# Computational Sensitivity Analysis to Identify Muscles That Can Mechanically Contribute to Shoulder Deformity Following Brachial Plexus Birth Palsy

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**Purpose** Two mechanisms, strength imbalance or impaired longitudinal muscle growth, potentially cause osseous and postural shoulder deformity in children with brachial plexus birth palsy. Our objective was to determine which muscles, via either deformity mechanism, were mechanically capable of producing forces that could promote shoulder deformity.

**Methods** In an upper limb computational musculoskeletal model, we simulated strength imbalance by allowing each muscle crossing the shoulder to produce 30% of its maximum force. To simulate impaired longitudinal muscle growth, the functional length of each muscle crossing the shoulder was reduced by 30%. We performed a sensitivity analysis to identify muscles that, through either simulated deformity mechanism, increased the posteriorly directed, compressive glenohumeral joint force consistent with osseous deformity or reduced the shoulder external rotation or abduction range of motion consistent with postural deformity.

**Results** Most of the increase in the posterior glenohumeral joint force by the strength imbalance mechanism was caused by the subscapularis, latissimus dorsi, and infraspinatus. Posterior glenohumeral joint force increased the most owing to impaired growth of the infraspinatus, subscapularis, and long head of biceps. Through the strength imbalance mechanism, the subscapularis, anterior deltoid, and pectoralis major muscles reduced external shoulder rotation by  $28^{\circ}$ ,  $17^{\circ}$ , and  $10^{\circ}$ , respectively. Shoulder motion was reduced by  $40^{\circ}$  to  $56^{\circ}$  owing to impaired growth of the anterior deltoid, subscapularis, and long head of triceps.

**Conclusions** The infraspinatus, subscapularis, latissimus dorsi, long head of biceps, anterior deltoid, pectoralis major, and long head of triceps were identified in this computational study as being the most capable of producing shoulder forces that may contribute to shoulder deformity following brachial plexus birth palsy.

**Clinical relevance** The muscles mechanically capable of producing deforming shoulder forces should be the focus of experimental studies investigating the musculoskeletal consequences of brachial plexus birth palsy and are potentially critical targets for treating shoulder deformity. (*J Hand Surg Am. 2014;39(2):303–311. Copyright* © *2014 by the American Society for Surgery of the Hand. All rights reserved.*)

Key words Biomechanics, brachial plexus, deformity, shoulder, simulation.

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NE IN 3 CHILDREN AFFECTED BY brachial plexus birth palsy (BPBP) sustains permanent osseous and postural deformity, 2 types of shoulder deformity that can interfere with upper limb strength and mobility.<sup>1</sup> Osseous deformity, characterized by posterior humeral head subluxation and glenoid retroversion, is thought to arise from a persistent, posteriorly directed glenohumeral joint force that both shifts the humeral head posteriorly and compresses the posterior glenoid margin.<sup>2-4</sup> In addition, children with BPBP develop contractures that cause postural deformity by reducing passive external shoulder rotation and abduction range of motion (ROM).<sup>1,5,6</sup> Both osseous and postural shoulder deformities are attributed to mechanical forces that, over time, alter the shape and behavior of bone and soft tissues at the shoulder.

Identifying the muscles that could produce the observed deformities is critical for developing treatments to prevent or reverse shoulder deformity following BPBP. Two mechanisms, strength imbalance and impaired longitudinal muscle growth, have been proposed to explain the biomechanical etiology of shoulder deformity.<sup>7–14</sup> Previous studies suggest that a strength imbalance between unimpaired internal shoulder rotator muscles and paralyzed external shoulder rotator muscles produces an internal rotation contracture.<sup>7-12</sup> A second, recently proposed mechanism posits that muscle paralysis impairs longitudinal muscle growth, increasing passive forces that muscles exert on the shoulder joint.<sup>13,14</sup> Both the strength imbalance and the impaired growth deformity mechanisms could change shoulder mechanical forces. However, the biomechanical etiology of shoulder deformity in children with BPBP has not been clearly established.

