Considerations When Testing and Training the Respiratory Muscles

W Darlene Reid and Gail Dechman

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The purpose of this review is to describe the muscles of respiration, mechanisms that lead to their dysfunction, and unique features of the respiratory muscles that should be considered when designing testing and training protocols for patients with respiratory compromise. The diaphragm's unique shape is an important determinant of its function. Respiratory pathophysiology combined with systemic abnormalities may result in inspiratory muscle weakness, fatigue, and injury. The specificity of training principle, as it applies to respiratory muscle training in patients with respiratory compromise, will be described. Precautions related to this type of training will also be outlined. In conclusion, a better understanding of the mechanisms contributing to inspiratory muscle dysfunction will assist physical therapists in designing the most appropriate training programs. This article, along with the other articles in this focus on respiratory muscle training, will provide therapists with scientifically based guidelines for rehabilitation of patients with impaired respiratory muscle function. [Reid WD, Dechman G. Considerations when testing and training the respiratory muscles. Phys Ther. 1995;75:971–982.]

Key Words: *Chronic obstructive lung disease, Diaphragm, Dyspnea, Exercise tolerance, Rehabilitation.*

Respiratory muscle function is essential for life. Respiratory muscles, like all skeletal muscles, improve their function in response to training. Unlike limb muscles, however, they must contract repetitively, approximately 12 to 20 times per minute every day of our lives. Because the inspiratory muscles are used so frequently, they have no opportunity to rest and may become fatigued or injured under conditions that overload the respiratory system. Physical therapists can play a major role in recognizing such respiratory muscle dysfunction and implementing appropriate training either to prevent fatigue or to facilitate recovery from fatigue and injury. Because of the vital function of these muscles, however, care must be taken in progression of exercise because undue fatigue could precipitate or exacerbate respiratory failure.[1] This review will describe the primary and some of the accessory muscles of respiration, conditions associated with respiratory muscle dysfunction, and unique features of the respiratory muscles that should be considered when testing and training the respiratory muscles of patients with respiratory compromise. The information in this article will provide a basis for the other articles in this focus on respiratory muscle testing and training.

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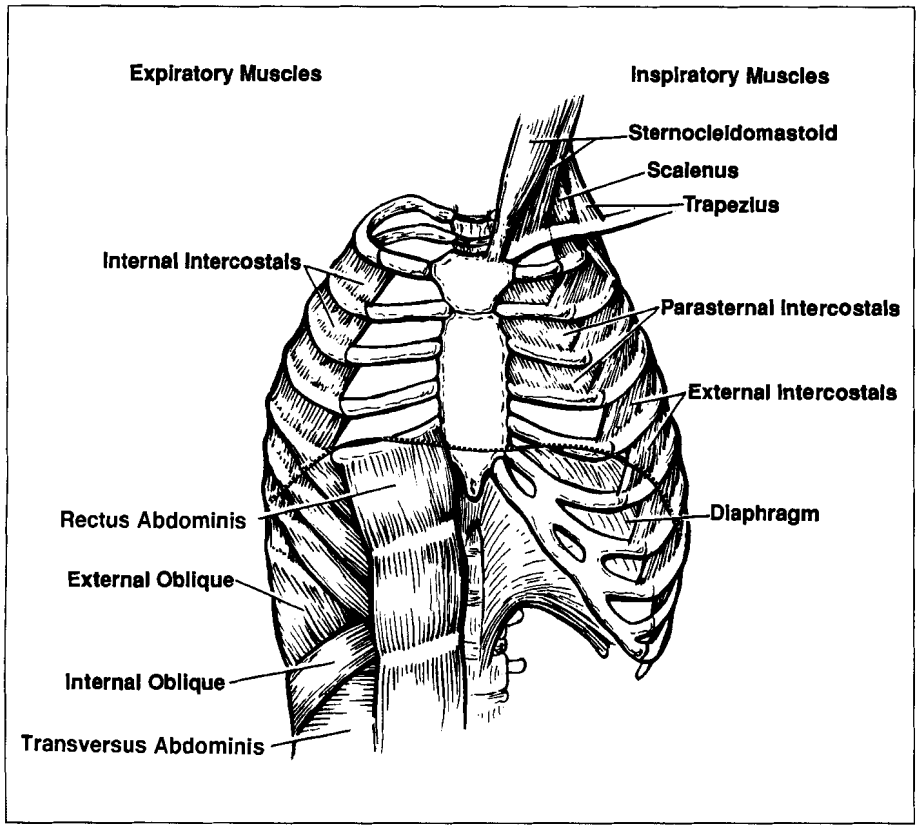

Figure 1. *Anatomy of inspiratory and expiratory muscles.*

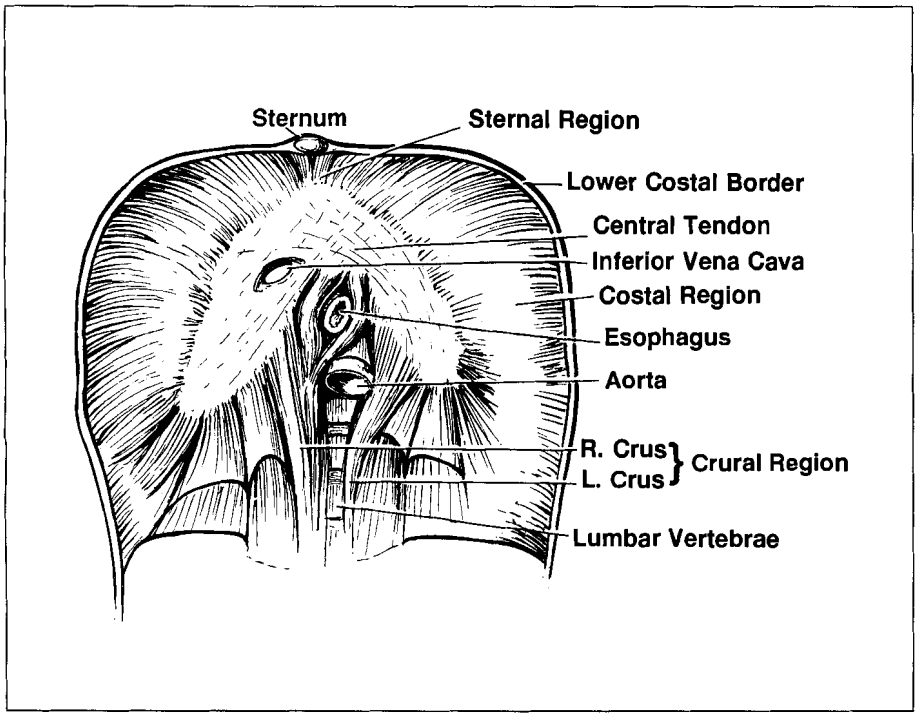

Figure 2. *Three anatomical regions of the diaphragm. The sternal, costal, and crural regions arise from the xyphoid process of the sternum, the upper margins of the lower six ribs, and the anterolateral aspect of the L1–L3 vertebrae, respectively. The muscle fibers from all three regions radiate inward and insert into the central tendon.*

