Baclofen Pump Intervention for Spasticity Affecting Pulmonary Function

Deanna Britton, MS, CCC-SLP, BC-NCD¹; Barry Goldstein, MD, PhD¹⁻³; Jill Jones-Redmond, MS, CCC-SLP^{1,4}; Peter Esselman, MD^{1,2}

¹Harborview Medical Center, Seattle, Washington; ²Department of Rehabilitation Medicine, University of Washington, Seattle, Washington; ³Spinal Cord Injury & Disorders Strategic Health Care Group, Veterans Administration Puget Sound Health Care System, Seattle, Washington; ⁴Overlake Medical Center, Bellevue, Washington

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Abstract

Introduction: Muscle spasticity may adversely affect pulmonary function after spinal cord injury (SCI). However, there is limited information regarding the treatment of spasticity as a determinant of pulmonary function. This study presents the case of a man with C4 tetraplegia who had severe spasticity and difficulty weaning from ventilatory support.

Methods: Case presentation.

Results: Severe spasticity likely contributed to respiratory compromise in this patient. Successful and rapid weaning from the ventilator occurred within 3 weeks of baclofen pump placement.

Conclusions: Randomized clinical trials among SCI patients with significant spasticity are needed to determine whether intervention with a baclofen pump facilitates earlier ventilatory weaning.

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Key Words: Spinal cord injuries; Spasticity; Ventilator weaning; Baclofen pump; Respiratory impairment; Tetraplegia

INTRODUCTION

Despite the overall decline in mortality for patients with spinal cord injury (SCI), pulmonary complications are a leading cause of mortality in patients in the acute and chronic phase after tetraplegia (1). Respiratory impairments after cervical SCI are caused by compromise of the respiratory muscles, such as the diaphragm, abdominal, and intercostal muscles. These changes result in decreased vital capacity, diminished cough strength, and increased risk of atelectasis, pneumonia, hypoxemia, and sleep disorders. Skeletal deformities and autonomic changes also contribute to a decline in pulmonary function after SCI (2).

Spasticity may also adversely affect pulmonary function. Silver & Lehr (3) described 3 patients with C4 and C5 SCI who experienced dyspnea associated with diaphragmatic spasticity. Spasticity of truncal muscles (intercostals and abdominals) has been reported to

impair pulmonary function. For instance, Laffront et al (4) described a patient with a C4 American Spinal Injury Association (ASIA) grade A SCI whose breathlessness was attributed to spastic abdominal contractions. However, there is little information regarding the treatment of spasticity as a determinant of pulmonary function after SCI. Treatment of spasticity with enteral medications may have limited effectiveness and may have side effects not tolerated by patients (5). The use of intrathecal baclofen is often an effective alternative to oral medications for the treatment of severe spasticity (6,7).

Ventilator weaning is often challenging for persons with high-level tetraplegia because of multiple factors. Respiratory failure results from decreased vital capacity, arterial hypoxemia and hypercarbia, pneumonia, atelectasis, and sleep disorders, and may present acutely after a SCI, but may also present years after the injury (8,9).

We describe a patient with C4 tetraplegia who had severe spasticity and difficulty weaning from ventilatory support until a baclofen pump was implanted for treatment of spasticity.

CASE REPORT

A 55-year-old man was injured in an accident resulting in C4 ASIA grade A tetraplegia, requiring ventilatory

Please address correspondence to Deanna Britton, MS, CCC-SLP, BC-NCD, Speech-Language Pathology Clinical Specialist, Harborview Medical Center, 325 Ninth Avenue, Box 359819, Seattle, WA 98104; phone: 206.731.2409 (e-mail: deanna@u.washington.edu).

support. He was admitted to an inpatient rehabilitation unit approximately 2 weeks after injury, at which time he was ventilator dependent with tracheostomy. He was not yet tolerating tracheostomy cuff deflation because he experienced episodes of bradycardia associated with cuff deflation. He was initially communicating via mouthing, yes/no responses with eye blinks, and use of an alphabet board. The patient's initial ventilation settings were FiO_2 at 40%, positive end expiratory pressure (PEEP) of 5, tidal volume of 1.5 L, and respiratory rate of 8 per minute.

Tolerance of cuff deflation was one of the initial goals for purposes of eventual ventilator weaning, communication, and swallowing. The speech pathologist, respiratory therapist, and physiatrist developed a program toward this goal. He tolerated progressively increased times of cuff deflation and use of "leak speech" coordinated with ventilatory (primarily inspiratory) phases for communication. With full-cuff deflation trials, he was taught to volitionally close his glottis as needed to maintain O_2 saturation by redirecting ventilator air to his lungs. Once the patient was consistently tolerating cuff deflation for a half hour, he participated in a swallowing evaluation and began eating; subsequently, he quickly progressed to a regular diet.

