Brachial Plexus Birth Injury Alters Bone Microstructure and Muscle Composition

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Brachial plexus birth injury (BPBI), the most common nerve injury in children1, causes muscle paralysis and often leads to shoulder deformities and lifelong arm impairment in up to 30% of these children2. BPBI causes muscle weakness and morphologic changes in the glenohumeral joint3, but little is known about changes in underlying bone and muscle tissue, especially how they are affected by injury location. Clinically, deformity is more severe with injuries distal to the dorsal root ganglion (postganglionic) than proximal (preganglionic)4. We use unique rodent models of both injuries to improve understanding of musculoskeletal changes with BPBI. Male and female Sprague-Dawley rat pups experienced postganglionic neurectomy3, preganglionic neurectomy5, or sham surgery on one side (affected) with the contralateral (unaffected) side serving as control. After sacrifice at 8 weeks, bones (humeri, scapulae) and muscles (biceps, subscapularis) were harvested. Fixed bones were scanned with micro-CT and analyzed for microstructure6. Snap-frozen, cryosectioned muscles were stained with Masson’s trichrome and analyzed for collagen deposition, indicative of fibrosis. Affected-to-unaffected ratios were computed and compared across groups (ANOVA/Tukey posthoc). Scapular bone microstructure was greatly deteriorated on the affected side for post- and preganglionic groups, significantly more than for sham, most notably with fewer, less connected, and more sparsely arranged trabeculae. Preganglionic bone was also less mineralized than postganglionic or sham. Muscle fibrosis was increased ~20-30% in post- and preganglionic biceps compared with sham. Underlying changes in bone and muscle tissue likely contribute to the unique clinical presentations of BPBI injuries, and understanding the effect of injury location is a crucial step for developing targeted treatments.