The effect of hyperinflation on respiratory muscle work in acute induced asthma

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ABSTRACT: To examine the relationship between end-expiratory lung volume and respiratory muscle work during acute bronchoconstriction, we measured the work of breathing in nine asthmatic subjects, in whom bronchoconstriction was induced with histamine aerosol. When the forced expiratory volume in one second (FEV₁) fell below 60% of the control value, work was measured at the spontaneously hyperinflated lung volume (Vls), at a volume equivalent to the control functional residual capacity (FRC) and at a volume 30% of vital capacity (VC) above the control FRC. Hyperinflation to Vls caused a 39% decrease in the total positive work per breath from 2.8±0.9 to 1.7±0.1 J, entirely due to a decrease in inspiratory work per breath from 1.6±0.4 to 0.8±0.3 J. Inspiratory work did not change at any lung volume, because the increase in inspiratory elastic work due to hyperinflation was offset by the decrease in flow resistive work. Breathing above Vls did not alter the total positive muscle work, but did increase the negative work of the inspiratory muscles from 0.4±0.1 to 0.8±0.1 J·breath⁻¹. We conclude that during induced asthma spontaneous hyperinflation minimizes the total respiratory muscle work and may constitute a mechanism for minimizing energy expenditure.

In patients with bronchial asthma, an elevation in end-expiratory lung volume is commonly seen during spontaneous attacks [1], as well as during bronchoconstriction induced by exercise [2, 3] and pharmacologic agents [4, 5]. This hyperinflation must increase the elastic component of inspiratory muscle work [6] which is additive to the increased flow-resistive component consequent upon airway narrowing. In fact, in a previous study it was concluded that the major part of the increase in inspiratory muscle work was the result of hyperinflation [6]. Thus, hyperinflation, which is presumably beneficial in terms of airway patency and hence gas exchange, is achieved at the expense of an increased mechanical load, that might lead to respiratory muscle fatigue and ventilatory failure.

During an acute attack of a disease in which expiratory airflow obstruction is prominent, there is surprisingly little expiratory muscle recruitment [7]. This raises the possibility that hyperinflation might be advantageous not only by improving airway patency and hence decreasing the inspiratory flow-resistive work, but also by decreasing expiratory muscle work. However, there is no published information as to the influence of different end-expiratory lung volumes on respiratory muscle work during acute bronchoconstriction. The purpose of this study, therefore, was to establish the relationship between end-expiratory lung volume and respiratory muscle work (both inspiratory and expiratory) during acute bronchoconstriction.

Methods

Nine non-obese asthmatic subjects (7 male, 2 female) with a long history of asthma were selected for the study. Morphometric data for the subjects are given in table 1. The average age of the subjects was 35 yrs (range 19-60 yrs). All subjects gave histories of regular episodic attacks of wheezing requiring bronchodilator treatment since childhood. They were in clinical remission and all had refrained from use of inhaled bronchodilator for more than 6 h prior to the study. Informed consent was obtained and the protocol was approved by the Medical Ethics Committee of the Institution.

Increasing bronchoconstriction was induced by sequentially administering doubling concentrations of an aerosol of histamine, according to the protocol of Cockcroft et al. [8]. Solutions of histamine diphosphate were aerosolized using a nebulizer which delivered 0.26-0.30 ml over 2 min at an air flow rate of 6 l·min⁻¹. Subjects commenced with an initial concentration of 0.03 mg·ml⁻¹ and doubling concentrations were
Table 1. – Anthropometric data and lung volumes

<table>
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<th>Subject</th>
<th>Sex</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>TLC (% TLC)</th>
<th>FEV₁ (% TLC)</th>
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TLC: total lung capacity; FRC: functional residual capacity; RV: residual volume; FEV₁: forced expiratory volume in one second; FEV₁/FVC: ratio of FEV₁ to forced vital capacity; BC: during bronchoconstriction.

The subjects were seated in a pressure compensated volume-displacement body plethysmograph and breathed into a mouthpiece connected through a three-way valve to a Fleisch No. 2 pneumotachograph. This was coupled to a Validyne MP45 (+0.5 kPa) differential pressure transducer to measure airflow. The other port of the three-way valve was used to deliver aerosols and measure the FEV₁ with an electronic autospirometer (Minato AS-800).

