Journal of Spine
ISSN: 2165-7939
Kumar, J Spine 2016, S7
DOI: 10.4172/2165-7939.S7-004
Review Article
Open Access

Respiratory Care in Traumatic Spinal Cord Injury

Kumar N*
Consultant in Spinal Injuries and Rehabilitation Medicine, Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, UK

Abstract

This article deals with the respiratory management of acute spinal cord injury patients discussing the mechanics of respiration, assessment and management strategies, respiratory care in the acute stage, invasive mechanical ventilation and weaning including survival following spinal cord injury.

The importance of the care in supine position, physiological instability of the injured cord effect of hypoxia and hypovolaemia is also discussed.

A review of relevant literature has been done to try and answer whether early mobilization following acute spinal cord injury is better than active physiological conservative care including slow weaning, reflecting the ethos of treatment for these problems at Oswestry.

Keywords: Trauma; Spinal cord injury; Respiratory care; Ventilation; Weaning; Survival; Life expectancy

Introduction

Spinal injuries without neurological damage have little effects on respiratory function unless associated with injury to the chest wall. Early verticalisation/mobilisation of these patients are safe and likely to improve vital capacity.

Spinal injury with cord damage (SCI) has a profound effect on the mechanics of respiration and on respiratory function particularly in cervical cord injuries. Early mobilisation of patients with high thoracic and cervical cord injuries especially during the stage of spinal shock is likely to cause further reduction in vital capacity added morbidity [1,2]. Respiratory complications are the leading cause of morbidity and death after SCI [3-5]. The degree of respiratory dysfunction depends on pre-existing pulmonary status, the level of SCI, and any associated chest wall or lung injuries as well as on the quality of the management of the physiologically impaired respiratory functions. The more rostral and complete the damage to the spinal cord, the greater the likelihood of major respiratory impairment. The impact of spinal shock on respiratory function following acute spinal cord injury can be severe necessitating a transient need for an artificial airway and mechanical ventilatory assistance. As spinal shock resolves the flaccid paralysis of the chest wall muscles is replaced by spasticity. The chest wall becomes rigid with loss of compliance, while the abdomen is hypercompliant, both contributing to the reduction in tidal volume in the sitting posture resulting in an improvement in respiratory function particularly during inspiration [6]. Additionally, pulmonary function may be impaired in SCI due to the loss of ventilatory muscle function from denervation, concomitant lung injuries such as pneumothorax; haemothorax; or pulmonary contusion and decreased central ventilatory drive that is associated with head injury or the effects of alcohol and drugs.

Around 40% of spinal cord injuries occur in the cervical spine, a trend that is steadily increasing, with respiratory causes being responsible for death in over 20% of individuals [7]. Loss of lung volumes and relative hypoxemia contribute to global hypoxaemia, exacerbating cord ischaemia in the acute period [7-11]. Respiratory compromise results in the loss of muscle strength generation capacity and reduced lung volumes and in particular vital capacity, of up to 70% ineffective cough and secretion clearance abilities [7-11]; reductions in both lung and chest wall compliance and an additional oxygen cost of breathing due to changes in respiratory mechanics, with obstructive sleep apnoea evident in over 50% of acute tetraplegics [12].

While some countries have specialist spinal Centres to manage such catastrophic trauma with a demonstrable improvement in health outcomes attributed to their contribution [13], many individuals are initially admitted to local hospitals where healthcare professionals are less likely to fully appreciate the significant and continued vulnerabilities of such individuals. This article is aimed at providing a basic understanding of the causes and identification of the main principles of the respiratory management strategies required to maintain pulmonary health for cervical SCI patients during the initial and early post trauma phase.

Respiratory Mechanics

Individuals with spinal cord injury exhibit reduced lung volumes and flow rates as a result of respiratory muscle weakness. These features have been investigated in relation to the combined effects of injury level and posture. Supine values of forced vital capacity and forced expiratory volume in 1s (FEV 1) were repeatedly and consistently shown to be larger in recumbence compared with the seated posture [14-17].

