Posterior Shoulder Dislocation in Infants with Neonatal Brachial Plexus Palsy

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Background: Glenoid dysplasia and posterior shoulder subluxation with resultant shoulder stiffness is a well-recognized complication in infants with neonatal brachial plexus palsy. It is generally considered to be the result of a slowly progressive glenohumeral deformation secondary to muscle imbalance, physeal trauma, or both. Recent publications about infantile posterior shoulder dislocation have suggested that the onset of dysplasia occurs at an earlier age than has been previously recognized. The prevalence of early dislocation in infants with this disorder has not been previously reported, to our knowledge.

Methods: We studied 134 consecutive infants with neonatal brachial plexus palsy who were seen at our institution over a period of two years. All infants were examined at monthly intervals to assess neurological recovery and the status of the upper extremity until recovery occurred or a treatment plan was established. The type of brachial plexus involvement was classified. Specific clinical signs associated with subluxation and dislocation were recorded. These included asymmetry of skin folds of the axilla or the proximal aspect of the arm, apparent shortening of the humeral segment, a palpable asymmetric fullness in the posterior region of the shoulder, or a palpable click during shoulder manipulation. The infants who were identified as having these clinical signs were evaluated with ultrasonographic imaging studies.

Results: Eleven (8%) of the 134 infants had a posterior shoulder dislocation. The mean age at the time of diagnosis was six months (range, three to ten months). There was no correlation between the occurrence of dislocation and the type of initial neurological deficit. A rapid loss of passive external rotation between monthly examinations indicated a posterior shoulder dislocation.

Conclusions: Posterior shoulder dislocation can occur earlier (before the age of one year) and more rapidly in infants with neonatal brachial plexus palsy than has been appreciated previously. As with developmental dysplasia of the hip, a high index of suspicion, recognition of clinical signs, and the use of ultrasonography will allow the diagnosis to be established. Following early diagnosis, attention should be focused on improving the stability and congruency of the shoulder joint.

Level of Evidence: Prognostic study, Level I-1 (prospective study). See Instructions to Authors for a complete description of levels of evidence.

The prognosis for shoulder function following neonatal brachial plexus palsy depends on two related, but independent, factors that limit both the active and the passive range of motion of the shoulder[1]. The first factor is the neurological recovery of the upper brachial plexus, which affects muscle reinnervation and the active range of motion of the shoulder. The second factor is the deformation of the osteocartilaginous shoulder articulation and the soft-tissue contractures that limit the passive range of motion of the shoulder.

Skeletal changes associated with neonatal brachial plexus palsy have received more attention with the introduction of modern imaging techniques[2-7]. Glenoid dysplasia, proximal humeral epiphyseal flattening, and posterior shoulder subluxation and dislocation can be reliably identified in children. These changes in the anatomical shape of the shoulder have, in the past, been attributed to progressive growth alteration as a consequence of untreated contractures or birth-related epiphyseal trauma[8,9].

Whitman[10] recognized the association of neonatal brachial plexus palsy with shoulder subluxation in infancy in 1905. In the study by Fairbank[11], fourteen of twenty-eight patients with shoulder dislocation were less than one year old. Fairbank[11] and Sever[12] attributed shoulder dislocation to initial articular trauma sustained at birth, to splinting devices that held the shoulder in abduction and external rotation, or to muscle imbalance. However, to our knowledge, only nine cases of posterior shoulder dislocation in infants with neonatal brachial plexus palsy have been reported[13-16]. Recent studies have suggested that the true prevalence of this deformity in infants may have been underestimated[2,14,15].

Most studies have indicated that an internal rotation contracture is an important indicator of posterior shoulder dislocation, but other clinical findings have not been stressed. The recommended method of imaging the shoulder of the very young child has been computerized tomography, but ionizing radiation and the need for sedation make this technique less than optimal[14].

We report on a series of eleven infants with neonatal brachial plexus palsy in whom dislocation of the shoulder was identified before the age of one year. The purpose of the present study is to report the prevalence of shoulder dislocation in a consecutive series of children with neonatal brachial plexus palsy and to describe the salient clinical findings and imaging studies that lead to an accurate early diagnosis.

