Authors:

Christopher Winslow, MD, FCCP
Julia Rozovsky, MD

Affiliations:

From the Division of Pulmonary and Critical Care Medicine, Evanston Hospital, The Feinberg Medical School of Northwestern University, Evanston, Illinois, and the Rehabilitation Institute of Chicago, Chicago, Illinois (CW); and Division of Pulmonary and Critical Care Medicine, The Feinberg Medical School of Northwestern University, Chicago, Illinois (JR).

Correspondence:

All correspondence and requests for reprints should be addressed to Christopher Winslow, MD, Evanston Hospital, Suite 5303, 2650 Ridge Avenue, Evanston, IL 60201.

0894-9115/03/8210-0803/0
American Journal of Physical Medicine & Rehabilitation

DOI: 10.1097/01.PHM.0000078184.08835.01

Literature Review

Effect of Spinal Cord Injury on the Respiratory System

ABSTRACT

Winslow C, Rozovsky J: Effect of spinal cord injury on the respiratory system. *Am J Phys Med Rehabil* 2003;82:803–814.

There are >200,000 persons living with a spinal cord injury in the United States, with approximately 10,000 new cases of traumatic injury per year. Advances in the care of these patients have significantly reduced acute and long-term mortality rates, although life expectancy remains decreased. This article will review the alterations in respiratory mechanics resulting from a spinal cord injury and will examine the contribution of respiratory complications to morbidity and mortality associated with various types of spinal cord injury.

Key Words: Spinal Cord Injury, Respiratory Muscles, Pneumonia, Respiratory Failure, Thromboembolism

A spinal cord injury (SCI) is among the most catastrophic injuries that a person can experience. The personal and societal effect of a significant SCI is profound because it confers lifelong disability on persons who are typically young adults. Fifty-four percent of all SCIs occur in individuals between 16 and 30 yr of age, with 75% of injuries occurring in those <45 yr old.[1]

The economic effect of SCI is considerable. Presently, there are >200,000 persons living with an SCI in the United States, with approximately 10,000 new cases of traumatic injury per year.[2,3] Acute SCI is the second most expensive condition treated in United States hospitals, with mean acute care hospital charges of $53,000, exceeded only by the respiratory distress syndrome of infants, with mean charges of $68,000.[4] Furthermore, it has been estimated that in the United States alone, $8 billion is spent annually in medical charges, household assistance, environmental modifications, and other support services attributable to SCI.[5]

Respiratory complications are frequent in persons with SCI and contribute significantly to associated morbidity, mortality, and economic burden.[4,6–10] In

this article, we will examine the alterations in respiratory mechanics resulting from an SCI, explore the contribution of respiratory complications to mortality in SCI, and review the data regarding respiratory complications in persons with SCI.

Frequency and Classification of SCI. The spinal level of neurologic injury, the type of neurologic deficit, and the duration of injury are all factors that determine the degree of respiratory compromise. In general, functional impairment worsens as the level of injury is more cephalad. In addition, a complete SCI, defined as the absence of motor or sensory function below the injury (classified as American Spinal Injury Association [ASIA] score A), results in a greater functional impairment than incomplete injuries (ASIA scores B–D) (Fig. 1) in which some residual function below the level of injury remains. An injury to the spine without a resulting neurologic deficit is classified as an ASIA E injury. Figure 1 stratifies 17,243 persons with neurologic deficits from a cervical, thoracic, or lumbar SCI entered into the National Spinal Cord Injury Database (NSCID) from 1993 through 1998 by admission level of injury and ASIA impairment score. Injuries involving the cervical spinal cord are very common, with levels of injury from C4 through C7 accounting for 8,132 (47%) of all injuries for this cohort. Given the frequency of cervical levels of injury, nearly half of all patients with a neurologic deficit from an SCI will experience significant respiratory compromise.

Respiratory Mechanics After SCI. The study of the alterations in respiratory mechanics resulting from SCI has furthered the understanding of normal respiratory muscle function. The main muscles known to contribute to respiration are shown in Figure 2. During quiet breathing, coordination of the diaphragm, the intercostals muscles, and the scalene muscles allow the chest

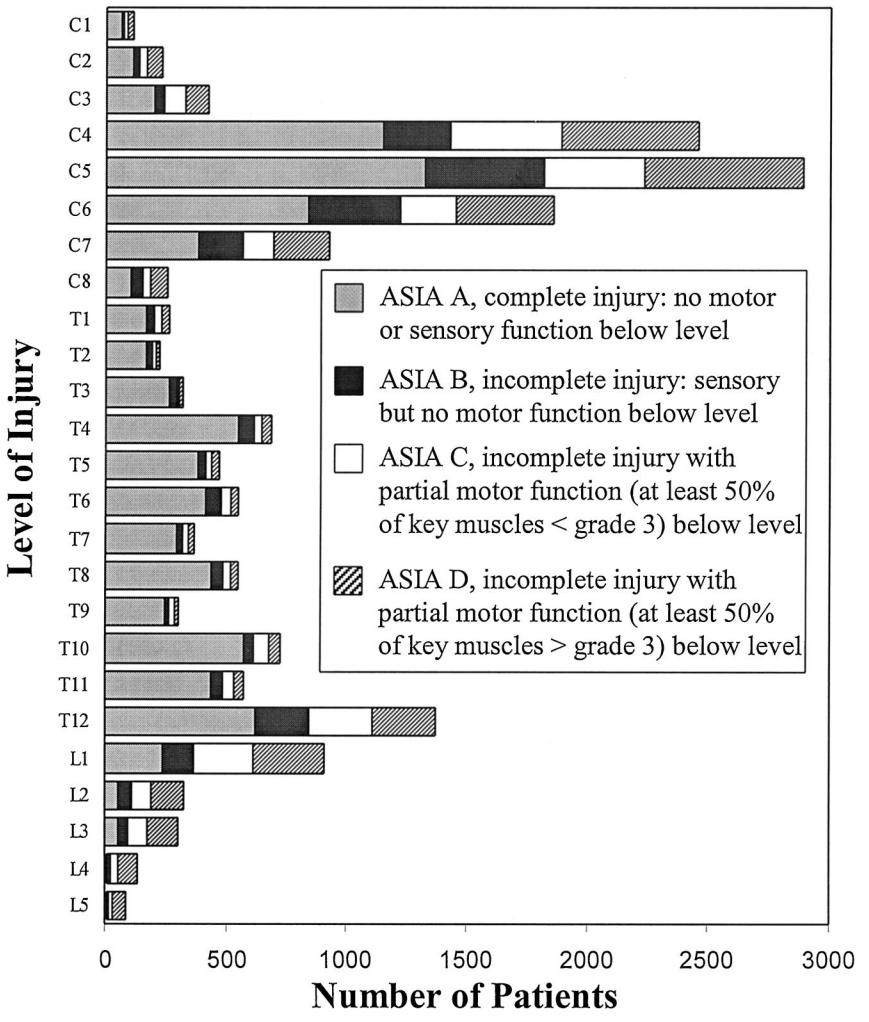

Figure 1: Level of injury and American Spinal Injury Association (*ASIA*) impairment scale at hospital admission. National Spinal Cord Injury Database, 1993–1998.

wall to move with a single degree of freedom along its relaxation characteristic.[11–13] The relaxation characteristic is a line that results from a plot of abdominal *vs.* rib cage movement during passive lung inflation. Breathing along this line represents optimal efficiency of the respiratory system. The study of subjects with selected types of SCI has allowed us to examine the effect of components of this respiratory system in a way not permitted in the intact subject and to demonstrate that each component muscle group acting in the absence of the others induces marked conformational changes of the chest wall that shifts the operational characteristic away from the relaxation line (Fig. 3). In general, the isolated contraction of the diaphragm, in the absence of intercostal and scalene muscle contraction, expands the lower rib cage but collapses the upper rib cage. Conversely, contraction of the strap muscles of the neck and the intercostals in the absence of diaphragmatic contraction expands the upper rib cage with deleterious effects on the lower cage. The next section will examine the contribution of each muscle to the respiratory pump both in the intact state and then after a significant SCI. The innervations of the main muscles of respiration are shown in Table 1.

