

Respiratory problems and management in people with spinal cord injury

Author

Berlowitz, David J, Wadsworth, Brooke, Ross, Jack

Published

2016

Journal Title

Breathe

Version

Version of Record (VoR)

DOI

[10.1183/20734735.012616](https://doi.org/10.1183/20734735.012616)

Rights statement

@ ERSpublications. This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License (<https://creativecommons.org/licenses/by-nc/4.0/>), which permits unrestricted, non-commercial use, distribution and reproduction in any medium, providing that the work is properly cited.

Downloaded from

<http://hdl.handle.net/10072/101158>

Griffith Research Online

<https://research-repository.griffith.edu.au>



¹Institute for Breathing and Sleep, Austin Health, Heidelberg, Australia.

²University of Melbourne, Faculty of Medicine, Dentistry and Health Sciences, Melbourne, Australia.

³School of Human Services and Social Work, Griffith University, Logan Campus, Australia.

⁴Physiotherapy Department, Princess Alexandra Hospital, Woolloongabba, Australia.

⁵Victorian Spinal Cord Service, Austin Health, Heidelberg, Australia.

Respiratory problems and management in people with spinal cord injury

Spinal cord injury (SCI) is characterised by profound respiratory compromise secondary to the level of loss of motor, sensory and autonomic control associated with the injury. This review aims to detail these anatomical and physiological changes after SCI, and outline their impact on respiratory function. Injury-related impairments in strength substantially alter pulmonary mechanics, which in turn affect respiratory management and care. Options for treatments must therefore be considered in light of these limitations.

Cite as: Berlowitz DJ, Wadsworth B, Ross J. Respiratory problems and management in people with spinal cord injury. *Breathe* 2016; 12: 328-340.



@ERSpublications

Spinal cord injury impairs respiratory function. The associated impairments can be well managed. <http://ow.ly/1Gcy305oQIY>

Spinal cord injury (SCI) is thankfully a relatively rare injury, but one with profound personal and community impacts. The personal disability is typically substantial, with significant reductions in community participation, employment and quality of life (QOL) [1-3]. Access Economics estimated the cost of SCI in Australia in 2009 to be \$2.0 billion: \$5 million per case of paraplegia (lifetime) and almost double that (\$9.5 million) for quadriplegia [4]. In Australia, Western Europe and North America, SCI most commonly occurs secondary to motor vehicle accidents, whereas falls from trees and rooftops are the most frequent cause in South-East Asia and Oceania [5]. Worldwide, the average person with traumatic SCI is most likely to be male, in his early 30s, with a 70% chance of being paraplegic, and a 50% likelihood of having a motor and sensory complete lesion [6]. However, alongside increases in general population age, the average age of injury is also increasing [7]. Traumatic SCI

incidence varies from 8 to 246 cases per million inhabitants per year with a global prevalence from 236 to 1298 per million [8, 9]. A SCI may also be secondary to nontraumatic causes such as vascular abnormalities, tumours or infection [10], and far less is known regarding incidence and prevalence in nontraumatic SCI.

Respiratory complications remain the most common cause of mortality following SCI [11, 12]. Patients are most vulnerable to respiratory illness in the first year after injury but continue to suffer from respiratory complications throughout life. The number of respiratory complications suffered during initial admission is more important than level of injury in determining length of stay and hospital costs [13].

The most important determinants of the extent of respiratory compromise after SCI are the patterns and level of motor, sensory and autonomic neurological impairment. Classification



© ERS 2016

of the individual SCI is made on the basis of clinical examination and comprises a two-stage process of neurological level of injury (NLI) determination and then motor and sensory function. The NLI is defined as the most caudal spinal segmental level with intact motor and sensory function. Motor and sensory impairment are scored and severity of SCI classified using the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) developed by the American Spinal Injury Association (ASIA) and the International Spinal Cord Society (www.iscos.org.uk/international-standards-and-spinal-cord-independence-measure-scim) [14]. The clinical examination to determine ISNCSCI score involves the assessment of 10 muscles (motor) and 28 dermatomes (sensory) bilaterally [14, 15]. Once the NLI is determined, the injury is classified according to the ASIA Impairment Scale (AIS) (table 1).

The effects of SCI on sensory and motor functions are directly related to the local site of the cord injury and any subsequent recovery. However, the effects of SCI on the autonomic nervous system are often more global because of the anatomical organisation of the autonomic nervous system, particularly the sympathetic nervous system. The ISNCSCI taxonomy does not include any assessment of the autonomic nervous system and, while it is likely that complete paralysis (*i.e.* ISNCSCI A) is associated with a complete sympathetic lesion, this may not be the case in all individuals [16].

The ISNCSCI and separate assessments of the integrity of the autonomic nervous system describe the impairment caused by a particular injury. However, the subsequent disability, handicap and QOL are determined by the relationship between

life satisfaction, perceived health status and any comorbidities that arise from the injury.

Acute SCI and respiration

Effects of SCI

The ability to breathe deeply and cough forcefully is impaired to varying degrees depending on the level and completeness of the SCI, with greater dysfunction seen at higher injury levels [17–19]. Although a number of authors have documented the changes in pulmonary function following SCI, care is required when these data are reviewed. Because pre-morbid spirometric values are generally not available in SCI, the magnitude of the drop immediately after SCI remains unknown. Similarly, the significance of any measurements obtained after injury is inferred from population values. A recent publication addressed this limitation through the development of SCI-specific prediction equations [20] and the model is currently being prospectively validated.

Immediately following a traumatic SCI, there is a period of spinal shock resulting in flaccid paralysis of the muscles below the level of injury, which lasts for a period of weeks to months [21]. Flaccid paralysis of the intercostal muscles creates an unstable chest wall such that during inspiration, the negative intrathoracic pressure causes paradoxical inward depression of the ribs [22, 23]. This mechanical imbalance and disadvantage result in less efficient ventilation, increased work of breathing, and a tendency towards distal airway collapse and microatelectasis [24]. Airway secretions may

Table 1 ISNCSCI impairment scale

ISNCSCI category	Description	Characteristics
A	Complete	No sensory or motor function in the sacral segments S4–S5
B	Sensory incomplete	Sensory but no motor function is preserved below the neurological level, including the sacral segments S4–S5, and no motor function is preserved more than three levels below the motor level on either side of the body
C	Motor incomplete	Motor function is preserved at the most caudal sacral segments for voluntary anal contraction or the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments S4–S5), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body Less than half of key muscle functions below the single NLI have a muscle grade ≥ 3
D	Motor incomplete	Motor incomplete status as defined above, with at least half of key muscle functions below the single NLI having a muscle grade ≥ 3
E	Normal	If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments and the patient had prior deficits, then the AIS grade is E Someone without an initial SCI does not receive an AIS grade

People with SCI have the severity of the motor and sensory impairment associated with their injuries scored using first a classification of the NLI (the most caudal spinal segmental level with intact motor and sensory function) and then with the ISNCSCI taxonomy. For more detail, please see the main text.

accumulate in the lungs either through increased production [25] or decreased clearance secondary to impaired cough. It is during this period that intubation and ventilation for respiratory support is highly likely [26].

With time and a reduction in active chest wall movement secondary to weakness, the tendons, ligaments and joints of the rib cage stiffen. Together with spasticity of the intercostals, this will stabilise the rib cage at a lower absolute lung volume, such that paradoxical breathing lessens [27, 28]. The resolution of spinal shock may also improve lung volumes as the thoracic and abdominal muscles begin to develop spasticity.

