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1. Introduction

The objectives of this book chapter are to describe the diagnostics, clinical assessment and surgical management of obstetric brachial plexus injury (OBPI) or brachial plexus birth palsy (BPBP).

OBPI occurs during delivery process. The incidence of OBPI has been reported to vary between 0.38 and 5.8 for every 1000 live births [1-5]. The occurrence has increased despite the advances in obstetrics, and medical technology [6, 7]. Reported risk factors for OBPI include shoulder dystocia, macrosomia (defined as birth weight greater than 4500 g) [8-11], instrument-assisted delivery, and downward traction of the fetal head [3, 12, 13]. Shoulder dystocia is the most prevalent risk factor in our patients [14]; almost all the children in our study had documented shoulder dystocia [14]. Shoulder dystocia is, therefore, closely associated with the most severe cases of permanent obstetric brachial plexus injuries [8, 9, 11]. However, permanent injury is not exclusive to large infants; 80% of the OBPI patients in our published study were not macrosomic and 43% (104/241) weighed less than 4000 g at birth [14]. OBPI that occur during breech deliveries may have a different mechanism of onset, and are more likely to be bilateral. Avulsions of the upper roots are more likely during breech than during vertex delivery [15, 16]. OBPI may also occur, although very rarely, during cesarean sections [17].

The most commonly affected roots are C5–6 (Erb’s palsy), because of their more superficial location in the neck, are more vulnerable to injury. Less frequently, the entire plexus (C5–T1) may be affected [18, 19]. The injury can be simple stretch or rupture or avulsion. Most of these injuries are transient; patients recover functions spontaneously within the 3 months of life. However, a significant proportion of these children tend to retain persistent limb deficits, never recover full function and develop permanent injuries [1, 20, 21]. Unlike adults, children may have complications from even the most simple nerve injury due to the growth issues that are
present. The mildest and most common OBPI is neurapraxia; the most severe is avulsion [20, 22]. Both types of injury have the potential to result in permanent disability.

2. Diagnostics

Diagnostic tools used to identify which lesions are permanent in OBPI include computed tomography (CT), magnetic resonance imaging (MRI), myelogram, and electromyography (EMG) as well as nerve conduction velocity (NCV) studies [23-27]. Distinguishing preganglionic (avulsion) from postganglionic (rupture) lesions is critical, and can be difficult at initial presentation based on clinical examination alone in these infants [26, 28]. Our experience with MRI for pre-operative assessment of the spinal roots has been unfavorable. EMG testing is the procedure of choice for preoperative evaluation of nerve-muscle integrity.

2.1. Radiological evaluation

In order to assess bony deformities of the shoulder joint, CT or MRI images of the patients are studied before and after triangle tilt surgery. Posterior humeral head subluxation, glenoid version, and SHEAR deformity are measured from the radiographs (CT/MRI scans). Glenoid version (normal value=0) is measured as described by Friedman et al. [29] using axial CT/MRI images (Figure 1). A scapular line connecting the mid-glenoid to the medial spine of the scapula is constructed using Universal Desktop Ruler (AVPSoft.com, Voronezh, Russia). The angle formed between the scapular line and a line drawn tangential to the glenoid surface interacting closely with the humeral head is calculated and 90° is subtracted from it to measure the glenoscapular angle. Posterior subluxation of the humeral head (Figure 1) is expressed as percentage of humeral head anterior to the glenoid (PHHA, normal value=50), and calculated from the ratio of the distance between the scapular line to the anterior aspect of humeral head and the greatest diameter of the head, multiplied by 100.

The scapular deformity, also referred as SHEAR deformity is measured from the 3D reconstructions of the CT images (Figure 2). The area of the scapula visible above the clavicle is measured and divided with the total area of the scapula for both affected and normal sides. The ratio of the affected side is subtracted from that of the normal side and multiplied with 100 to obtain SHEAR deformity (normal value=0).

2.2. Clinical assessment

Shoulder function is assessed through the modified Mallet scale through video recordings of patients performing the following movements: shoulder abduction, external rotation, hands to mouth, hands to neck, hands to spine, and supination. For each functional Mallet parameter, patients are scored on a scale of 1–5 with 5 as normal function, and 1 denoting lack of any movement (Figure 3). Despite continuing improvements in diagnostic technology, at this time, the final diagnoses must be made during surgery in complex or unclear cases.
Figure 1. Schematic drawing showing the method of calculating glenoscapular angle (glenoid version $\theta$), posterior subluxation of the humeral head and spinoscapular angle ($\zeta$) [40].

