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Synthesis Review

Action of the diaphragm on the rib cage

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De Troyer A, Wilson TA. Action of the diaphragm on the rib cage. *J Appl Physiol* 121: 391–400, 2016. First published June 9, 2016; doi:10.1152/japplphysiol.00268.2016.—When the diaphragm contracts, pleural pressure falls, exerting a caudal and inward force on the entire rib cage. However, the diaphragm also exerts forces in the cranial and outward direction on the lower ribs. One of these forces, the "insertional force," is applied by the muscle at its attachments to the lower ribs. The second, the "appositional force," is due to the transmission of abdominal pressure to the lower rib cage in the zone of apposition. In the control condition at functional residual capacity, the effects of these two forces on the lower ribs are nearly equal and outweigh the effect of pleural pressure, whereas for the upper ribs, the effect of pleural pressure is greater. The balance between these effects, however, may be altered. When the abdomen is given a mechanical support, the insertional and appositional forces are increased, so that the muscle produces a larger expansion of the lower rib cage and, with it, a smaller retraction of the upper rib cage. In contrast, at higher lung volumes the zone of apposition is decreased, and pleural pressure is the dominant force on the lower ribs as well. Consequently, although the force exerted by the diaphragm on these ribs remains inspiratory, rib displacement is reversed into a caudal-inward displacement. This mechanism likely explains the inspiratory retraction of the lateral walls of the lower rib cage observed in many subjects with chronic obstructive pulmonary disease (Hoover's sign). These observations support the use of a three-compartment, rather than a two-compartment, model to describe chest wall mechanics.

mechanics of breathing; diaphragm insertional force; zone of apposition; Hoover's sign

THE DIAPHRAGM IS A DOME-SHAPED partition separating the thoracic and abdominal cavities. It consists of a central aponeurosis, the "central tendon," and a peripheral muscle sheet made up of muscular fibers that take origin from the circumference of the thoracic outlet and converge to insert into the central tendon. Depending on their origins, the muscle fibers of the diaphragm are conventionally grouped into two components: *1*) the crural (or vertebral) portion that originates from the lumbar spine and the aponeurotic arcuate ligaments and *2*) the costal portion that originates from the xiphoid process of the sternum and the upper margins of the lower rib pairs. From their origins on the ribs, the muscle fibers of the costal portion run in the cranial-dorsal direction along the inner surface of the rib cage, as shown in Fig. 1. The region where they are directly apposed to the lower rib cage constitutes the so-called "zone of apposition," (47) which, in humans at resting end-expiration, represents about 40% of the total surface of the rib cage (48) and 60% of the total surface area of the muscle (28, 51).

When the muscle fibers of the diaphragm are activated during inspiration, they develop tension and shorten. As a result, the axial length of the apposed diaphragm diminishes, and the dome, which is primarily made up of the central tendon, descends. This descent produces both an expansion of the pleural cavity and a caudal displacement of the abdominal viscera leading to an outward motion of the ventral wall of the abdomen. Consequently, intrapleural pressure (Ppl) falls, lung volume increases, and abdominal pressure (Pab) rises. Tension in the diaphragm sheet generates an effective transdiaphragmatic pressure (Pdi) that balances the difference between Pab and Ppl ($Pdi = Pab - Ppl$).

Moreover, the diaphragm displaces the rib cage as it contracts. The characteristics of this displacement and the mechanisms involved have been controversial throughout medical history from Galen (approximately AC 130-200) through Vesalius (1514–1564) and Duchenne (24) to Goldman and Mead (32) [see Derenne et al. (16) for review]. However, observations in humans breathing with the diaphragm alone and recent studies in dogs have led to significant progress in the assessment and understanding of the action of the diaphragm on the rib cage. This review summarizes these recent developments. Thus, in the following sections, we first describe the rib cage displacements produced by isolated contraction of the diaphragm and analyze the mechanisms of these displacements. We also analyze the effect on these displacements of passive mechanical support of the abdomen, the influence of body position, and the effect of lung inflation, as occurs in subjects

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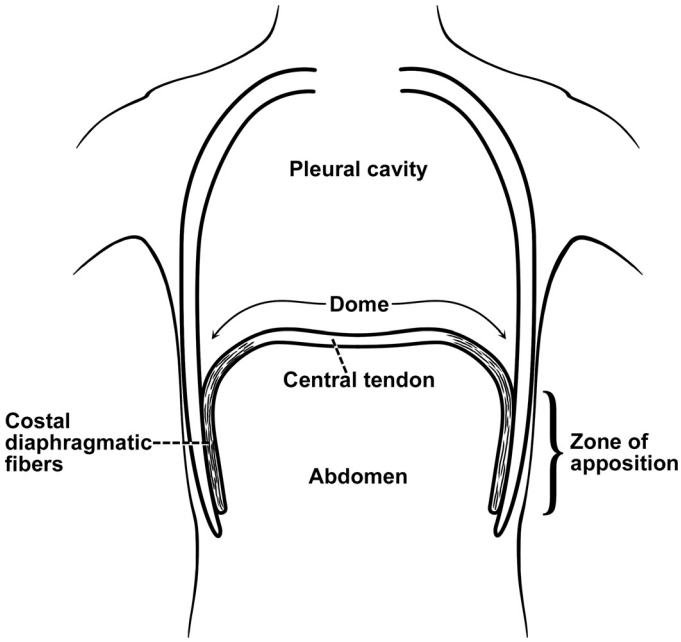

Fig. 1. Coronal section of the human chest wall at end-expiration. The muscle fibers of the diaphragm take origin from the circumference of the thoracic outlet, in particular the lower rib pairs, and converge to insert into a central aponeurosis (the central tendon). Note that from their origins on the lower ribs, the fibers run mainly cranially and are directly apposed to the inner aspect of the lower rib cage, constituting the "zone of apposition."

with chronic obstructive pulmonary disease (COPD). We finally discuss the application of these recent developments to modeling the chest wall and suggest future experiments that might allow key variables of the diaphragm-rib cage interaction in humans to be quantified.

