2.02 1.95 1.86 1.90</td>
<td>2.20 * 1.66</td>
</tr>
<tr>
<td>VEDP (ml)</td>
<td>48.7 (13.0)</td>
<td>47.5 60.4 53.8 43.9</td>
<td>31.5 * 71.4</td>
</tr>
<tr>
<td>VEAP (ml)</td>
<td>49.4 (63.6)</td>
<td>45.9 48.7 52.9 50.3</td>
<td>45.1 * 53.8</td>
</tr>
<tr>
<td>Inhaled volume (ml)</td>
<td>614 (358)</td>
<td>632 628 601 622</td>
<td>625 617</td>
</tr>
<tr>
<td>Inhaled duration (s)</td>
<td>4.3 (2.3)</td>
<td>4.34 4.36 3.84 4.28</td>
<td>4.19 4.22</td>
</tr>
<tr>
<td>Exposure index (l/s)</td>
<td>1.83 (2.24)</td>
<td>1.94 1.90 1.56 1.93</td>
<td>1.83 1.84</td>
</tr>
</tbody>
</table>

Puffs are very close to those from the selected ten puffs. With the exception of puff volume on visit 2 none of the variables investigated differed significantly in the group for any visit. Considering all studies five variables were statistically significantly different over the last five puffs compared with the first five. Thus, in smoking a cigarette, these subjects showed a progressive reduction in puff volume, duration and frequency. In addition, during the early puffs they expired a significantly lower volume between the puff and inhalation. There were no associated changes in the inhalation pattern as cigarettes were smoked.

The between-subject variability in smoking behaviour is presented in Fig. 3 and Table 2. Fig. 3 shows how these ten subjects differed in their intake of cigarette smoke into the mouth (i.e. that which was potentially available for delivery to the lungs). Individual differences in puff volume (PV) can be compensated for by the number of puffs taken (P/C) such that the total intake ($\Sigma$PV) is similar (subjects 1 and 3).

Conversely individuals with similar puff volumes may exhibit markedly different levels of smoke intake as a result of the number of puffs taken (subjects 5 and 8). When the number of cigarettes per day (C/D) is considered, smoke intake per day ($\Sigma$PV/D) can vary by as much as 20-fold (subjects 4 and 9).

Table 2 details the individual differences in the mean levels of the descriptors of smoking. Except for the volume expired during the puff, inter-subject variability and these differences are highly significant. This means that individuals differ consistently in their smoking pattern.

In general there is a relationship between puff duration and puff volume ($r = 0.74$), although puff volume and puff interval are not correlated ($r = 0.14$). The extent to which different subjects exhaled during, and immediately after, the puff could, on average, be greater (subject 6) or less (subject 9) than the corresponding puff volume.

The variability in the exposure of the lung surface to tobacco smoke is shown in terms of inhalation volume and duration, and an integral of the two, exposure index. All inhalation volumes were larger than the predicted anatomical dead space though some (subjects 9 and 10) were only about twice this dead space. The puff volume is diluted by the volume that is inhaled. Thus for similar puff volumes (45 ml and 48 ml respectively in subjects 8 and 9), the degree of dilution is 1:27 for subject 8, and 1:7 for subject 9; however, subject 8 holds the inhalation in for much longer than does subject 9.

All mean measurements (n = 4) of the first five and last five puffs for each subject in the form of an analysis of variance, have been deposited as Clinical Science Table 83/2 with the Librarian, the Royal Society of Medicine, 1 Wimpole Street, W1M 8AE, who will issue copies on request.