Diaphragm. The diaphragm is the most important muscle of inspiration. The radius (r) of curvature of the diaphragm is an important determinant of force development because

transdiaphragmatic pressure (P_{di}) relates to the tangential tension (T_{di}) developed by the diaphragm according to LaPlace's law: $P_{di} = T_{di}/r^2$.

Thus, a more tightly curved diaphragm results in a smaller r and more effective translation of diaphragmatic tension (T_{di}) to P_{di}.[14] Because of its curvature, the diaphragm abuts the lower ribcage forming the zone of apposition. This circumferential zone is greater at lower lung volumes and decreases as the diaphragm moves caudally during inspiration.[15] In the intact person, contraction of the diaphragm increases negative intrathoracic pressure by increasing thoracic volume with a compensatory displacement of the abdominal contents.

In a study of two C1 tetraplegic subjects with diaphragmatic pacers and denervated scalene and parasternal intercostal muscles, Danon et al.[13] showed that the anteroposterior and transverse diameters of the upper rib cage are reduced with diaphragmatic stimulation. The absence of scalene and intercostal muscle function in these subjects caused an inward movement of the upper rib cage during inspiration when negative pleural pressure was generated by electrically stimulated diaphragmatic contraction. This paradoxical motion of the upper rib cage diminishes the effectiveness of the inspiratory effort.

In the uninjured person, as the diaphragm moves caudally during inspiration, it presses on the abdominal contents that act as a fulcrum and transmit "appositional" forces laterally to expand the lower rib cage.[16] The magnitude of the appositional force depends on (1) the rise in abdominal pressure and (2) the area of the zone of apposition.[12] Studies of thoracoabdominal movements in persons with tetraplegia demonstrate that increases in the anteroposterior and transverse diameters of the abdomen are greater than the changes in comparable dimensions of the lower rib cage.[12,13] An examination of the effect of posture on thoracoabdominal function can help explain this observation.

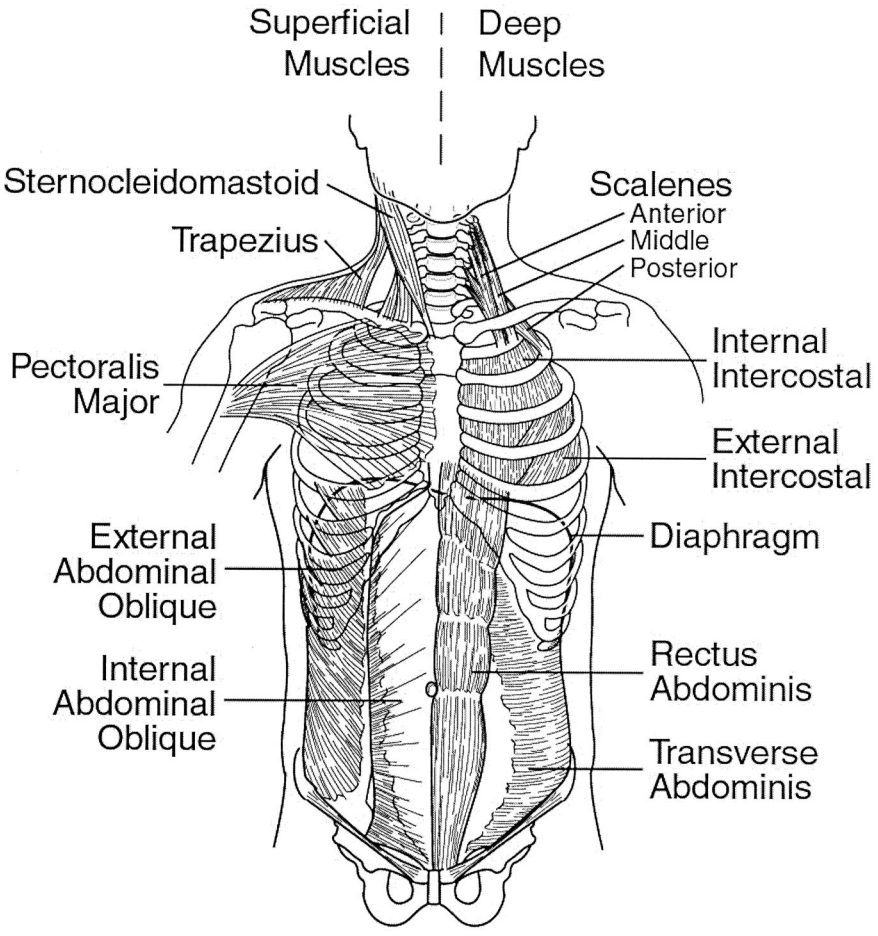

Figure 2: Main muscles of respiration.

In tetraplegic persons, tidal volume (V_T), P_{di}, and airway occlusion pressures are greater in the supine compared with the upright posture.[13] When persons with tetraplegia assume an upright posture, lung volume increases as the abdominal contents are shifted caudally due to gravitational forces and the lack of abdominal muscle tone. As shown in Figure 4, this caudal shift (1) shortens diaphragmatic muscle fiber length reducing P_{di}, (2) decreases the zone of apposition, and (3) increases r. All contribute to a reduction in T_{di}. As a result, V_T is lower in the upright compared with the supine posture. Therefore, in the upright posture, appositional forces are reduced in persons with tetraplegia both because of an inability to raise abdominal pressure due to a lack of abdominal muscle tone and a decrease in the zone of apposition. The relationship between resting lung volume, V_T, and posture is shown in Figure 5.

For subjects with tetraplegia, V_T increases about 16% with the addition of an abdominal binder, primarily due to an increase in both anteroposterior and lateral rib cage excursion during inspiration.[13] By supporting the abdominal wall, binders shift the abdominal contents cephalad. For persons with some residual function of the diaphragm, this cephalad shift restores the fulcrum effect of the abdominal contents facilitating expansion of the lower rib cage. Abdominal binders also allow the lung to operate at a lower functional residual capacity, perhaps placing the remaining functional inspiratory muscles in a position of greater mechanical advantage.

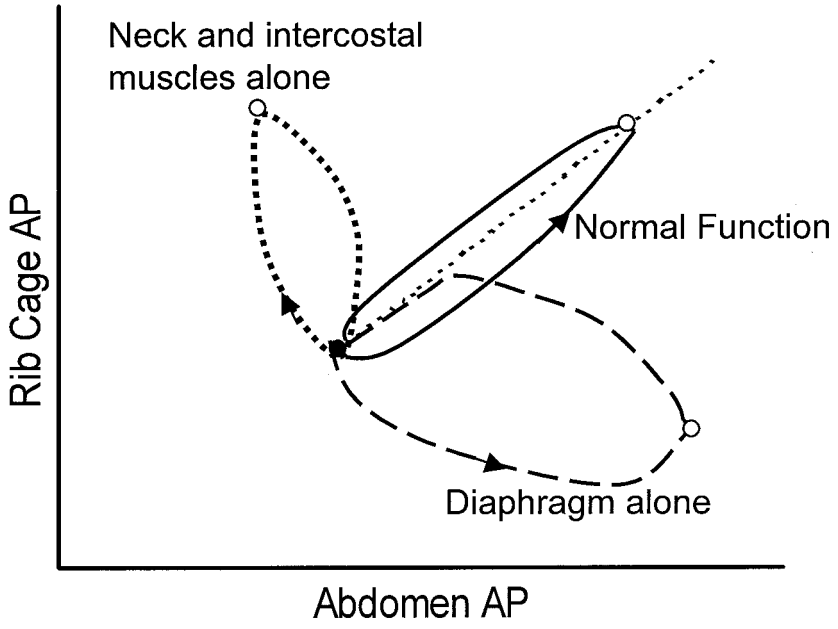

Figure 3: Konno-Mead plot of abdominal motion (*abscissa*) *vs.* rib cage motion (*ordinate*). *AP*, anteroposterior. *Straight dotted line* indicates relaxation characteristic; *solid circle* indicates end-exhalation; *open circle* indicates end-inhalation; *arrows* indicate the direction of motion. Adapted with permission.[13]

