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# Preganglionic and Postganglionic Brachial Plexus Birth Injury Effects on Shoulder Muscle Growth

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## Abstract

**Purpose—**Brachial plexus birth injury can differ in presentation, depending on whether the nerve ruptures distal to, or avulses proximal to, the dorsal root ganglion. More substantial contracture and bone deformity at the shoulder is typical in postganglionic injuries. However, changes to the underlying muscle structure that drive these differences in presentation are unclear.

**Methods—**Seventeen Sprague-Dawley rats received preganglionic or postganglionic neurectomy on a single limb on postnatal days 3 and 4. Muscles crossing the shoulder were retrieved once the rats were sacrificed at 8 weeks after birth. External rotation range of motion, muscle mass, muscle length, muscle sarcomere length, and calculated optimal muscle length were measured bilaterally.

**Results—**Average shoulder range of motion in the postganglionic group was 61.8% and 56.2% more restricted at 4 and 8 weeks, respectively, compared with that in the preganglionic group, but affected muscles after preganglionic injury were altered more severely (compared with the unaffected limb) than after postganglionic injury. Optimal muscle length in preganglionic injury was shorter in the affected limb (compared with the unaffected limb: −18.2% ± 9.2%) and to a greater extent than in postganglionic injury (−5.1% ± 6.2%). Muscle mass in preganglionic injury was lower in the affected limb (relative to the unaffected limb: −57.2% ± 24.1%) and to a greater extent than in postganglionic injury (−28.1% ± 17.7%).

**Conclusions—**The findings suggest that the presence of contracture does not derive from restricted longitudinal muscle growth alone, but also depends on the extent of muscle mass loss occurring simultaneously after the injury.

**Clinical relevance—**This study expands our understanding of differences in muscle architecture and the role of muscle structure in contracture formation for preganglionic and postganglionic brachial plexus birth injury.

### Keywords

Brachial plexus birth injury; contractures; muscle mass; range of motion; sarcomere length

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BRACHIAL PLEXUS BIRTH injury (BPBI) is the most common nerve injury among children,[1] most frequently affecting the C5-C6 nerve roots of the brachial plexus.[2] Although many children spontaneously recover nerve and muscle function, 20% to 30% of affected children do not have total neurological recovery.[3] Up to 33% of children affected by BPBI sustain permanent postural and osseous deformities,[4] which can have lifelong consequences for upper-limb function and quality of life.[5] Presentation in patients, however, can vary markedly according to nerve injury location relative to the dorsal root ganglion. Nerve root ruptures that occur distal to the ganglion (postganglionic) frequently result in paralysis with shoulder internal rotation (restricted external rotation) and elbow flexion (restricted extension) contractures.[6] Contractures in these patients correlate with osseous glenohumeral deformities, including retroversion, declination, and flattening of the glenoid fossa.[7–9] In contrast, avulsion injuries proximal to the ganglion (preganglionic) typically result in paralysis without shoulder or elbow contractures or substantial osseous deformity.[10]

The underlying mechanisms leading to these different presentations are unclear, but recent work in murine models of BPBI provide initial evidence that postganglionic contractures are associated with restricted growth of paralyzed muscles,[11] with 10% to 35% shorter optimal muscle lengths in all shoulder internal and external rotator muscles, biceps short head, and triceps long head in the affected limb compared with the unaffected limb. Optimal muscle length is the length at which underlying sarcomeres exhibit optimal actin-myosin overlap and produce peak force.[12] Postganglionic nerve injury is also associated with degenerative changes to muscle spindles and absence of important cell signals, such as ErbB, necessary for myogenesis and muscle regeneration.[13] In contrast, preganglionic injury retains ErbB signaling and spindle formation.[13] A prior study of biceps and brachialis muscle architecture in a preganglionic mouse model reported that reduced muscle cross-sectional area, reduced volume, and muscle fiber atrophy were similar in magnitude to those seen with postganglionic injury; the authors concluded that it is unlikely that differences in contracture resulted from a difference in motor denervation.[13] However, at the shoulder, changes to other muscles that may contribute to contracture formation are unknown. Prior simulation work suggested that subscapularis, infraspinatus, latissimus dorsi, long head of biceps, anterior deltoid, pectoralis major, and long head of triceps are critical contributors to shoulder external rotation range of motion (ROM).[14]

We conducted a study in a BPBI rat model to examine the effect of nerve injury location (preganglionic vs postganglionic) on passive external rotation ROM and shoulder muscle architecture, specifically optimal muscle length and muscle mass. We hypothesized that after postganglionic injury, animals would have more restricted ROM, and paralyzed muscles would exhibit restricted muscle longitudinal growth (ie, a shorter optimal muscle length) and lower muscle mass compared with preganglionic injuries.

## MATERIALS AND METHODS

All procedures were approved by the institutional animal care and use committee. In this study, 23 neonatal Sprague-Dawley rats were divided into preganglionic and postganglionic injury groups. Preganglionic neurectomy was performed on the left forelimb (Fig. 1) in 13 rats on postnatal day 3–4, wherein the C5 and C6 nerve roots were excised proximal to the

dorsal root ganglion via supraclavicular incision.[13] Because of the complex nature of the preganglionic surgeries, in which visual access to the nerve is limited, success of the procedure was defined as observation of the typical phenotype of an internally rotated and adducted limb during the 24 hours after surgery. The study relied on prior demonstration of the preganglionic injury model and further confirmed appropriate neurological deficit in the injured rats based on the injury phenotype.[13] On that basis, 7 of the 13 rats were analyzed in the study. Postganglionic neurectomy was performed on the right forelimb of 10 rat pups at postnatal day 4, in which C5 and C6 nerve roots were excised distal to the dorsal root ganglion via transverse infraclavicular incision and splitting of the pectoralis major muscle[15]; the nerve roots can be clearly visualized in this procedure. The surgeries were performed on pups under isoflurane anesthesia. After the procedures, incisions were closed with tissue adhesive and rat pups were given one prophylactic dose each of buprenorphine and carprofen. The rats were closely monitored for pain or distress, and no additional analgesics were needed. All pups were kept with their respective dams for the first 3 weeks and then weaned on postnatal day 21. Thereafter, they were housed 1–3 rats/cage with sex-matched littermates. The animals were kept at room temperature (21°C) and were provided standard rat chow (Purina, Woodstock, Ontario, Canada) and water ad libitum. Body mass was measured every other day for 2 weeks after neurectomy and biweekly thereafter.