Six weeks after initiation of cuff deflation, the patient had developed tolerance for a cuffless tracheostomy tube, and was continuing with a regular diet without signs or symptoms of aspiration pneumonia. T-piece trials, use of a speaking valve, and training with glosso-

pharyngeal breathing (GPB) began approximately 2 months after injury. GPB, a compensatory means of taking additional air into the lungs, consists of using the oral and pharyngeal structures to trap air into the oral cavity and pump it into the lungs (10,11). The patient's baseline pulmonary measures during GPB training included: vital capacity of 0.77 L, negative inspiratory force of -30, and a tidal volume of 450 L. He spoke with a low volume off the ventilator and had a weak cough. The patient demonstrated consistent vital capacity gains with use of GPB (Figure 1). The patient was also instructed on use of manual coughing techniques. Functional electrical stimulation was not attempted. During the next 6 weeks, the patient gradually weaned off ventilator support and did not need supplemental oxygen.

Subsequently, there were medical complications that resulted in further respiratory impairments. For this reason, the patient was placed back on the ventilator. In addition to symptoms of respiratory compromise, he presented with an earache, sore throat, and dysphagia, and was subsequently treated for oral candidiasis. During this same period, he developed severe spasms and spasticity of the lower extremities and trunk. When he had truncal spasms, he complained of difficulty breathing, shortness of breath, air hunger, insomnia, difficulty speaking, dysphagia, and head and neck pain. These symptoms progressively worsened over the next 2 months, despite aggressive treatment with antispasticity medications (20 mg of baclofen 4 times daily; 5 mg of diazepam once in the

Figure 1. Vital capacity (with and without GPB) before and after baclofen pump. The baclofen pump did not significantly affect vital capacity.

Figure 2. Number of hours per day tolerated without the ventilator. Despite no significant change in vital capacity, the baclofen pump appears to affect the patient's endurance with ventilator weaning.

morning and 20 mg every evening; and 8 mg of tizanidine every 6 hours). During this time, approximately 6 months after injury, the patient was able to participate with ventilator weaning during the day, typically ranging from 10 to 15 hours/day, but was still unable to tolerate being off the ventilator at night.

Approximately 6 months after injury, a baclofen pump was implanted between the L4-L5 spinous processes, with the tip terminating at T6-T7. The initial baclofen pump dosage was 150 µg/d. The dosage was gradually increased to 240 µg/d over the next month. After this procedure, symptoms of pain, insomnia, and spasticity were alleviated. Three weeks after baclofen pump implantation, the patient weaned from mechanical ventilation and has remained off the ventilator to date (Figure 2), for more than 3 years.

Management of pulmonary secretions and swallowing discomfort remained problematic after weaning from mechanical ventilation. The patient began using an in-exsufflator (12,13) as a means to improve clearance of secretions. Similar to previously documented reports (14), he reported that this method was superior to suctioning for removal of his secretions. He was subsequently decannulated, approximately 1 month after ventilator weaning. After decannulation, the patient's swallowing and secretion management improved significantly.

Additionally, episodes of sleep apnea were observed immediately after baclofen pump implantation. Thus, the patient completed a sleep study, and was diagnosed with severe obstructive sleep apnea (OSA) with marked sleep disruption and moderate oxygen desaturations. After this diagnosis, the patient was started with nighttime bi-level positive airway pressure (BiPAP) delivered via face mask, per the recommendations of the pulmonary consultant. The patient has since maintained good pulmonary health without the ventilator with use of the in-exsufflator to assist with clearance of secretions and nighttime BiPAP to manage sleep apnea.

DISCUSSION

This case illustrates the potential impact of severe spasticity on respiratory function and ability to wean from the ventilator after cervical SCI. Treatment with intrathecal baclofen therapy to decrease spasticity is one option in the treatment of spasticity that may improve pulmonary function. Clinical indications for intrathecal baclofen therapy include severe spasticity that interferes with function in patients who do not get satisfactory therapeutic results with oral medications (15).

Decreased respiratory function after cervical SCI is caused by compromise of the diaphragm, thoracic intercostal muscles, and abdominal muscles. Paralysis of the chest wall caused by SCI can result in paradoxical chest and abdominal movements during breathing

adding to the respiratory compromise. Outward movement of the abdominal wall and inward movement of the chest wall during inspiration characterize this paradoxical movement (16). In addition, decreased compliance of the pulmonary system probably occurs over time because of the chronically hypoventilated atelectatic lung.