Intercostal Muscles. Data concerning intercostal muscle function in tetraplegia reveal conflicting findings. Intercostal muscle activity, as measured by surface electromyographic (EMG) recordings, has been reported as absent[17–19] in phase with other inspiratory muscles[17] or continuous throughout the respiratory cycle, suggesting spasticity.[12,18,19] These patterns of activity do not correlate with level of SCI, duration of injury, or any measure of rib cage deformation. One possible explanation may be that none of these studies present any data regarding neurologic function other than the level of injury. Because SCI can be asymmetric and incomplete injuries are present in about half of those with tetraplegia,[20] differences in patterns of residual innervation between subjects may explain these findings.

In addition to alterations in intercostal muscle function as a result of denervation, the ribcage stiffens considerably in the months after cervical SCI.[17,18,21,22] Although the mechanism of these alterations is unclear, several factors have been proposed as contributory.[18] Chronically diminished thoracic excursions due to inspiratory muscle weakness is believed to induce stiffness of the tendons and ligaments and ankylosis in the joints of the rib cage.[18] Paralysis of the intercostal muscles decreases rib cage excursion by poorly transmitting and distributing the inspiratory forces throughout the rib cage.[19] A seemingly contradictory theory proposes that spasticity of the intercostal muscles causes chest wall stiffness.[12] Lastly, a decrease in the compliance of the lungs from atelectasis and an increase in compliance of the abdomen due to the lack of muscle tone in the abdominal wall further contribute to thoracic stiffness.[18]

Muscles of the Neck and Shoulder Girdle. Some muscles of the neck and shoulder girdle can also serve as muscles of respiration. When their extrathoracic insertion point is fixed, contraction of these muscles can exert changes in the shape of the chest, thereby assisting ventilation. Indirect evidence for the role of these muscles in respiration has been suggested by Banzett et al.,[23] who showed that normal subjects could achieve higher minute ventilations with their arms supported on a table, rather than unsupported at their sides. Depending on the level and completeness of the SCI, the function of these muscles may be preserved because they are innervated by cervical nerve roots and the accessory nerve (cranial nerve XI). These muscles include the scalene, the sternocleidomastoids, the trapezii, and the pectoralis major.

Studies of the scalene muscles using needle EMG in normal subjects have demonstrated that they are active during quiet breathing in both the supine and upright posture and, thus, should be considered primary, not accessory, muscles of inspiration.[11,24] Coordinated contraction of these muscles seems to be necessary for the rib cage to achieve a full range

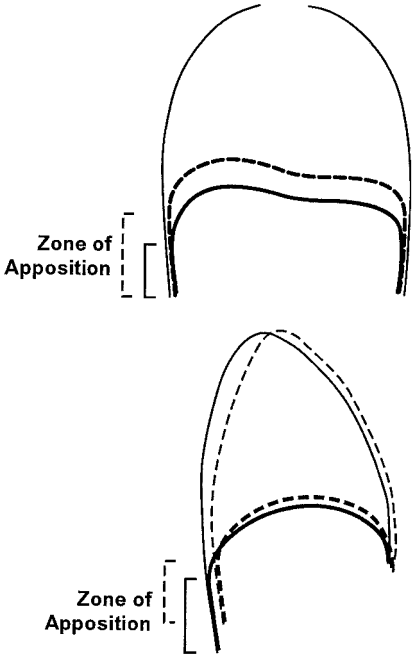

Figure 4: Posteroanterior (*upper panel*) and lateral (*lower panel*) views of the chest. *Dashed lines* represent the chest at functional residual capacity; *solid lines* represent the chest at end-inspiration; *thick lines* indicate the diaphragm. Adapted with permission.[12]

TABLE 1
Muscles of respiration and their innervation

Muscle	Innervation
Inspiratory, primary	
Diaphragm	C3–C5
Intercostals	T1–T11
Scalene	
Anterior	C3–C4
Middle	C5–C6
Posterior	C6–C8
Inspiratory, accessory	
Sternocleidomastoid	C2–C4 and accessory N. (XI)
Trapezius	C1–C4 and accessory N. (XI)
Expiratory	
Rectus abdominus	T6–T12
Transversus abdominus	T2–L1
Internal and external obliques	T6–L1
Pectoralis major	Medial and lateral pectoral N. (C5–T1)

C, cervical; T, thoracic; L, lumbar; N, nerve.

of movement along its relaxation characteristic.[11]

Because the scalenes are innervated by the lower six cervical nerves, some residual function of these muscles is often preserved in an SCI involving the lower cervical cord. Estenne and De Troyer[11,12] demonstrated that the pattern of scalene EMG activity in persons with tetraplegia predicted upper rib cage motion. Subjects with either a lack of phasic inspiratory EMG activity or continuous (spastic) activity were more likely to demonstrate a paradoxical decrease in the anteroposterior diameter of the upper rib cage during inspiration than subjects with phasic inspiratory activity of the scalenes. Their findings confirm the observations of Danon et al.,[13] who also found paradoxical movement of the upper and middle rib cage with isolated contraction of the diaphragm in the absence of scalene and intercostal muscle activity.

During quiet breathing in an uninjured person, the sternocleidomastoid muscle contributes little to respiration. However, as minute ventilation increases, phasic inspiratory EMG activity is found, suggesting its role as an accessory muscle of respiration.[24,25] Because it is innervated by the first and second cervical nerves and the accessory nerve, the function of the sternocleidomastoid muscle is generally preserved in all but the highest levels of cervical SCI. Both the sternocleidomastoids and the trapezii are important muscles of respiration in tetraplegia. With its origins on the clavicle and the sternum and its insertion on the mastoid process, contraction of the sternocleidomastoid can elevate the upper rib cage when the head is held fixed by the upper fibers of the trapezii. In subjects who were either ventilator or diaphragm-pacer dependent, isolated contraction of these muscles markedly increased the anteroposterior diameter of the upper chest with little change in transverse dimension, suggesting that these muscles act as a "pump handle" and elevate the upper rib cage during inspiration.[13,19] In addition, contraction of these muscles in the absence of diaphragmatic function, causes a paradoxical decrease in the anteroposterior diameter of the abdomen and the transverse diameters of both the abdomen and lower rib cage.[13,19] Thus, isolated contraction of these muscles has a positive effect on the upper rib cage and an adverse effect on the lower rib cage and abdomen, just as the converse is true for the diaphragm because isolated diaphragmatic contraction favorably affects the abdomen and lower rib cage and