Materials and Methods

We reviewed the records of 134 consecutive infants with the diagnosis of neonatal brachial plexus palsy who were less than one year old when first seen at our institution between April 1997 and April 1999. The patient history was obtained from the parents and any accompanying medical records. The examination of each infant included inspection and palpation of the head, neck, shoulders, chest, and upper extremities. The initial degree of brachial plexus involvement was classified according to the system of Narakas as type 1 (involvement of the C5 and C6 nerve roots), type 2 (involvement of the C5, C6, and C7 nerve roots), or type 3 (involvement of the entire plexus)[17]. At each visit, the active and passive ranges of motion of the shoulder, elbow, wrist, and digits were assessed bilaterally. Motor recovery was assessed by grading muscle-group function as absent, less than antigravity, greater than antigravity, or strong. The presence of contractures was determined by recording the passive range of shoulder motion. The patients were followed at monthly intervals until recovery occurred or a treatment plan was established.

The early management of all infants involved protection of the affected limb followed by range-of-motion exercises that were taught by physicians, supervised by occupational therapists, and performed by the parents at home several times a day. The parents were encouraged to pin the baby's sleeve to the front of the shirt to support the elbow in flexion and to limit internal rotation of the shoulder past the anterior part of the chest. This position is meant to prevent the so-called waiter's tip position, in which the subscapularis, teres major, and latissimus dorsi muscles are most tight. No splint was applied to the shoulder, although a small wrist cock-up splint was used for four children with weak C6 function and absent wrist extension.

External rotation exercises were carried out with the elbow flexed to ≥90° to tighten the medial collateral ligament of the elbow and to protect against valgus stress, with the forearm supinated to tighten the interosseous membrane and to protect the radial head, with the arm adducted to maximally stretch the internal rotators, and with the scapula supported to isolate the glenohumeral joint and to maintain external rotation. All infants who were diagnosed with shoulder dislocation at the initial visit received nonspecific occupational or physical therapy, but in no case was the attention focused on stretching to prevent an internal rotation contracture.

Internal rotation contracture of the shoulder was assessed by measuring passive external rotation with the shoulder adducted, which is the position in which the internal rotators, particularly the superior fibers of the subscapularis, are tightest[18]. Palpation identified changes in the symmetry of the anterior and posterior shoulder regions. Comparisons of axillary and humeral skin folds as well as of humeral segment length were made (Fig. 1). Dynamic signs of instability, such as translation or a click during rotation, were recorded. Infants who maintained passive external rotation and lacked the physical findings of dislocation continued to be managed with stretching exercises.

When abnormal findings were recognized on physical examination of the shoulder, diagnostic imaging studies were performed. In children less than one year old, the ossific nucleus of the humeral head is usually small enough to allow imaging of the glenohumeral joint with ultrasonography by a radiologist who is familiar with the technique (Fig. 2). Ultrasonography was performed in the axial plane through a posterior approach and could be accomplished without sedation with the infant held by the parent. The uninvolved shoulder served as the control. Dislocation was diagnosed when the center of the humeral head projected posterior to the axis of the scapula. Abnormal, excessive posterior sloping of the ossified glenoid was usually present. Standard axillary views were made for infants who were more than six to nine months old, in whom the proximal humeral epiphysis was likely to be well ossified.

Results

Posterior dislocation of the shoulder was identified before the age of one year in eleven (8%) of 134 patients with neonatal brachial plexus palsy (see Appendix). An additional infant who had transient anterior instability of the shoulder that responded to immobilization is not included in this group. There were five girls and six boys. All patients were born by cephalic presentation and vaginal delivery. The average birth weight was 4.2 kg (range, 3.6 to 4.8 kg). The right side was involved in five infants, and the left side was involved in six. None of the infants sustained associated skeletal or articular trauma to the shoulder during or after delivery. No patient had had previous shoulder or brachial plexus surgery or splint application. The average age at the time of diagnosis of the shoulder dislocation was six months (range, three to ten months).

Clinical Findings

Most infants with neonatal brachial plexus palsy have some tightness of the internal rotators of the shoulder and lose both active and passive external rotation. The salient clinical finding indicative of a posterior dislocation of the shoulder in our patients was the perception of a loss of passive external rotation between monthly visits. In the seven patients who had been followed prior to the diagnosis of the shoulder dislocation, the mean loss of passive external rotation was 61° (range, 40° to 90°). In the other four infants, the posterior shoulder dislocation was diagnosed at the first visit. The mean passive external rotation at the time of diagnosis of the posterior shoulder dislocation was −10° (range, −30° to 5°).

Palpation of the shoulder revealed definite posterior fullness caused by the displaced humeral head. This fullness was best appreciated in comparison with the normal shoulder. During external rotation maneuvers, a reduction click was identified in four infants.