Early mobilisation of patients with spinal neural tissue injury is associated with a reduction of vital capacity and a potential drop of oxygen saturation and/or postural hypotension. Individually or in combination these may further impair cord functions. The tetraplegic and high paraplegic patient's ability to cough is markedly impaired. It is more difficult to get rid of bronchial secretions with assisted coughing against gravity than when patients are in recumbence.

Complete injuries above the mid thoracic region will result in loss of the major respiratory muscle groups for both inspiration and expiration and thus an inability to either fully aerate the lungs or to clear pulmonary secretions, resulting in major vulnerabilities toward pulmonary collapse and infection. Intercostal and abdominal muscle paralysis result in paradoxical chest wall motion i.e., the thorax is pulled in while the hyper compliant abdomen moves out; and loss of diaphragmatic excursion through the zone of apposition [18]. The upright sitting posture results in lower lung volumes than supine

***Corresponding author:** Naveen Kumar, Consultant in Spinal Injuries and Rehabilitation Medicine, Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, UK, Tel: +44 1691 404000; E-mail: Naveen.Kumar@rjah.nhs.uk

Received February 25, 2016; **Accepted** March 08, 2016; **Published** March 10, 2016

Citation: Kumar N (2016) Respiratory Care in Traumatic Spinal Cord Injury. J Spine S7: 004.doi:10.4172/2165-7939.S7-004

lying since the diaphragm loses its ability to generate the same force of contraction [14-19].

Thus while somewhat counterintuitive to the respiratory clinician, the supine position should be adopted in times of respiratory compromise and throughout the process of weaning from mechanical ventilation in complete cord lesions. The use of abdominal binders applied over the lower ribs and abdomen, is common practise in the specialist Centres for use in the upright position [20], improving the VC by as much as 0.32 litres [21]. This application may in borderline cases, offset the need for respiratory support.

Assessment and Management Strategies

The clinical assessment of pulmonary function in acute spinal cord injury begins with a careful history regarding respiratory symptoms and a review of underlying cardiopulmonary co-morbidity such as chronic obstructive pulmonary disease or heart failure. Evaluation also includes respiratory rate, chest wall expansion, abdominal wall movement, and force of cough, chest, limbs and other associated injuries according to a detailed secondary survey. Arterial blood gas analysis and pulse oximetry are especially useful because the bedside diagnosis of carbon dioxide (CO_2) retention or hypoxia may be difficult.

Atelectasis and pneumonia pose significant morbidity and are reported in 40-70% of cases. Respiratory assessment should be vigilant, simple and repeated frequently at the bedside to warn of impending or frank respiratory failure. Aggressive respiratory management has been advocated for the prevention and treatment of pulmonary complications and has been associated with improved outcomes [3,22,23].

As a minimum, the Vital Capacity (VC), Respiratory Rate (RR) and oxygen saturation (SaO_2) should all be monitored regularly, and their trends considered, preferably with arterial blood gases (ABG's) performed at frequent intervals during the first few weeks post injury. The initial reduction of VC in the acute phase will increase steadily within the first five weeks post injury [8]. While a reduction in VC to 10 ml/kg body weight is accepted, further reductions due to loss of compliance or increased resistance (e.g. atelectasis and/or infection), will cause rapid deterioration while a peak cough flow rate (which reduces with lower VCs) of at least 1601/s is essential to shear mucus along the airway walls, for airway clearance [24]. The provision of assisted cough to increase the mucus clearance ability [25], either manually or mechanically is vital in reducing the risk of pulmonary complications and subsequent respiratory failure.

A VC < 700 ccs may be inadequate to sustain spontaneous breathing and is a major indicator to provide ventilatory support. Prompt support with Non Invasive Ventilation (NIV) may enable the avoidance of invasive tracheal intubation in acute SCI [26]. However, halo fixation may pose particular difficulties with mask fitting for NIV therapy while other risks associated with NIV in a non-specialised spinal Centre include the ASCI patient being susceptible to profound and rapid desaturation, silent fall into respiratory failure, paralytic ileus, and risk of air swallowing with an increased risk of acute vomiting and aspiration. The loss of arm and hand function must not be forgotten when selecting the NIV interface.