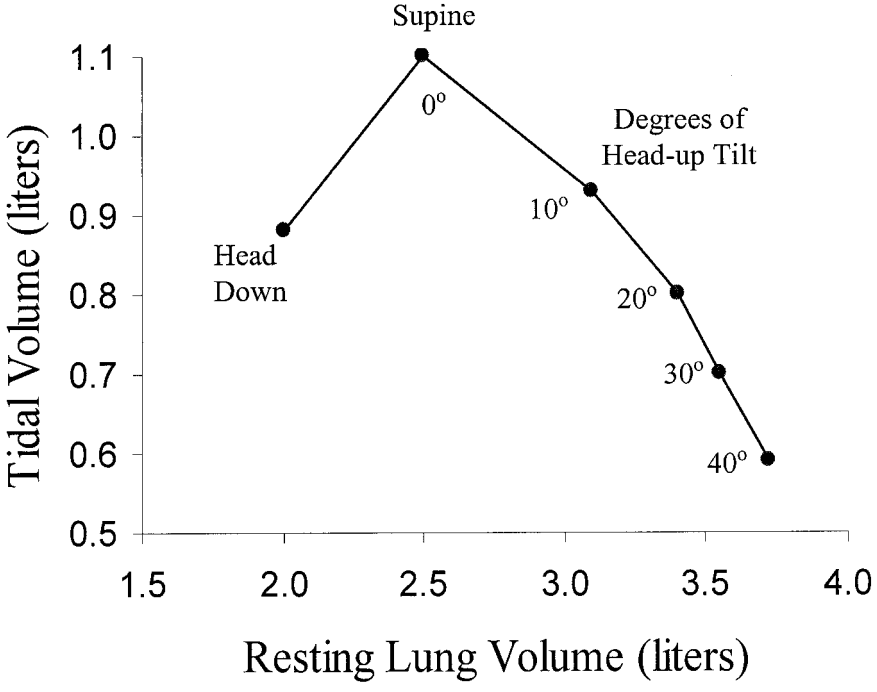

Figure 5: The relationship between lung volume, tidal volume, and posture. Used with permission.[13]

adversely affects motion of the upper rib cage.

The accessory muscles of respiration can also be recruited to assist in exhalation in persons with tetraplegia. The EMG activity of pectoralis muscle has been demonstrated during exhalation[26,27] and cough.[28] In addition, a randomized trial of 12 subjects with levels of injury between C5 and C8 showed that a 6-wk training program using repetitive isometric contraction of the pectoralis muscles resulted in improved exhalatory function as measured by a 47% increase in the expiratory reserve volume compared with controls.[27] This training effect persisted when subjects were retested 10 wk after discontinuing exercise.

Muscles of the Abdomen. During quiet breathing, exhalation is a passive process occurring in the absence of thoracic or abdominal muscle contraction. However, as minute ventilation increases, the abdominal muscles contract during exhalation pulling the chest caudally to facilitate thoracic emptying.[29] Contraction of the abdominal musculature is also essential in the development of the expulsive force needed for an effective cough. With their innervations arising from the thoracic and lumbar nerve roots, abdominal muscle function is absent in persons with complete injuries of the cervical and upper thoracic spinal cord.[12,30]

Pulmonary Function After SCI Spirometry. The nadir in lung function occurs immediately after an SCI with respiratory function, markedly improving during the first year after injury.[31,32] Serial measures of spirometry performed by Ledsome and Sharpe[31] showed improvement in both forced vital capacity (FVC) and 1-sec forced expiratory volume (FEV_1) by 5 wk postinjury, with a more gradual, but significant, improvement between 5 wk and 5 mo. Among 11 subjects with complete injuries between C5 and C6, mean FVC was 1.5 ± 0.49 liters (31.3 ± 6.3% of predicted values) after injury, improving to 2.2 ± 0.36 liters (44.9 ± 7.3% of predicted) at 5 wk postinjury. By 5 mo after injury, mean FVC had improved to 2.8 ± 0.73 liters (57.7 ± 11.5% of predicted). Comparable changes were noted in FEV_1.

Bluechardt et al.[32] extended these observations by performing spirometry every 2 mo from 90–120 days postinjury in 12 subjects with similar levels of injury. Mean FEV_1 improved 0.72 liters (40%, $P \leq 0.001$), although improvements in mean FVC (32.4%, $P \leq 0.07$) and mean maximum voluntary ventilation (16%, $P \leq 0.33$) did not significantly change over this period. The authors noted marked variability in the degree of recovery, with no relationship to level of injury.

Data regarding the long-term (>1 yr) effect of SCI on spirometric measures of lung function show an inverse relationship between FVC and level of injury, with FVC decreasing as the level of injury moves cephalad.[33,34] In general, persons with high tetraplegia (C2–C5) and spontaneous ventilation can have an FVC as high as 50% of their preinjury predicted value, depending on the type of injury.[33–35] For persons with low tetraplegia (C6–C8), the percentage of predicted FVC (FVC%) increases by 9% per vertebral level as the level of injury moves caudally. For thoracic and lumbar levels of injury, each caudal drop in level of injury by one vertebra increases the FVC% by 1%.[35]

The type of the injury also influences the FVC%. For persons with high tetraplegia and an incomplete (ASIA B through D) injury, the FVC% will be about 16% higher than those with complete (ASIA A) injuries. For persons with low tetraplegia (C6-C8), the FVC% will be about 10% higher compared with those with complete injuries. There is no difference in FVC% between incomplete and complete injuries for persons with injuries to the thoracic or lumbar spinal cord.[35]

As in the uninjured population, persons with SCI experience a gradual loss in lung function over time. Presently, there are no data to suggest that persons with SCI experience an accelerated loss of lung function as a result of their injury, although there are no longitudinal data involving large numbers of subjects stratified by cumulative cigarette exposure. In a recent study of 57 subjects with tetraplegia, spirometry performed at 10, 20, and >20 yr (mean, 26.3 ± 3.6 yr for 26 subjects) postinjury suggests that mean FVC is reduced by about 15 ml/yr between 10 and 20 yr postinjury, increasing to 33 ml/yr between 20 and >20 yr after injury.[36] There was no relationship between level of injury and loss of FVC. This rate of decline in FVC is not in excess of that reported for an uninjured population.[37]

Measures of Lung Volumes. Tetraplegia produces a restrictive ventilatory impairment that, like FVC and FEV_1, worsens as the level of injury ascends.[17,18,34,38,39] Figure 6 shows the total lung capacity and its subdivisions for 36 subjects with tetraplegia and motor-complete lesions who were at least 6 mo postinjury.[34] As the level of injury ascends, total lung capacity is progressively reduced. Functional residual capacity that is the sum of the residual volume and the expiratory reserve volume is also reduced, although not as significantly as total lung capacity. Notably, the reduction in functional residual capacity occurs at the expense of expiratory reserve volume, with a compensatory increase in residual volume. The loss of expiratory reserve volume can be explained by the denervation of the abdominal musculature and other muscles necessary for forced exhalation. As mentioned earlier, training of the pectoralis muscles has been shown

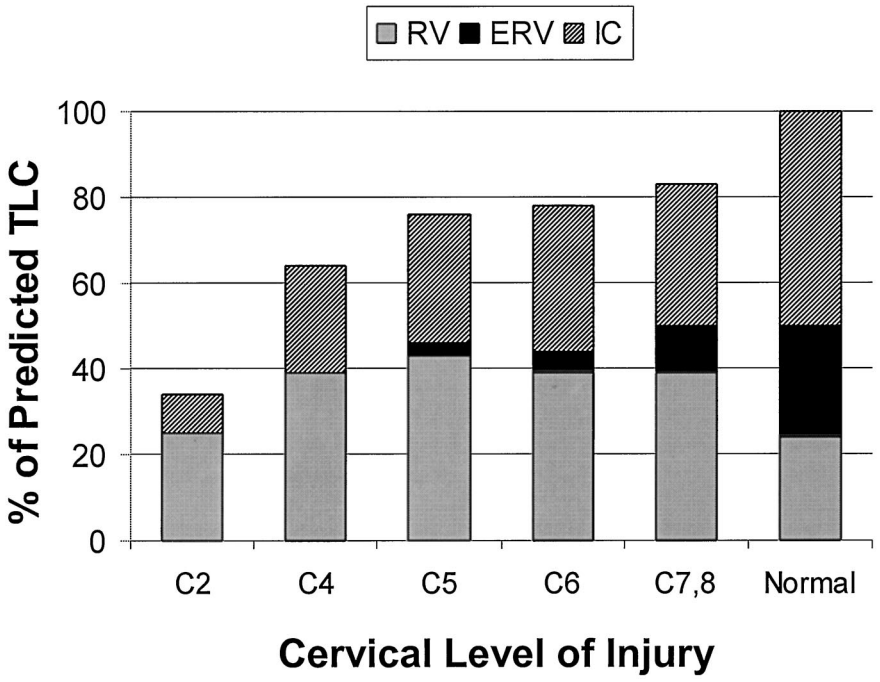

Figure 6: Total lung capacity (*TLC*) and the subdivisions of residual volume (*RV*), expiratory reserve volume (*ERV*), and inspiratory capacity (*IC*) as function of cervical level of injury (C2–C8) and normal subjects. Used with permission.[34]

to produce sustained increases in expiratory reserve volume by improving exhalation.[27] This is consistent with the observations of Loveridge et al.,[38] who showed that functional residual capacity remains constant and residual volume decreases in the first year after injury.

