

Breathing muscle activity during expiration in patients with chronic airflow obstruction

M.J. Morris, R.G. Madgwick, A.J. Frew, D.J. Lane

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ABSTRACT: During quiet ventilation in 10 patients with severe chronic airflow obstruction (AFO) there were large tidal swings of pleural pressure, 15.8 ± 5.1 cmH₂O, with high negative pressures achieved, 15.2 ± 5.2 cmH₂O. The pattern of pleural pressure mirrored that of tidal expiratory flow with a rapid rise to maximum of pleural pressure and flow at the beginning of expiration, a slow decline of both throughout most of expiration, and just before the onset of inspiration at flow reversal, a sharp fall in pleural pressure and expiratory flow. The shape of expiratory flow and pleural pressure tracings and the pattern of EMG recordings are compatible with loss of post-inspiratory muscle braking of flow. The generally negative pleural pressure and the EMG silence during expiration indicated relaxation of expiratory muscles throughout expiration. Extrapolation of the tidal expiratory flow curve to zero, and calculation of the area under the extrapolated curve showed the volume of dynamic hyperinflation to be a small proportion of the total increase in functional residual capacity above the predicted value in these patients.

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The knowledge that airways are narrower during expiration than during inspiration, and the audible wheeze during, and the long duration of expiration, have led physicians to the generally held belief that expiration is more difficult for patients with airflow obstruction (AFO) than is inspiration. However, most asthmatics claim the opposite to be true [1].

Previous work has shown tidal expiratory flow to be informative in assessing the presence and severity of AFO [2]. The rapid rise to maximal tidal expiratory flow correlates with other accepted indices of airflow obstruction [2]. We have speculated that this correlation occurs because of progressive loss of post inspiratory braking of flow in patients with increasingly severe AFO, or that it may be related to progressively early onset of flow limitation during tidal expiration as AFO becomes more severe [2].

In this study we have attempted to assess respiratory muscle activity by measuring pleural pressures and taking electromyogram (EMG) recordings in patients with severe AFO. In the literature there are almost no data on pleural pressures during resting ventilation in chronic AFO [3] although there are some in asthma [4, 5] and in histamine induced bronchoconstriction [6]. Similarly, there are very few data concerning EMG activity during resting tidal ventilation in patients with severe chronic AFO [7, 8].

Hyperinflation is a hallmark of AFO and is thought

to contribute to the patient's discomfort. There is no simple bedside technique to measure the volume of hyperinflation. The second part of our study was directed at seeing whether an index of hyperinflation could be determined from the expiratory tidal flow pattern.

Materials and methods

Two normal subjects and 10 patients with clinical and physiological evidence of chronic airflow obstruction (table 1) were studied after informed consent had been obtained. Lung volumes and inspiratory airways resistance were measured during quiet breathing in a Fenyves constant volume body plethysmograph [10, 11]. In six patients functional residual capacity (FRC) was re-measured using oesophageal pressure rather than mouth pressure [12, 13] to represent alveolar pressure and was not found to be different (paired t-test $p > 0.4$).

Tidal and maximum flow volume loops were recorded using flow measured by the Fleisch pneumotachograph and volume obtained by integration of the flow signal, tracings being recorded on a Bryan's X-Y recorder 50000 (fast mode).

An oesophageal balloon [14] was positioned in the lower oesophagus with no evidence of positive pressure on tracheal compression or abdominal contraction and

Table 1. – Lung function measurements

Normal subjects	Age yrs	Sex	Height cm	FEV ₁ l	% pred	VC l	% pred	TLC l	% pred	FRC l	% pred	RV l	% pred	Raw	sGaw	k
PV	29	F	169	3.9	115	4.3	112	5.8	108	3.1	111	1.7	109	1.2	0.270	
RT	30	M	180	4.25	100	5.5	104	8.3	114	4.30	108	2.8	139	1.7	0.139	
Patients																
BB	44	M	179	2.1	50	4.7	94	7.6	107	3.8	96	2.9	133	4.1	0.064	1.015
WS	66	F	159	0.7	35	1.6	58	6.0	142	4.6	180	4.5	266	7.1	0.030	0.711
AG	70	M	160	0.4	20	1.6	57	7.3	152	6.0	200	5.4	267	10.7	0.016	0.275
GP	37	M	173	0.5	15	2.2	49	10.3	160	8.3	229	8.1	420	8.4	0.014	0.623
AD	64	M	175	1.5	53	4.1	101	8.7	136	5.7	152	4.4	188	7.5	0.023	0.492
HF	59	M	175	1.2	41	2.4	59	6.4	100	4.7	126	4.0	172	20.8	0.010	0.344
PJ	52	M	175	0.6	19	2.9	67	9.3	142	7.7	205	6.5	289	8.7	0.015	0.686
MV	45	F	165	0.9	38	2.1	60	7.3	143	5.9	205	5.3	325	11.7	0.014	0.296
HB	56	M	166	0.5	17	1.6	40	8.1	134	6.8	195	6.4	315	15.0	0.010	0.565
JA	44	F	152	1.5	63	2.1	74	4.5	109	2.7	116	2.4	189	9.5	0.039	1.000
Mean	54		168	1.0	35	2.5	66	7.6	133	5.6	170	5.0	257	10.4	0.024	0.601
sd	11		9	0.6	17	1.1	19	1.7	20	1.7	45	1.7	87	4.7	0.017	0.251