Clinical and biological experiments are useful for measuring changes in muscle properties (such as muscle functional length, muscle size, and muscle path) and osseous deformity following BPBP, but it is difficult to directly quantify the mechanical consequences of these changes in vivo. In contrast, computational musculoskeletal models can be used to predict which muscles or anatomical changes are mechanically capable of producing posteriorly directed and compressive glenohumeral joint forces or limiting shoulder ROM consistent with deformity. Such an analysis would narrow the potential muscles and mechanisms underlying deformity and provide a basis for future experiments that would focus on evaluating these candidate muscles in a more targeted way. Upper limb computational models have been used to investigate the biomechanics of numerous orthopedic issues, including tendon transfers,<sup>15,16</sup> wheelchair propulsion,<sup>17</sup> nerve injury,<sup>18</sup> and clavicle fractures.<sup>19</sup>

The objective of this study was to identify the shoulder muscles that were mechanically capable of producing forces in directions that could produce the shoulder deformity observed in children with BPBP via either the strength imbalance or the impaired longitudinal muscle growth deformity mechanism. Upon performing a sensitivity analysis, we identified muscles that could increase the posteriorly directed, compressive glenohumeral joint force associated with osseous deformity or reduce the external shoulder rotation or abduction ROM consistent with postural deformity. We hypothesized that muscles crossing the posterior aspect of the shoulder could mechanically contribute to osseous deformity and that muscles that internally rotate or adduct the shoulder could mechanically contribute to postural deformity.

### **METHODS**

### **Musculoskeletal model**

We used a 3-dimensional computer model of the upper limb musculoskeletal system<sup>20</sup> to perform a sensitivity analysis to assess the impact of 2 potential deformity mechanisms, strength imbalance and impaired growth, on shoulder biomechanics. The model, implemented for dynamic musculoskeletal simulation in the OpenSim 3.0 software platform (Stanford University, Stanford, CA),<sup>21</sup> has been widely used to evaluate both healthy and pathological upper limb function.<sup>17,18,22-26</sup> Experimentally determined muscle properties were incorporated in the model to characterize the biomechanical action and forcegenerating behavior of 18 muscles and muscle compartments crossing the shoulder: anterior deltoid, middle deltoid, posterior deltoid, teres major, teres minor, supraspinatus, infraspinatus, subscapularis, pectoralis major (clavicular head), pectoralis major (sternocostal head, 2 compartments), biceps (long head), biceps (short head), triceps (long head), latissimus dorsi (3 compartments), and coracobrachialis. Origin-to-insertion muscle paths determined the direction in which muscles produced glenohumeral joint forces. The paths of muscles crossing the shoulder in our computational model match experimentally determined moment arms from numerous cadaveric studies.<sup>27,28</sup> In addition, muscle optimal length, pennation, physiological cross-sectional area, and tendon length were derived from cadaveric experiments<sup>29</sup> and magnetic resonance imaging,<sup>30</sup> whereas overall joint moment profiles throughout the ROM were derived from isometric joint moment measurements obtained using a dynamometer.<sup>31–33</sup> The glenohumeral joint allowed rotation only. Translation of the humeral head relative to the glenoid was not permitted. Joint moment, the mathematical product of muscle force and moment arm, indicated a muscle's tendency to augment or limit joint ROM along a given direction of movement. The total force produced by each muscle was the sum of both active and passive muscle forces; active force depends on the level of neural stimulation, muscle length, and muscle size, whereas passive force is generated by stretched muscles independent of neural stimulation.<sup>34</sup> Representations of ligaments and other passive structures at the shoulder were included in the model to limit movement at the extremes of unimpaired shoulder ROM.<sup>22</sup>

#### Sensitivity analysis

In a sensitivity analysis, we iteratively altered the properties of individual muscles crossing the shoulder in the musculoskeletal model to simulate muscle changes associated with either the strength imbalance or the impaired growth deformity mechanism. To simulate strength imbalance, each muscle crossing the shoulder, 1 muscle at a time, was iteratively activated to produce a submaximal force equal to 30% of its maximum active force; all other muscles were inactive and could generate passive forces only. This model of strength imbalance was based on observed decreases in muscle size<sup>7</sup> and increases in muscle activity<sup>35</sup> in the affected limbs of children with BPBP. We simulated impaired longitudinal muscle growth by reducing the functional length of each muscle crossing the shoulder, 1 muscle at a time, by 30%, based on changes in muscle length in a murine model of BPBP.<sup>13</sup> All muscles in the model were inactive and could generate passive forces only, and all other properties in the model were unchanged.

