Alexandria Journal of Medicine (2011) 47, 157–164

Alexandria University Faculty of Medicine

Alexandria Journal of Medicine

www.sciencedirect.com

ORIGINAL ARTICLE

Management of generalized spasticity of lower limbs by selective posterior rhizotomy

Wael Fouad

Alexandria Faculty of Medicine, Neurosurgery Department, Alexandria, Egypt

Received 24 May 2011; accepted 17 June 2011
Available online 9 August 2011

KEYWORDS

Spastic paraplegia;
Microsurgical DREZotomy;
Selective posterior rhizotomy

Abstract *Introduction:* Spasticity can be defined as a velocity-dependent resistance to passive movement of a joint and its associated musculature. Functional neurosurgery should be considered when spasticity cannot be controlled by physical therapy and medications.

Objective: This study was done to evaluate the functional results of microsurgical DREZotomy (MDT) in a consecutive series of 15 paraplegic patients suffered from generalized spasticity of lower limbs.

Methods: This retrospective study included 15 paraplegic patients (10 due to spinal cord injury and 5 due to disseminated sclerosis) who underwent MDT for treatment of spasticity. The male to female ratio was 2–1 (10 males and 5 females) and their ages ranged from 22 to 56 years with mean age of 39 years. Assessment of spasticity was done using modified Ashworth scale and global functional score. All cases underwent MDT that consisted of a longitudinal incision of the dorsolateral sulcus, performed ventrolaterally at the entrance of the rootlets into the sulcus, followed by bipolar coagulations performed inside the sulcus, down to the apex of the dorsal horn, continuously along all the spinal cord segments selected for surgery. Mean postoperative follow up examination period was 30 months.

E-mail address: waelfouad_67@hotmail.com

Peer review under responsibility of Alexandria University Faculty of Medicine.
doi:10.1016/j.ajme.2011.06.007

Production and hosting by Elsevier

Results: There was no operative mortality. Postoperatively, all the patients had improvement of their spasticity grade. Assessment of outcome after surgery was done by comparing global functional score for spastic paraplegic patients before and after surgery. At the last follow up examination period, excellent results were obtained in 33.3% of patients, good results in 60% of patients, and fair results in 6.7% of patients.

Conclusion: MDT typically had a dramatic effect on tone in lower limbs. Better long-term results were obtained in the spastic patients caused by spinal cord injury (excellent results in 40%) than in the spasticity caused by disseminated sclerosis (excellent results in 20%).

1. Introduction

Spasticity can be defined as a velocity-dependent resistance to passive movement of a joint and its associated musculature.[1] Spasticity is characterized by hyperexcitability of the stretch-reflex related to the loss of inhibitory influences from descending supraspinal structures.[2] Spasticity should not be treated just because it is present as it may be useful for compensating for loss of motor power.[3] Spasticity should be treated only when excess tone leads to further functional losses, impairs locomotion or induces deformities.[4] Functional neurosurgery should be considered when hyperspasticity cannot be controlled by physical therapy and medications.[5,6]

The methods of surgical management are classified according to whether their impact is general or focal and whether the effects are temporary or permanent.[1] They include not only intrathecal baclofen therapy (ITB) and botulinum toxin injections, but also destructive surgery directed to the peripheral nerves, dorsal roots, dorsal root entry zone or spinal cord.[7,8]

When harmful spasticity affects the whole limb in paraplegic patients, surgery involving the dorsal roots as dorsal rhizotomies or dorsal root entry zone as microsurgical DREZ-otomy (MDT) may be the solution.[9] MDT attempts to selectively interrupt the small nociceptive fibers (situated laterally and centrally respectively), while sparing the large leminiscal fibers, which are regrouped medially (Fig. 1).[2] Complementary orthopedic operations are frequently needed in patients with associated irreducible contractures, tendon retractions, and/or joint deformities.[10]