Neurological deficits may be asymmetrical so the all-important diaphragm should be considered as two separate halves. Paralysis of a single hemi-diaphragm in a complete cervical spine injury, which may go unnoticed by the untrained eye, may require longer term or at least part-time respiratory support, since all intercostal and abdominal muscle activity will be lost. (There has been identified more recently a crossed phrenic nerve pathway thought able to support the contralateral diaphragm, though the clinical implications of this have not yet been fully explored [27]). Where diaphragm function is uncertain, more detailed assessment in the form of fluoroscopic screening [28], M mode ultrasound [29] and surface EMG are all useful assessment tools.

Aspiration poses a significant risk in the tetraplegic patient. Kirshblum et al., [30] studied 187 acute SCIs. Forty two patients had signs of aspiration with video fluoroscopic confirmation in 31 of these. Kirshblum's independent predictors of dysphagia by VFSS were tracheostomy tube at the time of admission, recent cervical spine surgery particularly with an anterior approach, and age. Clinically, aspiration often goes unnoticed but may present as repeated respiratory infections or repeated/persistent lobar collapses. Assessment of swallowing with speech and language therapist input is vital, as salivary and/or food aspiration can have a major detrimental impact upon respiratory health and complicate the ongoing management. Medications should be reviewed due to the effects of some, on muscle fibres e.g. corticosteroids and lipid lowering agents. The profound psychological impact of denying oral intake in the medium and/or longer term, in a high SCI individual should not be overlooked.

Respiratory Care in the Acute Stage

An Oswestry experience of respiratory management in self-ventilating tetraplegia patients.

The Midland Centre for Spinal Injuries is one of twelve tertiary specialised spinal injury Centres within the United Kingdom. This 44 bedded centre is dedicated to the specialist care for patients with spinal injury and provides holistic acute management, comprehensive rehabilitation and lifelong care for those living with spinal cord injuries. The Centre caters to a wide geographic area including the West Midlands, north and mid-Wales and the south of the North West region (Cheshire) – a population of the order of approximately 10 million people. Approximately 120 'new' SCI patients are admitted each year.

As a preventative measure, the Centre has an intensive management programme of respiratory care. This includes three hourly high side turns on a mechanical bed with turning system or manual side turns; regular deep breathing exercises; use of incentive spirometry to optimise lung capacity, assisted coughing for secretion clearance; use of non-invasive biphasic positive airway pressure (BIPAP) as a routine prophylactic treatment for improving lung capacity and preventing atelectasis rather than as a mechanism for assisted ventilation. A Cough Assist machine is also used in selected patients. In later stages inspiratory training is used with the Train Air, which is a computer programme linked to an inspiratory mouthpiece. High tetraplegic patients use this as part of their gymnasium routine like a paraplegic would use the weights machines. The biggest result is increased voice projection. Close monitoring of respiratory function is also carried out relying on respiratory rate, pulse oximetry, regular use of micro spirometer to record vital capacity, monitoring peak flow where relevant and arterial blood gas analysis. Care is also taken to ensure adequate hydration and all oxygen delivered is humidified.

Almost all patients with a SCI are offered an active physiological conservative management for their spinal cord injury with a period of recumbence for about 6 weeks.

A previous internal audit in 2007 had looked into the respiratory complications in tetraplegic patients before and after transfer between 2003 and 2004 to this Centre. This had showed that such preventative measures were successful but identified certain areas to improve.

The re-audit was a retrospective study looking into the respiratory

Frankel grade	High Cervical (C1 – C4) (n=50)	Lower Cervical (C5-T1) (n=55)
A	6	18
B	16	16
C	62	37
D	16	29

Table 1: Showing the neurological level and density by Frankel grade.

complications amongst all acute tetraplegic patients admitted over three year period between 2007 and 2009. Patient and injury demographics, respiratory complications (i.e., pneumonic consolidation, collapse or atelectasis, pulmonary embolism, effusion) before and after admission to the Centre, method of management of the spinal column injury, the respiratory management and changes in vital capacity were reviewed. 105 patients with a tetraplegia Frankel Grade A to D was included. 73 were males and 32 were females. The mean age was 51 years (at the previous audit this was 44 years) with 31% over 65 years of age (Table 1).