We computed the effect of each applied muscle change on shoulder forces and identified muscles that were mechanically capable of contributing to shoulder deformity. We first identified muscles that, through either simulated deformity mechanism, increased the posteriorly directed, compressive glenohumeral joint force. The glenohumeral joint force was calculated as the sum of gravitational and muscle forces transmitted between the glenoid and the humeral head. During the force calculations, the torso assumed an upright posture, and the arm was in neutral abduction and internal/external shoulder rotation posture. We evaluated whether muscles increased glenohumeral joint force in the axial plane, because such forces are expected to contribute to posterior humeral head subluxation and glenoid retroversion

osseous deformities that occur in the axial plane. Changes in glenohumeral joint force produced by muscles were normalized to the largest individual force change to highlight relative differences among muscles. For muscles that produced a posteriorly directed glenohumeral joint force relative to the glenoid centerline, the joint force was resolved into posterior and compressive force components; compressive force components were along the glenoid centerline and toward the glenoid, and posterior force components were perpendicular to the glenoid centerline in the posterior direction. Posterior forces would tend to shift the humeral head posteriorly, whereas compressive forces would tend to compress the humeral head and glenoid together, increasing the mechanical loading within these structures.

Second, we identified individual muscles crossing the shoulder that could contribute to postural deformity by reducing the external shoulder rotation or abduction ROM through either simulated deformity mechanism. We simulated 2 clinical examinations that are commonly performed to determine if a BPBP patient has limited shoulder ROM (Fig. 1).<sup>6</sup> During the first simulated examination, the shoulder was externally rotated with the shoulder adducted and the elbow flexed to 90°. In the second examination, the shoulder was abducted in the coronal plane with the elbow extended. For each deformity mechanism, we calculated how much each individual muscle crossing the shoulder reduced ROM during the simulated examinations. We first computed the net shoulder rotation joint moments generated by muscles and ligaments crossing the shoulder as the shoulder was moved in the directions indicated in Figure 1. The end point, or limit, of ROM was defined as the joint angle at which the muscles and ligaments crossing the shoulder could prevent further shoulder rotation during a clinical examination.<sup>36</sup> We computed the amount that each muscle reduced shoulder ROM as the difference between the ROM limits with and without the deformity mechanisms applied to each muscle.

#### RESULTS

# Potential contributors to osseous deformity: strength imbalance mechanism

Through the simulated strength imbalance mechanism, several muscles increased the glenohumeral joint force in the axial plane and were therefore mechanically capable of contributing to osseous deformity (Fig. 2). Muscles that increased the posteriorly directed, compressive glenohumeral joint



**FIGURE 1:** The postures in which external shoulder rotation was evaluated, which were analogous to those used during clinical examinations.<sup>6</sup> The black arrows indicate the direction of movement.



FIGURE 2: A Glenohumeral joint forces produced by muscles in the axial plane through the strength imbalance deformity mechanism, superimposed on a diagram of the glenohumeral joint. Forces were normalized to that of the infraspinatus, which was most sensitive to the strength imbalance mechanism. Muscles that increased the posteriorly directed, compressive glenohumeral joint force relative to the glenoid centerline are shown as black arrows on the radial plot. B Components of the glenohumeral joint force; compressive force components were along the glenoid centerline and toward the glenoid, and posterior force components were perpendicular to the glenoid centerline in the posterior direction.

force included the infraspinatus, subscapularis, long head of biceps, latissimus dorsi, teres major, teres minor, and posterior deltoid. The infraspinatus produced the highest glenohumeral joint force through the strength imbalance mechanism, primarily along the glenoid centerline. Most of the increase in the posteriorly directed, compressive glenohumeral joint force was attributed to the subscapularis, latissimus dorsi, and infraspinatus. Of muscles that increased the posteriorly directed glenohumeral joint force, the infraspinatus and subscapularis increased the compressive force the most. Muscles that increased force



**FIGURE 3:** A Glenohumeral joint forces produced by muscles in the axial plane through the impaired longitudinal muscle growth deformity mechanism are superimposed on a diagram of the glenohumeral joint. Forces were normalized to that of the anterior deltoid compartment, which was most sensitive to the impaired growth mechanism. Muscles that increased the posteriorly directed, compressive glenohumeral joint force are shown as black arrows on the radial plot. **B** Components of the glenohumeral joint force; compressive force components were along the glenoid centerline and toward the glenoid, and posterior force components were perpendicular to the glenoid centerline in the posterior direction.

in the anterior direction relative to the glenoid centerline, including the anterior deltoid and pectoralis major, could counter muscles that increased force in the posterior direction.

