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Hyperinflation and respiratory muscle interaction

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Hyperinflation and respiratory muscle interaction. M. Decramer. ©ERS Journals Ltd 1997.
ABSTRACT: Hyperinflation clearly affects respiratory muscle interaction. It commonly increases the rib cage contribution to chest wall motion, whilst it reduces the abdominal contribution.

This change is thought to result from the fact that hyperinflation severely reduces the mechanical advantage of the diaphragm, whilst it affects the mechanical advantage of the neck and rib cage muscles to a lesser extent. The mechanical disadvantage in the diaphragm induced by hyperinflation is presumably primarily the result of the length changes undergone by the diaphragm in acute hyperinflation. Changes in diaphragmatic geometry are generally considered to be less important in the reduction of the diaphragm's force-generating capacity.

Further factors contributing to the mechanical disadvantage in the diaphragm include a reduction in the appositional component of diaphragmatic action (through reduction in the zone of apposition), and a reduction in the insertional component (through a shift in the alignment of the diaphragmatic fibres from axial to radial).

In chronic hyperinflation, the diaphragm adapts to the chronically hyperinflated state. This adaptation to chronic foreshortening is similar to the adaptation occurring in the skeletal muscle. It is caused by a dropout of sarcomeres in series along the muscle fibres. It restores the force-generating capacity of the muscle, in part, but it reduces the capacity of the muscle to undergo length changes.

The mechanical advantage of the parasternal intercostals and the scalenes is possibly less affected, because the length changes undergone by these muscles during hyperinflation are smaller. The factors determining the mechanical advantage of the parasternal intercostals are complex.

Variables related to the mechanical advantage of the parasternal intercostals include: length changes; changes in angle between the parasternal intercostals and the sternum and between rib and sternum; and changes in mechanical arrangement among different parasternals. At present, it is difficult to develop an integrated view of these factors and of their change with hyperinflation.

Finally, hyperinflation commonly results in recruitment of expiratory muscles. The functional significance of this expiratory muscle recruitment in patients is still debated.
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The effects of hyperinflation on respiratory muscle function and on respiratory muscle interaction have interested respiratory physiologists for more than 30 yrs [1]. This area has recently been revived because of the more widespread application of volume reduction surgery [2]. Volume reduction surgery frequently results in functional improvement. Whether this is the result of improved pulmonary mechanics [3], improved ventilation/perfusion relationships, improved respiratory muscle function, or a combination thereof remains to be elucidated. In any event, improvement in respiratory muscle function has been demonstrated by several independent groups of investigators [4, 5].

If volume reduction surgery improves respiratory muscle function, then to understand the mechanisms of this improvement, insight into how hyperinflation affects the respiratory muscles is required. Despite the fact that the effects of hyperinflation on the respiratory muscles have been studied for over more than 30 yrs [1], several important mechanisms have only been demonstrated in the last decade. The present review will attempt to summarize the effects of hyperinflation on respiratory muscle function and, more specifically, on respiratory muscle interaction, since the other aspects of respiratory function will be covered in accompanying reviews. We will first discuss the effects of hyperinflation on respiratory muscle interaction, and will then explain the observed effects by the presumed effects of hyperinflation on the individual respiratory muscles.

Previous articles in this series: No. 1: G.J. Gibson. Pulmonary hyperinflation a clinical overview. *Eur Respir J* 1996; 9: 2640–2649. **No. 2:** E.W. Russi, U. Stammberger, W. Weder. Lung volume reduction surgery for emphysema. *Eur Respir J* 1997; 10: 208–218. **No. 3:** R. Pellegrino, V. Brusasco. On the causes of lung hyperinflation during bronchoconstriction. *Eur Respir J* 1997; 10: 466–475. **No. 4:** A. de Troyer. Effect of hyperinflation on the diaphragm. *Eur Respir J* 1997; 10: 708–713.

Effects of hyperinflation on respiratory muscle interaction

Hyperinflation has well-known effects on respiratory muscle interaction [6–8]. It has been shown in animals [6], in normal humans [9], and in patients with chronic obstructive pulmonary disease (COPD) [10], that hyperinflation clearly increases the contribution of the rib cage and neck muscles, and decreases the relative contribution of the diaphragm to chest wall motion (fig. 1). It is also known that hyperinflation must be severe before it clearly affects respiratory muscle interaction [6]. Evidently, hyperinflation is pronounced in patients with COPD, particularly in emphysematous patients [8].

