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MAGNETIC RESONANCE IMAGING AND ULTRASONOGRAPHY
IN BRACHIAL PLEXUS BIRTH INJURY

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Academic Dissertation
To be presented with the permission of
The Faculty of Medicine of the University of Helsinki,
For public discussion in Small Hall, Main building of the University of Helsinki,
On 20th of May 2011 at 12 noon.

Helsinki 2011
To Reino
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II  Pöyhiä T, Nietosvaara Y, Remes V, Kirjavainen M, Peltonen J, Lamminen A.

III Pöyhiä T, Koivikko M, Peltonen J, Kirjavainen M, Lamminen A, Nietosvaara Y.

IV  Pöyhiä T, Lamminen A, Peltonen J, Willamo P, Nietosvaara Y.
    Accepted for publication.

The publishers of original publications kindly granted their permission to reproduce the articles in this thesis.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>BPBI</td>
<td>brachial plexus birth injury</td>
</tr>
<tr>
<td>CT</td>
<td>computed tomography</td>
</tr>
<tr>
<td>DW</td>
<td>diffusion-weighted</td>
</tr>
<tr>
<td>EMG</td>
<td>electromyography</td>
</tr>
<tr>
<td>ENMG</td>
<td>electroneuromyography</td>
</tr>
<tr>
<td>FCU</td>
<td>flexor carpi ulnaris</td>
</tr>
<tr>
<td>F0-F3</td>
<td>degree of fatty infiltration 0-3</td>
</tr>
<tr>
<td>GH</td>
<td>glenohumeral</td>
</tr>
<tr>
<td>GHJ</td>
<td>glenohumeral joint</td>
</tr>
<tr>
<td>GSA</td>
<td>glenoscapular angle</td>
</tr>
<tr>
<td>MR</td>
<td>magnetic resonance</td>
</tr>
<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
</tr>
<tr>
<td>PD</td>
<td>proton density</td>
</tr>
<tr>
<td>PER</td>
<td>passive external rotation</td>
</tr>
<tr>
<td>PHHA</td>
<td>percentage of humeral head anterior to the middle of the glenoid fossa</td>
</tr>
<tr>
<td>ROM</td>
<td>range of motion</td>
</tr>
<tr>
<td>SR0-SR2</td>
<td>size reduction 0-2</td>
</tr>
<tr>
<td>SE</td>
<td>spin echo</td>
</tr>
<tr>
<td>SEP</td>
<td>somatosensory evoked potential</td>
</tr>
<tr>
<td>STIR</td>
<td>short time to inversion recovery; short tau inversion recovery</td>
</tr>
<tr>
<td>T</td>
<td>tesla</td>
</tr>
<tr>
<td>TA</td>
<td>time of acquisition</td>
</tr>
<tr>
<td>TAM</td>
<td>total active motion</td>
</tr>
<tr>
<td>TE</td>
<td>time to echo</td>
</tr>
<tr>
<td>TM</td>
<td>teres major</td>
</tr>
<tr>
<td>TR</td>
<td>time of repetition</td>
</tr>
<tr>
<td>T1</td>
<td>longitudinal relaxation</td>
</tr>
<tr>
<td>T2</td>
<td>transverse relaxation</td>
</tr>
<tr>
<td>T2*</td>
<td>transverse relaxation obtained using gradient echo sequences</td>
</tr>
<tr>
<td>US</td>
<td>ultrasonography/ ultrasound</td>
</tr>
</tbody>
</table>
ABSTRACT

Brachial plexus birth injury (BPBI) is caused by traction of the head during delivery. The upper brachial plexus (C5-C6) is most commonly affected. Despite advances in obstetrics the incidence of BPBI varies from 1 to 4 per 1000 living births in western countries. Mild injuries, in which the nerve has only been stretched, will heal and patients will recovery completely, but in permanent palsy patients may require operative treatment. Diagnostic imaging is needed to determine the correct surgical treatment. Delayed diagnosis can lead to deterioration of the glenohumeral joint and poor patient outcome.

This study is focused on imaging findings in permanent brachial plexus birth injury. Such findings include ultrasound screening for posterior shoulder subluxation, and MRI evaluation of rotator cuff muscle changes in relation to glenohumeral joint pathology and muscle changes in BPBI with elbow flexion contracture. Additionally the outcome of surgical treatment of glenohumeral joint deformity was evaluated.

The results of the present population-based study show that in Helsinki during years 2003-2006, among 132 BPBI patients out of 41980 born neonates (3.1 per 1000), 27 (0.64 per 1000) of the BPBI cases did not heal during the first year of life and the palsy was considered permanent. One-third of these permanent BPBI patients developed posterior subluxation of the humerus during the first year of life. The rate of posterior subluxation was even higher among BPBI patients sent from the tertiary catchment area (I). All rotator cuff muscles, especially the subscapular muscle were atrophic in patients with internal rotation contracture (II). Every studied BPBI patient with elbow flexion contracture had fatty infiltration and size reduction of the supinator muscle, and pathological changes also occurred in the brachialis muscle (III). In all patients for whom the relocation operation was successful (10/13 of these undergoing the surgery) congruency of the glenohumeral joint improved, with mean glenoid version improvement of 33° (IV).

In conclusion, ultrasound (US) screening of the glenohumeral joint should be performed at 3 and 6 months of age in infants with persisting symptoms of BPBI, because the risk for shoulder instability is high during the first year of life (I). Imbalance of the shoulder muscles leads to progressive glenoid retroversion, subluxation of the humeral head and internal rotation contracture (II). Brachialis muscle pathology seems to be the main cause of elbow flexion contracture. The more severely affected the pronator teres muscle, the more restricted the prosupination movement (III).
Glenohumeral joint deformity in BPBI can be treated by shoulder relocation in young patients. Among patients younger than 5 years, successful relocation of the humeral head results in remodeling of the glenohumeral joint (IV).
INTRODUCTION

Brachial plexus birth palsy was described in 1779 by Smellie (Smellie 1779). Nearly one hundred years later Duchenne presented a case report of four neonates with upper plexus injuries (Duchenne 1872). Erb gave his name to typical upper root injury by localising the lesion at the junction of the C5 and C6 roots by electrical stimulation (Erb 1874). Klumpke, the first female medical intern in Paris, added to the medical knowledge of her day with a report of a lower plexus lesion with possible involvement of sympathetic fibres causing Horner’s syndrome (Klumpke 1885).

