

# CHEST<sup>®</sup>

Official publication of the American College of Chest Physicians



## Respiratory muscle function in health and disease

JM Luce and BH Culver

*Chest* 1982;81:82-90  
DOI 10.1378/chest.81.1.82

The online version of this article, along with updated information and services can be found online on the World Wide Web at:

<http://chestjournal.org>

CHEST is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright 2007 by the American College of Chest Physicians, 3300 Dundee Road, Northbrook IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder (<http://www.chestjournal.org/misc/reprints.shtml>). ISSN: 0012-3692.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S<sup>®</sup>

## CRITICAL REVIEW

# Respiratory Muscle Function in Health and Disease\*

John M. Luce, M.D., F.C.C.P.; and Bruce H. Culver, M.D.

The muscles of respiration comprise three groups: the diaphragm, the intercostal and accessory muscles, and the muscles of the abdomen (Fig 1). All three groups have inspiratory and expiratory function and work together in intricate ways. This article reviews respiratory muscle action and interaction. We also discuss respiratory muscle dysfunction and its treatment in several disease states, including chronic obstructive pulmonary disease, asthma, chest wall deformity, obesity, generalized neuromuscular disorders, cervical cord injury, and unilateral and bilateral diaphragmatic paralysis. We conclude with a discussion of how training may influence respiratory muscle fatigue. Other issues, including central nervous system control of the respiratory muscles, are covered by Derenne and colleagues.<sup>1</sup> Their article contains an extensive bibliography.

### THE DIAPHRAGM

In their comprehensive review of the respiratory muscles, Derenne and associates<sup>1</sup> note that the diaphragm is inserted onto the ribs and that at the periphery its fibers are directed upwards, parallel to the rib cage. When the diaphragm contracts, it pushes down on the abdominal viscera, displaces the abdominal wall outward, and increases abdominal pressure ( $P_{ab}$ ). At the same time, the diaphragm lifts the rib cage upward, causing an outward displacement because of its rib articulation, and generates a negative pleural pressure ( $P_{pl}$ ) that inflates the lungs (Fig 2). The increased abdominal pressure, applied to the lower rib cage below the diaphragm, also contributes to the outward displacement. The amount of respiratory work performed by the diaphragm during inspiration is reflected in the pressure gradient across

the diaphragm,  $P_{di}$ , which is the difference between  $P_{ab}$  and  $P_{pl}$ . These values can be obtained by simultaneously recording pressures in esophageal and gastric balloons. In normal persons,  $P_{di}$  ranges from 10 to 20 cm  $H_2O$  during quiet breathing.

Although the diaphragm is primarily an inspiratory muscle, it has an expiratory action on the rib cage when its dome is flattened and its peripheral fibers are directed centrally rather than towards the head. This happens normally at high lung volumes, but it also can occur during tidal breathing in hyperinflated patients with an increased functional residual capacity (FRC). In such persons, the negative  $P_{pl}$  generated by the diaphragm sucks in the lower part of the chest, and the rib cage actually may decrease in its lateral dimension. The expiratory action of the diaphragm at high lung volumes

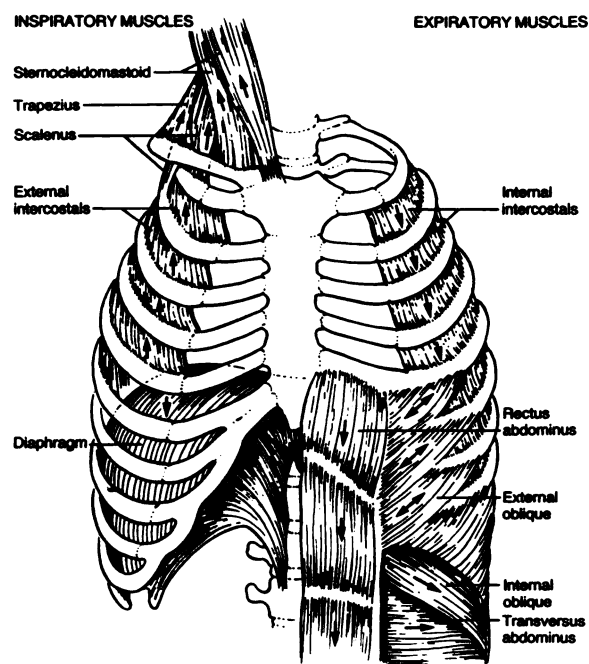
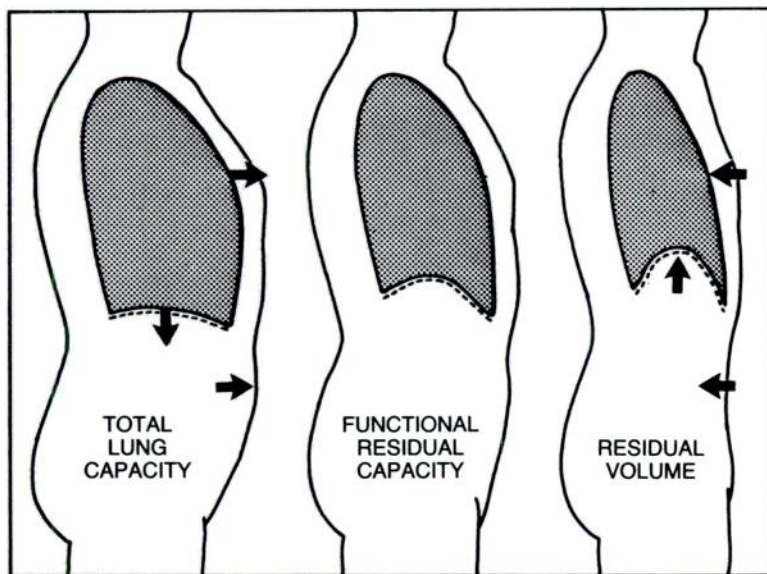


FIGURE 1. Inspiratory and expiratory muscles of respiration. See text for explanation.