Respiratory Muscle Anatomy and Function

During quiet inspiration in asymptomatic individuals, the respiratory muscles contract in a coordinated fashion such that the diaphragm descends in a pistonlike fashion and the ribs move upward and outward. The increase in the size of the thoracic cavity creates a negative intrathoracic pressure, which draws air into the lungs. The inspiratory muscles then relax, and expiration is accomplished passively, using the elastic recoil of the lungs. Activities such as exercise or even breathing at rest in individuals with respiratory disease demand increased levels of ventilation, which may require recruitment of both accessory inspiratory and expiratory muscles.[2]

Inspiratory Muscles

The primary muscles of inspiration, those required during quiet breathing, are the diaphragm, the scalenes, and the parasternal intercostals[3] (Fig. 1). The diaphragm is composed of three anatomically distinct regions[4] (Fig. 2). The costal portion arises from the upper margins of the lower six ribs and is closely associated with the sternal region, which originates from the posterior aspect of the xyphoid process (Fig. 2). The thicker crural portion arises from the anterolateral aspect of the L1–L3 vertebrae (Fig. 2). Fibers from all three regions of the diaphragm radiate inward, inserting into the central tendon.[4] The costal and crural components of the diaphragm may be recruited differently, especially during high levels of ventilation; however, different functions for these regions have not been clearly defined.[5]

Under quiet breathing conditions, the diaphragm performs about 70% to 80% of the work of breathing.[3] The dome shape of the diaphragm is essential for optimal performance of the appositional and insertional components of diaphragm action[5,6] (Fig. 3). The zone of apposition is that part of the diaphragm that is apposed to the inner aspect of the rib cage. When the diaphragm contracts, it shortens and

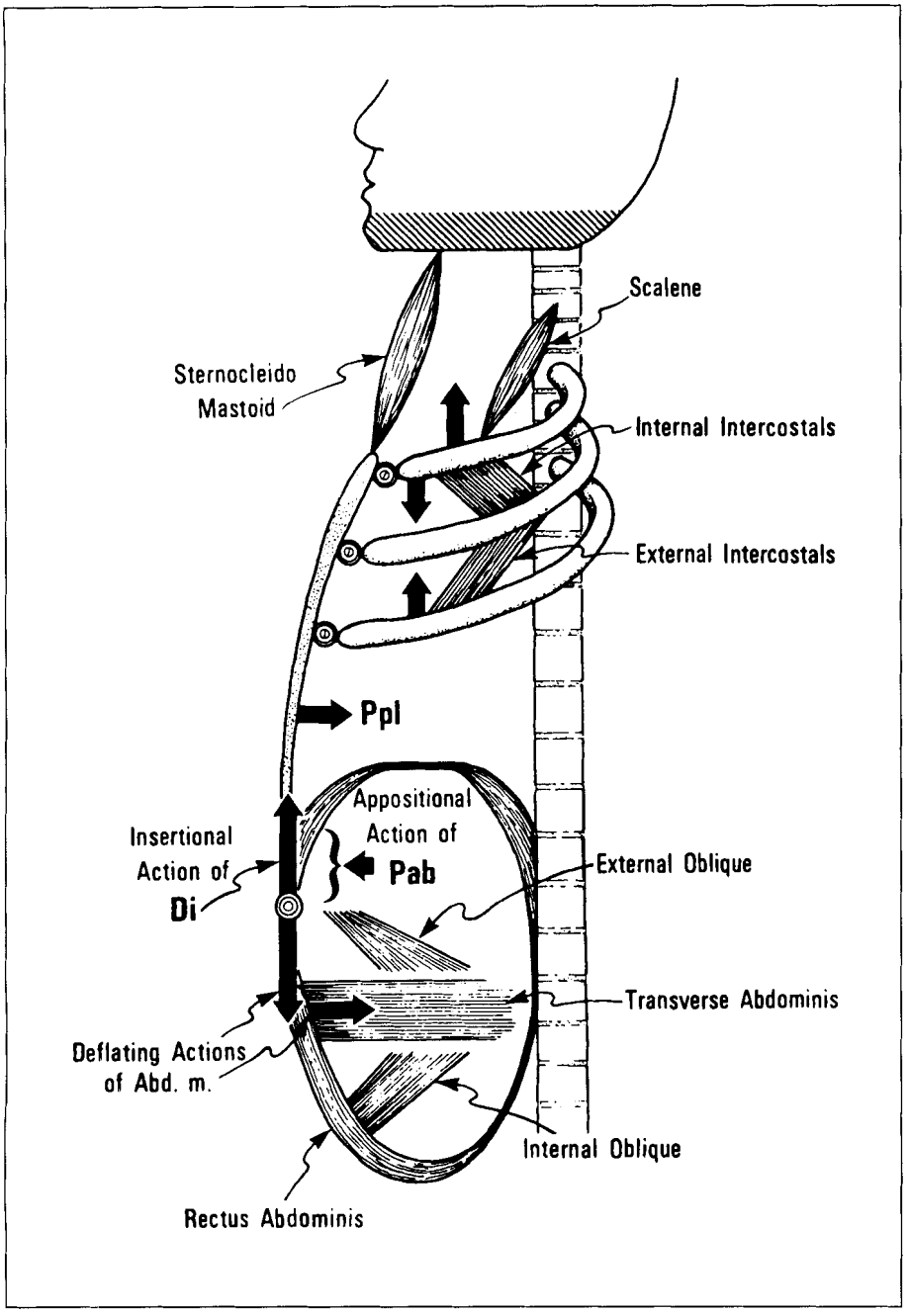

Figure 3. *Diagrammatic representation of inspiratory and expiratory muscles and their respective actions (arrows). Abd. m.=abdominal muscles, Di=diaphragm; Pab=abdominal pressure, Ppl=pleural pressure. (Reprinted with permission from Roussos C. Function and fatigue of the respiratory muscle.* Chest. *1985;88 [suppl]: 124s–132s.).*

the dome descends to compress the abdominal contents, increasing intra-abdominal pressure. This increased pressure is transmitted laterally to the rib cage via the zone of apposition and causes the lower rib cage to expand, which contributes to the decrease in intrathoracic pressure responsible for inspiration[7] (Fig. 3).

Therefore, abdominal muscle resting tension complements the inspiratory action of the diaphragm by facilitating an increase in pressure in the abdominal compartment rather than outward protrusion of the abdomen during diaphragmatic contraction.[5,8] In addition, the zone of apposition and dome shape of the diaphragm are maintained during inspiration by abdominal muscle resting tension supporting the abdominal viscera up against this muscle.[9]

Upward and outward rib movement during inspiration is dependent on the cranial orientation of the diaphragm's insertion[7] (Figs. 3 and 4). Because descent of the dome of the diaphragm is opposed by the abdominal contents, contraction of diaphragm fibers inserted into the ribs pulls them upward and outward in what has been described as a bucket-handle motion. If the dome of the diaphragm is flattened, as may be the case in patients with chronic obstructive lung disease or quadriplegia, the diaphragm fibers pull horizontally on the ribs rather than upward and outward. Thus, the diaphragm's ability to increase the dimensions of the thoracic cage is severely limited or lost. In persons with chronic obstructive lung disease, the dome of the diaphragm is flattened by hyperinflated lungs. In persons with quadriplegia, loss of abdominal muscle activity results in a decreased dome shape and a decreased zone of apposition of the diaphragm, and descent of the diaphragm is not adequately opposed by the viscera during inspiration.

