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ORIGINAL ARTICLE

Selective neurotomy of the tibial nerve for treatment of spastic foot

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KEYWORDS

Spastic foot;
Selective peripheral neurotomies;
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Abstract *Introduction:* Selective peripheral neurotomy (SPN) of the tibial nerve is indicated for the treatment of spastic deformities of the foot.
Objective: This retrospective study was done to evaluate the surgical results of SPN of tibial nerve in 16 patients who had spastic foot.
Methods: The male to female ratio was 2–1 (11 males and 5 females) and their ages ranged from 18 to 65 years.
All patients preoperatively had spasticity either G3 or G4 as measured by modified Ashworth scale. All cases underwent surgery in the form of variable combination of SPN of tibial nerve depending on the pattern and distribution of spasticity. Depending on the degree of preoperative spasticity, 50–80% of the isolated motor branches of fascicles were resected under the operating microscope. Mean postoperative follow up examination period was 24 months.
Results: There was no operative mortality. Two patients had wound infection. Transient paresis of flexors of the foot occurred in one patient that responded well to physical therapy. Sensor affections and trophic changes occurred in three patients. Postoperatively, all the patients had immediate improvement of their spasticity grade. After initial improvement, recurrence of spasticity occurred in one patient. Abnormal foot posture that was present in all cases improved in 15 patients (94%) postoperatively, while pain that was present in 25% of cases improved in all these cases postoperatively as measured by visual analog scale. Assessment of outcome after surgery was done by comparing modified Ashworth scale preoperatively and postoperatively. At the last follow up

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examination period, excellent results were obtained in 50% of patients, good results in 31% of patients, fair results in 13% of patients, and poor results in 6% of cases.

Conclusion: In well-selected patients, SPN of tibial nerve can yield good effects on refractory spasticity of the foot and its consequences.

1. Introduction

Spasticity was defined as velocity-dependent exaggeration of the stretch reflex.[1] This symptom is observed in case of pyramidal lesions due to cerebrovascular accidents and head or spinal cord injuries. One of the most frequent muscular compartments involved in this pathology is the posterior muscles of the leg, especially the triceps surae. This leads to spastic pes equines, possibly associated with varus posture and spastic claw of the toes.[2,3]

Several medical treatments exist (physiotherapy, perineural injections of alcohol or intramuscular injections of botulinum toxin).[4] In the case of failure of these treatments, orthopedic surgery (aponeurotomy of gastrocnemius or lengthening of calcaneus tendon) or neurosurgical treatment (neurotomy) may be proposed.[5] Neurotomies are indicated when the spasticity is localized to the muscle or muscular group supplied by a single or a few peripheral nerves that are easily accessible.[1,6] Neurotomies can cut all types of sensory and motor fibers and therefore these must be as selective as possible. Neurotomies re-equilibrate the tonic balance between agonist and antagonist muscles. This results in reduction of abnormal joint postures and improvement in residual voluntary movement.[7]

Selective peripheral neurotomies (SPN) were first introduced for the treatment of spastic deformities of the foot by Stoffel.[8] Selectivity is required to suppress the spasticity without excessive weakening of motor power and without producing exaggerated amyotrophy.[1,2] To achieve this goal at least one fifth of the motor fibers are preserved. For the treatment of spastic pes equines, selective fascicular neurotomy must involve mainly one of the three components of the triceps surae, the soleus muscle, which is supposed to be exclusively responsible for the spastic pes equines in 75% of the cases.[9]

The aim of this work was to evaluate the surgical results of selective peripheral neurotomy of tibial nerve in 16 patients who had spastic foot and to compare these results with the results of the other authors reported in the literature.

2. Methods

This retrospective study was carried out on 16 patients who had spastic foot. This study was done in Alexandria hospitals over a period of 6 years starting from January 2004 to December 2009. The male to female ratio was 2–1 (11 males and 5 females) and their ages ranged from 18 to 65 years with mean age of 47 years. The cause of spastic foot was cerebrovascular stroke in 10 patients, cerebral palsy in two patients, head injury in two patients and spinal cord injury in two patients. Spastic foot deformity was in the form of equinus in one patient, equinus with flexion of toes in two patients, equinovarus in eight patients, and equinovarus with flexion of toes in five patients (Table 1).