\*From the Department of Medicine, Division of Respiratory Diseases, University of Washington School of Medicine, and the USPHS Hospital, Seattle.

Reprint requests: Dr. Culver, Division of Respiratory Diseases, USPHS Hospital, Seattle 98144



**FIGURE 2.** Normal respiratory muscle function. In inspiration, diaphragmatic contraction pushes down on the abdominal viscera and displaces the chest outward. The diaphragm also lifts the rib cage upward and outward and inflates the lung towards its total capacity, while intercostal and accessory muscles stabilize the chest wall. In expiration, the diaphragm and other inspiratory muscles relax, and contraction of the abdominal muscles helps the lung achieve residual volume. In this illustration, as in those that follow, the arrows represent direction of movement, not muscular force applied.

explains Hoover's sign, an inspiratory indrawing of the lateral costal margins seen in some patients with chronic obstructive pulmonary disease (COPD).

#### INTERCOSTAL AND ACCESSORY MUSCLES

The intercostal muscles include the internal and external intercostals, whose fibers run in an anterior-posterior direction. The accessory muscles include the scalenes, the sternomastoids, and the trapezoids. The external intercostal and the accessory muscles serve primarily an inspiratory function and are responsible for increasing the anterior-posterior diameter of the thorax. Inspiratory activity can be observed in the external intercostals in the upper few intercostal spaces during quiet breathing, with the lower intercostals becoming active with increased ventilation. The scalenes, sternomastoids, and trapezoids are recruited at high levels of inspiratory activity, at which point other back muscles that support the chest cage also become involved.

#### ABDOMINAL MUSCLES

The abdominal respiratory muscles include the rectus and transverse abdominis and the external and internal obliques. These generally are regarded as expiratory muscles that augment the passive recoil of the lungs, especially during deep and forceful breathing. Yet, the abdominal muscles also play a facilitatory role in inspiration in that their contraction tends to lengthen the diaphragm and diminish its radius of curvature, allowing it to generate a greater tension as well as greater Pdi for a given tension. This improvement in the diaphragm's mechanical advantage is significant in the upright posture, particularly during exercise.

#### RESPIRATORY MUSCLE INTERACTION

Goldman and Mead<sup>2</sup> believe that the diaphragm performs all the body's respiratory muscle work during quiet breathing in the upright posture and have demonstrated that the rib cage is driven along its relaxation curve in experimental subjects trying to breathe with the diaphragm alone. In contrast to this viewpoint, Macklem et al<sup>3</sup> have argued that the intercostal and accessory muscles are never electrically silent and that their contribution is necessary to properly displace the chest wall. Danon and colleagues<sup>4</sup> studied three quadriplegic patients with lesions at C<sub>1</sub>, who for the purposes of the experiment breathed either with only their accessory muscles, with electrically-paced diaphragms, or with a combination of the two groups. They determined rib cage movement by means of magnetometry, in which magnets are placed on opposite sides of the chest and their displacement is recorded. The investigators demonstrated that the diaphragm functioning alone did not drive the rib cage along its relaxation curve, but instead caused rib cage distortion when the patients were semi-upright. Paradoxical movements in the upper rib cage due to the absence of accessory muscle activity also occurred in this position. These findings suggest that the other respiratory muscles stabilize the chest wall and convert diaphragmatic contraction into intrathoracic pressure and volume changes.

#### RESPIRATORY MUSCLE FATIGUE

In keeping with its prominent role in respiration, the diaphragm is well equipped for sustained activity, containing muscle fibers and mitochondrial enzymes that favor aerobic metabolism.<sup>5</sup> Rochester and Bettini,<sup>6</sup> who studied dogs breathing an

increased carbon dioxide (CO<sub>2</sub>) mixture through inspiratory resistances, found that the diaphragm's increased oxygen (O<sub>2</sub>) requirements were met by a combination of blood flow and O<sub>2</sub> extraction at high levels of work, at which point diaphragmatic blood flow continued to increase while oxygen extraction plateaued. Robertson et al<sup>7</sup> confirmed these findings in experimental animals and determined that conditions of extreme diaphragmatic work were associated with no limitation in diaphragmatic blood flow.

In spite of these studies, clinical experience suggests that respiratory muscle fatigue does occur at high levels of exertion, presumably because the diaphragm outstrips its blood supply. How this might occur was suggested in a recent study by Aubier and colleagues,<sup>8</sup> who measured the diaphragmatic electromyogram (EMGdi), Pdi, and diaphragmatic lactate production in dogs whose cardiac output was markedly reduced by tamponade. One group of animals was mechanically ventilated during this period; another breathed spontaneously. The investigators found that whereas the ventilated dogs survived several hours of experimentally-induced shock, the spontaneously breathing animals, which were expending large amounts of respiratory muscle work, died when they could no longer generate a measurable Pdi in spite of EMGdi activity. Since the animals' diaphragmatic lactate levels were increasing before death and lactate levels from other muscle groups were not rising, the investigators concluded that the dogs died with, if not from, diaphragmatic fatigue.