FEV₁: forced expiratory volume in one second; VC: slow vital capacity; TLC: total lung capacity; FRC: functional residual capacity; RV: residual volume; Raw: inspiratory airways resistance at FRC (cmH₂O·l·s⁻¹ conversion to SI units, multiply by 0.098); sGaw: specific conductance (l·cmH₂O⁻¹·s, conversion to SI units, multiply by 10.2); k: decay constant of tidal expiratory flow, (s⁻¹). Predicted values from COTES [9].

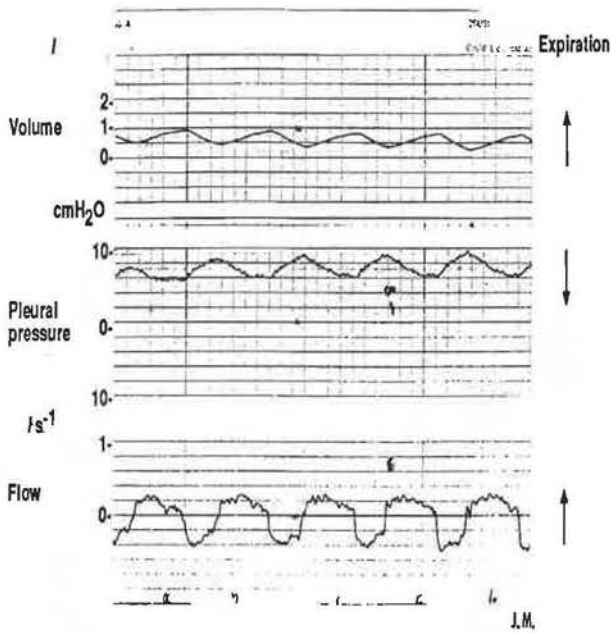


Fig. 1. - Tidal volume, pleural pressure and flow tracings vs time in a normal subject.

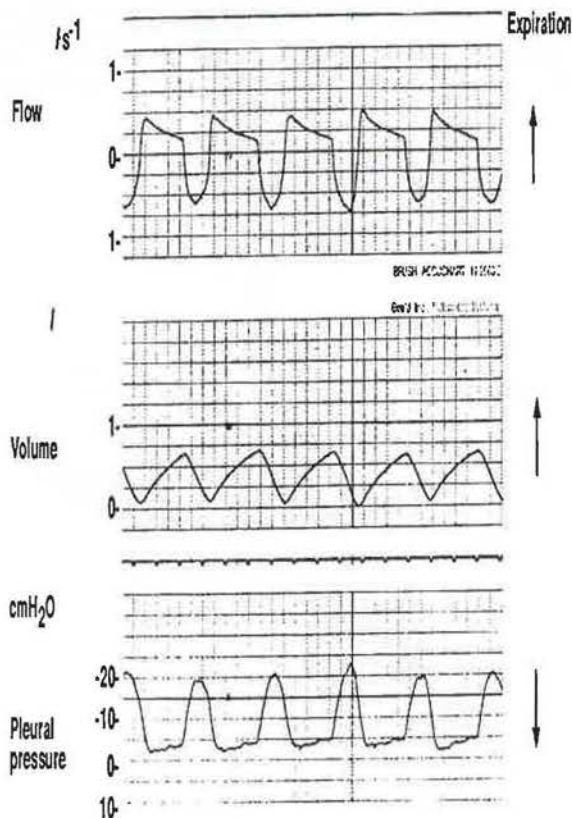


Fig. 2. - Tidal volume, pleural pressure and flow tracings vs time in a patient with airflow obstruction. Similar tracings were seen in all patients in this study.

with minimal cardiac artefact. The gas volume in the balloon was 0.5 ml and was checked frequently. Oesophageal pressure was measured with the mouth/oesophagus (m/oes) body plethysmograph transducer relative to atmospheric pressure (pleural pressure) or to mouth pressure (transpulmonary pressure) [15]. Oesophageal pressure, flow and volume were recorded synchronously on a Gould Brush recorder 260 which had a frequency response flat up to 60 Hz (figs 1 and 2). Pressure and flow recordings were in phase [16] as regards the apparatus and the pens were accurately lined up on the recording paper. The frequency response of oesophageal balloon, tubing, transducer, recorder system was flat up to 5 Hz. The pressure transducer was calibrated before each study with a water manometer and checked at the end and found in each case to be unchanged. Zero pressure was checked frequently during each study. Flow was measured through a Fleisch No. 3 pneumotachograph which gave a linear response up to 2 l.s⁻¹. The flow signal was integrated to give volume. There was no significant drift of the tidal volume tracing against time. Flow and volume were calibrated using a one litre syringe according to manufacturer's instructions (water displacement volume of calibrating syringe 1.03 l).