Selection and assessment of patients were performed carefully that included observation of posture, assessment of passive range of motion, orthopedic status (musculoskeletal contractures), and quantification of the degree of spasticity using modified Ashworth scale (Table 2). In this study all patients preoperatively had spasticity either G3 or G4 as measured by modified Ashworth scale (Table 2). The determination of the respective involvement in the abnormal posture of spasticity (treated with SPN) and only articular, muscular, tendinous, and/or ligamentous limitations (relieved by orthopedic procedures) was important. If doubt persisted after detailed clinical examination, nerve blocks with 3 mL of 0.25% xylocaine were used for testing of passive articular mobility. When spasticity plays the larger role in the articular limitations, abnormal postures were significantly diminished after the test. If this was not the case, orthopedic surgery may be the initial or the only treatment. Also motor power of planter flexors and dorsiflexors was assessed using motor scale (0 = flaccid; 5 = normal power).

All cases underwent surgery in the form of selective peripheral neurotomy of tibial nerve under general anesthesia but without long-lasting muscle relaxants so that the motor responses elicited by bipolar electrical stimulation of motor branches could be detected.

The patient is operated on in the prone position with the knees slightly flexed to relax the hamstrings and gastrocnemius muscles and thereby facilitating access to popliteal region. The

Table 1 Foot deformity in 16 patients who underwent SPN of tibial nerve.

Foot deformity	No.	%
Equinus	1	6.25
Equinus + flexion of toes	2	12.5
Equinovarus	5	31.25
Equinovarus + flexion of toes	8	50
Total	16	100

Table 2 Modified Ashworth scale.[2]

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

All patients had spasticity either G3 or G4 preoperatively.

leg is draped to allow the surgeon to get a sufficient view of the foot check its muscle responses. The skin incision is made in the midline vertically for 7 cm down from the transverse popliteal line. The incision may start 3 cm higher when the identification of gastrocnemius branches is necessary. To get an easy access to the flexor fascicles the distal tibial trunk under the soleus arch, it was necessary to extend the incision 3 cm downwards.

Tibial nerve dissection began by locating proximal segment of the nerve in the fat of the upper popliteal region. The tibial branches were dissected and identified proximally to distally using the operating microscope and the bipolar stimulation. When the correction of the flexion of the toes was needed, the epineurium of the tibial trunk was opened at the level of the soleus arch to allow dissection of the responsible motor fascicles (Figs. 1 and 2).

Depending on the degree of preoperative spasticity, 50–80% of the isolated motor branches of fascicles were resected under the operating microscope. The resection was 7 mm long from the proximal stump, which was coagulated with bipolar forceps to prevent re-growth of fibers.

The evaluation of treatment outcome was done by measuring some of the clinical consequence of spasticity like pain, abnormal posture and reduced functional ability. Mean postoperative follow up examination period was 24 months (range from 1 to 4 years).

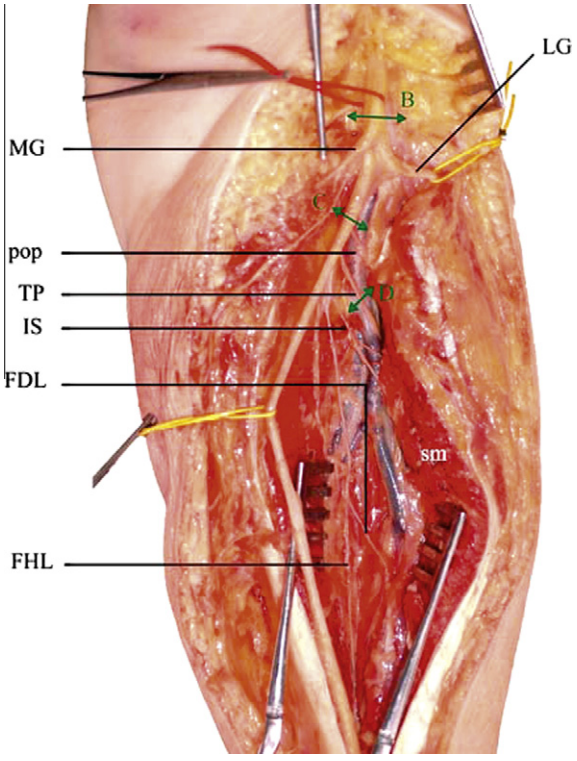

Figure 1 Operative view of the right popliteal region showing the tibial nerve and peroneal nerve. The sensory sural nerve lies superficially just beneath the subcutaneous aponeurosis between the two gastrocnemius muscles. (MG) Nerve to the medial head of the gastrocnemius, (LG) nerve to lateral head of gastrocnemius, (pop) nerve to popliteus, (sm) soleus muscle. The emergence of the muscular branches of the nerve (for the TP, FDL, FHL and IS) from a common muscular branch, arising close to the tendinous arch of the soleus.