In a study in supine anaesthetized and vagotomized dogs, we demonstrated that the alterations in respiratory muscle interaction induced by hyperinflation were not the result of alterations in neural output into the respiratory muscles, but instead resulted from alterations in mechanical effectiveness of respiratory muscles [6]. Indeed, although during breathing near total lung capacity (TLC) inspiratory changes in gastric pressure, diaphragmatic length changes and abdominal expansion, were all clearly reduced, signalling a clear reduction in the mechanical effect of diaphragmatic contraction, crural diaphragm integrated electromyographic activity (EMG) remained constant, and costal diaphragmatic EMG even increased by 25%. Conversely, although the relative inspiratory rib cage expansion increased with hyperinflation, parasternal intercostal integrated EMG was reduced by 25%. The present review will, thus, focus on the effects of hyperinflation on respiratory muscle interaction and not on the effects on respiratory muscle coordination.

In addition to increasing the relative contribution of the rib cage and neck musculature, expiratory muscle recruitment is commonly present in COPD patients, whether at rest [11, 12] or during exercise [13]. The functional

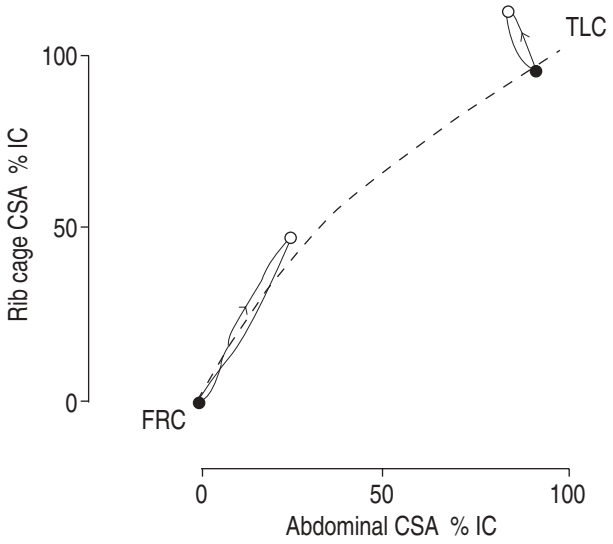

Fig. 1. – Supine anaesthetized dog breathing quietly at functional residual capacity (FRC) and total lung capacity (TLC). Rib cage cross-sectional area (CSA) as a percentage of inspiratory capacity (IC) vs abdominal CSA as a percentage of IC. Loop: tidal loop; dashed line: relaxation line. Note that the tidal loop falls close to the relaxation line at FRC, but clearly deviates from it at TLC. ●: end-expiration; ○: end-inspiration.

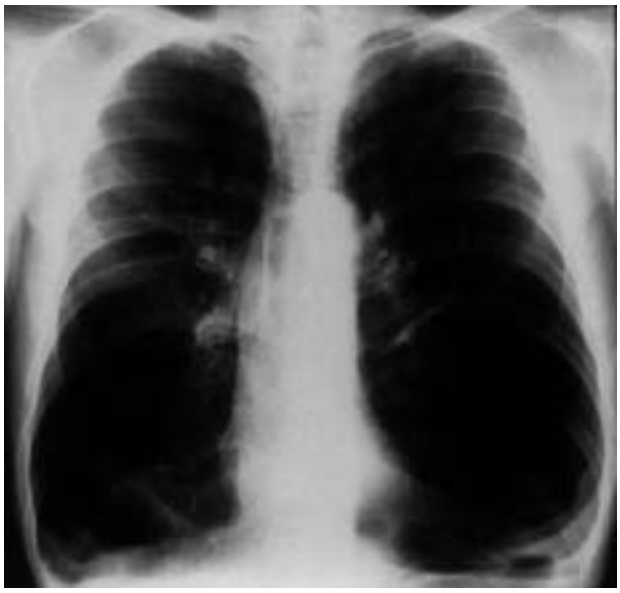

Fig. 2. – Chest radiograph of a patient with emphysema and severe hyperinflation. Notice excessive flattening of the diaphragm.

significance of this recruitment of expiratory muscles is not well understood. Indeed, in COPD patients expiration is often flow-limited, such that expiratory muscle recruitment cannot contribute to expiratory flow and, thus, cannot contribute to ventilation. Expiratory muscle recruitment, however, may affect the length and geometry of the diaphragm, such that the length at which the diaphragmatic contraction is initiated may be greater than it would otherwise be. Sudden relaxation at the onset of inspiration, however, is expected to quickly restore the diaphragm to its original length, such that the significance of the longer length at which contraction was initiated may be questioned.