To evaluate internal rotation contracture, maximum passive external shoulder rotation was measured bilaterally at 4 and 8 weeks of age using a previously developed custom device[15] with the animals under isoflurane anesthesia (0% to 5% inhalation). These time points approximately correspond to age 2 to 7 years of postnatal human musculoskeletal development,[16] by which time the shoulder deformity is established clinically.[16] The forelimb was placed at 90° shoulder abduction and elbow flexion, with the limb oriented ventrally at a neutral shoulder rotation posture.[17] The forearm was fixed against the rotating lever on the device. A 10-g weight was hung on the wrist of the rat to apply a constant external rotational force at the shoulder. The external rotation was measured to the nearest 5° using a protractor built onto the custom device, and the average of 3 measurements was used for analysis.[15]

At age 8 weeks, animals were sacrificed via $CO_2$ asphyxiation followed by bilateral thoracotomy. The intact upper torso between the neck and the diaphragm was extracted using a guillotine, degloved, fixed in 10% neutral-buffered formalin for 48 hours at 21°C, and then transferred to 70% ethanol at 4°C, where it was positioned in a consistent limb posture (neutral adducted shoulder and elbow flexed to 90°) using a custom fixture.[15] Muscles crossing the glenohumeral joint (ie, biceps short head, biceps long head, anterior deltoid, spinal deltoid, pectoralis major, supraspinatus, infraspinatus, teres major, and subscapularis) were carefully dissected[15] and stored in 70% ethanol at 4°C. For each muscle, muscle belly length was measured using a digital Vernier caliper with a 0.01-mm resolution. Muscle mass was measured using a digital scale with 0.01-g resolution (Sartorious AG, Göttingen, Germany). Sarcomere length was measured using a standard laser diffraction technique[18] with a 5.0-mW, 633-nm HeNe laser (Thorlabs, Newton, NJ). Optimal muscle length ($L_o$) was computed (Equation 1), in which muscle length ($L_m$) is scaled by the ratio of optimal sarcomere length for rat skeletal muscle, 2.4μm,[19] to the measured sarcomere length ($L_s$):

$$L_o = L_m\left(\frac{2.4\mu m}{L_s}\right) \quad (1)$$

We evaluated the normality of the data using Shapiro-Wilk test. To evaluate whether ROM, muscle mass, optimal muscle length, and sarcomere length for each muscle differed between the affected and unaffected limb, paired *t* tests were performed for normally distributed data and Wilcoxon signed rank tests for nonnormal data. To evaluate whether ROM, muscle mass, and optimal muscle length for each muscle differed between the preganglionic and postganglionic injury groups, unpaired *t* tests were performed, using the difference between affected and unaffected values expressed as a percentage of the unaffected value. For nonnormal data, Mann-Whitney nonparametric tests were performed to evaluate whether muscle mass differed between preganglionic and postganglionic injury groups. Significance was assessed at $\alpha = .05$.

## RESULTS

Passive external shoulder rotation ROM was significantly lower (Fig. 2) in the affected limb than in the unaffected limb for the postganglionic group at both 4 weeks (−40.7% ± 21.8%; $P < .05$) and 8 weeks (−29.6% ± 49.1%; $P < .05$). For the preganglionic group, external rotation ROM in the affected limb was significantly greater than in the unaffected limb at 4 weeks (21.1% ± 41.0%; $P < .05$), indicating less restricted movement in the affected forelimb. This comparison was not significant at 8 weeks (26.6% ± 45.8%; $P = .28$). Compared with the postganglionic group, the preganglionic group had less restricted passive external rotation at 4 weeks ($P < .05$) and 8 weeks ($P < .05$).

Both postganglionic and preganglionic injury groups exhibited shorter optimal muscle lengths in the affected compared with the unaffected limb for several muscles, suggesting restricted muscle growth in both cases (Fig. 3). Compared with the unaffected limb, optimal muscle length was an average of 5.1% shorter in the affected limb for the postganglionic group and 18.1% shorter for the preganglionic group. For the postganglionic group, the optimal muscle lengths were significantly shorter in the affected limb than in the unaffected limb for pectoralis major (−11.1% ± 11.7%; $P<.05$), spinal deltoid (−14.6%±17.9%;$P<.05$), and subscapularis (−9.0% ± 10.3%; $P < .05$). The biceps long head was significantly longer in the affected limb of the postganglionic group (5.7% ± 6.2%; $P < .05$). For the preganglionic group, optimal muscle lengths were significantly shorter in the affected limb compared with the unaffected limb for spinal deltoid (−22.4%±19.9%; $P<.05$), biceps long head (−23.0%±13.9%; $P<.05$), biceps short head (−19.8% ± 16.2%; $P < .05$), supraspinatus (−32.4% ± 9.8%; $P < .05$), and teres major (−15.0% ± 18.6%; $P < .05$). Compared with the postganglionic group, the preganglionic group had significantly shorter optimal muscle lengths for biceps long head (−28.7% vs postganglionic; $P < .05$), biceps short head (−15.9% vs postganglionic; $P < .05$), and supraspinatus (−29.0% vs postganglionic; $P < .05$). All muscles with significantly shorter optimal muscle lengths on the affected side in both postganglionic and preganglionic injury groups exhibited the same or longer mean sarcomere lengths on the affected side (Table 1). Sarcomere lengths in the affected muscles were significantly longer in teres major ($P < .05$) in the postganglionic group and in

pectoralis major ($P < .05$), biceps long ($P < .05$), biceps short ($P < .05$), and teres major ($P < .05$) in the preganglionic group.