# Potential contributors to osseous deformity: impaired growth mechanism

Muscles that increased the posteriorly directed, compressive glenohumeral joint force with simulated impaired growth included the infraspinatus, subscapularis, long head of biceps, and long head of triceps (Fig. 3); therefore, these muscles were mechanically capable of contributing to osseous deformity in children with BPBP. Nearly equal posteriorly directed glenohumeral joint forces were produced by the infraspinatus, subscapularis, and long head of biceps. The infraspinatus increased the com-pressive force component more than any other muscle through the impaired growth mechanism. The anterior deltoid and long head of triceps increased force in the anterior direction relative to the glenoid centerline and thus could counteract muscles that increased force in the posterior direction.

# Potential contributors to postural deformity: strength imbalance mechanism

Through the strength imbalance deformity mechanism, external shoulder rotation ROM was reduced by the subscapularis, anterior deltoid, and the combined heads of pectoralis major by  $28^{\circ}$ ,  $17^{\circ}$ , and  $10^{\circ}$ , respectively; therefore, these muscles were mechanically capable of contributing to BPBP-associated postural deformity (Fig. 4). The latissimus dorsi, teres major, and long head of triceps each reduced external rotation ROM by less than  $3^{\circ}$ . No muscle individually reduced abduction ROM through the strength imbalance deformity mechanism by more than  $2^{\circ}$ .

# Potential contributors to postural deformity: impaired growth mechanism

External shoulder rotation ROM was reduced by  $52^{\circ}$  and  $40^{\circ}$  by impaired growth of the anterior deltoid and subscapularis muscles, respectively (Fig. 5). In addition, the long head of triceps reduced abduction by  $56^{\circ}$ . Consequently, these muscles that reduced shoulder ROM were mechanically capable of contributing to postural deformity. Shoulder ROM was not reduced by more than  $2^{\circ}$  by either the pectoralis major or the latissimus dorsi.

#### DISCUSSION

Muscles that can alter shoulder forces may provide a mechanical stimulus for the development of shoulder deformity. In our computational simulations, the posteriorly directed, compressive glenohumeral joint force was increased the most by the infraspinatus, subscapularis, latissimus dorsi, and long head of biceps, which are therefore mechanically capable of



FIGURE 4: Individual muscles that reduced the external shoulder rotation and abduction through the strength imbalance deformity mechanism.



**FIGURE 5:** Individual muscles that reduced the external shoulder rotation and abduction through the impaired longitudinal muscle growth deformity mechanism.

contributing to osseous deformity. Likewise, because they reduced shoulder ROM the most, the subscapularis, anterior deltoid, pectoralis major, and long head of triceps are capable of contributing to postural deformity. The subscapularis was the only muscle capable of contributing to both osseous and postural deformity through either deformity mechanism.

Clinical and experimental evidence corroborate our findings, which indicate that the subscapularis is likely a key contributor to BPBP-associated shoulder deformity. The subscapularis receives innervation from the C5, C6, and C7 nerve roots.<sup>37</sup> The C5 and C6 roots are injured in up to 96% of children with BPBP,<sup>1,8</sup> and the subscapularis is frequently atrophic as a result.<sup>3,7</sup> Einarsson and colleagues<sup>38</sup> observed an increase in subscapularis muscle fiber stiffness, whereas in a mouse model of BPBP, the subscapularis was functionally shorter in the affected limbs than in the unimpaired contralateral limbs.<sup>13</sup> Surgical release of the subscapularis muscle is commonly performed to effectively relieve shoulder contractures in children with BPBP.<sup>39,40</sup>

Other muscles we identified as mechanically capable of contributing to shoulder deformity, including the deltoid, supraspinatus, infraspinatus, and pectoralis major, can be weakened or paralyzed by injury involving the C5 and C6 nerve roots. Atrophy and fatty degeneration has been observed clinically in the deltoid, infraspinatus, and supraspinatus,<sup>3,7</sup> whereas functional shortening of the biceps muscle was observed in a murine model of BPBP.<sup>14</sup> In cases of severe internal rotation contracture, pectoralis major lengthening in conjunction with subscapularis release improved shoulder ROM but had little effect on osseous deformity.<sup>41</sup> The latissimus dorsi, commonly transferred to restore external shoulder rotation function,<sup>41,42</sup> retains innervation following BPBP and thus could contribute to osseous deformity through the strength imbalance mechanism.