Although hyperinflation, which is potentially extremely severe in COPD patients (fig. 2), clearly affects respiratory muscle interaction, the mechanisms of these alterations remain relatively obscure. In order to better understand the nature of the effects of hyperinflation on respiratory muscle interaction, we will successively examine the effects of hyperinflation on the diaphragm, the parasternal intercostals, scalenes and sternomastoids, and on the expiratory muscles. Finally, we will try to integrate these elements to form an overall view of how hyperinflation affects respiratory muscle function and respiratory muscle interaction.

Effects of hyperinflation on the diaphragm

It has been clearly shown that hyperinflation detrimentally affects the diaphragmatic force-generating capacity [14, 15]. This is thought to result from five different effects: on diaphragmatic length; on diaphragmatic geometry; on the magnitude of the appositional component of diaphragmatic action; on the insertional component of diaphragmatic action; and potential effects on the interaction between different parts of the diaphragm.

Hyperinflation is expected to foreshorten the diaphragm and, thus, to displace it to a suboptimal position of its length-tension curve. Data from experimental animals

[16, 17] and normal humans [18], indicate that the diaphragmatic shortening occurring between functional residual capacity (FRC) and TLC averages about 30–40%. This is a substantial change in length, in terms of a length-tension curve, and is expected to affect the diaphragmatic force-generating capacity to a significant extent. Since a large proportion of COPD patients have a FRC exceeding the TLC of normal subjects, a substantial diaphragmatic shortening is also expected in these patients [8]. It should be stressed, however, that if hyperinflation is chronic, the diaphragmatic length-tension curve will adapt to the hyperinflated state. This adaptation consists of a dropout of sarcomeres in series, and the end result is that the whole length-tension curve including the optimal length shifts to a shorter length. As a consequence, as the actual operating length shortens, the optimal length also shortens, and a certain match between the *in situ* operational length and the optimal length remains [19].

This adaptation is represented schematically in figure 3. The mechanism of this adaptation is a reduction in the number of sarcomeres in series. This adaptation takes about 4–6 weeks to develop; so that it is an adaptation to chronic hyperinflation [20], and does not constitute an adaptation to acute hyperinflation. Although there is no direct evidence that this adaptation also occurs in patients with hyperinflation, there is convincing indirect evidence that this is the case [21]. It should be stressed that the adaptation, in part, restores the force-generating capacity

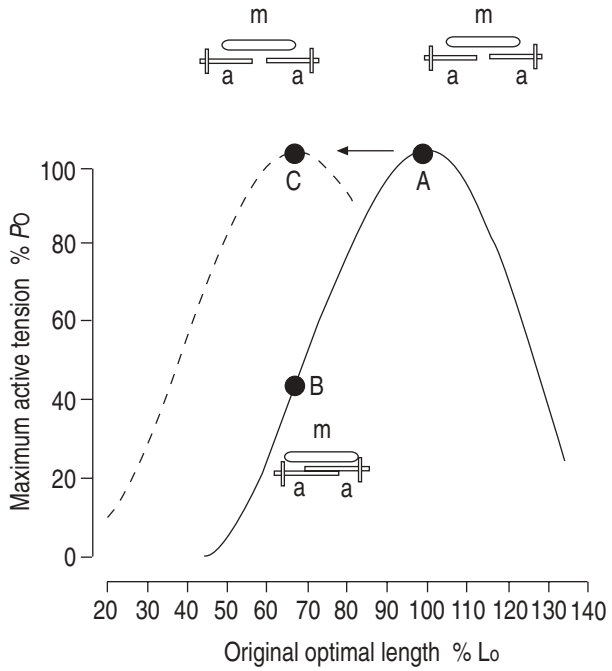

Fig. 3. – Diaphragmatic length-tension curve in normal (——) and emphysematous (– – –) hamsters. Length is expressed as a percentage of optimal length (Lo), and tension is expressed as a percentage of maximal tetanic tension (*P*o). A: control; B: acute shortening; C: chronic shortening. Degree of filament overlap between actin (a) and myosin (m) filaments in each of these conditions is shown. Note that the diaphragmatic length-tension curve in emphysematous hamsters is shifted to the left, which means that it shifts to shorter length. The consequence of this is that shortening with chronic hyperinflation does not occur from A to B as it does in acute hyperinflation, but from A to C. A and C are characterized by the same degree of filament overlap between the actin and myosin filaments, as shown. C is shorter, because at C there are fewer sarcomeres in series. (After Farkas [19]).