Compared with the unaffected limb, muscle mass was an average of 28.0% lower in the affected limb for the postganglionic group and 57.2% lower for the preganglionic group (Fig. 4). In the postganglionic group, muscle mass was significantly lower in the affected limb than the unaffected limb for pectoralis major (−25.4% ± 12.0%; $P < .05$), anterior deltoid (−38.0% ± 36.6%; $P < .05$), spinal deltoid (−41.5% ± 36.7%; $P < .05$), biceps long head (−54.39% ± 39.1%; $P < .05$), subscapularis (−24.0% ± 24.4%; $P < .05$), teres major (−36.9% ± 39.9%; $P < .05$), and triceps (−42.1% ± 39.0%; $P < .05$). In the preganglionic group, muscle mass was significantly lower in the affected limb compared with the unaffected limb for anterior deltoid (−70.6% ± 12.8%; $P < .05$), spinal deltoid (−64.7% ± 37.2%; $P < .05$), biceps long head (−75.6% ± 8.0%; $P < .05$), subscapularis (−69.9% ± 20.1%; $P < .05$), supraspinatus (−83.6% ± 9.8%; $P < .05$), infraspinatus (−78.8% ± 9.1%; $P < .05$), teres major (−54.5% ± 25.9%; $P < .05$), and triceps (−23.5% ± 15.5%; $P < .05$). Compared with the postganglionic group, the preganglionic group exhibited significantly lower muscle mass for anterior deltoid (−32.6% vs postganglionic; $P < .05$), subscapularis (−45.9% vs postganglionic; $P < .05$), supraspinatus (−72.2% vs postganglionic; $P < .05$), and infraspinatus (−68.5% vs postganglionic; $P < .05$).

## DISCUSSION

This work demonstrates that both postganglionic and preganglionic BPBI induce considerable reductions in muscle mass and optimal muscle length. Affected limb changes in muscle structure were more severe for preganglionic injury in both the magnitude and number of muscles affected, but shoulder external rotation ROM was more restricted after postganglionic injury, consistent with clinical presentation.[10] A previous study[15] using the same postganglionic rat model reported similar affected side reductions in muscle mass for pectoralis major, spinal deltoid, and subscapularis, as well as increases in optimal muscle length for biceps long head. Our study reveals marked loss of muscle mass and reduced optimal muscle lengths in many shoulder muscles after preganglionic injury. Another study in mice that looked only at volumetric changes for biceps and brachialis reported marked biceps atrophy with both preganglionic and postganglionic injury, but there was no significant difference between groups.[13] Although many muscles in the current study were more severely affected for preganglionic injury, biceps long head exhibited muscle atrophy in both groups similarly, consistent with the prior report. Our initial hypothesis that postganglionic injury would result in shorter optimal muscle length and lower muscle mass than preganglionic injury was derived from earlier work that reported greater contracture formation in postganglionic than preganglionic injury,[13] combined with observations of significant muscle mass loss and shorter optimal length in a postganglionic injury group,[15] but typical length biceps in a mouse model of preganglionic injury.[13] Our current work also observed a typical length biceps, consistent with the earlier report. However, unlike the earlier work, which considered only biceps, brachialis, and elbow contracture, our current work examined a broader group of muscles and shoulder contracture and found that many shoulder muscles were affected in a way contrary to the biceps after preganglionic injury. This suggests that the extent of growth disruption may differ among affected muscles. This

phenomenon is seen in other musculoskeletal disorders such as cerebral palsy and Duchenne muscular dystrophy, in which certain muscles may be more affected than others; this could be the result of factors such as the degree of nerve involvement, afferent signaling, or differences in mechanical loading.[20] Marked variability in biceps muscle mass was observed across animals in all studies, in part because the mass of the affected biceps short head is extremely small (sometimes at the resolution of the scale), and small changes result in a large relative percent change.

Previous postganglionic studies on biceps and subscapularis muscles in mice reported longer sarcomeres in the impaired limb compared with control, suggesting that restricted longitudinal growth could be responsible for more severe elbow contractures after postganglionic injury.[11,13] In our study, longitudinal growth (ie, shorter optimal muscle length) was also severely restricted in preganglionic injury and to a greater extent overall than in postganglionic injury. This suggests that shorter optimal muscle length cannot be solely responsible for contracture severity. Force developed by a muscle-tendon unit depends on both optimal muscle length, which is related to muscle excursion potential, and cross-sectional area, which is directly proportional to peak force production.[12] A muscle with short optimal length produces active force over a limited length range and high passive forces as it becomes quickly overstretched. However, the remarkably lower muscle mass in the preganglionic group would likely limit passive force production. To illustrate this interaction, we calculated the force produced by a representative muscle with shortened optimal muscle length and reduced muscle mass (Fig. 5) as it is stretched, using values consistent with the range of values reported here. This illustration considers only the effects of structural changes to a single representative muscle in isolation, but overall joint behavior that leads to contracture or joint morphological change will be governed by the combined effects of multiple muscles. The role of each muscle with regard to contracture development is further determined by its line of action relative to the joint, so structural changes to some muscles may have a larger influence on joint function than others, as illustrated in prior computational work by our group, which identified the muscles most likely to influence shoulder contracture and joint reaction force.[14,21] In those simulations, we illustrated that the combined effects of muscle paralysis or weakness and short muscles could lead mechanically to contracture and altered joint loading. More recent work by our group further illustrates that these changes can directly influence bone growth in such a way that glenohumeral dysplasia is predicted.[22,23] However, this simple calculation demonstrates that shorter optimal muscle length encourages passive forces that are higher than normal, whereas reduced muscle mass limits force production, and different combinations of muscle changes determine the overall change in passive force. Substantially greater muscle atrophy with preganglionic injury thus can explain the lack of contracture and greater passive external rotation ROM compared with the unaffected arm despite the shorter optimal muscle length. The balance between these 2 factors is essential for defining the mechanical underpinnings of contracture development with muscle architectural changes.