The scalene muscles originate on the transverse processes of the lower five cervical vertebrae and insert on the upper surface of the first and second ribs[5] (Fig. 1). These muscles lift and expand the rib cage during inspiration. Contrary to traditional belief, the scalene muscles are active during every inspiratory effort and therefore should be considered a primary and not an accessory inspiratory muscle group.[10] The parasternal muscles are the other important inspiratory muscle group (Fig. 1). These muscles are attached to the sternum and run between the costal cartilages in a downward and outward direction.[3] When they contract, the ribs are lifted and the anterior-posterior dimension of the rib cage increases.[5] This inspiratory action of the parasternal and scalene muscle groups on the upper thorax is important to counteract the expiratory action of the diaphragm on the upper

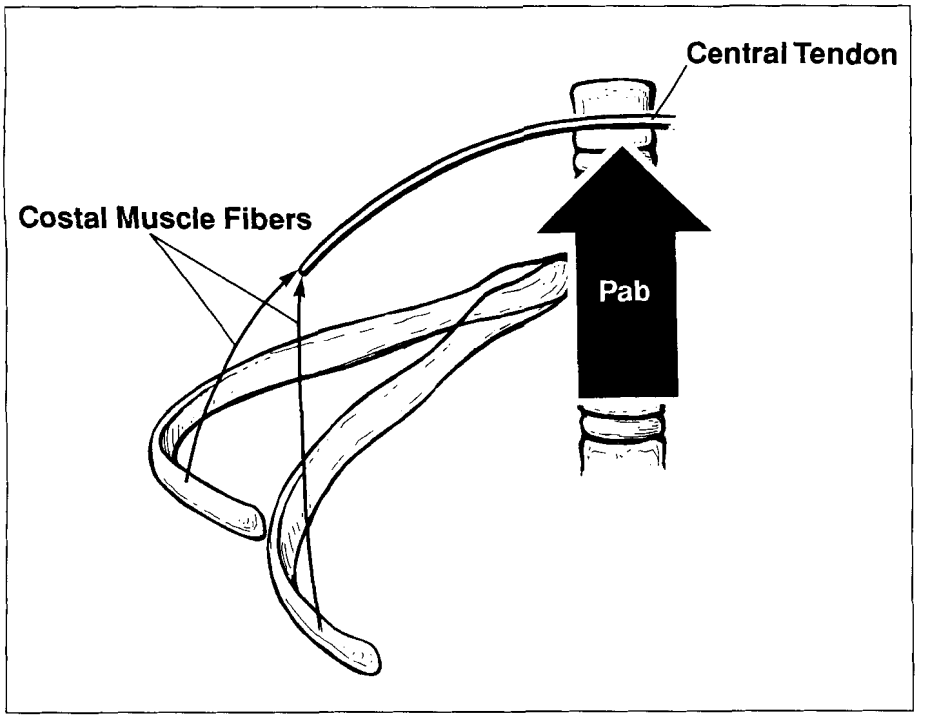

Figure 4. *The insertional component of diaphragm action. During inspiration, the costal fibers contract, exerting an action on the ribs. The abdominal contents provide a fulcrum for this action by apposing diaphragm descent, which results in an increase in abdominal pressure (Pab) (large black arrow) and helps to maintain the dome shape of the diaphragm. This dome shape allows contraction of the costal fibers to lift the ribs upward and outward during inspiration (thin arrows indicate direction of muscle action). The dome shape of the diaphragm is decreased or lost in states in which there is hyperinflation or in quadriplegia when the abdominal muscles no longer support the abdominal viscera. In these conditions, the contraction of the costal fibers may result in an inward rather than an upward and outward movement of the lower ribs.*

rib cage.[3,5] Descent of the diaphragm causes the decrease in pleural pressure necessary for inspiration. The decrease in pleural pressure is greatest in the cephalad regions around the apex of the lung and, if it is unopposed by the contraction of the parasternal and scalene muscle groups, will cause the upper rib cage to move inward in a manner that is characteristic of expiration. This breathing pattern is observed in individuals with high spinal cord lesions when the diaphragm is partially innervated and the scalene and parasternal muscles are not functional.

The sternocleidomastoid muscles are the most important accessory muscles of inspiration[5] (Figs. 1 and 3). These muscles run from the mastoid processes to insert along the medial third of the clavicle and ventral surface of the manubrium sterni.[5] As ventilatory demands increase, these muscles contract to lift the sternum and increase the anteroposterior diameter of the upper rib cage during inspiration. The role of the external intercostal muscles during inspiration is controversial, but if they contract during inspiration, their contribution is certainly minimal compared with that of the parasternal muscles.[11] The external intercostal muscles may have a more important function at high levels of ventilation.[11]

Expiratory Muscles

All expiratory muscles, to some extent, can be considered accessory muscles because tidal expiration is usually passive and achieved by elastic recoil of the lungs in asymptomatic individuals. The abdominal muscles (rectus abdominis, external and internal obliques, and transversus abdominis) (Figs. 1 and 3), however, assist expiration and facilitate diaphragmatic contraction under all circumstances. All of the abdominal muscles have attachments to the lower ribs.[3] Contraction of these muscles decreases the size of the rib cage to assist expiration. Activity in these muscles increases intra-abdominal pressure, which not only provides a fulcrum for diaphragm contraction during inspiration (see previous section) but also pushes the abdominal contents cranially, decreasing lung volume and lengthening the diaphragm at end-expiration (Fig. 3). Increased, phasic activation of the abdominal muscles further decreases lung volume and hence lengthens the diaphragm during exercise or other activities that require increased expiration.[3] Thus, the abdominal muscles play an important role in both expiration and inspiration as a result of the complex combination of respiratory muscle activities and their actions on the rib cage. Similar to the role of the external intercostal muscles, the role of the internal intercostal muscles is controversial, but they are thought to play a small role in expiration and are perhaps best considered accessory muscles of expiration.[11] Thus, in spinal cord lesions that result in a loss of innervation to the intercostal and abdominal muscles, the ability to ventilate at higher than resting levels is greatly hampered because these muscles cannot be actively recruited during expiration.

Respiratory Muscle Innervation

Motor innervation to the diaphragm is from the phrenic nerves (C3–C5 nerve roots). Most investigators agree that the costal and sternal portions of the diaphragm are supplied by the C3–C4 roots and that the crural region is supplied by the C4–C5 roots.[3,7] The significance of such segmental innervation is not clear because it is not known whether differential activation of the various portions of the diaphragm is possible. The phrenic nerve also carries sensory and proprioceptive information related to the diaphragm. Other inspiratory muscles are innervated by spinal and accessory

