Mechanisms and consequences of hyperinflation

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The mechanisms and pathophysiological consequences of hyperinflation are complex and not fully elucidated [1]. In the present issue of the journal, Wheatley *et al.* [2] and Cormier *et al.* [3] provide additional data contributing to our understanding of these aspects of hyperinflation in induced bronchoconstriction.

One of the mechanisms of hyperinflation may be changes in the static equilibrium between lung and chest wall as a consequence of a loss of elastic recoil of the lung and a shift of the static lung volume-pressure curve upward and to the left [4], or of a "resetting" of the passive elastic characteristics of the chest wall [5]. However, in conditions of bronchoconstriction, the hyperinflation is apparently determined mainly by dynamic factors [6]. The most important of these is the increased airway resistance with prolonged mechanical expiratory time constants and, thus, slow and incomplete lung emptying [1, 6]. Another factor is a persisting inspiratory muscle activity during expiration [7-10]. This is believed to be accessory and intercostal in nature rather than diaphragmatic [8, 9], although electrical activity has also been demonstrated in the latter muscle [10]. Furthermore, glottic constriction during expiration [11] and enhanced airway closure [12] have been demonstrated to accompany bronchoconstriction and have been suggested to contribute also to the hyperinflation. Finally, chronic adaptations of inspiratory muscles to shorter operating lengths and to permanently increased loads may occur in chronic obstructive pulmonary disease (COPD) [6] as occurs in experimentally induced emphysema in animals [13], and may thus also intervene in the hyperinflation.

In their present paper, Cormier *et al.* [3] have tried to shed more light on the mechanisms determining hyperinflation by investigating the extent to which the hyperinflation may be influenced by changes in the passive elastic loads, induced by applying extrathoracic pressures on asthmatic subjects lying inside an iron lung. After provoking a decrease in forced expiratory volume in one second (FEV,) of about 40% and an increase in functional residual capacity (FRC) of about 30% by methacholine inhalation they find that, while negative extrathoracic pressures lead to a further lung expansion, positive pressures can reduce the hyperinflation only from about 730 ml above the pre-challenge level to a lower plateau level of about 450 ml above the control FRC. They conclude that these data support the hypothesis that hyperinflation is, indeed, mainly due to prolonged expiratory time constants and persisting inspiratory muscle activity. It remains unclear whether this inspiratory muscle activity during expiration, found in transiently induced bronchoconstriction, also persists in spontaneously occurring, prolonged asthmatic episodes or in chronic hyperinflation in COPD.

The pathophysiological effects of hyperinflation are multiple, and beneficial as well as detrimental. On the one hand, hyperinflation improves airway patency and thus ventilation distribution [1, 5]. On the other hand, it is generally considered to profoundly alter respiratory muscle function [14], and to be very disadvantageous to inspiratory muscle function. It decreases the efficiency of the diaphragm in particular [5], due to a shift to the inefficient part of its force-length relationship [1], to a flattening of its curvature with a nearly horizontal orientation of its fibres so that their contraction pulls the rib margins inward rather than outward and upward [5], and to a decrease of the zone of apposition through which the lower rib cage is exposed to the outward pushing effect of increases in abdominal pressures. The mechanical disadvantages of hyperinflation on the parasternal and accessory muscles are much less evident [14]. From FRC to total lung capacity (TLC), the parasternals shorten less than the diaphragm [15-17] and apparently approach their optimal length near TLC [18]. This has been confirmed by findings in dogs that the relationship between electrical input and intramuscular pressure in the parasternal muscles improves with hyperinflation [19], and that the overall pressure generating capacity of the inspiratory muscles is relatively well-preserved near TLC [20], which indicates that other muscles compensate for the severe ineffectiveness of the diaphragm near TLC [14]. Hyperinflation, furthermore markedly increases the elastic work of breathing [6], but decreases the resistive work, and it also increases the negative work during expiration due to the tonic contraction of the inspiratory muscles [7, 8]. As a consequence of all previous effects, hyperinflation increases energy demands [1], while the energy supply to strongly contracting muscles may be impeded, all of which may preclude inspiratory muscle failure.

The mechanisms by which hyperinflation affects the work of breathing are analysed in more detail in the interesting paper by Wheatley *et al.* [2] in the present issue of the journal. A previous study from the same laboratory [7] demonstrated a tenfold increase in inspiratory work (to a value of about 3 J-breath') for a decrease in FEV, of about 50%, yet it remained unknown whether
this increase in respiratory work was due to the hyperinflation per se or to the primary bronchoconstriction. In the present paper, Wheatley et al. [2] specifically try to elucidate the mechanical effect of hyperinflation on the respiratory work as distinct from the effect of increased airway resistance. They find that after induced bronchoconstriction, causing a decrease of FEV₁, to below 60% of the control value and an increase of FRC of about 14% TLC, inspiratory work is 1.6 J/breath⁻¹ (56% of it being due to elastic work). Voluntary decrease of FRC to its control value of 51% TLC results in a nonsignificant decrease in inspiratory work to 1.2 J/breath⁻¹ (especially due to a decrease in elastic work to 28% of the inspiratory work) and a not unexpected, marked increase of expiratory work from 0.1 to 0.6 J/breath⁻¹. Hence, they draw the reasonable conclusion that the end-expiratory lung volume spontaneously selected in acute asthmatic attacks is such as to minimize the total work of breathing. In addition, they find that a further voluntary increase of end-expiratory lung volume does not change total or inspiratory work in comparison with the spontaneously chosen level of hyperinflation. Yet, this increases the elastic work further to 83% of the inspiratory work. It should, however, be stressed that the negative inspiratory work increases from 0.2 J at control FRC to 0.4 J at the spontaneous hyperinflation level, and to a significantly higher value of 0.8 J at the voluntary hyperinflation level. This suggests that, after all, a hyperinflation accompanied by higher energy requirements and decreasing efficiency of the mechanically disadvantaged inspiratory muscles at high volumes. Several components of total mechanical work are not measured in this analysis; these include the work to overcome the flow resistance of the chest wall and the distortion of thoracoabdominal structures which represent a major waste of effort [6]. In addition negative work by expiratory muscles is not calculated. Finally, it remains uncertain how far these data can be extrapolated to patients with spontaneous, episodic asthmatic attacks or especially those with COPD in whom adaptations to chronic changes in muscle length, chest configuration and mechanical loads may be expected to occur.

Clearly, much information is still needed before “an integrated view on how the vital pump is altered by increases in end-expiratory lung volume" will be obtained [14]. As more questions are answered and problems solved, our interpretation of the relative importance of different intervening factors in hyperinflation and of the secondary mechanical effects of it will vary, in a manner similar to that of a pendulum swinging back and forth until it reaches equilibrium.

References