During tidal breathing, pleural pressure was measured in each patient at five points in each of five consecutive representative breaths; at instants of zero flow, the most positive and the most negative pressure, and at the end of the long declining plateau of expiratory pressure immediately before the rapid fall in pressure heralding the onset of inspiration at flow reversal. Dynamic lung compliance was measured according to the method of FRANK *et al.* [17] on the same five consecutive representative breaths during quiet tidal breathing. Transpulmonary pressure (Pm-oes) was measured during interruptions to a slow expiration in 4 patients (static lung compliance) and throughout very slow expiration in three patients (quasi-static compliance). In three patients technically satisfactory measurements of compliance were not obtained. A constant volume history of two deep breaths immediately preceding the measurement was enforced, and the pressure volume line of best fit by eye was drawn through the pooled points obtained from at least three attempts. Static compliance in the tidal range was taken as the slope of the pressure volume line for one litre above FRC.

Alveolar pressures, near the beginning (1), and near the end (2) of expiration were calculated on the same five consecutive representative breaths as follows. Alveolar pressure = pleural pressure + lung recoil pressure. Pleural pressure we have measured as oesophageal pressure, and for the calculation of these 2 alveolar pressures we have taken (1) the maximum pleural pressure, which is achieved early in expiration (within 0.5 s), and (2) the pleural pressure just before the rapid fall in pleural pressure which heralds the onset of inspiration. At instants of no flow (end-inspiration and end-expiration) alveolar pressure = 0 and pleural pressures are equal and opposite to lung recoil pressures so we have taken the measured pleural pressures

at flow reversal to be the lung recoil pressures, at the beginning and end of expiration. The sum of these 2 pleural pressures (1) at and near the beginning of expiration, and (2) at and near the end of expiration, equals the alveolar pressure at each of these times. At the end of expiration this alveolar pressure has been described as intrinsic positive end-expiratory pressure (PEEP) (18). This rapid negative swing of oesophageal pressure up to the onset of inspiratory flow reflects the elastic recoil of the total respiratory system at that lung volume which has to be overcome before inspiratory flow can begin. In making these calculations it is assumed that mean alveolar pressure is zero at instants of no flow at the mouth and possible pendelluft is not taken into account. A further inaccuracy results from pleural pressure being measured at a lower lung volume (297 ± 140 ml) than the lung recoil pressure in the calculation of maximum expiratory alveolar pressure early in expiration. At this lower volume the mean reduction in lung recoil pressure from the value used, would be 1 cmH₂O water (static) or 2.5 cmH₂O (dynamic). At the end of expiration the volume difference is too small to be measured (fig. 2).

Satisfactory EMG tracings were obtained in 4 of 6 patients with chronic airflow obstruction and in 2 normal subjects using silver-silver chloride standard surface electrodes in the 7th right intercostal space (7RICS), Electromed 4880 isolated EMG pre-amplifier and Electromed MX216 recorder. It has been suggested that electrodes placed over the area of apposition of the diaphragm to the chest wall, act as surface electrodes for the diaphragm. It is our experience that these electrodes detect both inspiratory and expiratory muscle activity.

In the patients expiration was noted to end abruptly (fig. 2) while there was still measurable flow, and presumably pressure to drive it. We attempted to determine to what lung volume expiration would have continued if not interrupted by the next inspiration (*i.e.* the elastic equilibrium volume where lung and chest wall recoil are equal and opposite). We fitted an exponential curve to the decay phase of the flow in the same 5 consecutive breaths. We then extrapolated this curve to zero flow and calculated the area under the curve. Equation for the decay of flow was $y = e^{-kt}$ where y = flow rate, k = decay constant, t = time. Area under the extrapolated curve (A) was calculated as follows; $A = a/k$ where a = instantaneous flow just prior to end expiration (see Appendix).

Tidal breathing throughout this study was stimulated in that 1) each subject was wearing a nose clip and breathing through a mouthpiece during the measurements and 2) there was 140 ml apparatus deadspace.

Results

The tidal expiratory flow, volume and pressure patterns of 1 normal subject are shown in figure 1. It can be seen that both flow and pleural pressure tracings are more sinusoidal in appearance [19] than are the

