Passive expiration as a test of lung function¹

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Ashutosh, K, and Keighley, J F (1978). Thorax, 33, 740–746. Passive expiration as a test of lung function. Twenty-five normal subjects, 14 non-smokers and 11 smokers, passively expired into a spirometer after a maximal active inspiration, and after a passive inflation of the chest by a pressure cycled intermittent positive-pressure breathing (IPPB) machine. Acceptable passive expirations could be performed by all subjects after a passive inspiration but by only 12 after an active inspiration. Expired volume was found to change exponentially with time (r > 0.98), and the time constant of passive expiration (T_D) was obtained. There was no significant difference between the smokers and non-smokers in age, sex, forced vital capacity, FEV₁, FEV₁/FVC %, maximum mid-expiratory flow rate, maximum expiratory flow at 50% and 25% of the vital capacity, or the magnitude of the fall in the dynamic compliance with increasing frequency of breathing (Cdyn/f). T_p in smokers (1.06 s \pm 0.47 SD) was significantly longer than in the non-smokers (0.65 s \pm 0.25 SD P < 0.02). T_p had a significant correlation with $Cdyn/f(T_p = 0.6 + 161.81 Cdyn/f \pm 0.38 SE, r = 0.49$, P < 0.02). We conclude that satisfactory passive expiratory spirograms can be easily obtained after a mechanically assisted passive inspiration. T_p thus obtained is determined by the intrinsic properties of the respiratory system (lung plus thorax), and is significantly prolonged in smokers compared with non-smokers when other studies of pulmonary function including frequency dependence of compliance are unchanged.

Passive expiration, defined as the expiration when the lungs and thorax are allowed to return to functional residual capacity (FRC) by their own elastic recoil and no active muscular effort is applied, has been shown to be determined solely by the intrinsic properties of the respiratory system (DuBois and Ross, 1951). Although found to be a useful method of measuring total respiratory (lung+chest wall) compliance and resistance (Brody et al, 1960), it has not been widely utilised because of the difficulty in obtaining a passive expiration in unanaesthetised, untrained individuals (Agostoni and Mead, 1964).

We found that passive expirations were more consistently and easily obtained if preceded by a passive inflation of the chest by an intermittent positive-pressure breathing machine (IPPB). The present study was undertaken to determine the practicality of obtaining passive spirograms in untrained individuals by the above method. We also tested the ability of the passive spirogram to detect early abnormalities in lung function compared with other pulmonary function tests including frequency dependence of compliance and flow-volume curves.

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Methods

Twenty-five normal healthy volunteers were selected for the study. Four subjects, two of whom were smokers, had a mild morning non-productive cough. None had any known pulmonary illness at the time of the study. All had had a normal expiratory spirogram and a normal chest radiograph within one year of the study. Fourteen were non-smokers (men 8, women 6) and eleven were smokers (men 8, women 3). The difference in the age and sex composition between the smokers and non-smokers was not significant (see table).

Subjects reported to the laboratory in a fasting state. Simultaneous forced expiratory spirograms and expiratory flow volume curves were obtained with a low resistance wedge spirometer (Med Science Electronics—Wedge Spirometer Model 370), with electronic output fed into two X-Y plotters (HP X-Y Recorders, Model 7046, Hewlett Packard Co, Waltham, Mass).

Next, a 10 cm long latex oesophageal balloon attached to the end of a 100 cm polyethylene tube having multiple holes in its distal 9 cm was passed into the distal one-third of the oesophagus through

Table Results (mean \pm 1SD) of selected tests in smokers and non-smokers

| | Smokers | Non-smokers | Student's two-tailed t test |
|---|------------------|-----------------|-----------------------------|
| No of subjects | 11 | 14 | |
| Age (years) | 30.27 ± 8.88 | 25·64±4·72 | NS |
| FEV ₁ (l) | 3.43 ± 0.80 | 3.58 ± 0.57 | NS |
| FVC(I) | 4.31 ± 1.07 | 4.16 ± 0.68 | NS |
| FEV ₁ /FVC (%) | 80.36 + 8.84 | 84·93±7·05 | NS |
| FEF ₂₅₋₇₅ % | 3.59 ± 1.26 | 4·21±1·09 | NS |
| V max ₅₀ (1/sec) | 3.78 ± 1.31 | 4·45±1·28 | NS |
| V max ₂₅ (l/sec) | 1.56 ± 0.64 | 2.02 ± 0.72 | NS |
| PEV ₁ (Î) | 1.10 + 0.24 | 1.19 + 0.27 | NS |
| TP (s) | 1.06 ± 0.47 | 0.65 ± 0.25 | P < 0.02 |
| T _A (s) | 0.87 + 0.54 | 0·54±0·16 | NS |
| Co (l/cm water) | 0.22 ± 0.10 | 0.23 ± 0.08 | NS |
| Cdyn/f (l/cm H ₂ O/sec ⁻¹) | 1·7 ±1·3 | 1.2 ± 1.3 | NS |