Clinically, contracture severity is correlated with glenoid deformity.[10] Possible explanations for this relationship include impaired shoulder rotation with a deformed joint or similar mechanical underpinning driving the formation of both bony and postural deformities. Our previous computational modeling studies suggest that restricted muscle growth can lead to

posteriorly directed glenoid forces consistent with the development of glenoid retroversion as well as restricted external rotation ROM.[15,21,22] However, concurrent effects of atrophy were not considered in these prior simulations. Other groups have linked mechanical loading to morphogenesis of other joints, such as the hip.[24,25] Our study describes muscle architecture that can inform future mechanical analyses of shoulder loading and underlying bone growth after BPBI.

Although mechanisms driving the muscle changes after BPBI remain unclear and are outside the scope of this study, some considerations deserve future attention. Surgical approaches differ for the 2 neurectomies: postganglionic neurectomy requires an infraclavicular approach and division of the pectoralis major to expose the brachial plexus, and the preganglionic procedure requires a supraclavicular approach. We previously demonstrated that division of pectoralis major disrupts its growth compared with the unaffected limb.[15] This may explain differences in this muscle's growth and the passive external rotation between the 2 groups in the current study. The preganglionic procedure, which requires incision of the spinal cord dura and makes visibility of the nerve root more challenging, could affect more of the surrounding nerve supply in some cases, which may explain the larger number of muscles exhibiting structural changes. Nevertheless, reduced contracture observed in the preganglionic case is consistent with clinical presentation.[10] Although the neurectomy procedure is intended to create isolated C5-C6 disruption via transection, preganglionic BPBI is often an avulsion injury that may also disrupt other structures.[13] Prior work in a mouse model reported that postganglionic injury was associated with degenerative changes to muscle spindles and absence of important cell signals (eg, ErbB) necessary for myogenesis and muscle regeneration, while preganglionic injury retained ErbB signaling and spindle formation.11,13 While we did not observe normal longitudinal growth of muscle fibers with preganglionic injury, these differences may play an important role in other aspects of muscle growth.

The current study had several limitations. Although the neuromuscular anatomy of rats and humans is similar,[26] rats have a spinal deltoid muscle not present in humans, and glenohumeral loading may differ, because rats are lifelong quadrupeds and not transient quadrupeds like humans. Muscle architecture was investigated at only one time point in both injury groups (8 weeks), but deformity is known to be well-established by this time in this model.[15,17,27] Assessing injury progression over time will be critical in future work to inform timing of both deformity initiation and optimal treatment. The current study emphasizes the mechanical role of macrostructural changes to the architecture of muscle on shoulder range of motion. The underlying mechanisms driving these changes and the histological changes to muscle are a critical next step to investigate. Additional measures of bone morphology and histology and immunohistochemistry are ongoing in the current study. Prior work by others in models of mice with regard to histology and presence of satellite cells[11] report that there may be fibrotic changes and active satellite cells, suggesting more attention is needed in this area.

We also acknowledge that we relied on prior demonstration of the preganglionic injury model with histological confirmation of preganglionic injury, and confirmation of appropriate neurological deficit in the injured rats, rather than histological confirmation of a

preganglionic injury in these rats. We observed marked differences in passive external shoulder rotation ROM consistent with clinical differences between preganglionic and postganglionic injuries, which support the likelihood that a preganglionic injury was created as intended. In addition, although we found that many muscles were shorter in the preganglionic group, in contrast to our initial expectations, our current work observed a typical-length biceps, consistent with the only other prior report of preganglionic muscle change.[13] Moreover, both the preganglionic and postganglionic procedures have been previously presented alongside sham control surgeries in which the approach is performed without neurectomy.[11,13,15] Limited changes in muscle outcomes were observed owing to the approach without neurectomy. For example, after the postganglionic sham procedure, only pectoralis major (which is implicated in the surgical approach) was affected. This supports that observed changes are associated with successful neurectomy. This also supports our use of the unaffected limb as a control and basing all assessments on changes in the affected limb relative to the unaffected limb.

This study reveals muscle architectural changes after preganglionic and postganglionic BPBI. Although muscle mass loss and reduced optimal muscle length are present for both injuries, the effects are significantly more severe in preganglionic BPBI. However, contractures at the shoulder are more severe in postganglionic BPBI, which is consistent with previous clinical studies in humans. Our findings suggest that after injury, the extent of contracture depends inversely on the extent of muscle mass loss and directly on the amount restricted muscle growth.