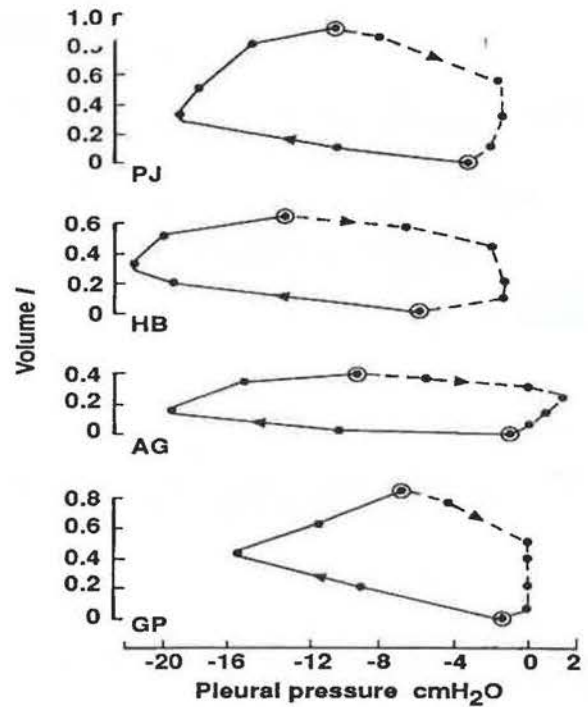


Fig. 3 - Tidal pressure volume loops in the 4 most severely obstructed patients have been constructed from analysis by hand of synchronous oesophageal pressure vs time and volume vs time tracings. The open circles show the relationship between volume and pressure at instants of flow reversal.

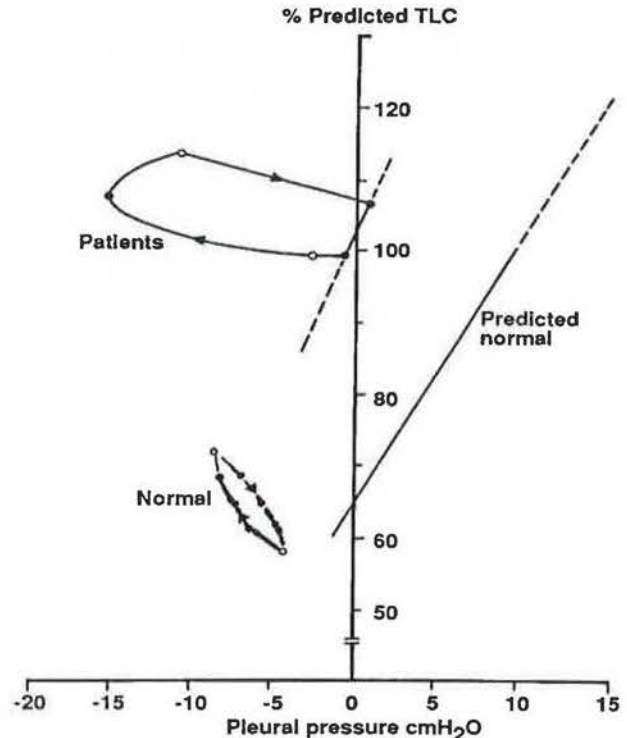


Fig. 4. - Mean tidal pressure volume loops are shown for the 2 normal subjects and for the 10 patients. — — — : Chest wall pressure volume relaxation line; o : Flow reversal; TLC: total lung capacity.

corresponding tracings of the representative patient with chronic airflow obstruction (fig. 2). The tidal expiratory flow pattern seen previously [2] in patients with airflow obstruction (fig. 2) was confirmed in these 10 patients and exhibited three distinct phases: 1) rapid rise to maximum flow; 2) slow decline in flow through most of expiration, 3. Abrupt ending of expiration with flow dropping rapidly through zero. These changes were mirrored in the synchronous oesophageal pressure tracing (fig. 2). At the beginning of expiration, pressure increased rapidly to its maximum near zero, then declined very gradually throughout much of the remainder of expiration. Then at the point where expiratory flow abruptly decreased there was a rapid fall in pressure which thus began milliseconds before flow reversal (fig. 2). Figure 3 shows tidal pressure volume loops constructed from the tracings of pressure vs time and volume vs time in the 4 subjects with the severest AFO. Figure 4 shows mean tidal pressure volume loops in the 2 normal subjects and the 10 patients. Again, the smooth contour of expiration in the normal subjects is in contrast to the 3 distinct phases of the expiratory volume pressure loop in the patients with airflow obstruction.