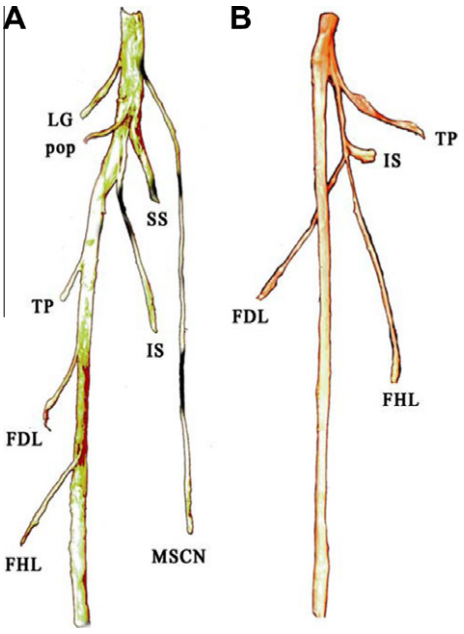

Figure 2 Anatomic view of dissected tibial nerves.[28] (A) Left tibial nerve with emergence of the muscular branches of the nerve from its ventrolateral aspect. (LG) nerve to the lateral head of the gastrocnemius, (pop) nerve to the popliteus, (TP) nerve to the tibialis posterior, (FDL) nerve to the flexor digitorum longus, (FHL) nerve to the flexor hallucis longus, (SS) superior nerve to the soleus, (IS) inferior nerve to the soleus, (MSCN) medial sural cutaneous nerve. (B) Right tibial nerve, emergence of the muscular branches of the nerve (for the TP, FDL, FHL and IS) from a common muscular branch, arising close to the tendinous arch of the soleus.

3. Results

SPN was performed on tibial nerve branches in all cases, while neurotomy of distal nerve trunk was performed in 10 cases with flexion deformity of the toes. 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 24 months (range 1–4 years).

Postoperative mortality and morbidity: There was no perioperative mortality. As regard the morbidity, two patients had wound infection that responded well to antibiotics after culture and sensitivity. Paresis of flexors of the foot because of excessive nerves sectioning occurred in one patient that was transient, and responded well to physical therapy. Recurrence of spasticity due to insufficient amount of sectioning could be observed in one case that was shifted to other modalities of treatment (Botulinum toxin injection). Recurrence occurred 3 months postoperatively after initial improvement of the spasticity. Sensory affections and trophic changes occurred in three patients that responded well to medical treatment.

Assessment of outcome after surgery was done by comparing modified Ashworth scale pre- and postoperatively. The patients were classified into four groups according to their

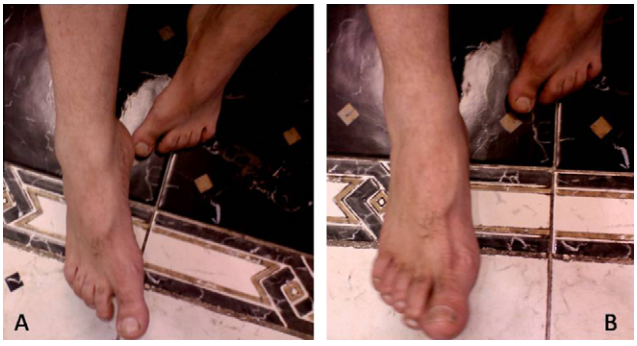

Figure 3 Equinovarus deformity with flexion of toes. Before (A) and after (B) surgery.

