References and Notes

1. M. V. L. Bennett, *Ann. N.Y. Acad. Sci.* **137**, 509 (1966).
2. ———, in *Structure and Function of Synapses*, G. D Pappas and D. P. Purpura, Eds. (Raven, New York, 1972), pp. 221–256.
3. C. A. G. Wiersma, *J. Neurophysiol.* **10**, 23 (1947).
4. E. J. Furshpan and D. D. Potter, *J. Physiol. (London)* **145**, 289 (1959).
5. J. J. Wine and F. B. Krasne, *J. Exp. Biol.* **56**, 1 (1972).
6. J. J. Robertson, *Ann. N.Y. Acad. Sci.* **94**, 339 (1961); J. S. Keeter, R. B. Hanna, G. D. Pappas, *J. Cell Biol.* **79**, 764 (1978); C. Peracchia, *Int. Rev. Cytol.* **66**, 81 (1980).
7. D. C. Spray, A. L. Harris, M. V. L. Bennett, *Science* **204**, 432 (1979).
8. A. Van Harreveld, *Proc. Soc. Exp. Biol. Med.* **34**, 428 (1936).
9. Since one current microelectrode was used to change membrane potential, the junctional membrane resistance R_J was calculated according to simplified equations derived elsewhere [Y. Asada and M. V. L. Bennett, *J. Cell Biol.* **49**, 159 (1971)].
10. R. Ochi, *Pfluegers Arch.* **311**, 131 (1969); W. J. Moody, *J. Physiol. (London)* **316**, 293 (1981).
11. E. J. Furshpan and D. D. Potter, *J. Physiol. (London)* **145**, 326 (1959).
12. W. R. Loewenstein, M. Nakas, S. J. Socolar, *J. Gen. Physiol.* **50**, 1645 (1967); B. Rose and R. Rick, *J. Membr. Biol.* **44**, 377 (1978).
13. A. A. Auerbach and M. V. L. Bennett, *J. Gen. Physiol.* **53**, 211 (1969). The junctional resistance in this preparation exhibits a voltage dependency similar to that at the GMS, but no plateau was reached in this preparation.
14. T. G. Smith and F. Baumann, *Prog. Brain Res.* **31**, 313 (1969).
15. G. L. Ringhmam, *J. Physiol. (London)* **251**, 395 (1975).
16. A. Arvanitaki and N. Chalazonitis, *Bull. Inst. Oceanogr. Fish.* **1143**, 1 (1959).
17. J. G. Nicholls and D. Purves, *J. Physiol. (London)* **209**, 647 (1970).
18. D. C. Spray, A. L. Harris, M. V. L. Bennett, *J. Gen. Physiol.* **77**, 77 (1981).
19. H. B. Mann and D. R. Whitney, *Ann. Math. Stat.* **60**, 50 (1947).
20. W. C. De Mello, *Cell Biol. Int. Rep.* **4**, 51 (1980); W. R. Loewenstein, *Physiol. Rev.* **61**, 829 (1981).
21. B. Mann and H. Kuhn, *J. Appl. Phys.* **42**, 4398 (1971); H. C. Pant and B. Rosenberg, *Biochim. Biophys. Acta* **225**, 379 (1971).
22. B. Rosenberg, *Discuss. Faraday Soc.* **51**, 190 (1971).
23. E. H. Walker, *Int. J. Quantum Chem.* **11**, 103 (1977).

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Mechanical Action of the Intercostal Muscles on the Ribs

Abstract. *The external and internal interosseous intercostal muscles were separately stimulated at end-expiratory lung volume in anesthetized dogs. These muscles were all found to elevate the ribs into which they insert. By attaching weights to the ribs, it was determined that the nonlinear compliance of the ribs was responsible for this phenomenon.*

The action of the intercostal muscles has been a subject of controversy throughout medical history (*1*). Up to the middle of this century, varying and opposite points of view found strong supporters (*2*). At present, the most widely held view is that associated with Hamberger (*3*), whose theory, inferred from the anatomical relations of the muscles (points of origin and insertions), is that the external intercostals and the interchondral portion of the internal intercostals (the parasternals) elevate the ribs to which they are attached and, accordingly, are inspiratory, while the interosseous portion of the internal intercostals lowers the ribs and, therefore, is expiratory.

Electrical recordings from the intercostal muscles in normal humans showed a phasic behavior of these muscles which was in accord with Hamberger's theory (*4*). Electromyographic observations, however, cannot be interpreted correctly as long as the mechanical action of the muscles remains unknown. Motions are frequently complex, requiring contraction not only of agonists but also of synergists, fixators, and even antagonists. Electrical activity of a muscle associated with a particular motion does not prove that the muscle is the agonist. The observation that one intercostal muscle contracts during inspiration and that this contraction is associated with an enlargement of the rib cage, therefore, does not prove that the muscle is inspiratory in function (that is, causes flow of air into the lungs), nor does it prove that the action of the muscle is to raise the lower rib into which it is inserted; the ribs could be displaced by other muscles and the electrical activity observed in the intercostals might be fixating or antagonistic (*2*). With the exception of the parasternals, which were recently shown to be inspiratory (*5*), there is no direct experimental evidence that establishes the mechanical action of the intercostal muscles. In this report we show that at end-expiratory lung volume, both the external and the interosseous internal intercostals elevate the ribs into which they insert. We also show that this phenomenon results from the nonlinear compliance of the ribs.