The amount of respiratory muscle work humans can sustain has been determined by Tenney and Reese.<sup>9</sup> These investigators measured the maximum voluntary ventilation (MVV) in five athletic subjects on the basis of 20 second efforts in which the subjects chose their own tidal volume and frequency. The subjects then breathed until they could not keep pace with these parameters or until they were completely exhausted. On the average, the subjects could breathe for less than one minute and to a minute ventilation of from 150 to 200 liters, an amount from 80 to 90 percent of their original MVV. However, they could breathe indefinitely if their minute ventilation was 55 percent or less of the MVV.

Roussos and Macklem<sup>10</sup> have provided another perspective on respiratory muscle fatigue by having their subjects breathe through inspiratory resistance while generating a Pdi amounting to a predetermined fraction of the maximum Pdi (Pdi<sub>max</sub>) they could generate at FRC. The investigators determined that the critical Pdi where fatigue devel-

oped in the subjects was some 40 percent of their Pdi<sub>max</sub>; below this level, they could breathe indefinitely. Gross and colleagues<sup>11</sup> demonstrated that diaphragmatic fatigue is heralded by a shift in the ratio of the amplitude of the high and low frequency components of the EMGdi. This shift is not present at a Pdi which is 25 percent of the Pdi<sub>max</sub> subjects can generate, but it does occur when the Pdi is around 50 percent of maximum. Because of this, the investigators believe that frequency spectrum analysis of the EMGdi can detect fatigue before the muscle fails entirely.

Using this new technique, Gross and co-workers<sup>12</sup> demonstrated a shift in the frequency spectrum of the diaphragm and sternomastoid EMG in C<sub>3-7</sub> quadriplegic patients breathing through inspiratory resistances, indicating inspiratory muscle fatigue. Muller et al<sup>13</sup> have recorded the same characteristic EMG changes in premature infants during rapid-eye-movement (REM) sleep, when the intercostal and accessory muscles are less active and unopposed diaphragmatic motion distorts the chest wall. Andersen and associates<sup>14</sup> have observed similar EMG patterns in patients with respiratory failure receiving mechanical ventilation; magnetometry revealed that many of these patients display thoracoabdominal discoordination during weaning from mechanical ventilation as well.

These findings suggest that discoordinate breathing is maladaptive, if not abnormal. However, Roussos and Macklem<sup>15</sup> measured the elevated activity of the diaphragmatic and intercostal and accessory muscles in five normal seated subjects breathing through inspiratory resistance until exhaustion and found that the subjects alternated their use of the two muscle groups. Since neither muscle group was entirely inactive, the investigators felt that at any one moment the less active group supported the chest wall to enhance the function of the other group. Thus, the apparent discoordination activity was a coordinated effort that served to protect both muscle groups from total exhaustion by alternating their contribution to the breathing task.

#### RESPIRATORY MUSCLE DYSFUNCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Patients with COPD must breathe at high lung volumes to maintain patency of their narrowed airways. The major abnormality of respiratory muscle function in patients with COPD is thought to be the mechanical disadvantage caused by this hyperinflation, which depresses the dome of the diaphragm, shortens its fibers, and forces it to work on an ineffective portion of its length-tension curve.

This explanation is supported by a study by Rousos et al<sup>16</sup> in which subjects breathing at FRC plus one-half of their inspiratory capacity generated an average mouth pressure of only  $-27$  cm H<sub>2</sub>O breath after breath compared with  $-80$  cm H<sub>2</sub>O in subjects inspiring at a normal FRC.

Sharp and associates<sup>17</sup> studied 17 patients with COPD, seven of whom reported that their dyspnea was reduced when they leaned forward while sitting or when they lay down. All seven patients had paradoxical inward abdominal motion in the standing and erect seating postures, suggesting diaphragmatic dysfunction; this disappeared when they assumed their preferred position. These seven patients had significantly greater lung volumes than the other ten patients. They also used their accessory muscles more than the other patients while standing or sitting erect and also generated a smaller P<sub>di</sub>. This suggested that extremely hyperinflated individuals relieve dyspnea through postures that enhance function of the diaphragm by improving its length-tension relationship.

A correlation between diaphragmatic dysfunction and the degree of airflow obstruction is suggested by the finding of Sharp et al<sup>18</sup> that asynchronous breathing is common in patients with severe COPD. Many of these patients show minimal abdominal movement coincident with outward rib cage motion during inspiration; a small number of them even manifest complete asynchrony of abdomen and rib cage. Braun and Rochester<sup>19</sup> measured maximum inspiratory and expiratory mouth pressure (P<sub>I<sub>max</sub></sub> and P<sub>E<sub>max</sub></sub>) in normal subjects and patients with moderate and severe COPD. They found that P<sub>I<sub>max</sub></sub> was reduced in both the moderately and the severely obstructed patients, who presumably had inspiratory muscle weakness; patients who could develop a P<sub>I<sub>max</sub></sub> of greater than  $-50$  cm H<sub>2</sub>O generally had normal arterial carbon dioxide (CO<sub>2</sub>) levels, while patients whose P<sub>I<sub>max</sub></sub> was less retained CO<sub>2</sub>.