Rib Cage Displacements Caused by Diaphragm Contraction

Human studies. Measurements of thoracoabdominal motion in supine healthy individuals requiring spinal or epidural anesthesia up to T1 for genitourinary surgery (26) and in seated subjects with quadriplegia due to complete, traumatic transection of the cervical cord at the C5–C6 level (27, 49, 57), i.e., in subjects in whom the diaphragm is the only muscle active during quiet inspiration, have shown that the diaphragm acting alone in humans causes an increase in the anteroposterior and transverse diameters leading to an increase in the cross-sectional area of the lower rib cage and a decrease in the diameters and cross-sectional area of the upper rib cage. Fig. 2*A* shows the records obtained during resting breathing in a representative quadriplegic subject with a complete C5 transection in the seated posture. Studies in subjects with traumatic transection of the upper cervical cord (C1–C2) and pacing of the phrenic nerves have also established that the diaphragm in humans has both an inspiratory action on the lower rib cage and an expiratory action on the upper rib cage (15, 55).

Animal studies. The mechanisms of these actions were initially investigated using tetanic, supramaximal stimulation of the phrenic nerves in the neck in supine anesthetized dogs and rabbits. In agreement with observations in quadriplegic subjects, phrenic nerve stimulation in both animals produced a caudal displacement of the upper ribs and a decrease in the cross-sectional area of the upper rib cage (14, 17). However, in contrast to quadriplegic subjects, phrenic nerve stimulation in animals also produced a caudal and inward displacement of the lower ribs and a decrease in the cross-sectional area of the lower rib cage (14, 44).

Tetanic, supramaximal stimulation of the phrenic nerves is useful to study the mechanics of the diaphragm because it provides a constant, well-defined level of muscle activation without simultaneous contraction of other muscles. However, this technique is unphysiological for two reasons. First, it produces a distribution of activity among the different areas of the diaphragm that is different from that during spontaneous contraction. In the dog, for example, whereas the entire diaphragm is activated during spontaneous breathing (9, 37), stimulation of the phrenic nerves in the neck involves the C5 and C6 but not the C7 nerve roots. Because the C7 roots supply a large part of the crural portion of the canine diaphragm (43), this implies that such stimulation causes nearly complete activation of the costal diaphragm but only partial activation of the crural diaphragm. Second, tetanic stimulation of the phrenic nerves (stimulation frequencies set between 20 and 100 Hz) causes extreme shortening of the muscle fibers of the costal diaphragm compared with quiet breathing. Specifically, whereas the muscle fibers in anesthetized or unanesthetized dogs shorten by 5–15% of their resting, end-expiratory length during quiet inspiration (8, 25, 54), during isolated supramaximal phrenic nerve stimulation at functional residual capacity (FRC), the muscle shortens by 40% or more (6, 20, 25, 36, 44). During stimulation, therefore, the dome of the diaphragm descends markedly, and the zone of apposition all but disappears, so that most of the lower rib cage becomes apposed to the lung, rather than the abdominal cavity.

The shortcomings of phrenic nerve stimulation, combined with the differences in lower rib cage motion observed during stimulation in animals and during spontaneous breathing in quadriplegic subjects, prompted the development of a new experimental preparation. In this preparation, the parasternal intercostal and external intercostal muscles in all interspaces were severed in anesthetized dogs, and measurements of rib displacement were made while the animals were in the supine posture and breathing spontaneously (17, 18). Consequently, as the inspiratory intercostals were eliminated from the act of breathing (19), the diaphragm was the only muscle active during inspiration, and the normal spatial distribution of neural drive among the different areas of the muscle was maintained. As is shown in Fig. 2*B*, the lower ribs in these animals moved cranially and outward during inspiration while the upper ribs moved caudally and inward; the sternum (not shown in the figure) was essentially stationary. The rib displacements in this preparation, therefore, were fully consistent with the changes in rib cage diameters observed in quadriplegic subjects, and this result was important because it confirmed that the lower rib displacements observed during phrenic nerve stimulation were related to the pattern and magnitude of diaphragm contraction during stimulation, rather than to a species difference in the diaphragm-rib cage interaction. The new preparation therefore appeared well suited to assess the mechanisms of the inspiratory action of the diaphragm on the lower rib cage and its expiratory action on the upper rib cage.

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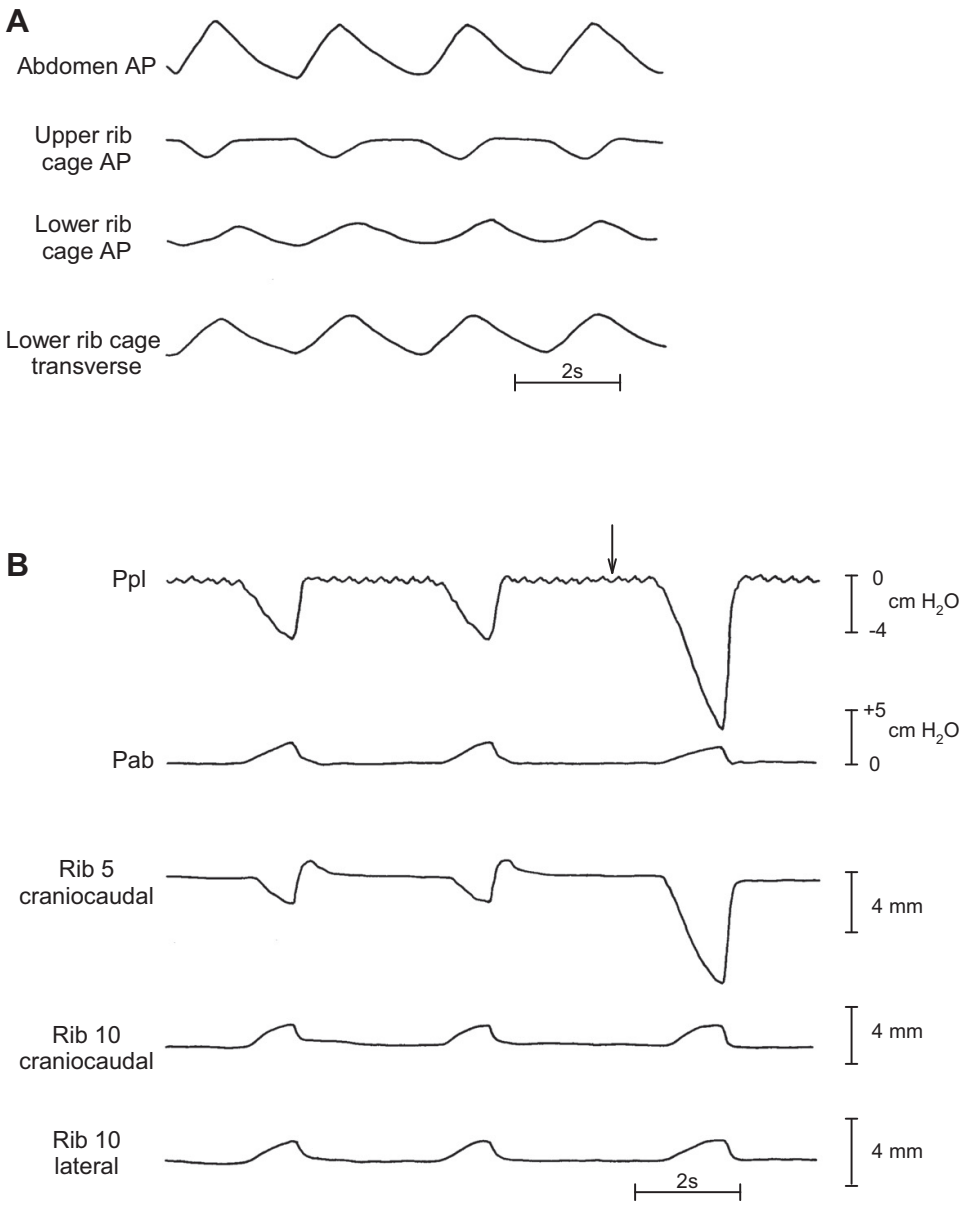

