

Energy Requirements of Breathing*

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The mass movement of gases into and out of the lungs is accomplished by muscular work. This requires energy expenditure, caloric consumption, oxygen utilization, and carbon dioxide production. Inspiration is always an active process, enlarging the volume of the thorax, thereby increasing the negative pressure so that air flows into the lungs. Expiration is ordinarily passive but may require muscular work.

The position of the chest wall at rest is a mechanically neutral point at which the tendency of the chest wall to expand is balanced by the tendency of the lungs to recoil. This is the end expiratory level or midposition, and the lung volume at this point is termed the functional residual capacity. An increase in chest size can be achieved only by exerting pressure greater than the elastic forces of the lungs. In addition, airway resistance to airflow and nonelastic tissue resistance must be overcome. In the normal individual, 60 to 70% of the total ventilatory work is required for overcoming elastic forces, the remainder for overcoming nonelastic resistance, largely due to airflow.

Expiration is passive in the normal individual during quiet breathing, the energy stored in the elastic lung tissue during inspiration being sufficient.

The diaphragm is the major muscle of inspiration and is probably always active even with so-called thoracic breathing. It arises from the xiphoid process, from the inner surface of the last six ribs and their cartilages, and from the lumbar vertebrae. By virtue of its position and attachments, it not only increases the vertical dimension of the chest by a downward movement, but also increases the transverse diameter by flaring the lower ribs.

The intercostal muscles are second in importance to the diaphragm. The external intercostal fibers run downward and forward and the internal intercostal fibers are directed upward and forward to the next rib, so that contraction elevates the rib with resulting increase in the anteroposterior diameter, transverse diameter, or both. Studies by Koepke *et al.* (1958) showed that the 1st intercostals are active in quiet breathing of most individuals. With increased ventilation, there is increased utilization of the intercostals, so that the majority of individuals use

all intercostal muscles when ventilation requirements approach 50% of the predicted vital capacity.

The diaphragm and intercostals are the only respiratory muscles used by the normal individual under ordinary circumstances. Other muscles are mobilized when ventilatory demands are increased, either because of increase in total ventilation in the normal individual or because of altered physiology of respiration in disease states. The so-called accessory muscles of respiration include particularly the scalenes, the sternomastoid, and the trapezius.

The scalene muscles arise from the transverse processes of the cervical vertebrae and insert in the 1st and 2nd ribs. They serve to stabilize and elevate the 1st and 2nd ribs. They are not ordinarily employed in quiet respiration.

Although a well trained subject was able to breathe 60 L per minute without electromyographic evidence of scalene activity, these muscles are ordinarily brought into play when an intrathoracic pressure of -5 to -6 cm of H₂O is exerted and chest volume is increased by 800 to 900 ml of air. On rapid inspiration they are brought into action at the very beginning of inspiration (Thompson, Patterson, and Shapiro, 1964).

While many muscles of the thorax may be used in severe dyspnea, only the sternomastoid and the trapezius will be considered. The former arises from the manubrium and the sternal end of the clavicle and inserts at the mastoid process and lateral half of the nuchal line. It can assist in lifting the thorax. Campbell (1955) showed that there was never any detectable activity of this muscle in any subject during quiet respiration, but that increasing intensity of contraction was exhibited as intrathoracic pressures progressed from -10 to -50 cm of H₂O.

The trapezius arises from the occiput, the ligamentum nuchae, and the spines of the last cervical and thoracic vertebrae. It inserts into the acromial 3rd of the clavicle, and the scapula. The upper fibers tend to lift the chest.

These muscles do not function with equal effectiveness as muscles of respiration, and they are described in order of decreasing efficiency. The diaphragm and the intercostals are peculiarly suited for their task of increasing the chest volume. The scalene muscles have a direct pull to lift the 1st rib. The sternomastoid and trape-

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zius are large muscles that have a poor mechanical advantage in lifting the thorax.

The normal individual at rest has an oxygen requirement of about 300 ml that must be furnished, and produces about 240 ml of carbon dioxide that must be eliminated. This is achieved by a minute volume of approximately 7 L. About 2% of the total oxygen consumption at rest, or some 6 ml, is used by the respiratory muscles.

An individual with normal heart and lungs is able to increase his ventilation considerably without much increase in total oxygen consumption. As respiratory effort increases, the oxygen cost of breathing becomes excessive in everyone, whether sick or well. Eventually the point is reached where the respiratory muscles consume so much oxygen that further increase in ventilation does not supply additional oxygen for utilization by other tissues of the body. With this there is a greatly increased production of carbon dioxide, and carbon dioxide retention may occur because alveolar ventilation cannot keep pace with the carbon dioxide produced by the respiratory muscles. This point is estimated to take place in the normal subject at ventilatory levels of about 140 L per minute.

In patients with lung disease, the ventilatory work is often increased, so that there may be a disproportionate oxygen utilization and carbon dioxide production by respiratory muscles even at rest. This is particularly apt to occur in patients with airway obstruction, especially in those with chronic obstructive pulmonary emphysema. The effective ventilatory level may be reached at 15 to 20 L per minute in such patients. Any efforts to increase ventilation further only add oxygen which is used by the respiratory muscles. Carbon dioxide is produced in excess of the ability to excrete it, and respiratory acidosis may be worsened.

The energy problems of patients with lung disease, particularly those with emphysema, are multiple. They often must use the less efficient accessory muscles at rest, a breathing pattern the normal subject uses only on extreme exertion. The mechanical efficiency of these muscles is poor, their oxygen consumption and carbon dioxide production being out of proportion

to their contribution to effective alveolar ventilation.

The diaphragm is a much less effective inspiratory muscle when it is flattened by overdistended lungs. Its contraction no longer increases the vertical dimension of the chest. Furthermore, it acts like a flat sheet of muscle, draws the lower ribs together, and thus reduces rather than increases the transverse diameter.

Expiration which is normally passive may require muscle activity. This is because of loss of elastic recoil of lung and because of obstruction to expiratory airflow. Unanswered is the question of whether there is structural or biochemical change in the respiratory muscles so that they require more oxygen and do less work.

Many patients with chronic lung disease are alive only because their respiratory muscles are able to perform increased work day in and day out. Greater force must be exerted if effective alveolar ventilation is to be maintained and blood gases kept at tolerable limits. This means that caloric intake must be adequate, nutrition must be good, and muscle tone and strength must be maintained. The nutritional care, particularly of patients with chronic pulmonary emphysema, has in the past received little attention. There is a tendency for attention to be centered on the more obvious pulmonary abnormalities and to disregard measures designed to improve and maintain the nutritional

status of the patient. In the past we have repeatedly observed such patients to exhibit rapid and progressive loss of weight with concomitant deterioration in their condition. Two factors appeared to be responsible primarily for the loss of weight in these patients: (1) loss of appetite, which seems, at least in part, to be related to infection and to gastric distention, and (2) patients with advanced pulmonary insufficiency frequently develop severe dyspnea while eating. The distress resulting from shortness of breath thus limits the time that these patients are willing to spend in chewing and swallowing their food. As a result they take smaller meals and their caloric intake may be severely compromised. This chain of events and the interaction of the different factors described above are graphically represented in figure 1.

References

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Fig. 1—Circular deterioration of pulmonary emphysema