of the diaphragm but that, concomitantly, it reduces the capacity of the diaphragm to undergo displacements [22]. Indeed, sarcomeres along diaphragmatic fibres are arranged in series, such that the displacements of the diaphragm are the sum of the displacements undergone by the individual sarcomeres. If the number of sarcomeres is reduced, then the total displacement will be smaller. Whether this reduction in the number of sarcomeres in series will affect diaphragmatic contractile properties to a clinically significant extent remains to be examined [22].

It should be stressed that, in the reasoning developed above, we have implicitly assumed that the optimal length (Lo) for the diaphragm corresponds to a lung volume close to FRC. Experimental evidence produced by Road *et al.* [23] demonstrated, in supine anaesthetized dogs, that at FRC the costal diaphragm was distended to 105% Lo, while the crural diaphragm was shortened to 92% Lo. It should be noted, however, that the deviations from Lo at FRC in the diaphragm are small.

The length change occurring in the diaphragm with acute hyperinflation may have another significant consequence. Indeed, data obtained in animals, *in vitro* [24] and *in vivo* [25], as well as in humans [26], clearly demonstrate that the inotropic effects of theophylline are considerably greater on foreshortened muscle than on muscle placed at its optimal length. This relates to the mechanism of the drop in force induced by muscle shortening. Although, classically, this force decline is related to alterations in overlap between actin and myosin filaments, it has been clearly demonstrated that with foreshortening excitation-contraction failure also occurs. This is due to compression of the T-tubulus, blocking exit-electrolyte flow, and consequent failure of inward spread of the action potential, such that the central sarcomeres contract less than the peripheral ones [27]. Caffeine restores T-tubular function, presumably because it causes hyperpolarization of the cell membrane [28]. Whether, as a consequence, inotropic agents may result in considerably greater effects in COPD patients with acute hyperinflation, and whether these effects result in clinically significant improvements, remains to be examined.

On the basis of Laplace's law [29, 30], it is conventionally accepted that hyperinflation will also flatten the diaphragm and, consequently, reduce its force-generating capacity. Laplace's law states that the pressure developed by diaphragmatic contraction is directly proportional to the tension developed in the fibres of the diaphragm and inversely proportional to the radius of curvature of the diaphragm. If the diaphragm flattens and, therefore, the radius of curvature increases, diaphragmatic contraction will become less effective because a greater tension will be required to produce the same change in pressure. Although this reasoning is without question valid, a number of observations point to the fact that geometrical changes may be clearly less important to the reduction in force-generating capacity observed in patients with hyperinflation. Indeed, Kim *et al.* [14] demonstrated that the reduction in force observed in dogs with hyperinflation from FRC to TLC was almost entirely due to the change in length, and that changes in geometry occurring between FRC and TLC did not appear to affect the decline in force output of the canine diaphragm. Moreover, Gauthier *et al.* [18] demonstrated

with nuclear magnetic resonance (NMR) spectroscopy in normal humans that diaphragmatic shortening occurring between residual volume (RV) and TLC was about 40%. In addition, the force decline between RV and TLC observed previously in normal humans [15] could be entirely explained by the shift along the diaphragmatic length-tension curve.

As a consequence, it is commonly believed that changes in diaphragmatic geometry do not affect the decline in the diaphragm force-generating capacity with hyperinflation to a significant extent. This is confirmed by data from GAUTHIER *et al*. [18], who directly demonstrated by three-dimensional reconstruction of NMR images that there was little change in diaphragmatic geometry between FRC and TLC (fig. 4). No such data are available in patients, and no real spatial construction of the diaphragm has been made in patients with severe hyperinflation. At present, it is thus not possible to correctly evaluate the role of changes in diaphragmatic geometry in patients with hyperinflation.