Fig. 2. *A*: pattern of rib cage motion in a representative C5 female quadriplegic subject breathing at rest in the seated posture. The respiratory changes in anteroposterior (AP) diameter of the lower rib cage, upper rib cage, and abdomen are shown, as well as the changes in the transverse diameter of the lower rib cage. Note that the lower rib cage diameters increase in phase with the abdomen (upward deflections) during inspiration, whereas the AP diameter of the upper rib cage decreases (downward deflection). *B*: displacement of the upper ribs (rib 5) and lower ribs (rib 10) in a vagotomized dog breathing with the diaphragm alone. The traces of pleural pressure (Ppl) and abdominal pressure (Pab) are also shown. Rib 10 moves in the cranial and outward direction (upward deflections) both during unimpeded inspiration and during single occluded breaths (breath after arrow), whereas rib 5 moves in the caudal direction (downward deflection).

Mechanism of Diaphragmatic Action on the Lower Rib Cage

Based on the observation that the costal and crural portions of the canine diaphragm displace the lower rib cage differently during selective stimulation (21) and on an analysis of a model of the chest wall in humans (45), the concept has emerged that the inspiratory action of the diaphragm on the lower rib cage is the result of two components of the force developed by the muscle. The first component, denoted the "insertional force," is related to the attachments of the muscle fibers of the costal portion of the diaphragm to the lower ribs. Thus, because the fibers run in the cranial-dorsal direction from their attachments, they exert a direct, cranially oriented force on these ribs when they contract. The second component, denoted the "appositional force," is due to the transmission of abdominal pressure to the lower rib cage in the zone of apposition. Indeed, as shown in Fig. 1, the existence of such a zone makes the lower rib cage, in effect, part of the abdominal container, and measurements in dogs have confirmed that during resting breathing, the changes in pressure in the pleural recess between the apposed diaphragm and the lower rib cage are nearly equal to the changes in Pab (56). The pressure rise in this pleural recess during inspiration therefore has the effect of pushing the lower ribs laterally.

In their model analysis, Loring and Mead (45) described the total effective pressure acting on the rib cage during diaphragm contraction (Peff) as the sum of Ppl and the effect of the force exerted by the muscle, which is proportional to Pdi, by the equation

$$\text{Peff} = \text{Ppl} + B \cdot \text{Pdi} \qquad (1)$$

From their modeling, they estimated that the coefficient B (describing the effect of a unit ΔPdi on rib displacement

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relative to the effect of a unit ΔPpl) was 0.65. They also estimated that the insertional component of diaphragm force contributed ~25% of the value of *B* and that the appositional component contributed ~75%.

These estimates, however, were obtained from a model of the chest wall that had two significant shortcomings. First, the model contained the assumption that the rib cage moves with a single degree of freedom, i.e., moves as a unit, so that its motion can be represented in terms of a single variable such as its anteroposterior dimension. The observations in humans with quadriplegia and in dogs breathing with the diaphragm alone have clearly established, however, that the diaphragm displaces the lower rib cage and the upper rib cage differently and, thus, that this assumption was invalid. Second, Loring and Mead (45) recognized that the insertional force of the diaphragm was only partly acting on the dorsal parts of the ribs (where the force has a small moment around the axis of rib rotation), but they did not take into account the fact that the effect of the appositional force is also smaller along the dorsal portion of the rib cage. Therefore it would be expected that the value of the coefficient *B* for the lower rib cage would be >0.65 and that the contribution of the appositional force would be lower than 75%.

On the basis of these considerations, Wilson and De Troyer (62) recently measured the displacements of the lower ribs along the craniocaudal and the mediolateral axis and the changes in Ppl and Pdi in supine anesthetized dogs during breathing with the diaphragm alone and during passive inflation. Following Loring and Mead (45), they described the total (cranial plus lateral) rib displacement (Z) as an equivalent of rib compliance, *A*, multiplied by the effective pressure (ΔPpl + *B*·ΔPdi).

$$Z = A \times \left(\Delta\mathrm{Ppl} + B \cdot \Delta\mathrm{Pdi}\right) \quad (2)$$

From the data obtained in the two maneuvers, they determined that *A* was 0.86 ± 0.09 mm/cmH_2O and that *B* was 0.98 ± 0.06. Thus, in supine dogs, *B* at FRC is ~1.0, and the effects of a unit change in Ppl and a unit change in Pdi on the lower ribs are equal. With these values of the parameters, the rib displacements that would be produced by ΔPpl acting alone and ΔPdi acting alone were then calculated. Figure 3 shows the displacements corresponding to the values of ΔPpl and ΔPdi measured during spontaneous diaphragmatic breathing and displays the key feature that the net displacement of the lower ribs, albeit relatively small, is determined by the balance between large, but nearly equal and opposite, effects of ΔPpl and ΔPdi. In other words, ΔPpl has a large expiratory effect on the ribs, but this effect is outweighed by the large inspiratory effect of ΔPdi, so that the ribs are displaced in the cranial and outward direction.