The risk of respiratory failure is directly associated with injury level. Patients may lose up to one AIS level within the first few days of injury as a result of cord swelling or bleeding, making this an especially high-risk period [29]. A patient with a complete injury above C5 will typically have impaired diaphragm function and is likely to require a period of endotracheal intubation and mechanical ventilation [30]. A C5 injury level may also involve diaphragm weakness but is more likely to be associated with the ability to breathe independently. Impaired inspiration, lack of cough strength, and no movement of the hands, trunk and lower limbs are seen. A patient with a complete T12 classification will have no observable inspiratory or expiratory impairment, full upper body strength, good trunk strength and balance but no movement of the lower limbs. An understanding of this classification allows the clinician to predict the likely needs and respiratory management of their patient (table 2).

Particularly in traumatic SCI, a number of factors may modify the degree of respiratory impairment. Patients with incomplete injuries have some feeling and, often, movement preserved below the level of injury; as such, they will relatively improve the remaining function of the muscles and systems below the lesion. Traumatic brain injury occurs in many patients with a primary diagnosis of cervical SCI [31], potentially modifying airway protection and motor control. Injuries that result in traumatic SCI are frequently high velocity, and may also result in thoracic cage trauma, rib fractures and internal organ damage.

Cardiovascular and autonomic impact on respiration

In the acute post-injury period of spinal shock, not only is there flaccid paralysis of the muscles below the injury, in lesions above T6, there is also a reduction in sympathetic nervous system activity [21] and an unopposed expression of parasympathetic activity *via* the vagus nerve [32–35]. Impaired control of the autonomic nervous system seen in individuals with high thoracic and cervical SCI can lead to hypotension and cardiac arrhythmias. The most common arrhythmia seen is bradycardia. Higher, more complete injuries will result in more significant arrhythmias and these are most common in the first 14 days [36]. Patients with recurrent bradycardia may benefit from early insertion of a cardiac pacemaker [37].

Unopposed parasympathetic activation of the airways has been demonstrated to increase

Table 2 Neurological level for complete SCI, typical respiratory impairment and support [123, 155, 159, 160]

Neurological level	Dysfunction
C1–C3	Likely full time, ventilator dependent secondary to severe diaphragm weakness (paralysis) May be able to come off ventilation for brief period if able to adequately self-ventilate using frog/GPB Potential candidate for diaphragm pacing [155]
C3–C4	Diaphragm function will be impaired, reducing tidal volume and vital capacity Periods of unassisted ventilation (ventilator-free time) are likely and may be adequately supported with nocturnal ventilation alone Domiciliary ventilatory support may be noninvasive, particularly if lung volumes are high enough during day while seated
C5	Independent respiration possible in long term although initial ventilatory support common Diaphragm function intact but intercostal and abdominal muscle paralysis results in decreased lung volumes, and cough strength and effectiveness
C6–8	Independent breathing People with lesions caudal to C7 typically can augment inspiration and cough with accessory muscles, particularly pectoralis major and minor
T1–T4	Inspiratory capacity and forced expiration supported by intercostal activity; however, cough efficacy remains reduced secondary to abdominal (expiratory) weakness
T5–T12	Progressive relative improvement in muscle strength at descending lesion levels Minimal disruption to autonomic dysfunction affecting the cardiovascular system below T6
T12	Respiratory function essentially comparable to that of an able-bodied person

bronchial reactivity [38–40] and the effect may be reversed with ipratropium bromide, an anticholinergic agent. The same authors also observed that anticholinergic pre-treatment completely abolished airway reactivity following methacholine challenge [40]. Notwithstanding the parasympathetic influence on the airways, the effect is minimal compared with the restrictive pulmonary deficit secondary to muscle paralysis, and appears to affect the forced expiratory volume in 1 s (FEV₁) and forced vital capacity (FVC) equally; hence, there is no effect on the forced expiratory ratio. It has been demonstrated that short-term β -agonist administration in acute tetraplegia may confer some benefit [41]; however, there is no clear evidence of longer-term benefit from bronchodilation in cervical SCI.

Pulmonary oedema and pulmonary embolism

Pulmonary oedema can affect as many as 50% of individuals with acute tetraplegia [42]. The causes are multifactorial and include excessive fluid resuscitation in the presence of hypotension in the acute post-injury setting. The risk of pulmonary embolism is increased following acute SCI, with an incidence of 4.5% and a mortality rate of 3.5% in the first 3 months following injury [43]. In the first month post-injury, SCI patients have 500 times the risk of death from pulmonary embolism compared to age- and sex-matched controls without SCI [44]. In a retrospective audit of 222 patients with SCI from 2010 to 2013, CLEMENTS *et al.* [45] found 33 had at least one pulmonary embolism and eight of these also had a deep venous thrombosis. They found occurrence of venous thromboembolism to be associated with increased weight, male sex, completeness of motor paralysis, length of stay, associated pelvic or lower limb fracture and delayed admission to the specialist SCI centre.

Lung volumes

Lung function and respiratory muscle pressure-generating capacity change over time, and are correlated with lesion level [46, 47]. The ventilatory pattern alters significantly during the first year following injury. Immediately following injury, there is flaccid paralysis of the intercostal and abdominal muscles, with marked paradoxical abdominal and thoracic movement, and a reduction in vital capacity to approximately 20–60% of the predicted value in tetraplegia and 80–90% in paraplegia [22, 48, 49]. The paradoxical ventilation is due to the diaphragm contracting against an unstable rib cage and is more marked following cervical injuries [50]. Truncal and intercostal tone increase with time [50], stabilising the rib cage and returning the vital capacity to approximately 60% of the pre-injury level [51]. A progressive reduction in the functional residual

capacity also occurs during this time, associated with atelectasis and basal pulmonary fibrosis [51]. The residual volume remains elevated compared with normal population values [51–54], especially in the sitting position with the abdomen unbound, although the magnitude of the elevation reduces with time [51, 52]. The total lung capacity does not appear to recover significantly during this time [51, 52].

Position

Moving from upright to supine affects the respiratory function of the tetraplegic and high paraplegic individual differently to the able-bodied person. FRISBIE and BROWN [55] compared the change in neck and abdominal circumference in 13 male subjects with tetraplegia with 11 male, age-matched controls. The waist size increased in both groups when sitting but increased by a significantly ($p < 0.001$) greater amount in the subjects with tetraplegia (4.1 ± 1.1 inches *versus* 1.6 ± 0.8). This increase in abdominal girth when sitting in tetraplegia is secondary to decreased abdominal muscle strength [56] and the associated increased abdominal wall compliance [56–58]. In the seated position, the abdominal contents are less supported by the decreased abdominal wall muscle tone and fall forward, increasing the waist size and lowering the diaphragm. In able-bodied subjects, the FVC is reduced in the supine position [52, 57, 59], whereas in tetraplegia it is increased [49, 52, 53, 57, 59]. Postural changes are associated with symptoms; patients with an acute, high SCI report less breathlessness when supine compared to sitting [60]. In the supine position, the weight of the abdominal contents forces the diaphragm to a higher resting level so that contraction produces greater absolute excursion of the diaphragm; an effect that can be increased when the person with tetraplegia is tipped 15° head down from supine such that the vital capacity rises by a further 6% [61].

Cough

The ability to produce an effective cough is severely impaired in patients with cervical or high thoracic SCI [62, 63]. Patients who have loss of innervation to the abdominal muscles and the internal intercostals lose the ability to produce a forced expiration [64]. DE TROYER and ESTENNE [65] have shown that patients with injuries at C5–C8 can utilise the clavicular portion of pectoralis major to generate an expulsive force, although the extent to which this is clinically important is unclear. LINN *et al.* [17] found that in a group of patients with high tetraplegia, peak expiratory flow rate was <50% of predicted normal values. The most effective peak cough flows can be achieved with a positive pressure-supported inspiration followed

by expiration augmented by negative pressure or an assisted cough [66, 67].

Sleep disordered breathing and sleep apnoea syndrome

People with SCI have marked impairment of both respiratory function and strength after injury, and as such, the normal ventilatory instability associated with sleep may further compromise their already marginal ventilator system. In the able-bodied, the loss of wakefulness coincides with a decrease in alveolar ventilation and an associated 2–7-mmHg increase in arterial carbon dioxide tension [68]. Sleep hypoventilation is secondary to a number of separate physiological processes and there is some evidence that it may be exaggerated at sleep onset in SCI [69].