Maternal diabetes is the main etiologic risk factor for macrosomia, which predisposes a fetus to shoulder dystocia (Acker et al. 1985, Bradley et al. 1988). Despite the increased glucose balance during pregnancies of diabetic mothers, macrosomia has not decreased during the last 25 years (Teramo 1998). It has been shown that shoulder dystocia leads to brachial plexus birth injury (BPBI) in 26% of deliveries of babies with a birth weight over 4500g (Rouse et al. 1996). In addition to macrosomic children (+ 2 SD) in vertex presentation, small children in breech presentation are also in danger of BPBI. The severity of the injury varies from mild nerve stretching of the brachial plexus, to rupture of the nerves or to total avulsion of the nerve roots from the spinal cord. The majority of children with BPBI recover within the first year of life, but 25% develop permanent palsy (Andersen et al. 2006). Muscle imbalance as a result of nerve injury may lead to internal rotation contraction of the shoulder, and retroversion of the glenoid with subluxation of the humeral head. If elbow flexion has not recovered at all within 3-9 months, a primary plexus reconstruction operation is performed (Clarke and Curtis 1995). In spite of possible primary operations, continued restriction of total active movement of the hand or secondary bony deformities may require additional surgical corrections (Leffert 1998).

In addition to clinical findings and electromyography (EMG), diagnostic imaging will help to evaluate the need for and type of treatment required. Magnetic resonance imaging (MRI) allows non-invasive visualisation of possible root avulsion without contrast agent, which is needed in myelography and computer tomography (CT) -myelography studies (Gasparotti et al. 1997). Plain radiographs demonstrate fractures and deformities of the bones. With ultrasonography (US), possible posterior subluxation of the humeral head can be detected without sedation of the patient (Saifuddin et al. 2002). MRI allows evaluation of the muscles, cartilage and unossified bony structures. In this study possible pathological shoulder and elbow joint changes in BPBI were analyzed using ultrasound or magnetic resonance imaging.
REVIEW OF THE LITERATURE

ETIOLOGY OF BRACHIAL PLEXUS BIRTH INJURY

Since 1779, when Smellie ascribed paralysis of the arm of neonate to delivery, the discussion about etiologic factors has been vigorous. Poliomyelitis, toxin agents, congenital syphilis and infective or ischemic factors have been suggested as reasons for brachial plexus palsy. Despite the colorful discussion in the past, the obstetric origin of brachial plexus injury among newborns has been widely accepted. Sever emphasized the role of traction of the brachial plexus during delivery as a cause of BPBI (Sever 1916). Diabetes, macrosomy, shoulder dystocia and operative vaginal delivery (forceps or vacuum extraction) have been accepted as important risk factors for BPBI. When birthweight increased from less than 3500g to more than 4500 g, the incidence of BPBI was 45 times higher in a Swedish study (Bager 1997). A previous child with obstetric brachial palsy and multiparity are regarded as additional risk factors (Gherman et al. 2003). Also, excessive maternal weight gain during pregnancy has been reported to predispose the mother to delivery problems and the infant to BPBI (Lewis et al. 1998). In a recent retrospective study based on the Swedish Medical Birth Registry, the rates of BPBI increased significantly during the period 1987-1997 (Mollberg et al. 2005). Thus maternal obesity in western countries seems to increase brachial plexus birth injuries. The incidence of BPBI increased from 0.1 % to 0.5 % when body mass index registered at first visit at the maternal center increased from under 19 to over 30 in the above mentioned study (ibid.). The majority of BPBI babies are delivered from vertex presentation (Wolf et al. 2000). Breech presentation along with high birth weight results in a higher risk of BPBI (Soni et al. 1985). McFarland et al. were the first to report BPBI after cesarean section (McFarland et al. 1986). Despite the known risk factors, shoulder dystocia remains partly unpredictable, besides after cesarean section (Backe et al. 2008). BPBI has also been reported after breech presentation of a child with 830g birth weight (McFarland et al. 1986).
EPIDEMIOLOGY OF BPBI

The reported incidence of BPBI varies over ten-fold in western countries (Adler and Patterson 1967, Hoeksma et al. 2000) (Table 1).

Table 1. Incidence of BPBI

<table>
<thead>
<tr>
<th>Author and year</th>
<th>Area</th>
<th>Incidence per 1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adler and Patterson 1967</td>
<td>New York/ USA</td>
<td>0.38</td>
</tr>
<tr>
<td>Hardy 1981</td>
<td>Auckland/ New Zealand</td>
<td>0.87</td>
</tr>
<tr>
<td>Levine et al. 1984</td>
<td>Ohio/ USA</td>
<td>2.6</td>
</tr>
<tr>
<td>Jackson et al. 1988</td>
<td>California/ USA</td>
<td>2.5</td>
</tr>
<tr>
<td>Sjöberg et al. 1988</td>
<td>Malmö/ Sweden</td>
<td>1.9</td>
</tr>
<tr>
<td>Alanen 1989</td>
<td>Turku/ Finland</td>
<td>1.8</td>
</tr>
<tr>
<td>Walle and Hartikainen-Sorri 1993</td>
<td>Oulu/ Finland</td>
<td>2</td>
</tr>
<tr>
<td>Bhat et al. 1995</td>
<td>Pondicherry/ India</td>
<td>1.0</td>
</tr>
<tr>
<td>Gilbert et al. 1999</td>
<td>California/ USA</td>
<td>1.5</td>
</tr>
<tr>
<td>Hoeksma et al. 2000</td>
<td>Amsterdam/ Holland</td>
<td>4.6</td>
</tr>
<tr>
<td>Donelly et al. 2002</td>
<td>Dublin/ Ireland</td>
<td>1.5</td>
</tr>
<tr>
<td>Evans-Jones et al. 2003</td>
<td>United Kingdom, Ireland</td>
<td>0.42</td>
</tr>
<tr>
<td>Dahlin et al. 2007</td>
<td>Malmö/ Sweden</td>
<td>3.8</td>
</tr>
<tr>
<td>Backe et al. 2008</td>
<td>Trondheim/ Norway</td>
<td>3</td>
</tr>
</tbody>
</table>

