

Preganglionic and postganglionic neonatal brachial plexus injury effects on muscle properties and range of motion

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INTRODUCTION: Neonatal brachial plexus injury (NBPI) is the most common nerve injury in children, affecting 0.4 to 4 per 1000 newborns [1], with most frequent injury to the C5-C6 level. The injury results in weakness or paralysis of the shoulder and elbow, with internal rotation and adduction contractures as frequent sequelae. Location of the nerve injury relative to the ganglion is known to influence the presentation of contractures [2,3], however the specific relationship between changes to the affected muscle and shoulder range of motion is not clear. The goal of this work was to examine the effect of injury location on muscle architecture and shoulder range of motion (ROM) in a rat model of NBPI.

METHODS: The protocol was approved by the NCState IACUC. An existing rat model of NBPI [4] was used to study the effects of injury location on muscle architecture in 3 experimental groups: preganglionic NBPI (N= 25 ROM, N= 7 architecture), postganglionic NBPI (N= 22 ROM, N= 4 architecture), and sham surgery (N=16 ROM). Neurectomies and sham surgeries were performed at 3-5 postnatal days on a single limb; the contralateral limb was an unaffected control. The preganglionic surgery was achieved through supraclavicular incision, whereas an incision through pectoralis major was performed to inflict the postganglionic injury. At 4 and 8 weeks, maximum passive external rotation (ER) range of motion was assessed using a previously described custom test fixture [5] while the rats were anesthetized under isoflurane gas. Rats were sacrificed after 8 weeks. The specimens were fixed in neutral buffered formalin, and muscles crossing the shoulder in both limbs were dissected and stored in 70% ethanol. Mass was measured using a scientific scale, muscle belly length was measured using calipers, and sarcomere lengths were measured using laser diffraction [6]. Optimal muscle fiber lengths were computed using the following equation: $L^{mo} = L^m * (2.4 \mu m / L^s)$; where L^{mo} is the optimal muscle length, L^m is the muscle length and L^s is the sarcomere length. Differences in ROM, muscle mass and optimal length between affected and unaffected limbs were compared with paired two-sample t-tests. Group differences in passive ER ROM were assessed using one-way ANOVA. Muscle mass and optimal muscle lengths between the preganglionic and postganglionic groups were assessed using unpaired two-sample tests ($\alpha=0.05$).

RESULTS: All three groups showed a tendency for restricted passive ER on the affected limb relative to the unaffected limb at 4 and 8 weeks (preganglionic at 4 weeks $p = 0.1$, 8 weeks $p = 0.12$; postganglionic and sham $p < 0.05$ at 4 and 8 weeks). The preganglionic group had the least restriction in ER (4-week mean = -19.45° ; 8 week mean = 13.24°) and postganglionic group had the most ER restriction (4 week mean = -62.72° ; 8 week mean = -25.60°) (Fig 1) . Severely reduced muscle mass in anterior deltoid, spinal deltoid, biceps long head, biceps short head, subscapularis, supraspinatus, infraspinatus and teres major was observed on the affected shoulder muscles compared to the unaffected shoulder in the preganglionic group ($p < 0.05$). Reduced muscle mass (Fig. 2) on the affected side relative to unaffected side in postganglionic group was observed only in pectoralis major, anterior deltoid, spinal deltoid, bicep long and triceps. Muscle mass was significantly lower in the preganglionic group relative to postganglionic especially in biceps short head, supraspinatus and infraspinatus ($p < 0.05$). Differences in optimal muscle length (Fig. 3) between the affected and unaffected limb, an indicator of restricted longitudinal muscle growth, was observed in the majority of muscles in the preganglionic group, with $p < 0.05$ in supraspinatus, infraspinatus and teres major. This difference was less marked in postganglionic group muscles with only supraspinatus and infraspinatus showing significantly lower ($p < 0.05$) optimal fiber lengths.

DISCUSSION: The postganglionic group exhibited severe restriction in passive ER relative to the preganglionic group [5]. This was also consistent with severe contractures in internal rotation and elbow flexion described in previous studies [2]. ROM is not substantially affected due to preganglionic nature of injuries (e.g. C5-6 nerve root avulsion or nerve root ruptures) due to notable absence of contractures [3]. Computational simulations exhibiting loss of contractures through muscle imbalance have also seen lower effects on ROM as opposed to other mechanisms causing higher contractures [7]. It was previously suggested that preservation of afferent innervation in preganglionic injuries may protect against contractures, and it has been shown to preserve muscle spindle development [2]. However, in the current study, muscle longitudinal growth was severely restricted in the preganglionic group. Other possible explanations for the lower ER restriction in the preganglionic group include the significantly lower muscle mass surrounding the shoulder joint, which may reduce joint stiffness, or the lack of incision in pectoralis major due to the supraclavicular approach. The difference in the muscle mass could therefore be responsible for the difference in shoulder and elbow contractures in the two injury groups, as opposed to differences in optimal muscle length. This work clarifies structural changes to muscle following NBPI, which plays a role in understanding the loads acting on the shoulder joint that contribute to osseous deformities.

SIGNIFICANCE/CLINICAL RELEVANCE: The study provides important insight into the differential effects of nerve injury location in NBPI and its effects on range of motion at the shoulder and the impact on muscle growth and structure. This work provides new evidence critical for understanding the underlying contributions to shoulder and elbow contractures causing deformity and loss of function at the shoulder in NBPI.

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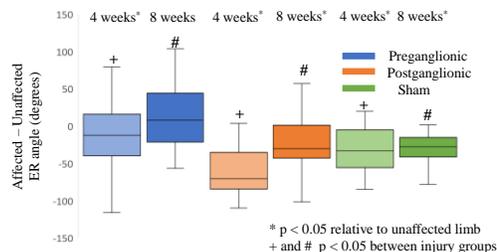


Fig 1: Passive ER range of motion

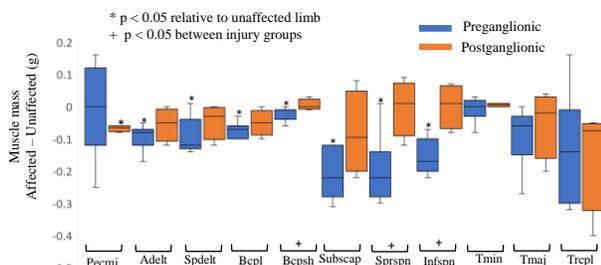


Fig 2: Muscle mass

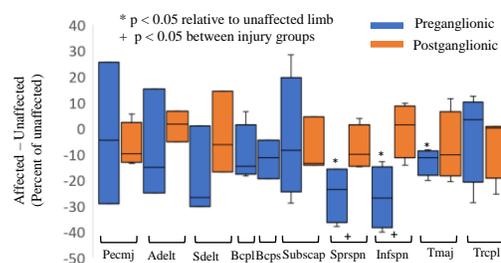


Fig 3: Muscle optimal length