The most significant sleep-related respiratory problem post SCI is obstructive sleep apnoea (OSA) [70, 71]. The OSA appears as a direct consequence of cervical injury [72] and is up to 83% prevalent in the first year. Chronically, OSA has a prevalence of 40–91% [73–78]. The prevalence in paraplegia does not appear significantly different to that of the able-bodied population; however, the literature is far less comprehensive in this group. In the general population, untreated sleep disorders are associated with cardiovascular disease and impaired cognition [79]. Neurocognitive impairments including decreased memory and attention have also been linked to nocturnal hypoxia in tetraplegic subjects with untreated OSA [80]. In chronic SCI, most authors have observed associations between increasing age, body mass index (BMI) and neck circumference and OSA prevalence [73, 74, 77, 81], but these relationships appear weaker acutely [72]. It may be that OSA in tetraplegia is a biphasic disorder, acutely caused by the cervical SCI that partially resolves only to increase in prevalence again as people with SCI age and gain weight [72, 73]. An individual with acute tetraplegia and undiagnosed or untreated OSA may struggle to participate in the demanding process of rehabilitation [82], and any ongoing cognitive impairment [80] will also likely limit future employment opportunities that are skewed towards computer and desk based tasks due to the physical SCI disability.

Respiratory assessment

As with all clinical practice, background anatomical, pathophysiological and disease knowledge inform the clinical assessment of patients. In SCI, much can be gleaned by careful assessment of the patient's breathing pattern to identify paradoxical or unequal chest wall movement. Qualitative diaphragm movement can be assessed when viewing the patient in the supine position from the foot end of the bed to assess symmetry on deep inspiration.

Simple spirometry, particularly serial measures, provides clinically relevant information regarding the vital capacity and any airflow obstruction, and can be undertaken regularly at the bedside. Measures of muscle strength, such as the maximal inspiratory pressure (MIP) and the most sensitive, noninvasive measure of functional respiratory muscle strength, the sniff nasal inspiratory pressure, can also be readily performed at the bedside [83]. The ability to cough has been routinely assessed by measuring peak cough flow using an "asthma" peak flow meter; however, the reliability of these devices is poor [84].

Respiratory treatment

Early surgical stabilisation of the spine is typical practice in the majority of spinal units globally [85, 86]. Patients are typically intubated for surgery, admitted to intensive care post-operatively and once surgically stabilised, able to be sat out of bed. However, this upright position whilst in spinal shock may lead to increased work of breathing and hypotension, which can be countered to a degree through the use of an abdominal binder and antihypotensive medication.

Lung volume maintenance

Restoration of lung volume is a mainstay of treatment for the person with acute tetraplegia or high paraplegia. Intermittent positive pressure breathing (IPPB) *via* a mouthpiece or facemask to support inspiration prior to manually assist coughing can augment lung volume and increase exhalation flow. Similarly, insufflation using a portable, noninvasive ventilator or an in/exsufflation device will also boost inspiratory volumes prior to assisted coughing or exsufflation [66, 67]. Both IPPB and in/exsufflation can be delivered *via* a variety of interfaces including mouthpiece, facemask and tracheostomy connector. The introduction of many new ventilators for noninvasive ventilation (NIV) has overcome some of the limitations of IPPB machines such as a lack of choice of interface and the requirement for pressurised gas [87].

Breath stacking is a technique where a resuscitation bag is used with a mouth piece or facemask to deliver two or more breaths prior to exhalation in order to augment lung volume and aid in secretion clearance. This low-cost treatment can be provided at home or in the subacute environment [88–90] and while it has been shown to slow the decline in FVC when introduced for those with neuromuscular disorders [91], controlled trial evidence for efficacy is lacking.

Glossopharyngeal breathing (GPB) can be used to increase lung volumes and assist secretion clearance in the person with high tetraplegia [92]. Vital capacity may be increased by as much

as 1000 mL [58] and in ventilator users, GPB can provide security in case of ventilator failure or for brief periods of ventilator-free breathing [93].

An effective cough

A large inspiratory effort followed by a quick and forceful expiration is required in order to achieve a successful cough. A compressive force directed inwards and upwards under the diaphragm, and compression of the rib cage may provide manual, expiratory assistance, and replace the work of the abdominal and internal intercostal muscles. FROWNFEILER and MASSERY [94] describe various methods of achieving assisted cough. The therapist must synchronise the applied compressive force with the expiratory effort of the patient. Mechanical in/exsufflation devices to assist coughing have been documented to assist with cough in patients with neuromuscular disorders and respiratory muscle weakness [66, 67, 95–97]; however, there are essentially no well-controlled, longer-term comparison data.

Respiratory muscle training and the position of the diaphragm

A recent Cochrane and associated review and meta-analysis have demonstrated that respiratory muscle training (RMT) can significantly improve respiratory muscle strength, function and endurance during the period of training in tetraplegia [98, 99]. A significant benefit of RMT was demonstrated for vital capacity (mean difference (95% CI) 0.41 (0.17–0.64) L) MIP (10.66 (3.59–17.72) cmH₂O), maximal expiratory pressure 10.31 (2.80–17.82) cmH₂O), maximum voluntary ventilation (17.51 (5.20–29.81) L·min⁻¹) and inspiratory capacity (0.35 (0.05–0.65) L). Unfortunately, no high-quality, long-term data are available and as with all muscle training, the effect on strength appears to decay or be lost with cessation of training.

Elasticated abdominal binders are used to minimise the effect of postural hypotension and aid respiration [100–103]. Abdominal binding decreases abdominal compliance, and restores pressure transference across the thorax and abdomen, allowing the diaphragm to assume a more normal resting position in the upright posture [58]. WADSWORTH *et al.* [104] found that not only does an abdominal binder improve vital capacity, FEV₁, peak expiratory flow and MIP but also increases the time the patient can sustain voice.

Secretion and ventilation management of individuals with acute cervical SCI differs from that required by individuals with pulmonary dysfunction due to non-neurological injuries [105]. Secretions accumulate secondary to increased production [25], poor cough and, in some instances, aspiration of saliva. Atelectasis leads to impaired aeration, infection and pneumonia [35]. A literature review of respiratory

management during the first 6 weeks following cervical SCI showed a protocol using a combination of techniques, which may include IPPB, manually assisted coughing, respiratory muscle resistance training, NIV and/or a clinical pathway is most likely to provide positive patient outcomes [106].

Extubation

Readiness for weaning from mechanical ventilation is indicated by a vital capacity of 15 mL·kg⁻¹ or greater [107, 108], a decreasing sputum load, ability to co-operate, a patent upper airway, a relatively clear chest radiograph with no new changes, and reduction in the requirement for ventilator assistance [35, 109, 110]. Ideally, extubation of the patient with SCI is a co-ordinated decision involving medical, surgical, nursing and physiotherapy staff. Extubation should occur early in the day to allow for a period of intensive observation and chest physiotherapy in order to minimise the risk of respiratory deterioration and re-intubation. Adequate secretion clearance and techniques aimed at volume maintenance/augmentation are recommended [105, 106, 110, 111]. Extubation failure is a poor patient outcome, associated with increased mortality, tracheostomy rate and length of hospital stay [112, 113].

Tracheostomy

High-level tetraplegic patients may be safely managed without invasive ventilation or tracheostomy in units with significant SCI and NIV expertise [114]. Whilst BERNEY *et al.* [111] showed early extubation and intensive physiotherapy may decrease the intensive care length of stay in suitable patients, this and the previous study by BACH [114] reflect the experience of specialist SCI and NIV services and, as such, may not be generalisable. Failure to intubate in a timely way in the presence of tetraplegia can lead to the need for emergency airway intervention [29, 109] or catastrophic airway loss and death [30]. In a generalist unit not familiar with SCI-specific management, intubation and invasive ventilation followed by the use of a tracheostomy to facilitate weaning from ventilation may provide a safer option for the patient.