NS=Not significant. For abbreviations, see text.

the mouth or nose. The proximal end of the polyethylene tube was connected to a pressure transducer, and the pressure was recorded on a multichannel electronic recorder (HP Sanborn Recorder, Hewlett Packard). The final position of the balloon was that which showed the least amount of cardiac artifact and maximum deflections in the pressure signals during respiration.

With the oesophageal balloon in position, the passive spirogram was recorded by two different methods: (1) the subject inspired maximally and at end-inspiration suddenly relaxed his chest muscles, glottis, and pharynx, and let his chest deflate passively; (2) a Bennett PR2 IPPB machine (Puritan Bennett Co, Berkeley, California) was set at a pressure of 20 cm of water and was connected to the subject through a mouthpiece. The subject sealed his lips tightly around the mouthpiece, and the system was checked for any leaks. The subject initiated an inspiration and then immediately relaxed his respira. ry muscles, pharynx, and glottis as completely as possible, and allowed the machine to force air into his chest at the preset pressure. At the end of the positive-pressure inspiration he continued to relax and allowed his chest to deflate passively without using any effort. Thus in this method both inspiration and expiration were passive. During these manoeuvres simultaneous intraoesophageal pressure recordings, volume-time spirograms, and flow volume curves were obtained by connecting the expiratory line of the IPPB machine to a wedge spirometer-X-Y recorder system as in the case of the active spirogram.

Two criteria were used to determine whether the expirations were completely passive. One was a rapid (within 0.4 seconds from the onset of the expiration) rise or fall of the intraoesophageal pressure to the pressure previously recorded at the FRC, usually -1 or -2 cm of water. The second criterion was a linear expiratory limb of the flow

volume curve of the spirogram. We analysed only those expirations that satisfied both the criteria, except in the one instance when oesophageal pressure was not recorded (figs 1 and 2).

To measure the frequency dependence of compliance, the oesophageal pressure and the air flow at the mouth were recorded on a multichannel (Sanborn Div, Hewlett-Packard Co, Waltham, Mass) recorder, and the volume signal was obtained by integrating the flow signal from a pneumotachometer. The subjects, who were asked to breathe at different frequencies (f) with tidal volume in the range of 0.6 to 1.2 l, inspired to the maximum inspiratory capacity and then expired to FRC between breathing at different frequencies. An effort was made to keep the end expiratory lung volume constant by instructing the patient to control his breathing so as to keep the end-expiratory point in the volume tracing at the same level. Neither the volume nor the endexpiratory point, however, could be kept completely identical at all the breathing frequencies. Compliance at different frequencies was measured by dividing the change in volume by the change in pressure between two zero flow points in each respiratory cycle. Later, we used a computer channel (HP 8816A—Respiratory Analyzer, Hewlett Packard) that directly recorded dynamic lung compliance at each breathing frequency. Readings of compliance obtained by the computer were always randomly checked, at least once in each breathing frequency, against the value obtained by dividing volume by pressure change as displayed in the volume and pressure recording. Frequency dependence of compliance could not be measured in one subject because of her inability to swallow the oesophageal balloon.

Age, sex, and smoking history in pack years were recorded. Forced vital capacity (FVC), FEV₁, maximal mid-expiratory flow rate (FEF₂₅₋₇₅%), and FEV₁/FVC% were calculated from the active spirogram. Maximum flow at 50% of vital capacity

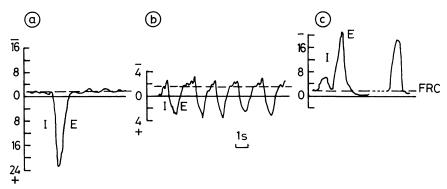


Fig 1 Examples of oesophageal pressure recordings in acceptable passive expirations. Time is recorded along X axis and pressure (cm of H_2O) is displayed on Y axis. I and E denote inspiration and expiration respectively. (a) and (b) were obtained by method of passive inspiration and (c) after active inspiration. Note rapid change of oesophageal pressure to FRC.