In the second stage of their study, Wilson and De Troyer (62) applied external forces to the lower ribs first in the cranial-dorsal direction, then in the lateral direction to simulate, respectively, the effects of the insertional and appositional forces, and the rib trajectories for these external forces were then compared with the rib displacement during spontaneous diaphragmatic breathing. As shown by the data obtained in a representative animal in Fig. 4, pulling the lower rib pair (the 10th pair in this case) in the lateral or the craniodorsal direction displaced the ribs both cranially and outward, but the outward rib displacement associated with a given cranial displacement

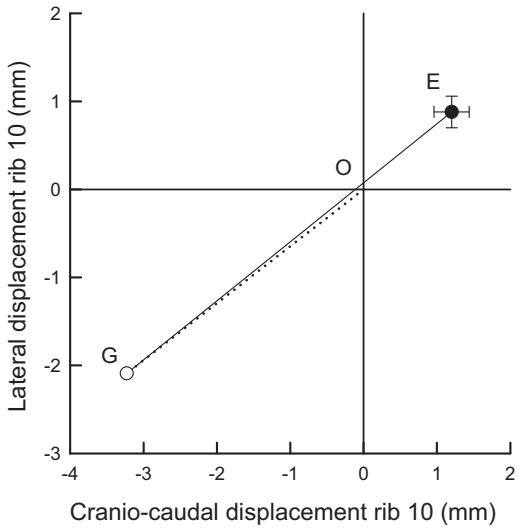

Fig. 3. Trajectory of rib 10 displacement produced by pleural and transdiaphragmatic pressure during spontaneous diaphragmatic breathing in dogs. The displacement of the rib along the craniocaudal axis is shown on the abscissa, and its displacement along the mediolateral axis is shown on the ordinate; positive values correspond to displacements in the cranial and outward direction, and negative values correspond to displacements in the caudal and inward direction. ● (point E) represents the mean ± SE displacement of the rib measured during spontaneous unimpeded diaphragmatic breathing in eight dogs, and ○ (point G) represents the position of the rib that would have occurred if the change in transdiaphragmatic pressure (ΔPdi) were zero. The dotted line O-G therefore is the rib displacement produced by the change in pleural pressure (ΔPpl) alone, and the continuous line G-E is the rib displacement due to ΔPdi alone. [Reproduced from Wilson and De Troyer (62).]

was about twice as large when the external force was applied in the lateral than the craniodorsal direction. In addition, the rib trajectory during spontaneous diaphragmatic breathing lay between the two trajectories produced by external forces. This confirmed that the force exerted by the diaphragm on the lower ribs has both an insertional component and an appositional component, and when the trajectories for external forces were used as the basis for a vector analysis to obtain the relative contributions of the insertional and appositional forces to the rib displacement driven by the diaphragm during spontaneous diaphragmatic breathing, the conclusion emerged that in supine dogs, the two components contribute nearly equally to the lower rib displacement produced by the diaphragm.

Mechanism of Diaphragmatic Action on the Upper Rib Cage

As previously pointed out, selective, tetanic stimulation of the phrenic nerves in dogs and rabbits produces a caudal displacement of the upper ribs (14, 17). However, when the stimulation is repeated after bilateral pneumothoraces have been introduced so as to uncouple the rib cage from the lung, the upper ribs remain stationary. As a result, the view has long been held that the action of the diaphragm on the upper rib cage is exclusively determined by the fall in Ppl.

To be sure, it would be expected that the effect of the insertional and appositional forces would decrease as one moves cranially, away from the muscle origins on the lower ribs and the zone of apposition. Studies in dogs breathing with the diaphragm alone, however, have shown that the caudal displacement of the upper ribs during inspiration is slightly increased when an external force is applied to the lower ribs to

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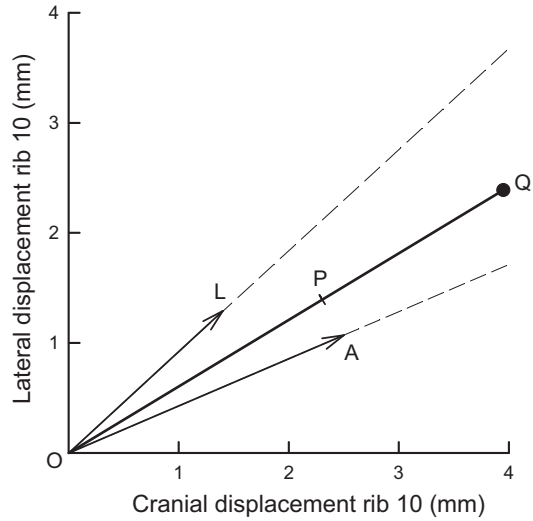

Fig. 4. Comparison between the trajectory of rib 10 during spontaneous diaphragmatic breathing and the trajectories during the application of external forces. The continuous line O-Q corresponds to the rib trajectory driven by a ΔPdi of 5 cmH_2O in a representative animal breathing with the diaphragm alone, and the dashed lines correspond to the rib trajectories obtained during the application of external forces to the rib in the lateral direction (upper line) and the cranial-dorsal direction (lower line). The lines O-A and O-L are the vectors along these trajectories that are the components of the resultant O-Q, and the segment O-P is the component O-A projected onto the resultant O-Q. The ratio of the length of this segment to the length O-Q therefore is the fraction of rib displacement contributed by the insertional force, and the ratio of the length of the segment P-Q to the length O-Q is the fraction of the rib displacement contributed by the appositional force. [Reproduced from Wilson and De Troyer (62).]

prevent them from moving cranially (18). Also, when the animals were made apneic and the lower ribs were displaced in the cranial direction by external forces, the upper ribs moved cranially even though the maneuver was associated with a fall in Ppl. When the maneuver was then repeated in the presence of pneumothorax, so that the fall in Ppl was eliminated, the cranial displacement of rib 5 was ~25% of the displacement of rib 10 (18).