Smoking patterns and absorption

Each subject’s increases in alveolar CO concentration, and heart rate, as a result of smoking one
TABLE 2. Individual results

Mean values of smoking variables over four visits for each subject. *P*, Calculated probability on *F* statistic; the null hypothesis is that, as a group, the subjects are not different. LSD, Least Significant Difference between individuals at a level of *P* = 0.05. VEDP, Volume expired during the puff; VEAP, volume expired immediately after the puff.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subject no.</th>
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<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Puff interval (s)</td>
<td>29.2</td>
<td>47.3</td>
<td>30.9</td>
<td>26.4</td>
<td>33.6</td>
<td>19.3</td>
<td>31.2</td>
<td>13.6</td>
<td>9.6</td>
<td>25.1</td>
<td>&lt;0.0001</td>
<td>7.7</td>
<td></td>
</tr>
<tr>
<td>Puff volume (ml)</td>
<td>35.2</td>
<td>55.9</td>
<td>50.6</td>
<td>26.0</td>
<td>43.3</td>
<td>43.6</td>
<td>42.8</td>
<td>44.3</td>
<td>50.7</td>
<td>52.3</td>
<td>&lt;0.0001</td>
<td>8.8</td>
<td></td>
</tr>
<tr>
<td>Puff duration (s)</td>
<td>1.63</td>
<td>2.18</td>
<td>3.20</td>
<td>0.87</td>
<td>1.53</td>
<td>1.83</td>
<td>2.09</td>
<td>1.86</td>
<td>1.83</td>
<td>2.32</td>
<td>&lt;0.0001</td>
<td>0.36</td>
<td></td>
</tr>
<tr>
<td>VEDP (ml)</td>
<td>57.4</td>
<td>41.7</td>
<td>26.1</td>
<td>58.0</td>
<td>101.7</td>
<td>72.9</td>
<td>79.3</td>
<td>6.5</td>
<td>9.2</td>
<td>54.8</td>
<td>0.30</td>
<td>37.4</td>
<td></td>
</tr>
<tr>
<td>VEAP (ml)</td>
<td>7.7</td>
<td>97.3</td>
<td>19.8</td>
<td>31.3</td>
<td>62.4</td>
<td>162.6</td>
<td>41.0</td>
<td>40.4</td>
<td>46.8</td>
<td>6.4</td>
<td>&lt;0.0001</td>
<td>16.4</td>
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<tr>
<td>Inhaled volume (ml)</td>
<td>749</td>
<td>1136</td>
<td>630</td>
<td>396</td>
<td>618</td>
<td>389</td>
<td>505</td>
<td>1028</td>
<td>299</td>
<td>358</td>
<td>&lt;0.0001</td>
<td>94</td>
<td></td>
</tr>
<tr>
<td>Inhaled duration (s)</td>
<td>5.1</td>
<td>5.5</td>
<td>5.7</td>
<td>1.7</td>
<td>3.8</td>
<td>3.2</td>
<td>2.5</td>
<td>7.3</td>
<td>2.8</td>
<td>4.6</td>
<td>&lt;0.0001</td>
<td>0.47</td>
<td></td>
</tr>
<tr>
<td>Exposure index (I * s)</td>
<td>2.9</td>
<td>4.1</td>
<td>2.4</td>
<td>0.4</td>
<td>1.3</td>
<td>0.7</td>
<td>0.7</td>
<td>4.1</td>
<td>0.6</td>
<td>1.2</td>
<td>&lt;0.0001</td>
<td>0.64</td>
<td></td>
</tr>
</tbody>
</table>

**FIG. 3.** Mean subject levels of smoking variables taken over four visits. All bars are shown when values are derived from all puffs (n = 39 to n = 91), but not when values are the mean of four measurements. Subjects nos. 1, 4, and 6 smoked low-tar cigarettes; the others smoked middle-tar brands.

**FIG. 4.** Increase in alveolar carbon monoxide concentration (CO boost) and heart rate (AHR) in each subject after smoking one cigarette. Pre = mean values before smoking; Subject number. Pre-CO = subjects 1, 2, and 3; Pre-HR = subjects 2, 4, and 6; AHR = subjects 1, 2, and 3. Missing values are those from technically unsatisfactory measurements. 