Over the first decade of unimpaired development, endochondral ossification progresses from the center of the scapula toward the cartilaginous glenoid.<sup>43</sup> The humeral head and glenoid support compressive forces generated by muscles and soft tissues crossing the shoulder, which stabilize the glenohumeral joint at rest and during movement.<sup>44,45</sup> However, according to the Hueter-Volkmann law, abnormally high static, continuous compression of the epiphyseal plate reduces the rate of longitudinal bone growth.<sup>46</sup> Posterior subluxation and compression of the humeral head against the posterior glenoid margin could locally inhibit ossification and bone growth and produce osseous deformity. Biomechanical studies investigating the forces needed to produce osseous deformity would be useful for developing treatments to prevent or reverse deformity.

When multiple muscles are affected by BPBP simultaneously, shoulder ROM may be reduced by more than the sum of each muscle's individual effect on reduction of shoulder motion. To illustrate this effect, we applied the strength imbalance mechanism simultaneously to all muscles that internally rotate the shoulder in an adducted posture. When internal rotator muscles were affected simultaneously, external rotation was restricted by  $82^\circ$ , whereas the sum of each muscle's individual restriction from Figure 4 was only  $60^\circ$ . Therefore, it may be important to target multiple muscles to effectively treat postural deformity in children with extensive nerve injury.

There were several limitations to our study. First, we used an adult musculoskeletal model to evaluate the biomechanical consequences of BPBP in children. We normalized muscle forces and computed the reduction in shoulder ROM to account for differences in shoulder strength and ROM, respectively, between an adult and a child. In addition, we assumed that the geometric arrangement of muscles about the shoulder, which affects the direction of muscle forces acting on the glenohumeral joint, are similar between a human adult and an infant. Others have used adult musculoskeletal models to evaluate pediatric orthopedic conditions such as cerebral palsy.<sup>47</sup>

Second, the posterior and compressive glenohumeral joint forces we calculated were determined relative to the glenoid centerline, and we did not represent changes in glenoid version. In these simulations, the shoulder was in a fixed posture, and translation of the humeral head against the glenoid was not allowed. Therefore, glenoid retroversion angle would not affect our outcome measures as we evaluated them in the model. However, changes to glenoid version could change the effect of these directional forces on the bones. This warrants future study.

We simulated 2 possible deformity mechanisms, 1 mechanism at a time. However, BPBP-associated shoulder deformities are often complex and idiosyncratic among patients; and other mechanisms, including those not evaluated in this study, may concurrently contribute to shoulder deformity. Other proposed deformity mechanisms include cross-innervation of agonist-antagonist muscle pairs,<sup>48</sup> abnormal development of the glenohumeral joint capsule and ligaments,<sup>42,49</sup> muscle fibrosis,<sup>50</sup> and direct birth trauma to the shoulder. Further research using biological models, such as murine models of BPBP,<sup>13,14,50–53</sup> is needed to identify other mechanisms and determine their relative contributions to shoulder deformity.

We applied the same muscle changes to all muscles to identify the relative potential contributions among muscles crossing the shoulder to BPBP deformity. However, multiple muscles are frequently affected in the same patient. Furthermore, the extent that muscle forces are altered and the effect of those forces on shoulder deformity depend on several factors, including the extent and severity of nerve injury, musculoskeletal geometry, and patient anthropometry and stage of development. The results from this study provide a foundation for future biomechanical analyses that will extend the current work to explore in more detail the effects of combined patterns of muscle injury and concomitant clinical factors on shoulder deformity.

The muscles most capable of producing forces that may contribute to shoulder deformity following BPBP, the infraspinatus, subscapularis, latissimus dorsi, long head of biceps, anterior deltoid, pectoralis major, and long head of triceps, should be the focus of experimental studies investigating the musculoskeletal consequences of BPBP and are potentially critical targets for treating shoulder deformity.

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