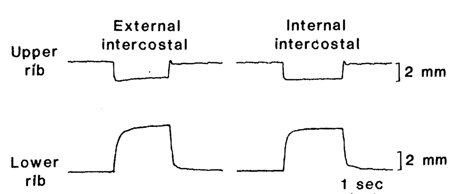

Fig. 1. Effect of stimulating separately the external and the internal interosseous intercostal muscle in one intercostal space on the axial displacements of the ribs situated immediately above (upper trace) and below (lower trace). In the two channels, upward deflections indicate a cephalad displacement, and downward deflections the reverse. In this record, the stimulation frequency was 100 Hz.

Experiments were performed on supine dogs anesthetized with sodium pentobarbital (25 mg/kg), intubated, and maintained under deep general anesthesia with supplementary doses. The rib cage and the intercostal muscles were exposed from the second to the tenth rib by deflection of the skin and the consecutive layers of muscles. Hooks were screwed into two adjacent ribs on the anterior or midaxillary line and connected by inextensible threads to linear displacement transducers positioned along the longitudinal body axis of the animal in order to measure the axial displacements of the ribs (*5*). A pair of stimulating electrodes spaced 2 cm apart was then inserted superficially in the fibers of the external intercostal muscle connecting the two ribs. The stimulus (20 to 100 Hz, 0.2 msec) was adjusted from 5 to 10 V for maximum effect without activation of the other intercostal muscles of the same interspace (*6*). After the external intercostal was studied, the muscle was removed and the internal interosseous intercostal was exposed and stimulated. All measurements were obtained at end-expiratory lung volume during apnea induced by hyperventilation. Studies were made on 28 intercostal spaces in 12 animals.

Representative records are shown in Fig. 1. Electrical stimulation of the external intercostal resulted in a cephalad displacement of the rib situated below and a caudad displacement of the rib situated above the muscle. The cephalad displacement of the lower rib was, however, twice as large as the caudad displacement of the rib above. For the ribs to which it is attached, therefore, the net effect of contraction of the external intercostal muscle was inspiratory. Stimulation of the interosseous portion of the internal intercostal also resulted in a cephalad displacement of the lower rib and a caudad displacement of the rib above. Here also, the cephalad displacement of the lower rib was about twice as large as the caudad displacement of the upper rib. The net action of the internal interosseous intercostal muscle, therefore, was also inspiratory for the ribs into which it inserts. Almost identical records were obtained for all the interspaces investigated. There was no difference between the intercostal spaces situated in the upper and lower parts of the rib cage.

Stimulating the intercostal muscles in the dorsal part of the rib cage, 4 to 5 cm lateral to the spine, rather than in the midaxillary line did not affect the results; whatever intercostal muscle was stimulated, the cephalad motion of the rib below was larger than the caudad motion of the rib above. Finally, stimulating the external or internal intercostal muscles simultaneously in three adjacent interspaces resulted in a cephalad displacement of the two ribs situated in between. The rib's displacements were then associated with increases in lung volume and falls in pleural pressure.

These data show that in the dog, the external and internal intercostal muscles have a similar effect on the ribs. At end-expiratory lung volume the net effect of their contraction is to elevate the ribs to which they are attached. The results thus disagree with Hamberger's hypothesis (*3*). They support the observation by Duchenne (*7*) of a man in whom stimulation of the external intercostal with or without the internal intercostal in one interspace always elevated the lower above the upper rib, and they indicate that neither the orientation of the intercostal muscle fibers nor the distance between their insertions and the center of rotation of the ribs plays a predominant role in determining the mechanical action of the muscles. We reasoned that instead, the primary determinant of the action of the intercostal muscles was the inherent tendency of the ribs to be more easily displaced cephalad than caudad.

To test this hypothesis, we measured the stress-strain relation of the ribs (with their anatomical attachments) along the cephalocaudal axis of the rib cage. While the animal was apneic, weights (100 to 500 g) were applied to one rib successively in the cephalad direction and then in the caudad direction. The resulting axial displacement of the rib was measured with a linear displacement transducer. Ranging between the third and the eighth rib, 12 ribs from three animals were studied. Figure 2A shows representative results. For any given weight, the rib was displaced about twice as much in the cephalad direction as in the caudad direction. As shown in Fig. 2B, this was true for all 12 ribs investigated, regardless of the fact that in each animal the cephalad and caudad rib compliance (*8*) progressively increased from the upper to the lower rib cage.

These findings are consistent in all respects with our hypothesis. They show that, at end-expiratory lung volume, the ribs are nonlinearly compliant and that, in response to a given load, they move cephalad more easily than caudad. This is consistent with the fact that in this volume range the rib cage is below its neutral position (*9*) and also with the anatomy of the ribs, which, given their configuration and their vertebral and sternal articulations, appear to have less constraint on their motion in the lower than in the upper part of the rib cage (*10*). As a result, if one rib is submitted to the same muscle tension simultaneously in both directions, no matter which intercostal muscles are active, it will be elevated.