The area of apposition, which is the zone of the diaphragm immediately apposed to the rib cage, is an important feature in relation to the expansion of the rib cage by the diaphragm. It has been well-demonstrated

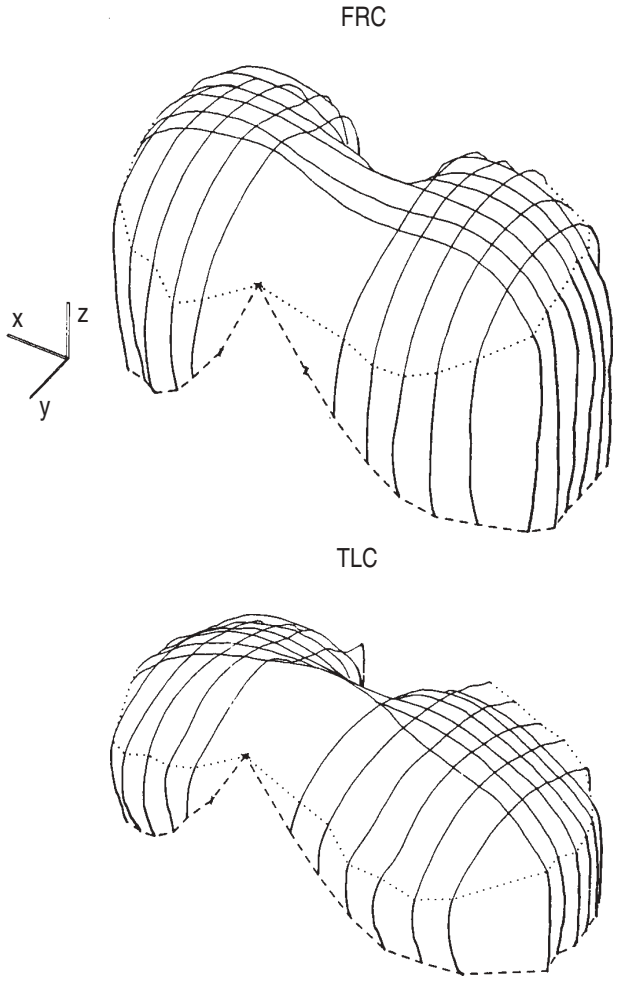

Fig. 4. – Three-dimensional reconstruction of diaphragmatic geometry at functional residual capacity (FRC) and at total lung capacity (TLC). Note that diaphragmatic geometry is relatively constant with hyperinflation from FRC to TLC. (After GAUTHIER *et al*. [18]).

that the area of apposition is largest at RV, smaller at FRC, and even smaller at TLC in supine anaesthetized dogs, as well as in normal subjects [31]. It is, thus, expected that the action of rib cage expansion is related to the magnitude of the zone of apposition, and will decrease progressively from RV to FRC and from FRC to TLC. At present, in patients with hyperinflation, it is difficult to evaluate how the reduction in the appositional component of diaphragmatic action is related to the overall reduction in force-generating capacity.

The action of the diaphragm in expanding the lower rib cage is dependent not only upon the appositional component mentioned above but also upon the insertional component, which is the action of the diaphragm on the lower rib cage through its insertions into the lower rib cage. With hyperinflation, the diaphragmatic fibres are expected to be progressively oriented in a more radial fashion than the normal axial alignment and, as a consequence, the insertional component may be expected to be reduced [29]. It should be noted, however, that the diaphragmatic geometry remains relatively constant during hyperinflation [18]. There is, however, no doubt that the action of the diaphragm in expanding the lower rib cage is clearly reduced with hyperinflation. Indeed, in patients with severe hyperinflation, there is inspiratory indrawing of the lower rib cage margin instead of inspiratory expansion [32, 33]. At present, it is difficult to evaluate the significance of the reduction in insertional component in patients with hyperinflation relative to the changes in the other components of diaphragmatic action.

Finally, MACKLEM *et al*. [34] hypothesized that if hyperinflation changed the mechanical arrangement between the costal and crural parts of the diaphragm, shifting them from a parallel arrangement at FRC to a series arrangement at TLC, the force-generating capacity of the diaphragm would be reduced in a way that was independent of its force-length relationship. Indeed, muscles arranged in parallel are designed to develop large forces, since the forces produced by the individual muscles are additive. Muscles arranged in series share the same force, but the displacements undergone are additive. If the costal and crural parts of the diaphragm were arranged in parallel at FRC and gradually move to a series arrangement at TLC, then the force-generating capacity of the diaphragm would be reduced with hyperinflation, on the basis of this alteration in mechanical arrangement. In keeping with this concept, DECRAMER and co-workers [35] demonstrated, in supine anaesthetized dogs, that the two parts of the diaphragm were arranged in parallel at FRC and progressively moved to a series arrangement at higher lung volumes. Whether such mechanism may be of significance in patients with COPD has, so far, not been studied.