#### ASTHMA

Sternomastoid and intercostal muscle retraction frequently are observed in patients during severe asthma attacks. An explanation for why inspiratory muscles would be called upon in a disease that affects expiration was sought by Martin and co-workers,<sup>20</sup> who studied the lung and chest wall mechanics of subjects in whom bronchoconstriction was induced by progressively larger doses of inhaled histamine. An increase in airflow resistance was associated with a linear increase in FRC up to 75 percent of TLC in these subjects. At each level of hyperinflation the most positive expiratory pleu-

ral pressures recorded during spontaneous breathing were less than the predicted chest wall relaxation pressure, suggesting persistent inspiratory muscle activity during expiration. The investigators concluded that this activity resulted in a chest wall configuration that decreased airway resistance and optimized the function of the diaphragm.

#### CHEST WALL DEFORMITY

Decreased chest wall compliance is found in ankylosing spondylitis, kyphoscoliosis, and other processes that distort the normal configuration of the chest wall. The chests of patients with ankylosing spondylitis also are held in an inspiratory position throughout the respiratory cycle; these patients make more use of their abdominal muscles than do other individuals, perhaps because their intercostal and accessory muscles fatigue more readily. The diaphragm functions normally in patients with ankylosing spondylitis, and they can maintain normal lung volumes, MVV, and arterial blood gases unless their abdominal muscles cannot function properly, as might occur after abdominal surgery. By contrast, kyphoscoliosis is characterized by small lung volumes, especially the expiratory reserve volume (ERV) and FRC; a low MVV; and hypoxemia and hypercapnia in many patients. Although the contribution of respiratory muscle dysfunction to the CO<sub>2</sub> retention of patients with severe kyphoscoliosis had not been quantitated, their hypercapnea might be attendant in part to fatigue related to the stiff chest wall.

#### OBESITY

Massive obesity also causes a decrease in chest wall compliance that is associated with a reduced ERV, FRC, and vital capacity (VC); a low MVV; hypoxemia; and hypercapnea in certain patients with the obesity-hypoventilation syndrome. Rochester and Enson<sup>21</sup> have noted that VC and MVV are significantly lower in patients with the obesity-hypoventilation syndrome than in simple obesity, either because they have greater mass loading of the respiratory muscles or their muscles cannot handle a ventilatory load. To test this latter hypothesis, Arora and Rochester<sup>22</sup> measured MVV and P<sub>I<sub>max</sub></sub> and P<sub>E<sub>max</sub></sub> in obese patients with and without CO<sub>2</sub> retention and expressed them as percent predicted; respiratory muscle strength was calculated as  $P_{I_{max}}\% + P_{E_{max}}\%/2$ . These parameters were reduced more in the hypercapneic patients than in those without CO<sub>2</sub> retention, suggesting that impaired strength and endurance of the respiratory muscles is characteristic of the obesity-hypoventilation syndrome.

Respiratory muscle dysfunction also may con-

tribute to the sleep apnea disorders that seem to cause chronic respiratory failure in the obese. Strohl and co-workers<sup>23</sup> have described an obese family in whom an airway obstruction was related to relative inactivity of the genioglossus muscle. Muller and colleagues<sup>24</sup> recently observed in normal subjects and patients with cystic fibrosis that arterial oxyhemoglobin desaturation was correlated with recurrent periods of REM sleep; these periods were associated with a loss of intercostal and diaphragmatic EMG activity and a decrease in the end-tidal position of the rib cage and abdomen that resulted in a reduced FRC. The investigators speculated that this reduction in FRC might result in airway closure in dependent lung regions that might contribute to the hypoxemia of certain patients with massive obesity.

#### GENERALIZED NEUROMUSCULAR DISORDERS

Respiratory failure has been reported in disorders that involve the anterior horn cell, such as poliomyelitis and amyotrophic lateral sclerosis; peripheral nerve disorders, such as Guillain-Barré syndrome and peripheral neuropathy; motor end-plate disorders, such as myasthenia gravis, tetanus, and *Clostridium botulinum* infection; and muscle disorders, such as polymyositis, muscular dystrophy, and trichinosis. Also documented is respiratory failure due to certain drugs, such as the polymyxins and aminoglycosides in large amounts, and in severe hypophosphatemia. Since most of these disorders cause diffuse muscle weakness, it is difficult

to assess which muscles are most affected. Nevertheless, diaphragmatic dysfunction has been observed with many of the conditions, and it may contribute to characteristic pulmonary function abnormalities, including a decrease in  $P_{I_{max}}$ , VC, and MVV.

#### CERVICAL CORD INJURY

Respiratory muscle dysfunction should be anticipated in patients suffering injuries to the cervical cord. The lower intercostals and abdominal muscles are affected in all such patients, although the accessory muscles and upper intercostals remain active; the accessory muscles were responsible for a minimum of 22 percent of ventilation in eight quadriplegic individuals studied by McKinley and colleagues.<sup>25</sup> The diaphragm also continues to function unless the phrenic nerves (cord level C<sub>3</sub>-C<sub>5</sub>) are impaired, but abdominal wall laxness interferes with the normal inflating action of the diaphragm on the rib cage. Respiratory mechanics also are interfered with by paradoxical inward motion of the upper thorax during upper intercostal contraction in inspiration (Fig 3). Because of these abnormalities, quadriplegic patients generally are most comfortable while supine, since the abdominal contents elevate the diaphragm into the chest and lengthen its fibers in this position. If they must sit up, these patients may be aided by abdominal binding.