Patterns of smoking
cigarette are shown in Fig. 4. A stepwise-inclusion multiple regression analysis (SPSS) with CO boost as the dependent variable fitted the smoking variables in the following order, with (a) simple, and (b) multiple correlation coefficients shown: number of puffs (a) -0.31, (b) 0.31; puff volume (a) 0.21, (b) 0.37; exposure index (a) -0.15, (b) 0.42; inhalation duration (a) -0.14, (b) 0.46; volume expired during the puff (a) -0.07, (b) 0.49. Puff duration, volume expired after the puff and inhaled volume were not fitted by the programme since they caused no significant improvement in the correlation. At no stage did either the simple or multiple correlation coefficients reach the level of statistical significance. An analogous multiple regression analysis with change in heart rate as the dependent variable fitted the smoking variables as follows: number of puffs (a) -0.29, (b) 0.29; puff volume expired after the puff (a) -0.23, (b) 0.39; inhaled volume (a) 0.18, (b) 0.45; inhalation duration (a) -0.10, (b) 0.61. The remaining variables were not fitted. In this case the multiple correlation coefficient just achieved statistical significance after the fourth step. There was no overall correlation between these two indices of absorption \(r = 0.37\), not significant). In addition, inspection of Fig. 4 does not show any obvious relationship between two variables when looked at on an individual basis.

**Perception of degree of inhalation**

Subjects' assessment of their degree of inhalation ranged from a score of 35 to 100% on the visual analogue scale. There was, however, no significant correlation with the measured mean inhaled volume (range 0.3-1.1 litres; \(r = 0.04\)). Normalization of inhaled volume for vital capacity made no impression on this lack of correlation \(r = 12.6 \text{ to } 26.2\%\); \(r = 0.06\).

**Discussion**

It is well recognized that, in observing smoking behaviour, an experimenter may affect that which he is trying to measure. It could be argued that this is particularly the case in the present study where the attempt at getting adequate sensitivity of methods has necessitated an unnatural smoking environment. The general opinion of the subjects was that 'smoking' under such artificial conditions did not differ greatly from their usual experience. The attempt at diversion by reading, and listening to music, must have played some part in this. There was no sense of resistance to breathing, nor did the addition of the pneumotachograph subjectively increase the sense of resistance normally offered by a filter cigarette. The normal resting ventilations and heart rates suggested that the subjects were relaxed.

Although the dead space of the holder was small with the insert effectively increasing the length of the filter rod by only about 30%, these additions may have affected delivery of smoke constituents, causing compensatory changes in smoking patterns. However, none of our subjects commented on differences in ‘taste’ or ‘satisfaction’ when smoking a cigarette under experimental conditions. Moreover, the fact that subjects were somewhat constrained, aware they were being observed and obliged to handle the holder rather than the cigarette, may have interfered with their ‘natural’ smoking behaviour; we have no way of knowing to what extent different individuals were affected in this respect.

Our results on the basic smoking variables of puff volume and duration, interval between puffs, inhalation volume and duration do not differ appreciably from values in the literature [6-13]. Most other studies in which puffing characteristics have been quantified have employed a modified cigarette-holder as in the present study. Recently, Tobin & Sackner [6] have suggested that such an approach results in an increased puff volume and frequency when compared with measurements made using an inductance device around the cheeks. However, our mean levels of puff volume agree closely with those made by these authors using their inductance device and although the average number of puffs was higher, others have reported both low frequencies [14] and high frequencies [7] with a cigarette-holder pneumotachograph. The inhalation volumes and durations reported by Tobin & Sackner [6] using a less-constraining chest pneumogram are similar to those observed here with plethysmography; measurements made with magnetometry [8] are also in close agreement. Although the techniques used in the present study permit exact quantification of nuances in smoking behaviour, there is no evidence that the results obtained differ markedly from those where less precise methodology has been employed.

The most significant aspect of this study was that it has been possible to show that patterns of smoking are relatively repeatable within subjects and yet considerably different between subjects. This conclusion is true both for puffing and inhalation behaviour, which could always be identified as discrete events. A further significant finding was that, as a cigarette is smoked, puffing became less intense whereas inhalational behaviour remained unchanged. These results confirm and
extend the observations of others who have shown decreases in puff frequency [14] and puff duration [15] with decreasing length of unsmoked cigarette. This may reflect a smoker's tendency to compensate for the increased concentration of tobacco smoke components delivered as the burning end approaches the mouth or it may be a response to the initial satisfaction achieved in the early puffs.