It therefore follows that mechanical ventilation with or without tracheostomy is usual for most patients with acute traumatic cervical SCI. Patients are more likely to progress to tracheostomy if they experience respiratory complications, or have a complete motor SCI or an admission AIS motor score of ≤22. Lesion level, as well as completeness, is important. Changes on magnetic resonance imaging at the C3 level are predictive of the need for tracheostomy [115] and Como *et al.* [29] found a tracheostomy rate of 81–83% in patients with a complete SCI above the level of C5. Overall tracheostomy incidence in tetraplegia is

reported to be between 10% and 60% [116, 117] with FVC being an important predictor of the need for tracheostomy, with cut-points of <830 and ≤500 mL. BERNEY *et al.* [106] also found volume of pulmonary secretions and gas exchange to be predictive of airway management. Tracheostomy may be performed as early as day 4 following anterior cervical surgery with no increase in the incidence of wound or implant infection [118], and has been associated with fewer days on the ventilator and a shorter hospital stay in selected patients [119].

Weaning

The best method of weaning from ventilation in SCI is unclear. Reports suggest it is best performed with the patient supine [28, 59, 110] or with a head down tip [120, 121], to optimise diaphragm function. COHN [122] suggests that weaning should be thought of as a conditioning process for the diaphragm and warns that fatigue of the muscle should be avoided. WALLBOM *et al.* [110] suggest a regime of twice-daily trials with a 3-4-h rest period in between trials and gradual increases for each session until weaning is achieved. An evidence-based protocol to guide weaning of ventilator-dependent cervical SCI patients has been shown to increase mean maximal inspiratory and expiratory pressure, mean vital capacity, and mean off-ventilator breathing times [120].

Speech options with a tracheostomy

Communication options for patients with tracheostomy in the intensive care setting should be explored as soon as practicable. Patients with intact hand function can write or type whilst ventilated, but this option is either impossible or very difficult for the person with tetraplegia. Lip reading, or use of eye gaze boards or eye blinks are also useful communication methods; however, all of these methods are slow, often frustrating for the patient and carer, and give rise to frequent miscommunication. Full or partial deflation of the tracheostomy cuff can allow for leakage of air across the vocal cords and therefore speech. "Leak speech" can be optimised by taking a multidisciplinary approach consulting with the speech pathologist, physiotherapist or respiratory therapist and physician [123, 124].

Ventilator adjustments to compensate for loss of air through the mouth and nose during cuff deflation will ensure the patient remains adequately ventilated [125]. Increasing tidal volume, positive end-expiratory pressure and inspiratory time will facilitate speech by compensating for leak and allowing the patient to remain stable on ventilation [126]. It is also possible to place a one-way speaking valve in line with the patient's breathing circuit to redirect all exhaled air across the vocal folds [127].

This requires the circuit to be reconfigured in order to facilitate speech in addition to cuff deflation and ventilator adjustment. Cuff deflation may also allow for use of a one-way speaking valve in the nonventilated, tracheostomised patient [128]. Speaking valve use may increase work of breathing and monitoring for fatigue is required.

Tracheostomy decannulation

Removal of the tracheostomy tube in the patient with SCI requires assessment of cough effectiveness, airway patency and adequacy of the swallow [129, 130]. Other considerations such as co-operation of the patient, oxygen requirements, medical stability, presence of respiratory infection or pending surgery should be taken into account [131, 132]. As the incidence of aspiration is moderately high in acute tetraplegia (16-41%), speech pathology assessment is required [133, 134]. Speaking valve use during oral intake has been shown to decrease aspiration risk in nonventilated patients [135] and persistent aspiration following cervical SCI may not be an obstacle to decannulation if a risk management approach is taken [136]. Management of the SCI patient by a specialist multidisciplinary tracheostomy team can decrease total cannulation time and acute length of stay with associated cost savings [137]. While most patients will be able to clear secretions post-tracheostomy removal by use of mechanical in/exsufflation or manually assisted coughing [110, 111], insertion of a minitracheostomy may be considered. Minitracheostomy is also useful where manually assisted coughing is contraindicated such as in the presence of intra-abdominal trauma or fractured ribs.

Long-term ventilation

With increased survival after high SCI, long-term ventilation is now 6-8% prevalent in the developed world [119, 138-140]. It is a common assumption amongst the able-bodied that life with a high SCI would not be worth living [3]; however, research into perceived QOL in the ventilator-associated SCI group indicates that this is not the case. The majority of those living in the community report good or excellent QOL and, in some cases, higher QOL than their non-ventilator associated counterparts [141]. There is however, a discrepancy in QOL in the first year following discharge from hospital in ventilator users and non-ventilator users with SCI [142]. This may indicate a period of increased stress for those with SCI and their carers as they adapt to life in the community with high care needs. Discharging a person with full time ventilation into the community requires a coordinated multidisciplinary team and liaison with the person's local community. Comprehensive training, back-up equipment and planning for unusual events such as power failures

need to be considered to ensure the safety of the ventilated person. Peer support can be useful to allay fear and assist with practical planning. Annual medical evaluations of the patient's ability to breathe may identify delayed diaphragm recovery [143].

Readmissions to hospital with a chest infection

As survival of individuals following SCI increases, the impact of ageing on respiratory health and wellbeing has become more evident [144, 145]. POSTMA *et al.* [146] found that pulmonary function tests were a stronger predictor of respiratory infection in the first year following injury than lesion level or completeness and similarly, RAAB *et al.* [147] observed a linear relationship between inspiratory muscle strength and pneumonia. Patients with a vital capacity of <2 L are at greatest risk of developing late-onset respiratory failure [148], and an association between decline in vital capacity over time with increased BMI and an age at injury >30 years has been described [46].

Smoking cessation is a clear priority for those with SCI, particularly for those with higher lesions and impaired cough effectiveness. SAUNDERS *et al.* [149] found a higher incidence of smokers compared to the rest of the population in their study of 833 American adults with SCI. They also found that those with lower socioeconomic status were more likely to smoke, as were those with poor access to care.

Treatment of OSA

The gold standard for treatment of OSA remains continuous positive pressure therapy (CPAP), but only 20–50% of people with SCI and OSA are able to adhere with CPAP [74, 76, 77, 150]. People with tetraplegia are far more likely to need CPAP for OSA, yet upper-limb motor dysfunction, reduced independence for donning and doffing of masks, and the associated need for increased carer support time challenge adherence. The less claustrophobic nature of nasal pillows may mean that they are well

sued to tetraplegic users and the lower pressures required by many tetraplegic CPAP users allows for greater flexibility in interface choice as leakage is less likely at lower pressures [151]. These practical challenges to the use of CPAP for OSA in tetraplegia and the uncertainty as to the real benefit of the therapy in SCI limit the ability of clinicians to provide evidence-based advice to their patients. The results of a current study investigating the effect of CPAP for OSA in acute tetraplegia will assist with informing future practice recommendations in this area [82].

Electrical and magnetic stimulation of breathing

A paralysed diaphragm can be stimulated *via* a phrenic nerve pacer or by direct motor point pacing of the diaphragm if the phrenic nerve is intact, and the cell bodies of C3, C4 and C5 at the spinal cord are viable [152, 153]. Full pre-operative assessment is required to ascertain a patient's suitability for stimulation [154]. Extensive post-operative training is necessary to increase diaphragmatic endurance, and to teach the patient, family and carers the necessary skills and understanding of the device. The conditioning period may be complicated by initial feelings of dyspnoea, particularly if the patient has previously been ventilated with large tidal volumes and associated low bicarbonate [152]. For some patients, phrenic nerve pacing will provide a full-time alternative to the ventilator and the tracheostomy will no longer be required, but for others, the ventilator and tracheostomy will be required for additional hours during the day or night [155].