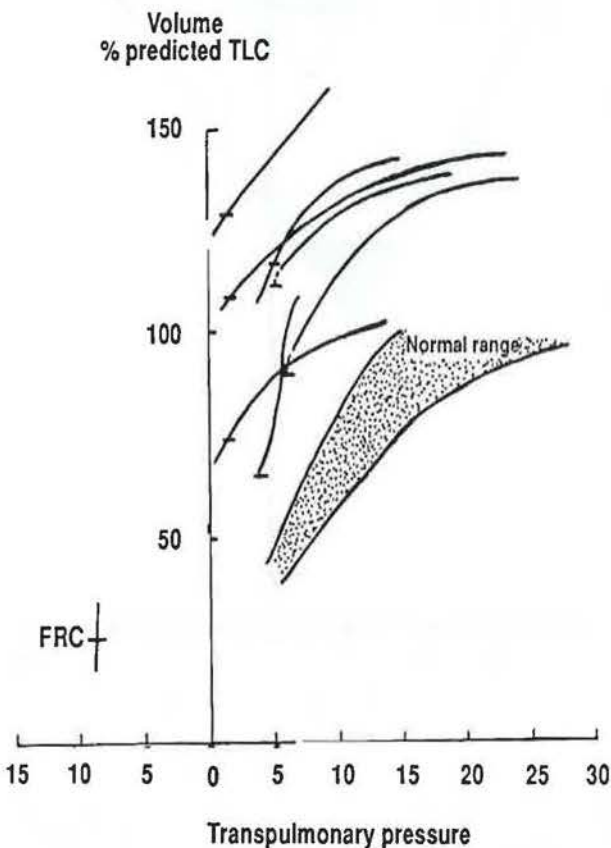


Fig. 5. - Lung pressure-volume curves in 7 patients with airflow obstruction. Normal range (drawn from data in COTES [9]) is shown for comparison. Transpulmonary pressure at FRC and over the tidal range is similar in these patients to that in normal subjects over their tidal range. TLC: total lung capacity; FRC: functional residual capacity.

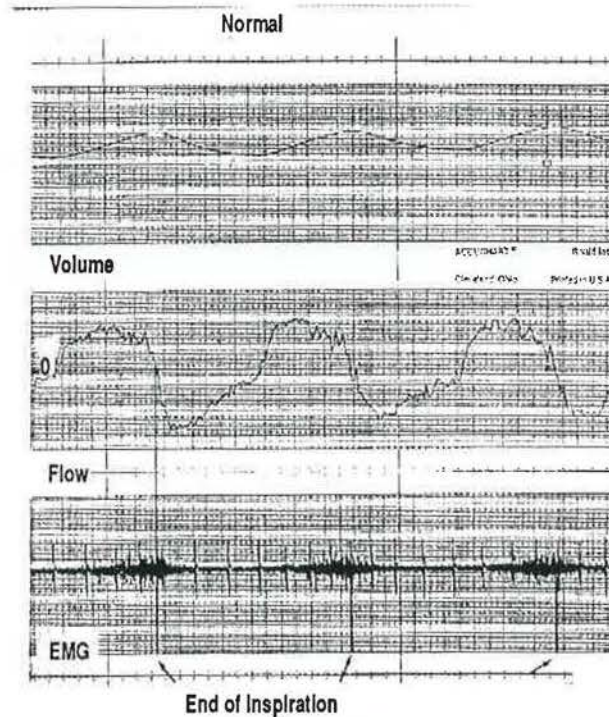
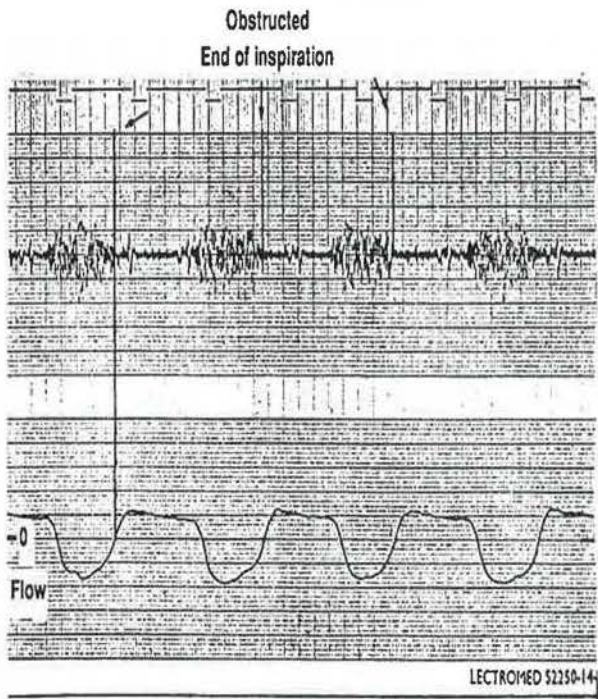
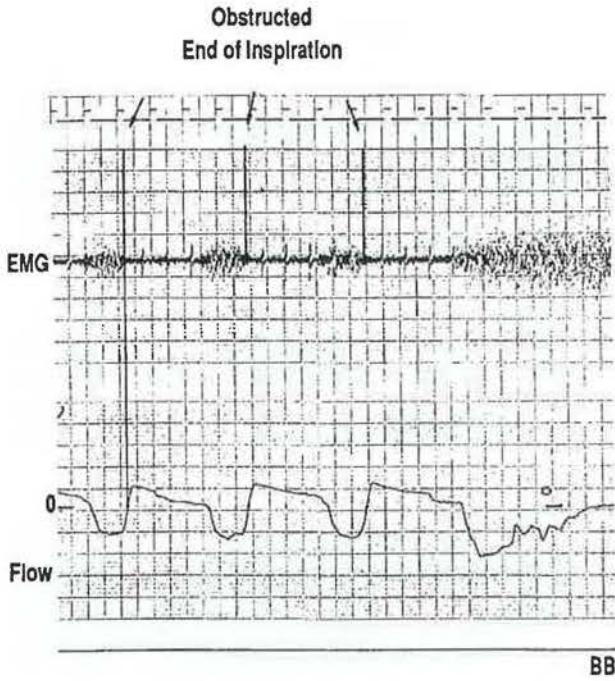


Fig. 6. - EMG from surface electrodes in 7th right intercostal space in normal subject during tidal breathing. EMG: electromyograph.