The number of permanent cases among BPBI has been estimated to vary from 14% to 20% (Rust 2000, Noetzel et al. 2001), while in the population-based study 25% of children with BPBI in Sweden had persistent functional and cosmetic abnormalities of the upper limb (Sjöberg et al. 1988). Andersen et al. also concluded that in 25% of cases of BPBI, symptoms remain permanent (Andersen et al. 2006). Although risk for shoulder subluxation associated with BPBI was reported at the beginning of the last century (Whitman 1905, Fairbank 1913, Sever 1925), few subsequent reports have been published. Babitt and Cassidy considered the shoulder instability to be rare (Babitt and Cassidy 1968). Polloc and Reed reported 4 posterior subluxations out of 11 patients with BPBI (Polloc and Reed 1989). 62% (26/42) of patients imaged with either CT or MRI in order to evaluate associations between functional limitations, muscular deformity and persistent palsy had posterior subluxation in a study performed by Waters et al. (Waters et al. 1998). In the population-based study from Malmö, Sweden, the incidence of posterior dislocation of the shoulder has been calculated as being 0.28 per 1000, (7.3%, 6/82) of all BPBI patients during a 6-year study period (Dahlin et al. 2007). In the North American study Moukoko et al. reported posterior shoulder instability in eleven (8%) out of 134 neonates with BPBI (Moukoko et al. 2004).
ANATOMY OF THE BRACHIAL PLEXUS

The brachial plexus is a complicated cable network which is formed by the ventral rami of the lowest four cervical and first thoracic spinal nerve roots (C5-T1) (Appendix, Fig.1). These roots fuse to three trunks: upper (C5-C6), middle (C7) and lower (C8-T1). Each trunk divides into two parts, resulting altogether in six divisions: anterior and posterior, each having upper, middle and lower trunks. These six divisions further form three cords: posterior, lateral and medial. Anterior divisions of the upper and middle trunks (C5-C7) unite to form the lateral cord. Posterior divisions of all three trunks (C5-T1) join to form the posterior cord, while the anterior division of the lower trunk (C8-T1) continues as the medial cord. The cords are then divided into 5 nerves: the ulnar nerve originates from the medial cord, the median nerve from the medial and lateral cords, the musculocutaneous nerve from the lateral cord, and the axillary and radial nerves from the posterior cord. Through this network creation every nerve gets fibers from several spinal roots, which minimizes the risk of possible damage for example in the case of root avulsion. According to the microscopic study, the adult brachial plexus consists of an average of 118 047 myelinated nerve fibers (range 85 566-166 214) (Bonnel 1984). Roughly, C5 and C6 innervate the muscles of the shoulder region, arm and elbow, and C7 muscles of the forearm and partly of the wrist. C8 and T1, in turn, innervate muscles of the hand and fingers. The brachial plexus is located under the sternocleidomastoid muscle, between the anterior and medial scalene muscles (Appendix, Fig.2). The clavicle protects the brachial plexus at the level of plexus divisions (Kawai 2000).

PATHOMECHANICS OF BPBI

Fetomaternal disproportion may lead to lateral traction of the head away from the shoulder causing nerve injury during labor. This is confirmed in the study of Walle and Hartikainen-Sorri, who found that two-thirds of shoulder injuries involved the anterior shoulder after strong traction of the head in order to liberate the shoulder behind the symphysis in delivery. The posterior shoulder, which was affected in one-third of cases, was most probably compressed against the sacral promontory of the mother (Walle and Hartikainen-Sorri 1993). In breech presentation, if the body is pulled out strongly during delivery while the arm has been slipped up over the bent head, the pulling forces over-stretch or even tear the nerves. C8-T1 roots are especially vulnerable to avulsions, because there is poor connective tissue support around these nerves. On the other hand C5 and C6 roots seldom avulse, because they are fixed with ligaments to the osseus margins of the foraminas.
Bonnard and Anastakis 2001). Nonetheless, when rare upper root avulsions appear, they have been strongly associated with breech presentation (Geutjens et al. 1996).

Depending on the degree of damage, the conductivity of the nerves may be diminished or completely destroyed. At the neural level the injury may lead to demyelination, axonal degeneration or avulsion of the nerve root from the spinal cord. The clinical consequences of the nerve injury include disruption of the motor and possibly also sensory function. Seddon classified nerve injury into three types: neurapraxia lesions, axonotmesis and neurotmesis (Seddon 1942). The mildest type of injury is neuropraxia (the myelin sheath is damaged around the intact axon), where stretching of the nerve resolves completely (remyelination) within the first months of life. In the more severe injury, called axonotmesis, Wallerian degeneration of the axon takes place distal to the site of injury. While the endoneural tube remains intact, the axon regenerates at a rate of 1 to 2 mm per day (Brushart 1999). This axonal regeneration usually results in recovery. In the most severe type of neural injury, neurotmesis, the axon as well as the surrounding connective tissue are damaged. In the case of total nerve rupture or avulsion spontaneous recovery does not occur (Hentz and James 1999).

