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Perspectives on glenohumeral joint contractures and shoulder dysfunction in children with perinatal brachial plexus palsy

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ABSTRACT

Shoulder joint deformities continue to be a challenging aspect of treating upper plexus lesions in children with perinatal brachial plexus palsy (PBPP). It is increasingly recognized that PBPP affects the glenohumeral joint specifically, and that abnormal scapulothoracic movements are a compensatory development. The pathophysiology and assessment of glenohumeral joint contractures, the progression of scapular dyskinesia and skeletal dysplasia, and current shoulder imaging techniques are reviewed.

Introduction

Perinatal Brachial Plexus Palsy (PBPP) represents a challenging part of our pediatric practices. Secondary shoulder deformities in particular are a problem. Natural history studies have shown that 20–30% of children with PBPP will have residual neurologic deficits at 3 years of age.[1] Hoeksma et al reported a 65% incidence of shoulder dysfunction in children with incomplete neurological recovery.[2] However, children with delayed (more than 3 weeks after birth) but ultimately full neurologic recovery still had a 30% incidence of shoulder joint contracture.[2,3] The etiology and treatment of secondary deformities of the shoulder are controversial. Contractures and deformities occur as a child undergoes skeletal and muscular growth, as well as while developing motor control. Loss of motion at the glenohumeral joint (GHJ) stems from the initial nerve injury and from abnormal muscle development coupled with poor movement patterns and altered motor planning. This sequence of events results in glenohumeral and scapulothoracic altered movement rhythms and bony changes. The purpose of this paper is to describe the anatomical changes we see at the GHJ and the subsequent sequelae of movement patterns.

Glenohumeral joint

Pathophysiology of joint and muscle contractures

Adduction and internal rotation contractures of the shoulder are reported as the most common and most severe secondary deformities in PBPP.[4–7] For upper plexus injuries, the most accepted explanation is that of an imbalance between internal and external rotators of the shoulder. According to this explanation, the external rotators and abductors are weak or paralyzed and overpowered by the subscapularis, which receives some of its innervation from the lower plexus.[8] With prolonged unopposed action of the pectoralis major, teres major and latissimus dorsi, posterior subluxation of the humeral head at the GHJ occurs, with retroversion of the glenoid and subsequent deformity of the humeral head and the glenoid.[9]

Recent studies, however, have demonstrated that the subscapularis muscle is also structurally affected, similar to the external rotators. This second hypothesis is supported by an MRI study by Poyhia et al that found amyotrophy of all the muscles of the rotator cuff, with the subscapularis having the most severe

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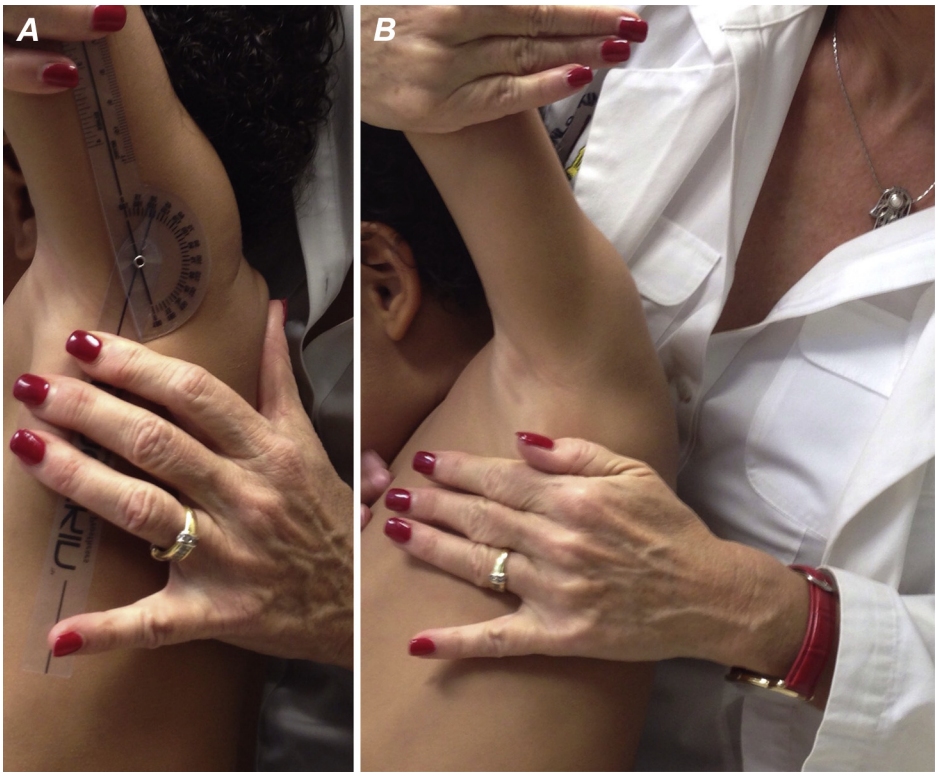