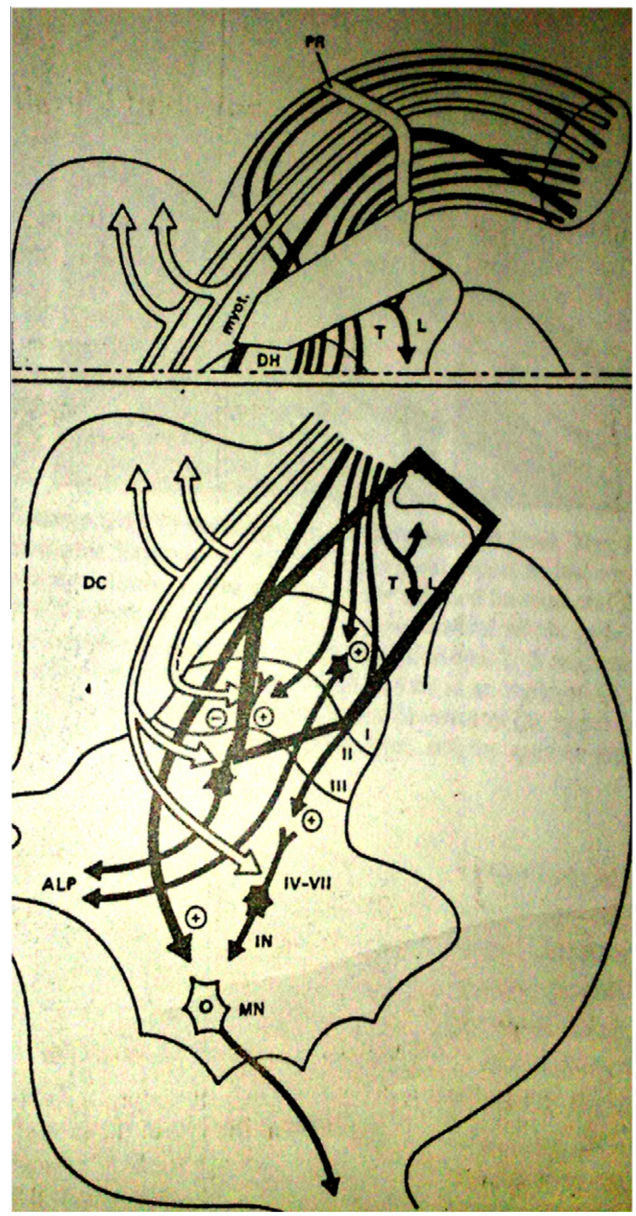

Figure 1 Schematic representation of organization of fibers at DREZ area and target of MDT.[2] DH = dorsal horn, TL = tract of Lissauer myot = myotatic fibers, DC = dorsal column. PR = pial ring. MDT (arrowhead) cuts most of the fine fibers and myotatic fibers and enters the medial (excitatory) portion of TL and the apex of the dorsal horn.

1.1. Objective

This study was done to evaluate the functional results of MDT in a consecutive series of 15 paraplegic patients suffered from generalized spasticity of lower limbs and to compare these results with the results of the other authors reported in the literature.

2. Methods

This retrospective study included 15 paraplegic patients [10 due to spinal cord injury (SCI) and 5 due to disseminated sclerosis (DS)] who underwent MDT for treatment of spasticity. This study was done in Alexandria hospitals over a period of 5 years starting from January 2004 to December 2008. The male to female ratio was 2–1 (10 males and 5 females) and their ages ranged from 22 to 56 years with mean age of 39 years. Craniospinal MRI was done for all cases of disseminated sclerosis. Spinal MRI was not done in the two patients with lower dorsal fracture at D12 who had previous operation in the form of plate and screws fixation. Preoperative urodynamic studies and cystomyography were done for all patients.

Selection and assessment of patients were performed carefully. Once the spastic state had been demonstrated as harmful, one has to determine the respective involvement in the abnormal posture of spasticity (treated with MDT) and only articular, muscular, tendinous, and/or ligamentous limitations (relieved by orthopedic procedures). If doubt persists after detailed clinical examination, a testing of passive articular mobility should be performed under anesthesia. When spasticity plays the larger role in the articular limitations, abnormal postures were significantly diminished during the test. If this was not the case, orthopedic surgery may be the initial or the only treatment. Nerve blocks with xyloacaine may also be used for testing.

Preoperative assessment of the patients by measuring the grade of spasticity was done using both modified Ashworth scale (Table 1) and global functional score that measured the degree of disability related to spasticity (Table 2).