Effects of hyperinflation on the parasternal intercostals

Hyperinflation produces clear effects on the parasternal intercostals, which are the most important inspiratory portion of the intercostal musculature [16, 36–38]. The effects of hyperinflation on the parasternal intercostals may be analysed in terms of changes in length, in

geometry or mechanical advantage, and in mechanical arrangement. The parasternal intercostals were shown to shorten by only about 7% between FRC and TLC in supine anaesthetized dogs [36]. This relatively modest shortening contrasts to the more substantial shortening observed in the diaphragm (see above). It should be noted, however, that this shortening may be underestimated. Indeed, DE TROYER and LEGRAND [37] demonstrated that, in supine anaesthetized dogs, the part of the parasternal intercostals that was most consistently active during the inspiration was the medial portion, which was always active more and before the middle portion, which in turn was activated more than the lateral portion. The recruitment threshold, thus, appeared to exhibit a gradient from medial to lateral. In addition, they demonstrated that the length changes with passive inflation were clearly greater in the medial bundles than in the lateral and middle bundles. In the medial bundles, they obtained a shortening with 1 L passive inflation of about 10%. This is more than observed previously [36], but still clearly less than diaphragmatic shortening occurring with similar hyperinflation.

A number of additional factors may influence the geometry and the mechanical advantage of the parasternal intercostals, and the change occurring between FRC and TLC or with hyperinflation. These include: the shape of the length-tension curve of the parasternal intercostals; the position of the optimal length of the parasternal intercostals; and the ratio of the angle between the parasternal intercostals and the sternum, and the angle between the ribs and the sternum.

With respect to the shape of the length-tension curve, FARKAS *et al.* [39] demonstrated that the range of operating lengths for the parasternal intercostals was considerably more narrow than the diaphragmatic range of operating lengths. This signifies that although the shortening occurring with hyperinflation is small, if the range of the operating lengths is considerably smaller as well, even a modest change in length might induce a severe mechanical disadvantage in the parasternal intercostals. In addition, FARKAS *et al.* [39] and JIANG *et al.* [40] demonstrated that, in supine anaesthetized dogs, the optimal length for the parasternal intercostals did not occur at a lung volume close to FRC, in contrast to the diaphragm [23]; instead, it occurred at a lung volume close to TLC. If these data are correct, hyperinflation may move the parasternal intercostals to their optimal length and, thus, may be beneficial for the parasternal intercostals rather than detrimental. These data have been disputed by DIMARCO and co-workers [41], who found, with a technique of ventral root stimulation, that there was a similar force decline in the parasternal intercostals and the interosseous intercostals with hyperinflation. As a consequence, the effects of hyperinflation on parasternal intercostal length and its relationship to its optimal length should be regarded as controversial.

An interesting point which remains is the extent to which geometrical alterations in the parasternal intercostals may affect their force-generating capacity over the vital capacity range. As demonstrated by DE TROYER and LEGRAND [37], the mechanical advantage of the parasternal intercostals is dependent on the angle between the parasternal intercostal and the sternum (β), and also on the angle between the rib and the sternum (α). The

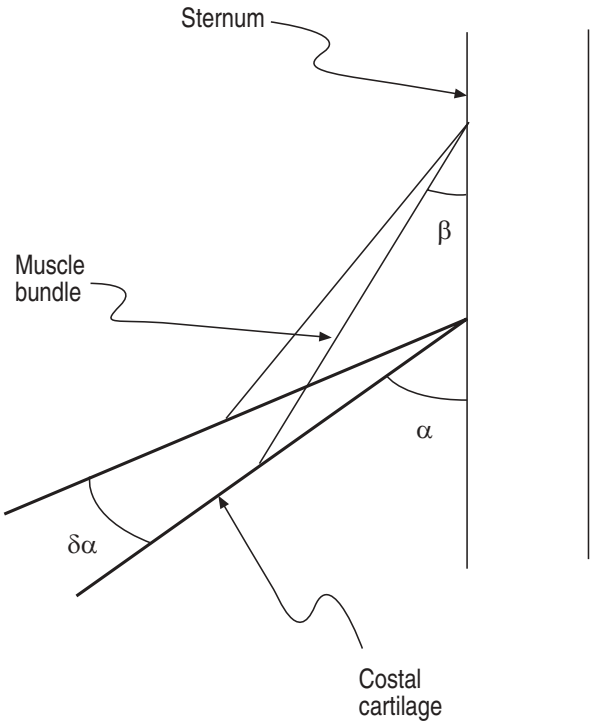

Fig. 5. – Diagram illustrating factors determining the mechanical advantages of the parasternal intercostals. Note that the mechanical advantage is dependent upon the relationship between the angles: α, between the rib (costal cartilage) and the sternum; and β, between the parasternal intercostals and the sternum. (After DE TROYER and LEGRAND [37]).

