Passive mechanical properties of muscle fibers in hamstring contractures of children with spastic cerebral palsy

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INTRODUCTION

Contractures develop in patients with spastic cerebral palsy (CP). However, the mechanism of contracture is unknown. Contractures represent an inability of the muscle to increase length. The elements that are responsible for this stiffness are also unknown [1]. It could result from changes on multiple scales:

• Protein: Titin is a major load bearing protein in muscle
• Cell: Muscle cells mechanical properties could be altered
• Tissue: Tissue level includes ECM elements and connections
• Structural: The organization of the muscle effects mechanics

Previous work has shown that single fibers from contracted muscle tissue have increased passive stiffness that could lead to the overall muscle stiffness [2]. The opposite result was observed when scaled to muscle fiber bundles as bundles from typically developing (TD) children are stiffer than contracted bundles [3]. Studies have also demonstrated in the upper extremity that larger sarcomeres are seen in-vivo of contracted muscles [4]. Our study investigates these effects in specific hamstring muscles evolved in gait, gaits (GR) and somnolent (ST).

METHODS

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CONCLUSIONS

• Protein level: Titin isoform is not altered in CP
• Cell level: CP fibers are not stiffer than control fibers
• Tissue level: Bundle stiffness does not correlate with titin isoform
• Tissue level: Bundle stiffness does account for increased passive tension of contracture tissue in CP muscle
• Collagen Content is also increased in contracture of CP muscle
• Structure level: CP muscle in contracture has longer sarcomere lengths
• When combined with tissue changes, indicates much larger in vivo stress of CP muscle
• Sarcomere length is correlated with CP severity measures

REFERENCES