2.1. Surgical technique

Surgery was performed under general anesthesia, with an initial short lasting curarization to allow intraoperative observation of motor responses to electrical stimulation of the nerve roots. A laminectomy was performed from T10 to L2. The dura and arachnoid were opened longitudinally, and the filum terminale was isolated. Identification of roots was performed by electrical stimulation. MDT consisted of a longitudinal incision of the dorsolateral sulcus, performed ventrolaterally at the entrance of the rootlets into the sulcus, and of microbipolar coagulations, performed inside the sulcus, down to the apex of the dorsal horn, continuously along all the spinal cord segments selected for surgery (Fig. 2).

3. Results

MDT was performed on both sides from L1 to S1 in eight cases, from L2 to S1 in four cases and from L2 to S4 in two cases who had spastic bladder as revealed by preoperative urodynamic studies and cystomyography that showed decreased bladder capacity and accelerated uninhibited contraction. MDT was done on the right side only in one case from L2 to S1.

All patients were followed up after surgery, at the discharge from the hospital then at 3 months postoperatively. The mean postoperative follow-up period was 2.5 years (range, 1–5 years).

There was no perioperative mortality. As regards the morbidity, three patients had post-operative CSF leak. Two patients had wound infection. One further patient had bacteremia due to urinary tract infection, which was treated with systemic antibiotics.

Assessment of outcome after surgery was done by comparing global functional score for spastic paraplegic patients before and after surgery (with a total of 20/20 denoting a bedridden and totally dependent patient). Improvement of more than 10 points was considered excellent, between 5 and 10 points was considered good and less than 5 points was considered fair results. At the last follow up period, excellent results were obtained in 33.3% of patients, good results in 60% of patients, and fair results in 6.7% of patients. In all patients with excellent or good results, the reduction of spasticity was accompanied by complete withdrawal of medications.

In this study all patients preoperatively had spasticity either G3 or G4 as measured by modified Ashworth scale, that improved postoperatively by a least three grades in all of them.

Better long-term results were obtained in the spastic patients caused by SCI (excellent results were obtained in 40% of cases) than in the spasticity caused by DS (excellent results were obtained in 20% of cases).

In this study the two patients with spastic bladder were improved after surgery as marked both clinically by stoppage of urine leakage around the catheter and by postoperative urodynamic studies and cystomyography.

Summary data and outcome of these 15 patients are listed in Table 3.

4. Discussion

Spasticity is a motor disorder characterized by an increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex, as one of the components of upper motor neuron (UMN) syndrome.[11] The clinical features of UMN syndrome can be divided into two main groups which are positive phenomenon (spasticity, clonus, rigidity) and negative phenomenon (weakness, incoordination, loss of dexterity).[12] UMN modulates important segmental motor reflex activity in the spinal cord. The majority of positive phenomenons of the UMN syndrome may arise due to interruption of the supraspinal control of these spinal reflexes.[13] Pathophysiology of spasticity and UMN syndrome continues to be a complex and an incompletely understood area.[14]

Generally patients do not complain about spasticity; they are more likely to be aware of stiffness, deformity and limitations in functional abilities.[15] After a period of time, the patients will have a mixture of spasticity and muscle shortening or contracture. There are two types of deformities, dynamic caused by spasticity and fixed described as contracture that remains present under local blocks or anesthesia. The differentiation between dynamic from fixed deformities is of prime importance before deciding any surgical treatment. The dynamic range of motion measures are useful starting points, supplemented with instrumental measures of spasticity and its effects on function, such as motion.[16] Spasticity should not be treated just because it is present; it should be treated because it is harmful. Patients may use spasticity in functional

Table 1 Modified Ashworth scale.

Grade	Patient's status
0	No increase in muscle tone
1	Slight increase in tone with a catch and release or minimal resistance at end of range of motion
1+	Slight increase in tone but with minimal resistance through range of motion following catch
2	More marked increase in tone but limb easily moved
3	Considerable increase in tone, passive movement is difficult
4	Affected part is rigid

Table 2 Global functional score for paraplegic patients with spasticity in the lower limbs.