Benefits include greater wheelchair mobility, elimination of the fear of accidental ventilator disconnection, loss of social stigma associated with being attached to a ventilator, improved speech, no noise from the ventilator, reduced need for carer input, and improved well-being and overall health [155, 154, 156, 157]. Disadvantages include the need for major surgery involving thoracotomy, risk of surgical damage to the phrenic nerve and failure of the implanted device [154, 158].

Conflict of interest

Disclosures can be found alongside this article at breathe.ersjournals.com

References

1. Müller R, Peter C, Cieza A, *et al.* Social skills: a resource for more social support, lower depression levels, higher quality of life, and participation in individuals with spinal cord injury? *Arch Phys Med Rehabil* 2015; 96: 447–455.
2. van Koppenhagen CF, Post MW, van der Woude LH, *et al.* Changes and determinants of life satisfaction after spinal cord injury: a cohort study in the Netherlands. *Arch Phys Med Rehabil* 2008; 89: 1733–1740.
3. Hammell KW. Exploring quality of life following high spinal cord injury: a review and critique. *Spinal Cord* 2004; 42: 491–502.
4. AccessEconomics. The economic cost of spinal cord injury and traumatic brain injury in Australia: report by Access Economics Pty Limited for The Victorian Neurotrauma Initiative; 2009 June. <https://www.tac.vic.gov.au/about-the-tac/our-organisation/research/tac-neurotrauma-research/vni/the20economic20cost20of20spinal20cord20injury20and20traumatic20brain20injury20in20australia.pdf>
5. Cripps RA, Lee BB, Wing P, *et al.* A global map for traumatic spinal cord injury epidemiology: towards a living data repository for injury prevention. *Spinal Cord* 2011; 49: 493–501.

6. Wyndaele M, Wyndaele JJ. Incidence, prevalence and epidemiology of spinal cord injury: what learns a worldwide literature survey? *Spinal Cord* 2006; 44: 523-529 527p.
7. DeVivo MJ. Epidemiology of traumatic spinal cord injury: trends and future implications. *Spinal Cord* 2012; 50: 365-372.
8. Lee BB, Cripps RA, Fitzharris M, *et al.* The global map for traumatic spinal cord injury epidemiology: update 2011, global incidence rate. *Spinal Cord* 2014; 52: 110-116.
9. Furlan JC, Sakakibara BM, Miller WC, *et al.* Global incidence and prevalence of traumatic spinal cord injury. *Can J Neurol Sci* 2013; 40: 456-464.
10. New PW, Sundararajan V. Incidence of non-traumatic spinal cord injury in Victoria, Australia: a population-based study and literature review. *Spinal Cord* 2008; 46: 406-411.
11. van den Berg ME, Castellote JM, de Pedro-Cuesta J, *et al.* Survival after spinal cord injury: a systematic review. *J Neurotrauma* 2010; 27: 1517-1528.
12. Waddimba AC, Jain NB, Stolzmann K, *et al.* Predictors of Cardiopulmonary Hospitalization in Chronic Spinal Cord Injury. *Arch Phys Med Rehabil* 2009; 90: 193-200.
13. Winslow C, Bode RK, Felton D, *et al.* Impact of respiratory complications on length of stay and hospital costs in acute cervical spine injury. *Chest* 2002; 121: 1548-1554.
14. Maynard FM Jr, Bracken MB, Creasey G, *et al.* International Standards for Neurological and Functional Classification of Spinal Cord Injury. American Spinal Injury Association. *Spinal Cord* 1997; 35: 266-274.
15. Nesathurai S, ed. *The rehabilitation of people with spinal cord injury*. 2nd Edn. Boston, Blackwell Science, 2000.
16. Previnaire JG, Soler JM, El Masri W, *et al.* Assessment of the sympathetic level of lesion in patients with spinal cord injury. *Spinal Cord* 2008; 47: 122-127.
17. Linn WS, Adkins RH, Gong H Jr, *et al.* Pulmonary function in chronic spinal cord injury: a cross-sectional survey of 222 southern California adult outpatients. *Arch Phys Med Rehabil* 2000; 81: 757-763.
18. Vázquez R, Sedes P, Fariña M, *et al.* Respiratory management in the patient with spinal cord injury. *BioMed Res Int* 2013; 168757-168757.
19. Benditt JO, Boitano LJ. Pulmonary issues in patients with chronic neuromuscular disease. *Am J Respir Crit Care Med* 2013; 187: 1046-1055.
20. Mueller G, de Groot S, van der Woude LH, *et al.* Prediction models and development of an easy to use open-access tool for measuring lung function of individuals with motor complete spinal cord injury. *J Rehabil Med* 2012; 44: 642-647.
21. Ditunno JF, Little JW, Tessler A, *et al.* Spinal shock revisited: a four-phase model. *Spinal Cord* 2004; 42: 383-395.
22. Lucke KT. Pulmonary management following acute SCI. *J Neurosci Nurs* 1998; 30: 91-104.
23. Menter RR, Bach J, Brown DJ, *et al.* A review of the respiratory management of a patient with high level tetraplegia. *Spinal Cord* 1997; 35: 805-808.
24. Fishburn MJ, Marino RJ, Ditunno JF Jr. Atelectasis and pneumonia in acute spinal cord injury. *Arch Phys Med Rehabil* 1990; 71: 197-200.
25. Bhaskar KR, Brown R, O'Sullivan DD, *et al.* Bronchial mucus hypersecretion in acute quadriplegia. Macromolecular yields and glycoconjugate composition. *Am Rev Respir Dis* 1991; 143: 640-648.
26. Claxton AR, Wong DT, Chung F, *et al.* Predictors of hospital mortality and mechanical ventilation in patients with cervical spinal cord injury. *Can J Anaesth* 1998; 45: 144-149.
27. Axen K, Pineda H, Shunfenthal I, *et al.* Diaphragmatic function following cervical cord injury: neurally mediated improvement... vital capacity measurements. *Arch Phys Med Rehabil* 1985; 66: 219-222.
28. Mansel JK, Norman JR. Respiratory complications and management of spinal cord injuries. *Chest* 1990; 97: 1446-1452.
29. Como JJ, Sutton ERH, McCunn M, *et al.* Characterizing the need for mechanical ventilation following cervical spinal cord injury with neurologic deficit. *J Trauma Acute Care Surg* 2005; 59: 912-916.
30. Hassid VJ, Schinco MA, Tepas JJ, *et al.* Definitive establishment of airway control is critical for optimal outcome in lower cervical spinal cord injury. *J Trauma* 2008; 65: 1328-1332.
31. Michael DB, Guyot DR, Darmody WR. Coincidence of head and cervical spine injury. *J Neurotrauma* 1989; 6: 177-189.
32. Bravo G, Rojas-Martínez R, Larios F, *et al.* Mechanisms involved in the cardiovascular alterations immediately after spinal cord injury. *Life Sciences* 2001; 68: 1527-1534.
33. Yardley CP, Fitzsimons CL, Weaver LC. Cardiac and peripheral vascular contributions to hypotension in spinal cats. *Am J Physiol Heart Circ Physiol* 1989; 257: H1347-H1353.
34. Garstang SV, Miller-Smith SA. Autonomic nervous system dysfunction after spinal cord injury. *Phys Med Rehabil Clin N Am* 2007; 18: 275-296.
35. Berly M, Shem K. Respiratory management during the first five days after spinal cord injury. *J Spinal Cord Med* 2007; 30: 309-318.
36. Lehmann KG, Lane JG, Piepmeier JM, *et al.* Cardiovascular abnormalities accompanying acute spinal cord injury in humans: incidence, time course and severity. *J Am Coll Cardiol* 1987; 10: 46-52.
37. Moerman J, Christie D, Sykes L, *et al.* Early cardiac pacemaker placement for life-threatening bradycardia in traumatic spinal cord injury. *J Trauma Inj, Infect Crit Care* 2011; 70.
38. Spungen AM, Dicipinigitis PV, Almenoff PL, *et al.* Pulmonary obstruction in individuals with cervical spinal cord lesions unmasked by bronchodilator administration. *Paraplegia* 1993; 31: 404-407.
39. Almenoff PL, Alexander LR, Spungen AM, *et al.* Bronchodilatory effects of ipratropium bromide in patients with tetraplegia. *Paraplegia* 1995; 33: 274-277.
40. Dicipinigitis PV, Spungen AM, Bauman WA, *et al.* Bronchial hyperresponsiveness after cervical spinal cord injury. *Chest* 1994; 105: 1073-1076.
41. Barratt DJ, Harvey LA, Cistulli PA, *et al.* The use of bronchodilators in people with recently acquired tetraplegia: a randomised cross-over trial. *Spinal Cord* 2012; 50: 836-839.
42. Lanig IS, Peterson WP. The respiratory system in spinal cord injury. *Phys Med Rehabil Clin N Am* 2000; 11: 29-43.
43. Aito S, Pieri A, D'Andrea M, *et al.* Primary prevention of deep venous thrombosis and pulmonary embolism in acute spinal cord injured patients. *Spinal Cord* 2002; 40: 300-303.
44. Christie S, Thibault-Halman G, Casha S. Acute pharmacological DVT prophylaxis after spinal cord injury. *J Neurotrauma* 2011; 28: 1509-1514.
45. Clements R, Churilov L, Wahab AL, *et al.* Exploratory analysis of factors associated with venous thromboembolism in Victorian acute traumatic spinal cord-injured patients 2010-2013. *Spinal Cord* 2016; in press [DOI: 10.1038/sc.2016.94].
46. van Silfhout L, Peters AEJ, Berlowitz DJ, *et al.* Long-term change in respiratory function following spinal cord injury. *Spinal Cord* 2016; 54: 714-719.
47. Mueller G, de Groot S, van der Woude L, *et al.* Time-courses of lung function and respiratory muscle pressure generating capacity after spinal cord injury: a prospective cohort study. *J Rehabil Med* 2008; 40: 269-276.
48. Ledsome JR, Sharp JM. Pulmonary function in acute cervical cord injury. *Am Rev Respir Dis* 1981; 124: 41-44.
49. Baydur A, Adkins RH, Milic-Emili J. Lung mechanics in individuals with spinal cord injury: effects of injury level and posture. *J Appl Physiol* 2001; 90: 405-411.
50. Morgan MD, Gourlay AR, Silver JR, *et al.* Contribution of the rib cage to breathing in tetraplegia. *Thorax* 1985; 40: 613-617.
51. Haas F, Axen K, Pineda H, *et al.* Temporal pulmonary function changes in cervical cord injury. *Arch Phys Med Rehabil* 1985; 66: 139-144.
52. Loveridge B, Sanii R, Dubo HI. Breathing pattern adjustments during the first year following cervical spinal cord injury. *Paraplegia* 1992; 30: 479-488.
53. Forner JV. Lung volumes and mechanics of breathing in tetraplegics. *Paraplegia* 1980; 18: 258-266.
54. Bruschi C, Cerveri I, Zoia MC, *et al.* Reference values of maximal respiratory mouth pressures: a population-based study. *Am Rev Respir Dis* 1992; 146: 790-793.