The balance between the opposing recoil forces of the lung and chest wall determines functional residual capacity. At functional residual capacity, the tendency of the chest wall to recoil outward to a larger volume is equal to the tendency of the lung to collapse inward to a smaller volume. The reduction in functional residual capacity seen in persons with tetraplegia is due to both an increase in lung recoil pressure and a decrease in chest wall recoil forces.

De Troyer and Heilporn[17] demonstrated that lung compliance is reduced in persons with tetraplegia. However, specific compliance (static expiratory compliance/total lung capacity) was normal. This suggests that most of the changes in the pressure-volume curve was due to loss of gas-containing alveoli, as would occur with atelectasis, whereas the remainder of the ventilated alveoli retained normal elastic properties. The authors reasoned that had the change in compliance been attributable to a uniform increase in alveolar surface tension throughout the lung, then the change in compliance would have been disproportionately large compared with the change in lung volume, and this was not observed.

Although lung compliance is reduced in these patients, the majority of the reduction in functional residual capacity is attributable to a decrease in chest wall recoil forces. This reduction is due to multiple factors, including a loss of phasic inspiratory intercostal muscle activity,[17] a stiffening of the rib cage,[17,18,21,22] and increase in the compliance of the abdominal wall.[18] The increase in compliance of the abdomen may act to reduce rib cage contribution to tidal volume generation because the lack of abdominal muscle tone produces a more distensible abdominal compartment, reducing the fulcrum effect of the contracting diaphragm on visceral contents.

SCI resulting in paraplegia produces similar alterations in lung volumes, although to a lesser degree. In seven patients with T6–T7 level of injury, mean total lung capacity was minimally reduced (77.1 ± 3.5% of predicted), and functional residual capacity was normal. Mean expiratory reserve volume was reduced to 36.0 ± 2.6% of predicted, with a compensatory increase in mean residual volume to 129.9 ± 6.8% of predicted.[30]

Mortality Associated with SCI. Recent medical advances in the care of these patients have significantly reduced both acute and long-term mortality rates.[1,6,40–42] Earlier studies showed acute mortality rates between 15% and 40% for persons with SCIs involving the lower cervical cord, with respiratory failure being a common cause of death.[7,43,44] DeVivo et al.[1] recently queried the NSCID for first-year mortality among persons admitted to a Model SCI Care System or Shiner's hospital SCI unit within 24 hr of injury (n = 9,085). An examination of consecutive 5-yr periods revealed that the odds of dying between 1993 and 1998 were reduced by 66% compared with the period from 1973–1977.

Despite the improvements in 1-yr mortality, life expectancy is reduced for persons with a significant functional impairment from an SCI. An individual who sustains an injury at age 20 with a neurologic deficit between C1 and C4 can be expected to live 32.9 yr (38.5 yr for an injury resulting in a neurologic level between C5 and C8 and 44.1 yr for a level of injury between T1 and S5). The life expectancy for an individual with ventilator dependency from SCI is 15.3 yr. These values compare with an average life expectancy of 56.8 yr for an uninjured 20-yr old (based on 1994 United States government life tables, not corrected for race or sex).[1]

TABLE 2
Five most common causes of death (% of total)

First Year Postinjury $n = 100$		Beyond First Year Postinjury $n = 481$	
Cardiac	32.0	Respiratory	22.0
Respiratory	28.0	Cardiac	20.6
Septicemia	8.0	Suicide	16.0
Digestive	7.0	Cancer	11.9
Ill-defined	5.0	Septicemia	9.8

From the National Spinal Cord Injury Database (1993–1998). Adapted from DeVivo (1).

Until recently, renal failure and complications of the urinary tract were the leading causes of death among persons with SCI.[1,45–50] But improvements in bladder care and catheter regimens have reduced mortality to the point that respiratory diseases have surpassed urinary disorders as the leading cause of death.[6] Table 2 summarizes a recent assessment of the most common causes of death both within and beyond the first year after injury. Deaths due to respiratory disease are the most common cause of death beyond 1-yr postinjury and are second only to cardiac causes of death in the first year after injury.[1] The combination of respiratory and cardiac causes account for 60% of the deaths occurring in the first year and over 42% of the deaths occurring beyond the first year. Despite the fact that 80% of all SCIs occur in men,[20] the high incidence of cardiac causes of death is surprising in this relatively young population that is well below the average age of onset of coronary artery disease in the uninjured population. These findings are consistent with a 1993 report that found pneumonia to be the leading cause of death irrespective of patient age and the length of survival after injury. Pneumonia is also the most common cause of death in those with tetraplegia.[6]

Respiratory Complications of SCI. In general, the frequency of pulmonary complications parallels the degree of respiratory impairment, increasing with progressively higher levels of injury. Earlier studies reported pulmonary complications occurring in 36% of those with any SCI,[8] and 100% of patients with injuries involving the cervical spine.[7] More recent studies have shown that respiratory complications occur in 50–67% of persons with any SCI.[9,10] Similar to the improvements in survival, the prevalence of respiratory complications also seems to have decreased over the last 25 yr. This improvement may be attributable to many factors, including better prevention, recognition, and treatment of respiratory complications, general improvements in intensive care medicine, and the utilization of multi-disciplinary treatment teams in the care of these patients.[52] The most common pulmonary complications are atelectasis, pneumonia, and respiratory failure. Complications involving the pleura and thromboembolism will also be discussed.

Atelectasis. Atelectasis is perhaps the most common respiratory complication and is virtually ubiquitous after a cervical SCI. However, atelectasis has been reported in only 37% of subjects with cervical or thoracic levels of injury.[9] This report probably underestimates the actual prevalence of atelectasis as it was based on the interpretation of plain radiographs. The true prevalence of atelectasis is difficult to assess because of the lack of consistently applied diagnostic and quantitative criterion and because episodes of atelectasis and pneumonia are combined into a single data item in the NSCID. Interventions aimed at reducing atelectasis have generally focused on increasing lung inflation[52,53] or enhancing secretion removal through assisted-cough techniques.[54,55]

The use of intermittent positive-pressure breathing treatments in persons with acute tetraplegia has not been shown to improve either vital capacity or FEV_1.[52] The effect of intermittent positive-pressure breathing treatments on other measures of lung inflation such as functional residual capacity or atelectasis has not been studied. A retrospective analysis by Peterson et al.[53] of 42 persons with a C3–4 complete injury and ventilator dependence demonstrated that mechanical ventilation with a higher (25.3 ml/kg) *vs.* a lower (15.5 ml/kg) mean tidal volume decreased the incidence of atelectasis (16% *vs.* 52% respectively, $P = 0.01$) and shortened the duration of mechanical ventilation (37.6 days *vs.* 58.7 days, $P = 0.02$). The mean peak airway pressure of 35.2 cm H_2O in the high tidal volume group was comparable with that reported by Rothen et al.,[56] who studied re-expansion of atelectasis using serial computed tomography in subjects without lung disease undergoing general anesthesia for elective procedures. Based on these data, ventilation strategies utilizing larger tidal volumes have been suggested for person with SCI.[57] This strategy seems to be commonly used among those treating ventilatory failure after SCI.