Other clinical signs that were appreciated in all eleven infants included an apparent shortening of the humeral segment of the involved extremity; asymmetry of the soft-tissue folds of the proximal part of the arm; and a deep, asymmetrical axilla (Fig. 1).

Pattern of Plexus Involvement and Recovery

There appeared to be no relationship between the location or extent of the nerve lesion in the brachial plexus and the presence of a shoulder dislocation. Six infants had involvement of the C5 and C6 nerve roots (Narakas type-1 involvement), two infants had involvement of the C5, C6, and C7 nerve roots (Narakas type-2 involvement), and three infants had involvement of the entire plexus (Narakas type-3 involvement). One of the patients with type-3 involvement had a Horner sign. However, the common persistent motor deficit, seen in all eleven patients, was absence of active external rotation of the shoulder.

The degree and timing of the pattern of neurological recovery also varied. At the time of diagnosis of the shoulder dislocation, five of the six patients with Narakas type-1 palsy had already recovered antigravity elbow flexion. Antigravity function of the anterior deltoid was identified in two patients in this group. Of the two patients with Narakas type-2 palsy, one exhibited early recovery of antigravity elbow flexion and deltoid function. The other infant had less than antigravity elbow flexion and no deltoid function when the posterior shoulder dislocation was diagnosed at the age of five months. Two of the three infants with Narakas type-3 palsy had begun to recover elbow flexion by the time that the posterior shoulder dislocation was recognized.

Ultrasonographic Findings

The initial patients were studied with use of ultrasonography in the axial plane from the lateral aspect of the shoulder. This view produced an image of the rounded cartilaginous humeral head sitting atop the glenoid, simulating a scoop of ice cream on a cone. These images were highly dependent on the angle of imaging, and the ability to visualize the glenoid decreased greatly with advancing ossification of the humeral head; consequently, this technique was found to be difficult and somewhat subjective. The posterior approach as described by Hunter et al.[15] was adopted for the evaluation of the later patients. This technique produced better static images that were more easily reproduced and interpreted. In addition, the technique was more readily learned and understood by our sonographers because of its remarkable similarities with hip ultrasonography, a frequently performed study.

Images that are made with the posterior approach demonstrate the humeral head in relation to the posterior surface of the scapula (the posterior scapular line). Ossification of the humeral head does not interfere with these anatomic landmarks. In normal shoulders, the center of the humeral head lies anterior to this line, whereas in subluxed and dislocated

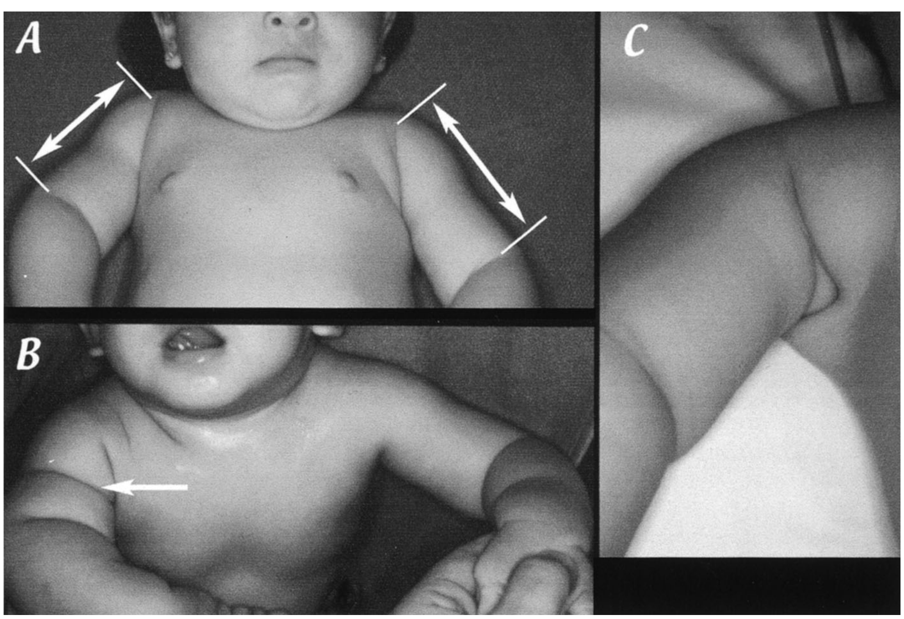

Fig. 1

Case 9. *A,* Apparent shortening of the humeral segment is seen on the involved, right side. *B,* An extra skin fold (arrow) on the involved side is caused by telescoping of the humerus posteriorly in the soft-tissue envelope. *C,* The axilla on the involved side appears deeper, with additional skin folds.