In the patients, pleural pressure remained negative throughout expiration or became only slightly positive, the most positive pressure being 0.6 ± 2.1 cmH₂O (mean \pm SD). Pleural pressure at the end of the slow decay of tidal expiratory flow was -0.6 ± 2.0 cmH₂O and was -2.6 ± 2.0 cmH₂O at the point of flow reversal. Mean difference in pleural pressure between end-expiration and end-inspiration (-10.9 ± 4.7 cmH₂O) at instants of flow reversal was 8.3 ± 3.8 cmH₂O. Tidal volume was 0.8 ± 0.3 l, frequency 16.9 ± 3.1 and dynamic compliance 0.11 ± 0.07 l \cdot cmH₂O⁻¹. There were large tidal swings of pleural pressure, 15.8 ± 5.1 cmH₂O, with high negative inspiratory pressure achieved (15.2 ± 5.2 cmH₂O). There was no correlation between end-expiratory pleural pressure and the degree of over-inflation as measured by the plethysmographic lung volume (FRC) expressed as % predicted total lung capacity (TLC). End-expiratory pressure stayed close to zero no matter how marked the over-inflation. Calculated alveolar pressures were markedly positive, 11.7 ± 4.9 cmH₂O (range 6.1–21.2), at the beginning of expiration and still positive, 2.0 ± 0.9 cmH₂O, at the end of expiration just before flow reversal (fig. 2). Static compliance was 0.371 ± 0.273 l \cdot cmH₂O⁻¹, transpulmonary pressure at held TLC was 16.1 ± 6.4 cmH₂O and transpulmonary pressure at FRC was 3.3 ± 2.3 cmH₂O (fig. 5). Dynamic lung compliance in the 7 patients in whom both were measured (0.117 ± 0.066 l \cdot cmH₂O⁻¹ was less than static lung compliance (Wilcoxon signed rank test $p < 0.02$). Maximum flow volume measurements showed marked decrease in maximum expiratory flows in the 7 patients



Figs 7 and 8. - Typical EMG tracings from 2 representative patients with chronic airflow obstruction during tidal breathing. EMG: electromyograph.

in whom the measurement was made. Over the tidal volume range in 6 the maximum expiratory flow lay within the tidal flow tracing, and in 1 the maximum expiratory flow was $0.1 \text{ l}\cdot\text{s}^{-1}$ greater than tidal flow at expiratory mid-tidal volume.

Raw EMG tracings during tidal breathing showed phasic inspiratory activity which in the 2 normal

subjects (fig. 6) continued during expiration [20, 21], and which in the patients with chronic airflow obstruction stopped fairly abruptly at the end of inspiration (figs 7, 8). A flat tracing was seen throughout expiration until immediately before flow reversal. These surface electrodes did not detect expiratory activity during tidal breathing, but immediately detected any voluntary expiratory effort. After the completion of this study, it was found in similar patients with AFO that surface electrodes over external oblique were silent during quiet tidal ventilation, but showed phasic expiratory activity during tidal ventilation when expiration was forced as did 7RICS electrodes. Both external oblique electrodes and 7RICS showed expiratory activity during slow expiratory vital capacity manoeuvre.