Pleural pressure (Ppl) was measured with a 10 cm latex oesophageal balloon [9] coupled to a Validyne MP45 (+9.8 kPa) differential pressure transducer. Volume changes were obtained from the spirometer bell of the body plethysmograph. All signals were recorded on a Hewlett-Packard stripchart recorder and stored on magnetic tape. Prior to bronchoconstriction, absolute lung volumes were measured in the body plethysmograph using the Boyle's Law method [10].

[Fig. 1. – Schematic representation of the breathing manoeuvre performed after bronchoconstriction. VLS, VLC and V LH are the spontaneous, control and high end-expiratory lung volumes, respectively; TLC: total lung capacity; RV: residual volume.]
Fig. 2. — A schematic representation of the relationship between pleural pressure and volume during a bronchoconstricted breath with end-inspiratory and end-expiratory points shown. Arrows denote the direction of the loop. Pstw: static pressure-volume curve of the relaxed chest wall; Cldyn: the dynamic compliance of the lung. Shaded areas to the left of Pstw denote total positive inspiratory muscle work and to the right denote expiratory muscle work. Flow-resistive inspiratory muscle work (Wres) is the shaded area to the left of Cldyn. Elastic inspiratory muscle work (Wel) is the dotted area between Cldyn and Pstw. Expiratory muscle work (Wt) is the shaded area to the right of Pstw. Negative inspiratory muscle work is the area enclosed by ABCA, ZZ: Wres; ZZ: Wt; ZZ: Wel.

The subjects were assisted in keeping end-expiratory lung volume constant, and the tidal volume fixed at 750 ml by a visual display of the volume trace on an oscilloscope. They breathed with an inspiratory flow of 0.5 l·s⁻¹ at each of the 3 lung volumes, but expiratory flow was not controlled due to the presence of expiratory flow limitation. This pattern of breathing was adopted in an attempt to match ventilation at the three different end-expiratory volumes. Attention to maintaining constant subject posture was aimed at throughout the experiment.

Total lung capacity was assumed not to change throughout the study, including the period of induced bronchoconstriction [11]. Vls, Vls and Vli were subsequently derived from the difference between the respective end-expiratory lung volume and TLC.

The static relaxation pressure-volume curve of the chest wall (Pstw) was measured in 8 asthmatic subjects (2 included in this study) prior to bronchoconstriction. They were studied in our laboratory during clinical remission and had a group mean FEV₁ of 80% predicted. Pleural and gastric (Pga) pressures were measured with 10 cm latex balloons, in the oesophagus and stomach, respectively, coupled to Validyne MP45 (+9.8 kPa) differential pressure transducers. When the pressure difference (Pga-Ppl) during deflation was similar to or less than that at end-expiration, the subject was assumed to be relaxed. At least three technically adequate deflation curves from near TLC were performed in each subject and a mean chest wall relaxation curve was obtained from the 8 subjects. The Pstw curve was then applied to the data obtained in this study. The mean value for chest wall compliance was 38±3% TLC·kPa⁻¹, which was within the tidal breathing range of 31–64% TLC·kPa⁻¹ quoted by Turner et al. [12] for normal subjects.

Mean tidal volume-pleural pressure loops were obtained from 5 tidal breaths at each of the lung volumes. The work of breathing was determined graphically [13] by relating changes in Ppl to the Pstw (fig. 2). This figure shows a schematic representation of a volume-pleural pressure loop through inspiration and expiration with Pstw and the measured dynamic compliance (Cldyn) indicated. The work done by the respiratory muscles is represented as the area under a pressure-volume curve. Pressure generation for areas to the left of the chest wall relaxation curve is by the inspiratory muscles, and areas to the right by the expiratory muscles. Positive inspiratory muscle work can be further subdivided into an elastic component (shown as Wel) and a flow-resistive component (shown as Wres) by using Cldyn. The areas (measured by planimetry) of the elastic and flow-resistive components added together give the total positive inspiratory muscle work (Wt). Expiratory muscle work is to the right of Pstw (shown as Wt). Total respiratory muscle work was calculated by adding the positive inspiratory and expiratory components. The negative work of the inspiratory muscles (Wres), which corresponds to the work performed by the muscles during expiration while being lengthened by potential energy stored in the chest wall, is denoted in figure 2 by the area ABCA. From the dynamic pressure-volume loops at each lung volume, the minimum (or most negative) inspiratory Ppl (Pplmin), and maximum (or most positive) expiratory Ppl (Pplmax) were measured. Similarly, inspiratory and expiratory muscle pressures (Pimus and Pmus) were measured as the maximum pressure difference between Pstw and the volume-pressure loop, with Pmus to the left of Pstw and Pmus to the right (when present).