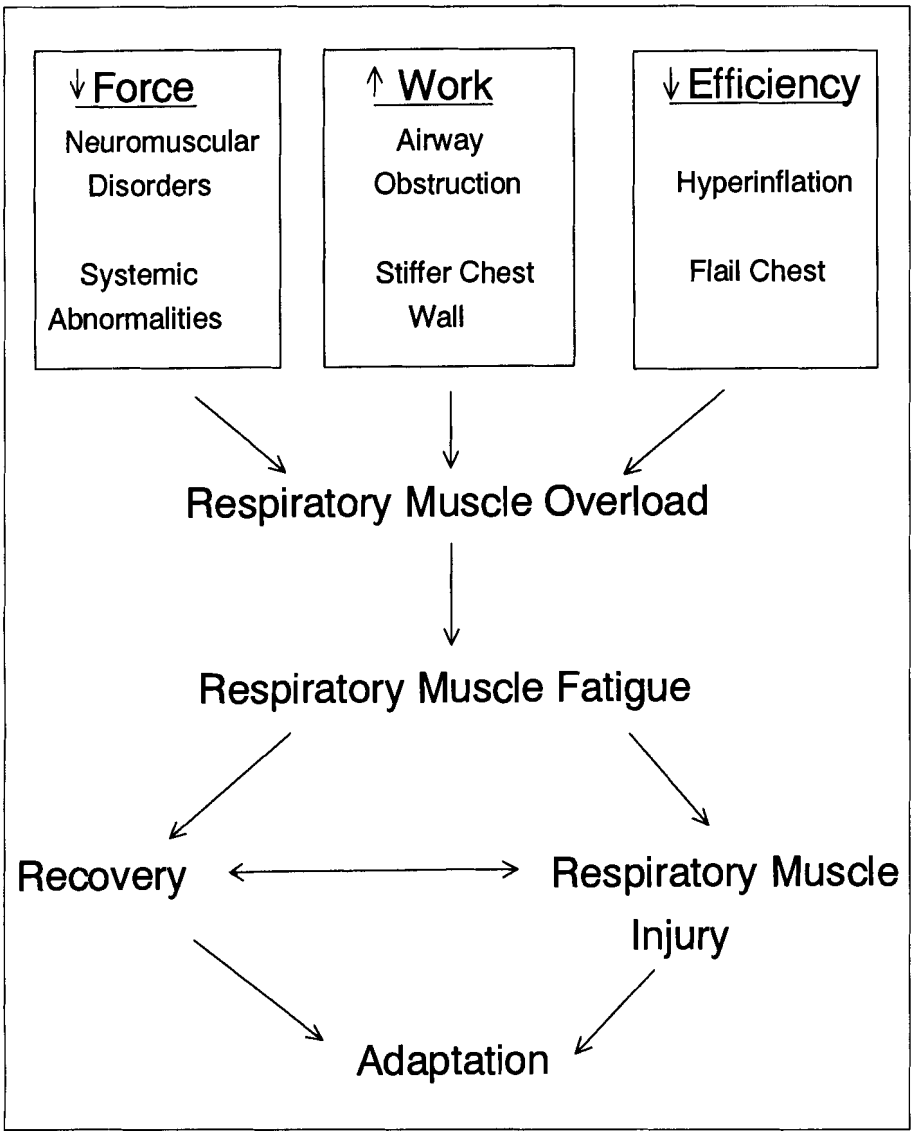

Figure 5. *Factors contributing to respiratory muscle dysfunction. In several different conditions, decreased force, increased work of breathing, or decreased efficiency of the respiratory muscles contributes to an increased relative overload to the respiratory muscle. This excessive loading may contribute to respiratory muscle fatigue and injury. Recovery from these entities may occur, followed by a positive adaptive or maladaptive response of the respiratory muscles.*

nerves. The scalene muscles are innervated by C2–C7,[12] and the sternocleidomastoid muscles are innervated by the accessory nerve and the C2 and C3 roots.[13] The intercostal muscles receive afferent and efferent innervation from the anterior primary rami of the intercostal nerves (T1–T11). Similar to the intercostal muscles, the abdominal muscles receive segmental innervation from spinal nerves, but from only the lower levels. The rectus abdominis, external and internal oblique, and transversus abdominis muscles all receive their supply from the lower six thoracic nerves (T7–T12).[3] The transversus abdominis and internal oblique muscles also receive their supply from the L1 root.

Conditions Associated With Respiratory Muscle Dysfunction

In many different situations, respiratory muscle dysfunction may contribute to impaired exercise tolerance, dyspnea, and respiratory failure in individuals with respiratory compromise. A better understanding of the factors contributing to respiratory muscle dysfunction could enable physical therapists to design more effective treatment plans. This section will outline factors that contribute to respiratory muscle dysfunction. These factors can be broadly grouped into those that decrease the force of the respiratory muscles, those that increase the work of breathing due to changes in the lungs or chest wall, and those that decrease respiratory muscle efficiency such that a relative overload is imposed on the respiratory muscles (Fig. 5).

Fatigue of the inspiratory muscles has been defined as the loss of force-generating ability or the ability to shorten, which is reversible by rest.[14] The reversibility of fatigue is an important component that differentiates fatigue from weakness, which is the loss of force-generating ability present in the rested muscle.[14] Although these definitions appear quite clear, it is often difficult to differentiate between weakness and fatigue for two major reasons. First, mouth or transdiaphragmatic pressure rather than inspiratory muscle force is measured for an estimate of inspiratory muscle force, and there are many factors that contribute to both intrasubject and intersubject variability in this measurement. Second, *fatigue* is defined as impaired performance by the inspiratory muscles, which is reversible by rest, and it is very difficult to rest the inspiratory muscles in order to truly distinguish fatigue from weakness. Although it is difficult to differentiate between weakness and fatigue, it is quite possible that both of these entities may be present in some patients.

Weakness of the inspiratory muscles caused by acute or chronic disorders may result in the inability to cope with the normal loads of respiration or increased loads associated with respiratory disease. Conditions contributing to this inspiratory muscle weakness can include metabolic abnormalities[15] (Table), shock,[16] sepsis,[17] infection,[18] malnutrition,[19–21] steroid administration,[22] and disuse.[20] There are also many chronic conditions associated with respiratory muscle weakness

Table. *Conditions Sometimes Associated With Respiratory Muscle Weakness*[a]

Neural damage	Central nervous system	Quadriplegia
		Amyotrophic lateral sclerosis
	Peripheral nervous system	Guillain-Barré syndrome
		Traumatic injury of phrenic nerve during surgery
		Hereditary motor and sensory neuropathy (eg, Charcot-Marie-Tooth Disease)
Neuromuscular junction		Myasthenia gravis
		Botulism
		Lambert-Eaton myasthenic syndrome
Myopathies		Duchenne's muscular dystrophy
		Steroid-induced myopathy
		Alcoholic myopathy
		Acid maltase deficiency
		Nemaline myopathy
		Cytoplasmic body neuropathy
		Rhabdomyolysis
Connective tissue disorders		Rheumatoid arthritis
		Ankylosing spondylitis
		Scleroderma
		Systemic lupus erythematosus
		Dermatomyositis
Systemic abnormalities	Endocrine disorders	Hypothyroidism
		Hyperthyroidism
		Cushing's disease
	Metabolic abnormalities	Hypophosphatemia
		Hypomagnesemia
		Hypokalemia
		Hypoxia
		Hypercapnia
		Metabolic acidosis

[a]Reprinted from Reid[15] by courtesy of Marcel Dekker Inc.

such as neuromuscular disorders; connective tissue disorders; and systemic abnormalities (Table), including malnutrition.[15,20]