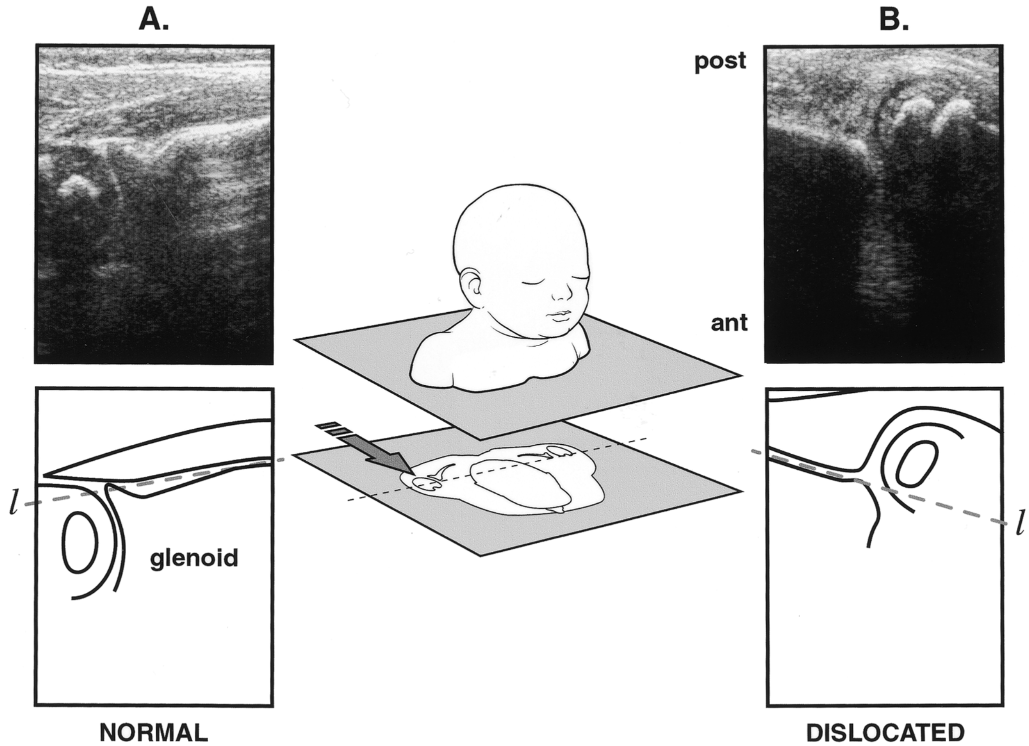

Fig. 2

A, Ultrasonogram (and corresponding illustration) showing congruous reduction of the glenohumeral joint. The ossific nucleus lies anterior to the axis of the scapula. *B,* Ultrasonogram (and corresponding illustration) showing the dislocated shoulder with the humeral head clearly seen posterior to the line of the scapula (l). The glenoid appears to be sloping posteromedially.

shoulders there are varying degrees of posterior displacement of the head relative to this line. The posterior rim of the glenoid is normally sharply defined, but it becomes flattened and slopes posteriorly, indicating retroversion or pseudoglenoid formation.

Of the eleven patients with suspected posterior shoulder dislocation, six initially were evaluated with use of the lateral approach and five were evaluated with use of the posterior approach. Varying degrees of posterior displacement of the humeral head were confirmed in all patients. The distinction between marked subluxation and dislocation was not easily made in some of the shoulders as there appears to be a continuum of dysplastic findings.

Discussion

At the beginning of the last century, clinical descriptions of posterior dislocation of the shoulder were frequently reported[10,11,19]. At that time, the radiographic evaluation of infants was not routine and ultrasonography was not available. Perhaps at a time with less reliance on sophisticated imaging and more reliance on well-practiced skill in physical examination, the diagnosis was better appreciated. Royal Whitman, considering what he termed the "congenital dislocation," concluded that there were three different variants[10]. The first variant was a true congenital dislocation sustained in utero. The second variant was a traumatic form that was due to extraction maneuvers and was considered by Whitman to be very uncommon. The third, and by far the most common, variant was dislocation due to muscle imbalance resulting from a brachial plexus palsy. Fairbank described twenty-eight cases of posterior shoulder dislocation in thirty-seven patients with brachial plexus palsy[11]. Fourteen of the infants with shoulder dislocation were less than one year old. Fairbank incriminated muscle imbalance as the primary pathogenic mechanism. Sever, in his report on 1100 cases of birth-related brachial plexus palsy, emphasized the importance of dealing with the posteriorly dislocated shoulder early on, although he did not report the prevalence of posterior dislocation in infants[12].