Component	Score
Pain	
Absent	0
Mild and rare; no influence on daily life	1
Mild, but frequent; inducing depression	2
Marked and frequent; participating to the disability	3
permanent and severe; inducing suicide desire	4
Spasms	
Absent	0
Mild and rare spasm, only during mobilization; no disability	1
Mild but frequent spasms during mobilization; moderate disability	2
Marked and frequent, spontaneous spasms; making sitting position uncomfortable	3
Almost constant severe spasms; making sitting position impossible	4
Sitting position	
Normal and comfortable	0
Normal posture, but slightly uncomfortable	1
Marked difficulty, causing reduction of sitting periods	2
Severe difficulty; patients to be tied down in position	3
Impossible	4
Body transfers	
Normal	0
Mild difficulty	1
Moderate difficulty; can do but only from bed to chair	2
Marked difficulty; need for a person helping	3
Severe difficulty; need for two persons helping	4
Washing and dressing	
Normal	0
Mild difficulty	1
Moderate difficulty; can do but slowly	2
Marked difficulty; need for a person helping	3
Severe difficulty; totally dependant	4

activities. Spasticity should only be treated when excess muscular tone leads to further functional losses, impairs locomotion, or induces deformities, or chronic pain.[17] So in patients with no residual motor function, the objectives of the treatment are improvement in function, prevention of deformities, or alleviation of discomfort and pain. This should be explained carefully to patients what can be gained and what will not be obtained by surgery.[18]

In this study, the most common cause of spastic paraplegia was SCI (spasticity of spinal origin) that was found in 10 patients, of these five patients had previous traumatic cord contusion at lower cervical level, while the other five patients had previous dorsal cord injury (1 upper dorsal, 2 middorsal and 2 at lower dorsal), Only the two patients with lower dorsal spine injury had previous operation in the form of plate and screw fixation due to fracture of dorsal 12 vertebra. The second common cause was DS that was found in five cases (spasticity of both cerebral and spinal origin). Other causes of spastic paraplegia of cerebral origin include cerebral palsy, cerebrovascular stroke and traumatic brain injury which were not included in this study.[19]

In this study, the male to female ratio was 2–1 (10 males and 5 females) and their ages ranged from 22 to 56 years with mean age of 39 years. This could be explained by the fact that SCI that constitute the majority of this series was more common in young males (males constitute 80% of cases of SCI and their mean age was 34 years).

For measuring the spasticity, Ashworth developed a standardized scale to document resistance to passive movements.[20] It is a simple, easy to use test. The scale has been shown to correlate with other more reliable measures such as electromyography measurements. The disadvantage of this scale is the fact that it measured the combined effect of both biochemical and neural components of tone also the lake of standardization technique in performing the tone measurements. Repetitive movement of the limb may modify stretch reflex and influence more dynamic biochemical properties of the muscle. The force applied in moving the limb should also be standardized.[21]

Preoperative assessment of the patients by measuring the degree of disability related to their spasticity was done using global functional score for paraplegic patients with spasticity in lower limbs. This score developed by Millet and associate quantifies five components that are directly influenced by spasticity, abnormal postures, and articular limitations and are part of the patient's everyday life (Table 2).[22] The score goes from 0 to 4 for each component, with a total of 20/20 denoting a bed ridden and totally dependent patient. A score of 10/20 was seen to correspond reproducibly to the threshold between a minimally acceptable and an unacceptable condition and thus was identified as the lowest position at which to consider surgery. The score was also used for the assessment of outcome after surgery by comparing the score before and after surgery. All patients in this study had preoperative scores more than 10/20.

Table 3 Summary data and outcome of 15 paraplegic patients who underwent MDT.