![Schematic drawing showing the method of calculating glenoscapular angle (glenoid version $\theta$), posterior subluxation of the humeral head and spinoscapular angle ($\zeta$) [40].](image)

(C/(C+D) – A/(A+B)) x 100 = scapular elevation (%)

Figure 2. Measuring scapular elevation to quantitate the extent of the SHEAR deformity. Shown here is the CT for a patient with 37% scapular elevation [42].
Figure 3. Modified Mallet scale evaluation of function and arm appearance. In addition to assessing the classical shoulder functions of the classical Modified Mallet system, supination and the resting position are evaluated. [19]

3. Non-surgical interventions

Non-Surgical interventions are physical and occupational therapies, electrostimulation, neuromotor therapy, BTX-A injections and splinting. Among the most promising of these methods is BTX-A treatment. This has been shown to treat biceps/triceps co-contraction [30-32], and shown to improve biceps movement and strength [33]. BTX-A treatment for OBPI has not been effective over long-term, in our experience.

4. Primary surgical solutions

There are three primary surgical solutions: neurolysis, neurotization (nerve transfer) and nerve grafting. These may be performed alone or in combination with each other. The choice of which peripheral nerve surgery technique is appropriate is based on which method will maximize
and encourage the natural regenerative process of the nerve. We use nerve transfer and grafting, depending on the intra-operative findings and with our surgical experiences. Injuries of C5-C6 do not always warrant surgery if injury to C7 is minor. In patients where C8 and T1 are involved in addition to C5-C7, hand function is also affected, therefore nerve repair for the upper and middle trunks is more likely to be required.

5. Surgical management

The decision of whether to surgically repair the nerves, however, does typically need to be made in the first 6 months of life. Delay beyond this age in these patients lead to long-term morbidity by causing muscle imbalances and weakness around the shoulder (the deltoid and external shoulder rotators) [34-37], and bony deformities at the shoulder joint (glenohumeral dysplasia and joint incongruity) [20, 38, 39]. These anatomical changes subsequently severely impair the bone growth and development [20]. The major bony deformity that develops is termed as the SHEAR (scapular hypoplasia, elevation, and rotation) deformity, which is caused by the elevation and extrusion of the affected scapula beyond the clavicle [40]. The abnormal anterior rotation of the clavicle together with the protracted scapula causes the acromioclavicular plane to tilt forward and thereby lead to the impingement of the acromion upon the humeral head [41, 42]. Significant secondary deformities that follow include medial rotation contracture (MRC) and elbow flexion.

Early surgical interventions have been shown to improve the limb functions in this group of patients [43, 44]. Management of secondary deformities in OBPI has typically been through the performance of various operative procedures including tendon transfers, muscle releases, axillary nerve decompression, humeral osteotomy, biceps tendon lengthening, glenohumeral capsulorrhaphy and anterior capsule release [45-51].

5.1. Z-lengthening

Biceps tendon lengthening/ the Z-lengthening is an option in C5-C7 (asymmetric) nerve injury, where the biceps recovers faster, thereby overpowering the triceps. The added length that is achieved allows straightening of the elbow and provides additional length to the arm [19].

5.2. Ilizarov bone lengthening

In severe OBPI patients, who is left with severe bony rotational and shortening deformities that are functionally limiting, the use of Ilizarov bone distraction technique is appropriate. This technique is used for rotation and lengthening of the humerus as well as the forearm. Functional gains are significant as the hand is placed into a more useful position [19].

5.3. Posterior glenohumeral capsulorrhaphy

Posterior glenohumeral capsulorrhaphy tightens the posterior capsule surrounding the humeral head and repositions it anteriorly. This procedure does not address the SHEAR
deformity [40] and its central influence in the pathophysiology of the medial rotation contracture. In our experience, on its own, posterior capsulorrhaphy is often not sufficient to address the glenohumeral subluxation, as is predictable when taking the SHEAR into consideration. In our experience, successful restoration of position and function in failed humeral osteotomy patients has followed from surgically addressing the SHEAR deformity. It may be inferred that the SHEAR correction, the Triangle Tilt surgery is a more specific operation because it addresses the root cause of the medial rotation.

