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DOES FASCICULAR NEUROTOMY HAVE LONG-LASTING EFFECTS?

Hervé Collado, MD, Laurent Bensoussan, MD, Jean-Michel Viton, MD, Virginie Milhe De Bovis, MD and Alain Delarque, MD

From the Department of Physical and Rehabilitation Medicine, Faculté de Médecine, Université de la Méditerranée, Assistance Publique Hopitaux de Marseille, University Hospital La Timone, Marseille, France

Objective: **To determine whether fascicular neurotomy has long-lasting effects on spasticity.**
Design: **We present 4 clinical cases and a critical analysis of the literature.**
Patients: **This is a retrospective study on 4 patients referred to our department for spastic equinovarus foot deformity. For all 4, neurotomy was successful not long after surgery, but spasticity reappeared after a few months.**
Methods: **We compared our results with those in the PubMed database.**
Results: **Most publications acknowledge the immediate effectiveness of this surgery, but do not study the long-term effects of neurotomy. No publication proved long-lasting effects of neurotomy for spastic equinovarus foot deformity. The only long-term follow-up with a sufficient population is the one of Berard et al. (Pediatr Orthop B 1998, 7:66), who reported 61% recurrence.**
Conclusion: **There is no study showing that tibial nerve neurotomy has long-lasting effects. The 4 cases reported are an illustration that recurrence of spasticity may occur after neurotomy. These findings have to be taken into account for treatment decision-making and for provision of information to patients.**

Key words: neurotomy, spasticity, recurrence, equinovarus foot, stroke, brain injury.

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Correspondence address: Hervé Collado, Fédération de Médecine Physique et de Réadaptation, CHU Timone, 264 rue Saint-Pierre, FR-13005 Marseille, France. E-mail: hervecollado@neuf.fr

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INTRODUCTION

Selective neurotomy is considered safe, effective and long-lasting for treating patients with spastic equinovarus foot deformity. In their review, Deltombe et al. (1) concluded that neurotomy can cure spasticity over the long-term. For this reason, our medical department was surprised to receive 4 outpatients presenting with recurrence of spasticity after neurotomy. The aim of this study was to question the widely held notion that neurotomy has long-lasting effects on spasticity. We present here 4 clinical cases and a critical analysis of the literature.

PATIENTS AND METHODS

This is a retrospective study on 4 patients referred to our department for spastic equinovarus foot deformity. In 2 patients the spasticity was due to an ischaemic stroke, and in the other 2 due to traumatic brain injury. Three patients underwent surgery in adulthood. The remaining patient had traumatic brain injury in childhood and underwent neurotomy at 11 years of age. A different team performed the surgery for each patient. Impairments and disabilities were assessed, both before and after neurotomy. The degree of spasticity was assessed using the modified Ashworth scale (2, 3).

RESULTS

Case 1

A 62-year-old man presented with right carotid artery thrombosis resulting in a left hemiplegia. After 5 months he was disabled by a left hemiplegia with hemi-neglect and spasticity of the left lower limb, associated with hypoesthesia. A 10-m walk was possible using an ankle foot orthosis (AFO) and a tripod cane. Twenty-eight months after the stroke, spasticity increased and gait became impossible. Both the AFO and anti-spastic drugs became ineffective in controlling his equinus deformity and a chemical motor nerve block was carried out. His spasticity decreased and the gait improved and he proceeded to a selective left posterior tibial nerve fascicular neurotomy 34 months after the stroke.

Before neurotomy, soleus spasticity was 4/5 and gastrocnemius was 4/5 (Ashworth scale). Ankle flexion was measured to -10°, with knee extended and flexed, with marked varus in both cases. Standing and walking were impossible. Immediately afterwards, the spasticity of soleus was 1/5, gastrocnemius 3/5. Ankle flexion was 0° with the knee flexed and extended. Walking was now possible with an AFO and a simple cane.

His spasticity recurred 7 months after surgery. The spasticity of soleus rose to 3/5 and gastrocnemius to 4/5. Ankle flexion was measured to -10°, with knee extended and flexed and the patient was unable to walk. A few months later, impairments and disabilities increased. The assessment 14 months after surgery showed greater spasticity (soleus at 4/5 and gastrocnemius at 5/5 with clonus). Ankle flexion was -20°, with knee extended and flexed, with recurrence of varus. Walking was impossible. Thanks to an injection of botulinum toxin in the triceps surae, the spasticity and the equinus decreased and the patient could walk with a cane and an AFO.