Already at the beginning of the last century, Whitman classified shoulder dislocation in young children as belonging to three categories: 1. True congenital displacement of the humeral head, which he regarded as very rare; 2. traumatic dislocations occurring during delivery; and 3. acquired subluxation resulting from injury to the brachial plexus (Whitman 1905). Intrauterine or congenital dislocation of the humeral head is very rare (Heilbonner 1990). Zancolli and Zancolli regarded humeral epiphyseolysis to be due to obstetric trauma, and is often present in the case of internal rotation contracture of the shoulder, joint deformity and posterior subluxation of the humeral head (Zancolli and Zancolli 1993). They also presumed that during labor a direct muscle lesion occurs at the same time as the plexus injury, which is followed by fibrosis of the muscles and scar contractions (Zancolli and Zancolli 2000). The above theory is not widely accepted. Only a few studies support the theory that the humeral head dislocates during delivery at the same time as brachial plexus injury occurs (Thomas 1914) and is maintained by the muscle imbalance (Dunkerton 1989, Troum et al. 1993). Most authors think that the posterior subluxation of the humeral head develops gradually as a result of the muscle imbalance (Fairbank 1913, Gilbert 1993, Waters et al. 1998). In most BPBI patients, the strength of the internal rotators dominate, leading to an internally rotated position of the arm and to a reduced range of motion (Gilbert 1993). Bone and cartilage remodel according to the mechanical relation to the muscle action and
joint reaction forces (Johnstone and Foster 2001). During prolonged internal rotation the pressure of the humeral head is directed to the posterior corner of the glenoid, resulting in a more posterior position of the humeral head and increased retroversion of the glenoid (Pearl et al. 2003).

Pathomechanics in the elbow is analogous to that in the shoulder region: muscle imbalance leads to a decreased range of motion, which results in soft tissue contractures, bony deformities and possibly dislocations (Waters 2005). The initial muscle imbalance leading to pronation of the forearm may change in the long-term depending on the recovery process. Normal prosupination movement requires functioning of the biceps brachii, brachioradialis, pronator teres, pronator quadratus and supinator. Pronation or supination contracture may develop with or without interosseous membrane contraction and subluxation/dislocation of the radius or the ulna (Zancolli and Zancolli 2000). Aitken reported 27 (25.2%) posterior dislocations of the radial head with bowing of the ulna out of 107 patients with BPBI. An additional 6 (5.6%) patients had anterior dislocation of the humeral head in the same study, resulting in an incidence of 30.8% for bony deformity in the elbow region (Aitken 1952). The first signs of bony malformation and incipient posterior subluxation of the radial head were seen radiographically as early as two months of age in the above mentioned study (ibid).

Ballinger and Hoffer followed 121 patients with Erb’s C5-6 palsies for at least 2 years (average follow-up was over 6 years). Patients with elbow surgery, radial head dislocations, elbow subluxations or mixed palsy with affision of the lower roots were excluded from the series giving 38 patients with classic C5-6 palsy for follow-up. 34 of these 38 patients had elbow flexion contracture (Ballinger and Hoffer 1994). Elbow extension deficit has been reported in 90% of permanent BPBI patients in Sweden (Strömbeck et al. 2007). Patients with permanent BPBI have frequently limitations of forearm rotation. 86% patients with permanent BPBI symptoms had active pronation beneath normal values and 64% had active supination below normal values in the study of Sibinski et al. (Sibinski et al. 2007).

Delayed maturation of the bony structures has been reported in association with BPBI (Polloc and Reed 1989). Bae et al. measured size differences in the affected and uninvolved upper extremities (Bae et al. 2008). Their study demonstrated that the affected upper extremity was approximately 95% of the length and girth of the contralateral upper limb. The difference was statistically significant (p<0.01). The nerve injury and weakened power of the muscles may be reasons for the decreased growth potential (ibid.).
PROGNOSIS OF BPBI

According to the study of Gherman et al. the outcome of BPBI is difficult to predict on the basis of ante-or intrapartum characteristics (Gherman et al. 2003). Patients with permanent brachial plexus injury had a higher mean birth weight, but otherwise there were not significant differences in ante-or intrapartum characteristics between temporary (transient) and permanent brachial plexus injuries (ibid.). Hoeksma et al. concluded that a complete neurological recovery occurs in 66 % of patients, and the extent of recovery seemed not to be predictable on the basis of direct initial post partum symptoms (Hoeksma et al. 2004). In other studies the rate of full recovery in BPBI has been reported to be between 13%-80% (Wickström et al. 1955, Gordon et al. 1973, Lindell-Iwan et al. 1995, Evans-Jones et al. 2003). Bennet and Harrold reported permanent palsy in 25% of patients; the higher the number of affected roots, the worse the recovery rate (Bennet and Harrold 1976). In the case of full recovery, which occurred among three out of every four of their patients, the symptoms disappeared within the first five months. Also, patients with persistent symptoms showed partial recovery (ibid.).

In a population-based retrospective study with a mean follow-up time of 13.3 years, the extent of the brachial plexus birth injury was the most important prognostic factor for predicting the final outcome in 112 patients who had undergone brachial plexus surgery (Kirjavainen et al. 2007). ROM and strength of the affected upper limb was better preserved in patients with C5-C6 injury than in those with C5-T1 injury in a 12 year follow-up study (Kirjavainen et al. 2011).