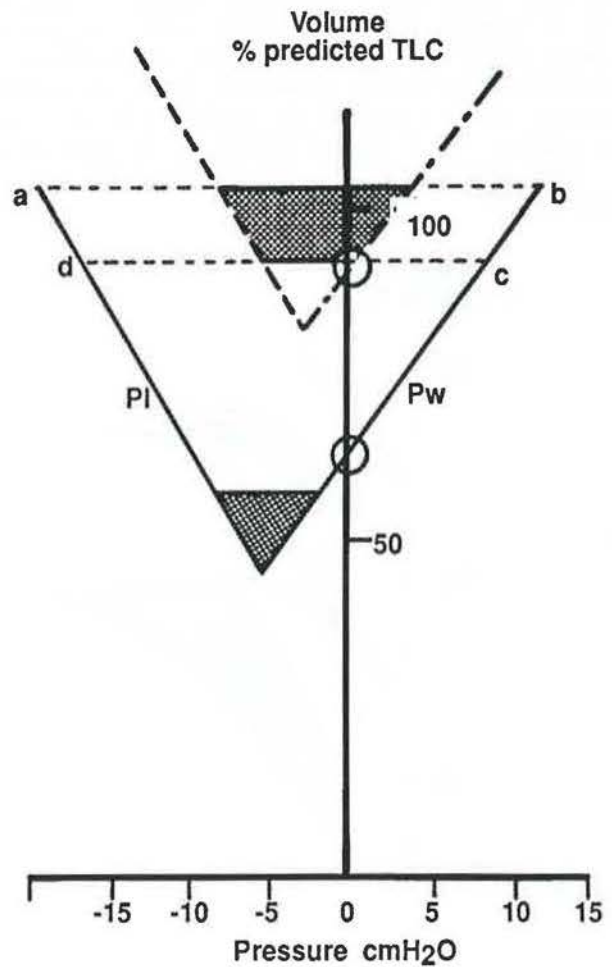


Fig. 9. - Schematic P-V work diagram using mean data from 10 patients with AFO shows measured loss of lung recoil and suggested loss of chest wall recoil with new elastic equilibrium volume. Volume of dynamic hyperinflation is less than total volume of over-inflation, FRC - predicted elastic equilibrium volume. — : predicted chest wall and lung relaxation lines [9]; - - - : measured lung pressure volume line; — · — · — : Suggested chest wall relaxation line; o : neutral position of chest wall; ⊞ : elastic work of breathing; abcd: area denoting predicted elastic work when breathing at high lung volume, elastic properties of lungs and chest wall unchanged.

Although a single exponential is unlikely to describe fully the pattern of lung emptying in patients with air-flow obstruction, the correlation coefficient of the linear regression of $\log(\text{flow vs time})$ was between 0.953 and 0.990, $p < 0.001$, for each of the 10 patients.

The mean extrapolated volume calculated from the decay of flow during tidal breathing was 0.43 ± 0.29 l and was significantly less than the volume of hyperinflation as assessed by the plethysmographic measurements, 2.13 ± 1.5 l. There was a significant positive correlation between k (decay constant) and specific airways conductance ($sGaw$) ($r = 0.834$, $p < 0.01$), but not between k and FEV_1 % predicted ($p > 0.1$) (table 1).

Discussion

These patients all suffered from chronic AFO, and measurements of pressure-volume and flow-volume relationships and of alveolar pressures have been presented to characterize them further.

Lung compliance (fig. 5) showed a shift upwards of lung pressure-volume lines with loss of lung recoil pressures although static lung compliance remained similar over the tidal range to that of normal subjects over their tidal range. The significant difference between dynamic and static lung compliance could be in part attributed to the different volume history prior to each measurement, but dynamic compliance may be decreased in these patients with chronic AFO as a manifestation of their frequency dependence of dynamic compliance [22, 23].

The mean pressure volume loop (fig. 4) shows the mean expiratory pressures lying on a line roughly parallel to the predicted normal chest wall relaxation line, but shifted upwards in a similar manner to the measured lung pressure-volume lines. Such a shift upwards of chest wall relaxation pressures has been demonstrated by SHARP *et al.* [24] in their series of patients with chronic airflow obstruction. Pleural pressures at the end of tidal expiration are not more positive in the patients who are breathing at higher lung volumes. This finding is compatible with loss of chest wall recoil occurring with longstanding over-inflation.

In similar patients in whom maximum expiratory flow does not exceed tidal flow, O'DONNELL *et al.* [25] have shown that flow limitation is occurring during tidal breathing. If we assume from the flow volume curves that this is true in these subjects, expiratory flow over the tidal volume range is reaching its maximum when pleural pressure is quite negative, -3.8 ± 2.7 cmH_2O . It may be that the intrinsic narrowing and increased compliance of the airways are enough to decrease pressure wave speed at some level in the airway walls sufficiently to limit gas flow, without the additional narrowing due to dynamic compression which is necessary for flow limitation to occur in normal airways.

Alveolar pressures were markedly positive during expiration as would be expected in AFO. Some of these patients resemble normal subjects in that positive

alveolar pressures are associated with sub-atmospheric pleural pressures throughout expiration (fig. 2). In others pleural pressures are slightly positive early in expiration. In these stable upright patients with airflow obstruction, end-expiratory alveolar pressure (intrinsic PEEP) ranged from 0.2–3.2 cmH_2O , mean 2.0 cmH_2O , which is less than that described by PETROF *et al.* [18] in their apparently similar patients during weaning after an exacerbation requiring ventilation (4.6 ± 0.6 cmH_2O). It is however similar to the value of 2.5 ± 1.5 cmH_2O reported by DAL VECCHIO *et al.* [26].

AGOSTONI [27] has argued that the normal delay in peak tidal expiratory flow is produced by continued contraction of inspiratory muscles and that this is reflected in a smooth decline in pleural pressure. He further comments that removal of inspiratory muscle braking would produce a rapid rise in pleural pressure early in expiration and this has been demonstrated by MCILROY *et al.* [29] and by PIERCE [29] in voluntarily relaxed expiration in trained subjects. This is exactly the pattern of flow and pleural pressure seen in the present study in the patients with AFO (fig. 2) and the EMG tracings in the patients with AFO show phasic inspiratory muscle activity to stop abruptly at the end of inspiration (figs 7 and 8). CITTERIO *et al.* [7] has also shown in 8 patients with chronic AFO more rapid decay of inspiratory muscle activity from its peak during inspiration than in normal subjects.