The data at high lung volume (Vls) came from only 6 of the 9 subjects. This was because in 3 subjects, the spontaneous hyperinflation reached or exceeded the predetermined high lung volume.

Statistical analysis was performed using one-way analysis of variance, coupled with the Scheffe F-test for determination of significance [14]. All data are expressed as mean±1se.

Errors of the method

There were several components of total mechanical work we did not measure. These included the work to overcome the flow-resistance of the chest wall and the elastic distortion of thoraco-abdominal structures. Similarly, any negative work by the expiratory muscles has not been calculated. The compressibility of alveolar gas may also be important in subjects with high flow resistances. However, any increase in work due to gas decompression is likely to be small. Unless the subject is breathing rapidly (>30 breaths·min⁻¹), end-inspiratory
pleural pressures are unlikely to reach -3.9 kPa, in which case the volume of decompression would be less than 4% of FRC [15]. All calculations refer to work performed by the inspiratory and expiratory muscles, and do not consider any muscle internal work.

The inter-subject variability in the relaxation pressure-volume curve of the chest wall was small, and we assumed that large changes do not occur with bronchoconstriction. There is only one case reported with apparent loss of chest wall recoil during bronchoconstriction [16] and this could be explained by errors in calculation of total lung capacity. Also, any measurements of Pstw during severe asthma would be in doubt because of the extreme difficulty in achieving adequate subject relaxation. We therefore used a mean chest wall relaxation curve from a group of asthmatics in remission for this analysis. Large inter-individual differences, were they to occur, could have influenced some of our results.

Dynamic compliance has been used to partition inspiratory work into its elastic and flow resistive components. However, some of the reduction in dynamic compliance is due to flow resistive pressure drops between asynchronously ventilated units.

Finally, we controlled tidal volume and inspiratory flow rate during the study. Theoretically, the subjects could have decreased the level of work at any given end-expiratory lung volume by adopting different patterns of breathing. Therefore, we do not claim to have measured the spontaneous work of breathing at different lung volumes. Our aim was to keep ventilation matched, and to study only the mechanical effects of lung volume on the work of breathing.

Results

Prior to bronchoconstriction the group mean FEV₁, FEV₁/FVC ratio and TLC were within the normal range (table 1). The control end-expiratory lung volume (VOLD) constituted 57±3% of TLC. Following bronchoconstriction the FEV₁ decreased to 52±3% predicted (p<0.001) and the end-expiratory lung volume (VOLD) increased substantially to 71±2% TLC (p<0.001). RV also increased from 28±2 to 51±3% TLC. The high end-expiratory lung volume (VH) was predetermined at 80±2% TLC. The imposed pattern of breathing at VOLS did not significantly alter minute ventilation compared with the preceding spontaneous tidal breaths (14.7 l.min⁻¹ and 13.6 l.min⁻¹, respectively).

The spontaneous hyperinflation associated with bronchoconstriction resulted in a decrease in total positive respiratory muscle work per breath (WT) from 2.8±0.4 J at VOLD to 1.7±0.1 J at VOLS (p<0.001). However, the further increase in end-expiratory lung volume to VH did not decrease WT any more (table 2). There was no significant change in the inspiratory work muscle work per breath (WI) with increasing lung volume (table 2, fig. 3). The inspiratory elastic component increased from 28±5% of WI at VOLD to 83±3% at VH (p<0.001), whereas the flow-resistive component decreased from 73±6% at VOLD to 17±3% at VH (p<0.001) (table 2). In fact, over the 3 lung volumes, Wt rose linearly as a function of increasing end-expiratory lung volume (r=0.99) and Wt fell linearly (r=0.99). Inspiratory muscle work per breath (Wir) constituted 57% of Wt when breathing at VOLS during bronchoconstriction (table 2, fig. 3). Spontaneous hyperinflation markedly decreased We from 1.6±0.4 J at VOLT to 0.1±0.05 J at VOLS (p<0.001). The further increase in lung volume from VOLS to VH completely abolished the small remaining amount of We. Therefore, it is obvious that the reduction in Wt with increasing end-expiratory lung volume was entirely due to a reduction in its expiratory component. Since expiration was not controlled, the duration of expiration (TE) was greatest at VOLS (2.9±0.3 s) and least at VH (1.8±0.4 s). Necessarily mean expiratory flow was lower at VOLS than at VH.