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Case 2

A 45-year-old man presented a left ischaemic stroke (thrombosis of left carotid artery) resulting in a right hemiplegia with aphasia. After 8 months, he was unable to walk alone because of a right equinovarus foot. During the stance phase, the initial contact was on the lateral part of the forefoot, and during the entire gait cycle, there was no heel contact. He was able to walk only a few metres with both a cane on the left side and assistance on the other side. Four years after the stroke, a botulinum toxin injection showed good results on spasticity. He could walk alone, barefoot, with a cane. So, after further assessments, a neurotomy was planned after 53 months to maintain these good results in the long-term.

Pre-neurotomy, the spasticity of the soleus was 3/5 with clonus and gastrocnemius 2/5. Ankle dorsiflexion was measured to -30°, when the knee was extended and to -5° when the knee was flexed. He was able to walk only a few metres with both a cane on the left side and human assistance on the other side. During the stance phase, the initial contact was on the lateral part of the fore foot, and during the entire gait cycle, there was no contact of the heel (Fig. 1A). A fascicular neurotomy of 4/5 of soleus nerve and lateral and medial gastrocnemius nerve was performed. Post-operatively, the spasticity of soleus was 1/5 and was 0/5 in gastrocnemius muscles. Ankle dorsiflexion was measured to -10° (knee extended) and 0° (knee flexed). Functional ability had also improved and he was able to walk alone, barefoot and without a cane. During the stance phase, the initial contact of the right foot was plantargrade (Fig. 1B).

The recurrence of spasticity in soleus muscle (3/5) and gastrocnemius (4/5) was observed 19 months after surgery with clonus of the toe flexors. Ankle dorsiflexion was again -30° (knee extended) and -10° (knee flexed). During the stance phase, the initial contact was on the lateral part of the fore foot. Furthermore, during the swing phase, we observed an equinovarus right foot deformity with toe flexor spasms and a circumduction (Fig. 1D). The patient complained of ankle instability, a footdrop during the swing phase of gait and toe flexor spasms with pain on contact with the shoe. Walking barefoot was possible for only a few metres with frequent contacts between fore foot and ground (foot drag). Nonetheless, using an AFO, he could maintain the initial heel contact with the ground. Botulinum toxin was used to decrease spasticity (spasticity of soleus and gastrocnemius: 1/5) to achieve a more comfortable gait with less foot drag and less falls.

Case 3

A 27-year–old man presented with a right subdural haematoma, cerebral contusion and cerebral oedema following a traumatic brain injury. At 8 months after the injury, he still had a right hemiparesia and an equinovarus foot with prevalent spasticity in the soleus muscle. The patient had memory difficulties and features of a frontal lobe syndrome. Oral antispastic medication and a botulinum toxin injection were both unsuccessful. A chemical motor nerve block was thus carried out, which reduced spasticity and improved gait and a neurotomy was carried out at 12 months for longer term benefits. Pre-operative spasticity in soleus muscle was 4/5 with clonus, and 1/5 in gastrocnemius. Ankle dorsiflexion was measured to -35° (knee extended) and -15° (knee flexed). He walked with a cane. During stance phase, the initial contact