Mean delay in transfer from the referring hospitals to the Centre was 24 days (although almost 25% were admitted within one week of injury and approximately 48% within two weeks). 28% (29/105) had a respiratory complication prior to transfer to the Centre, majority of which was pneumonia /consolidation (90%). 48% of those with a respiratory complication (14/29) had required a period of invasive ventilation prior to transfer.

All patients had undergone the same preventative management programme of respiratory care. Of the 20 patients who did develop a respiratory complication after transfer to the Centre, eleven had already had respiratory complication prior to their transfer. Only 9 out of 105 patients had a 'new' respiratory problem (5 infections, 4 of whom required antibiotic therapy; whilst pulmonary embolus, postoperative period of ventilation following a gastro-intestinal surgery, pulmonary effusion and a pneumothorax accounted for the remaining cases). This study had shown that respiratory complications are potentially preventable in self-ventilating tetraplegic patients with a comprehensive management programme.

Invasive Mechanical Ventilation, Weaning and Life Expectency

The likelihood of tracheostomy requirement for ventilation post-surgical fixation [31] is increasingly common outside the specialist Centres. When diaphragm function is lost, invasive mechanical ventilatory support is essential, though recovery has been seen to occur as late as 24 months post injury [32]. Regardless of the timing, the method of ventilation for SCI patients requires larger tidal volumes [33] (at least 10-15 mls/kg), to ensure effective aeration of the lung bases and avoidance of atelectasis and infection. This is well tolerated by SCI patients, with no known evidence to demonstrate pulmonary damage in the absence of acute lung injury. The effect of large volume ventilation is that of respiratory alkalosis, with no long term detrimental effect from this [34]. Electrolyte monitoring in the acute stabilisation phase is required.

The discontinuation of mechanical ventilatory support is likely to take some weeks to achieve. Consistent factors underpinning successful weaning after spinal cord damage have been attributed to accurate neurological assessment; prevention of pulmonary atelectasis by regular and frequent respiratory physiotherapy; ventilator free breathing (VFB) graduated according to VC; rest periods with controlled ventilation; cuff deflation allowing translaryngeal air flow, and regular tracheostomy tube changes [35]. It may be useful to highlight the significant incidence of sleep apnoea (both central and obstructive in nature) in tetraplegia immediately post injury [36,37] which increases over time [38], as this is likely to complicate the respiratory picture and even delay weaning if unrecognised.

Watt et al. compared the long-term survival of 262 patients who were having mechanical ventilation on discharge from a single Spinal Injury Centre with the cohort who had been weaned from mechanical ventilatory support prior to discharge, and examined the causes of death and contributory factors. Mean survival was better amongst weaned compared to ventilated patients [39]. The survival from initial ventilation was poor for the older age group, and for the middle age group who remained on ventilation. Patients with any comorbidity had substantially poorer survival. Groups defined by the AIS scale did not differ strongly, and survival did not differ significantly by neurological level. Pre-existing comorbidities increased the mortality rate by 3.3 [40].

Conclusions

In summary acute SCI may be one of the most devastating acute conditions with respiratory dysfunction providing a major cause of mortality and morbidity; the level and completeness of injury being major determinants of the extent of respiratory dysfunction. Other concomitant injuries and co-morbidities not incorporated here will have further detrimental impacts. Spinal cord injuries are often admitted to a local hospital or trauma centre, so early referral and consultation to a specialist centre when available, where improved health outcomes are achieved, is of paramount importance. Good respiratory health is more likely by ensuring full aeration of the lungs, with proactive chest clearance regimens and monitoring in the acute stage (though vulnerabilities are lifelong). This will also reduce the likelihood of secondary hypoxic cord damage. The minimum basic strategies with complete lesions should include the adoption of large volume ventilation while ventilator-dependant; the supine lying position for maximal spontaneous tidal volume exchange and throughout the weaning process; the monitoring of the vital capacity and use of an abdominal binder when upright. Advice, guidance and support from the local tertiary spinal centre should be sought as soon as cord damage is suspected/realised, to ensure the best management strategies are utilised from the outset for all systems and aspects of care.