| Table 2. - Respiratory muscle work during bronchoconstriction |
|-----------------|----------------|----------------|----------------|
|                 | WT             | WI             | Wt             |
|                 | J              | J              | J              |
| VOLS            | 2.8*           | 1.2            | 28*            |
| (n=9)           | ±0.4           | ±0.1           | ±5             |
| VOLS            | 1.7            | 1.6            | 56             |
| (n=9)           | ±0.1           | ±0.1           | ±4             |
| VH              | 1.6            | 1.6            | 83*            |
| (n=6)           | ±0.1           | ±0.1           | ±3             |

Data expressed as mean±se. VOLS, VOLS and VH are the control, spontaneous and high end-expiratory lung volumes, respectively. WT, WI, Wt, and Wt are positive total, inspiratory, expiratory and negative inspiratory muscle work per breath, respectively. Wt and Wt are the elastic and flow resistive components, respectively, of WI. Statistical comparisons between VOLS, VOLS and VH were with one-way ANOVA and a Scheffe F-test. *: p<0.01 for VOLS and VH to VOLS.

Fig. 3. - The positive total, expiratory and inspiratory muscle work during bronchoconstricted tidal breathing at the 3 lung volumes. VOLS, VOLS and VH are the control, spontaneous and high end-expiratory lung volumes, respectively. Bars represent 1 se. Note: i) the similar total inspiratory work at the 3 volumes; and ii) the decreasing expiratory work with increasing volume. : Total; : expiratory; : inspiratory.
There was a doubling of $P_{\text{m}}$ from $V_L$ to $V_L$. With increasing lung volume $P_{\text{p}}$ and $P_{\text{e}}$ varied. However, there did not increase when lung volume increased from $V_L$ to $V_L$. The negative work of the inspiratory muscles $W_{\text{i}}$ showed a progressive rise with increasing lung volume. There was a doubling of $W_{\text{i}}$ per breath from 0.4±0.1 kPa·L at $V_L$ to 0.8±0.1 kPa·L at $V_L$ (p<0.01). Thus, even though $W_{\text{i}}$ did not increase when lung volume increased from $V_L$ to $V_L$, there was more negative work being done.

At each lung volume $P_{\text{p}}$, $P_{\text{m}}$, and $P_{\text{e}}$ were measured during tidal breathing (table 3). The changes in $P_{\text{p}}$ and $P_{\text{m}}$ are illustrated for one subject in figure 4. With increasing lung volume $P_{\text{p}}$ became less negative. However, $P_{\text{e}}$ remained unchanged. During expiration, there was more subject variability with a range of $P_{\text{p}}$ from 0.9–10.7 kPa at $V_L$. There was a large decrease in $P_{\text{p}}$ after spontaneous hyperinflation and a non-significant further decrease at $V_L$. $P_{\text{m}}$ behaved similarly with all subjects having large positive expiratory muscle pressures at $V_L$. At $V_L$, four subjects no longer generated expiratory muscle pressures, and at $V_L$ no positive expiratory muscle pressures were recorded in any subject.

The dynamic compliance ($C_{\text{dyn}}$) was 1.6±0.1 kPa·L and was substantially reduced following bronchoconstriction to 0.4±0.1 kPa·L at $V_L$ (p<0.01). Spontaneous hyperinflation increased $C_{\text{dyn}}$ to 0.6±0.1 kPa·L (at $V_L$) and at $V_L$ it was 0.7±0.1 kPa·L (fig. 4). These changes did not reach statistical significance.

### Discussion

The increase in end-expiratory lung volume that almost invariably accompanies acute asthma has a number of adverse mechanical effects, which include the increase in elastic work on both the chest wall and the lung. In fact, it has been postulated that hyperinflation is the cause of the major portion of the increase in inspiratory muscle work during acute bronchoconstriction [6]. In addition, the hyperinflation may disadvantage the inspiratory muscles by causing them to contract from shorter initial lengths, leading to reductions in tension generating capacity [17] and efficiency [18].

During acute asthma, respiratory work may be increased to many times normal involving both an increase in power and in work per breath [19]. The traditional view is that the increased work is due to both an increase in airway resistance, and hyperinflation [20]. However, there is only one published study in which an attempt has been made to quantitate and partition this increased work. Martin et al. [6] found that the inspiratory muscle work rate...
increased tenfold from control to maximal bronchoconstriction. At that point elastic work accounted for 57% of the total. On this basis, the authors concluded that elastic work contributed the major portion of inspiratory work, and that the increase in elastic work was largely the result of hyperinflation. They did not measure expiratory muscle work, but on a separate occasion, reported an increase in negative work of the inspiratory muscles during induced asthma [7].