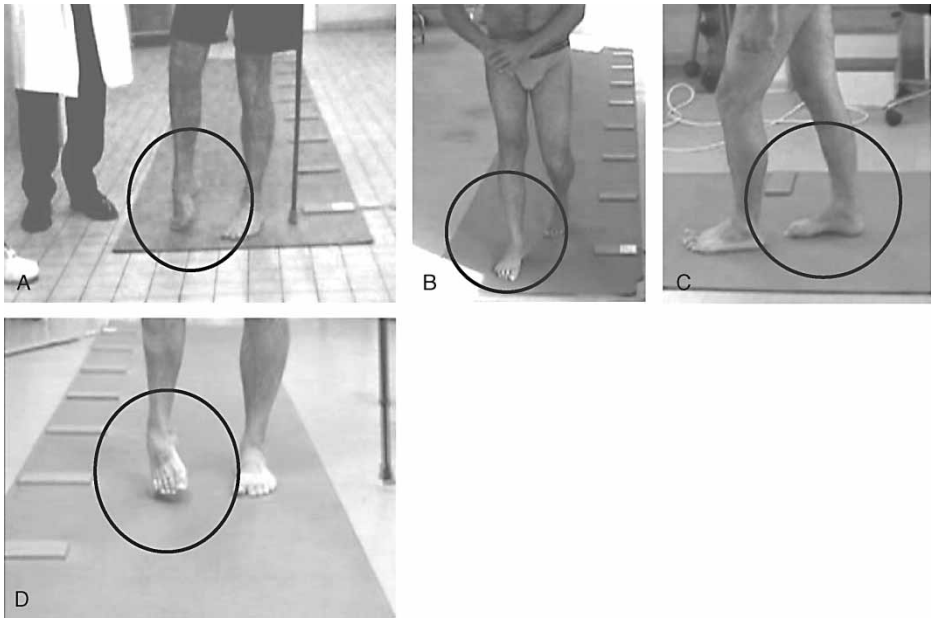

Fig. 1. Case 2. (A) Before neurotomy. Equinovarus foot, walk with human assistance and a cane. (B and C) Immediately after neurotomy. Regression of the equinovarus foot, walk with plantigrade contact with ground without assistance. (D) Recurrence of spasticity and equinovarus foot 19 months after surgery. Walk with a cane and the first contact with ground again with the fore foot.

with ground was on the forefoot and, during swing phase, there were forefoot dragged on the ground. A selective neurotomy of 4/5 of soleus nerve and nerve to tibial posterior muscle immediately produced a complete resolution in spasticity (0/5), clonus, and in the equinus (0° knee flexed and extended), and varus deformities. He was able to walk barefoot without a cane and with plantargrade contact with ground.

Spasticity recurred 29 months after surgery to a level of 3/5 in soleus and 1/5 in gastrocnemius muscles. Ankle dorsiflexion was measured to −45° (knee extended) and −15° (knee flexed). He needed a cane in the left hand for walking and the first contact with ground was on the fore foot. Botulinum toxin was thus injected 31 months after the injury, which reduced spasticity and improved ankle dorsiflexion to 0° knee flexed and extended. He could walk alone using an AFO with no foot drag.

Case 4

A 6-year-old boy was the victim of a traumatic brain injury and spent 13 days in an intensive care unit while comatose. He needed artificial ventilation and then tracheostomy. Imaging showed ischaemia of the middle cerebral artery territory and an intracranial haemorrhage (right thalamus and right Rolando area). At 12 months, he had a right hemiplegia with a non-functional right upper and lower limb, language difficulties and cognitive deficits. He suffered from a gradual increase in spasticity and the emergence of a direct equinovarus foot. Because walking was impossible and oral anti-spastic treatment was ineffective, a chemical motor nerve block was performed. Spasticity decreased and gait improved and a posterior tibial neurotomy was performed at 11 years of age. Pre-operatively, spasticity in soleus and gastrocnemius were 4/5 and 1/5, respectively. Ankle dorsiflexion was −5° (knee extended) and −10° (knee flexed). He was unable to walk alone. Immediately after tibial posterior neurotomy, he had better functioning and was able to walk independently barefoot with plantargrade contact.

Recurring spasticity was observed 11 years after surgery. The spasticity of the soleus and gastrocnemius was 3/5 and ankle dorsiflexion was measured to −10° with knee extended and flexed. Walking became impossible and a triple arthrodesis of the posterior foot with lengthening of Achilles tendon and tenotomy of toe flexor was carried out. Four years later, there was improvement of ankle dorsiflexion (−5°, with knee extended and flexed) and the same level of spasticity. He was able to walk only a few metres with both a cane on the left side and human assistance on the other side (Fig. 2). During stance, the initial contact was on the forefoot and there was no contact of the heel during the entire gait cycle.

