Effects of hyperinflation on the respiratory muscles

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Chronic obstructive pulmonary disease (COPD) has a detrimental effect on the respiratory muscles, as is evident from reduced respiratory muscle strength [1-4], fibre size [5-7], and glycolytic enzyme activity [8]. These effects are presumably obtained through different mechanisms to which hyperinflation [9, 10], increased work of breathing [11], hypoxaemia [12], hypercapnia [13], and malnutrition [4, 14-17] may all contribute.

It is generally accepted that hyperinflation profoundly alters respiratory muscle function, but it also has the marked beneficial effect of decreasing airways resistance, thereby improving ventilation distribution and allowing an increase in minute ventilation in patients who are using maximal expiratory flow during resting ventilation [9]. However, it presumably shortens inspiratory muscles and thus displaces them to a less advantageous position of their length-tension curve. Moreover, it is thought to change inspiratory muscle geometry at functional residual capacity (FRC) [10] and, finally, to affect the mechanical interaction between respiratory muscles in a way which is still not fully understood [9, 18-20].

This editorial will focus on some new basic insights into how hyperinflation affects respiratory muscle length and function. It should be emphasized that hyperinflation may be extremely severe in COPD. Thus, a plethysmographically determined FRC equalling or even exceeding the predicted total lung capacity (TLC) is a relatively common finding in patients with severe airflow obstruction (fig. 1). From the point of view of inspiratory muscle mechanics these patients are, thus, similar to normal subjects breathing at or even above their TLC and, hence, the inspiratory muscles are expected to operate at a severely disadvantageous position of their length-tension curve. Recent animal experiments specifically addressing this question may contribute to a better understanding of how the respiratory muscles are affected by this enormous increase in FRC.

Firstly, the significance of the distinction between acute and chronic hyperinflation has become evident, since in the latter state adaptive changes to chronic shortening presumably occur in the diaphragm. Thus, in emphysemaous hamsters the diaphragm drops out sarcomeres, resulting in a shift of the whole length-tension curve to a shorter length [21-24], such that the muscle adapts to the shorter in situ operating length. Whether this adaptation also occurs in emphysemaous humans remains open to question. Indeed, ARORA and ROCHESTER [17] failed to obtain evidence for chronic diaphragmatic shortening and sarcomere drop-out in patients with COPD. This apparent discrepancy with animal experiments is not readily explained. It may relate to the fact that in the patients studied, hyperinflation was less severe than in the emphysemaous hamsters.

In acute hyperinflation, insufficient time is available for this adaptation to develop and, as a consequence, a severe mechanical disadvantage for the inspiratory muscles is expected. We examined the changes in length undergone by the diaphragm and by the parasternal intercostals during hyperinflation in supine anaesthetized dogs, using piezoelectric crystals [25, 26]. During inflation from FRC to TLC the parasternals shortened by about 7% [25], and the diaphragm by more than 30% [26]. As a consequence, hyperinflation is expected to induce a mechanical disadvantage, which is much more pronounced in the diaphragm than in the parasternal intercostals. Moreover, although the optimal length in the diaphragm is expected to correspond to a lung
volume close to supine FRC [27, 28], the parasternals were found to be longer than optimal at FRC and to approach their optimal length near TLC [29]. This was recently confirmed by experiments in which the relationship between electrical input and force output, estimated by changes in parasternal intramuscular pressure, was shown to improve with hyperinflation [30].

Although surprising at first sight, the latter findings are in keeping with the breathing pattern observed at elevated end-expiratory lung volume in supine anaesthetized and vagotomized dogs [30, 31]. Indeed, whereas dogs at FRC breathe with proportional rib cage and abdominal expansion close to the relaxation line, near TLC chest wall motion almost exclusively becomes rib cage motion, frequently even associated with abdominal indrawing and a fall in gastric pressure during inspiration. Since these changes were associated with increased diaphragmatic electromyographic (EMG) activity [30, 31], the latter finding is indicative of ineffective diaphragmatic contraction near TLC. The fact that in these experiments the pressure-generating capacity of the inspiratory musculature as a whole was relatively well preserved near TLC, supported our contention that the optimal length of the parasternal intercostals was probably close to TLC rather than to FRC [31]. Indeed, since hyperinflation induces a severe ineffectiveness of the diaphragm, other muscles have to compensate for it, in order to keep the pressure generating capacity of the global inspiratory musculature relatively constant.

Although, undoubtedly, the aforementioned experiments have contributed to our conceptual understanding of how hyperinflation affects inspiratory muscle function, it is appropriate to underline the limitations of the reasoning developed above. The analysis should not be limited to the diaphragm and the parasternal intercostals, which are primary muscles of inspiration in humans also [32], but other respiratory muscles need to be considered. These include, the triangularis sterni [33, 34], the transversus abdominis [35], the external and internal intercostals, the levators costae [36], the scalenes [37, 38] and the sternocleidomastoids [38]. The scalenes and sternocleidomastoids are not electrically active during quiet breathing in supine anaesthetized dogs at FRC or approaching TLC, but they may play a major role during respiration in patients with COPD [39, 40]. The scalenes should be considered as primary muscles of inspiration in man [37]. Their shortening with hyperinflation is also considerably less than the diaphragmatic shortening [41].

Hyperinflation may also modify the function of respiratory muscles in a way which is not related to length-tension considerations. The diaphragm flattens with hyperinflation and the zone of apposition diminishes [10] and these factors further reduce its mechanical effectiveness in generating pressures and expanding the rib cage. Hyperinflation further changes the mechanical interaction among costal and crural parts of the diaphragm, such that they become arranged more in series and less in parallel, which may contribute to the diaphragm’s failure as a pressure generator at high lung volumes [20]. Only scanty data are available on how hyperinflation affects the action of the external and internal intercostals [42, 43], whereas no data are presently available on how parasternal intercostal or accessory muscle force relates to rib cage motion and rib cage expansion at FRC and how these relationships are altered at elevated end-expiratory lung volume.

These new insights were obtained in experimental animals, and it remains uncertain how far these concepts can be extrapolated to humans and to patients with hyperinflation. Nevertheless, several studies have provided evidence to support the contention that in humans similar concepts presumably apply. Firstly, although maximal inspiratory pressure decreases sharply with increasing lung volumes [3, 44, 45], the actual pressure generated by the inspiratory musculature (i.e., the difference between maximal inspiratory pressure and the elastic recoil curve of the total respiratory system [45]) is clearly more independent of lung volume. This suggests that the pressure generating capacity of the inspiratory muscles is relatively well preserved with hyperinflation in humans also. Secondly, the pattern of chest wall motion and gastric pressure development during inspiration in normal subjects breathing at elevated end-expiratory lung volume is very similar to that observed in supine anaesthetized dogs, [46, 47]. Finally, in patients with COPD and severe hyperinflation, diaphragmatic shortening is more pronounced than intercostal and accessory muscle shortening [48], and these patients exhibit signs of clear diaphragmatic ineffectiveness correlating with the degree of airflow obstruction [48–50].

Our understanding of how respiratory muscle function is altered in patients with hyperinflation, however, remains limited and more data are required on the basic alterations induced in the extra-diaphragmatic respiratory muscles in patients. Recent evidence obtained in animal experiments clearly indicates that hyperinflation is detrimental to the mechanical effectiveness of the diaphragm but may be beneficial to the mechanical effectiveness of the parasternal intercostals. More information on other respiratory muscles and on the interaction between them is needed, however, to come to an integrated view on how the action of the vital pump is altered by increases in end-expiratory lung volume.

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References

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