Acknowledgements

The author would like to thank Sue Pieri-Davies, Prof W El Masri, Mr A Osman and Mr J R Chowdhury, for their guidance and support without which the publication of this review article would not have been possible.

References

1. El Masri WS (2010) Management of traumatic spinal cord injuries: current standard of care revisited ACNR 10: 37-40.
2. El Masry WS (1993) Physiological instability of the injured spinal cord paraplegia. Paraplegia 31: 273-275.
3. Claxton R, Wong D, Chung F, Fehlings M (1998) Predictors of hospital mortality and mechanical ventilation in patients with cervical spinal cord injury. Can J Anaesth 45: 114-149.
4. Jackson AB, Groomes TE (1994) Incidence of respiratory complications following spinal cord injury. Arch Phys Med Rehabil 75: 270-275.
5. Winslow C, Bode RK, Felton D, Chen D, Meyer PR Jr. (2002) Impact of respiratory complications on length of stay and hospital costs in acute cervical spine injury. Chest 121: 1548-1554.
6. Estenne M, De Troyer A (1986) The effects of tetraplegia on chest wall statics. Am Rev Respir Dis 134: 121-124.
7. Mansel JK, Norman JR (1990) Respiratory complications and management of spinal cord injuries. Chest 97: 1446-1452.
8. Ledsome JR, Sharp JM (1981) Pulmonary function in acute cervical cord injury. Am Rev Respir Dis 124: 41-44.

9. Lu K, Lee TC, Liang CL, Chen HJ (2000) Delayed apnea in patients with mid-lower cervical spinal cord injury. Spine 25: 1332-1338.

10. McMichan JC, Michel L, Westbrook PR (1980) Pulmonary dysfunction following traumatic qadriplegia: Recognition, prevention, and treatment. JAMA 243: 528-531.

11. Reines HD, Harris RC (1987) Pulmonary complications of acute spinal cord injuries. Neurosurgery 21: 193-196.

12. Berlowitz DJ, Brown DJ, Campbell DA, Pierce RJ (2005) A longitudinal evaluation of sleep and breathing in the first year after cervical spinal cord injury. Arch Phys Med Rehabil 86: 1193-1199.

13. Smith M (2002) Efficacy of specialist versus non-specialist management of spinal cord injury within the UK. Spinal Cord 40: 10-16.

14. Cameron GS, Scott JW, Jousse AT, Botterell EH (1955) Diaphragmatic respiration in the quadriplegic patient and the effect of position on his vital capacity. Ann Surg 141: 451-456.

15. Baydur A, Adkins RH, Milic-Emili J (2001) Lung mechanics in individuals with spinal cord injury: effects of injury level and posture. J Appl Physiol 90: 405-411.

16. Alvisi V, Marangoni E, Zannoli S, Uneddu M, Uggento R, et al. (2012) Pulmonary function and expiratory flow limitation in acute cervical spinal cord injury. Arch Phys Med Rehabil 93: 1950-1956.

17. Morgan MDL, Silver JR, Williams SJ (1986) The respiratory system of the spinal cord patient. Management of spinal cord injury. Baltimore: Williams and Wilkins 78-117.

18. Winslow C, Rozovsky J (2003) Effect of spinal cord injury on the respiratory system. Am J Phys Med Rehabil 82: 803-814.

19. Estenne M, De Troyer A (1987) Mechanism of the postural dependence of vital capacity in tetraplegic subjects. Am Rev Respir Dis 135: 367-371.

20. Goldman JM, Rose LS, Williams SJ, Silver JR, Denison DM (1986) Effect of abdominal binders on breathing in tetraplegic patients. Thorax 41: 940-945.

21. Wadsworth BM, Haines TP, Cornwell PL, Paratz JD (2009) Abdominal binder use in people with spinal cord injuries: a systematic review and meta-analysis. Spinal Cord 47: 274-285.