6. Muscle and bone deformities and their management

We have been less aggressive to nerve reconstruction and paying more attention to the secondary and tertiary consequences of the initial nerve injury, based on developing and quite compelling literature and on our own experience with several thousand patients [52]. We described our preferred management for muscle injury as a result of OBPI, with supporting clinical and literature evidence. The traditional muscle release operations do not adequately address the pathophysiology of the shoulder in OBPI patients in our experience. Therefore, the surgeon and the lead author (RKN) [53-55] has modified the previously described soft tissue release operation [56], by coupling neurolysis and decompression of the axillary nerve with an untethering release of soft tissue contractures (modified Quad, figure 4) [53-55]. We have demonstrated that modified Quad [53-55] lead to better shoulder abduction and flexion through releasing the existing contractures.

6.1. Modified Quad (figure 4)

1. Transfer of the latissimus dorsi muscle to give external rotation and abduction.
2. Transfer of the teres major muscle to stabilise the scapula.
3. Release of the subscapularis, pectoralis major and minor contractures.
4. Decompression and neurolysis of the axillary nerve [53, 54].

However, these procedures may not address the glenohumeral dysplasia and joint incongruity. Restoration of glenohumeral congruity is therefore a primary objective in treating OBPI, which then allows for maximum functional range of motion and improved limb growth. A bony surgical procedure, Triangle Tilt (figure 5) [42, 44, 57-66] was therefore developed by the lead author and surgeon (RKN).

6.2. The triangle tilt surgery consists of

1. osteotomy of the clavicle at the junction of the middle and distal thirds,
2. osteotomy of the acromion process at its junction with the spine of the scapula,
3. ostectomy of the superomedial angle of the scapula to reduce scapular winging,
4. splinting of the extremity in adduction, external rotation and forearm supination. This triangle tilt detaches the distal acromio-clavicular triangle-humeral head complex from the abnormally positioned scapula, and tilts the acromio-clavicular plane back to neutral position. This relieves the impingement of the acromio-clavicular triangle on the humeral head and allows the head to be repositioned passively into a neutral position within the glenoid fossa (Figure 5), resulting in improved gleno-humeral joint congruency [42, 44, 57-66].

We have demonstrated the short (1 year), and long-term (2 years), and extended long term (5 years) benefits of triangle tilt surgery in OBPI patients (age between 0.9 and 17 year old) by examination of their radiological reports as well as the modified Mallet functional scale [42, 44, 57-66]. In addition, triangle tilt surgery is a salvage procedure in failed humeral osteotomy patients [58, 65]. Minor elements of the procedure include bone grafting of the acromion process and clavicular osteotomy sites and semi-rigid fixation of the clavicular osteotomy segments to prevent nonunion [19].
Figure 5. Artist's rendering of the triangle tilt surgery and anterior capsule release. Illustrated are osteotomies of the clavicle, scapula, and acromion process, along with glenohumeral capsulodesis and anterior capsule release. [19]
7. Conclusions

Based on our own experience with several thousand OBPI patients, we address primarily the muscle and bony operations, and we are less aggressive to nerve reconstruction. These procedures directly address the anatomy of the glenohumeral joint, and thereby resulting in the best possible overall functional outcome. In addition, this minimize the morbidity, expense and the invasiveness of surgery. Nerve reconstruction is reserved for those less common cases where the C5 and C6 nerve roots will not recover.

Modified Quad surgery improves median nerve conduction, and active abduction in young, as well as teen OBPI patients. The triangle tilt surgery improves all shoulder functions significantly in short (1 year), and long-term (2 years), and extended long term (5 years) follow-up, and further the functions are maintained over the extended time in these patients. We recommend that the triangle tilt surgery be performed before the age of 2 years for optimal improvements in outcomes of clinical functioning. This surgery can be performed as early as nine months, and up until late adolescence (16-17 years). Optimal clinical outcomes are achieved if this surgical procedure is performed before the age of 2 years, but improvements in functioning are observed if the surgery is performed after this age as well.

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