Subarachnoid-to-Subarachnoid Shunt for Correction of Nonfunctioning Baclofen Pump in a Severe Case of Chronic Debilitating Post–Spinal Cord Injury Spasticity

Adewale A. Bakare[1], Jonathan Weyhenmeyer[1], Albert Lee[1,2]

Key words
- Intrathecal baclofen
- Multilevel scar tissue
- Neuroablative
- Spasticity
- Spinal cord injury
- Subarachnoid-to-subarachnoid shunt

Abbreviations and Acronyms
CSF: Cerebrospinal fluid
ITB: Intrathecal baclofen
SCI: Spinal cord injury

From the [1]Department of Neurological Surgery, Indiana University School of Medicine, Indianapolis and [2]Goodman Campbell Brain and Spine, Indiana University School of Medicine, Indianapolis, Indiana, USA

To whom correspondence should be addressed: Albert Lee, M.D. [E-mail: alee@goodmancampbell.com]

Citation: World Neurosurg. (2018) 110:26-29. https://doi.org/10.1016/j.wneu.2017.10.127

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

BACKGROUND: **Perhaps the most disabling condition seen in patients with spinal cord injury (SCI) is spasticity. Spasticity is characterized as hyperreflexia and hypertonicity as a result of damage to the supraspinal tracts in the aftermath of SCI. Intrathecal baclofen (ITB) is the mainstay therapy for spasticity unresponsive to oral baclofen. One of the problems associated with post-SCI spasticity unresponsive to ITB is the development of scar tissue that prevents the diffusion of baclofen in the desired spinal cord area. This case offers a unique strategy to deal with multilevel scar tissue.**

CLINICAL PRESENTATION: **This 46-year-old paraplegic male with a T8 SCI whose spasticity had been well managed with ITB therapy for many years recently suffered intractable spasticity necessitating multiple reoperations for a nonfunctioning ITB catheter secondary to extensive scar tissue and intrathecal adhesions. Placement of a subarachnoid-to-subarachnoid shunt eliminated the problem of extensive scar tissue preventing adequate baclofen therapy.**

CONCLUSIONS: **After undergoing multilevel thoracic and lumbar laminectomies with subarachnoid-to-subarachnoid spinal shunt, the patient's spasticity was finally brought under control with adequate daily baclofen infusion. This case demonstrates a creative way to address ITB catheter failure before considering other measures, such as neuroablative procedures (e.g., rhizotomy, myelotomy). This case reinforces the recommendation that ablative procedures, which have far greater complications, should be reserved for patients who have failed medical or other nonablative therapies.**

INTRODUCTION

In 2016, the National Spinal Cord Injury statistical center estimated a prevalence of 54 cases of spinal cord injury (SCI) per 1 million persons in the United States.[1] Damage to the upper motor neurons in SCI results in poor regulation of the alpha motor neuron response to tonic muscle stretch reflexes that are constantly relayed to the spinal cord.[2,3] Subsequently, the alpha motor neurons persistently fire action potentials to opposing muscle groups. As such, damage to the upper motor neuron tracts causes hyperexcitable states that manifest as spasticity, hypertonia, and hyperreflexia.[2-4] Unfortunately, spasticity is an inevitable, chronic neurologic sequela of SCI.[3,5] Patients with SCI in a chronic spastic state experience debilitating quality of life, characterized by pressure ulcers, poor ambulation, wheelchair distress, and impaired activities of daily living.[2]

A number of medications have been shown to improve spasticity caused by upper motor neuron lesions, including baclofen, benzodiazepines, and dantrolene.[2,6] Oral baclofen has long been known to reduce spasticity and hypertonicity, but the dosage required often leads to intolerable adverse effects.[2,7] As a result, intrathecal baclofen (ITB) has become a mainstay in the management of spasticity in patients with SCI and cerebral palsy.

Here we present the case of a patient with spastic paraplegia with extensive scar tissue. The patient's spasticity had been well managed with ITB therapy for many years, but he recently suffered severe unremitting truncal and lower extremity spasticity necessitating multiple reoperations for a nonfunctioning ITB catheter secondary to extensive scar tissue and intrathecal adhesions. We describe a novel approach to dealing with scar tissue to improve patient spasticity due to SCI.

CLINICAL PRESENTATION

This 46-year-old male has a past medical history significant for paraplegia, neurogenic bowel and bladder, and chronic spasticity secondary to severe traumatic brain injury and a T8 SCI sustained in a motor vehicle accident. The patient's spasticity was previously well managed with ITB infusion for almost 20 years. His original intrathecal catheter entered the thecal sac at the L2-3 interspace and was threaded up to T10. Over the last 3 years, the patient presented to our neurosurgery clinic on several occasions with intractable torso and lower extremity spasticity. The persistent spasticity resulted in multiple operations for intrathecal catheter revision. Unfortunately, residual and

increasing spasticity was not relieved by these interventions.

During an ITB catheter revision in early 2015, significant subarachnoid scarring was noted, which was preventing the intrathecal catheter from threading to the desired spinal level. Postoperatively, the patient continued to complain of disabling upper trunk spasticity in the area extending from the xyphoid to the shoulder. Given his persistent symptoms, we elected to pursue an additional operation 3 months later in an attempt to mitigate the increased spasticity.