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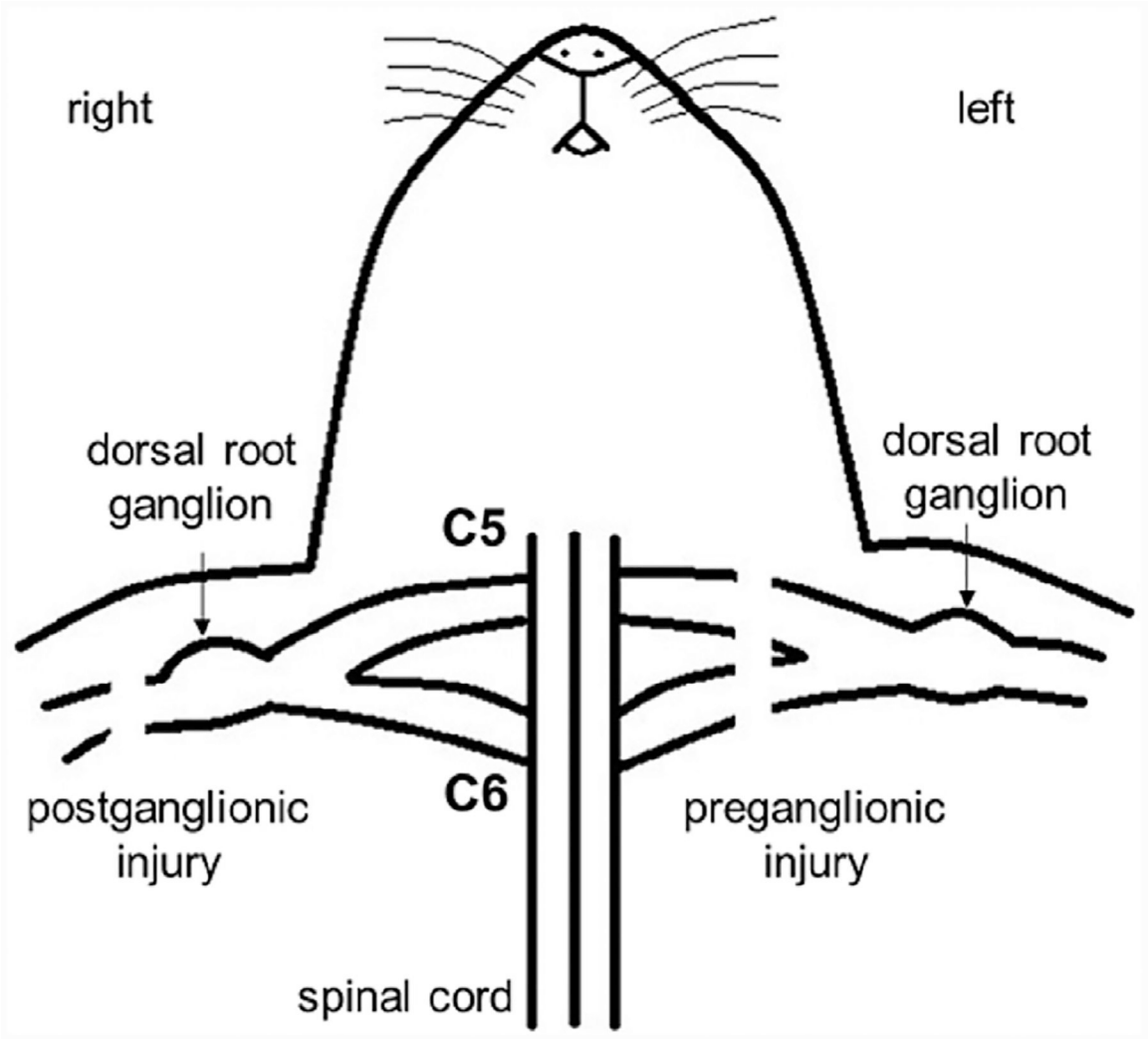