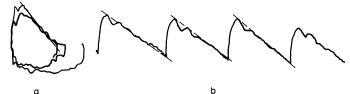


Fig 2 Flow volume curves of passive expiration: (a) is after an active inspiration while (b) is after successive passive inspirations.

(V max₅₀) and at 25% of vital capacity (V max₂₅), and peak flow (V max) were obtained from the maximum expiratory flow volume curve. Passive expired volume in one second (PEV₁) was obtained from the passive expiratory spirogram.

The active and passive spirograms were replotted on a semilogarithmic paper, the time on the X axis, and the unexpired volume as a percentage of the total expired volume

total expired volume - expired volume to time t%

total expired volume

on the Y axis at 0.2 second intervals.

The change in expired volume was exponentially related to time $(r \ge 0.98)$. The relationship between the expired volume and time is described by the equation $V_t = V_0 e^{-t/T}$ where $V_t =$ unexpired volume at a given time t, V_0 the initial volume and T = the time constant of the total respiratory system (lung+thorax). At the instant when t = T, $V_T = V_0 \cdot e^{-1} = V_0 \cdot 37$ ($V_T =$ unexpired volume up to the instant when t = T). Therefore, the time at which the unexpired volume falls to 0.37 of the original value equals the time constant. Time constants of active expiration (T_A) and passive expiration after a maximal inspiration (T_{pmax}) and after a passive inspiration by IPPB device (T_p) were thus calculated.

Dynamic compliance on Y axis was plotted against different breathing frequencies on X axis. The regression line extrapolated to zero yielded the estimated compliance of the lung at zero f (CO). The slope of the regression line (Cdyn/f) was taken as the measure of the frequency dependence of compliance.

Results

All subjects produced many successive, satisfactory passive expirations after a few trials when the inspiration was passive and accomplished by IPPB. On the other hand, it was difficult for them to relax adequately for an acceptable passive expiration when the inspiration was active. Thirteen subjects (52%) could not produce a single satisfactory passive expiration after active inspiration, and many subjects could learn to relax sufficiently only after many trials. Passive expirations after passive inflation of the chest by IPPB always met the criteria for adequate passive expirations if the multiple spirograms thus obtained were fully superimposable.

The intraoesophageal pressure at the end of a passive inflation of the chest ranged between +4 and +10 cm H₂O in most subjects. In one subject the end-inflation pressure rose to 22 cm H₂O, probably from the subject resisting the positive pressure

inflation by contracting his thoracic muscles (fig 1a). Nevertheless, the following expiration still met the criteria for an adequate passive expiration.

Subjects were divided into two groups—smokers and non-smokers. The table summarises the results of selected tests in the two groups.

Time constant of passive expiration (T_p) was significantly prolonged in smokers compared to the non-smokers (P < 0.05, table). Figure 3(a) shows that 73% of the smokers and only 21% of the non-smokers had a T_p of 0.7 or more (P < 0.05). Mean Cdyn/f was also lower in the smokers than in the non-smokers, but the difference was not statistically significant (see table).

No other difference between the two groups was observed.

The relation between dynamic compliance and respiratory frequency was linear (Mean r=0.86) except in one subject in whom the dynamic compliance at highest breathing frequency was unchanged from that at $f \le 10$.

 T_p and Cdyn/f showed a significant correlation $(T_p=0.60+161.81 \text{ Cdyn/f}\pm0.38 \text{ SE}, r=0.49, p<0.02)$ (fig 4).

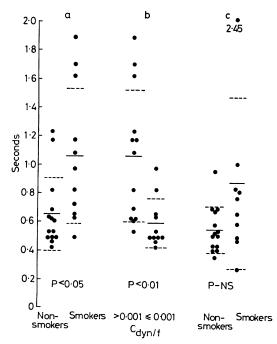


Fig 3(a) Time constant of passive expiration (T_p) in smokers and non-smokers; (b) T_p in subjects with Cdyn/f of more, and less than 0.001; and (c) Time constant of active expiration (T_A) in smokers and non-smokers. Mean and ± 1 SD are shown by solid and broken lines respectively.