No	Age	Sex	Etiology	Spasticity grade Ashworth scale		Level of DREZ lesion		Complications	Outcome using global functional score		
				Preop.	Postop. 1 year	Right side	Left side		Pre-operative score	Score at 3 months postoperatively	Score at last follow up postoperative period in months (m)
1	32	M	Cervical SCI	G 3	G 1	L1–S1	L1–S1		12	5 Good result	4 Good result 36 m
2	41	M	D.S.	G 4	G 0	L1–S1	L1–S1	CSF leak	16	5 Excellent result	4 Excellent result 48 m
3	25	M	Dorsal SCI	G 3	G 1	L2–S1	L2–S1		13	4 Good result	4 Good result 60 m
4	45	M	D.S	G 3	G 1	L1–S1	L1–S1		13	6 Good result	6 Good result 30 m
5	39	M	Cervical SCI	G 3	G 0	L2–S1	L2–S1		15	4 Excellent result	4 Excellent result 34 m
6	29	M	Dorsal SCI	G 4	G 1+	L1–S1	L1–S1	CSF leak	17	6 Excellent result	9 Good result 34 m
7	51	F	D.S.	G 3	G 1	L2–S1	L2–S1		13	5 Good result	5 Good result 40 m
8	22	F	Dorsal SCI	G 3	G 1	L2–S1		Wound infection	11	7 Fair result	7 Fair result 31 m
9	31	M	Cervical SCI	G 3	G 1	L1–S1	L1–S1		14	3 Excellent result	3 Excellent result 24 m
10	47	M	Dorsal SCI	G 3	G 1	L1–S1	L1–S1		16	5 Excellent result	8 Good result 22 m
11	48	F	D.S.	G 4	G 1+	L1–S4	L1–S4	Wound infection	18	7 Excellent result	9 Good result 28 m
12	46	F	Cervical SCI	G 3	G 0	L1–S1	L1–S1		15	6 Good result	6 Good result 24 m
13	56	F	D.S.	G 3	G 0	L1–S1	L1–S1	CSF leak	12	5 Good result	5 Good result 12 m
14	36	M	Cervical SCI	G 3	G 1	L2–S1	L2–S1		14	3 Excellent result	3 Excellent result 22 m
15	34	M	Dorsal SCI	G 4	G 1	L1–S4	L1–S4	Bacteremia due to UTI	17	6 Excellent result	6 Excellent result 15 m

SCI = spinal cord injury, D.S. = disseminated sclerosis. Preop. = preoperative, Postop. = postoperative.

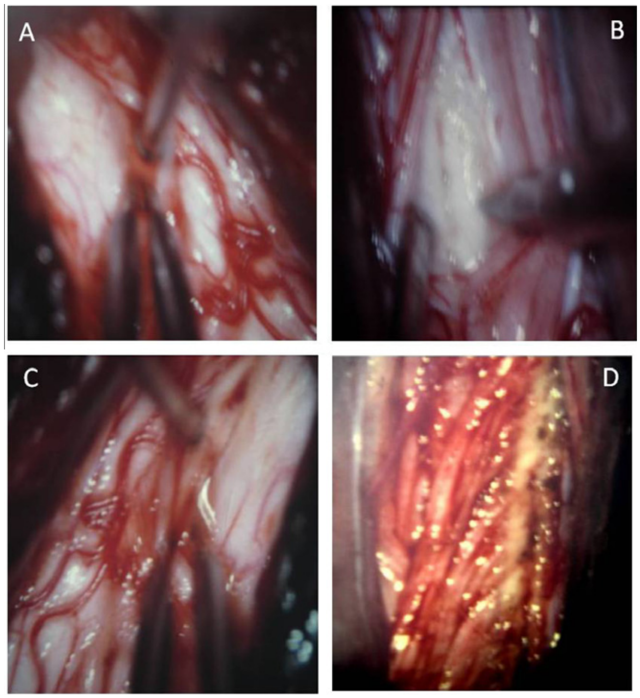

Figure 2 MDT operative technique at the lumbosacral level. (A) The dorsal roots are retracted dorsomedially and held with a small hook, to gain access to the dorsolateral sulcus. (B) Incision is performed using a micro-knife, along the dorsolateral sulcus. (C) Micro-coagulations inside the sulcus down to the apex of the dorsal horn. (D) The lesion is completed and apex of dorsal horn is exposed.