**FIGURE 1:**
Schematic figure of preganglionic and postganglionic injury models.

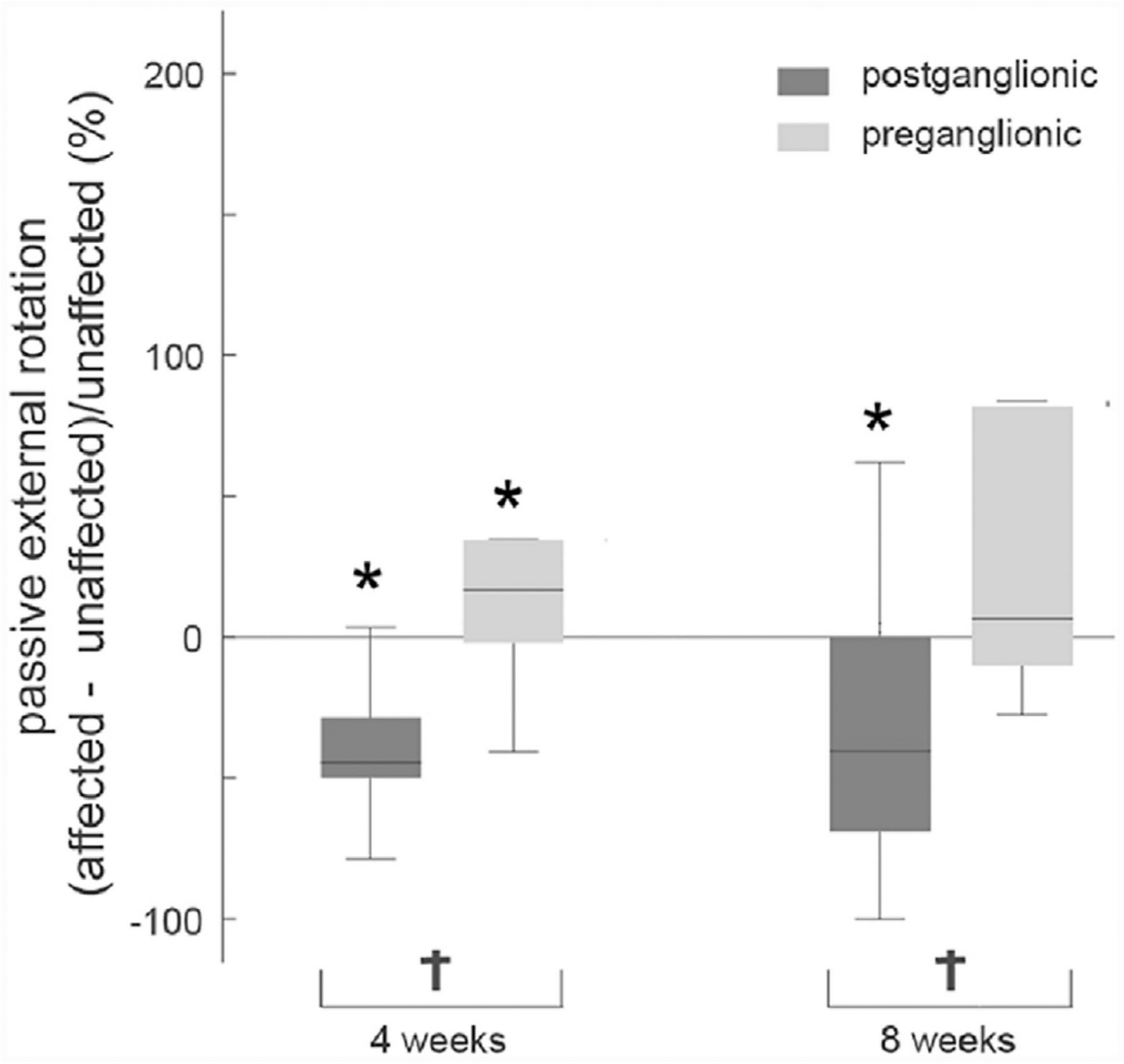


**FIGURE 2:**

Passive external rotation of the affected limb relative to the unaffected limb at 4- and 8-week points. Postganglionic injury resulted in significant restriction of passive external rotation in the affected limb (relative to unaffected), and the restriction was significantly more pronounced (more negative) than after preganglionic injury. *$P < .05$ for affected versus unaffected limb. †$P < .05$ for postganglionic versus preganglionic group.

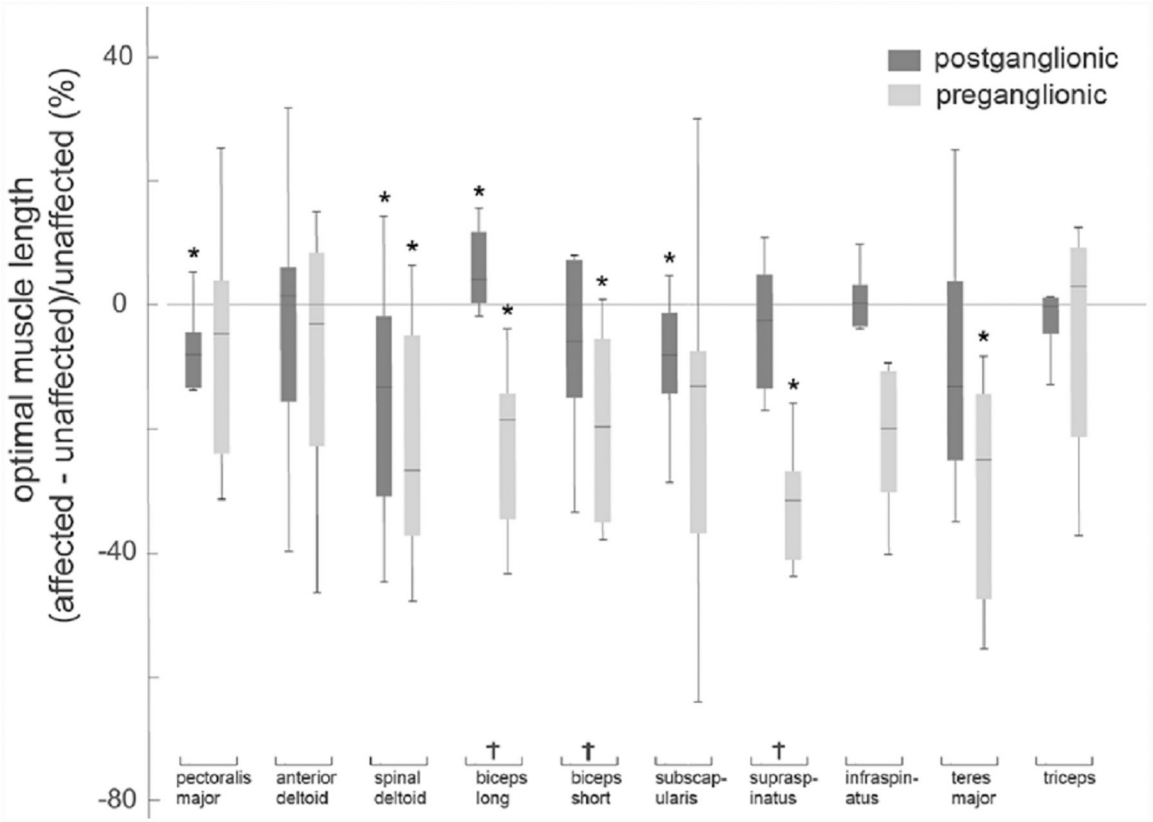


**FIGURE 3:**

Optimal muscle length for muscles in the affected limb relative to the unaffected limb. Postganglionic injury resulted in significant restriction of muscle length in the affected limb (relative to unaffected), and the restriction was significantly more pronounced (more negative) in more muscles after preganglionic injury. *$P < .05$ for affected versus unaffected limb. †$P < .05$ for postganglionic versus preganglionic group.