These observations confirm that the force exerted by the diaphragm on the lower ribs is transmitted, via the rib cage tissues and the sternum, to the upper ribs, and they lead to the conclusion that the action of the diaphragm on the upper ribs is determined not exclusively by the fall in Ppl as conventionally thought but also by the displacement of the lower ribs. In the dog, whereas the coefficient of proportionality of ΔPdi (*B* in *Eq. 2*) is ~1.0 for the lower ribs, for the upper ribs, it is ~0.25. It also follows that if the caudal displacement of the upper ribs during isolated phrenic nerve stimulation disappears after pneumothorax, this is not only because the direct effect of ΔPpl on these ribs is eliminated but also because the caudal displacement of the lower ribs due to ΔPpl is abolished.

Effect of Abdominal Support

When the abdomen in quadriplegic subjects is given a passive mechanical support by a pneumatic cuff or an elastic binder placed below the epigastrium, the expansion of the lower rib cage during inspiration increases, and the inward displacement of the upper rib cage is reduced (15, 55, 57). With abdominal support, quadriplegic subjects also have an increase in ΔPab and ΔPdi during inspiration and during transcutaneous stimulation of the phrenic nerves with single twitches (34, 59, 60). Similarly, in supine dogs breathing with the diaphragm alone, application of a plate on the ventral abdominal wall at end-expiration causes an increase in ΔPab and ΔPdi during inspiration, a marked increase in the cranial and outward displacement of the lower ribs, and a decrease in the caudal displacement of the upper ribs while neural inspiratory drive remains unchanged (23).

The facilitatory effect of abdominal support on the inspiratory action of the diaphragm on the lower rib cage has traditionally been attributed to an increase in the appositional force. To be sure, because such a support causes an increase in abdominal elastance, the descent of the dome of the diaphragm in response to a given muscle activation is reduced, so that the zone of apposition throughout inspiration is larger, and ΔPab is greater. The appositional force therefore is greater. However, by reducing the descent of the dome, the abdominal support also opposes the shortening of the diaphragmatic muscle fibers. Consequently, it allows the fibers to operate on a more advantageous portion of their length-tension curve and, thus, to exert greater force at their attachments to the lower ribs. Indeed, in dogs breathing with the diaphragm alone, the plate applied on the ventral abdominal wall caused both an increase in ΔPdi and an increase in the coefficient *B* for the lower ribs (from ~1.0 to ~1.2), and the vector analysis of the rib trajectory showed that the insertional force and the appositional force each contributed about half of the increased inspiratory displacement of these ribs (23). The increased displacement of the lower ribs, in turn, appeared to be the primary determinant of the decreased caudal displacement of the upper ribs.

Effect of Body Position

The rib cage displacements observed in quadriplegic subjects are also affected by changes in body position. Specifically, when such subjects are tilted from the seated to the supine posture, the lower rib cage continues to expand along its transverse diameter during inspiration, but the increase in anteroposterior diameter is nearly abolished. In many supine quadriplegic subjects, the anteroposterior diameter of the lower rib cage, in fact, slightly decreases during the first part of inspiration (15, 27, 49). In addition, although the anteroposterior diameter of the upper rib cage decreases during inspiration in both postures, the magnitude of the decrease is commonly greater in the supine than in the seated posture (49, 55).

The smaller expansion of the lower rib cage and larger retraction of the upper rib cage produced by the diaphragm in the supine posture are likely related to the substantially lower abdominal elastance in this posture (10, 13, 28, 42). That is, with a decrease in abdominal elastance, the descent of the dome of the diaphragm in response to a given muscle activation is greater, so that lung expansion and ΔPpl are greater. Measurements in subjects with transection of the upper cervical cord and pacing of the phrenic nerves have provided clear evidence of this mechanism (15, 55). When the subjects were supine, the unassisted paced diaphragm was able to generate an adequate tidal volume, but when they were tilted head-up or moved to the seated posture, the tidal volume (and ΔPpl) produced by pacing decreased by 40% or more relative to the

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supine posture. In addition, a decrease in abdominal elastance leading to an increase in the descent of the dome of the diaphragm implies that the decrease in the zone of apposition and the amount of muscle shortening are greater and also that ΔPab is smaller. As a result, even though the zone of apposition along the dorsal portion of the rib cage is larger at end-expiration in the supine than in the seated posture (27), it is possible that the insertional and appositional forces of the diaphragm on the lower ribs are less than in the seated posture. The rib displacements produced by the diaphragm in head-up dogs, however, have not been studied, so this possibility remains to be tested.

Effect of Lung Inflation

Like subjects with quadriplegia, healthy individuals have an expansion of the lower rib cage during inspiration, but subjects with severe COPD (GOLD 3 or 4) and lung hyperinflation commonly have an inspiratory inward displacement of the lateral walls of the lower rib cage both at rest and during exercise (2, 4, 30, 38). This paradoxical displacement, conventionally called "Hoover's sign," has traditionally been attributed to the direct action of the diaphragm on the lower ribs (31, 35, 38). That is, because the diaphragm in these subjects is low and the zone of apposition is small, it was argued that the muscle fibers of the costal diaphragm became radially oriented during inspiration and therefore pulled the ribs inward, rather than cranially. This mechanism, however, has not been validated. More important, studies of diaphragm configuration using posteroanterior and lateral chest radiographs have shown that in subjects with COPD and severe hyperinflation, the zone of apposition at FRC [near predicted total lung capacity (TLC)] is smaller than in healthy individuals, but not eliminated (46, 53). In fact, the diaphragm in such subjects is still apposed to the lower rib cage at higher lung volumes, midway between FRC and TLC (53). Recent studies using ultrasonography have also shown that when breathing at rest, subjects with COPD, including those with Hoover's sign, still have a zone of apposition at end-inspiration (33, 52). Apparently, therefore, contrary to the conventional wisdom, Hoover's sign would not be the result of an inward pull of the lower ribs by radially oriented diaphragm muscle fibers.