In our study, we have specifically tried to evaluate the mechanical effects of hyperinflation (as distinct from the increased pulmonary resistance) on the increased respiratory work during bronchoconstriction.

Spontaneous hyperinflation

Let us first compare the effect of breathing at the initial, control lung volume (assuming no hyperinflation) with that at the spontaneously selected volume (Vls). When overall inspiratory muscle work is considered, hyperinflation had no significant effect. The inspiratory work remained similar due to a large decrease in its flow-resistive component (which accounted for the majority of the inspiratory work at Vlc). In agreement with the findings of Martin et al. [6], the elastic component constituted the majority of inspiratory work at Vls. However, the increase in elastic work during bronchoconstriction was not solely due to hyperinflation but also to a decrease in dynamic compliance. This was related to the greater inequality of time constants in the lung caused by bronchoconstriction. After hyperinflation the inspiratory muscles must have been disadvantaged by operating at a shorter length. Presumably, this necessitated more activation and more metabolic activity to generate a given force [21].

Although expiratory and inspiratory work were similar and substantial at the control lung volume, hyperinflation markedly decreased expiratory muscle work. Indeed, it is remarkable that a relatively small increase in end-expiratory lung volume (14% TLC) almost abolished the expiratory muscle load. This decrease was partly due to a lower expiratory airway resistance attributable to the larger airway calibre at the higher lung volume. In addition, the increased passive elastic recoil of the lung and chest wall, after hyperinflation, may have provided an increased expiratory driving pressure which contributed to the decrease in expiratory muscle work. A recent study suggests that expiratory flow-resistive work is approximately half as efficient as inspiratory flow-resistive work [22]. If expiratory muscle work were twice as costly, then, in terms of total energy consumption, it would be advantageous to minimize expiratory muscle work, as achieved by the spontaneous hyperinflation. Thus the total positive respiratory muscle work following spontaneous hyperinflation was much less than that at control lung volume due to the large decrease in expiratory muscle work. It is worth noting that Te' at Vlc was greater than at Vls (as Te was not controlled). If Te' at Vlc had been maintained similar to that at Vls, the higher expiratory flows would have required much greater expiratory muscle pressures. Hence Wt would have increased at Vlc even further both due to resistive and compressive work. Some of the work that we were unable to measure, such as that due to chest wall distortion (both inspiratory and expiratory) was also likely to be greater at Vlc due to the higher expiratory pressures. Similarly, gas compressive work must have been greater at Vlc due to the higher pulmonary resistance [15]. In two of our subjects, gas compression when breathing at Vlc was so marked that it constituted a significant proportion of the plethysmographic volume signal displayed to the subject on the oscilloscope. Consequently, the actual inspired volume at Vlc was less than 750 ml, so that ventilation was not matched and thus the inspiratory muscle work at Vlc was also underestimated. All of these potential errors tended to underestimate total work at Vlc relative to that at Vls, so that the effects of spontaneous hyperinflation on minimizing total respiratory work may have been even greater.

As in other studies [6, 20], we found that dynamic compliance was markedly reduced by bronchoconstriction. However, relative to the control volume (Vlc), hyperinflation actually increased compliance, presumably due to a decrease in the inequality of time constants of different lung units [23]. Hence, this also represented a beneficial effect of spontaneous hyperinflation. The less negative Pplmin, when Pmus remained the same, implies that inspiratory work on the lung was decreased by hyperinflation, but the load on the inspiratory muscles did not change. However, since the largest changes occurred in the expiratory pressures, our results suggest that in the absence of hyperinflation and with no change in the pattern of breathing, the increased load during induced asthma would predominantly affect the expiratory muscles, and have little effect on the inspiratory muscles.

It is interesting to note, in figure 4, that the relaxation volume of the respiratory system during bronchoconstriction was higher than the control end-expiratory lung volume. This finding was consistent among all subjects, and may be explained by the change in position of the pressure-volume curve of the lung during bronchoconstriction. The upward shift of the curve, with no change in Pstw, would tend to increase the relaxation volume.