55. Frisbie JH, Brown R. Waist and neck enlargement after quadriplegia. *J Am Paraplegia Soc* 1994; 17: 177-178.
56. Estenne M, Pinet C, De Troyer A. Abdominal muscle strength in patients with tetraplegia. *Am J Respir Crit Care Med* 2000; 161: 707-712.
57. Estenne M, De Troyer A. Mechanism of the postural dependence of vital capacity in tetraplegic subjects. *Am Rev Respir Dis* 1987; 135: 367-371.
58. Alvarez SE, Peterson M, Lunsford BR. Respiratory treatment of the adult patient with spinal cord injury. *Phys Ther* 1981; 61: 1737-1745.
59. Chen CF, Lien IN, Wu MC. Respiratory function in patients with spinal cord injuries: effects of posture. *Paraplegia* 1990; 28: 81-86.
60. Arora S, Flower O, Murray NPS, et al. Respiratory care of patients with cervical spinal cord injury: a review. *Crit Care Resusc* 2012; 14: 64-73.
61. Cameron GS, Scott JW, Jousse AT, et al. Diaphragmatic respiration in the quadriplegic patient and the effect of position on his vital capacity. *Ann Surg* 1955; 141: 451-456.
62. Roth EJ, Lu A, Primack S, et al. Ventilatory function in cervical and high thoracic spinal cord injury: relationship to level of injury and tone. *Am J Phys Med Rehabil* 1997; 76: 262-267.
63. Wang AY, Jaeger RJ, Yarkony GM, et al. Cough in spinal cord injured patients: the relationship between motor level and peak expiratory flow. *Spinal Cord* 1997; 35: 299-302.
64. Gounden P. Static respiratory pressures in patients with post-traumatic tetraplegia. *Spinal Cord* 1997; 35: 43-47.
65. De Troyer A, Estenne M. The expiratory muscles in tetraplegia. *Paraplegia* 1991; 29: 359-363.
66. Bach JR. Mechanical insufflation-exsufflation: comparison of peak expiratory flows with manually assisted and unassisted coughing techniques. *Chest* 1993; 104: 1553-1562.
67. Chatwin M, Ross E, Hart N, et al. Cough augmentation with mechanical insufflation/exsufflation in patients with neuromuscular weakness. *Eur Respir J* 2003; 21: 502-508.
68. Olson EJ, Simon PM. Sleep-wake cycles and the management of respiratory failure. *Curr Opin Pulm Med* 1996; 2: 500-506.
69. Bascom AT, Sankari A, Goshgarian HG, et al. Sleep onset hypoventilation in chronic spinal cord injury. *Physiol Rep* 2015; 3: e12490.
70. Castriotta RJ, Wilde MC, Sahay S. Sleep disorders in spinal cord injury. *Sleep Med Clin* 2012; 7: 643-653.
71. Giannoccaro MP, Moghadam KK, Pizza F, et al. Sleep disorders in patients with spinal cord injury. *Sleep Med Rev* 2013; 17: 399-409.
72. Berlowitz DJ, Brown DJ, Campbell DA, et al. A longitudinal evaluation of sleep and breathing in the first year after cervical spinal cord injury. *Arch Phys Med Rehabil* 2005; 86: 1193-1199.
73. Berlowitz DJ, Spong J, Gordon I, et al. Relationships between objective sleep indices and symptoms in a community sample of people with tetraplegia. *Arch Phys Med Rehabil* 2012; 93: 1246-1252.
74. Burns S, Kapur V, Yin K, et al. Factors associated with sleep apnea in men with spinal cord injury: a population-based case-control study. *Spinal Cord* 2001; 39: 15-22.
75. Leduc BE, Dagher JH, Mayer P, et al. Estimated prevalence of obstructive sleep apnea-hypopnea syndrome after cervical cord injury. *Arch Phys Med Rehabil* 2007; 88: 333-337.
76. Sankari A, Bascom A, Oomman S, et al. Sleep disordered breathing in chronic spinal cord injury. *J Clin Sleep Med* 2014; 10: 65-72.
77. Stockhammer E, Tobon A, Michel F, et al. Characteristics of sleep apnea syndrome in tetraplegic patients. *Spinal Cord* 2002; 40: 286-294.
78. Tran K, Hukins C, Geraghty T, et al. Sleep-disordered breathing in spinal cord-injured patients: A short-term longitudinal study. *Respirology* 2010; 15: 272-276.
79. Punjabi NM. The epidemiology of adult obstructive sleep apnea. *Proc Am Thorac Soc* 2008; 5: 136-143.
80. Sajkov D, Marshall R, Walker P, et al. Sleep apnoea related hypoxia is associated with cognitive disturbances in patients with tetraplegia. *Spinal Cord* 1998; 36: 231-239.
81. McEvoy RD, Myktyyn I, Sajkov D, et al. Sleep apnoea in patients with quadriplegia. *Thorax* 1995; 50: 613-619.
82. Berlowitz DJ, Ayas N, Barnes M, et al. Auto-titrating Continuous Positive Airway Pressure Treatment for Obstructive Sleep Apnoea after Acute Quadriplegia (COASA): study protocol for a randomized controlled trial. *Trials* 2013; 14: 181.
83. Polkey MI, Green M, Moxham J. Measurement of respiratory muscle strength. *Thorax* 1995; 50: 1131-1135.
84. Stefan Tino K, Victoria M, Surinder Singh B, et al. Accuracy of portable devices in measuring peak cough flow. *Physiol Meas* 2015; 36: 243-257.
85. Fehlings MG, Rabin D, Sears W, et al. Current practice in the timing of surgical intervention in spinal cord injury. *Spine* 2010; 35: Suppl., S166-S173.
86. Fehlings MG, Vaccaro A, Wilson JR, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). *PLoS One* 2012; 7: e32037.
87. Bott J, Keilty SEJ, Noone L. Intermittent positive pressure breathing - a dying art? *Physiotherapy* 1992; 78: 656-660.
88. Torres-Castro R, Vilaro J, Vera-Urbe R, et al. Use of air stacking and abdominal compression for cough assistance in people with complete tetraplegia. *Spinal Cord* 2014; 52: 354-357.
89. Armstrong A. Developing a breath-stacking system to achieve lung volume recruitment. *Br J Nurs* 2009; 18: 1166-1169.
90. McKim D. Keeping ventilated and 'at-risk' patients out of intensive care unit. *Can Resp J* 2008; 15: Suppl. C, 9C-10C.
91. McKim DA, Katz SL, Barrowman N, et al. Lung volume recruitment slows pulmonary function decline in duchenne muscular dystrophy. *Arch Phys Med Rehabil* 2012; 93: 1117-1122.
92. Pryor J. Physiotherapy for airway clearance in adults. *Eur Respir J* 1999; 14: 1418-1424.
93. Dail CW, Zumwalt M, Adkins H. A manual for instruction of glossopharyngeal breathing. 1956. <https://catalog.hathitrust.org/Record/002066560>
94. Frownfelter D, Massery M, eds. *Facilitating Airway Clearance with Coughing Techniques*. Amsterdam, Elsevier Health Sciences, 2014.
95. Sancho J, Servera E, Diaz J, et al. Efficacy of mechanical insufflation-exsufflation in medically stable patients with amyotrophic lateral sclerosis. *Chest* 2004; 125: 1400-1405.
96. Vianello A, Corrado A, Arcaro G, et al. Mechanical insufflation-exsufflation improves outcomes for neuromuscular disease patients with respiratory tract infections...including commentary by Goncalves MR and Bach JR. *Am J Phys Med Rehabil* 2005; 84: 83-91.
97. Whitney J, Harden B, Keilty S. Assisted cough: a new technique. *Physiotherapy* 2002; 88: 201-207 207p.
98. Berlowitz DJ, Tamplin J. Respiratory muscle training for cervical spinal cord injury. *Cochrane Database Syst Rev* 2013; 7: CD008507.
99. Tamplin J, Berlowitz DJ. A systematic review and meta-analysis of the effects of respiratory muscle training on pulmonary function in tetraplegia. *Spinal Cord* 2014; 52: 175-180.
100. Goldman JM, Rose LS, Williams SJ, et al. Effect of abdominal binders on breathing in tetraplegic patients. *Thorax* 1986; 41: 940-945.
101. McCool FD, Pichurko BM, Slutsky AS, et al. Changes in lung volume and rib cage configuration with abdominal binding in quadriplegia. *J Appl Physiol* 1986; 60: 1198-1202.
102. Scott M, Frost F, Supinski G. The effect of body position and abdominal binders in chronic tetraplegic subjects more than 15 years post-injury. *J Am Paraplegia Soc* 1993; 16: 117.
103. Wadsworth BM, Haines TP, Cornwell PL, et al. Abdominal binder use in people with spinal cord injuries: a systematic review and meta-analysis. *Spinal Cord* 2009; 47: 274-285.
104. Wadsworth BM, Haines TP, Cornwell PL, et al. Abdominal binder improves lung volumes and voice in people with tetraplegic spinal cord injury. *Arch Phys Med Rehabil* 2012; 93: 2189-2197.
105. Wong SL, Shem K, Crew J. Specialized respiratory management for acute cervical spinal cord injury: a retrospective analysis. *Top Spinal Cord Inj Rehabil* 2012; 18: 283-290.