The effect of lung inflation on the action of the diaphragm on the lower ribs was assessed in dogs breathing with the diaphragm alone (22). Thus a bilateral cervical vagotomy was made to avoid the apnea and prominent expiratory muscle activity that lung inflation would otherwise elicit (5, 39), and the displacements of the lower ribs, ΔPpl and ΔPdi, were measured while the animals performed single inspiratory efforts against an occluded airway at FRC and after passive inflation to different levels of transrespiratory pressure (Ptp). The ribs moved cranially and outward during occluded breaths at FRC, as shown in Fig. 2*B*. As lung volume (Ptp) before occlusion was progressively increased, however, the changes in Ppl and Pdi during occluded breaths decreased progressively and continuously, and the cranial-outward displacement of the ribs decreased rapidly and was then reversed into a caudal-inward displacement (22).

The coefficients in *Eq. 2* were evaluated for each value of Ptp, and the rib displacements produced by ΔPpl and ΔPdi acting alone were calculated. These are shown in Fig. 5. The rib displacement produced by ΔPpl was caudal and inward at all lung volumes and gradually decreased in magnitude as lung volume before diaphragm contraction was progressively increased above FRC. The rib displacement produced by ΔPdi also decreased with increasing lung volume, and the direction of this displacement was cranial and outward at all lung volumes, including when TLC (Ptp = +25 cmH_2O) was approached. However, the displacement of the lower ribs is the result of the balance between the inward force exerted by ΔPpl and the cranial and outward force exerted by the diaphragm, and with increasing lung volume, the balance between the effects of the two forces changes. In supine dogs at FRC, *B* for the lower ribs equals 1.0, and the effect of the force exerted by the diaphragm dominates, so that the net rib displacement is cranial and outward. At Ptp = +10 cmH_2O, $B = 0.85$, and the effects of the two forces are equal. As a result, although the effect of the force exerted by the diaphragm remains inspiratory, the net cranial-outward rib displacement is abolished. At Ptp > +10 cmH_2O, $B < 0.85$, and the net rib displacement is caudal and inward. These results, combined with the radiographic observations by McKenzie et al. (46) and Singh et al. (53) and the ultrasonographic observations by Gorman et al. (33) and Priori et al. (52), lead to the conclusion that Hoover's sign is, in fact, merely caused by the decrease in the zone of apposition that is known to occur with increasing lung volume (7, 50, 51) and, thus, by the dominant effect of ΔPpl on the lower ribs.

Modeling of the Chest Wall

The chest wall and the respiratory muscles constitute a complex system. Studies over the last four decades have generated large amounts of data on restricted aspects of the system, in particular the pressure changes and the rib cage displacements produced by isolated contraction of different muscle groups. Integrating from these data the various mechanisms that govern the displacements of the system during breathing, however, remains difficult. A model of the chest wall that incorporates and integrates the information from the different studies would provide a comprehensive understanding of the system.

In 1967, Konno and Mead (41) measured the displacement of a number of points on the ventral surface of the rib cage and abdominal wall for different breathing maneuvers in healthy individuals. They found that the points on the rib cage moved synchronously and that the ratio of the displacements of different points was the same for all maneuvers. A nearly similar finding was made for the points on the abdominal wall. Since this work, the concept of the chest wall as consisting of two compartments, the rib cage and abdomen, each with a single degree of freedom, has been a cornerstone of the study of chest wall mechanics.

A schematic diagram of a two-compartment model of the chest wall is shown in Fig. 6. The cross-hatched walls in the diagram represent the stationary foundation, and the solid walls are pistons that represent the rib cage (rc), diaphragm (di), and abdomen (ab). The pistons have displacements X_i and are connected to the foundation by springs with spring constants k_i. The rib cage inspiratory muscles (RCM) are connected to the rib cage piston and to the foundation, and the diaphragm muscle (DIA) is connected to the diaphragm piston and to a

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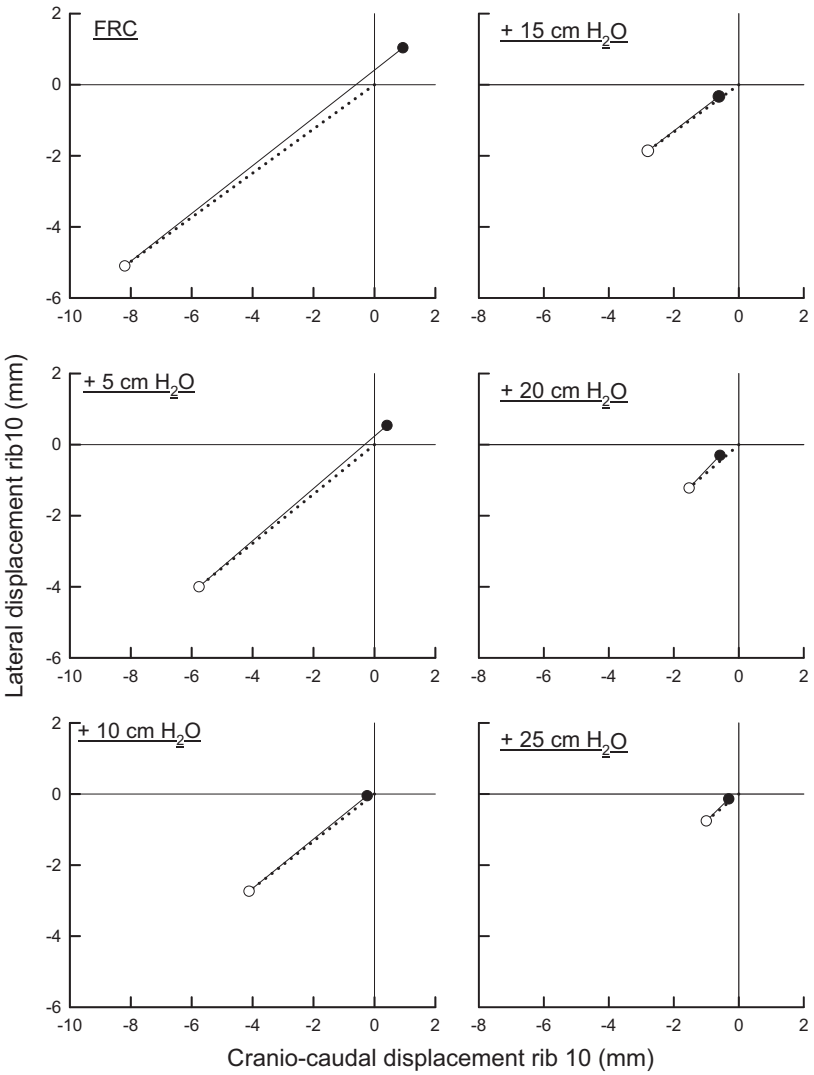