The relevance of these data to the actual function of the intercostal muscles during breathing is still not known. However, our results may lead to a new insight into that question. With the mechanical action of the intercostal muscles on the ribs established, it should be possible to interpret the electrical observations more reliably. In particular, the electrical activity observed during quiet expiration in the interosseous portion of the lowermost internal intercostals (*4*) may be regarded as an antagonistic activity which tends to prevent collapse of the lower rib cage, rather than as an agonistic activity which deflates the rib cage (*11*).

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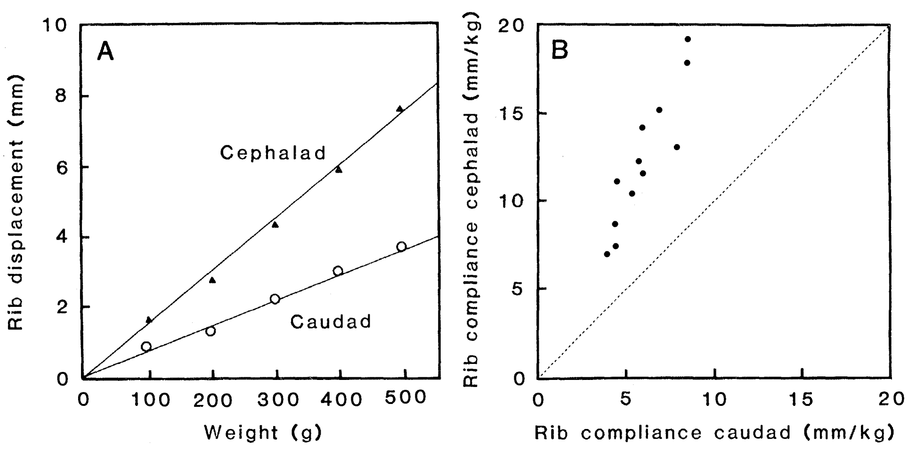

Fig. 2. Identification of the mechanism responsible for the inspiratory action of the external and internal intercostal muscles. Weights were applied to one rib successively in the cephalad and the caudad direction. The resulting axial displacement of the rib was measured by a displacement transducer. (A) Representative results. (B) Results obtained in the 12 ribs investigated; the dashed line is the identity line (*8*).

References and Notes

1. J. H. S. Beau and J. H. Maissiat, *Arch. Gen. Med.* **1**, 265 (1843); L. Luciani, in *Human Physiology* (Macmillan, London, 1911), vol. 1, p. 411.
2. R. Gesell, *Am. J. Physiol.* **115**, 168 (1936); E. J. M. Campbell, *J. Physiol. (London)* **129**, 12 (1955).
3. G. E. Hamberger, *De Respirationis Mechanismo* (Iena, 1727).
4. A. Taylor, *J. Physiol. (London)* **151**, 390 (1960); M. H. Draper, P. Ladefoged, D. Whitteridge, *Br. Med. J.* **1**, 1837 (1960); L. Delhez, *Contribution électromyographique à l'étude de la mécanique et du contrôle nerveux des mouvements respiratoires de l'homme* (Vaillant-Carmanne, Liège, 1974), pp. 103–174.
5. A. De Troyer and S. Kelly, *J. Appl. Physiol.* **53**, 373 (1982).
6. In several intercostal spaces, external intercostal stimulation was repeated after a thin plastic sheet had been introduced between the two intercostal muscle layers. Rib motion results obtained after such insulation could never be distinguished from those recorded before, indicating that external intercostal stimulation did not produce significant internal intercostal activation.
7. G. B. Duchenne, *Physiologie des mouvements* (Baillière, Paris, 1867), p. 646.
8. Rib compliance actually refers to the strain-stress relation of the rib cage at a given point.
9. K. Konno and J. Mead, *J. Appl. Physiol.* **24**, 544 (1968).
10. H. E. Evans and G. C. Christensen, *Miller's Anatomy of the Dog* (Saunders, Philadelphia, ed. 2, 1979); C. M. Goss, *Gray's Anatomy of the Human Body* (Lea & Febiger, Philadelphia, ed. 28, 1966).
11. Since this report was submitted, we stimulated the intercostal muscles and measured the cephalad and caudad compliance of the ribs at various lung (rib cage) volumes. We found that the net elevating effect of the intercostal muscles on the ribs was accentuated as lung volume fell below end expiration. Conversely, as lung volume increased above end expiration, this net elevating effect progressively decreased; it was eventually reversed into a net lowering effect above approximately half inspiratory capacity. This was true for both the external and the internal interosseous intercostal muscles. Rib compliance measurements were consistent with these findings. As lung volume increased, the tendency of the ribs to be displaced cephalad progressively decreased while the tendency to be displaced caudad gradually increased, so that at high lung volumes the ribs were more easily displaced in the caudad than in the cephalad direction. These additional experiments support the hypothesis that the nonlinear compliance of the ribs, rather than the orientation of the muscle fibers, is the primary determinant of mechanical action of the intercostal muscles.
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