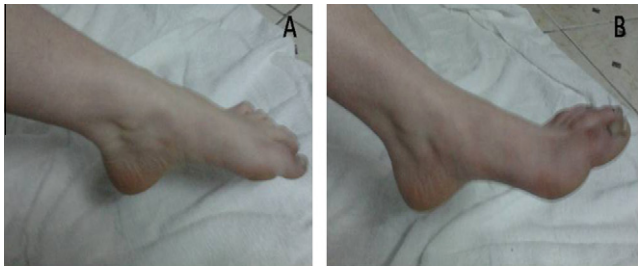

Figure 4 Equinus deformity with flexion of the toes. Before (A) and after (B) surgery.

results. Group 1: Reduction of three or more grades was considered excellent, group 2: reduction of two grades was considered good, group 3: reduction of one grade was considered faire, and group 4: no grade reduction was considered poor. All patients had immediate initial improvement of their spasticity. At the last follow up examination period, excellent results were obtained in 50% of patients, good results in 31% of patients, fair results in 13% of patients, and poor results in 6% of cases. In excellent and good results, the reduction of spasticity was accompanied by complete withdrawal of medications.

Operation improved equinus deformity in 94% of patients, varus deformity in 77% of patients and flexion of toes in 70% of patients (Figs. 3 and 4), while pain that was present in 25% of cases improved in all these cases as measured by visual analog scale.

Also, comparison of pre- and postoperative motility was evaluated for ankle planter flexors using motor scale, the improvement of motor power was observed in 75% of case while stationary in 19%, and deteriorated in 6%. As regards the motor power of dorsiflexors the improvement was in 87.5% of cases, while remained stationary in 12.5% of cases.

Summary data and outcome of these 16 patients are listed in Table 3.

4. Discussion

Spasticity of the lower limbs causes adduction of the thigh, plantar flexion and inversion of the foot resulting in reduction, or even loss of neuro-functional capacities.[10] Generally patients do not complain about spasticity; they are more likely to be aware of stiffness, deformity and limitations in functional abilities. 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. Spasticity should only be treated when excess muscular tone leads to further functional losses, impairs locomotion, or induces deformities, or chronic pain.[11] This should be explained carefully to patients what can be gained and what will not be obtained by surgery.[12] Selective fascicular neurotomy is appropriate in cases of localized spasticity, with absence of associated muscle–tendon retraction, and when a preoperative motor block using local anesthetic or botulinum toxin has given good functional results.[13] This strategy allows the patient to appreciate the benefit that can follow a selective neurotomy. Objectives may be cosmetic.[14]

In this study, the most common cause of spastic foot was cerebrovascular stroke that was found in 10 patients (62, 5%), cerebral palsy in two cases, cervical cord injury in two cases and traumatic brain injury in another two cases. In this study, the male to female ratio was 2–1 (11 males and 5 females) and their ages ranged from 18 to 65 years with mean age of 47 years. Cerebrovascular stroke is more common in old age while cerebral palsy and trauma are more common in young age and all of them are more common in males.

Assessment of the degree of spasticity in each muscle of the leg is essential to plan the surgical procedure. Thus, a variable combination of SPN can be performed, depending on the pattern and distribution of spasticity. The operative program includes designation of the spastic muscles to be targeted and the amount of denervation of the respective muscles needed.[15] For measuring the spasticity, Ashworth developed a standardized scale to document resistance to passive movements (Table 2).[16] 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.[17]

Surgery should be performed so that excessive hypertonia is reduced without the suppression of useful muscular tone or impairment of residual motor and sensory functions.[18] SPN aims at re-equilibrating the tonic balance between agonist and antagonist muscles by reducing excess spasticity. Decreased spasticity is obtained by sectioning both afferents and efferent fibers of the stretch–reflex at the level of a motor nerve.[19]

According to the previously published data, the branches to the medial and lateral heads of the gastrocnemius were found to arise either separately from both sides of the tibial nerve trunk or posteriorly from a common origin, sometimes including the medial sural cutaneous nerve. One or two nerves to the soleus may arise from a common origin or separately from the tibial nerve. The nerve to the tibialis posterior, like the nerves

Table 3 Summary data and outcome of 16 patients who underwent SPN of tibial nerve.

