# Case report Adductor tenotomy and selective obturator neurectomy for the treatment of spasticity in a man with paraplegia

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**Context:** Excessive hip adductor spasticity in patients with spinal cord injury (SCI) can produce scissoring effect of the thighs which can interfere with various activities of daily living. If noninvasive measures do not produce desired results, surgical treatment may be considered. One surgical option for the management of adductor spasticity includes adductor tenotomy and selective obturator neurectomy. This procedure was performed in an individual with T11 paraplegia having severe adductor spasticity in both thighs. **Method:** Case report.

**Findings:** Adductor spasticity at hips was identified as the main barrier in functional activities and rehabilitation of our patient. After a trial of noninvasive treatment, a selective obturator neurectomy and adductor tenotomy were carried out, which completely relieved his adductor spasticity at both thighs without any complications. With comprehensive rehabilitation, the patient showed enhanced functional independence in various activities of daily living. This helped the patient to achieve functional indoor ambulation using orthosis in spite of complete paraplegia.

**Conclusion/clinical reference:** Severe adductor spasticity can have debilitating effects and could be a major barrier in rehabilitation of patients with SCI. Surgical treatment for adductor spasticity can be considered when patients are unresponsive to noninvasive treatment. Relief from adductor spasticity can improve functional outcomes and decrease dependency. Although this procedure is commonly performed in patients with cerebral palsy, it remains a viable surgical alternative in carefully selected patients with SCI when other modes of treatment are contraindicated, failed, or not available.

Keywords: Spinal cord injuries, Paraplegia, Muscle spasticity, Tenotomy, Neurectomy, Rehabilitation

## Introduction

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Adductor spasticity at the hips can have debilitating effects in individuals with spinal cord injury (SCI). It can affect daily activities, interfere with rehabilitation, and increase the burden of care on the caregivers. It is difficult to maintain good perineal hygiene because of the scissoring effects of thighs. Toileting becomes more problematic leading to neglected bladder management. Similarly, inappropriate positioning in the bed or wheel chair promotes formation of pressure ulcers, further incapacitating the patient. Spasticity interferes with transfers and sometimes hampers the ability to ambulate as well, making the patient prone to falls with subsequent fractures. Severe cases may result in hip joint dislocations or subluxations. Sexual intercourse is not easy because of abnormal positioning and spasms of thighs. Daily activities become much more complex as adductor spasticity becomes intractable to noninvasive treatment. This necessitates the consideration of other treatment options.

Patients with spasticity are in general difficult to treat. First-line treatment for spasticity remains noninvasive management (medications and physical therapy). Whereas considering surgical options, we have to clearly define the clinical goals, i.e. increase comfort, decrease pain, improve function and autonomy, and prevent complications.<sup>1</sup> The selection is based on rigorous clinical assessment to determine the role of

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spasticity and to differentiate between disabling and useful spasticity.<sup>1</sup> In patients with SCI, spasticity in adductor muscles of the thighs may be focal spasticity or a manifestation of generalized spasticity depending on level and nature of lesion. Management options vary from patient to patient and selection is guided by the goals for each patient. When only the adductor group of muscles at the hips is affected, this can be treated as focal spasticity.

Focal spasticity is primarily treated with injections of botulinum toxin into the affected muscles and intensive rehabilitation therapy. For patients who do not respond to conventional medical management, peripheral neurotomy is an established treatment option.<sup>2-4</sup> Although clonus and spasticity may be diminished by neurectomy, further orthopedic surgery may be required, e.g. adductor tenotomy with anterior obturator neurotomy to correct spasticity in severe cases of adductor spasticity in patients with cerebral palsy.<sup>5</sup> Similar principles are reflected in treatment of adductor spasticity in patients with SCI.<sup>6,7</sup> The objective of this procedure is to re-equilibrate the tonic balance between the agonist and antagonist muscles at the hip. The adductor group of muscles includes the adductor longus, adductor magnus, adductor brevis, gracilis, and pectineus. The obturator nerve (L2, L3, and L4) leaves the obturator foramen and after entering the thigh, divides into an anterior and a posterior branches. The anterior branch usually supplies the adductor longus, adductor brevis, and gracilis. The adductor magnus receives innervations both from posterior branch of obturator nerve and a branch from tibial division of the sciatic nerve, whereas pectineus is supplied by the femoral nerve. In the past, complete obturator neurectomies resulted in uncontrolled hip abduction contracture due to complete loss of opposing adduction. For this reason complete neurectomy was superseded by selective neurectomy of the anterior branch of obturator nerve, with good results reported in 80% of patients.<sup>8</sup> Hence, denervating the adductor longus, brevis, and gracilis, while innervation remains intact for adductor magnus and pectineus. This procedure is aimed not only to relieve spasticity but to maintain joint stability as well. It is a safe procedure for spasticity management and to improve functional outcomes.9