106. Berney SC, Gordon IR, Opdam HI, *et al.* A classification and regression tree to assist clinical decision making in airway management for patients with cervical spinal cord injury. *Spinal Cord* 2011; 49: 244–250.
107. Chevreton JC, Deléamont P. Repeated vital capacity measurements as predictive parameters for mechanical ventilation need and weaning success in the Guillain-Barré syndrome. *Am Rev Respir Dis* 1991; 144: 814–818.
108. Mahanes D, Lewis R. Weaning of the neurologically impaired patient. *Crit Care Nurs Clin North Am* 2004; 16: 387.
109. Ball PA. Critical care of spinal cord injury. *Spine* 2001; 26: S27–S30.
110. Wallbom AS, Naran B, Thomas E. Acute ventilator management and weaning in individuals with high tetraplegia. *Top Spinal Cord Inj Rehabil* 2005; 10: 1–7.
111. Berney S, Stockton K, Berlowitz D, *et al.* Can early extubation and intensive physiotherapy decrease length of stay of acute quadriplegic patients in intensive care? A retrospective case control study. *Physiother Res Int* 2002; 7: 14–22 19p.
112. Epstein SK. Extubation failure: an outcome to be avoided. *Crit Care* 2004; 8: 310–312.
113. Harrop JS, Sharan AD, Scheid EH Jr, *et al.* Tracheostomy placement in patients with complete cervical spinal cord injuries: American Spinal Injury Association Grade A. *J Neurosurg* 2004; 100: Suppl., 20–23.
114. Bach JR. Noninvasive respiratory management of high level spinal cord injury. *J Spinal Cord Med* 2012; 35: 72–80.
115. Hou YF, Lv Y, Zhou F, *et al.* Development and validation of a risk prediction model for tracheostomy in acute traumatic cervical spinal cord injury patients. *Eur Spine J* 2015; 24: 975–984.
116. Berney S, Bragge P, Granger C, *et al.* The acute respiratory management of cervical spinal cord injury in the first 6 weeks after injury: a systematic review. *Spinal Cord* 2011; 49: 17–29.
117. Yugué I, Okada S, Ueta T, *et al.* Analysis of the risk factors for tracheostomy in traumatic cervical spinal cord injury. *Spine* 2012; 37: E1633–E1638.
118. Berney S, Opdam H, Bellomo R, *et al.* An assessment of early tracheostomy after anterior cervical stabilization in patients with acute cervical spine trauma. *J Trauma* 2008; 64: 749–753.
119. Leelapattana P, Fleming JC, Gurr KR, *et al.* Predicting the need for tracheostomy in patients with cervical spinal cord injury. *J Trauma Acute Care Surg* 2012; 73: 880–884.
120. Gutierrez CJ, Harrow J, Haines F. Using an evidence-based protocol to guide rehabilitation and weaning of ventilator-dependent cervical spinal cord injury patients. *J Rehabil Res Dev* 2003; 40: 99–110.
121. Gutierrez CJ, Stevens C, Merritt J, *et al.* Trendelenburg chest optimization prolongs spontaneous breathing trials in ventilator-dependent patients with low cervical spinal cord injury. *J Rehabil Res Dev* 2010; 47: 261–272.
122. Cohn JR. Pulmonary management of the patient with spinal cord injury. *Trauma Q* 1993; 9: 65–71.
123. Brown R, DiMarco AF, Hoyt JD, *et al.* Respiratory dysfunction and management in spinal cord injury. *Respir Care* 2006; 51: 853–870.
124. MacBean N, Ward E, Murdoch B, *et al.* Optimizing speech production in the ventilator-assisted individual following cervical spinal cord injury: a preliminary investigation. *Int J Lang Commun Disord* 2009; 44: 382–393.
125. Hoyt JD, Banzett RB, Brown R. *Ventilator-Supported Speech. Principles and Practice of Mechanical Ventilation.* New York, McGraw Hill, 2008; pp. 1163–1172.
126. Brown DJ, Cameron TS, Donoghue FJ, *et al.* Outcomes of patients with spinal cord injury before and after introduction of an interdisciplinary tracheostomy team. *Crit Care Resusc* 2009; 11: 14.
127. Hoyt JD, Banzett RB, Lohmeier HL, *et al.* Clinical ventilator adjustments that improve speech. *Chest* 2003; 124: 1512–1521.
128. Suiter DM, McCullough GH, Powell PW. Effects of cuff deflation and one-way tracheostomy speaking valve placement on swallow physiology. *Dysphagia* 2003; 18: 284–292.
129. Stelfox HT, Crimi C, Berra L, *et al.* Determinants of tracheostomy decannulation: an international survey. *Crit Care* 2008; 12: R26.
130. Budweiser S, Baur T, Jorres RA, *et al.* Predictors of successful decannulation using a tracheostomy retainer in patients with prolonged weaning and persisting respiratory failure. *Respiration* 2012; 84: 469–476.
131. Marchese S, Corrado A, Scala R, *et al.* Tracheostomy in patients with long-term mechanical ventilation: a survey. *Respir Med* 2010; 104: 749–753.
132. Santus P, Gramegna A, Radovanovic D, *et al.* A systematic review on tracheostomy decannulation: a proposal of a quantitative semiquantitative clinical score. *BMC Pulm Med* 2014; 14: 201.
133. Seidl RO, Nusser-Müller-Busch R, Kurzweil M, *et al.* Dysphagia in acute tetraplegics: a retrospective study. *Spinal Cord* 2010; 48: 197–201.
134. Shem K, Castillo K, Wong SL, *et al.* Dysphagia and respiratory care in individuals with tetraplegia: incidence, associated factors, and preventable complications. *Top Spinal Cord Inj Rehabil* 2012; 18: 15–22.
135. Prigent H, Lejaille M, Terzi N, *et al.* Effect of a tracheostomy speaking valve on breathing-swallowing interaction. *Intensive Care Med* 2012; 38: 85–90.
136. Ross J, White M. Removal of the tracheostomy tube in the aspirating spinal cord-injured patient. *Spinal Cord* 2003; 41: 636–642.
137. Cameron TS, McKinsty A, Burt SK, *et al.* Outcomes of patients with spinal cord injury before and after introduction of an interdisciplinary tracheostomy team. *Crit Care Resusc* 2009; 11: 14–19.
138. Alderson J. Spinal cord injuries. *Care Crit Ill* 1999; 15: 48–52.
139. Carter RE. Experience with ventilator dependent patients. *Paraplegia* 1993; 31: 150–153.
140. Quesnel A, Veber B, Proust F, *et al.* What are the perspectives for ventilated tetraplegics? A French retrospective study of 108 patients with cervical spinal cord injury. *Ann Phys Rehabil Med* 2015; 58: 74–77.
141. Hall KM, Knudsen ST, Wright J, *et al.* Follow-up study of individuals with high tetraplegia (C1–C4) 14 to 24 years postinjury. *Arch Phys Med Rehabil* 1999; 80: 1507–1513.
142. Charlifue S, Apple D, Burns SP, *et al.* Mechanical ventilation, health, and quality of life following spinal cord injury. *Arch Phys Med Rehabil* 2011; 92: 457–463.
143. Oo T, Watt J, Soni B, *et al.* Delayed diaphragm recovery in 12 patients after high cervical spinal cord injury. A retrospective review of the diaphragm status of 107 patients ventilated after acute spinal cord injury. *Spinal Cord* 1999; 37.
144. Burns SP, Weaver FM, Parada JP, *et al.* Management of community-acquired pneumonia in persons with spinal cord injury. *Spinal Cord* 2004; 42: 450–458.
145. Capoor J, Stein AB. Aging with spinal cord injury. *Phys Med Rehabil Clin N Am* 2005; 16: 129–161.
146. Postma K, Bussmann JB, Haisma JA, *et al.* Predicting respiratory infection one year after inpatient rehabilitation with pulmonary function measured at discharge in persons with spinal cord injury. *J Rehabil Med* 2009; 41: 729–733.
147. Raab AM, Krebs J, Perret C, *et al.* Maximum inspiratory pressure is a discriminator of pneumonia in individuals with spinal-cord injury. *Respir Care* 2016; in press [DOI: 10.4187/respcare.04818].
148. Peterson P, Kirshblum S, eds. *Pulmonary management of spinal cord injury.* Lippincott, Williams & Wilkins, Philadelphia, 2002.
149. Saunders LL, Krause JS, Saladin M, *et al.* Prevalence of cigarette smoking and attempts to quit in a population-based cohort with spinal cord injury. *Spinal Cord* 2015; 53: 641–645.
150. Berlowitz DJ, Spong J, Pierce RJ, *et al.* The feasibility of using auto-titrating continuous positive airway pressure to treat obstructive sleep apnoea after acute tetraplegia. *Spinal Cord* 2009; 47: 868–873.
151. Le Guen MC, Cistulli PA, Berlowitz DJ. Continuous positive airway pressure requirements in patients with tetraplegia and obstructive sleep apnoea. *Spinal Cord* 2012; 50: 832–835.
152. Jarosz R, Littlepage MM, Creasey G, *et al.* Functional Electrical Stimulation in Spinal Cord Injury Respiratory Care. *Top Spinal Cord Inj Rehabil* 2012; 18: 315–321.