MDT was indicated only when spasticity has resisted all forms of physiotherapy and drug treatments.[5] The microsurgical lesions were performed at the selected spinal segments that correspond to the lower limb myotomes. It was performed on both sides from L1 to S1 in eight cases, from L2 to S1 in four cases and from L2 to S4 in two cases because there was a spastic bladder. MDT was done on the right side only in one case from L2 to S1 because he had flexion deformity on the right knee joint but the opposite side was controlled enough by physiotherapy and antispasmodics. The longitudinal incision was made with a microknife followed by bipolar coagulations in dorsolateral sulcus. If the laxity of the root was sufficient, the incision was performed continuously, If not, a partial incision was made successively on each rootlet of the root, after isolation of each one by separating the tiny arachnoid membranes that hold them together.[23]

Intraoperative neurophysiologic monitoring was done by observation of muscular responses to stimulation of the dorsal roots from L1 to S1. Stimulation of the S2 to S4 dorsal roots could be assessed by recording of the motor anal response by a finger introduced into the anus.[24]

Some authors marked that intraoperative monitoring of somatosensory evoked potentials (SEP) make localization of dorsal root entry zone more accurate and minimize the possibility of damage of adjacent long tracts.[25,26] Also they found that intraoperative neurophysiologic monitoring were time-consuming, instead they measured 30 mm above the exit from the conus of the tiny coccygeal root which was the landmark between the S1 and S2 that could be sufficient in the patients who already have severe preoperative impairment of their vesicoanal functions.[27] In this study SEP were not performed, only intraoperative electrical stimulation of the nerve roots and observation of motor response in the specific muscle groups innervated by this root was done.

In this study the mean operation time was 4.5 h and the mean volume of blood loss was 850 ml. The excess blood loss was due to rich vasculature of the conus. The dorsolateral spinal artery courses along the dorsolateral sulcus. Its diameter is 0.1–0.5 mm, and it is fed by the posterior radicular arteries and joins caudally with the descending anterior branch of the Adamkiewicz artery. The dorsolateral spinal artery had to be preserved by being freed from the sulcus. MDT was difficult especially in the two cases with previous injury at lower dorsal region due to previous adhesion and scaring and necrotic cysts inside the cord. Similar to this study, Mertens et al. had reported a mean operation time of 4 h and a mean volume of blood loss of 650 ml.[28]

Like other series, there were no postoperative mortalities.[28,29] Two cases had superficial wound infection. One of them was treated successfully using the proper antibiotics after performing culture and sensitivity from the wound. While the second patient was diabetic and required a surgical debridement with the antibiotics. Also three patients had CSF leak that were treated with positioning and compression. Lastly one further patient had bacteremia due to urinary tract infection that was treated with systemic antibiotics. Similarly Sindou et al.[30] who retrospectively reviewed 44 cases with MDT in SCI reported two cases with wound infection treated and cured with antibiotics and three patients had post-operative CSF leak; of them, two were treated by positioning and compression, while an operation was needed for dural repair in the third patient. One patient had subcutaneous hematoma that required surgical drainage. One further patient had bacteremia due to urinary tract infection that was treated with systemic antibiotics.

In this study the spasticity had improved in all cases post operatively, as regards grade of spasticity measured by Ashworth scale and improvement in the functional score. All patients in this study had more comfort, less pain and ability to resume physical therapy with more functional gain. The muscular tone was diminished after MDT due to interruption of the afferent component of the myotatic monosynaptic and the nociceptive polysynaptic reflexes, which would deprive the somatosensory relays of the dorsal horn from most of its excitatory inputs.[5]

It is necessary to provide neurosurgical treatment together with intensive physical therapy for several weeks. To take the maximal benefit out of surgery, patients have to optimize their new neurological status by a rigorous and intensive program of physical therapy. The result of surgery and its impact on the daily life of patients can be appreciated only after a follow-up of not less than 3 months.[31] In this study, at 3 months follow up period, excellent results were obtained in eight patients (53.3%), and good results in six patients (40%). At the last follow up period (mean 2.5 years) excellent results had obtained in only 33.3% of cases while good results in 60% of cases. The decline of excellent results with time (from 53.3% at 3 months follow up, to 33.3% at last follow up period) might be due to secondary soft tissue changes like stiffness of muscles and tendons presumably occurred as a result of muscle not being placed through a normal range of movement.[32] Also better excellent results were obtained in cases of spasticity due to SCI than in cases of spasticity due to DS (60% versus 40% at 3 months follow up, 40% versus 20% at last follow up period). This could be related to the origin of spasticity, it had a spinal origin in cases of SCI but it had both cerebral and spinal origin in cases of DS. These results were nearly the same like other authors, but many authors did not correlate their results to the cause or the origin of the spasticity.[5,19] In this study two cases with spastic bladder were improved after surgery as marked both clinically by stoppage of urine leakage around the catheter, electrophysiologically and by using urodynamic studies, this might due to increase in the bladder capacity because the detrusor muscle was not irreversibly fibrotic.[33]

