MECHANICS OF CHEST WALL
OPERATIONAL LENGTH COMPENSATION—A NEW RESPIRATORY REFLEX

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Every time you get up in the morning the mere process of assuming the upright posture causes you to double or even triple your nervous output to respiratory muscles. If you did not do this you would seriously underventilate. You make this adjustment automatically and without chemical stimulation.

But it should be obvious that you have to increase the drive to the inspiratory muscles every time you get up. Why do I say this? First, certainly every medical student learns that lung volume increases when you get up in the morning. In the upright posture you breathe at a higher lung volume. Second, a higher lung volume means shorter inspiratory muscles. Third, in the physiologic range of their length changes shortened muscles require greater strengths of excitation than lengthened ones in order to develop the same force. Upon shifting from supine to upright, similar forces are required to ventilate the lungs. It follows then, that in the upright posture the shortened inspiratory muscles will require greater degrees of excitation to achieve a given ventilation.

Do such adjustments occur? To our knowledge no one had said so until we made our measurements. About ten years ago we were interested in the effects of mechanical loads on breathing. We had a servo-controlled blower with which we could change pressure at the mouth in a versatile fashion. One of our interventions was simply to bias the pressure at the mouth. That is to produce a steady positive pressure — so called positive pressure breathing. At a steady pressure of plus ten centimeters of water, which increased lung volume about one tidal volume's worth — a modest shift in the inspiratory direction — the intensity of the diaphragm EMG signals increased substantially.

Was the response chemical? End-tidal CO₂ had not changed, or, if anything, changed paradoxically. That is, it decreased slightly while EMG activity increased. But might not arterial PCO₂ have increased because of altered blood flow distribution? Recently we have checked this point. There was no significant change in arterial PCO₂ in the presence of the response. So it isn't chemically mediated.

Was the response simply behavioral? Was awareness a necessary part? We studied this in quadriplegics. We used abdominal cuirasses with negative pressure to produce inspiratory shifts. The patients had no sensation of the cuirass or of the surface EMG electrodes, and we screened the equipment from their vision. They had no idea that their breathing was being studied — no equipment of any sort at the mouth. And they all showed brisk responses. We conclude from this that the response is automatic and reflex in nature. At present we are studying the afferent features of this reflex.

What does this response have to do with getting up in the morning? The associated inspiratory shift in lung volume is entirely comparable to the shift that we produce with positive pressure breathing. Recently we have shown that standing individuals require more than three times as much diaphragmatic activation to develop a given inspiratory pressure as do supine individuals.

What does this response have to do with respiratory disease? Is it possible that patients with chronic obstructive lung disease and associated increases in lung volume are utilizing and ultimately exhausting this mechanism? Perhaps some of you will be stimulated to find out if this is the case.