Chronic obstructive lung disease can result in respiratory muscle dysfunction because of respiratory muscle weakness, increased work of breathing due to changes in the lungs, and inefficiency of the inspiratory muscles because of hyperinflation. Respiratory muscle weakness may occur because of systemic abnormalities (Table) such as poor nutrition,[19–21] abnormal arterial blood gases,[23,24] electrolyte imbalances,[20] and disuse.[20] Increased work of breathing results from airway obstruction due to mucus in the airway, inflamed airway walls, bronchospasm, and alveolar destruction (Fig. 6). This airway obstruction results in airway compression and hyperinflation (Fig. 7). Hyperinflation (which is abnormally large lung volumes) causes all of the inspiratory muscles to operate at lengths shorter than normal. Because of the length-tension curve, this condition places these muscles at a disadvantage for tension generation, with the diaphragm being affected to the greatest degree[25] (Fig. 8). In patients with chronic obstructive pulmonary disease (COPD), respiratory muscle dysfunction may contribute to dyspnea and exercise intolerance, and as the disease progresses, to hypercapnic ventilatory failure.[2] *Hypercapnic ventilatory failure* is defined as an increase in arterial partial pressure of carbon dioxide ($Paco_2$) above 45 mm Hg and a proportionate decrease in arterial partial pressure of oxygen (Pao_2).[20]

In kyphoscoliosis, most of the increased work of breathing is due to an increased stiffness of the chest wall and not due to an increased stiffness of the lungs.[20] This increased work of breathing may contribute to respiratory muscle fatigue and ultimately to hypercapnic ventilatory failure.[20,26] In time, the lungs also become stiffer because of lung infection and atelectasis,[27] which may further increase the work of breathing. Systemic abnormalities such as poor arterial blood gases may further exacerbate inspiratory muscle function.

In contrast to kyphoscoliosis, the primary pathology in interstitial lung disease results in an increased stiffness of the lungs and not an increased stiffness of the chest wall. The work of breathing imposed by the less compliant lungs, however, is not usually considered great enough to result in inspiratory muscle fatigue.[28] Individuals with this condition may develop respiratory muscle compromise when treated with high-dose steroids to alleviate inflammation of the lungs.[22] High-dose steroids induce a myopathy that may affect the respiratory muscles. A recent study[29] showed that respiratory muscle training can prevent the loss of respiratory muscle force and endurance that occurs in individuals taking high-dose steroids.

In summary, inspiratory muscle weakness, increased work of breathing due to changes in the lungs or chest wall, or decreased efficiency of the inspiratory muscles may result in a relative overload being imposed on the respiratory muscles, leading to fatigue and possibly injury of the inspiratory muscles (Fig. 5). In patients with poor respiratory muscle function, it is difficult to differentiate between weakness and fatigue of the inspiratory muscles. Regardless, both of these entities can manifest as decreased exercise tolerance, dyspnea, and respiratory failure, any of which may be very debilitating.

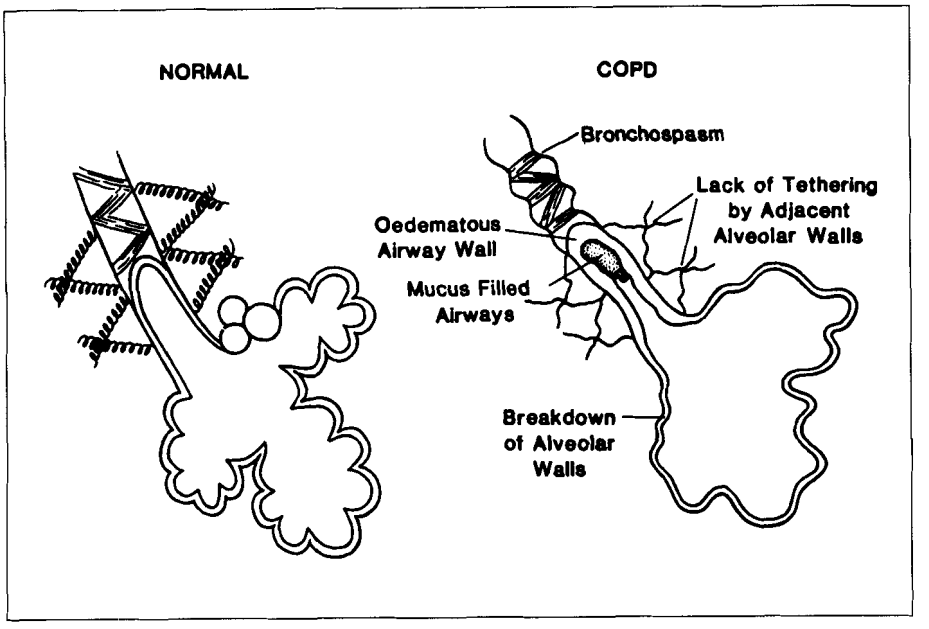

Figure 6. *Pathophysiology of chronic obstructive pulmonary disease (COPD). (Reprinted with permission from Reid WD, Loveridge BM. Physiotherapy management of patients with chronic obstructive airway disease.* Physiotherapy Canada. *1983;35:183–195.)*

Problems Associated With Respiratory Muscle Dysfunction

Exercise Intolerance

In many patients, the combination of increased respiratory loads and exercise may greatly hamper the ability of the inspiratory muscles to perform. Although there are many different conditions in which respiratory muscle dysfunction may contribute to exercise intolerance, the contribution of poor respiratory muscle function to exercise intolerance has primarily been explored in patients with COPD and interstitial lung disease.

In patients with COPD, the respiratory muscles may be weak because of many metabolic abnormalities (Table) or because of other factors such as prolonged inactivity,[30] steroid use,[22] poor nutrition,[19–21] and hyperinflation.[30] During exercise and other daily activities that increase ventilatory rates, inspiratory muscle function may be further weakened because of higher speeds of shortening and shorter operating lengths of the inspiratory muscles during the high ventilatory levels required by these activities. Higher amounts of ventilation increase the frequency of breathing, which shortens inspiratory and expiratory times for each breath. Shorter expiratory times in combination with airway obstruction and loss of structural stability of the small airways (due to alveolar destruction) results in greater dynamic compression of the airways than at rest and increased air trapping (Fig. 7). The resulting hyperinflation places all the inspiratory muscles, including the diaphragm, in shorter positions[2,30] (Fig. 8). Thus, the preexisting mechanical disadvantage of the inspiratory muscles is increased during exercise. Expiratory times, therefore, cannot be shortened to any great extent during high levels of ventilation. To overcome this limitation, the shortening speed of the inspiratory muscles increases greatly in order to reduce inspiratory time and raise inspiratory flow rate.[30] Thus, the combination of exercise and increased respiratory loads in patients with COPD requires the inspiratory muscles to work at greater lung volumes and high speeds, which can accentuate the weakness already present in these muscles. This exacerbation of weakness may result in situations in which the respiratory muscles are unable to generate the forces necessary to pump air in and out of the lungs, which may limit the ability to perform exercise and other activities.[30]

Whether increased ventilatory loads contribute to inspiratory muscle fatigue in patients with COPD during exercise is controversial. Changes in the electromyographic frequency spectrum consistent with respiratory muscle fatigue have been found in patients with COPD during exercise by some researchers[31,32] but not by others.[28,33] Although the presence of respiratory muscle fatigue is equivocal, there is no doubt that the demands placed on the respiratory muscles during exercise are very high. Improvement in inspiratory muscle force and endurance by training may reduce the perception of dyspnea and improve exercise tolerance. This outcome has been demonstrated in a few studies, but not all studies that have examined the benefits of respiratory muscle training have found a beneficial effect (see article by Reid and Samrai in this issue).

