DO OVERSTRETCHED SARCOMERES CAUSE MUSCLE CONTRACTURE IN CEREBRAL PALSY?

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INTRODUCTION

Long term spasticity may lead to muscle 'contracture', which is defined as a permanent tightening of the muscle This permanent tightening is alleged to be associated with several intra-muscular processes of adaptation such as an increase of intra-muscular connective tissue (Booth et al, 2001 and Vattanasilp et al,2000), and a reduction of the number of sarcomeres arranged in-series within a muscle fiber (Farmer and James, 2001, Tardieu et al, 1982 Vattanasilp et al, 2000). However, such adaptation is controversial: the suggested adaptation of in-series sarcomeres has never been proven *in vivo* to contribute to the contracture of the spastic human muscle. Still, because the success of surgical interventions such as tenotomy and tendon lengthening in decreasing joint deformities suggests that spastic muscles are indeed shorter than non-spastic muscles, the aim of the present study is to test the hypothesis that an altered force-length curve, due to a reduced number of in-series sarcomeres, may cause muscle contracture.

MATERIALS AND METHODS

Ten patients participated. Prior to tenotomy, FCU muscle length was measured in maximally flexed, neutral, and maximally extended position of the wrist. After tenotomy and surgical dissection active and passive isometric length-force curves were measured, using an instrument designed for this purpose (Fig 1).

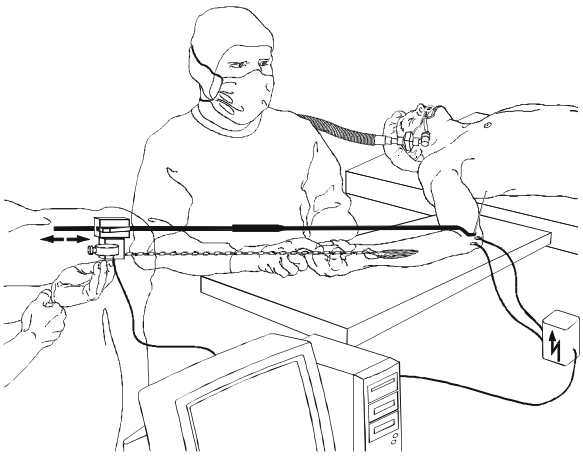

Figure 1: A method to measure length-force curves during transposition surgery, using a force transducer fixed to the humerus, transcutaneous, supra-maximal electrical stimulation and a data-acquisition system (Smeulders MJC et al, unpublished).

RESULTS AND DISCUSSION

Optimum length (l_{opt}, i.e. the length at which optimal force, mean Fmao = 87.4 N is exerted) did not differ from the FCU length in neutral position of the wrist (l_{neut}) (Fig 2). For muscle lengths, corresponding to the full range of wrist joint motion (from maximal flexion to maximal extension), mean active FCU force never decreased below 70 % of optimum force. This indicates that FCU length range is arranged symmetrically around optimum length for this movement, i.e. a small part of the ascending limb and a small part of the descending limb of the length force curve was used. Even at maximal wrist extension, passive FCU force was never excessively high (Fmp<10.6%Fmao). This implicates abundant overlap, rather than maximal stretching, of the sarcomere's myofilaments. Therefore, the limited wrist extension was not caused by over-stretching of the sarcomeres.

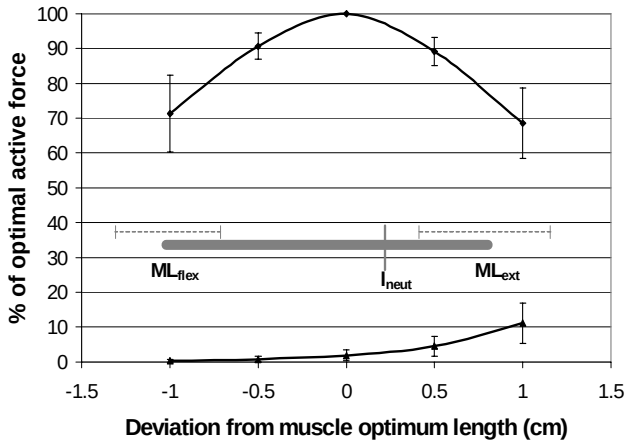

Figure 2: Average force length profile (black line) of the spastic FCU muscle in relation to the FCU muscle length during wrist range of motion (grey bar) of eleven patients. The error bars show the 95% confidence interval of force-data at four selected lengths. The dashed lines show the 95% confidence interval for the average muscle length with the wrist in flexion (MLflex) and the wrist in extension (MLext). The vertical grey line shows the muscle length with the wrist in neutral position (lneut).

Because 1) the l_{opt} of the spastic FCU did not differ significantly from its l_{neut}, 2) the FCU still exerted considerable force at maximal wrist extension, and 3) the passive resistance at maximum wrist extension was low, the hypothesis that the spastic muscle causing a joint deformity in cerebral palsy patients has an altered force-length curve as a result of a reduced number of in-series sarcomeres was rejected. It is concluded that the properties of the surgically dissected FCU muscle do not explain the flexion deformity of the wrist. It is hypothesized that the deformity is explained by the interactions of the FCU muscle with neighbouring muscles and extramuscular tissues connected to the bones.

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