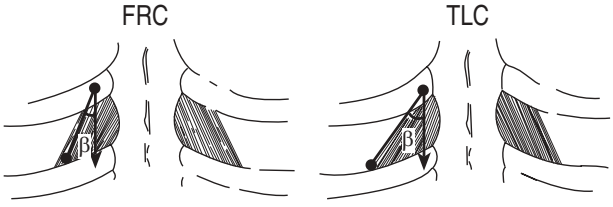

Fig. 6. – Change in the angle between the parasternal intercostals and the sternum (β) from functional residual capacity (FRC) to total lung capacity (TLC) in supine anaesthetized dogs (n=7). Note that with hyperinflation from FRC to TLC, β clearly increased (from 29±0.8 to 42±1.4°). This increase in β alone induces a reduction in mechanical advantage of about 15%. (After DECRAMER [7]).

relationship between these two angles determines the mechanical advantage of the parasternal intercostals, as demonstrated in figure 5. We measured the angle between the parasternal intercostals and the sternum at FRC and at TLC in seven supine anaesthetized dogs. These data are summarized in figure 6. We found that the angle between the parasternal intercostals and the midline clearly increased from 29±0.8° at FRC to 42±1.4° at TLC. This alone is expected to reduce the mechanical advantage of the parasternal intercostals by 15%. At present, we have no data on the geometrical alterations induced in the parasternal intercostals in patients with hyperinflation.

A final consideration on parasternal intercostal geometry relates to Laplace's law mentioned above. Indeed, the parasternal intercostals are situated at the periphery of the rib cage. For the parasternal intercostals to

produce changes in lung volume, they need to produce changes in pleural pressure or pressure changes at the inside of the rib cage cavity. The relationship between the tension developed at the periphery and the pressure changes inside the cavity will be given by Laplace's law, and will be directly proportional to the tension developed by the muscles and inversely proportional to the radius of curvature or, in other words, to the diameters of the rib cage. It is intuitively likely, if not obvious, that with hyperinflation the mechanical effectiveness of the parasternal intercostals is expected to change, such that a greater tension will be required at the periphery to produce the same change in pressure and, hence, the same change in lung volume. This mechanism was indirectly demonstrated by NINANE and GORINI [42], who demonstrated that with hyperinflation the fall in pleural pressure obtained after parasternal stimulation was clearly reduced, despite the fact that the mechanical effects on the ribs and the sternum were relatively well-preserved. Indeed, the sum of the cranial rib motion and the caudal sternum motion, reflecting the tension developed during breathing or during stimulation in their animals, remained relatively constant, while the swing in pleural pressure was clearly reduced. In addition, the relationship between the change in lung volume and the change in pressure, *i.e.,* the lung compliance, may change with hyperinflation. At present, it is unclear how these mechanisms may affect the mechanical effectiveness of the parasternal intercostals in patients with COPD, and how improvements after volume reduction surgery may be related to it. However, it appears likely that reducing the volume of the rib cage would beneficially alter the mechanical effectiveness of the rib cage muscles on the basis of such a mechanism.

As pointed out by MACKLEM *et al.* [34], the final element that may affect the characteristics of the vital pump is the mechanical arrangement among the different muscles. If the mechanical arrangement changes between the crural and the costal parts of the diaphragm, or between the diaphragm and the parasternal intercostals, or between different portions of the parasternal intercostals [43], then this is also expected to affect the outcome of the system as a whole. At present, there are no clear data on the relative significance of these mechanisms in affecting the mechanical output of the vital pump in COPD patients.

The respiratory role of the interosseous external and internal intercostal is not yet fully understood [41, 44, 45]. They play an important postural role in rotation of the trunk [44, 46]. Whether they play an important respiratory role with hyperinflation or in severely hyperinflated patients is unclear. In stimulation experiments in supine anaesthetized dogs, DE TROYER and co-workers [47, 48] demonstrated that hyperinflation may profoundly affect the action of the internal and external intercostals. How hyperinflation affects interosseous intercostal muscle function in patients is unclear.