If symptoms persist, any developing internal rotation contracture is commonly followed by glenoid deformity. Pearl and Edgerton recommend early imaging with a modality that will visualize the skeletally immature glenohumeral joint. According to their experience, the passive external rotation may become restricted as early as in the first six months of life (Pearl and Edgerton 1998). Hoeksma et al. detected a strong association (P=0.004) between shoulder contracture and osseus deformity in a retrospective study done in Amsterdam evaluating the outcomes for 52 children with BPBI at a mean age of 3.7 years, born between years 1991 and 1998 (Hoeksma et al. 2003). In this study, the prevalence of a shoulder contracture (>10º) was 56% (29/52) and osseus deformity 33% (16/48 patients with complete radiographic follow-up) (ibid.).
DIAGNOSIS OF BPBI

Clinical findings

It is important to have a thorough patient history. Type of delivery, presentation, possible use of forceps or suction cup, shoulder dystocia, birth weight and Apgar scores give valuable information needed to evaluate neonates. The strength of all muscles, possible contractures, range of motion and especially shoulder abduction and external rotation are assessed. Palpation gives information regarding possible clavicle or humeral fractures. Among older children who can play or follow instructions, range of motion is graded with the Mallet scale (Figure 1) in order to evaluate shoulder function. In this assessment, shoulder function is evaluated in 5 different movements: abduction, external rotation, hand to neck, hand to back and hand to mouth. Each movement is then graded for 1 to 5 points, where 1 indicates no function and 5 normal function (Mallet 1972). A goniometer is used for measurements of both active and passive ranges of motion of the forearm (American Academy of Orthopaedic Surgeons 1988). Sensibility may be assessed for each dermatomy using filament tests and stereognosis when needed.

Table 2. Sequels of BPBI

<table>
<thead>
<tr>
<th>Functional deficit* in</th>
<th>Main muscles affected</th>
<th>Extent of root injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>external rotation of the shoulder, abduction, elbow flexion, supination, (±) wrist extension</td>
<td>deltoid, supraspinous, infraspinous, biceps, brachial, coracobrachial, brachioradial, supinator, (±) radial wrist extensors</td>
<td>C5-C6</td>
</tr>
<tr>
<td>all above movements and extension of the elbow, wrist, and fingers</td>
<td>all above muscles and teres major, triceps, extensors/ flexors of wrist, extensors of the fingers</td>
<td>C5-C7</td>
</tr>
<tr>
<td>flexion of the fingers</td>
<td>flexor digitorum superficiale/ profundus, flexor pollicis longus, interossei and lumbrical muscles</td>
<td>C8, T1#</td>
</tr>
<tr>
<td>all above movements → flail hand</td>
<td>all the above muscles</td>
<td>C5-T1</td>
</tr>
</tbody>
</table>

* Degree of the functional deficit vary from mild to severe. # Horner’s syndrome may follow. (Leffert 1998). (±) may or may not be present.
The strength of the unaffected muscles dominates the clinical findings, resulting in the so-called “waiter’s tip” position, characteristic for upper plexus lesions in BPBI. This typical posture is due to the injury in the upper trunk (C5 and C6), which results in weakness of the abductors and external rotators of the shoulder, flexors and supinators of the elbow and extensors of the wrist (Table 2). At the same time unaffected internal rotators and adductors, elbow extensors and wrist flexors (Appendix, Figs. 3-6) remain powerful by innervation received from the middle trunk and C7. As a result of this muscle imbalance, the shoulder is adducted and internally rotated, elbow extended with forearm pronated and the wrist and fingers are flexed. If the upper plexus injury is extended to root C7, the involvement of the radial nerve may cause a sight flexion of the elbow. In the case of injury to the entire plexus, all the muscles may be affected, resulting in flail arm with no movement. An isolated lower plexus lesion with decreased grip power of the hand is very rare (Sever 1916). Horner syndrome (ptosis, miosis, enophtalmos, anhidrosis) may be associated with avulsion of T1. Clavicle fracture has to be taken into account as a differential diagnostic possibility in cases where there is a lack of shoulder movements of the newborn baby. Neonates with BPBI findings also have to be evaluated for possible associated injuries such as facial or phrenic nerve palsy.
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**Figure 1.** Mallet’s classification of function in brachial plexus birth injury (From Gilbert 1993, with permission).
Electromyography (EMG)

Terzis et al. has recommended electromyography (EMG) for assessing brachial plexus injury (Terzis et al. 1986). EMG measures the function of the motor unit, which includes the anterior horn cell in the spinal cord, the axon, neuromuscular synapse, and muscle connected with it. Function of peripheral sensory fibres can be assessed with EMG. EMG may be used to assess the extent of the injury, possible need for surgery and progression of recovery (Smith 1996). In the case of denervation, the nerve does not conduct electrical signals any more, and muscle cells develop fibrillation as a sign of denervation activity. Due to gradual Wallerian degeneration it takes 2 to 3 weeks before the fibrillation appears. This spontaneous muscle activity disappears when muscle fibres either degenerate or innervate. It may be challenging to insert EMG needles into neonates, because EMG diagnosis requires measurements of several muscles. Denervation appears and disappears earlier among neonates than adults (Vredeveld 2001) and can lead to underestimation of the severity of the nerve injury (Vredeveld et al. 2000). Recovery can be observed earlier among neonates using EMG, because distances are shorter although the axons grow at the same speed as in adults (Vredeveld 2001).

Somatosensory evoked potential (SEP)

With somatosensory evoked potentials, the electrical stimulation of the peripheral nerve is measured from the cortex. If there is a lesion in the somatosensory pathway, the stimulation does not reach the cortex. Using SEP the proximal root damage can beneficially be detected and the conductivity of nerve stumps assessed prior to grafting (Landi et al. 1980). Intraoperatively the functional continuity between proximal stump of the ruptured root and cortex can be assessed (ibid.). Jones reported that surgical findings were in good agreement with those seen on SEP (Jones 1979).