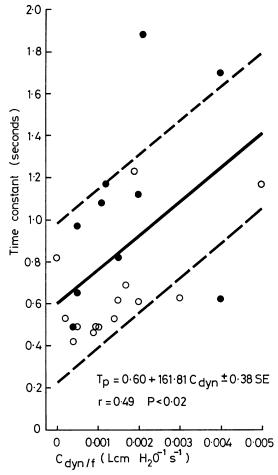


Fig 4 Correlation between T_p and Cdyn/f. Regression line and \pm 1 SE are shown. Closed circles denote smokers and open circles non-smokers.

Eleven subjects had Cdyn/f of equal to or less than 0.001, and 13 had values larger than 0.001. Although a higher proportion (54%) of subjects with Cdyn/f >0.001 smoked than of those with Cdyn/f \leqslant 0.001 (27%), the difference was not significant. T_p was significantly longer in the subjects with Cdyn/f >0.001 (1.06 s \pm 0.46 SD) than in the group with Cdyn/f \leqslant 0.001 (0.59 s \pm 0.17 SD, p <0.01). Figure 3(b) shows that 54% of the subjects with Cdyn/f of more than 0.001 and none of the subjects with Cdyn/f of 0.001 or less had a T_p greater than one second (p <0.025). Similarly, of the 17 subjects with $T_p > 1$ s 65% had Cdyn/f of 0.001 or less. Of the seven subjects with $T_p > 1$ s none had a Cdyn/f of 0.001 or less.

No significant difference was noted between T_p and T_{pmax} . Although mean time constant of active

expiration (T_A) was shorter in both smokers (0.87 s \pm 0.59 SD) and non-smokers (0.54 s \pm 0.16 SD) compared to T_p , the difference between T_A and T_p was not significant (fig 3(c)).

Discussion

Free collapse of the lung, that is, when the lungs deflate solely by the force of their elastic recoil, has been shown to reflect the intrinsic properties of the respiratory system. A passive expiration, when no muscular force is applied, can be used as a model reflecting free collapse of the lung plus thorax (Ebert and Haddy, 1954; Nissel and DuBois, 1954). Dixon and Brodie (1903) were first to use free collapse of the lung in animal experiments to show changes in lung mechanics. Since then passive expiration has been studied in isolated lungs (Nissel, 1948), anaesthetised human subjects (Newman et al, 1959), and trained volunteers (Otis et al, 1950), and has been recommended as a means for measuring total compliance and resistance of the lung plus thorax in man (Comroe et al, 1954).

It can be shown by mathematical analysis that during a passive expiration the change in volume in relation to time is exponential, and the ratio between the change in volume and the change in time at any given instant must be constant and equal to the product of the total respiratory (lungs+chest wall) resistance and compliance, that is, the time constant. Furthermore, the volume time course of a passive expiration is determined solely by the volume elastic and flow resistive characteristics of the respiratory system, and it has a fairly constant half-time predictable on the basis of the above properties (Pierce, 1959; McIlroy et al., 1963).

Despite the above works confirming the theoretical and experimental validity of the tests based on passive expiration, we are not aware of any study using passive spirometry as a test of pulmonary function in man and evaluating its relative sensitivity in comparison with other tests of the lung function, although the clinical usefulness of passive spirometry in patients with acute respiratory failure was suggested by Ashutosh et al (1975). One reason for this may be the difficulty in obtaining a reliable passive expiration in unanaesthetised subjects. Brody (1954) has shown that a voluntary passive expiration is virtually impossible because of persisting inspiratory tone in the respiratory muscles during a relaxed expiration. This tone of the inspiratory muscles becomes negligible only towards the end of the relaxed expiration if the previous inspiration is an active process. Probably when inspiration is a passive process there will be much less or even absent inspiratory tone during the following 'relaxed' expiration. This would explain the ease with which our subjects could perform satisfactory passive expirations after IPPB inspirations.

The criteria defining an acceptable passive expiration have been previously established by other workers. Pierce (1959) considered a rapid fall of oesophageal pressure to the baseline as denoting a passive expiration. McIlroy *et al* (1963) showed that a linear descending slope of the expiratory flow volume curve would correspond to a completely passive expiration. Both of the above criteria were met in all the acceptable passive expirations in our study.