No.	Age	Sex	Etiology	Deformity of foot	Operative program of SPN (Amount of denervation + spastic muscle)	Complications	Spasticity outcome using Ashworth scale	
							Preop.	Postop. + follow up period (ms)
1	48	M	Cervical cord injury	Equinus + flexion of toes	80% of fibers to soleus, motor fascicles of distal trunk of nerve + 65% fibers to tibialis posterior	Hyperesthesia and trophic changes lateral side of sole	G 3	G1 + good result 42 m
2	58	M	Cerebral stoke	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of nerve		G 4	G1 excellent result 48 m
3	19	F	Cerebral palsy	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of nerve	Wound infection	G3	G1 + good result 23 m
4	31	M	Head injury	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of nerve		G 3	G1 excellent result 18 m
5	61	F	Cerebral stoke	Equinovarus	80% of fibers to soleus, tibialis posterior + 65% fibers to gastrocnemius	Recurrence of spasticity	G 3	G 3 poor result 25 m
6	45	M	Cerebral stoke	Equinus	80% of fibers to soleus, gastrocnemius + 65% fibers to tibialis posterior		G 4	G1 excellent result 21 m
7	49	M	Cerebral stoke	Equinovarus	80% of fibers to soleus and tibialis posterior	Dysesthesia and trophic changes of sole.	G 3	G1 + good result 12 m
8	58	M	Cerebral stoke	Equinovarus	80% of fibers to soleus and tibialis posterior		G 3	G2 fair result 22 m
9	18	M	Cerebral palsy	Equinovarus	80% of fibers to gastrocnemius, soleus and tibialis posterior		G 4	G0 excellent result 20 m
10	34	F	Cervical cord injury	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of nerve	Transient paresis of wrist flexors	G 3	G1 + good result 17 m
11	54	F	Cerebral stoke	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of nerve	Dysesthesia and trophic changes of sole	G 4	G1 excellent result 20 m
12	65	M	Cerebral stoke	Equinovarus	80% of fibers to gastrocnemius,soleus + 65% of fibers to tibialis posterior		G 4	G1 excellent result 21 m
13	53	M	Cerebral stoke	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of		G 4	G1 excellent result 25 m
14	54	F	Cerebral stoke	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of nerve	Wound infection	G 3	G2 fair result 22 m
15	49	M	Cerebral stoke	Equinus + flexion of toes	80% of fibers to soleus, motor fascicles of distal trunk of nerve + 65% of fibers to tibialis posterior		G 4	G1 excellent result 19 m
16	53	M	Head injury	Equinovarus + flexion of toes	80% of fibers to soleus, tibialis posterior and motor fascicles of distal trunk of nerve		G 4	G2 good result 20 m

to the soleus, originates from the ventral lateral aspect of the tibial nerve, but more distally at the level of the tendinous arch of the soleus. Also that the distal part of the tibial nerve was found to contain five to eight fascicles, averaging 1 mm diameter each; two-thirds of these are motor fascicle and one-third are sensory fascicles.[20] Then arise the nerves to the tibialis posterior (TP), the flexor digitorum longus (FDL) and the flexor hallucis longus (FHL).[21] This precise knowledge of the distribution of the tibial nerve below the knee makes selective fascicular neurotomies possible beyond the nerve trunk, by selecting collateral motor branches.[22]

After identifying them by electrical stimulation, the neurotomy is performed under microscope. Each motor branch is partially sectioned (between 50% and 80%). The percentage of fibers to be sectioned depends on the degree of spasticity, and on the response to intraoperative electrical stimulation after sectioning 50% of the fibers. The resection is 5–10 mm long to prevent re-growth of fibers, and the proximal stump of the resected fascicle is coagulated with the goal of avoiding proliferative neuroma formation.[23]

Previous clinical studies have demonstrated that spasticity of the soleus muscle was exclusively responsible for the spastic (pes equines) flexion of the ankle in 75% of cases, whereas spasticity of the gastrocnemius muscle was predominantly involved in only 12.5% of the cases.[23–25] When equinus and/or ankle clonus are significantly decreased by flexing the knee which reduces the gastrocnemius tension, it may be assumed that this muscle plays an important role in the abnormal posture. If this maneuver is negative, spasticity may be considered predominant in the soleus muscle. During the surgical procedure using a skin incision 2 cm above and 6 cm below the popliteal knee flexion crease, after resection of four-fifths of the upper nerve to the soleus, one must get an immediate disappearance of the ankle clonus or hypertony. If it persists, that might indicates that the gastrocnemius muscle is involved in the spastic pes equines and that surgery should be completed with a gastrocnemius neurotomy. Some authors[24,26] reported that the neurotomy of the lower nerve to the soleus does not seem to be necessary to make all clonus and/or hypertony of the ankle disappear, while other authors believed that it must be done on a case-by-case basis.[23,27]

In this study, the soleus muscle alone was responsible for the equinus deformity in 81% of patients that disappeared after neurotomy of both upper and lower branches of soleus muscle, while gastrocnemius with soleus were responsible for equinus deformity in 19% of cases. One can also associate a neurotomy of the tibialis posterior nerve for a varus posture, or a neurotomy of the nerves to the flexor hallucis longus and/or flexor digitorum longus for a spastic claw of the toes.[28]