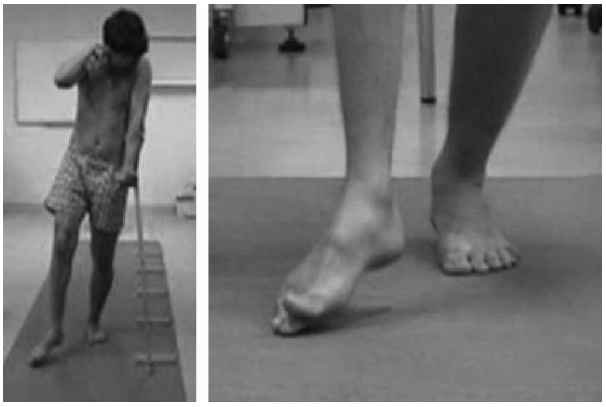

Fig. 2. Case 4. Recurrence of spasticity 11 years after neurotomy. Equinovarus foot with hallux erectus.

LITERATURE REVIEW

We reviewed the literature in this field with PubMed data using the keywords, "neurotomy", "spasticity", "anti-spastic treatment", "functional neurosurgery", "spastic foot" and "tibial nerve" (Table I). We found publications of 7 prospective studies. Berard et al. (4) and Sindou & Mertens (5) performed pre- and post-operative clinical assessments. Roujeau et al. (6), Caillet et al. (7), and Féve et al. (8) carried out clinical and electromyographic (EMG) evaluations. Privat & Privat. (9) and Decq et al. (10) reported on a large number of patients. Most publications reported the immediate effectiveness of surgery, but they did not study the long-term effects of neurotomy. Unfortunately, none specified their protocol and follow-up time was often short.

Only one study discussed the long-lasting effects of neurotomy. Berard et al. (4) carried out a prospective study including 13 unilateral selective tibial neurotomies for spastic foot in hemiplegic children among 100 hemiplegic children regularly followed up from 1989 to 1992. All patients were clinically and pre-operatively assessed by the same rehabilitation team and were postoperatively assessed 6, 12, 26 and 36 months after surgery and from then once each year. Spasticity was graded according to resistance to passive stretch, mobilization and ankle clonus. Gait data were pooled across patients for each limb involved, and video recordings were made with close attention to gait patterns. The results were considered excellent in 3 cases. Two cases needed additional corrective postoperative casts, and 8 cases had poor results with recurrence of the same deformity. The authors concluded that the effects of tibial neurotomy for spastic foot in hemiplegic children were transient in 61% of the cases, with a recurrence of the same deformity 5–17 months postoperatively. Histological data demonstrated that previously denervated muscle fibres were re-innervated into extensive motor units. The neurotomized muscles exhibited the characteristic patterns of denervated muscles that had been re-innervated by a collateral sprouting of a healthy axon nearby (anarchic mosaic pattern of the types 1, 2A and 2B fibres). They also found that among the 8 children with hemiplegic cerebral palsy the outcomes were poor (poor result for 6 patients and good result for 2 patients). In contrast, excellent results were found for acquired lesions and among the older children (5 cases: excellent result for 3 patients

