ACUTE EFFECTS OF INTRAMUSCULAR APONEUROTOMY ASSESSED BY FINITE ELEMENT MODELING

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INTRODUCTION

Aponeurotomy is a surgical technique executed to correct functional problems due to contractures occurring secondary to spastic paresis. Despite its clinical potential importance, the mechanical mechanism of this intervention is not clearly understood. The goal of this study was to investigate this mechanism using finite element modeling. The specific point addressed is modeling changes in lengths of sarcomeres within aponeurotomized muscle due to intramuscular myofascial force transmission [e.g., 1].

METHODS

Finite element modeling of extensor digitorium longus muscle of the rat [see 2 for a description of the model] was performed for two conditions (1) intact and (2) aponeurotomized.

In surgery, aponeurotomy is performed by cutting the intramuscular aponeurosis transversely to its line of pull. In the present study, proximal aponeurotomy was modeled by disconnecting the common nodes of two neighboring aponeurosis elements located in the middle of the proximal aponeurosis of the modeled muscle. Earlier experiments on dissected aponeurotomized rat muscle showed after isometric lengthening that below the location of the intervention a progressive tear occurred in the intramuscular connective tissue in the direction of muscle fibers [3]. As a consequence, a gap opened between the cut ends of the aponeurosis. Present modeling of aponeurotomized muscle allows such a gap and a limited tear.

RESULTS AND DISCUSSION

Major changes in muscle length-active force characteristics were found (Figure 1) after aponeurotomy (1) both active slack length (by approximately 1.2 mm) and optimum length (by 2.0 mm) shifted to higher muscle lengths and the length range of force exertion increased. (2) Muscle active force decreased (e.g., optimum force decreased by 21%).

On lengthening after aponeurotomy, two cut ends of the proximal aponeurosis were separated by a gap and tearing of intramuscular connective tissue along the fiber interface caused the muscle to be divided into a proximal and a distal population of muscle fibers.

Unlike the fairly homogeneous strains in the fiber direction of the intact muscle (Figure 2a), for the aponeurotomized muscle myofascial force transmission was shown to lead to a major strain distribution and thus sarcomere length distributions. Note the contrasting effects for the two fiber populations (Figure 2b): (1) In the distal population (fibers with no myotendinous connection to the muscles' origin), sarcomeres were much shorter than the ones of the proximal population (fibers with intact myotendinous junction at both ends) (2) From proximal ends of muscle fibers to distal ends, the serial distribution of sarcomere lengths varied from the lowest length to higher lengths in the distal population and in a reversed manner in the proximal population. Such distributions of sarcomere lengths explain the shifts in muscle active slack and optimum lengths. Muscle force reduction was explained primarily by the short sarcomeres in the distal population. However, fiber stress distributions showed that majority of the sarcomeres (even in the distal fiber population) still contribute to muscle force: myofascial force transmission prevents them from shortening to their active slack lengths.

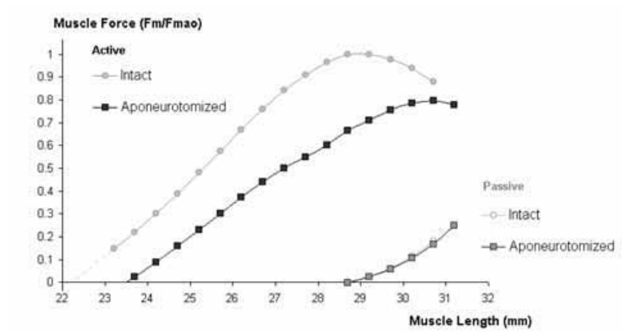

Figure 1: The isometric muscle length-force characteristics of modeled intact and aponeurotomized EDL muscles.

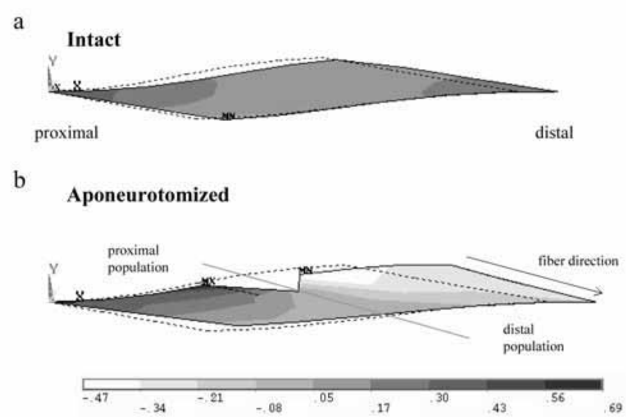

Figure 2: Distributions of fiber direction strain within modeled (a) dissected intact and (b) dissected aponeurotomized EDL muscles at high muscle length.

CONCLUSIONS

We conclude that the mechanism mechanically determining the effects of aponeurotomy is dominated by myofascial force transmission. Accounting for the general characteristics of sarcomere length changes shown in the proximal and distal populations of muscle fibers may help the surgeon make a more controlled decision on the location of the intervention.

REFERENCES

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