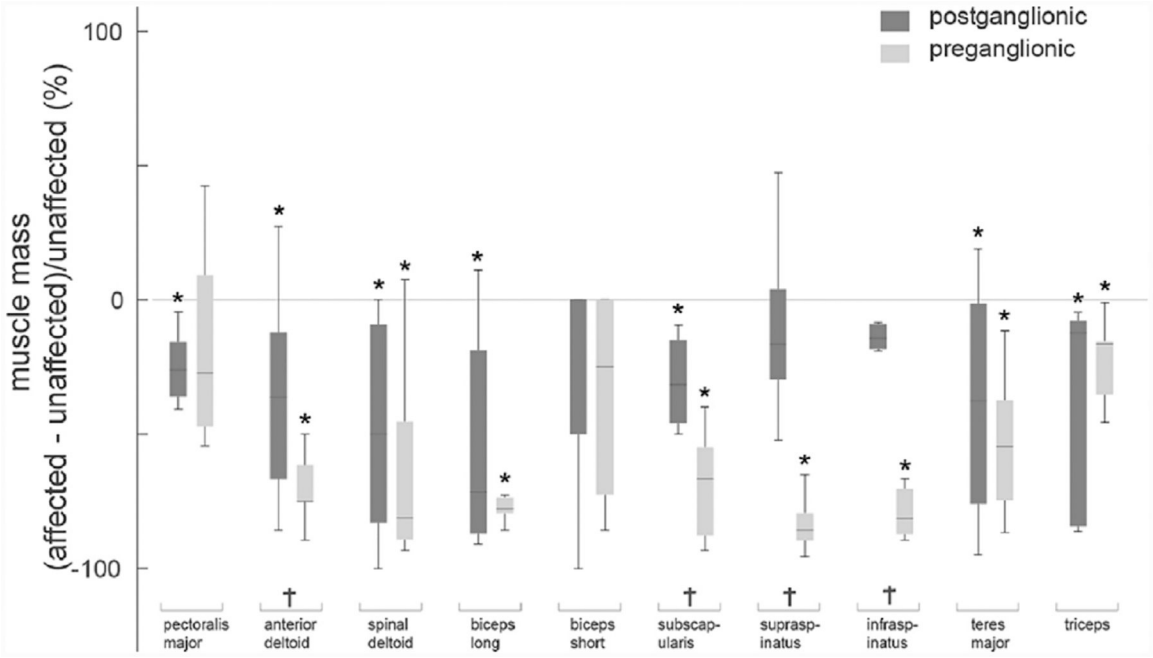


**FIGURE 4:**

Muscle mass for muscles in the affected limb relative to the unaffected limb. Postganglionic injury resulted in significantly lower muscle mass in the affected limb (relative to the unaffected limb), and lower mass was significantly more pronounced (more negative) in more muscles after preganglionic injury. $^{*}P < .05$ for affected versus unaffected limb. $^{\dagger}P < .05$ for postganglionic versus preganglionic group.

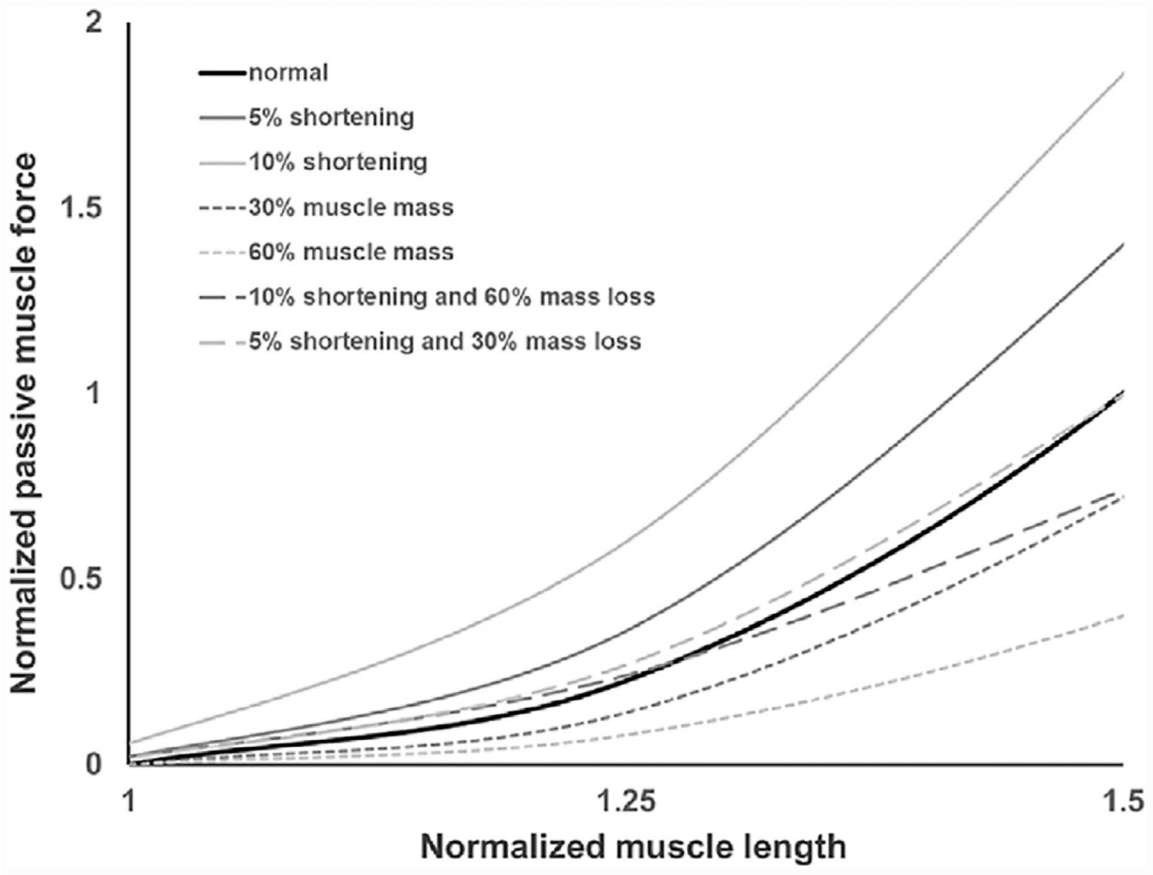


**FIGURE 5:**

Passive muscle force capacity of a representative muscle with altered architecture. Isolated shortening cases (dark and light gray solid lines), with optimal muscle length reduced by 5% and 10% relative to the nominal muscle (black solid line), increase passive force. Isolated mass loss (dark and light gray short dashed lines), with maximum isometric force reduced by 30% and 60% relative to nominal, reduce passive force. Combined changes to length and mass (dark and light gray long dashed lines) result in variable passive force magnitude and slope.