#### Case description

A 22-year-old man with SCI T11 ASIA "A" was admitted for the treatment of pressure ulcers and comprehensive rehabilitation.<sup>10</sup> A motor vehicle accident 2 years before admission resulted in wedge fracture of T11 causing compressive myelopathy and resulting in complete paraplegia. After spinal fixation, he received short-term physical therapy and was discharged home. One and half years later, he presented with six stage IV pressure ulcers and generalized spasticity of the lower extremities. Over the previous 8 months, he had remained mostly bedridden. He used a wheelchair for long durations without pressure relief and had very poor compliance with medications, exercise, and positioning. He preferred to use an indwelling urinary catheter and had recurrent urinary tract infections. There was no functional improvement during his stay at home and he continued to require assistance in transfers, toileting, and lower body dressing.

Initial assessment revealed multiple stage IV infected pressure ulcers at the sacral area and both lower limbs. Neurological examination showed no sensations below T11 (sensory level) including sacral segments and complete loss of motor strength in both lower limbs. Generalized spasticity in both lower limbs was observed mainly in flexor pattern. He had modified Ashworth grade II spasticity in the hip, knee, and ankle planter flexors and adductor muscle group of both thighs.<sup>11</sup> Range of motion was full at all joints, and upper limb examination was unremarkable.

The patient had a prolonged hospital stay and was treated for infected pressure ulcers and urinary tract infection. He underwent multiple plastic surgeries for treatment of pressure ulcers, which was an obstacle to rehabilitation. He was started on oral anti-spasticity medications and physical therapy as tolerated. Adductor spasticity at both hips increased from grade II to grade III and he also developed bilateral grade II spasticity in the knee extensors. Oral anti-spasticity medications were adjusted till he reached maximum tolerable doses of baclofen (25 mg orally, four times daily) and tizanidine (6 mg orally, thrice daily). Once his pressure ulcers were healed and infections were treated, patient was started on a comprehensive rehabilitation program mainly focusing on active physical and occupational therapy. Severe adductor spasticity at both hips remained a barrier to bed mobility, transfers, bladder management, and lower body dressing. Botulinum toxin was injected in the hip adductors on both sides. A dilution of 1 ml normal saline in each vial (100 units) of botulinum toxin. He received a total of 600 units, 300 units on each thigh at six sites (100 units each for adductor longus, brevis, and magnus on either side).

Over the next 2 months there was no improvement in spasticity or functional level. As a result of marked prominence of the greater trochanters, he remained at a higher risk of developing pressure ulcers of the hips. Owing to lack of response to conservative measures including physical therapy and maximal daily doses of two oral anti-spasticity medications, other options were explored. Percutaneous neurolytic procedures using phenol or ethanol were not possible due to lack of availability of medications and nerve locator equipment. Intrathecal baclofen pump was not considered, as the goal was to achieve good control of spasticity in relevant areas with a relatively low-cost permanent solution without need for frequent visits for refills. As he had a complete paraplegia with adductor spasticity as the main barrier, bilateral selective obturator neurectomy and adductor tenotomy were considered suitable. The objective was to free the adductors from spasticity and increase his independence in activities of daily living. Residual spasticity in other muscles groups might allow standing and pedal mobility with orthosis.