Since these early descriptions, only nine cases of posterior shoulder dislocation in infants with neonatal brachial plexus palsy have been reported[13-16]. Another four cases of anterior dislocation have been reported[20,21]. Troum et al. stated that "It is difficult to diagnose a posterior dislocation of the humeral head in an infant; the condition is rare and therefore is not immediately considered."[13]

There was little or no prereferral selection bias in the present study. The majority of the infants were referred directly by the newborn nurseries or by the pediatricians for the initial evaluation and treatment. Therefore, the prevalence of posterior shoulder dislocation in the present study (8%; eleven

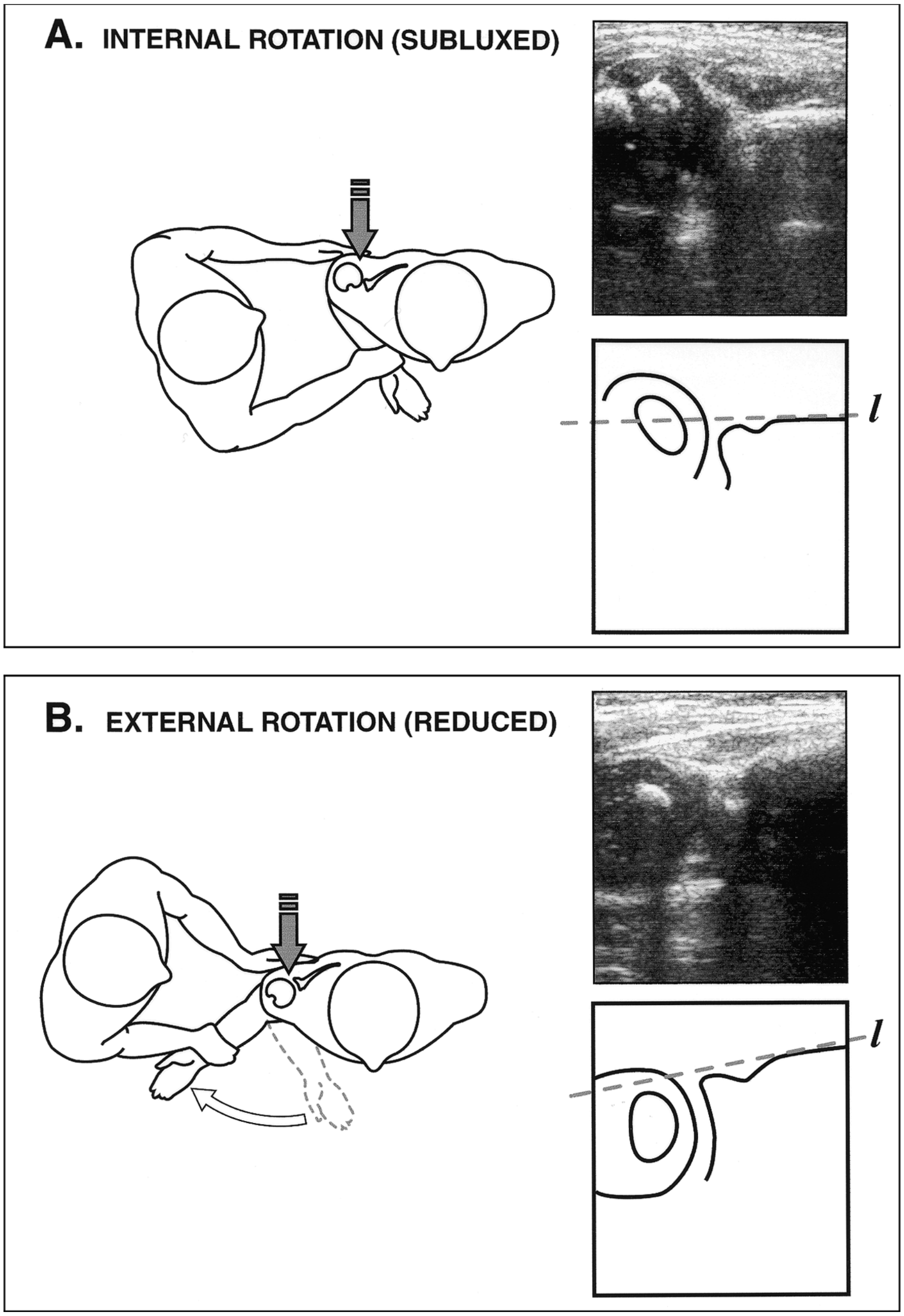

Fig. 3

Ultrasonography allows for a dynamic assessment of the reducibility of the dislocation. Note that the shoulder is subluxed in internal rotation *(A)* and reduced in external rotation *(B).*

of 134) may be low, depending on the referral base of the individual practice.