#### UNILATERAL DIAPHRAGMATIC PARALYSIS

Unilateral diaphragmatic paralysis most often

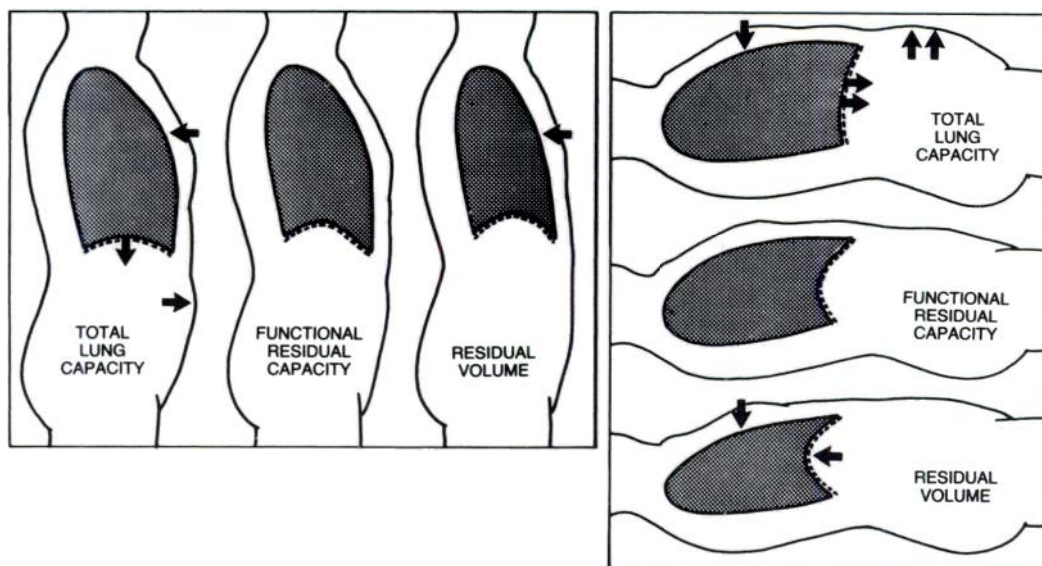


FIGURE 3. Diaphragmatic function is maintained in quadriplegics with cervical cord injury, but these patients lack lower intercostal and abdominal muscle activity. Inspiration to total lung capacity, therefore, is complicated by paradoxical inward motion of the upper thorax, whereas normal residual volume cannot be achieved in expiration due to abdominal muscle dysfunction. Quadriplegic patients are more comfortable in the supine position, where the abdominal viscera push up on the diaphragm and enhance its activity.

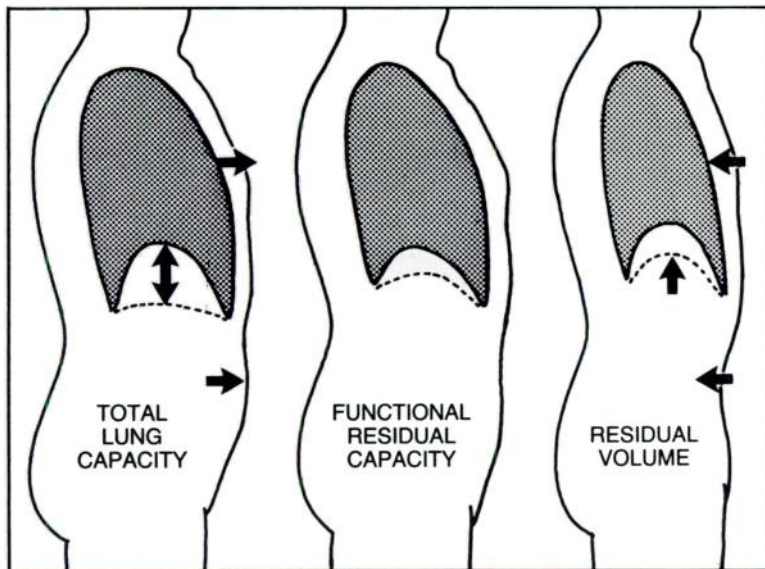


FIGURE 4. Unilateral diaphragmatic paralysis causes paradoxical upward displacement of the involved hemidiaphragm during inspiration to total lung capacity. The paralyzed hemidiaphragm usually rides higher in the chest at functional residual capacity.

results from involvement of the phrenic nerve by primary or metastatic tumor. It also may result from trauma, surgery, neurologic disease, and infection, as well as unknown causes. Few patients report symptoms referable to unilateral diaphragmatic paralysis, and most cases are picked up on routine chest x-ray examination. Although unilateral diaphragmatic paralysis usually induces immobility of the ipsilateral anterior abdominal wall and a lag in the upward and outward movement of the costal margin in deep inspiration, this frequently is obscured by the proper function of the contralateral hemidiaphragm (Fig 4). Paradoxical upward

movement of the paralyzed diaphragm usually can be detected during a "sniff" test under fluoroscopy, however.

Arborelius and co-workers<sup>26</sup> studied regional lung function by means of radioactive <sup>133</sup>Xe scans in patients with unilateral diaphragmatic paralysis and showed a reduction in perfusion of 19 percent, in ventilation of 20 percent, and in volume of 7 percent in the affected side. Arterial Po<sub>2</sub> was reduced from 83 mm Hg to 75 mm Hg when these patients went from an upright to a supine position and fell slightly more when they laid on the affected side. Although this relatively small drop in

