SCIENTIFIC ARTICLE

Contributions of Muscle Imbalance and Impaired Growth to Postural and Osseous Shoulder Deformity Following Brachial Plexus Birth Palsy: A Computational Simulation Analysis

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Purpose Two potential mechanisms leading to postural and osseous shoulder deformity after brachial plexus birth palsy are muscle imbalance between functioning internal rotators and paralyzed external rotators and impaired longitudinal growth of paralyzed muscles. Our goal was to evaluate the combined and isolated effects of these 2 mechanisms on transverse plane shoulder forces using a computational model of C5−6 brachial plexus injury.

Methods We modeled a C5−6 injury using a computational musculoskeletal upper limb model. Muscles expected to be denervated by C5−6 injury were classified as affected, with the remaining shoulder muscles classified as unaffected. To model muscle imbalance, affected muscles were given no resting tone whereas unaffected muscles were given resting tone at 30% of maximal activation. To model impaired growth, affected muscles were reduced in length by 30% compared with normal whereas unaffected muscles remained normal in length. Four scenarios were simulated: normal, muscle imbalance only, impaired growth only, and both muscle imbalance and impaired growth. Passive shoulder rotation range of motion and glenohumeral joint reaction forces were evaluated to assess postural and osseous deformity.

Results All impaired scenarios exhibited restricted range of motion and increased and posteriorly directed compressive glenohumeral joint forces. Individually, impaired muscle growth caused worse restriction in range of motion and higher and more posteriorly directed glenohumeral forces than did muscle imbalance. Combined muscle imbalance and impaired growth caused the most restricted joint range of motion and the highest joint reaction force of all scenarios.

Conclusions Both muscle imbalance and impaired longitudinal growth contributed to range of motion and force changes consistent with clinically observed deformity, although the most substantial effects resulted from impaired muscle growth.

Clinical relevance Simulations suggest that treatment strategies emphasizing treatment of impaired longitudinal growth are warranted for reducing deformity after brachial plexus birth palsy. *(J Hand Surg Am. 2015;40(6):1170−1176. Copyright © 2015 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Brachial plexus birth palsy, shoulder deformity, computer simulation, muscle strength, impaired growth.

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BRACHIAL PLEXUS BIRTH PALSY IS A perinatal injury to the peripheral nerves supplying the muscle of the upper limb, estimated to affect 0.4 to 4 per 1,000 newborns.[1] Injury most commonly occurs at the C5−6 level, resulting in paralysis of elbow flexors and muscles crossing the shoulder.[2] Postural and osseous deformities, including contractures of the shoulder and elbow,[3,4] abnormal morphology of the scapula and humerus, and subluxation of the humeral head,[5−7] are common sequelae of the nerve injury, even when spontaneous nerve regeneration is observed. Such deformity has a substantial negative impact on patient function and quality of life and motivates improved understanding of the etiology of the deformity to enhance treatment.

Recent literature suggests 2 potential muscular mechanisms underlying the clinically observed postural and osseous deformity: muscle imbalance and impaired longitudinal muscle growth. One long-held hypothesis suggests that the imbalance between paralyzed external rotators and intact internal rotator muscles at the shoulder leads to the internal rotation contractures, or restricted range of motion (ROM) and increased joint stiffness, that have been observed.[1,2,8] More recently, experiments in rodent models suggested that perinatal nerve injury may result in impaired longitudinal growth of the paralyzed muscles, resulting in reduced resting fiber length and overstretched sarcomeres.[9,10] A recent computational sensitivity analysis[11] investigating the potential biomechanical influence of these mechanisms on the joint reaction forces at the glenoid and on shoulder ROM suggested that both mechanisms have the potential to influence glenoid force, whereas impaired longitudinal growth may influence ROM. However, in that study, the roles of individual muscles were studied; it is unknown whether the combined effect of both mechanisms acting through multiple muscles has a potentially synergistic influence.

The goal of this study was to evaluate the combined and isolated effects of impaired longitudinal growth and muscle imbalance using a computational model of C5−6 brachial plexus injury on transverse plane mechanical forces at the shoulder. The model represented the clinically observed pattern of injury and muscle changes based on the best available descriptions of normal neuroanatomy, data from experimental animal models, and previously published clinical findings. In particular, we evaluated the effect of each mechanism on shoulder rotation ROM and transverse plane joint reaction forces at the glenohumeral joint.

MATERIALS AND METHODS

We implemented simulations of brachial plexus birth palsy using a computational model of the upper limb,[12,13] as previously augmented for dynamic simulation of brachial plexus palsy.[11] Simulations were performed in OpenSim (version 3.1, Stanford, CA),[14] an open-source musculoskeletal simulation software platform. Briefly, the model integrates representations of bone and joint geometry for the shoulder, elbow, forearm, and wrist with models of the path and force-generating capacity of the 32 muscles crossing these joints. The muscles crossing the glenohumeral joint include the anterior deltoid, middle deltoid, posterior deltoid, teres major, teres minor, supraspinatus, infraspinatus, subscapularis, pectoralis major (clavicular head and 2 compartments of sternocostal head), biceps (long and short heads), triceps (long head), latissimus dorsi (3 compartments), and coracobrachialis. The subscapularis muscle was augmented from a previous implementation of the model to include 2 compartments: an upper compartment that accounts for one third of the total muscle mass, and a lower compartment accounting for two thirds of the muscle mass.[15] The origin-to-insertion paths of the muscles are based on anatomical description and experimental measurements of muscle moment arm,[12] which is the distance from the muscle line of action to the center of rotation of the joint. The force of each muscle actuator depends on posture of the arm, the level of muscle activation from unactivated (0%) to fully activated (100%), and architectural characteristics of the individual muscle. These characteristics include optimal muscle fiber length, peak isometric force (related to muscle physiologic cross-sectional area), pennation angle, and tendon length, and are derived from experimental measurements made from cadaveric specimens and MRIs of living subjects.[16−22] Representations of ligaments and other passive structures were included to limit movement at the extremes of the ROM.[13,23] The model permits calculation of joint moment, the product of muscle force and moment arm, which indicates a muscle's ability to contribute to or resist rotation at a joint.

We classified muscles crossing the shoulder as either affected or unaffected by a C5−6 level injury (Table 1), according to neuroanatomy, available clinical findings, magnetic resonance imaging (MRI), electrodiagnostic testing, muscle biopsies, and motion analysis.[6−10,15,24-30] "Affected" refers to initial denervation from the neurological injury, regardless of whether reinnervation occurs. "Unaffected" refers to muscles without interruption of innervation from