Voluntary further hyperinflation

No expiratory muscle work was performed by any subject at Vlh. However, when compared with Vls, this did not constitute a significant reduction in expiratory work. Similarly, there was no change in the inspiratory muscle work between Vls and Vlh, again due to reciprocal changes in elastic and flow resistive components. The increase in negative inspiratory muscle work from Vls to Vlh suggested higher energy requirements. However, as the energy costs of negative work are only 30% of those for positive work of an equal magnitude [24], this increase represented a small rise in total energy costs. Although Pplmin was less negative at Vlh, so that inspiratory work done on the lung decreased, Pmus
remained unchanged and hence the load on the inspiratory muscles remained similar. Thus, the mechanical effects of breathing at a lung volume higher than that of spontaneous hyperinflation conferred no further advantage. There was no decrease in total positive muscle work, and negative work increased. Also, the inspiratory muscles were operating at a still shorter length than that during spontaneous hyperinflation, and would have been further disadvantaged.

The linear rise in $W_{\text{clastic}}$ over the three lung volumes was unexpected because the shape of the static pressure-volume curve of the lung predicted an a linear relationship. Clearly, the increased dynamic compliance at higher volumes tended to linearize the relationship under the conditions of our study. The linear fall in $W_{\text{res}}$ with increasing lung volume was even more surprising in view of the complex relationship between elastic recoil and airway size. Because of the shape of the pressure-volume curve of the lung, breathing at a higher lung volume will result in larger end-expiratory pressures than breathing at a lower volume. This should cause a relatively greater airway dilatation at higher lung volumes, and hence a relatively greater decrease in $W_{\text{res}}$. The general trend, however, would be predicted from the hyperbolic relationship of pulmonary resistance and lung volumes seen in normal subjects [25].

The implication in terms of a patient with acute airflow obstruction is that the spontaneously hyperinflated lung volume is the most appropriate for that patient to minimize mechanical work. Similar breathing patterns which require the patient to breathe at a higher or lower end-expiratory volume may be more costly in terms of work done. In an obese asthmatic subject with acute bronchoconstriction, the spontaneous hyperinflated end-expiratory lung volume may not be as high, which would tend to make the inspiratory muscle work greater than if the subject was not obese. We speculate that the total mechanical work may be increased in obese asthmatic subjects.

In summary, during acute bronchoconstriction in asthmatic subjects, spontaneous hyperinflation minimized the total positive respiratory muscle work entirely by decreasing the expiratory muscle component. An increasing end-expiratory lung volume induced reciprocal changes in inspiratory elastic and flow-resistive work so that the total inspiratory muscle work did not change substantially. Thus, hyperinflation did not result in an increase in inspiratory muscle work. However, a further voluntary increase in end-expiratory lung volume above that of spontaneous hyperinflation did not change the total positive work, but increased negative work and presumably disadvantaged the inspiratory muscles further due to their shorter length. Our results suggest that the end-expiratory lung volume spontaneously selected in acute asthma constitutes a mechanism for minimizing the work of breathing and energy expenditure.

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RÉSUMÉ: Pour examiner les relations entre le volume pulmonaire à la fin de l'expiration et le travail musculaire respiratoire au cours de la bronchoconstriction aiguë, nous avons mesuré le travail respiratoire chez 9 sujets asthmatiques chez lesquels une bronchoconstriction avait été induite par des aérosols d'histamine. Lorsque le volume expiratoire maximal seconde (VEMS) baisse en dessous de 60% des valeurs de départ, le travail a été mesuré au volume pulmonaire d'hyperinflation spontanée (Vls), au volume équivalent à la capacité résiduelle fonctionnelle de contrôle et à un volume de 30% de la capacité vitale au-dessus de la valeur de la capacité résiduelle fonctionnelle de contrôle. L'hyperinflation jusqu'à Vls provoque une chute de 39% du travail positif total par cycle (2.8±0.4 à 1.7±0.1 J), totalement attribuable à une diminution du travail expiratoire par cycle qui passe de 1.6±0.4 à 0.10±0.05 J. Le travail inspiratoire ne se modifie à aucun volume pulmonaire, parce que l'augmentation du travail élastique inspiratoire due à l'hyperinflation est annihilée par la diminution du travail résistif au débit. Une respiration au-dessus de Vls ne modifie pas le travail musculaire positif total, mais augmente le travail négatif des muscles inspiratoires de 0.4±0.1 à 0.8±0.1 J par cycle. Nous concluons qu'au cours de l'asthme induit, l'hyperinflation spontanée diminue le travail musculaire respiratoire total et pourrait constituer un mécanisme pour minimiser la dépense énergétique.