# Traumatic Spinal Cord Injury

## Pulmonary Physiologic Principles and Management

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**KEYWORDS**

• Pulmonary function • Spinal cord injury • Respiratory muscle strength
• Restrictive airway dysfunction • Airway dynamics • Respiratory symptoms • Sleep apnea

**KEY POINTS**

- Respiratory complications, principally pneumonia, are the primary cause for premature mortality among individuals who have suffered traumatic spinal cord injury, both during the early acute post-injury period and thereafter.
- Due to paralysis of respiratory muscles, traumatic injury to the cervical and upper thoracic spinal cord is associated with restrictive pulmonary dysfunction and respiratory muscle weakness, with greater compromise of expiratory as compared with inspiratory muscle function.
- A significant number of persons with cervical spinal cord injury manifest obstructive physiology characterized by reduction in baseline airway caliber, bronchodilator responsiveness, and nonspecific airway hyperreactivity, although the clinical significance of these findings are unclear.
- Chest physiotherapeutic techniques appear to be effective early adjuncts to prevent atelectasis and promote respiratory clearance during weaning attempts and to help prevent respiratory complications, such as pneumonia, among subjects with high cervical spinal cord injury.
- The prevalence of sleep-disordered breathing among subjects with tetraplegia far exceeds that witnessed in the general population, and implies a unique underlying physiology among these individuals.

## INTRODUCTION

Traumatic injury to the cervical and upper thoracic spinal cord is associated with variable degrees of pulmonary dysfunction and disability dependent on the level and completeness of injury. The purpose of this article was to detail the pulmonary function and mechanisms of pulmonary physiologic impairment associated with traumatic spinal cord injury (SCI), and interventions to prevent pulmonary complications associated with attendant decreases in respiratory muscle strength and impaired cough. We

Disclosure: None of the authors have any financial interests in subject matter or materials discussed in this article.
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Clin Chest Med 39 (2018) 411–425
https://doi.org/10.1016/j.ccm.2018.02.002
0272-5231/18/Published by Elsevier Inc.

examine temporal changes in pulmonary function following traumatic SCI and focus on the physiologic principles that govern and affect the ability to breathe spontaneously without ventilatory support. Various interventions discussed include chest physiotherapy, pharmacologic and nonpharmacologic techniques to improve respiratory muscle strength, and electrical pacing techniques. Sleep-disordered breathing also is discussed because of its high prevalence in this population, including current thoughts regarding pathophysiology and management. This review does not address spinal shock, the acute care of the spinal cord–injured patient (including ventilator or tracheostomy management), pulmonary embolism, or management of respiratory complications. With regard to diaphragmatic pacing, the reader is directed to Anthony F. DiMarcos' article, "Diaphragm Pacing," in this issue.

## EPIDEMIOLOGY

Significant and lifelong neurologic deficits are all too often the dramatic consequence of traumatic SCI. According to 2017 estimates compiled by the National Spinal Cord Injury Data Center, the annual incidence of traumatic SCI in the United States is approximately 54 cases per 1 million people, or 17,500 new cases per year, and affects approximately 285,000 persons (range 245,000–345,000).[1] Compared with the 1970s, men still comprise approximately 80% of victims of SCI, although the average age at injury has increased from 28.7 years to 42.2 years, a consequence of our aging population and an increase in injuries resulting from falls among older individuals.[2] There has been a corresponding decrease in the percentage of SCIs resulting from vehicular crashes (47% to 38%), although this remains the most common etiology, followed by falls (31%), acts of violence (14%), sports-related injuries (9%), medical/surgical complications (5%), and others (4%).[1,2] Notwithstanding the attendant emotional and physical challenges of SCI, the socioeconomic impact across a lifetime is substantial; according to the National SCI Statistical Center, the estimated lifetime medical cost for an individual injured at age 25 with low tetraplegia is approximately $3.5 million.[1]

The past 40 years have witnessed a substantial improvement in the acute management and short-term 2-year survival in persons with SCI, although the mortality risk remains high during this period, ranging from 3.1% to 21.0%.[3–10] Pulmonary complications pose the greatest risk during the first 2 years postinjury, and include pneumonia, pulmonary edema, respiratory failure, and thromboembolism.[11,12] Despite improved early survival and shorter initial hospital lengths of stay, according to a recent study of data from the National Spinal Cord Injury Model System, the life expectancy for those surviving beyond 2 years postinjury compared with an age-matched noninjured population has declined slightly over the past 30 years, and overall long-term survival for persons with SCI remains significantly less than that of the general population regardless of injury level.[1,13] Historical data also identify a shift in the principal cause of mortality during the chronic phase of SCI; mortality related to urosepsis and renal failure has now been supplanted by sepsis and pulmonary complications, particularly pneumonia.[1,14,15] Thus, pulmonary complications are now a primary cause for morbidity and mortality in the SCI population, regardless of time postinjury.

## OVERVIEW OF RESPIRATORY MUSCLE FUNCTION

The principal muscle of inspiration is the diaphragm innervated by the phrenic nerve arising from cervical nerve roots C3 to C5. Dome-shaped, the diaphragm consists of a central tendon and skeletal muscle fibers that insert laterally along the inner surface of the lower 6 ribs and anteromedially along the costal cartilages. The region of diaphragm that closely abuts the lower ribs at functional residual capacity defines the zone of apposition which normally constitutes 30% of total rib cage surface area.[16] With inspiration and muscle shortening, the diaphragm descends and the zone of apposition decreases, thereby increasing the thoracic cavity, displacing abdominal contents caudally, and elevating the lower rib cage.[17] The external intercostal muscles and parasternal portion of the internal intercostals supplied by corresponding thoracic spinal nerves have a synergistic action with the diaphragm during inspiration, serving to elevate the 2nd through 12th ribs.[18,19] Accessory muscles of inspiration, including the sternocleidomastoid (cranial nerve [CN] XI), scalene (C2–C7), and upper trapezius (CN XI) muscles, function to elevate the upper ribs and sternum.[16]

Generally, during quiet breathing, expiration is a passive process, although recruitment of expiratory muscles is essential for force generation during active processes, such a cough or exercise. The principal muscles of expiration are the internal intercostals and the abdominal