Posterior shoulder dislocation has been commonly reported in older children with neonatal brachial plexus palsy. Wickstrom et al. reported a prevalence of 9% in a series of fifty-four older children[22]. Torode and Donnan, on the basis of a statistical calculation that was based on the number of births, the incidence of brachial plexus palsy, and the findings in their patient population, believed that the prevalence of shoulder dislocation in children with neonatal brachial plexus palsy could be as high as 40%[14]. Zancolli and Zancolli described 148 patients with brachial plexus palsy, of whom seventy-eight (53%) had "upper arm sequelae," including sixty-four who had a shoulder contracture[9].

Torode and Donnan reported a high rate of posterior shoulder dislocation in their series, which included both children and infants[14]. They suggested that the diagnosis of dislocation might have been missed in early infancy because the accuracy of traditional radiographic studies was impeded by the primarily cartilaginous nature of the shoulder.

Since we have become aware of this condition and now actively look for the clinical signs, our experience has suggested that glenohumeral dysplasia with shoulder dislocation in infants with neonatal brachial plexus palsy is much more frequent than generally has been appreciated.

The hallmark clinical sign of shoulder dislocation is loss of external rotation. The other clinical signs can easily be overlooked in a routine examination of an uncooperative infant. Apparent shortening of the humeral segment of the involved extremity combined with asymmetrical skin folds becomes more obvious when specifically sought. True shortening due to impaired growth of the involved extremity in a child with neonatal brachial plexus palsy may occur gradually over a period of months to years. This is in contrast to the apparent shortening that is seen when the humeral head lies posterior to the glenoid. Such shortening results in telescoping of soft tissues about the shoulder, producing extra skin folds and creases and a deep axilla. The third useful clinical sign is the fullness in the posterior part of the shoulder that is created by the prominence of the humeral head. Comparison with the uninvolved side reveals both visible and palpable fullness.

The interpretation of radiographic images of an infant's shoulder can be challenging. With use of standard radiographic imaging techniques, the three-dimensional relationship of the glenoid to the humeral head must be inferred from the ossified humeral and scapular elements.

Ossification of the proximal humeral epiphysis normally begins during the third month after birth. The secondary ossification center is located in the cartilaginous humeral head in an eccentric medial and slightly posterior position with respect to the humeral shaft[23]. The radiographic projection of the ossified nucleus of the proximal humeral epiphysis on the anteroposterior radiograph is markedly influenced by the rotation of the proximal part of the humerus[24]. On the scapular side, the osseous contour of the normally concave glenoid fossa appears convex on the axillary lateral view during the first year of postnatal development[25]. The inferior margin of the glenoid does not ossify until puberty[23]. In a normal shoulder, the axis of the scapular body bisects the humeral head. In an infant with posterior dislocation or subluxation of the shoulder, the proximal humeral ossific nucleus lies posterior to the mediolateral axis of the scapula[14].

Arthrography, computerized tomography, and magnetic resonance imaging are not preferred methods of diagnosis because they all require general anesthesia or sedation of the infant. At our institution, ultrasonography has become the imaging study of choice for these patients. It is free of ionizing radiation, requires no sedation, and allows dynamic assessment of both the ability to reduce the shoulder and the degree of instability[26]. Ultrasonographic evaluation from the lateral aspect of the shoulder can be used to visualize the glenoid through an unossified, sonolucent humeral head. This view is later limited by the size of the ossific nucleus. We found it to be reliable until the age of approximately six months. Hunter et al. described the posterior technique for ultrasonographic evaluation of the infant's shoulder[15] (Fig. 2). Ossification of the proximal humeral epiphysis does not impair visualization of the glenohumeral relationship with this approach. This method can be very sensitive for establishing the diagnosis of dysplasia or early subluxation and for performing a dynamic analysis of shoulder rotation (Fig. 3).