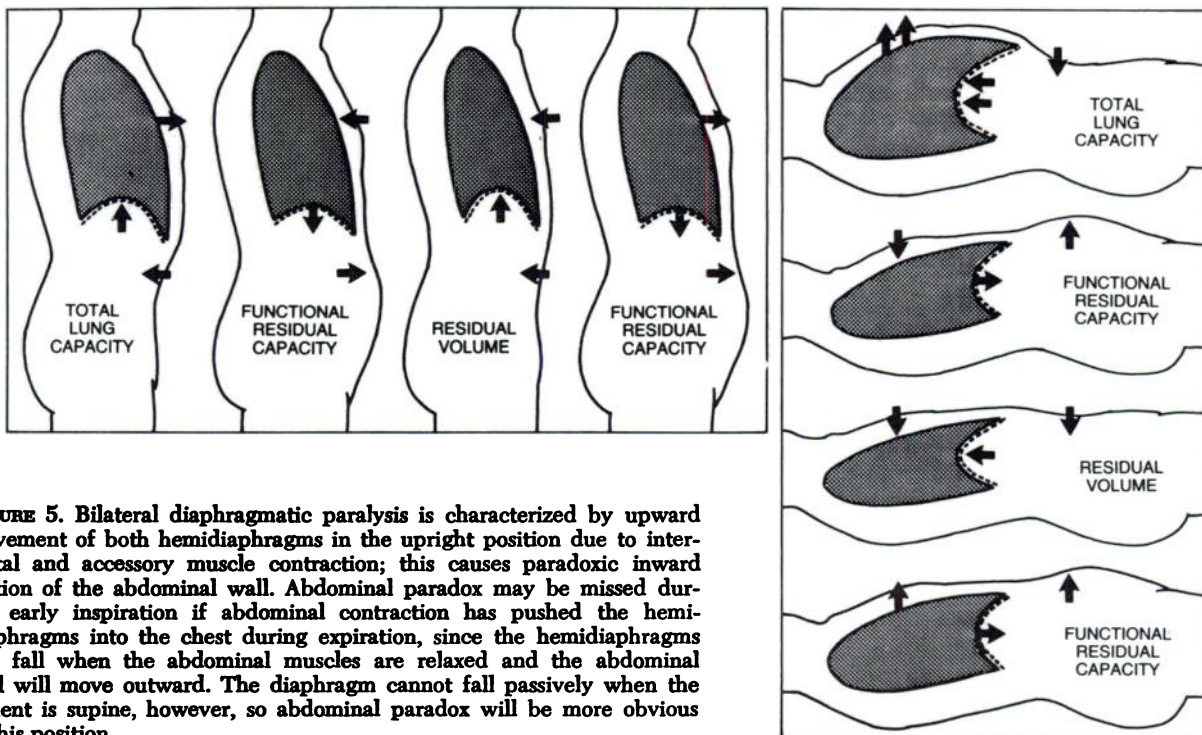


FIGURE 5. Bilateral diaphragmatic paralysis is characterized by upward movement of both hemidiaphragms in the upright position due to intercostal and accessory muscle contraction; this causes paradoxical inward motion of the abdominal wall. Abdominal paradox may be missed during early inspiration if abdominal contraction has pushed the hemidiaphragms into the chest during expiration, since the hemidiaphragms will fall when the abdominal muscles are relaxed and the abdominal wall will move outward. The diaphragm cannot fall passively when the patient is supine, however, so abdominal paradox will be more obvious in this position.

Po<sub>2</sub> should not be clinically significant in most adults, respiratory failure has been described in infants with unilateral diaphragmatic paralysis as a sequelae of cardiac surgery, several of whom required surgical plication of the hemidiaphragm.

#### BILATERAL DIAPHRAGMATIC PARALYSIS

If one breathes only with the intercostal and accessory muscles, as is the case in patients with bilateral diaphragmatic paralysis, Pdi equals zero and Pab equals Ppl. As a result, with each inspiration, the decrease in Ppl not only expands the lung, but also sucks in the abdomen and the diaphragm (Fig 5). This paradoxical inward motion of the abdomen may not be apparent in the upright position if the abdominal muscles have contracted and elevated the diaphragm to aid expiration, for in this situation the diaphragm may descend passively in early inspiration as the previously-tensed abdominal muscles relax. Nevertheless, paradox should be apparent if the patient is supine because in this position the diaphragm cannot fall passively. The upward motion of the diaphragm in inspiration is expiratory in direction and opposes lung inflation. Thus, breathing with only the intercostal and accessory muscles is quite inefficient, and the work of breathing is inordinately high.

Bilateral diaphragmatic paralysis may occur either in association with generalized neuromuscular disease, as noted above, or as an isolated phenomenon. Patients with this disorder characteristically complain of orthopnea, disturbed sleep, daytime fatigue, and morning headaches, and they may be misdiagnosed as having left ventricular dysfunction, idiopathic hypoventilation, or sleep apnea. According to McCredie and colleagues,<sup>27</sup> bilateral diaphragmatic paralysis causes hypoxemia, hypercapnea, and decreases in VC, RV, and TLC that are accentuated in the supine position, when the abdominal viscera push the inactive diaphragm into the chest and limit the effectiveness of the other respiratory muscles, and when paradoxical inward movement of the abdomen is most apparent during inspiration. Bilateral diaphragmatic paralysis should be suspected in the presence of such paradox; it is suggested by fluoroscopy and confirmed by transdiaphragmatic pressure recordings.