In interstitial lung disease, the primary pathophysiology results in noncompliant alveoli[34] in contrast to the airway obstruction found in patients with COPD. Many of the metabolic abnormalities and systemic factors such as poor nutrition and abnormal arterial blood gases that contribute to weak respiratory muscles in patients with COPD may be present in interstitial lung disease, but the worsening due to flow limitation during exercise is not. In interstitial lung disease, the major factors contributing to ventilatory impairment appear to be related to lung and not respiratory muscle function; worsening of ventilation and perfusion matching and pulmonary diffusion defects cause decreased saturation of oxygen.[35] Ventilatory impairment during exercise in this group of individuals results from both an increase in minute ventilation relative to oxygen consumption and a reduction in the overall capacity to increase ventilation compared to asymptomatic people.[35] Although the inspiratory muscles of these patients

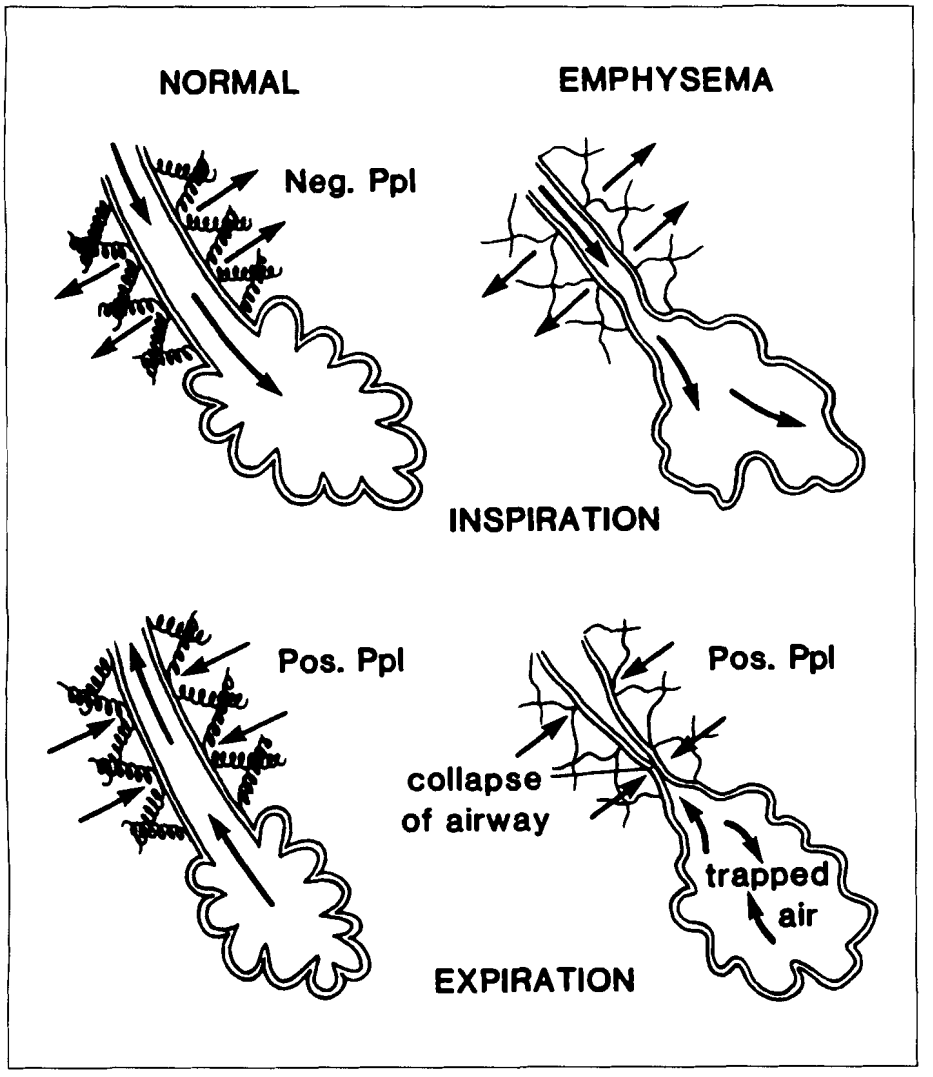

Figure 7. *Dynamic compression of airway. The breakdown of alveolar walls in emphysema decreases the tethering action of alveoli adjacent to the small airway. During inspiration, the negative pleural pressure (Ppl) tends to keep airway open. During expiration, the reverse situation occurs; pleural pressure becomes more positive, tending to collapse the smaller, more compliant airway, which results in air trapping in the distal alveoli. This trapping of air causes hyperinflation (abnormally large lung volumes) in patient with chronic obstructive pulmonary disease. (Reprinted with permission from Reid WD, Loveridge BM. Physiotherapy management of patients with chronic obstructive airways disease.* Physiotherapy Canada. *1983;35:183–195.)*

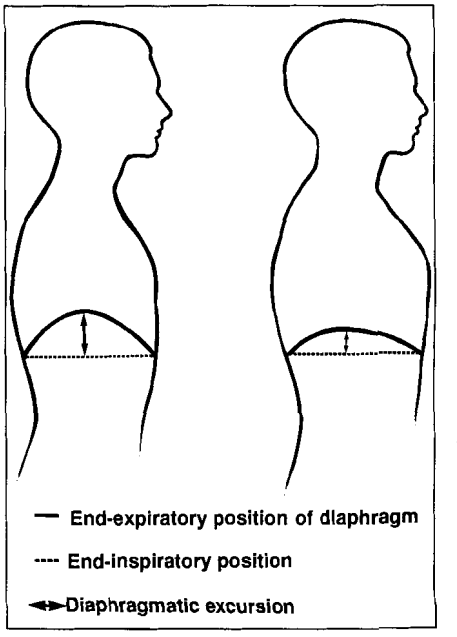

Figure 8. *Hyperinflation flattens the diaphragm and decreases its pumping ability. In an asymptomatic individual (left), the diaphragm is dome shaped at end expiration and has a large potential for diaphragmatic excursion. In the individual with chronic obstructive pulmonary disease (right), hyperinflation flattens the end-expiratory position and decreases the potential for diaphragmatic excursion.*

may be compromised, the demands placed on the respiratory muscles during exercise are less than in patients with COPD, and evidence of respiratory muscle fatigue in individuals with interstitial lung disease has not been demonstrated.[28]

Dyspnea

Dyspnea is the most common symptom limiting exercise and activities of daily living in individuals with conditions such as COPD[30] and interstitial lung disease.[35] Despite the considerable amount of research exploring the etiology of dyspnea, the relationship among dyspnea, respiratory muscle dysfunction, and exercise intolerance is unclear.

Dyspnea was thought to be caused by increases in muscle tension related to increases in the work of breathing, stimulation of the pulmonary J receptors, or alteration in the Pa_{O_2} or Pa_{CO_2} homeostasis.[36] Studies have shown, however, that none of these phenomena are essential for the perception of dyspnea. Persons with C1–2 quadriplegia[37] and subjects with drug-induced paralysis[38] were able to identify increases in Pa_{CO_2} by the respiratory discomfort experienced during hypercapnia, which suggests that breathlessness is not purely due to increases in muscle tension or the work of breathing. Sensory feedback from pulmonary receptors is also not essential for the perception of dyspnea, as shown by studies that blocked all pulmonary receptors by pharmacologic agents[39] or surgery[40] and failed to eliminate exercise-induced breathlessness. Both hypercapnia and hypoxia also show a lack of specificity with regard to the sensation of dyspnea they create.[41,42] Therefore, there is not a specific relationship between dyspnea and input to the central nervous system from a specific set of peripheral receptors such as respiratory muscle tension, pulmonary stretch receptors, or chemoreceptors.