Pneumonia. In general, the incidence of pneumonia seems to be decreasing over the last decade, although there is considerable variability among reported rates. In a multicenter study conducted from 1985 to 1990 involving 179 persons with a cervical SCI, Jackson and Groomes[10] described 97

episodes of pneumonia in 70 (54%) patients. Fishburn et al.[9] reported atelectasis or pneumonia in 17 of 30 patients (56.6%) with a cervical SCI over a similar period. A more recent study reported 49 of 413 patients (12%) with an episode of pneumonia for the period 1993–1997.[4] The lower incidence of pneumonia in the latter study may be explained by the use of a more rigorous definition of pneumonia.

Analyses of the NSCID also show a similar trend. Chen et al.[58] noted that before 1992, the overall incidence of atelectasis was 23.6% and pneumonia was 18.4% for persons with an SCI during the initial acute care or rehabilitation hospitalization. Since 1996, the incidence of atelectasis or pneumonia was 12.9% during the initial rehabilitation hospitalization.

Respiratory Failure. The cause of acute respiratory failure (RF) after an SCI can be attributed to hypoxemia due to ventilation/perfusion mismatch from retained secretions, atelectasis, or pneumonia; hypercarbia resulting from respiratory muscle weakness; or both. The standard treatment of RF after an acute SCI is endotracheal intubation followed by mechanical ventilation. Although invasive ventilation offers the advantage of direct access to the lower respiratory tract facilitating the removal of secretions, the presence of endotracheal and tracheostomy tubes bypass natural defense mechanisms, increasing the likelihood of nosocomial pneumonia.[59,60] Noninvasive ventilation has been successfully used to manage acute RF due to traumatic tetraplegia.[61] Currently, noninvasive ventilation for acute RF is likely underutilized and merits further study to delineate its appropriate application.

The risk of acute RF after an SCI correlates with the level of injury. In a prospective multicenter study, RF occurred in 23 of 56 persons (40%) with high tetraplegia (C1–C4) and 28 of 123 patients (23%) with low tetraplegia (C5–C8). RF was present in 8 of 81 persons (9.9%) with injuries to the thoracic spine.[10] However, bedside measures of respiratory function may be better predictors of RF than level of injury. A retrospective analysis of 54 subjects with acute cervical SCI showed that a tidal volume of $\leq$6 ml/kg was a better predictor of RF than either a vital capacity of $\leq$15 ml/kg or level of injury.[62] In addition, patients with tetraplegia who require surgical repair experience RF, defined as the need for mechanical ventilation for $>$48 hr, more frequently than those who do not need surgery.[4]

Acute RF may also be the most important complication associated with SCI. Among pulmonary complications, RF has the longest duration, lasting a mean of 35.9 days.[10] RF is also the most important contributor to both initial acute care hospital costs and length of stay in acute cervical SCI when compared with other respiratory complications and nonrespiratory variables, such as level of injury, age, and the need for surgery.[4]

The ability to resume spontaneous ventilation after RF has been examined as a function of level and type of spinal injury and of patient age. A retrospective analysis of 409 consecutive enrollees into the NSCID with a cervical level of injury requiring mechanical ventilation revealed that 89% of those with incomplete injuries (ASIA B through D) were able to resume spontaneous ventilation at discharge from their initial rehabilitation hospitalization compared with 76% of patients with complete injuries. Although completeness of injury did not influence the rate of weaning for patients with levels between C5 and C8, patients with a C4 level and incomplete injuries were more likely to wean than patients with complete injuries ($P = 0.046$).[63] An earlier retrospective study of 134 patients with tetraplegia reported that 76 patients (57%) resumed spontaneous ventilation.[64] This lower rate of weaning in the latter study may be explained by a greater percentage of subjects with high tetraplegia. Furthermore, among patients of $>$50 yr of age, the weaning rate was only 20%.[64]

From 1973 through 1998, 18,883 patients were entered into the NSCID. At the time of discharge from their initial rehabilitation hospitalization, 567 patients (3%) were ventilator dependent, with an additional 60 patients (0.3%) requiring short-term mechanical ventilation after discharge. The distribution of these patients by level of injury and ASIA impairment score is shown in Figure 7. In addition to invasive ventilation via a tracheostomy, alternative methods of ventilatory support have been used for ventilator-dependent patients, including noninvasive ventilation via either nasal mask or mouthpiece[65] and electrophrenic pacing.[66,67]

Pleural Complications. Pleural effusion, pneumothorax, and hemothorax are less common respiratory complications. They have been reported to occur in 70% of patients with a thoracic levels of injury compared with 16% of those with cervical levels of injury, suggesting that these complications are most likely due to concurrent thoracic trauma.[10]

Thromboembolic Disease. Advances in the thromboprophylaxis have significantly reduced the prevalence of deep venous thrombosis and pulmonary embolism after SCI. This may be due in part to the use of low-molecular-weight heparin (LMWH), which has been shown to be superior to standard heparin, with less bleeding complications.[68,69] Summarizing data from eight studies involving a total of 1,362 patients published between 1965 and 1985, Weingarden[70] reported clinically apparent deep venous thrombosis in a mean of 16.3% (range, 12–64%) of patients with SCI. In 1990, results of an 8-wk pro-

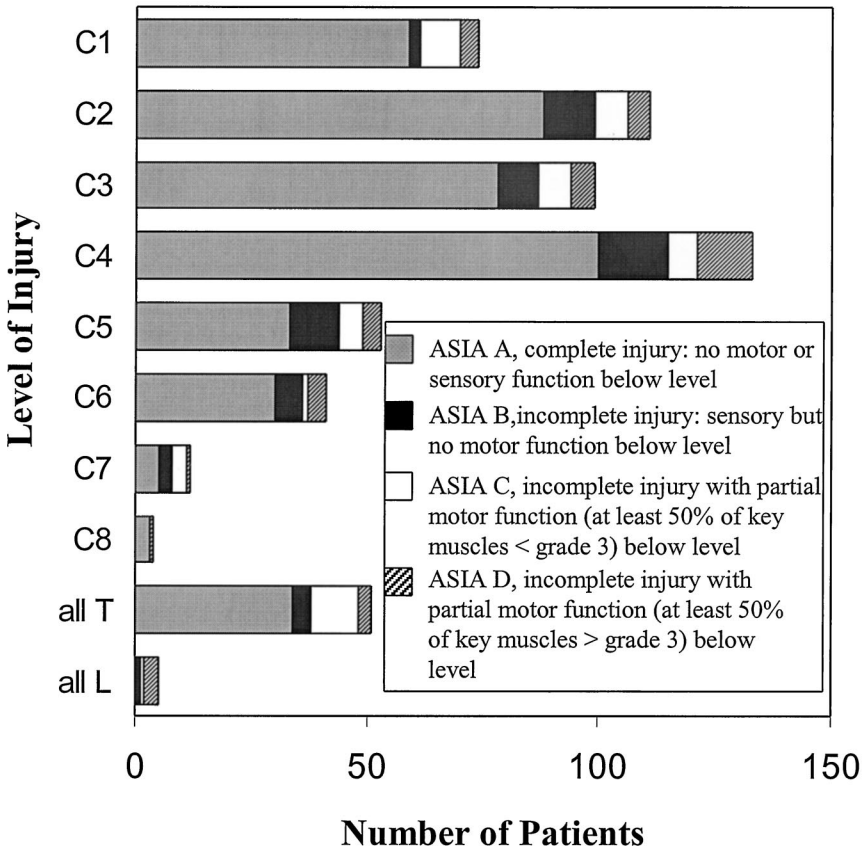

Figure 7: Level of injury and American Spinal Injury Association (*ASIA*) impairment scale for patients discharged from the initial rehabilitation hospitalization with ventilator dependence. National Spinal Cord Injury Database, 1993–1998.