Although we did not have sequential studies that documented the entire progression of subluxation to dislocation in any single patient, comparison of different degrees of posterior subluxation and dislocation showed progressive posterior sloping and deformation of the posterior glenoid lip. Waters et al. measured percentages of translation of the humeral head relative to the scapular axis on computerized tomographic scans or magnetic resonance images to better define the degree of subluxation and dislocation in forty-two patients, only one of whom was less than one year old[7]. The distinction between posterior subluxation and dislocation is more difficult to define with ultrasonography; however, our findings correlate best with Waters type-VI infantile dislocation of the humeral head.

Just as the term "dysplasia of the hip" has supplanted "congenital dislocation of the hip" to encompass "a dynamic disorder potentially capable of getting better or worse as the child develops . . . depending on the multidisciplinary care,"[27] the term "dysplasia of the shoulder" may be appropriate here. Dysplasia, when applied to the hip, includes both subluxation and dislocation and is defined by deficient development of the acetabulum[28]. When progressive displacement occurs in the shoulder, contact between the articular components may not be lost completely but is accompanied by cartilaginous deformation of both the glenoid and the humeral head followed by osseous remodeling. In the setting of acquired neuromuscular imbalance due to brachial plexus palsy, both dislocation and subluxation will contribute to poor functional outcomes.

It is reasonable to consider the mechanism of this glenohumeral dysplasia in the context of the clinical findings. Muscle imbalance combined with the plasticity of the glenohumeral joint in the infant appears to be responsible for the dysplasia and dislocations. In a normal shoulder, the concentric alignment of the humeral head within the glenoid fossa is made possible by the balance between the normal shape of the joint and the muscles of the rotator cuff combined with the integrity of afferent proprioceptive and efferent motor tracts. These are likely to be abnormal in the early course of recovery from neonatal brachial plexus palsy, and we postulate the following mechanism. Unopposed internal rotation brings the deltoid tuberosity into a position anterior to the glenohumeral joint. Pectoralis major muscle function positions the shoulder in forward flexion. Any contraction of the anterior deltoid then becomes particularly effective for pushing the humeral head directly posteriorly. As the humeral head moves posteriorly, the posterior aspect of the labrum and the articular surface of the glenoid first flatten and then are pushed posteriorly and medially. As the deltoid insertion moves closer to the acromion, the anterior deltoid muscle becomes shorter and the humeral segment appears shorter. This mechanism is consistent with Zancolli and Zancolli's[9] findings of a residual abduction contracture due to shortening of the anterior deltoid in children who have posterior displacement of the humeral head as well as our operative findings at the time of open reduction of these shoulders. Weight-bearing on the involved elbow in a crawling position may exacerbate the subluxating force; however, dislocation occurred in some of our children before they had reached that point of motor development.

In conclusion, the present study suggests that posterior shoulder dislocation in infants with neonatal brachial plexus palsy is more frequent than has generally been realized. Our review of 134 consecutive children with neonatal brachial plexus palsy who were seen over a two-year period demonstrated that eleven infants (8%) had a subsequent posterior shoulder dislocation. Physical examination of these infants revealed loss of passive external rotation, skin-fold asymmetry, apparent shortening of the humeral segment, and palpable abnormalities in the axilla and the posterior part of the shoulder. These findings were most definitively confirmed with ultrasonography. If the dysplastic process is recognized early, it may be possible to alter the outcome by early reduction of the joint and correction of the deforming forces.

Appendix

eA A table presenting the clinical findings for all eleven patients with posterior dislocation of the shoulder is available with the electronic versions of this article, on our web site at www.jbjs.org (go to the article citation and click on "Supplementary Material") and on our quarterly CD-ROM (call our subscription department, at 781-449-9780, to order the CD-ROM). ■