**TABLE 1.**

Muscle Sarcomere Length, Optimal Muscle Length, and Muscle Mass Data for Affected and Unaffected Limbs in Postganglionic and Preganglionic Injury Groups

| | Affected | | | | Unaffected | | | |
|---|---|---|---|---|---|---|---|---|
| **Muscle** | **Sarcomere Length, μm** | **Optimal Length, mm** | **Measured Muscle Length, mm** | **Mass, g** | **Sarcomere Length, μm** | **Optimal Length, mm** | **Measured Muscle Length, mm** | **Mass, g** |
| Postganglionic | | | | | | | | |
| Pectoralis major | 2.4 ± 0.2 | 18.0 ± 2.2 | 17.7 ± 2.0 | 0.22 ± 0.09 | 2.3 ± 0.2 | 20.4 ± 2.0 | 19.6 ± 2.7 | 0.30 ± 0.10 |
| Anterior deltoid | 2.4 ± 0.2 | 10.7 ± 1.8 | 10.5 ± 1.5 | 0.08 ± 0.05 | 2.5 ± 0.2 | 11.0 ± 1.4 | 11.6 ± 1.2 | 0.13 ± 0.04 |
| Spinal deltoid | 2.0 ± 0.1 | 16.5 ± 3.4 | 13.5 ± 2.6 | 0.07 ± 0.05 | 2.0 ± 0.1 | 19.2 ± 1.3 | 15.8 ± 1.1 | 0.12 ± 0.04 |
| Biceps long | 2.2 ±0.2 | 16.6 ± 2.3 | 15.8 ± 2.6 | 0.04 ± 0.04 | 2.3 ± 0.2 | 15.7 ± 1.6 | 15.0 ± 2.1 | 0.09 ± 0.02 |
| Biceps short | 2.2 ± 0.1 | 17.5 ± 2.8 | 15.6 ± 2.1 | 0.02 ± 0.01 | 2.1 ± 0.2 | 18.1 ± 1.6 | 15.6 ± 1.6 | 0.02 ± 0.01 |
| Subscapularis | 2.2 ± 0.2 | 17.8 ± 1.9 | 16.6 ± 2.0 | 0.19 ± 0.07 | 2.1 ± 0.1 | 19.3 ± 1.3 | 17.3 ± 1.2 | 0.25 ± 0.05 |
| Supraspinatus | 2.4 ± 0.2 | 20.6 ± 2.1 | 20.5 ± 2.5 | 0.18 ± 0.06 | 2.4 ± 0.1 | 21.4 ± 2.2 | 21.8 ± 2.1 | 0.20 ± 0.05 |
| Infraspinatus | 2.3 ± 0.2 | 21.4 ± 1.7 | 20.5 ± 1.9 | 0.19 ± 0.07 | 2.3 ± 0.1 | 21.7 ± 1.6 | 21.3 ± 1.5 | 0.22 ± 0.05 |
| Teres major * | 2.0 ± 0.1 | 21.0 ± 0.1 | 17.1 ± 2.5 | 0.13 ± 0.09 | 1.8 ± 0.1 | 23.9 ± 2.5 | 18.0 ± 1.4 | 0.21 ± 0.07 |
| Triceps long | 2.0 ± 0.2 | 23.5 ± 0.7 | 20.2 ± 2.4 | 0.40 ± 0.30 | 2.0 ± 0.2 | 23.9 ± 2.4 | 20.2 ± 1.3 | 0.65 ± 0.11 |
| Preganglionic | | | | | | | | |
| Pectoralis major * | 2.4 ± 0.4 | 20.6 ± 4.2 | 19.7 ± 4.3 | 0.25 ± 0.11 | 2.1 ± 0.2 | 22.4 ± 4.5 | 20.9 ± 5.9 | 0.30 ± 0.10 |
| Anterior deltoid | 2.6 ± 0.2 | 10.8 ± 2.8 | 11.7 ± 1.8 | 0.03 ± 0.01 | 2.4 ± 0.1 | 12.1 ± 2.4 | 13.9 ± 6.2 | 0.12 ± 0.04 |
| Spinal deltoid | 2.2 ± 0.2 | 16.1 ± 4.0 | 15.8 ± 3.6 | 0.04 ± 0.05 | 2.0 ± 0.1 | 21.4 ± 6.1 | 19.1 ± 7.0 | 0.13 ± 0.02 |
| Biceps long * | 2.6 ± 0.2 | 12.5 ± 2.2 | 15.0 ± 2.8 | 0.02 ± 0.01 | 2.3 ± 0.1 | 16.3 ± 2.1 | 17.2 ± 5.9 | 0.10 ± 0.02 |
| Biceps short * | 2.4 ± 0.2 | 15.7 ± 2.9 | 16.2 ± 2.3 | 0.01 ± 0.01 | 2.0 ± 0.1 | 20.0 ± 3.8 | 18.2 ± 6.1 | 0.03 ± 0.02 |
| Subscapularis | 2.4 ± 0.6 | 15.5 ± 5.2 | 16.6 ± 3.4 | 0.09 ± 0.07 | 2.3 ± 0.2 | 19.3 ± 1.6 | 19.6 ± 5.9 | 0.29 ± 0.07 |
| Supraspinatus | 2.6 ± 0.2 | 14.2 ± 2.8 | 18.2 ± 4.0 | 0.04 ± 0.02 | 2.5 ± 0.1 | 21.2 ± 4.0 | 24.3 ± 6.5 | 0.23 ± 0.08 |
| Infraspinatus | 2.4 ± 0.2 | 17.0 ± 1.5 | 18. 8 ± 2.6 | 0.04 ± 0.02 | 2.4 ± 0.2 | 20.9 ± 3.8 | 22.6 ± 6.3 | 0.19 ± 0.05 |
| Teres major * | 2.1 ± 0.1 | 16.5 ± 3.9 | 16.5 ± 3.5 | 0.09 ± 0.08 | 1.8 ± 0.1 | 23.8 ± 3.6 | 19.1 ± 7.0 | 0.18 ± 0.08 |
| Triceps long | 2.1 ± 0.3 | 23.6 ± 5.2 | 20.6 ± 2.4 | 0.59 ± 0.20 | 2.1 ± 0.1 | 25.1 ± 3.0 | 23.4 ± 6.0 | 0.76 ± 0.15 |

Data are shown as mean ± SD.

* Muscles with significantly longer sarcomeres in affected versus unaffected limbs ($P < .05$).