There are ethical difficulties in using needle electrodes for research measurements in severely disabled patients, and because of the specific nature of the signal many different muscle groups would have to be investigated. By using surface electrodes which detect muscle activity from any sizeable muscle mass in the vicinity, we have attempted to get a global view of phasic respiratory muscle activity. It is from electrical silence that we are drawing our conclusions, and we believe that silence from surface electrodes which do detect phasic inspiratory and expiratory muscle signals, is meaningful.

In normal subjects no electrical activity of the abdominal muscles is found during quiet breathing in the supine position [30, 31] and GORINI *et al.* [32] found no increase in abdominal EMG activity with histamine induced bronchoconstriction. With bipolar needle electrodes TAYLOR [33] found only a very small area in which there was activity of the internal intercostals during expiration in quiet breathing. With more vigorous breathing and with forced expiration expiratory activity occurred throughout the intercostals. We have interpreted the EMG silence we found over 7R1CS and over the external oblique, and the generally negative and decreasing pleural pressures during expiration as indicating that there is no significant expiratory muscle activity in these patients during expiration. Phasic inspiratory activity begins just before flow reversal and opposes the alveolar pressure (intrinsic PEEP) still available to drive unopposed expiratory flow. A patient would probably sense this as work associated with inspiration. It is not surprising, if in expiration there is relaxation of both inspiratory and expiratory muscle

groups, and in inspiration high negative pressures are generated, that patients perceive inspiration as being more difficult.

Extrapolation of the tidal flow tracing leads to an elastic equilibrium volume which is much greater than the predicted one (predicted FRC). Even if these patients are experiencing flow limitation during tidal expiration, this does not invalidate this calculation; flow should continue, even if at a limited rate, while there is pressure available to drive it. One interpretation of these data is that because of loss of lung elastic recoil (measured) and loss of chest wall recoil (demonstrated by SHARP *et al.* [24] in similar patients and suggested in this study by the measured tidal expiratory pleural pressures), in these patients elastic equilibrium occurs at a mean volume approximately 0.5 l below dynamic FRC (fig. 9). Because of the chronic nature of the AFO with the upward shift of the elastic equilibrium volume, this measurement of dynamic hyperinflation does not indicate at what level above the predicted FRC the patient is breathing, which is the measurement of hyperinflation made by other methods.

Appendix

During tidal expiration in patients with airflow obstruction (fig. 2), if we assume that the decay of flow is exponential the equation describing the flow at any instant is:

$$\text{Flow} = a e^{-kt}$$

where a is flow at time zero and t is time elapsed since time zero. If the expiratory flow is extrapolated to zero flow which it will reach at time ∞ , the area under the extrapolated curve will be the volume that would have been expired if expiration had continued to zero flow with the same exponential decline of flow.

This volume can be obtained by integrating ae^{-kt} from 0 to ∞ , a being the flow just before the rapid decline of flow which heralds the onset of inspiration.

$$\begin{aligned} A &= \int_0^{\infty} (a e^{-kt}) \\ &= -a/k \int_0^{\infty} [e^{-kt}] \\ &= -a/k (e^{-k\infty} - e^{-k\cdot 0}) \end{aligned}$$

But $e^{-\infty} = 0$ provided k is positive as it must be in a decay curve and $e^0 = 1$.

$$\begin{aligned} A &= -a/k (0-1) \\ A &= a/k \end{aligned}$$

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Activité des muscles respiratoires au cours de l'expiration chez les patients atteints d'obstruction chronique de courant aérien. M.J. Morris, R.G. Madgwick, A.I. Frew, D.I. Lane
 RÉSUMÉ: Au cours de la respiration calme, chez 10 patients atteints d'obstruction chronique sévère du débit aérien (AFO), l'on a noté de larges variations de pression pleurale en respiration au volume courant (15.8 ± 5.1 cmH₂O), avec de fortes pressions négatives (15.2 ± 5.2 cmH₂O). Le type de pression pleurale se développe en miroir avec celui du débit expiratoire au volume courant, avec une augmentation rapide vers le maximum de la pression pleurale et du débit au début de l'expiration, et une diminution lente des deux pendant la plus grande partie de l'expiration, ainsi que, juste avant le début de l'inspiration, au moment du changement de débit, une chute brutale de la pression pleurale et du débit expiratoire. La forme des tracés de débit expiratoire et de pression pleurale, ainsi que le type d'enregistrement d'électromyogramme, sont compatibles avec la perte du freinage musculaire post-inspiratoire du débit. La pression pleurale généralement négative, et le silence électromyographique au cours de l'expiration, ont indiqué une relaxation des muscles expiratoires pendant toute l'expiration. L'extrapolation de la courbe de débit expiratoire à volume courant vers zéro, et le calcul de la zone sous la courbe extrapolée, ont montré que le volume de l'hyperinflation dynamique n'est qu'une petite proportion de l'augmentation totale de la capacité résiduelle fonctionnelle au dessus des valeurs prédites chez ces patients. *Eur Respir J*, 1990, 3, 901-909.