Table I. *Review of literature*

	Patients	Methods	Results	Discussion
Berard et al. (4)	13 unilateral selective tibial neurotomies for spastic foot in hemiplegic children.	Clinical examination and video record. Preoperatively and postoperatively (6, 12, 26 and 36 months after the surgery and from then each year).	Excellent: 3 cases. Additional corrective postoperative casts: 2 cases.	61% of recurrence
Sindou & Mertens (5)	53 patients. – Lesions of the spinal cord: 12 – Cerebral hemisphere(s): 41	Clinical examination and video record (preoperative). Postoperative examination not detailed Re-examination at "regular" intervals (10 years).	Poor results: 8 cases. Spasticity improvement (92% of cases). Pain decrease. Increase of active ankle flexion (87% of cases). Functional improvement (82% of cases).	47% of the patients were followed only for 1 year, and only 28% were followed more than 4 years. 18% of unimproved cases (recurrence of equinus and toe flexor spasm). Functional improvement but they needed high shoes.
Roujeau et al. (6)	6 patients	Clinical examination. Follow-up: average of 29 months EMG	Stretch reflex: 0 (Held-Tardieu scale) in 7 limbs. But the exaggerated stretch reflex recurred in 3 limbs. Two unsuccessful patients among 6 (operated on again because of early spastic and functional recurrence).	Two unsuccessful patients among 6. Probative EMG results (4 patients). Small population.
Caillet et al. (7)	9 hemiplegic patients	Clinical examination. Recordings (VICON®) of kinematics parameters (with muscular electric activity recordings). Follow-up: 6 months	Stance knee hyperextension was corrected in 5 patients. Ankle dorsiflexion improved in 5 patients. Residual motricity improved in 2 patients. Subjective increase in gait comfort too.	No objective functional improvement was reported in the 6-month follow-up.
Fève et al. (8)	12 patients	Clinical examination. EMG Follow-up: 1 month.	Decrease of spasticity, but no increase in gait speed, cadence, and step length. Probative EMG results.	Short follow-up.
Privat & Privat (9)	159 patients	Method not described. Time of follow-up not known.	Spasticity decreased in all cases. A present function was improved in 44% of cases. A new function (gait) emerged in 22% of cases. Comfort increased in 27% of cases.	Different origins of spasticity and different types of neurotomies
Decq et al. (10)	392 neurotomies in 277 patients	Clinical examination. Kinematic analysis. Method not described and time of follow-up not known.	Disappearance of ankle clonus in all patients. Improvement of the angular variation of the second rocker of the ankle	

EMG = Electromyography.

and poor result for 2 patient). From this study, one can conclude that there are no long-lasting effects in 61% of the patients.

Sindou & Mertens (5) performed an important prospective study on 53 patients with a 2–17-year history of foot spasticity following spinal cord lesions in 12 patients and cerebral hemisphere lesions in 41 patients. Preoperative assessment consisted of a clinical analysis in stance and on walking with and without shoes and joint range of motion using photography and radiological imaging. There were however, no postoperative assessments, but outcomes 10 days after surgery were carried out just before discharge. All patients were then re-examined at regular intervals as outpatients. The authors recorded 62 tibial posterior neurotomies, that improved spasticity in 92% of cases (mean Ashworth score changed from 3.84 to 1.53), decreased pain and increased active ankle flexion in 87% of cases. The authors report functional improvement in 82% of cases, but orthopaedic shoes and/or splints could be discarded, only if the patients were wearing above-ankle boots to maintain ankle stability. Recurrence of equinus and toe flexor spasms occurred in the 18% of failures. Although follow-up was designed for 10 years, 47% of the patients were followed only for one year and only 28% were followed more than 4 years.

Roujeau et al. (6) reported on 6 fascicular tibial neurotomy patients (diplegic, paraplegic, tetraplegic and hemiplegic) with spastic equinus foot. Pre- and post-operative included the passive range of joint motion, the Held-Tardieu scale as a stretch reflex score and the quality of motor control of dorsiflexion. Immediately after operation, the stretch reflex with the knee extended was scored as 0 on the Held-Tardieu scale in 7 limbs. However, exaggerated stretch reflexes recurred in 3 limbs. The authors reported failure in 2 of the 6 patients, who were re-operated because of early recurrence of spasticity, which interfered with function. Patient follow-up was adequate (average 29 months), but the numbers were small. In addition to spasticity and functional improvement, the authors reported some EMG results (decrease in H_{max} and in the H_{max}/M_{max} ratio) in 4 patients.

Other studies had shorter follow-ups. Caillet et al. (7) followed up 9 hemiplegic patients for 6 months. In addition to clinical examination, three-dimensional recordings (VICON® Vicon Peak, California, USA) of kinematics parameters and muscle electric activity recordings (both before neurotomy and 6 months post-neurotomy) were performed. The latter showed that stance knee hyperextension was corrected in 5 patients. On the affected side, during stance phase, ankle dorsiflexion improved in 5 patients, and during the swing phase residual motricity improved in 2 patients. They also describe a subjective increase in gait comfort, but objective functional improvement was not seen at 6-months.

Similarly, Fève et al. (8) confirmed a decrease in spasticity over a 1-month period on the basis of EMG parameters (decrease in H_{max} and in the H_{max}/M_{max} ratio) in 12 patients. Spasticity reduced slightly, but gait speed, cadence and step length were not increased.