Radiography

During the neonatal period, radiographs are obtained to identify fractures and dislocations. Discontinuity of the cortex can be seen in cases of nondislocated clavicle fractures. The hump-like elevation of the bone contour is evident in 8 or 9 days after fracture as a sign of callus formation (Silverman and Kuhn 1993). Fractures of the clavicle or humerus usually heal without complications, but they may sometimes hide the symptoms of BPBI.
The position of the unossified humeral head and possible posterior subluxation are difficult to depict on plain radiographs in neonates. Odgen et al. studied the radiologic development of the humerus among 23 children, from newborn to 14 years old. A proximal humeral ossification center was radiographically invisible in all three of the studied full-term stillborn babies, but it had appeared by 3 months of age among the other three studied children (Odgen et al. 1978). Goddard states that the humeral ossification center is visible on plain radiographs in approximately 20% of newborns during the first week of life, and the ossification center of the greater tuberosity appears between 6 to 8 months of age (Goddard 1993). The main ossification center is always located medially with respect to the line going along the long axis of the humerus. True epiphyseolysis of the humeral head may be challenging to diagnose among young children because capital epiphysis may imitate epiphyseolysis due to internal rotation associated with BPBI (ibid.). Posterior dislocation of the ossified humeral head can be verified with axial radiographs (Figure 2), which will show the humeral head lying posterior to the glenoid (Dunkerton 1989). BPBI-associated bony anomalies including hypoplastic humeral head, an inferiorly directed coracoid and tapered acromion can additionally be visualized with plain radiographs (Sever 1925). BPBI patients may present delayed ossification of the epiphyseal centers on the affected side (Pollock and Reed 1989). The findings for acquired glenoid deformity resulting from BPBI may be similar to those for congenital glenoid deformity seen, for example, in Apert syndrome, Hurler syndrome, mucopolysaccharidosis VI, oculo-mandibulo-melic dysplasia, Pierre Robin syndrome, and TAR syndrome (Lachman 2007). The glenoid deformity seen in BPBI is usually unilateral, while underdevelopment of the glenoid is bilateral in most of the above-mentioned syndromes (Currarino et al. 1998). BPBI may result in up to 6-8 cm growth retardation of the arm (Narakas 1987). McDaid et al. used plain radiographs to evaluate the length discrepancy of the affected upper limb compared to the unaffected side in 22 children with BPBI. The length of the affected upper limb was on average 92% of that of the upper limb on the healthy uninvolved side. The retardation in size is most probably due to the lack of biomechanical function and stress that is needed for optimal development (McDaid et al. 2002).

If there is concern about possible diaphragmatic paralysis, conventional chest films may provide useful information. This information is especially important before anesthesia for surgical procedures and also medico-legally.

Intraoperative fluoroscopy or plain radiographs are used to verify the position of the bones as well as fixation material needed in humeral rotation osteotomy (Bae and Waters 2007).
Figure 2. Radiograph demonstrates posterior dislocation of the humeral head on the left side in AP (B) and axial (D) views compared to the right healthy side (A,C). Patient has C5-7 injury, no previous surgical procedures performed.
Conventional arthrography

Conventional arthrography has been used in evaluation of birth injuries of the shoulder (White et al. 1987). Pearl and Edgerton performed intraoperative arthrograms to assess BPBI-associated pathological changes of the glenohumeral joint and they classified the shape of the glenoid as being normal, flat, biconcave or pseudoglenoid (Pearl and Edgerton 1998). The glenoid surface was regarded as pseudoglenoid when the posterior aspect of the glenoid was clearly retroverted both in relation to the unoccupied anterior concavity and to the plane of the scapula (ibid.). A few years later they compared MRI, intraoperative arthrography and arthroscopic findings with each other and concluded that if MRI is not available, arthrography at the time of the surgery is informative (Pearl et al. 2003). Kon et al. correlated intraoperative arthrography findings with the degree of passive external rotation among 64 children with internal rotation contracture secondary to BPBI. They concluded that intraoperative arthrography correlated with the degree of internal rotation contracture (Kon et al. 2004). Nowadays invasive arthrography has been replaced with preoperative MRI.

Ultrasonography (US)

Musculoskeletal ultrasound is a widely used, non-invasive and inexpensive imaging modality, which can be used without sedation for neonates and infants (Keller 2005). Ultrasonography (US) has proved to be useful in detecting glenohumeral effusion in septic arthritis (ibid.), slipped humeral epiphyses (Zieger et al. 1987, Broker and Burbach 1990), posterior displacement of the humeral head in BPBI (Hunter et al. 1998, Saifuddin et al. 2002) and rotator cuff pathology (Daenen et al. 2007). US is therefore a good method in making differential diagnosis and assessment of shoulder joint pathology in patients with BPBI. The accuracy of US in clavicular fractures has been assessed by Graif et al. 1988, and Grissom and Harcke 2001. In the case of new clavicle fractures, soft tissue swelling and discontinuity of the cortex may be seen. After a couple of weeks a healing fracture with callus is seen as a hump-like elevation of the bone contour, while the normal clavicle has a mildly s-shaped structure on US (Katz et al. 1988).

US of the plexus area

A few studies have reported the use of US in assessment of the brachial plexus. Nerve bundles are seen as hypoechoic structures (Sheppard and Lyer 1998, Shafighi et al. 2003). Haber et al. demonstrated the use of US in detection of brachial plexus traction injuries among adults (Haber et al. 2006). US succeeded in visualizing scar tissue seen as a hyperechoic area, but could not distinguish the nerve from the scar and failed to follow the continuity of the nerve. Furthermore,
US was unsuccessful in the detection of the neuroma, later found during operation. Visualization of C5-C7 levels was possible using US, but not nerve roots C8 and T1, which are located too deep and caudally. The attachments of the nerve roots to the spinal cord were also hidden behind bone shadows (ibid.). With these limitations, US is a challenging tool even in experienced hands for detection of brachial plexus nerve lesions among neonates, who have thinner nerve structures than adults.

US of the shoulder girdle

US enables dynamic investigation of shoulder instability (Bianchi et al. 1994, Schydlowsky et al. 1998). US is therefore a suitable method for differential diagnosis and assessment of shoulder joint pathology in patients with BPBI. Although US is a widely-used method for assessing hip dysplasia and instability screening (Harcke and Grissom 1990, Holen et al. 1994, Holen et al. 1999), it is infrequently used as a screening method for BPBI-associated posterior subluxation of the humeral head. Hunter et al. presented the use of a posterior approach with US to detect posterior subluxation of the humeral head (Hunter et al. 1998). Moukoko et al. tried to verify posterior shoulder dislocation with a lateral approach, and noticed that the growing ossification center obscures visibility of the glenoid area (Moukoko et al. 2004). In their study, posterior shoulder instability was detected visually with US at a mean age of 6 months. In the case of posterior displacement, the center of the humeral head was located posterior to the axis of the scapula (Moukoko et al. 2004). Vathana et al. described measurement of the α-angle to assess the posterior subluxation of the humeral head. The α-angle is measured between the line along the posterior margin of the scapulae and the line tangent to the humeral head. Normally the α-angle is 30º or less, while posterior subluxation increases the angle value (Vathana et al. 2007).