Fig. 1. The scapulohumeral angle in abduction (Courtesy of Shriners Hospital for Children, Houston). A) Technique of measurement with goniometer. B) Demonstration of stretching the inferior GHJ contracture.

atrophy.[10] Van Gelein et al reported similar findings.[11] In addition, they reported that a severe posterior subluxation was predictable by a low infraspinatus to subscapularis volume ratio. These changes may be attributed solely to secondary abnormal forces about the shoulder joint. As the internal rotation posturing progresses, the external rotators (e.g., infraspinatus and teres minor) lengthen, and the internal rotators (e.g., subscapularis) shorten.[12] With muscle length changes, the humeral head internally rotates and shifts posteriorly in the glenoid fossa,[9] altering the line of muscle pull, or action line. This biomechanical alteration could diminish the contraction strength of the external rotators even if they become re-innervated.[12] The muscle changes noted on imaging studies could also be the result of abnormal muscle development resulting from interrupted neural input in the neonatal period. In muscle biopsy studies of the subscapularis muscle of patients with PBPP, the subscapularis muscle demonstrated shorter slack sarcomere length and increased stiffness when compared to healthy muscle.[5,13] Nikolaou et al reproduced brachial plexus injuries in a neonatal mouse model. They reported that muscle growth was impaired, contributing to formation of muscle contracture and limitation of shoulder external rotation.[14]

Assessment of glenohumeral joint limitation

An assessment of functional motion of the total shoulder complex fails to identify the restricted motions of the GHJ because it does not take into account substitutions and excessive motion at the scapulothoracic joint (STJ).[15] In order to assess motion of the GHJ in isolation, we perform measurements of the GHJ while firmly stabilizing the scapula in neutral position.

We evaluate 5 specific motions between the humerus and the scapula which directly correlate to identified GHJ limitations:

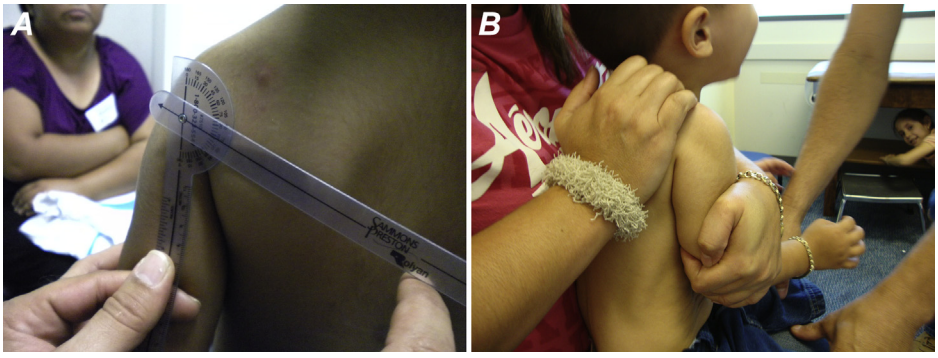

Fig. 2. The scapulohumeral angle in adduction (Courtesy of Shriners Hospital for Children, Houston). A) Technique of measurement with goniometer. B) Demonstration of stretching the superior GHJ contracture.