Effects of hyperinflation on the scalenes and sternomastoids

There are few data on the effects of hyperinflation on the action of the scalenes and sternomastoids [49]. In a recent paper, DE TROYER and co-workers [49] demonstrated that there is rarely activity in the sternocleidomastoids in COPD patients with severe hyperinflation and airflow obstruction. Moreover, PECHE *et al.* [50] demonstrated that, in contrast to conventional thinking, there is no hypertrophy of the sternocleidomastoids in these patients. Their function as accessory muscles of inspiration might, thus, be questioned, at least in COPD patients. The scalenes are important primary muscles of inspiration in normal subjects, as well as in COPD patients [49, 51], and the length change they undergo with hyperinflation is relatively small, such that one would expect the mechanical advantage of these muscles to be less disturbed than the mechanical advantage of the diaphragm [52]. In humans, however, their position and the shape of their length-tension curve is not clearly known.

Effects of hyperinflation on the expiratory muscles

It is clear that in patients with severe hyperinflation and COPD, activity is often present in the expiratory muscles [11, 12]. The likelihood of this activity is greater as airflow obstruction proceeds [11]. The functional significance of this activity, however, is relatively poorly understood. As expiration in COPD patients is, on average, flow-limited, expiratory muscle activity cannot contribute to expiratory flow and, therefore, cannot contribute to ventilation. Expiratory muscle activity may be involved in maintaining diaphragmatic shape and diaphragmatic length [53]. The pattern of relaxation of the expiratory muscles at the onset of inspiration is expected to quickly restore diaphragmatic length and shape to the precontraction status. Expiratory muscle activity undoubtedly contributes to intrinsic positive end-expiratory pressure (PEEPi), and must be taken into account in its measurement [12].

Summary

Hyperinflation in COPD patients is, without question, detrimental to diaphragmatic function. The relative contribution to chest wall motion of the rib cage and neck muscles is clearly increased, while the diaphragmatic contribution is decreased. Data obtained in supine anaesthetized dogs demonstrate that these changes are not due to changes in neural input into the respiratory muscles, but rather to changes in mechanical effectiveness of the respiratory muscles. This suggests that hyperinflation would affect the mechanical advantage of the rib cage muscles and the accessory muscles in these patients less than the diaphragmatic mechanical advantage.

The effects of hyperinflation on the mechanical advantage of the parasternal intercostals are complex, and are difficult to evaluate comprehensively at present. The length changes undergone by the parasternal intercostals with hyperinflation appear to be considerably smaller than the diaphragmatic length changes. In addition, data obtained in supine anaesthetized dogs suggest that the optimal length for the parasternal intercostals may be close to TLC rather than FRC. If this were unequivocally the case, then hyperinflation may even be beneficial to the parasternal intercostals. Geometric alterations,

including the alterations in the angle between the parasternal and the sternum, and between the rib and the sternum, are likely to detrimentally affect the mechanical advantage of the parasternal intercostals with hyperinflation.

There are few data concerning the sternocleidomastoids and the scalenes. The sternocleidomastoids are rarely active in COPD patients. The length changes undergone by the scalenes with hyperinflation appear to be small, indicating that their mechanical advantage may be well-maintained.

It appears clear that hyperinflation has a very pronounced effect on the diaphragm. The most important factor affecting the diaphragm with hyperinflation appears to be the substantial shortening (30–40%) occurring in the diaphragm. It should be stressed that the diaphragm adapts to the chronic hyperinflated state by a dropout in sarcomeres, which restores the force-generating capacity at foreshortened length. Geometrical alterations are less important in reducing the force-generating capacity of the diaphragm. Hyperinflation also clearly reduces the effects of diaphragmatic contraction on the lower rib cage, through a reduction both in the insertional and appositional component of diaphragmatic action.

Hyperinflation is often associated with expiratory muscle activity, but the functional significance of expiratory muscle activity in COPD patients is poorly understood.

Finally, although lung volume reduction surgery unequivocally results in improved force-generating capacity of the respiratory muscles [4, 5], it is at present difficult to determine the precise mechanism by which this improved respiratory muscle function is obtained. Geometric factors beneficially altering the mechanical advantage of the parasternal intercostals and other rib cage muscles, and the volume reduction process altering the relationship between the tension developed at the periphery of the rib cage and the pressure resulting from it at the inside, may be important. But, clearly, more research is needed to understand the effect of volume reduction surgery on the respiratory muscles.

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