Dyspnea on exertion seems to originate from the amount of central nervous system output to the respiratory muscles instead of input to the central nervous system from a specific set of peripheral receptors.[43] It is likely that several factors such as increased muscle tension, changes in speed of muscle contraction, and alterations in the body's acid-base balance may increase the output from the respiratory center such that once a threshold is reached, any further increase in this output is perceived as dyspnea. Respiratory muscle training may alleviate dyspnea via adaptation to the sensation, or by decreasing the relative output from the respiratory center by decreasing inspiratory muscle weakness or improving efficiency of breathing.

Hypercapnic Ventilatory Failure

Hypercapnic ventilatory failure can occur in several acute and chronic conditions associated with a relative overload imposed on the respiratory muscles and may, at least in part, be attributed to respiratory muscle dysfunction.[20,26] *Respiratory failure* can be defined as the inability to ventilate adequately, as demonstrated by a decrease in the Pao_2 below 55 mm Hg or a rise in the $Paco_2$ above 45 mm Hg.[20] Respiratory failure can arise due to a problem within the lungs that is known as hypoxemic respiratory failure, or it may be due to a problem with the respiratory muscles or chest wall that is known as hypercapnic respiratory failure or hypercapnic ventilatory failure. *Hypoxemic respiratory failure* can originate from conditions such as pneumonia or pulmonary edema. The primary arterial blood gas disturbance in this type of respiratory failure is a low Pao_2. This hypoxemia is usually accompanied by a normal or even a low $Paco_2$. If the disease is severe, however, the $Paco_2$ may rise to elevated levels. In contrast, *hypercapnic ventilatory failure* can arise from multiple origins, including a failure of the respiratory muscle pump or an alteration in breathing pattern. This type of respiratory failure is manifested by an increase in $Paco_2$ and a proportionate decrease in the Pao_2 (usually the $Paco_2$ is increased 1 mm Hg for every 1-mm Hg decrease in the Pao_2).[20]

Respiratory muscle dysfunction has been implicated as a cause of hypercapnic ventilatory failure; however, the specific etiology of this dysfunction is unclear at this time. Roussos[26] hypothesized several years ago that respiratory muscle fatigue leads to hypercapnic ventilatory failure. More recently, Rochester[44] proposed that weakness rather than fatigue contributes to hypercapnia. He postulated that patients with COPD have weak respiratory muscles, and hence they breathe with smaller tidal volumes to avoid fatigue. This type of breathing pattern has a larger dead space-to-tidal volume ratio, which increases $Paco_2$ levels. Reid and colleagues[45,46] demonstrated that increased resistive loading in animal models is associated with diaphragm muscle injury and hypercapnic ventilatory failure. Extensive studies have not been performed in humans, but a few reports have documented injury of the diaphragm in infants[47] and in adults with COPD.[47,48] Although it is not clear whether respiratory muscle dysfunction contributes to hypercapnic ventilatory failure, respiratory muscle training could potentially decrease or prevent ventilatory failure by alleviating fatigue, improving endurance, and preventing injury of the respiratory muscles.

Special Considerations for Respiratory Muscle Testing and Training

Because of anatomical and functional differences between the respiratory and limb muscles, some aspects of testing and training must be approached differently. These special considerations will be discussed.

Anatomical Location

Because the inspiratory muscles surround the thoracic cavity and act to pump air in and out of the cavity rather than crossing and moving a single joint, their function is more difficult to assess than that of many limb muscles and cannot be examined by determining torque output on devices such as an isokinetic dynamometer. The most common measurements of respiratory muscle force are the maximal inspiratory or expiratory mouth pressures (see the article by Clanton and Diaz in this issue for a full explanation of this technique) (Fig. 9). These inspiratory and expiratory pressures are estimates of the force produced by all the inspiratory muscles or all the expiratory muscles, respectively. More specifically, dia-

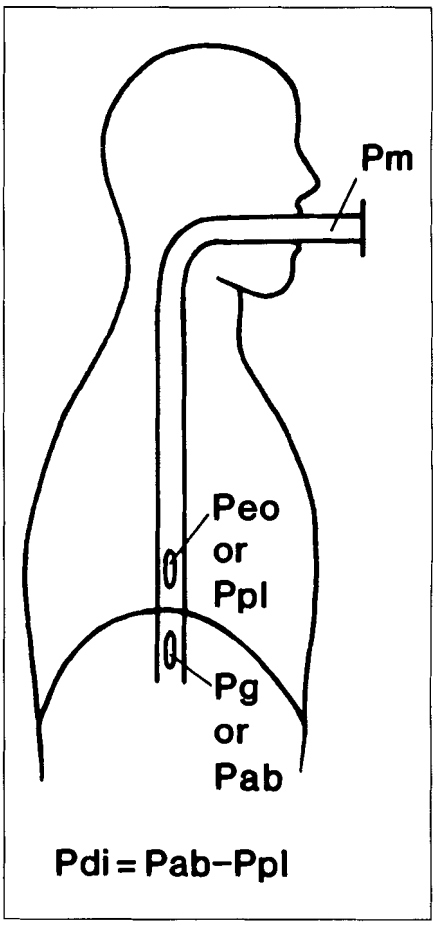

Figure 9. *Mouth pressure (Pm) and transdiaphragmatic pressure (Pdi). Mouth pressure is the force exerted by the respiratory system (respiratory muscles, chest wall, and lungs) as measured at the mouth. Transdiaphragmatic pressure, the force exerted by the diaphragm, is the difference between esophageal pressure (Peo) or pleural pressure (Ppl) and gastric pressure (Pga) or abdominal pressure (Pab), which is calculated by Pdi=Pab−Ppl. (Reprinted with permission from Reid WD, Loveridge BM. Physiotherapy management of patients with chronic obstructive airways disease.* Physiotherapy Canada. *1983;35:183–195.)*

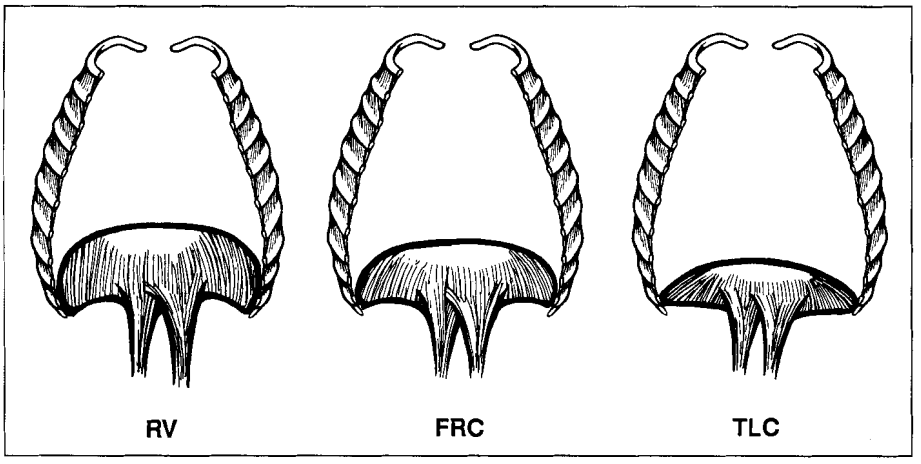

Figure 10. *Starting (end-expiratory) length of the diaphragm at residual volume (RV), functional residual capacity (FRC), and total lung capacity (TLC). Note the more dome-shaped and longer length of the diaphragm at RV compared with TLC.*

phragmatic muscle force can be estimated by measuring transdiaphragmatic pressure, which is the difference between the esophageal and abdominal pressures (Fig. 9). This, however, is a much more uncomfortable test because it requires balloon-tipped catheters to be inserted down into the lower third of the esophagus and the stomach. Thus, transdiaphragmatic pressure is usually measured only for research purposes.