It is clear from these findings that the smoke intake to which smokers subject themselves, depends not only on cigarette consumption but also on the number of puffs taken from a cigarette and the volume of each puff. This fact has also been recognized by others [16–18]. The interaction between lung volume changes and smoke intake during smoking is complex but probably important in the delivery of smoke to the lungs. A larger inhaled volume will both increase the dilution of tobacco smoke and distribute it to a larger exchange area; theoretically these constitute opposing effects on the degree of absorption by diffusion of materials such as nicotine and carbon monoxide. On the other hand a small inhalation, sufficient to cover the anatomical dead space, would tend to maximize airways deposition of tobacco smoke components. An increased duration of inhalation will allow increased time for airway deposition or absorption from the alveoli.

To investigate the effect of smoking patterns on gaseous absorption, a pilot study was performed in two subjects ‘puffing’ known volumes of 5% CO in air (equivalent to the concentration in cigarette smoke) with sequential known inhalation volumes and durations (R. Rawbone & L. Adams, unpublished work). The amount of CO absorbed (CO boost) was linearly related to the puff volume and primarily dependent on it. The duration of inhalation also had a positive influence on the CO boost but this factor was of much less importance than the puff volume. The depth of inhalation, always greater than the dead space, had no influence.

The inability in this study to predict CO absorption from a quantified smoking pattern may be a reflection of both the failure to define all important smoking variables and the uncertainty of how these variables interact to promote or minimize absorption. For the index of nicotine absorption (change in heart rate) a significant 38% (i.e. $r^2$) of the variability can be accounted for by four of the smoking variables. Surprisingly, however, puff volume is not included and the number of puffs shows an inverse relationship. Although it was found that an individual smoked in a consistent way, the measured increases in alveolar CO concentration and heart rate from smoking one cigarette were both variable between studies, and uncorrelated with one another.

There is some degree of uncertainty in normalizing expired CO measurements for cigarette yields under 'machine' conditions, which themselves may poorly reflect actual delivery; however, such corrections were small and the outcome was identical when they were not applied. Equally, the measurement of an increase in heart rate as an indirect assessment of nicotine absorption from one cigarette lacks specificity and makes interpretation of these results difficult.

Ashton et al. [19] have shown that 'trough' (pre-cigarette) levels of carboxyhaemoglobin correlate better with absorption from one cigarette (assessed by increase in blood nicotine levels) than boost levels. Pre-test mean alveolar CO levels obtained in the present study were measured after various periods of cigarette deprivation (sometimes overnight) and could therefore not be used to assess absorption. Similarly, Sutton et al. [16] have found a better correlation between total smoke intake from a cigarette and plasma nicotine than with the level of carboxyhaemoglobin after smoking; although these authors did not consider pre-smoking levels, they argue that carboxyhaemoglobin levels provide a relatively insensitive index of absorption.

Smokers' estimates of the extent of inhalation obtained during population studies do not correlate with the severity of smoking-induced diseases [20]. Recently Stepney [21] has shown that such estimates correlate poorly with increases in alveolar CO levels. In itself this is not surprising since there is preliminary evidence that measured depth of inhalation has little influence on absorption (see discussion above). However, both the present study and that of Tobin & Sackner [6] have shown that perceived inhalation is an unreliable estimate of actual inhalation. In this respect, its use in studies of smoking habits is of questionable value.

The relative health risk of smoking has usually been considered in terms of cigarette consumption and the tar/nicotine yields that these cigarettes produce under standard conditions. Health education aimed at reducing the former has been largely unsuccessful and attempts to reduce the latter are felt by many to be ineffective because of smokers' tendency to compensate for reduced yields by adopting a 'more intense' pattern of smoking [22–24]. So far, little attention has been paid to the role of an individual's smoking pattern as a determinant of the aetiology of smoking-related disease. It is apparent from the present work that, in a random group of young asymptomatic smokers, patterns of smoking are markedly and
consistently different. Although these patterns may affect long-term clinical risk, it is unlikely that the prospective study necessary to confirm this could be realized.

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References