On each side, a groin incision was made near the inguinal ligament in the proximal thigh over the adductor longus tendon. The adductor longus tendon was exposed and sectioned and anterior branch of obturator nerve was identified and a short segment excised. Following surgery, the patient remained on bed rest for 10 days. Postsurgical assessment showed that spasticity was completely relieved in the adductor group of thigh muscles, whereas spasticity in other muscle groups remained unchanged. He underwent intensive physical and occupational therapy starting with three sessions a week and increasing to three sessions twice daily for a total of 7 weeks. Rehabilitation focused on upper limb strengthening and improving balance, endurance, flexibility, and range of motion at all joints. Initially, most activities were aimed to improve self-confidence and functional outcomes in terms of self-care, bed mobility, wheelchair mobility, and transfers. Later bilateral kneeankle-foot orthosis was provided and he was advanced to sit-to-stand training, achieving satisfactory results. Weight-bearing activities continued and he showed good joint stability with improvement in standing activities. Owing to his progress, the patient was stepped up to a gait-training program. With continued therapy he showed improvement in his walking index for SCI from scale 0 to 9. (0 = client is unable to stand and/or participate in assisted walking, 9 = ambulates with walker, with braces and no physical assistance, 10 meters).<sup>12</sup> At the time of discharge, he was totally independent in all the activities of daily living, including transfers and wheelchair mobility. With relief of adductor spasticity at thighs, he was able to perform clean intermittent catheterization independently and he could ambulate for short distances using a walker with knee-ankle-foot orthosis.

### Discussion

Spasticity is a sequela of SCI that may have lifelong effects. Patients with posttraumatic spasticity have high incidence of problems such as flexion contractures, decubitus ulcers, and poor perineal hygiene because of adductor spasms.<sup>13</sup> Botulinum toxin is touted as the pharmacological treatment of first choice for focal spasticity.<sup>14</sup> Chemical neurolysis with alcohol or phenol is frequently used to manage spasticity, but is associated with the risk of neuralgia, deafferentation pain, and unintentional soft-tissue injury secondary to neurolytic agent spread to adjacent tissues.<sup>15</sup> If failure of noninvasive pharmacological treatments and botulinum toxin, focal spasticity can be treated with peripheral neurectomy.<sup>1</sup> Neurectomy involves cutting the nerve branches as they enter the target muscle. The technique is irreversible as there is no fiber discrepancy at the periphery. When a peripheral nerve is cut, not only the sensory, but also the motor neurons are cut. For this reason, it must be done as distally as possible.<sup>1</sup> Ablative procedures, although technically more challenging, are still effective procedures to consider when treating patients with severe spasticity.<sup>13</sup> One of the main indications for peripheral neurectomy is spastic hip.<sup>1,16,17</sup> Selective neurectomy of anterior branch of obturator nerve can be supplemented with adductor tenotomy. In this case, adductor spasticity was identified as the main barrier in rehabilitation. After a trial of noninvasive treatment, surgical treatment of spasticity benefitted the patient and he showed significant improvement in functional outcomes. In spite of complete paraplegia, the patient was able to achieve functional level of ambulation without any surgical complications. In children who have adductor contractures (e.g. cerebral palsy) the use of selective obturator neurectomies can lead to loss of walking ability and to abduction contractures.<sup>18</sup> Because the procedure decreases central nervous system activity, it may result in residual hypertonia or loss of muscle function.<sup>19,20</sup> Hence, appropriate selection of treatment becomes very important as effects of surgery may vary from one patient to another.

In underdeveloped health sectors, treatment options are limited due to lack of resources and poor patient follow-up. A surgical intervention that permanently addresses resistant focal spasticity without the need for frequent follow-up and having a low potential for complications can be a dependable choice.

#### Conclusion

Severe adductor spasticity can have debilitating effects and can be a major barrier in rehabilitation of patients with SCI. Surgical treatment can be considered when spasticity becomes unresponsive to noninvasive treatment. Relief from adductor spasticity can improve functional outcomes and decrease dependency. Although this procedure has been commonly performed in cerebral palsy for decades, less attention has been given to it in SCI. It could be a viable surgical alternative in patients with SCI when other modes of treatment are contraindicated, failed, or unavailable.