153. Garner DJ, Berlowitz DJ, Douglas J, *et al.* Home mechanical ventilation in Australia and New Zealand. *Eur Respir J* 2013; 41: 39–45.
154. DiMarco AF. Restoration of respiratory muscle function following spinal cord injury. Review of electrical and magnetic stimulation techniques. *Respir Physiol Neurobiol* 2005; 147: 273–287.
155. Onders RP, Khansarinia S, Weiser T, *et al.* Multicenter analysis of diaphragm pacing in tetraplegics with cardiac pacemakers: positive implications for ventilator weaning in intensive care units. *Surgery* 2010; 148: 893–898.
156. DiMarco AF. Diaphragm pacing in patients with spinal cord injury. *Top Spinal Cord Inj Rehabil* 1999; 5: 6–20.
157. Wolfe LF. Point: should phrenic nerve stimulation be the treatment of choice for spinal cord injury? Yes. *Chest* 2013; 143: 1201–1203.
158. Gay PC. Counterpoint: should phrenic nerve stimulation be the treatment of choice for spinal cord injury? No. *Chest* 2013; 143: 1203–1206.
159. Chiodo AE, Scelza W, Forchheimer M. Predictors of ventilator weaning in individuals with high cervical spinal cord injury. *J Spinal Cord Med* 2008; 31: 72–77.
160. Jackson AB, Grooms TE. Incidence of respiratory complications following spinal cord injury. *Arch Phys Med Rehabil* 1994; 75: 270–275.