spective study of 41 patients randomized to prophylaxis with either LMWH or standard heparin reported thrombosis in seven subjects (17%), all of whom were randomized to receive standard heparin.[68] Furthermore, both subjects who experienced bleeding complications were randomized to the standard heparin arm. A follow-up report after 68 patients had been treated with LMWH revealed thrombosis in seven (10.3%).[69]

Combinations of LMWH and either compression stockings or external pneumatic devices may be another factor that has contributed to the reduction in the incidence of thromboembolism. Although multimodality thromboprophylaxis using heparin and compression stockings has been shown to be superior to either modality alone in patients undergoing elective surgery,[71,72] there are no data for patients with acute SCI. However, comparing the incidence of thromboembolism in two recent retrospective studies utilizing multimodality prophylaxis with other studies using LMWH alone suggests that multimodality prophylaxis may also be superior in acute SCI. In these studies, multimodality prophylaxis reduced the incidence of deep venous thromboses to 1.2% of 423 patients with a cervical injury[4] and 2.1% of 276 subjects with any type of SCI.[73]

A similar reduction in the incidence of pulmonary embolism is seen in three studies published from our institution during the last 12 yr. In two 8-wk prospective randomized trials of LMWH or standard heparin, pulmonary embolism occurred in 5%[68] and 4%[69] of persons with an SCI in studies conducted from 1988 through 1992. More recently, there were no cases of pulmonary embolism noted from 1993 through 1997, although this retrospective analysis included only the initial acute care hospitalization. For this study, the mean length of stay was 22.2 ± 17.6 days for patients with a motor level of injury and 8.7 ± 8.5 days for patients with an injury to the spinal column who were neurologically intact.[4]

Prophylaxis should be initiated as soon as possible because the incidence of thromboembolism increases significantly after the first 72 hr after injury.[74] The optimal duration of prophylaxis is unknown, although there are data to support a minimum of 8 wk of therapy.[68,69,75]

Studies of the alterations in respiratory mechanics caused by SCI have led to improvements in the care of these patients and have expanded the understanding of normal respiratory physiology. Despite the important advances that have occurred in the prevention, diagnosis, and treatment of respiratory complications, they continue to significantly affect persons with SCI. Additional studies of interventions aimed at reducing respiratory complications are likely to further decrease the morbidity and mortality associated with these injuries.

Acknowledgment

I thank Rita Bode, PhD, for queries of the National Spinal Cord Injury Database.

References

1. DeVivo MJ, Krause S, Lammertse DP: Recent trends in mortality and causes of death among persons with spinal cord injury. *Arch Phys Med Rehabil* 1999;80: 1411–9

2. Go BK, DeVivo MJ, Richards JS: The epidemiology of spinal cord injury, in Stover SL, DeLisa JA, Whiteneck GC (eds): *Spinal Cord Injury: Clinical Outcomes from the Model Systems*. Gaithersburg, MD, Aspen Publishers, 1995, pp 21–55

3. Acute traumatic spinal cord injury

surveillance: United States, 1987. *MMWR Morb Mort Wkly Rep* 1988;37:285–6

4. Winslow C, Bode RK, Felten D, et al: The impact of respiratory complications upon length of stay and hospital costs in acute cervical spinal injury. *Chest* 2002; 121:1548–54

5. DeVivo MJ: Causes and costs of spinal cord injury in the United States. *Spinal Cord* 1997;35:809–13

6. DeVivo MJ, Black KJ, Stover SL: Cause of death during the first 12 years after spinal cord injury. *Arch Phys Med Rehabil* 1993;74:248–54

7. Bellamy R, Pitts FW, Stauffer ES: Respiratory complications in traumatic quadriplegia. *Neurosurgery* 1973;39: 596–600

8. Reines HD, Harris RC: Pulmonary complications of acute spinal cord injury. *Neurosurgery* 1987;21:193–6

9. Fishburn MJ, Marino RJ, Ditunno J: Atelectasis and pneumonia in acute spinal cord injury. *Arch Phys Med Rehabil* 1990; 71:197–200

10. Jackson AB, Groomes TE: Incidence of respiratory complications following spinal cord injury. *Arch Phys Med Rehabil* 1994;75:270–5

11. DeTroyer A, Estenne M: Coordination between rib cage muscles and diaphragm during quiet tidal breathing in humans. *J Appl Physiol* 1984;57:899–906

12. Estenne M, De Troyer A: Relationship between respiratory muscle electromyogram and rib cage motion in tetraplegia. *Am Rev Respir Dis* 1985;132:53–9

13. Danon J, Druz WS, Goldberg NB, et al: Function of the isolated paced diaphragm and the cervical accessory muscles in C1 quadriplegics. *Am Rev Respir Dis* 1979;119:909–19

14. Rochester DF: The diaphragm: Contractile properties and fatigue. *J Clin Invest* 1985;75:1397–402

15. Minh V, Dolan GF, Korrapka RF, et al: Effect of hyperinflation on inspiratory function of the diaphragm. *J Appl Physiol* 1976;40:67–73

16. Mead J, Loring SL: Analysis of volume displacement and length changes of the diaphragm during breathing. *J Appl Physiol* 1982;53:750–5

17. De Troyer A, Heilporn A: Respiratory mechanics in quadriplegia: The respiratory function of the intercostal muscles. *Am Rev Respir Dis* 1980;122: 591–600

18. Estenne M, De Troyer A: The effect of tetraplegia on chest wall statics. *Am Rev Respir Dis* 1986;134:121–4

19. De Troyer A, Estenne M, Vincken W: Rib cage motion and muscle use in high tetraplegia. *Am Rev Respir Dis* 1986;133: 1115–9

20. Nobunaga AI, Go BK, Karunas RB: Recent demographic and injury trends in people served by the Model Spinal Cord Injury Care System. *Arch Phys Med Rehabil* 1999;80:1372–82

21. Estenne M, Heilporn A, Delhez L, et al: Chest wall stiffness in patients with chronic respiratory muscle weakness. *Am Rev Respir Dis* 1983;128:1002–7

22. Bergofsky EH: Mechanism for respiratory insufficiency after cervical cord injury: A source of alveolar hypoventilation. *Ann Intern Med* 1964;61:435–47

23. Banzett RB, Topulos GP, Leith D: Bracing arms increases the capacity for sustained hyperpnea. *Am Rev Respir Dis* 1988;138:106–9

24. Raper AJ, Taliaferro-Thompson W, Shapiro W, et al: Scalene and sternomastoid muscle function. *J Appl Physiol* 1966;21:497–502

25. Moxham J, Wiles CM, Newham D, et al: Sternomastoid muscle function and fatigue in man. *Clin Sci* 1980;59:463–8

26. De Troyer A, Estenne M, Heilporn A: Mechanism of active expiration in tetraplegic subjects. *N Engl J Med* 1986;314: 740–4

27. Estenne M, Knoop C, Vanvaerenbergh J, et al: The effect of pectoralis muscle training in tetraplegic subjects. *Am Rev Respir Dis* 1989;139:1218–22

28. Estenne M, De Troyer A: Cough in tetraplegic subjects: An active process. *Ann Intern Med* 1990;112:22–8

29. Campbell EJM, Agostoni E, Newsome-Davis J: *The Respiratory Muscles: Mechanics and Neural Control*, ed 2. London, Lloyd-Luke, 1970

30. Gorini M, Corrado A, Aito S, et al: Ventilatory and respiratory muscle response to hypercapnia in patients with paraplegia. *Am J Respir Crit Care Med* 2000;162:203–8