Sindou et al.[34] reviewed their series that consisted of 175 paraplegic patients who underwent MDT at the lumbosacral level for excessive spasticity. MDT was performed bilaterally from L2 to S2 in 158 cases and unilaterally in 17 cases. Also it was performed from L2 to S4 in 15 cases with spastic bladder. Mean follow-up was 9 years. They obtained a significant decrease in spasticity (at least a 2-point reduction in Ashworth score) allowing the withdrawal of antispasmodic medications in 75% of patients. Bladder capacity was significantly improved in 85%; in the patients who improved. They reported better results of their cases caused by pure spinal origin (80%) followed by disseminated sclerosis (75%) and least improvement in spasticity caused by cerebral origin (60%). They concluded that reduction in spasticity usually results in a significant improvement of abnormal postures and articular limitations that was achieved in 90% of their patients.[34]

5. Conclusions

MDT typically has a dramatic effect on tone in lower limbs. Surgical indications must be restricted to paraplegic patients with severe disability who are unable to walk comfortably in a wheelchair or are exposed to pressure sores in bed, especially if additional pain from spasms, contractures, or neurotrophic disturbances is present. MDT can be indicated to treat neurogenic bladders when there is no voluntary micturation and if uninhibited detrusor contractions result in voiding around the catheter or in between intermittent self-catheterization. Better long-term results were obtained in the spasticity caused by spinal cord injury (excellent results in 40%) than in the spasticity caused by disseminated sclerosis (excellent results in 20%).