Conventional myelography and computed tomography myelography

Conventional myelograms with intrathecal contrast medium were previously used to verify root avulsions (Murphey et al. 1947). In the case of root avulsion, a dural covered diverticulum containing cerebrospinal fluid may be formed (Figure 3), even extending through the intervertebral foramen and this can be seen on a myelogram (Petras et al. 1985). However, nerve root avulsions have also been reported without dural abnormalities (Davies et al. 1966). Nagano et al. have even reported normal roots despite a meningocele configuration on myelogram, most probably due to dural rupture without discontinuity of the nerve rootlets (Nagano et al. 1989). That is why visualization of the nerve root itself is necessary when interpreting scans. Since Sir Godfrey
Hounsfield invented CT in the early 1970s, CT has little by little become a diagnostic tool for nerve root injuries (Marshall and De Silva 1986, Cobby et al. 1988). However, even in the beginning of the 1990s Hashimoto et al. concluded in their study that myelography is necessary for preoperative evaluation of cervical nerve root avulsion associated with birth palsy, because using CT myelography it was difficult to verify nerve root avulsions with no associated traumatic meningocele (Hashimoto et al. 1991). The possible reason for these difficulties with CT diagnostics was that their CT-study was performed using 5 mm thick sections and only axial images were available. Since that time the CT-technique has developed considerably (Volle et al. 1992). CT myelography images were obtained by performing scanning 90 to 120 minutes after intrathecal injection of a water-soluble contrast agent into the lumbar spinal canal (Carvalho et al. 1997). Walker et al. obtained 95% sensitivity and 98% specificity for complete nerve root avulsions with CT myelography using 1 mm contiguous axial images in infants and 3 mm slice thickness in adults (Walker et al. 1996). Nowadays, conventional myelography has been replaced by cross-sectional imaging techniques. Three-dimensional rotational CT-myelography has been successfully used especially among adults following cervical puncture at C1/C2 level (Kufeld et al. 2003). The possible risk related to allergic reactions, radiation, sedation and the time needed to complete the imaging procedure are limitations for CT myelography in small children (Birchansky and Altman 2000).

Figure 3. Radiographs demonstrate meningocele at the level of C7 root on the left side in myelogram obtained with intrathecal contrast medium.
**Computed tomography (CT)**

Computed tomography has been used to evaluate the brachial plexus from its origin to the axillary area (Fishman et al. 1986, Armington et al. 1987, Cooke et al. 1988). CT does not reveal detailed information of the thin nerves of the plexus area, although it can delineate the surrounding landmarks: anterior and middle scalene muscles, subclavian and axillary arteries and veins, clavicle and neural foramina.

The bony structures of the shoulder girdle and possible dislocation of the humeral head can be visualized easily with CT (Hernandez and Dias 1988, Beischer et al. 1999). Friedman et al. first described of technique to measure the glenoid version of the shoulders in the axial plane (Friedman et al. 1992). Mintzer et al. demonstrated that the glenoid is normally most retroverted in the first two years of life and after that the retroversion diminishes gradually, reaching adult glenoid orientation (-1.7º±6.4º) during the 10 first years of life (Mintzer et al. 1996). Waters et al. assessed the amount of posterior subluxation using CT or MRI by measuring the percent of the humeral head situated anterior to the line drawn through the medial tip of the scapula and the midpoint of the glenoid (Waters et al. 1998). Terzis et al. were the first to demonstrate on CT that glenohumeral joint congruency can be restored by palliative surgery for shoulder deformities in BPBI. Their study included pre-and postoperative CT scans of 28 patients treated in their clinic over 18 years (Terzis et al. 2003). Due to radiation exposure, CT has nowadays often been replaced with MRI, which additionally allows visualization of the cartilage and soft tissues.

**Magnetic resonance imaging (MRI)**

After Sir Peter Mansfield and Paul C. Lauterbur developed magnetic resonance for medical imaging in the 1970s, MRI has added imaging capabilities with multiplanar images and good tissue contrast (Mansfield and Maudsley 1977, Lauterbur 1980). With a strong magnet, using radiofrequency transmit and receiver coil system images are obtained without ionizing radiation. The potential of MRI in the musculoskeletal system imaging aroused great interest already in the beginning of the 1980s (Pettersson et al. 1985).

Magnetic resonance imaging is a non-invasive imaging modality for both traumatic and nontraumatic brachial plexopathies (Miller et al. 1993, Sureka et al. 2009) and gives good soft tissue contrast (Flanders et al. 1990). T1-weighted images with a short echo time (TE) and short repetition time (TR) allow good resolution of anatomical details due to a relatively high signal-to-
noise ratio. T2-weighted images (long TE, long TR) demonstrate water content in various tissues, while the signal-to-noise ratio is usually lower than in T1-weighted images. Fat-suppression techniques are used to reduce or null the signal from fat, which is often utilized in the detection of increased fluid concentration in tissues. T1-weighted fat-suppression techniques are utilized in contrast enhanced studies (Tamttu and Sepponen 1996).