Fig. 5. Trajectories of rib 10 displacement produced by pleural and transdiaphragmatic pressure during isolated diaphragmatic contraction at different lung volumes. The different panels correspond to the different lung volumes, and numbers at the top left refer to transrespiratory pressure (Ptp) before diaphragm contraction. In each panel, the ● represents the mean rib displacement measured during single occluded breaths in seven dogs, and ○ corresponds to the mean position of the rib that would have occurred if the change in transdiaphragmatic pressure (ΔPdi) were zero. The dotted lines therefore represent the rib displacement produced by the change in pleural pressure (ΔPpl) alone, and the continuous lines represent the rib displacement produced by ΔPdi alone. Note that the rib displacement produced by ΔPdi decreases with increasing lung volume but remains cranial and outward at all lung volumes. The cranial and outward net rib displacement at FRC (Ptp = 0), however, decreases rapidly as lung volume increases from FRC to +10 cmH_2O and is reversed into a caudal and inward displacement as lung volume increases further. [Reproduced from De Troyer and Wilson (22).]

shelf on the rib cage. As a result, the force exerted by RCM during contraction displaces the rib cage piston upward and causes a fall in Ppl, while the force exerted by DIA displaces both the diaphragm piston downward, leading to a fall in Ppl and a rise in Pab, and the rib cage piston upward. The resultant displacement of the rib cage piston during isolated diaphragm contraction therefore depends on the balance between the force exerted by the muscle and the force generated by Ppl.

For coordinated activation of the diaphragm and the rib cage inspiratory muscles in normal subjects, this two-compartment model represents the mechanics of the chest wall reasonably well. The model, however, imposes the constraint that the rib cage moves as a single unit. Consequently, it cannot account for the observation that the upper and lower ribs move in opposite directions during breathing with the diaphragm alone or the observation that in subjects with COPD and hyperinflation, simultaneous contraction of the diaphragm and rib cage inspiratory muscles may displace the lateral walls of the lower rib cage inward and the upper rib cage outward.

These two observations prompted the proposal that the rib cage be divided into upper and lower compartments to form a three-compartment model of the chest wall (3, 40, 58). A schematic diagram of the model recently developed by Wilson (61) is shown in Fig. 7. In this model, although the upper rib cage (urc) and lower rib cage (lrc) pistons are connected by a spring (k_c) representing the coupling between the two pistons, the rib cage inspiratory muscles act only on the upper rib cage piston while the diaphragm acts only on the lower rib cage piston. This action is due, in part, to the connection of the muscle to a shelf on the piston (insertional force), but in addition, the lower rib cage piston is subdivided into two parts; one part (A_1) is exposed to Ppl, and the other part (A_2), representing the zone of apposition, is exposed to Pab. When the diaphragm contracts alone, therefore, it develops both an insertional force and an appositional force that displace the lower rib cage piston upward and oppose the displacement due to ΔPpl. Also, although the upper rib cage piston is displaced downward by the action of Ppl, this displacement is affected by the displacement of the lower rib cage piston through the connection k_c.

The equations of static equilibrium of the four pistons in the model have been formulated, and the solutions to the equations for breathing with the diaphragm alone matched

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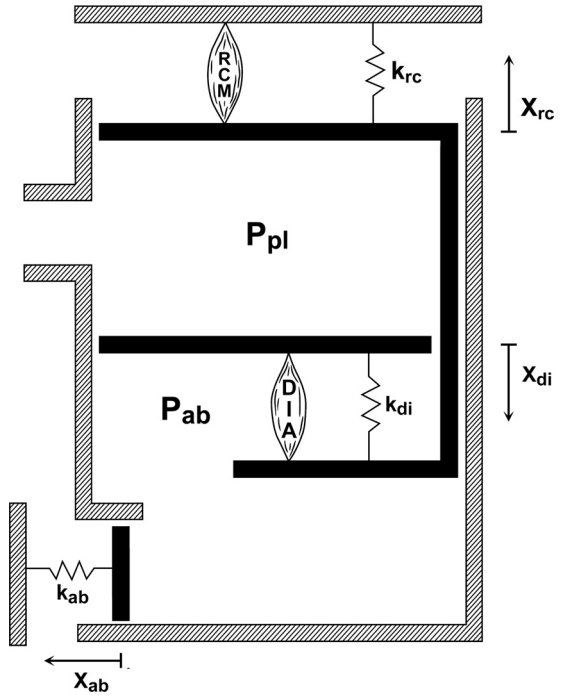

Fig. 6. Schematic diagram of the two-compartment model of the chest wall. The cross-hatched walls in the diagram are the stationary foundation, and the solid walls are pistons representing the rib cage (rc), diaphragm (di), and abdomen (ab). The pistons are connected to the foundation by springs with spring constant k_i and have displacements X_i; the arrows indicate positive displacements. The force exerted by the rib cage inspiratory muscles (RCM) displaces the rib cage piston upward, and the force exerted by the diaphragm muscle (DIA) acts both on the rib cage piston and the diaphragm piston. See text for further explanation.