References

1. Katz RT. Management of spasticity. *Phys Med Rehabil* 1988;**67**:108–16.
2. Sindou M, Quoex C, Baleydier C. Fiber organization at the posterior spinal cord-rootlet junction in man. *J Comp Neurol* 1974;**153**:15–26.
3. O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following motor stroke. *Brain* 1996;**119**:1737–49.
4. Abbott R, Forem SL, Johann M. Selective posterior rhizotomy for the treatment of spasticity. *Childs Nerv Syst* 1989;**5**:337–46.
5. Sindou M, Jeanmonod D, Mertens P. Surgery in the DREZ: microsurgical DREZotomy for treatment of spasticity. In: Sindou R, Abbott R, Keravel Y, editors. *Neurosurgery for spasticity*. Wien: Springer; 1991. p. 165–82.
6. Sindou M, Jeanmonod D. Microsurgical DREZ-tomy for the treatment of spasticity and pain in the lower limbs. *Neurosurgery* 1989;**24**:655–70.
7. Albricht AL, Cervi A, Singletary J. Intrathecal baclofen for spasticity incerebral palsy. *JAMA* 1991;**265**:1418–22.
8. Sindou M, Abdennebi B, Sharkey P. Microsurgical selective procedures in the peripheral nerves and the posterior root-spinal cord junction for spasticity. *Appl Neurophysiol* 1985;**48**:97–104.
9. Albright AL, Tyler-Kabara EC. Combined ventral and dorsal rhizotomies for dystonic and spastic extremities. Report of six cases. *J Neurosurg* 2007;**107**(4 Suppl.):324–7.
10. Bleck EE. Management of the lower extremities in children who have cerebral palsy. *J Bone Joint Surg* 1990;**72**:140–4.
11. Corcos DM, Cottleib GL, Penn RD. Movement deficit caused by hyperexcitable sterch reflex in spastic humans. *Brain* 1986;**109**:1043–58.
12. Thilmann AF, Fellows SJ, Garns E. The mechanism of spastic muscle hypertonus. *Brain* 1991;**114**:233–44.
13. North J. Trends in the pathophysiology and pharmacotherapy of spasticity. *J Neurol* 1991;**238**:131–9.
14. Brown P. Pathophysiology of spasticity. *J Neurol Neurosurg Psychiatry* 1994;**57**:773–7.
15. Sindou M. Neurosurgical management of disabling spasticity. In: Spetzler RF, editor. *Operative techniques in neurosurgery*, vol. 7. Philadelphia: Elsevier; 2004. p. 95–174.
16. Sindou M, Millet MF, Mortamais J, et al. Results of selective posterior rhizotomy in the treatment of painful and spastic paraplegia secondary to multiple sclerosis. *Appl Neurophysiol* 1982;**45**:335–40.
17. Sindou M, Fischer G, Mansuy L. Posterior spinal rhizotomy and selective posterior rhizidiotomy. In: Krayenbühl H, Maspes PE, Sweet WH, editors. *Progress in neurological surgery*, vol. 7. Karger: Basel; 1976. p. 201–50.
18. Lagalla G, Danni M, Reiter F, et al. Post stroke spasticity management. *Am J Phys Med Rehabil* 2000;**79**(4):377–84.
19. Sindou M, Merten P. Neurosurgery for spasticity. *Stereotact Funct Neurosurg* 2000;**74**:217–21.
20. Pisano F, Miscio G, Del CC, et al. Quantitative measures of spasticity in post stroke patients. *Clin Neurophysiol* 2000;**111**(6):1015–22.
21. Pandyan AD, Johnson GR, Price CL, et al. A review of the properties and limitations of the Ashworth and modified Ashworth scales as a measure of spasticity. *Clin Rehabil* 1999;**13**(5):373–83.
22. Millet M, Sindou M, Abbott R, Keravel Y. In: Sindou M, Abbott R, Keravel Y, editors. *"Neurosurgery for spasticity." A multidisciplinary approach*. Wien: Springer; 1991. p. 165–82.
23. Guenot M, Bullier J, Sindou M. Clinical and electrophysiological expression of deafferentation pain alleviated by dorsal root entry zone lesions in rats. *J Neurosurg* 2002;**97**:1402–9.
24. Jeanmonod D, Sindou M, Magnin M, et al. Intra-operative unit recordings in the human dorsal horn with a simplified floating microelectrode. *Electroencephalogr Clin Neurophysiol* 1989;**72**:450–4.
25. Guenot M, Hupe JM, Mertens P, et al. New type of microelectrode for obtaining unitary recordings in the human spinal cord. *J Neurosurg (Spine)* 1999;**91**:25–32.
26. Jeanmonod D, Sindou M. Somatosensory function following dorsal root entry zone lesions in patients with neurogenic pain or spasticity. *J Neurosurg* 1991;**74**:916–32.
27. Guenot M, Bullier J, Rospars J, et al. Single-unit analysis of the spinal dorsal horn in patients with neuropathic pain. *J Clin Neurophysiol* 2003;**20**:142–50.
28. Mertens P, Sindou M. Surgical management of spasticity. In: Barnes MP, Johnson GR, editors. *Clinical management of spasticity*. Cambridge University Press: Cambridge; 2008. p. 239–65.
29. Peacock WJ, Arens LJ. Selective posterior rhizotomy for relief of spasticity in cerebral palsy. *S Afr Med J* 1982;**62**:119–24.

30. Sindou M, Mertens P, Mosa W. Microsurgical DREZotomy for pain due to spinal cord and/or cauda equina injuries: long-term results in a series of 44 patients. *Pain* 2001;**92**:159–71.
31. Meythaler JM. Concepts of spastic hypertonia. *Phys Med Rehabil Clin N Am* 2001;**12**(4):725–32.
32. Kamper DG, Schmit BD, Ryma WZ. Effects of muscle biomechanics on the quantification of spasticity. *Ann Biomed Eng* 2001;**29**(12):1122–34.
33. Beneton C, Mertens P, Sindou M. The spastic bladder and its treatment. In: Sindou M, Abbott R, Keravel Y, editors. *Neurosurgery for spasticity: a multidisciplinary approach*. New York: -Springer; 1991. p. 193–9.
34. Sindou M. Microsurgical DREZotomy (MDT) for pain, spasticity and hyperactive bladder: a 20 year experience. *Acta Neurochir* 1995;**137**:1–5.