Unlike treatment with botulinum toxin and the intrathecal baclofen pump, which require regular follow-up for reassessment and dosage adjustment, this surgical treatment offers longer relief for focal spasticity and may be a cost-effective option when treatment choices are limited. This could be of particular importance in health systems around the world where affordability and expertise for advanced treatment of spasticity are not optimal yet. Many developing countries often need to adopt various treatment options that may not be currently considered in developed countries.

#### References

- Lazorthes Y, Sol JC, Sallerin B, Verdie JC. The surgical management of spasticity. Eur J Neurol 2002;9(Suppl 1):35–41; discussion 53–61.
- 2 Mandigo CE, Anderson RC. Management of childhood spasticity: a neurosurgical perspective. Pediatr Ann 2006;35(5):354–62.
- 3 Maarrawi J, Mertens P, Luaute J, Vial C, Chardonnet N, Cosson M, et al. Long-term functional results of selective peripheral neurotomy for the treatment of spastic upper limb: prospective study in 31 patients. J Neurosurg 2006;104(2):215–25.
- 4 Rousseaux M, Buisset N, Daveluy W, Kozlowski O, Blond S. Comparison of botulinum toxin injection and neurotomy in

patients with distal lower limb spasticity. Eur J Neurol 2008; 15(5):506-11.

- 5 Samilson RL. Current concepts of surgical management of deformities of the lower extremities in cerebral palsy. Clin Orthop Relat Res 1981;(158):99–107.
- 6 Sangwan SS, Chand S, Siwach RC, Gupta IS. Treatment of intractable spasticity in spinal cord injured patients. Indian J Med Sci 1992;46(6):169–73.
- 7 Eltorai I, Montroy R. Muscle release in the management of spasticity in spinal cord injury. Paraplegia 1990;28(7):433–40.
- 8 Sgouros S. Surgical management of spasticity of cerebral origin in children. Acta Neurochir Suppl 2007;97(Pt 1):193–203.
- 9 Haftek I. Clinical and electromyographic evaluation of obturator neurectomy in severe spasticity. Paraplegia 1987;25(5):394–6.
- 10 American Spinal Injury Association. International Standards for Neurological Classification of Spinal Cord Injury. Reprinted 2002. Chicago, IL: ASIA; 2006.
- 11 Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 1987;67(2):206–7.
- 12 Morganti B, Scivoletto G, Ditunno P, Ditunno JF, Molinari M. Walking index for spinal cord injury (WISCI): criterion validation. Spinal Cord 2005;43(1):27–33.
- 13 Burchiel KJ, Hsu FP. Pain and spasticity after spinal cord injury: mechanisms and treatment. Spine 2001;26(Suppl. 24):S146–60.
- 14 Adams MM, Hicks AL. Spasticity after spinal cord injury. Spinal Cord 2005;43(10):577–86.
- 15 Wassef MR. Interadductor approach to obturator nerve blockade for spastic conditions of adductor thigh muscles. Region Anesth 1993;18(1):13–7.
- 16 Buffenoir K, Roujeau T, Lapierre F, Menei P, Menegalli-Boggelli D, Mertens P, *et al.* Spastic equinus foot: multicenter study of the long-term results of tibial neurotomy. Neurosurgery 2004; 55(5):1130–7.
- 17 Sindou MP, Simon F, Mertens P, Decq P. Selective peripheral neurotomy (SPN) for spasticity in childhood. Child Nerv Syst 2007; 23(9):957–70. Epub, 2007 June 29.
- 18 Chambers HG. The surgical treatment of spasticity. Muscle Nerve 1997;20(Suppl. 6):S121–8.
- 19 Koman LA, Smith BP, Shilt JS. Cerebral palsy. Lancet 2004; 363(9421):1619–31.
- 20 Abbott R. Neurosurgical management of abnormal muscle tone in childhood. Pediatr Clin North Am 2004;51(2):457–75.