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LETTER TO THE EDITOR

DOES FASCICULAR NEUROTOMY HAVE LONG-LASTING EFFECTS?

Sir,

We read with interest the recent article by Collado et al. (1) on the recurrence of spasticity after tibial neurotomy observed in 4 cases. Although the methodology of the study is controversial (e.g. how many patients undergoing neurotomy were followed?), we agree that precise information about the long-lasting effects of our treatment is essential, especially in the rehabilitation field. Most of all, we think that the term "recurrence of spasticity" is inappropriate. The term "deformity recurrence" (the exact term used by Berard in the article cited by Collado et al.) would be more appropriate, as the recurrence observed by Collado et al. is probably not related to spasticity but to pathological motor activation pattern and musculo-tendinous retraction. Several facts lead us to this conclusion.

First, spasticity is usually defined as a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex (2). Neurotomy results in a section of the afferent fibres mediating the spastic monosynaptic reflex arc, leading to reduction of spasticity and osteo-tendinous reflex and clonus disappearance. The long-lasting effect of neurotomy to reduce the monosynaptic reflex arc has been demonstrated in 3 studies by means of H_{max}/M_{max} ratio permanent reduction, with a mean follow-up of 5 months, 24 months and 29 months, respectively (3–5). The related functional improvement obtained after neurotomy has been confirmed in a multicentre study with a mean follow-up of 10 months (6). Neurotomy also results in sectioning of the efferent motor fibres, which is responsible for a transient muscle weakness. Such weakness recovers thanks to collateral re-innervation, which is correlated with the return of the Mmax amplitude (corresponding to the sum of the motor units) to baseline value 8 months after the neurotomy (5). The recovery of the voluntary (and involuntary) muscle strength explains the recurrence of the pathological motor activation pattern, which is sometimes implicated in the equinovarus deformity. This also explains the recurrence of deformity after neurotomy in the case of dystonic patterns that are not related to an increase in tonic stretch.

Secondly, Collado et al. evaluated triceps spasticity with the Ashworth scale (all the patients were graded Ashworth 3 or 4). Although the Ashworth scale is commonly used in the literature, it is confounded by contracture, as increased resistance to movement is not exclusively dependent on stretch reflex activity, but is also due to increased stiffness as a result of contracture. The Tardieu scale seems more appropriate, especially to evaluate triceps spasticity (7). Moreover, all the patients had ankle dorsal flexion limitation in the pre-operative evaluation (ranging from –5° to –35°), which had worsened in the post-operative evaluation (ranging from –10° to –45°) leading to the suggestion that the triceps muscle shortening noted before the neurotomy is enhanced after it. In Berard's article, cited by the authors, the equinovarus deformity recurrence was correlated with triceps muscle shortening, while the spasticity evaluation was not detailed. Moreover, Berard evaluated children with hemiplegia with growth potential and higher risk of muscle shortening. As a denervated muscle risks retraction, triceps muscle shortening is a relative contra-indication to neurotomy, and special attention must be paid to the rehabilitation program, with stretching and posture training of the triceps muscle.

There is no doubt that Collado et al. noted equinovarus deformity recurrence after neurotomies. The recurrence can be caused by the logical recovery of a pathological motor activation pattern associated with a muscular retraction following the denervation. That is why experienced surgical teams prefer to section the motor nerve branches to the soleus muscle (which is, in most cases, responsible for the triceps clonus) and to spare the motor nerve branches to the gastrocnemius muscles which, as a bi-articular and fusiform muscle, are at higher risk of retraction (8). We have doubts as to the spasticity implication in such recurrence. If the spasticity is considered as a hyper-excitability of the stretch reflex, regarding the literature and our personal experience, neurotomy undoubtedly has long-lasting effects. We have never seen a clonus recurrence in a muscle whose nerve has been partially sectioned. The main questions are what are the frequency and causes of the equinovarus deformity sometimes recurring: a pathological motor pattern and a triceps muscle shortening (especially when the gastrocnemius nerves are treated) may explain such recurrence.

Collado et al.'s observation emphasizes the need for long-term clinical follow-up after neurotomy, for a well-defined rehabilitation programme, and the need for an interdisciplinary approach (integrating physical medicine and rehabilitation specialist, neurosurgeon and orthopaedic surgeon) to select the patients.

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RESPONSE TO LETTER TO THE EDITOR BY DELTOMBE ET AL.

We read with interest the letter to the Editor by Deltombe et al. about our paper entitled: *Does fascicular neurotomy have long-lasting effects?* The comments focus on the physiology of spasticity and associated altered motor patterns in stroke patients before and after tibial nerve neurotomy. This issue remains, however, controversial. The aim of our study was not to discuss this point, but rather to determine if there is evidence that tibial nerve neurotomy has long-lasting effects on the impairments and disabilities related to spasticity and associated altered motor patterns. Indeed, the literature review showed that there was no study proving long-lasting effects of tibial nerve neurotomy. Even in the study by Buffenoir et al. (6, above), the mean interval after neurotomy is 10 months, which is rather short when discussing the effects of a surgical procedure. This is of major importance for treatment decision-making and for patient information.

When going into details, we are aware that the population included in our study was rather small, but since we had noted the recurrence of spasticity among patients who had undergone neurotomy, we felt it was worth reviewing the literature on this topic.

As regards the term "deformity recurrence", we have noted that when speaking about the recurrence of spasticity, Berard et al. (1) reached the following conclusion in his summary: "Histologic data clearly demonstrate that previously denervated muscle fibers were reinnervated carrying into extensive motor units. This finding can explain the recurrence of the foot spasticity and deformity in neurotomized children."

In addition, close examination of the results we obtained on spasticity and clonus in the 4 neurotomized patients mentioned in our paper showed that, in addition to the systematic recurrence of spasticity, 2 of them (patients 2 and 3) also had the recurrent clonus. Along similar lines, Roujeau et al. (5, above) reported the complete disappearance of the clonus immediately after neurotomy, but at the end of the study (24 months later), the neurotomy had effectively abolished the clonus in only 6 of the 7 operated lower limbs. In this same study, using the Held Tardieu scale, the authors reported that the exaggerated stretch reflex recurred in 3 of the 7 operated lower limbs. It should be pointed out, however, that the Tardieu scale has not yet definitely been proved to be a relevant means of assessing spasticity (2).

As regards the electromyographic approach to spasticity, although we feel that clinical methods should always be the main techniques used to assess spasticity, several studies have indeed shown the immediate effectiveness of this surgical approach, in terms of the decrease in the H_{max}/M_{max} ratio obtained. However, in the study by Feve et al. (3, above), the authors carried out electromyography only one month after surgery. In the study by Roujeau et al. (5, above), electromyography was performed up to 24 months after surgery, and in the study by Deltombe et al. (4, above), it was performed after an interval of 29 months. In view of these rather short intervals, we do not feel one can be absolutely sure of the long-term effectiveness of this type of surgery.

To assess spasticity in our clinical practice, we use the Ashworth scale, although, like many of the other scales and scores currently used in medicine, it is not perfect. Many authors, such as Feve et al. (3, above), Sindou & Mertens (3) and Decq et al. (4) have used this scale, and the Tardieu scale has not yet definitely been proved to be a relevant means of assessing spasticity (2).

Further studies are now required on much larger populations, and sufficient hindsight is also necessary to enable us to assess the neurotomy technique more closely. Studies of this kind should be carried out jointly by neurosurgical, orthopaedic and rehabilitation departments.

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