Silver & Lehr (3) reported that dyspnea could be associated with spasms of the diaphragm. Intercostal muscle spasticity may limit chest wall expansion, but it is also reported that increased activity in the intercostal muscles may contribute to improved ventilatory function by decreasing the paradoxical chest and abdominal movement through stabilization of the chest wall (3,17). Other studies have noted electromyographic activity of the intercostal muscles during respiration in individuals with tetraplegia that is probably related to stretch reflexes (18,19). Cooling of the chest wall in an individual with tetraplegia, which results in decreased intercostal muscle spasticity, has been shown to cause an increase in paradoxical motion, causing a decrease in pulmonary function (20). If this is the case, treatment of spasticity with intrathecal baclofen may result in increased paradoxical motion, decreased vital capacity, and reduced ability to functionally use spasms of the abdomen and chest walls to enhance cough. In this case, as shown in Figure 1, the patient's vital capacity decreased after the placement of the baclofen pump and then gradually increased over the next 8 weeks back to the vital capacity reached before the baclofen pump.

Laffont et al (4) reported dyspnea associated with spastic contractions of paralyzed abdominal muscles, resulting in an increased load imposed on inspiratory muscles. In the reported case, we observed that spasticity of trunk muscles appears to have resulted in worsening of pulmonary function by decreasing chest wall compliance and increasing intra-abdominal pressure, reducing diaphragmatic excursion. Oral spasmolytic medications were ineffective and sedating, which may have further exacerbated the patient's respiratory compromise. Concomitant insomnia and pain resulting from severe spasticity further reduced his overall tolerance or endurance for weaning from mechanical ventilation. Given the mid-thoracic placement of the baclofen pump catheter tip, it is likely that the mid-to-lower trunk (intercostal and abdominal muscles) was affected, thereby resulting in decreased intra-abdominal pressures, greater diaphragmatic excursion, and greater compliance of the thoracic wall.

Respiratory compromise may initially present during sleep as chronic alveolar hypoventilation with sleep apnea. Assessment during sleep is important to determine the degree of respiratory compromise. The patient in this case report was diagnosed with OSA after placement of the baclofen pump. OSA may have been present to some degree before the baclofen pump placement, but it was not recognized because of the ongoing mechanical ventilation at night. Sleep apnea is

often present after SCI, but the exact prevalence is not known. Burns et al reports sleep apnea in up to 40% of individuals with SCI (21,22). Flavell et al (23) diagnosed severe nocturnal hypoxic episodes in 3 of 10 individuals with tetraplegia, and Cahan et al (24) reported on 6 of 16 patients with tetraplegia who had normal oxygen saturations during the day and demonstrated desaturation at night. Antispasticity medications may contribute to OSA, but Burns et al (21) reported that, in patients with tetraplegia, the daily oral baclofen dose was not correlated with the presence of sleep apnea.

The manufacturer of the baclofen pump reports that in clinical trials of patients with spasticity of spinal origin, hypoventilation was reported in 0.2% of patients after administration of a test bolus, in 0.8% of patients in the first 2 months after pump placement, and in 2.1% of patients after the initial 2 months. Because of the uncontrolled nature of the clinical trials, a clear association between the hypoventilation and the intrathecal baclofen treatment was not established (15). In a study of 5 individuals with tetraplegia who were monitored with overnight oximetry after intrathecal baclofen therapy, there were no desaturations below 90% and no evidence of sleep apnea (25).

It is interesting to note that the patient's vital capacity and other measures of ventilator weaning parameters (eg, tidal volume, negative inspiratory force, etc) alone did not adequately predict his ability to wean from mechanical ventilation. For example, this patient's vital capacity was consistently greater than 1 L during the period that he had significant difficulty with weaning from the ventilator. Subsequent to baclofen pump placement, endurance with ventilator weaning was achieved despite mildly lower vital capacity measures (Figure 1). Vital capacity, however, was monitored only during the daytime under optimal conditions; it may have been reduced at night when the patient's spasms were more severe.

In the reported case, the patient used several methods to augment his pulmonary function. GPB was used to increase his vital capacity, as shown in Figure 1. Nighttime BiPAP was used to treat sleep apnea and an in-exsufflator was used to assist with clearance of secretions. Additional treatments to consider in this population are the use of nasal-mask positive-pressure assistive ventilation (26), functional electrical stimulation of the abdominal muscles to enhance the patient's cough (27,28), and inspiratory/expiratory muscle training to strengthen the nonaffected or partially affected respiratory muscles (29,30).

CONCLUSIONS

Severe spasticity likely contributed to respiratory compromise and difficulty weaning from the ventilator in this patient. Maximal oral medications were ineffective and possibly contributed to fatigue and respiratory failure during weaning trials. After the baclofen pump in-

tervention, rapid weaning from the ventilator occurred. The patient reported immediate relief of severe spasms, pain, and breathing difficulty after this intervention. We hypothesize that the reduction in spasticity resulted in improved overall muscular endurance for purposes of ventilator weaning. Further investigations are needed to evaluate the effects of spasticity on pulmonary function and response to treatment, including studies comparing intrathecal baclofen with oral medications.

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