22. McMichan JC, Michel L, Westbrook PR (1980) Pulmonary dysfunction following traumatic quadriplegia. Recognition, prevention, and treatment. JAMA 243: 528-531.

23. Wallbom AS, Naran B, Thomas E (2005) Acute ventilator management and weaning in individuals with high tetraplegia. Top Spinal Cord Inj Rehabil 10: 1-7.

24. Bach JR, Saporito LR (1996) Criteria for extubation and tracheostomy tube removal for patients with ventilatory failure. A different approach to weaning. Chest 110: 1566-1571.

25. Jaeger RJ, Turba RM, Yarkony GM, Roth EJ (1993) Cough in spinal cord injured patients: Comparison of three methods to produce cough. Arch Phys Med Rehabil 74: 1358-1361.

26. Tromans AM, Mecci M, Barrett FH, Ward TA, Grundy DJ (1998) The use of the BiPAP biphasic positive pressure airway system in acute spinal cord injury. Spinal Cord 38: 481-484.

27. Zimmer MB, Nantwi K, Goshgarian HG (2007) Effect of spinal cord injury on the respiratory system; basic research and current clinical treatment options. J Spinal Cord Med 30: 319-330.

28. Simon G, Bonnell J, Kazantzis G, Waller RE (1969) Some radiological observations on the range of movement of the diaphragm. Clin Radiol 20: 231-233.

29. Lloyd T, Tang YM, Benson MD, King S (2006) Diaphragmatic paralysis: the use of M mode ultrasound for diagnosis in adults. Spinal Cord 44: 505-508.

30. Kirshblum S, Johnston MV, Brown J, O'Connor KC, Jarosz P (1999) Predictors of dysphagia after spinal cord injury. Arch Phys Med Rehabil 80: 1101-1105.

31. Hassid VJ, Schinco MA, Tepas JJ, Griffen MM, Murphy TL, et al. (2008) Definitive establishment of airway control is critical for optimal outcome in lower cervical spinal cord injury. J Trauma 65: 1328-1332.

32. Oo T, Watt JWH, Soni MB, Sett PK (1999) Delayed diaphragm recovery in 12 patients after high spinal cervical cord injury. A retrospective review of the diaphragm status of 107 patients ventilated after acute spinal cord injury. Spinal Cord 37: 117- 122.

33. McMichanJC, Michel L, Westbrook PR (1980) Pulmonary dysfunction following traumatic quadriplegia : Recognition, prevention, and treatment. JAMA 243: 528-531.

34. Watt JP, Silva P (2001) Respiratory alkalosis and associated electrolytes in long-term ventilator dependent persons with tetraplegia. Spinal Cord 39: 557-563.

35. Atito-Narh E, Pieri-Davies S, Watt JWH (2008) Slow ventilator weaning after cervical spinal cord injury. IJIC 18: 13-19.

36. Lu K, Lee TC, Liang CL, Chen HJ (2000) Delayed apnea in patients with mid- to lower cervical spinal cord injury. Spine (Phila Pa 1976) 25: 1332-1338.

37. Bach JR, Wang TG (1994) Pulmonary function and sleep disordered breathing in patients with traumatic tetraplegia. A longitudinal Study. Arch Phys Med Rehabil 75: 279-284.

38. Müller G, de Groot S, van der Woude L, Hopman MT (2008) Time-courses of lung function and respiratory muscle pressure generating capacity after spinal cord injury: a prospective cohort study. J Rehabil Med 40: 269-276.

39. Watt JW, Wiredu E, Silva P, Meehan S (2011) Survival after short- or long-term ventilation after acute spinal cord injury: a single-centre 25-year retrospective study. Spinal Cord 49: 404-410.

40. Hutton JL, Watt JH, Wiredu E (2012) Letter commenting on 'Survival after short- or long-term ventilation after acute spinal cord injury: a single-centre 25-year retrospective study'. Spinal Cord 50: 859-860.

This article was originally published in a special issue, **Spinal Cord Injury Rehabilitation** handled by Editor(s). Dr. Alessandro Landi, University of Rome, Italy