31. Ledsome JR, Sharp JM: Pulmonary function in acute cervical cord injury. *Am Rev Respir Dis* 1981;124:41–4

32. Bluechardt MH, Wiens M, Thomas SG, et al: Repeated measurements of pulmonary function following spinal cord injury. *Paraplegia* 1992;30:768–74

33. Almenoff PL, Spungen AM, Lesser M, et al: Pulmonary function survey in spinal cord injury: Influences of smoking and level and completeness of injury. *Lung* 1995;173:297–306

34. Anke A, Aksnes AK, Stanghelle JK, et al: Lung volumes in tetraplegic patients according to cervical spinal cord injury level. *Scand J Rehabil Med* 1993;25:73–7

35. Linn WS, Spungen AM, Gong J, et al: Forced vital capacity in two large outpatient populations with chronic spinal cord injury. *Spinal Cord* 2001;39:263–8

36. Tow AMPE, Graves DE, Carter RE: Vital capacity in tetraplegia twenty years and beyond. *Spinal Cord* 2001;39:139–44

37. Xiping X, Dockery DW, Ware JH, et al: Effects of cigarette smoking on rate of loss of pulmonary function in adults: A longitudinal assessment. *Am Rev Respir Dis* 1992;146:1345–8

38. Loveridge B, Sanii R, Dubo HI: Breathing pattern adjustments during the first year following cervical spinal cord injury. *Paraplegia* 1992;30:479–88

39. Manning HL, Brown R, Scharf SM, et al: Ventilatory and P0:1 response to hypercapnia in quadriplegia. *Respir Physiol* 1992;89:97–112

40. Apple DF, Hudson LM: *Proceedings on the National Consensus Conference on Catastrophic Illness and Injury in Spinal Cord Injury: The Model*. Atlanta, 1989

41. Burke MH, Hicks AF, Robbins M, et al: Survival of patients with injuries to the spinal cord. *JAMA* 1960;172:121–4

42. Frisbie JH, Kache A: Increasing survival and changing causes of death in myelopathy patients. *J Am Paraplegia Soc* 1983;6:51–6

43. Cheshire DJE: Respiratory management in acute traumatic tetraplegia. *Paraplegia* 1964;1:252–61

44. Silver JR, Gibbon NOK: Prognosis in tetraplegia. *Br J Med* 1968;4:79–83

45. Barber KE, Cross J: The urinary tract as a cause of death in paraplegia. *J Urol* 1952;67:494–502

46. Whiteneck GC, Charlifue SW, Frankel HL, et al: Mortality, morbidity, and psychosocial outcomes of persons spinal cord injured more than 20 years ago. *Paraplegia* 1992;30:617–30

47. Bunts RC: Preservation of renal function in the paraplegic. *J Urol* 1959;81: 720–7

48. Dietrick RB, Russi S: Tabulation and review of autopsy findings in fifty five paraplegics. *JAMA* 1958;166:41–4

49. Nyquist RH, Bors E: Mortality and survival in traumatic myelopathy during

nineteen years from 1946 to 1966. *Paraplegia* 1967:5:22–48

50. Tribe CR: Causes of death in the early and late stages of paraplegia. *Paraplegia* 1963;1:19–47

51. McMichan JC, Michel L, Westbrook PR: Pulmonary dysfunction following traumatic quadriplegia. *JAMA* 1980;243: 528–31

52. Stiller K, Simionato R, Rice K, et al: The effect of intermittent positive pressure breathing on lung volumes in acute quadriparesis. *Paraplegia* 1992;30:121–6

53. Peterson WP, Barbalata L, Brooks CA, et al: The effect of tidal volumes on the time to wean persons with high tetraplegia from ventilators. *Spinal Cord* 1999; 37:284–8

54. Kirby NA, Barnerias MJ, Siebens AA: An evaluation of assisted cough in quadriparetic patients. *Arch Phys Med Rehabil* 1966;47:705–10

55. Braun SR, Giovannoni R, O'Connor M: Improving the cough in patients with spinal cord injury. *Am J Phys Med* 1982; 63:1–10

56. Rothen HU, Sporre B, Engberg G, et al: Re-expansion of atelectasis during general anesthesia: A computed tomography study. *Br J Anaesth* 1993;71:788–95

57. Peterson P, Brooks CA, Mellick DC, et al. Protocol for ventilator management in high tetraplegia. *Top Spinal Cord Inj Rehabil* 1997;2:101–6

58. Chen D, Apple DF, Hudson LM, et al: Medical complications during acute rehabilitation following spinal cord injury: Current experience of the Model Systems. *Arch Phys Med Rehabil* 1999;80:1397–401

59. Niederman MS, Ferranti RD, Ziegler A, et al: Respiratory infection complicating long-term tracheostomy: The implications of persistent gram-negative tracheobronchial colonization. *Chest* 1984; 85:39–44

60. Johanson WG, Seidenfeld JJ, Gomez P, et al: Bacteriologic diagnosis of nosocomial pneumonia following prolonged mechanical ventilation. *Am Rev Respir Dis* 1988;137:259–64

61. Tromans AM, Mecci M, Barrett FH, et al: The use of BiPAP biphasic positive airway pressure system in acute spinal cord injury. *Spinal Cord* 1998;36:481–4

62. Kuagoolwongse C, Bode RK, Felten D, et al: Tidal volume is the best predictor of respiratory failure in acute cervical spinal cord injury. *Am J Respir Crit Care Med* 2000;161:A391

63. Winslow CJ, Bode RK, Felten D, et al: *The Likelihood of Assuming Spontaneous Ventilation After Respiratory Failure Following Acute Cervical Spinal Cord Injury*. Chicago, American Spinal Injury Association, 2000

64. Wicks AB, Menter RR: Long-term outlook in quadriplegic patients with initial ventilator dependency. *Chest* 1986; 90:406–10

65. Bach JR: Alternative methods of ventilatory support for the patient with ventilatory failure due to spinal cord injury. *J Am Paraplegia Soc* 1991;14:158–74

66. Moxham J, Shneerson JM: Diaphragmatic pacing. *Am Rev Respir Dis* 1993; 148:533–6

67. Elefteriades JA, Quin JA: Diaphragm pacing: Key references. *Ann Thorac Surg* 2002;73:691–2

68. Green D, Lee MY, Lim AC, et al: Prevention of thromboembolism after spinal cord injury using low-molecular-weight heparin. *Ann Intern Med* 1990;113:571–4

69. Green D, Chen D, Chmiel J, et al: Prevention of thromboembolism in spinal cord injury: Role of low molecular weight heparin. *Arch Phys Med Rehabil* 1994;75: 290–2

70. Weingarden SI: Deep venous thrombosis in spinal cord injury: Overview of the problem. *Chest* 1992;102:636S–9S

71. Agnelli G, Piovella F, Buoncristiana C, et al: Enoxaparin plus compression stockings compared with compression stockings alone in the prevention of venous thromboembolism after elective neurosurgery. *N Engl J Med* 1998;339: 80–5

72. Wille-Jorgensen P, Thorup J, Fischer A, et al: Heparin with and without graded compression stockings in the prevention of thromboembolic complications of major abdominal surgery: A randomized trial. *Br J Surg* 1985;72:579–81

73. Deep K, Jigajinni MV, McLean AN, et al: Prophylaxis of thromboembolism in spinal injuries: Results of enoxaparin used in 276 patients. *Spinal Cord* 2001; 39:88–91

74. Green D, Rossi EC, Yao JS, et al: Deep vein thrombosis in spinal cord injury: Effect of prophylaxis with calf compression, aspirin, and dipyridamole. *Paraplegia* 1982;20:227–34

75. Green D: Prophylaxis of thromboembolism in spinal cord-injured patients. *Chest* 1992;102:649S–51S