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References

1. **Tachdjian MO.** *Pediatric orthopedics.* Philadelphia: WB Saunders; 1972. Affections of the peripheral nerves; p 1036-56.
2. **Beischer AD, Simmons TD, Torode IP.** Glenoid version in children with obstetric brachial plexus palsy. *J Pediatr Orthop.* 1999;19:359-61.
3. **Gudinchet F, Maeder P, Oberson JC, Schnyder P.** Magnetic resonance imaging of the shoulder in children with brachial plexus birth palsy. *Pediatr Radiol.* 1995;25 Suppl 1:S125-8.
4. **Hernandez RJ, Dias L.** CT evaluation of the shoulder in children with Erb's palsy. *Pediatr Radiol.* 1988;18:333-6.
5. **Pearl ML, Edgerton BW.** Glenoid deformity secondary to brachial plexus birth palsy. *J Bone Joint Surg Am.* 1998;80:659-67. Erratum in: *J Bone Joint Surg Am.* 1998;80:1555-9.
6. **Pollock AN, Reed MH.** Shoulder deformities from obstetrical brachial plexus paralysis. *Skeletal Radiol.* 1989;18:295-7.
7. **Waters PM, Smith GR, Jaramillo D.** Glenohumeral deformity secondary to brachial plexus birth palsy. *J Bone Joint Surg Am.* 1998;80:668-77.
8. **Gilbert A.** Obstetrical brachial plexus palsy. In: Tubiana R, editor. *The hand.* Volume IV. Philadelphia: WB Saunders; 1993. p 575-601.
9. **Zancolli EA, Zancolli ER Jr.** Palliative surgical procedures in sequelae of obstetrical palsy. In: Tubiana R, editor. *The hand.* Volume IV. Philadelphia: WB Saunders; 1993. p 602-23.
10. **Whitman R.** The treatment of congenital and acquired luxations at the shoulder in childhood. *Ann Surg.* 1905;42:110-5.
11. **Fairbank HAT.** Birth palsy: subluxation of the shoulder-joint in infants and young children. *Lancet.* 1913;1:1217-23.
12. **Sever JW.** Obstetric paralysis. Report of eleven hundred cases. *JAMA.* 1925; 85:1862-5.
13. **Troum S, Floyd WE 3rd, Waters PM.** Posterior dislocation of the humeral head in infancy associated with obstetrical paralysis. A case report. *J Bone Joint Surg Am.* 1993;75:1370-5.
14. **Torode I, Donnan L.** Posterior dislocation of the humeral head in association with obstetric paralysis. *J Pediatr Orthop.* 1998;18:611-5.
15. **Hunter JD, Franklin K, Hughes PM.** The ultrasound diagnosis of posterior shoulder dislocation associated with Erb's palsy. *Pediatr Radiol.* 1998; 28:510-1.
16. **Dunkerton MC.** Posterior dislocation of the shoulder associated with obstetric brachial plexus palsy. *J Bone Joint Surg Br.* 1989;71:764-6.
17. **Narakas AO.** Injuries of the brachial plexus. In: Bora FW Jr, editor. *The pediatric upper extremity: diagnosis and management.* Philadelphia: WB Saunders; 1986. p 247-58.
18. **Kendall FP, McCreary EK.** *Muscles, testing and function.* 3rd ed. Baltimore: Williams and Wilkins; 1983.
19. **Steindler A.** *Reconstructive surgery of the upper extremity.* New York: Appleton; 1923.
20. **Babbitt DP, Cassidy RH.** Obstetrical paralysis and dislocation of the shoulder in infancy. *J Bone Joint Surg Am.* 1968;50:1447-52.
21. **Flores M, Burgess DM.** Obstetric brachial plexus palsy with anterior dislocation of the shoulder. *Injury.* 1997;28:148-9.
22. **Wickstrom J, Haslam ET, Hutchinson RH.** The surgical management of residual deformities of the shoulder following birth injuries of the brachial plexus. *J Bone Joint Surg Am.* 1955;37:27-36, passim.
23. **Wilkins KE.** Special problems with the child's shoulder. In: Rockwood CA, Matsen FA, editors. *The shoulder.* Philadelphia: WB Saunders; 1990. p 1033-87.
24. **Goddard N.** The development of the proximal humerus in the neonate with particular reference to bony lesions around the shoulder. In: Tubiana R, editor. *The hand.* Volume IV. Philadelphia: WB Saunders; 1993. p 624-31.
25. **Ogden JA, Phillips SB.** Radiology of postnatal skeletal development. VII. The scapula. *Skeletal Radiol.* 1983;9:157-69.
26. **White SJ, Blane CE, DiPietro MA, Kling TF Jr, Hensinger RN.** Arthrography in evaluation of birth injuries of the shoulder. *Can Assoc Radiol J.* 1987; 38:113-5.
27. **Klisic P, Zivanovic V, Brdar R.** Effects of triple prevention of CDH, stimulated by distribution of "baby packages." *J Pediatr Orthop.* 1988;8:9-11.
28. **Herring JA.** Developmental dysplasia of the hip. In: Herring JA, editor. *Tachdjian's pediatric orthopaedics.* 3rd ed, vol 1. Philadelphia: WB Saunders; 2002. p 513-654.