Other complicating factors when examining respiratory muscle function are the unusual dome shape and internal location of the diaphragm, which make resting length or changes in length impossible to determine except with sophisticated imaging techniques. Clinically, lung volumes are used to standardize starting length (Fig. 10). Theoretically, it is preferable to measure maximal inspiratory and expiratory pressures at functional residual capacity because the recoil of the lungs or chest wall will not influence the respiratory muscle force produced. The therapist cannot be sure, however, that functional residual capacity is achieved without using sophisticated pulmonary function equipment. Practically, it is much easier to standardize residual volume and total lung capacity without special pulmonary function equipment; thus, maximal inspiratory and expiratory pressures are commonly performed at residual volume and total lung capacity, respectively. The amount of movement produced by the respiratory muscles (ie, distance traveled) cannot be measured directly, so minute ventilation is used to estimate this value. The speed of respiratory muscle contraction is estimated by the flow of air moving in and out of the mouth (as measured by a pneumotach or flowmeter distal to a mouthpiece).

Type of Load

The function of the inspiratory muscles epitomizes the ultimate in an endurance load. The inspiratory muscles work against low-intensity loads throughout the life span. Thus, endurance testing is more informative and endurance training is more beneficial in most patient groups than strength testing and training. In skeletal muscle, improved endurance performance is associated with increased oxidative capacity due to higher levels of oxidative enzymes, larger substrate stores of lipid and glycogen, and increased numbers of capillaries.[49,50] Because the principle of training specificity applies to respiratory muscles, training protocols should focus on endurance activities that facilitate these kinds of subcellular changes within the muscle fibers.

The respiratory muscles can be strengthened[51,52]; however, the benefit of stronger respiratory muscles for asymptomatic individuals and most patient groups is not obvious, because the greater loads imposed by most respiratory conditions primarily require a higher level of endurance rather than greatly increased strength. If the respiratory muscles are very weak, some strengthening may be a necessary part of the endurance training program. In this case, an inspiratory muscle training technique with an element of strengthening such as threshold training should be chosen rather than the static, quasi-isometric strength training techniques used previously.[51,52] The term "quasi-isometric" has been used to describe "static" contractions of the inspiratory or expiratory respiratory muscles performed against an occluded airway because a small amount of muscle shortening occurs due to intrathoracic gas decompression or compression, respectively. This intrathoracic gas decompression or compression, and hence the amount of respiratory muscle shortening, is greater with higher respiratory muscle forces. In contrast, there may be some groups of patients who benefit from quasi-isometric respiratory muscle strength training. For instance, expiratory muscle strength is important for improving cough in patients with quadriplegia.[53] Those patients with weak expiratory muscles may benefit from specific strength training of the expiratory muscles.

Because training of respiratory muscles is activity specific, respiratory muscles should be trained using conditions that simulate those encountered during breathing. Respiratory muscles must shorten during contraction and work against both resistive and elastic loads. These two types of loads will be described.

Resistance is the part of the pressure change that occurs during respiration that is related to flow and can be increased due to changes in both the airway and tissues of the respiratory system. Narrow airway diameter and increasing flow are the most common causes of increased airway resistance.[54] Stiffening of lung or chest wall

tissues and impeding movement of these tissues will also contribute to an increase in respiratory system resistance. Some examples of increased resistive loads during breathing are a decrease in the airway diameter during bronchospasm, increasing flow during high levels of ventilation, and stiffening of the lung tissue in conditions such as interstitial pulmonary fibrosis. By definition, resistive loads increase proportionally to the speed of muscle shortening (ie, a higher flow rate will increase the resistive load to the respiratory muscles). Inspiratory muscle training should be performed at similar or higher resistive loads to those experienced by patients during an acute exacerbation or during high levels of ventilation required during exercise or other daily activities.

Elastance is the inverse of compliance, and an increase in elastance implies that the system is becoming stiffer. Elastance is the part of the pressure change during respiration that is related to volume change. As volume of the respiratory system increases, the elastic load increases.[54] For example, the elastic load is larger at total lung capacity versus functional residual capacity in asymptomatic people and may also be increased in patients with COPD or asthma during hyperinflated states. Elastic loads increase proportional to the amount of muscle shortening (ie, as lung volumes increase, the elastic loads imposed on the respiratory muscles increase). Ideally, inspiratory muscle training should incorporate elastic loading for those patients where it might be relevant to the demands that they face in their daily activities.

Mechanical efficiency is influenced by the ability of the central nervous system to control the generation of tension and movement patterns.[55] Therefore, it is essential that training fosters that neurological recruitment pattern required for the inspiratory muscles to meet the repetitive elastic and resistive loads performed. Little is known, however, about the type of training needed to optimize this outcome. Perhaps the best guideline is to use an activity that simulates a particular patient's breathing pattern and load during high ventilatory demands. The specifics of respiratory muscle testing and training prescription will be discussed in the article by Reid and Samrai and the article by Clanton and Diaz in this issue.

Special Needs of Patients

Careful monitoring of oxygen saturation during testing or training the inspiratory muscle is essential because the therapist is usually dealing with patients with some sort of respiratory compromise. These patients have decreased sensitivity such that they are less likely to increase ventilation in response to an increase in $Paco_2$. Thus, they could very easily experience desaturation of hemoglobin and allow their end-tidal carbon dioxide to rise when presented with additional inspiratory loads during testing and training (Pardy RL, MD, Fairbarn MS, MSc; unpublished research; 1994). Oximetry can be used to monitor oxygen saturation. Oximetry is noninvasive and very easy to perform, and therefore will not affect patient performance during testing and training.

Care should be taken when progressing the work load to avoid undue inspiratory muscle fatigue or injury because of the essential function of the respiratory muscles. We have demonstrated in two different animal models that increased resistive loading over several days results in diaphragm injury and inflammation.[45,46] It is possible that overloading human inspiratory muscles during training could induce inspiratory muscle fatigue and injury, and precipitate ventilatory failure.

Conclusion

Many patients with respiratory compromise may experience respiratory muscle dysfunction that could contribute to exercise intolerance, dyspnea, and ultimately hypercapnic ventilatory failure. Respiratory muscles, similar to limb muscles, improve their function in response to training. Thus, exercise intolerance, dyspnea, and hypercapnic ventilatory failure may be prevented or alleviated by effective training regimens. By understanding the similarities and the differences between limb muscles and respiratory muscles, physical therapists can more effectively design the most appropriate training programs.

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Considerations When Testing and Training the Respiratory Muscles

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