Dissecting fascicles inside the tibial nerve at the level of the soleus arch, after the epineural envelope has been opened, needs an atraumatic manipulation. Their identification is helped by optic magnification, and low intensity bipolar electrical stimulation that allows sparing of the motor fascicles from the sensory ones. This identification is of utmost importance to prevent any damage to the sensory fibers, which could lead to loss of proprioceptive sense, sensory disturbances and neurogenic pain in the plantar area, and thus act as an irritating thorn in spasticity. Sensory and trophic complications are the consequence of excessive manipulations of the sensory fascicles during surgery.[29] In this study sensory and trophic complications had occurred in three patients postoperatively.

It is safe and preferable to dissect the nerve after it arises from the trunk of the tibial nerve, and that is the reason why knowledge of the distance of the emergence of the nerves from the femorotibial articular line is necessary.[28,30] In one-third of cases, when the common muscular branch is present, the operative procedure is simpler and safe. In the other cases, the surgeon can either dissect the nerve trunk, with the help of electrostimulation, to find the corresponding fascicles inside the tibial nerve, or extend the approach to perform the neurotomy directly on the branches after they arise from the tibial nerve.[23] It is possible to reach the deep part of the posterior compartment through a long longitudinal approach. In this study, a common muscular branch was found in only two cases, while in the other 14 patients the individualized motor branches were identified. The motor fibers inside the distal trunk of tibial nerve were dissected in 10 patients who had flexion deformity of the toes.

Tibial nerve can also be approached through a small popliteal fossa incision that gives easy access to the nerves to both heads of the gastrocnemius and the upper nerve to the soleus muscle. Through this approach, access to the lower nerve to the soleus muscle and those to the TP, FDL and FHL is impossible. Searching the corresponding fascicles inside the tibial nerve can lead to sensory and trophic disturbances.[29]

In this study the spasticity and abnormal foot posture had improved in 94% of cases, as measured by Ashworth scale. Most patients in this study (94%) had more comfort, less pain and ability to resume physical therapy with more functional gain. The same results were reported by Sindou et al.[3] who performed SPN of tibial nerve in 53 (62 sides) patients with spastic foot. In this study motor power was improve in both planter and dorsiflexors postoperatively, the same results were obtained by many authors.[23–26]

The muscular tone was diminished after SPN due to interruption of the afferent component of the both afferents and efferent fibers of the stretch–reflex at the level of a motor nerve. Recurrence of spasticity occurred in one patient after initial improvement because of insufficient amount of sectioning and was treated by botulinum toxin injections and physiotherapy. The recurrence could be explained by the fact that the nerve endings of intact α motor neurons sprout in an attempt to compensate for the partial denervation as regrowth has been prevented by the resection performed during neurotomy. New motor endplates appear, and the size of motor units increases in proportion to the degree of denervation.[31] On average, motor units increase by about five times their original size, allowing them to compensate for a loss of as much as 80% of the motoneuron pool.[8]

The outcome of SPN also intimately depends on the postoperative care after a successful surgery. The lower limb can be raised to avoid edema and mobilized as soon a possible, physical therapy beginning on the second postoperative day. An anti-thrombotic therapy is administered for 5 days after surgery. Immediately thereafter, a programme of physical and occupational therapy is undertaken for at least 6 weeks.[3]

5. Conclusion

In well-selected patients, SPN can yield good effects on refractory spasticity of the foot and its consequences. SPN can un-

mask residual motor function of muscles, make easier passive movements, increase comfort in daily activity, decrease pain because of spasticity and improve cosmetic appearance of the foot. Operative planning with a detailed clinical assessment of each spastic muscle is essential to determine the degree of nerve section, thus avoiding insufficient surgery or the loss of motor strength in the concerned muscles. A common muscular branch arising from near the tendinous arch of the soleus must be search during or prior to tibial neurotomy. In the two-thirds of cases where it does not exist, the surgeon must choose between an intra-fascicular dissection, especially in the antéro-lateral part of the nerve trunk, with the risk of postoperative sensory disturbances, or an extended approach to perform a safe neurotomy of the lower nerve to the soleus, the nerves to the tibialis posterior, the flexor digitorum longus and flexor hallucis longus.

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