Before the operation there was concern that an intrathecal catheter extending from the lumbar spine would not be able to be threaded past the arachnoid adhesions and dural scar in the thoracic spine. With this in mind, we elected to perform bilateral laminectomies at T8 and T7, along with a partial laminectomy at T6, to facilitate direct placement of an intrathecal catheter in the thoracic spine. The intraoperative microscope was used to improve visualization to ensure that the catheter was not infiltrating the spinal cord parenchyma. The intraoperative microscope allowed for direct visualization of the old intrathecal catheter and arachnoid scar that had extended from the T8 level to the T9-10 level. We initially attempted to enter the thecal sac at the T8 spinal level and thread the intrathecal catheter rostral to the T6 level. Owing to the extensive scarring, we could not thread the intrathecal catheter higher than the T6 level; thus, we elected to place the catheter at T6 using fluoroscopic X-ray guidance and then ligated the old, nonfunctional lumbar catheter through a separate incision.

Although the patient experienced some relief of spasms in the upper trunk area postoperatively, spasticity worsened in the lower extremities and abdominal area. The lower truncal spasticity was so bad that the patient frequently complained of worsening diarrhea-type symptoms with incomplete evacuation despite an adequate bowel regimen. The patient was started on bolus dosing of ITB, 40 mg of oral baclofen daily, and large scheduled doses of oral valium. His high oral valium intake produced a significant cognitive decline. Unfortunately, however, his lower extremity symptoms persisted. The spasticity continued to be readily apparent on physical examination with lower extremity scissoring. Computed tomography myelography revealed a myelographic block impeding cerebrospinal fluid (CSF) flow (Figure 1), and preventing the ITB from providing adequate coverage in the lower extremities.

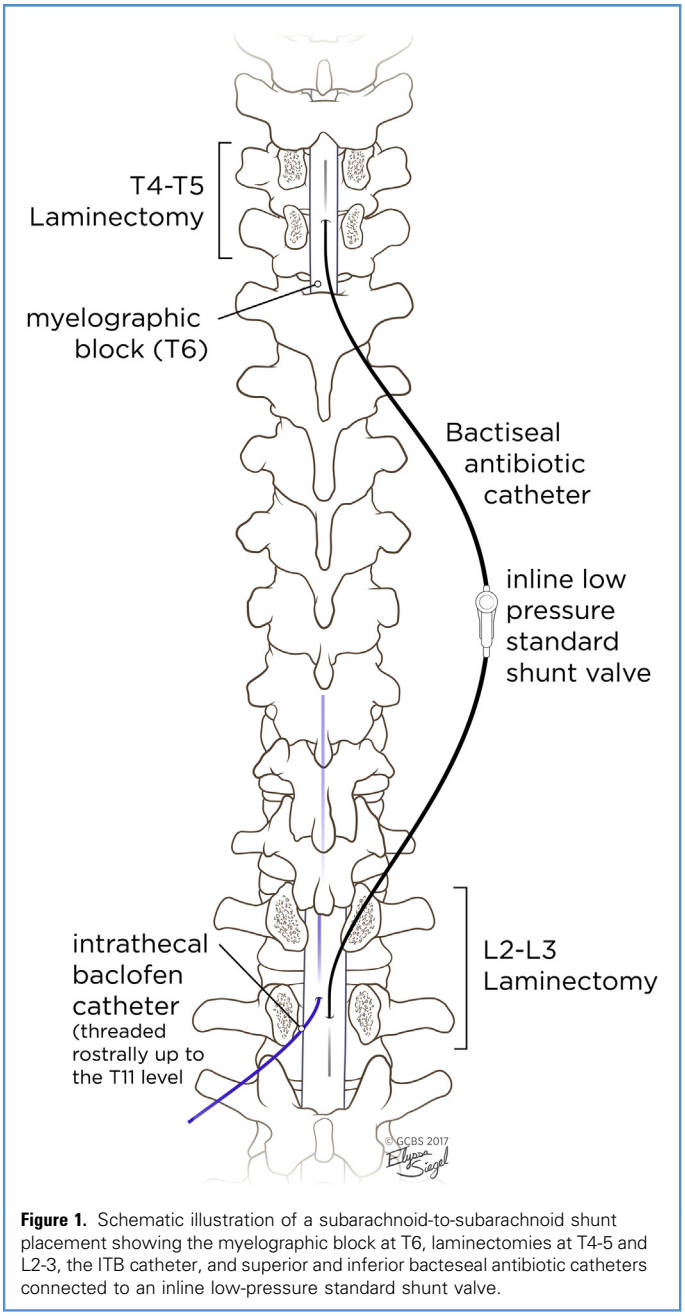

Figure 1. Schematic illustration of a subarachnoid-to-subarachnoid shunt placement showing the myelographic block at T6, laminectomies at T4-5 and L2-3, the ITB catheter, and superior and inferior bacteseal antibiotic catheters connected to an inline low-pressure standard shunt valve.

Given the patient's persistent spasticity, 2 surgical options were discussed over several clinic visits: placement of a second baclofen pump with catheterization of the caudal intrathecal space, or a subarachnoid-to-subarachnoid shunting procedure with a shunt placed between the thoracic and lumbar spinal levels. After extensive consultation, the patient opted for the subarachnoid-to-subarachnoid shunting procedure.

The patient was returned to the operating room in late 2016 for placement of the subarachnoid-to-subarachnoid shunt (Figure 1). The procedure began with a T4-5 laminectomy and an L2-3