Finally, 2 studies reported on a large series of patients, but, unfortunately, the period of follow-up is not known. Privat & Privat (9) reported on 159 heterogeneous patients following peripheral sectorial neurotomies (different causes of spasticity and different types of neurotomies). Spasticity decreased in all cases. Function improved in 44% of cases with a new function (gait) emerging in 22% of cases and increased comfort in 27% of cases. The study method was not described.

Decq et al. (10) performed 392 neurotomies in 277 patients (66% posterior tibial neurotomies). This led to the disappearance of ankle clonus in all patients and kinematic analysis showed that, during stance phase, the procedure improved angular variation of the second ankle rocker. As in the last study, neither the method nor the period of follow-up was described.

In these 2 studies, the methods used were unable to prove long-lasting effects of neurotomy.

None of the studies have shown any long-lasting effects of neurotomy in the treatment of spastic equinovarus foot deformity and all were based on short-term outcomes and impairments rather than on activities. The sole study providing long-term follow-up in a sufficiently large population is that of Berard et al. (4), who reported a 61% recurrence.

DISCUSSION

The aim of this study was to question the widely held notion that fascicular neurotomy had long-lasting effects on spasticity. We reported on 4 clinical cases and critically reviewed the literature to show that recurrence is high.

Patients 1 and 2 both had a stroke and evolved in a similar way through a successful neurotomy, the reappearance of spasticity after 7 months and 19 months, respectively, and rapid loss of the post-operative functional improvements. Botulinum toxin and anti-spastic oral medication were unsuccessful in patient 3 and a decrease in spasticity after a motor nerve block led to a neurotomy, which quickly resolved clonus, varus, and equinus. Twenty-nine months later, triceps hypertonia, clonus and a limited range of motion were noted and the patient showed the same functional status as before surgery.

Patient 4 had a neurotomy in childhood. Its eventual failure is in line with the results of Berard et al. (4), who was working only with children and found less favourable outcomes than with adults. In addition, they also found marked differences between children and children with congenital hemiplegic cerebral palsy who did worse, whereas there were better outcomes for older children with acquired hemiplegia, as for adults. These observations raise questions about the influence of the age and of the cause of spasticity on the long-term efficiency of neurotomy.

Why spasticity reappears after neurotomy remains unclear. Together with persisting lesions of the central nervous system, a reorganization of peripheral circuits is likely to be involved in

this phenomenon. Two mechanisms may explain the reinnervation, as shown by Decq (11); firstly, the axonal growth of the cut motor neurones and secondly the "sprouting phenomenon" described by Hoffman in the 1950s (12). The sprouting of motor neurone endings leads to the development of new motor endplates on denervated muscles and this phenomenon increases motor unit size, as shown by Rafuse et al. (13). This physiological phenomenon, however, concerns essentially motor reinnervation, which explains the recovery of weakness after neurotomy. Sensory reinnervation of afferent fibres, responsible for the segmentary reflex arc and thus for spasticity, may be more uncertain (origin of neurotomy effectiveness). This reinnervation seems to be anarchic and not specific for muscular receptors (14). Collins et al. (15) stated that this proprioceptive sensory reinnervation is not functional and seems to be responsible for the success of neurotomy, as described in the literature. But from a histological point of view, how can clinical and functional recurrences be explained? Deltombe et al. (1) hypothesized about agonist–antagonist imbalance, but this hypothesis does not explain the real spastic recurrence in the 4 patients assessed with Ashworth score measure.

Our patients are not representative of the whole group and only those who fail following neurotomy are referred to the Department of Rehabilitation Medicine. They therefore expect other therapeutic solutions. But do neurosurgical teams take the whole group of patients into account? And is the duration of longitudinal studies sufficient? It appears that we need studies reporting long-term follow-up to establish the efficiency of neurotomy.

The review presented in this article shows that no study has proved that tibial nerve neurotomies have long-lasting effects on spasticity. The 4 cases reported are an illustration that spasticity may recur after neurotomy. These findings have to be taken into account for the treatment decision-making process and for providing information to patients. The explanation for this recurrence remains unclear and will need further study.

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