the experimental observations (61). Therefore the model was also used to simulate dynamic maneuvers and to compute the volume changes of the chest wall compartments in subjects with COPD. That is, A_1 in the model was increased and A_2 was decreased to represent the decrease in the area of the zone of apposition that occurs with lung inflation. Moreover, airway resistance was increased, so that ΔPpl during inspiration was greater. With these changes, the model predicted that coordinated contraction of the diaphragm and rib cage inspiratory muscles would cause expansion of the abdomen and upper rib cage, but that the volume of the lower rib cage would progressively decrease during the first half of inspiration and partly recover during the second half. The dynamic solutions to the model thus closely reproduced the compartmental volume trajectories observed in subjects with Hoover's sign (4). By contrast, in the static solution to the model, for the same values of the parameters, the volume of the lower rib cage at end-inspiration was slightly larger than at end-expiration. This model analysis is significant because it further supports the conclusion that Hoover's sign is the result of the decrease in the zone of apposition and the dominant effect of Ppl on the lower rib cage, rather than an inward pull of the lower ribs by the diaphragm. In addition, the analysis predicts that the sign is a dynamic phenomenon that would be substantially decreased, if not suppressed, for slow inspirations. This prediction, however, remains to be tested.

Summary and Future Research Perspectives

Two forces act on the rib cage when the diaphragm contracts, namely the force related to ΔPpl and the force exerted by the muscle, which is proportional to ΔPdi. The displacements of the rib cage during diaphragm contraction therefore are defined by the product of rib cage compliance and the effective pressure (Peff) acting on the cage (Peff = Ppl + B·Pdi). The recent development of an experimental preparation in the dog in which the diaphragm is the only muscle active during inspiration has provided the data that were required to solve for B and, thus, to determine Peff.

In the control condition at FRC, B for the lower ribs equals 1.0, and Peff is positive because the magnitude of the increase in Pdi is slightly larger than the magnitude of the fall in Ppl. The force exerted by the diaphragm also acts on the upper ribs through the coupling between the lower rib cage and the upper rib cage, but the value of B for the upper ribs is considerably smaller, ~0.25. As a result, Peff for the upper rib cage is negative. When the abdomen is given a passive mechanical support, the area of the zone of apposition is larger and the elastance of the abdomen is greater, so that B for the lower ribs is >1.0, and ΔPdi is increased relative to ΔPpl. Consequently, Peff for the lower rib cage is more positive than in the control condition, and Peff for the upper rib cage is less negative. In contrast, when lung volume is elevated above FRC, the zone of apposition is smaller, and B for the lower ribs is <1.0. Therefore, although the relative magnitudes of ΔPdi and ΔPpl

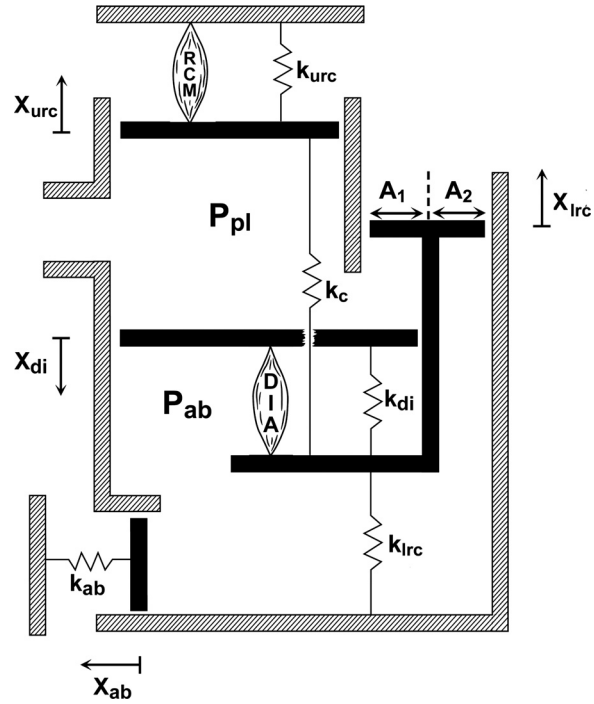

Fig. 7. Schematic diagram of the three-compartment model of the chest wall. In this model, the piston representing the rib cage piston is replaced by two pistons, one representing the upper rib cage (urc) and one representing the lower rib cage (lrc). Part of the lower rib cage (area A_1) is exposed to pleural pressure (Ppl), and part (area A_2) is exposed to abdominal pressure (Pab). A spring with spring constant k_c extends between the upper and lower rib cage pistons and represents the coupling between the upper and lower rib cage. See text for further explanation.

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are nearly the same as at FRC, Peff for the lower rib cage becomes negative.

It must be pointed out, however, that the mechanics of the rib cage in humans may be somewhat different from that in the dog. Also, as discussed in EFFECT OF BODY POSITION, the forces exerted on the ribs by the diaphragm may depend on body position. Consequently, these results in supine dogs cannot be extended to upright humans without caution. Because it allows for regional chest wall volumes to be measured, the optoelectronic plethysmography technique developed by Aliverti and colleagues (1, 11, 40, 52) might provide a useful method for quantifying the forces exerted by the diaphragm on the rib cage in humans. Thus, by measuring the volume displacements of the lower rib cage and the changes in Ppl and Pab during passive inflation and during spontaneous breathing in subjects with quadriplegia, the value of *B* for the lower ribs could be determined. These measurements could also be performed with quadriplegic subjects placed in the upright and the supine position to evaluate the effect of posture on the value of *B*. Moreover, the volume displacements of the upper rib cage during passive inflation and during spontaneous breathing in quadriplegic subjects could also be measured. The value of *B* for the upper ribs therefore could be determined, and this would provide an estimate of the magnitude of the coupling between the lower rib cage and the upper rib cage in humans that would be more accurate than the one inferred from measurements during phrenic nerve stimulation with single and paired twitches in normal subjects (12, 40).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

A.D. and T.A.W. prepared figures; A.D. and T.A.W. drafted manuscript; A.D. and T.A.W. edited and revised manuscript; A.D. and T.A.W. approved final version of manuscript.

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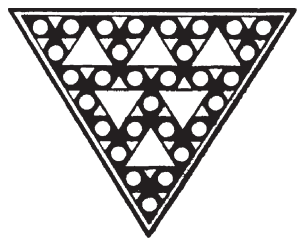

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