Newsom-Davis and associates<sup>28</sup> studied eight patients with bilateral diaphragmatic paralysis and neuromuscular disease, all of whom demonstrated paradoxical abdominal motion during inspiration in the supine position. None of these patients could generate a Pdi greater than 6 cm H<sub>2</sub>O, and all had an elevated arterial PCO<sub>2</sub> that climbed during sleep but could be normalized by voluntary hyperventil-

ation. These authors speculated that their patients' blood gases worsened during sleep because they had a central insensitivity to hypoxia and hypercapnea that could only be overcome by the stimulus of wakefulness. Another explanation recently was provided by Skatrud and colleagues<sup>29</sup> who described a patient with muscular dystrophy, bilateral diaphragmatic paralysis, chronic CO<sub>2</sub> retention, and hypersomnolence. Awake hypoventilation was associated with a weak inspiratory effort that could be overcome by forced hyperventilation only until the patient tired; during sleep, his hypoventilation worsened during REM-induced intercostal and accessory muscle inhibition.

Treatment of patients with bilateral diaphragmatic paralysis involves attention to underlying disorders, if present, and relief of respiratory failure. Although some patients require continuous ventilatory support with positive or negative pressure respirators, others can be assisted, particularly at night, with such devices as the rocking bed, which imparts motion to the abdominal contents in time with the respiratory cycle; the chest cuirass, a negative pressure device that is affixed to the front of the chest; and the pneumobelt, which intermittently compresses the abdomen and pushes the diaphragm into the thorax. Diaphragmatic pacing by radiofrequency stimulation has been used in patients with idiopathic hypoventilation and cervical cord injuries, in addition to those with COPD, kyphoscoliosis, and the obesity-hypoventilation syndrome; it may be suitable in some patients with bilateral diaphragmatic paralysis and intact phrenic nerves.

#### RESPIRATORY MUSCLE TRAINING

Patients with bilateral diaphragmatic paralysis and other disorders characterized by respiratory muscle fatigue also may be helped by respiratory muscle training, a technique introduced by Leith and Bradley.<sup>30</sup> Their study involved four "strength trainers" who performed repeated static maximum inspiratory and expiratory pressure maneuvers while breathing through an obstructed mouthpiece for fifteen minutes a day over a three-month period. At the same time, four "endurance trainers" performed repeated runs of normocapneic hyperpnea to exhaustion while breathing through a rebreathing system that allowed them to try to meet a preset maximum sustainable ventilatory capacity (MSVC) based on their MVV. The strength trainers increased their peak inspiratory and expiratory mouth pressure by 55 percent, but did not change their lung volumes or MSVC. By contrast, the endurance trainers increased their MSVC from 81 to 96 percent without affecting peak mouth pressures. The



improvement in strength and endurance fell off as the subjects stopped exercising; their values returned to pre-study levels after one year.

The improvement in respiratory muscle function after endurance training in these and other patients suggests that such training may help mechanically-ventilated patients, especially those in whom asynchronous breathing indicates respiratory muscle fatigue. In the study cited above, Andersen et al<sup>13</sup> found that the thoracoabdominal discoordination and diaphragmatic fatigue in 18 patients during conventional weaning from continuous mechanical ventilation was abolished by weaning with intermittent mandatory ventilation (IMV), in which ventilator breaths are augmented by the patient's spontaneous breathing. Downs and co-workers<sup>31</sup> believe that IMV reinforces the proper sequence of muscular efforts necessary for effective ventilation while providing respiratory muscle exercise.

At the same time, some patients who are ventilated at a low IMV rate complain of extreme dyspnea. Rochester and associates<sup>32</sup> have demonstrated that diaphragmatic contraction can be reduced to zero in patients with severe COPD who are treated with negative pressure ventilation; coincident with this is a loss of dyspnea independent of improvement in arterial blood gases. These investigators feel that resting overburdened muscles with negative pressure breathing may aid patients with chronic respiratory failure. It appears that respiratory muscle relaxation is preferable to aggressive training in certain persons, and that not all patients with respiratory failure profit from the increased work of breathing associated with IMV.