Because asymptomatic smokers with normal routine pulmonary function tests are known to have more abnormalities compared with normal nonsmokers when more sensitive tests are applied, the ability of a test to detect deviations from normal in smokers is widely considered to reflect its ability to detect early changes in pulmonary mechanics, presumably due to small airways obstruction (Dosman et al, 1975). FEF_{25-75%}, V max₂₅, closing volume, isoflow volumes, and frequency dependence of compliance are some of the tests that have been shown to detect a difference between smokers and non-smokers (McFadden et al. 1974; Knudson et al. 1976) when other pulmonary function test results are normal. On the other hand, some other workers could not detect any difference between smokers and non-smokers on the basis of FEF₂₅₋₇₅%, V max₅₀ and V max₂₅ (Tockman et al, 1967; Marcq and Minette, 1976), and in this respect agree with the results obtained in our subjects.

Frequency dependence of compliance is considered to be the most definitive test for small airways disease and has been the standard test against which all others have been compared (Woolcock et al, 1969). Guyatt et al (1975) pointed out that although all workers agree that dynamic compliance (Cdyn) falls at higher frequency of breathing (f) in the presence of obstructive lung disease, only a few have quantified frequency dependence. Furthermore, the method of expressing frequency dependence of Cdyn has varied with different observers. Mills et al (1963) used the slope of the regression line of Cdyn against f, Woolcock et al (1969) measured the value of Cdyn at the fastest f attained in a subject as a percentage of the static compliance of lung (Cst), McFadden et al (1974) have used the same value but at f of 60/min, while Ingram and O'Cain (1971) divided the values of Cdyn at all the breathing frequencies by Cst, for quantifying the magnitude of frequency dependence of Cdyn. Comparison of Cdyn with Cst may produce inaccuracies because of the difference in the tissue and surface hysteresis of the lung between static and dynamic conditions (Saibene and Mead, 1969).

In view of the uncertainties discussed above, Guyatt et al (1975) considered the best way to quantify the magnitude of the frequency dependence of Cdyn to be some measure of the slope of the regression line of Cdyn against f (Cdyn/f). They analysed four different ways of expressing frequency dependence of Cdyn based on the above approach including the measurement of the slope itself, and found them all to be acceptable.

We have used the slope Cdyn/f to quantify frequency dependence of compliance in our study. The slope was linear in all subjects except in one in whom Cdyn remained unchanged from f < 10 to the highest f attained. The slope in this subject was considered to be 0.

Although there was greater magnitude of frequency dependence of compliance in smokers as compared to non-smokers, the overlap between the two groups was such that no separation between the two groups could be obtained on the basis of this test. Our results are in disagreement with Woolcock et al (1969) who have shown that a complete separation between smokers and non-smokers is possible by this test. Other workers, however, have shown that results from this test may be normal in many subjects who are definitely abnormal by other criteria, and vice versa (Mills et al, 1963; Ingram et al, 1967). Poor reproducibility of this test in the same individuals has also been reported (Guyatt et al, 1975).

If the value of Cdyn/f of 0.001 was taken as the normal lower limit of frequency dependence of compliance, then a significant difference was observed in the values of T_p between the subject having a 'normal' Cdyn/f and those with 'abnormal' Cdyn/f values. This would suggest that both frequency dependence of compliance and time constant of passive expiration measured similar but not necessarily identical functions.

Our results show that T_p derived from a passive expiratory spirogram separated the smokers and non-smokers when $FEF_{25-75\%}$, \dot{V} max₅₀, \dot{V} max₂₅, and T_A were identical, and even the tests for frequency dependence of compliance did not bring about a clear cut separation between the two groups. T_p showed significant correlation with frequency dependence of compliance, higher values of T_p being associated with higher values of Cdyn/f.

We conclude that the time constant of the passive expiratory spirogram is a practical, feasible, and reproducible test in an unanaesthetised, untrained individual if the preceding inspiration is made passive by mechanical assistance. It depends on the intrinsic properties of the respiratory system and is able to detect abnormalities in pulmonary function in smokers when other tests including routine active

In view of the uncertainties discussed above—spirometry, MEFV curves, and frequency dependence upatt et al (1975) considered the best way to of compliance fail to do so.

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