#### REFERENCES

- 1 Derenne J-Ph, Macklem PT, Roussos Ch. The respiratory muscles: Mechanics, control and pathophysiology, Parts I, II, and III. *Am Rev Respir Dis* 1978; 118:119-33, 373-90, 581-601
- 2 Goldman MD, Mead J. Mechanical interaction between the diaphragm and rib cage. *J Appl Physiol* 1973; 35: 197-204
- 3 Macklem PT, Gross D, Grassino A, Roussos C. Partitioning of inspiratory pressure swings between diaphragm and intercostal/accessory muscles. *J Appl Physiol* 1978; 44:200-08.
- 4 Danon J, Druz WS, Goldberg NB, Sharp JT. Function of the isolated paced diaphragm and the cervical accessory muscles in C<sub>1</sub> quadriplegics. *Am Rev Respir Dis* 1979; 119:909-19
- 5 Rochester DF, Braun NMT. The respiratory muscles. *Basics of RD* 1978; 6:1-6
- 6 Rochester DF, Bettini G. Diaphragmatic blood flow and energy expenditure in the dog. *J Clin Invest* 1976; 57:661-72
- 7 Robertson CH, Jr., Foster GH, Johnson RL, Jr. The relationship of respiratory failure to the oxygen consumption of, lactate production by, and distribution of blood among respiratory muscles during increasing inspiratory resistance. *J Clin Invest* 1977; 59:31-42
- 8 Aubier M, Viires N, Sillye G, Roussos Ch. Effect of mechanical ventilation on lactic acidosis in shock. *Am Rev Respir Dis* 1980; 121:311 (abstract)
- 9 Tenney SN, Reese RE. The ability to sustain great breathing efforts. *Respir Physiol* 1968; 5:187-201
- 10 Roussos CS, Macklem PT. Diaphragmatic fatigue in man. *J Appl Physiol* 1977; 43:189-97
- 11 Gross D, Grassino A, Ross WRD, Macklem PT. Electromyogram pattern of diaphragmatic fatigue. *J Appl Physiol* 1979; 46:1-7
- 12 Gross D, Riley E, Grassino A, Ladd H, Macklem PT. Influence of resistive training on respiratory muscle strength and endurance in quadriplegia. *Am Rev Respir Dis* 1978; 117:343 (abstract)
- 13 Muller N, Gulston G, Cade D, Whitton J, Bryan MH, Bryan AC. Respiratory muscle fatigue in infants. *Clin Res* 1977; 25:714A (abstract)
- 14 Andersen JB, Kann T, Rasmussen JP, Howardy P, Mitchell J. Respiratory thoraco-abdominal coordination and muscle fatigue in acute respiratory failure. *Am Rev Respir Dis* 1978; 117:89 (abstract)
- 15 Roussos CS, Macklem PT. Response of the respiratory muscles to fatiguing loads. *Am Rev Respir Dis* 1976; 113:200 (abstract)
- 16 Roussos CS, Fixley MS, Gross D, Macklem PT. Respiratory muscle fatigue in man at FRC and higher lung volumes. *The Physiologist* 1976; 19:345 (abstract)
- 17 Sharp JT, Druz WS, Moisan T, Foster J, Machnach W. Postural relief of dyspnea in severe chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1980; 122:201-13
- 18 Sharp JT, Goldberg NB, Druz WS, Fishman HC, Danon J. Thoracoabdominal motion in chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1977; 115: 47-56
- 19 Braun NMT, Rochester DF. Respiratory muscle function in chronic obstructive pulmonary disease (COPD). *Am Rev Respir Dis* 1977; 115:91 (abstract)
- 20 Martin J, Powell E, Shore S, Emrich J, Engel LA. The role of respiratory muscles in the hyperinflation of bronchial asthma. *Am Rev Respir Dis* 1980; 121:441-47
- 21 Rochester DF, Enson Y. Current concepts in the pathogenesis of the obesity-hypoventilation syndrome: Mechanical and circulatory factors. *Am J Med* 1974; 57: 402-20
- 22 Arora NS, Rochester DF. Respiratory muscle function in obesity and obesity-hypoventilation syndrome. *Clin Res* 1979; 27:394A (abstract)
- 23 Strohl KP, Saunders NA, Feldman NT, Hallett M. Obstructive sleep apnea in family members. *N Engl J Med* 1978; 299:969-73
- 24 Muller NL, Francis PW, Gurwitz D, Levison H, Bryan AC. Mechanism of hemoglobin desaturation during rapid-eye-movement sleep in normal subjects and in patients with cystic fibrosis. *Am Rev Respir Dis* 1980; 121:463-69
- 25 McKinley AC, Auchincloss JH, Jr., Gilbert R, Nicholas JJ. Pulmonary function, ventilatory control, and respiratory complications in quadriplegic subjects. *Am Rev Respir Dis* 1969; 100:526-32
- 26 Arborelius M, Jr., Lilja B, Senyk J. Regional and total lung function studies in patients with hemidiaphragmatic paralysis. *Respiratory* 1975; 32:253-64
- 27 McCredie M, Lovejoy FW Jr, Kaltreider NL. Pulmonary function in diaphragmatic paralysis. *Thorax* 1962;

- 17:213-17
- 28 Newsom-Davis J, Goldman M, Loh L, Casson M. Diaphragm function and alveolar hypoventilation. *Quart J Med* 1976; 45:87-100
- 29 Skatrud J, Iber C, McHugh W, Rasmussen H, Nichols D. Determinants of hypoventilation during wakefulness and sleep in diaphragmatic paralysis. *Am Rev Respir Dis* 1980; 121:587-93
- 30 Leith DE, Bradley M. Ventilatory muscle strength and endurance training. *J Appl Physiol* 1976; 41:508-16
- 31 Downs JB, Klein EF, Jr., Desautels D, Modell JH, Kirby RR. Intermittent mandatory ventilation: A new approach to weaning patients from mechanical ventilators. *Chest* 1973; 64:331-35
- 32 Rochester DF, Braun NMT, Laine S. Diaphragmatic energy expenditure in chronic respiratory failure: The effect of assisted ventilation with body respirators. *Am J Med* 1977; 63:223-32



## Respiratory muscle function in health and disease

JM Luce and BH Culver  
*Chest* 1982;81;82-90  
DOI 10.1378/chest.81.1.82

This information is current as of November 12, 2007

<b>Updated Information &amp; Services</b>	Updated information and services, including high-resolution figures, can be found at: <a href="http://chestjournal.org">http://chestjournal.org</a>
<b>Citations</b>	This article has been cited by 2 HighWire-hosted articles: <a href="http://chestjournal.org">http://chestjournal.org</a>
<b>Permissions &amp; Licensing</b>	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: <a href="http://chestjournal.org/misc/reprints.shtml">http://chestjournal.org/misc/reprints.shtml</a>
<b>Reprints</b>	Information about ordering reprints can be found online: <a href="http://chestjournal.org/misc/reprints.shtml">http://chestjournal.org/misc/reprints.shtml</a>
<b>Email alerting service</b>	Receive free email alerts when new articles cite this article sign up in the box at the top right corner of the online article.
<b>Images in PowerPoint format</b>	Figures that appear in CHEST articles can be downloaded for teaching purposes in PowerPoint slide format. See any online article figure for directions.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S<sup>®</sup>