**Imaging of the nerves**

If a lesion of the central nervous system is suspected, evaluation with MRI is warranted. MRI has proven superior in the detection of small extra-axial fluid collections, and white matter and brainstem injuries associated with birth trauma (Barkovich 2005). MRI can visualize intradural lesions, for example intra-medullary odema and haemorrhage, without radiation and contrast agents (Flanders et al. 1990). Cerebrospinal fluid (CSF) acts as a natural contrast agent around affected nerve roots (Rapoport et al. 1988 Popovich et al. 1989, Posniak et al. 1993, Yilmaz et al. 1999). Nerve roots are clearly visualized in the axial plane as they exit the foramina (de Verdier et al. 1993). Doi et al. reported in their study 92.9% sensitivity and 81.3% specificity for detecting root avulsions with MRI among adults (Doi et al. 2002). However, some studies have shown a low accuracy for MRI. The study by Ochi et al. in adults reported accuracies of 73% for C5 and 64% for C6 root avulsions using MRI (Ochi et al. 1994). Medina et al. reported only 50% sensitivity but 100% specificity for nerve root avulsions in cases of pseudomeningocele among children less than 18 months of age. In the above mentioned study they used FSE T2- and FSE T1-weighted sequences with slice thicknesses of 1.5 to 4 mm. (Medina et al. 2006). Due to the small dimensions of newborns, imaging of the nerves is very challenging. MR images are sensitive to motion artefacts, which may interfere with the interpretation of the images (Carvalho et al. 1997). Sedation or general anesthesia is needed to guarantee that the little child does not move during the examination procedure. Imaging protocols vary depending upon the facilities in different institutions. Recently, the technology has developed considerably. 3 D MR myelography has been recommended as an imaging modality of first choice, because it showed in traumatic brachial plexus injuries 92% diagnostic accuracy, 89% sensitivity and 95% specificity (Gasparotti et al. 1997). CT myelography can then be reserved as an imaging possibility in cases where there are discrepancies between clinical, electromyographic and 3D MR myelographic findings (ibid.). With 3D heavily T2-weighted MR myelography sequences (CISS 3 D, True FISP 3D, BFFE and FIESTA) root avulsions and nerve retraction balls are better assessed (Vargas et al. 2010). Modern
technology can allow 0.5 mm slice thickness, which allows good visualization of the preganglionic roots and possible avulsions (Figure 4).

![Coronal BFFE-image with 0.5 mm slice thickness showing an avulsion of the right C8-root with a meningocele of a 4-month old girl with BPBI on the right side.](image)

**Figure 4.** Coronal BFFE-image with 0.5 mm slice thickness showing an avulsion of the right C8-root with a meningocele of a 4-month old girl with BPBI on the right side.

MR neurography has been used to evaluate peripheral nerves of the brachial plexus (van Es 2001, Filler et al. 2004, Smith et al. 2008). The brachial plexus is difficult to visualize because it runs obliquely to all of the three standard orthogonal planes. Trunks are best visualized in an oblique coronal plane and the cords in either an oblique coronal or sagittal plane (Panasci et al. 1995). Blair et al. described in 1987 the anatomic details of the brachial plexus assessed with MRI for the first time. The brachial plexus was seen best in the sagittal plane, and signal intensity was similar to that of the muscle: low in both T1- and T2- weighted images (Blair 1987). The neurovascular bundle is surrounded by fat, which gives a good contrast to the nerves (Sherrier and Sostman 1993). Gupta et al. reported that operative findings confirmed focal fibrosis, neuromas and scarring in complete MR evaluation of the brachial plexus (Gupta et al. 1989). The sensitivity and specificity of MRI have been reported to be 97% and 100% respectively in BPBI-associated post-traumatic neuroma.
Neuroma can be seen as a high signal intensity mass on T2-weighted and STIR images (Medina et al. 2006).

**Imaging of the muscles**

MRI is the only imaging method that shows the early phase of muscle denervation before fatty infiltration develops (Shabas et al. 1987). MRI shows muscle edema as a high signal intensity on T2-weighted images highlighted by the fat-suppression technique. The short tau inversion recovery (STIR) sequence is ideal for detecting skeletal muscle edema because increased edema is additive with signal intensity (Fleckenstein et al. 1991). MRI can detect the signal intensity changes in denervated muscles earlier than EMG, i.e. already 4 days after injury (West et al. 1994). MRI of the muscle has been suggested to be particularly useful among children who may be difficult to assess by clinical and electrodiagnostic examination (ibid). In chronically denervated muscles, fatty infiltration is seen on T1-weighted images as high signal intensity in addition to muscle atrophy (Fleckenstein et al. 1993). Hence MRI is a suitable method for grading muscle pathology, as shown in the study of Mahjneh et al. (Mahjneh et al. 2004). Nakagaki et al. used the largest width of the supraspinatus muscle belly and the length of the muscle measured from coronal oblique MRI images to estimate size and atrophy of the muscle atrophy after rotator cuff tear (Nakagaki et al. 1995). Thomazeau et al. measured supraspinatus belly atrophy by calculating the ratio between the cross-section of the supraspinatus muscle and the largest bony limits for supraspinatus fossa in a Y-shaped position (Thomazeau et al. 1996). A Y-shaped view in the oblique-sagittal plane is obtained at the most lateral point where the scapular spine is in contact with the rest of the scapula. Zanetti et al. extended the quantification of the muscles to the cross-sectional areas of the subscapular and infraspinous/teres minor muscles in the same Y position (Zanetti et al. 1998). Rotator cuff muscle volumes were assessed using shoulder MRI in the study performed by Lehtinen et al. (Lehtinen et al. 2003).

Bredella et al. correlated MRI findings with electrophysiologic findings and concluded that MRI can supplement information obtained from EMG by allowing the age (acute/chronic) of the muscle lesion to be approximated, possibly showing the morphologic cause of the lesion (Bredella et al. 1999). Nerve entrapment syndromes, such as posterior interosseus nerve and radial tunnel syndromes, have to be taken into account as different diagnostic possibilities in the evaluation of muscle edema or atrophy of the supinator and extensor muscles (Rosengren et al. 1997, Bordalo-Rodriques and Rosenberg 2004). The posterior interosseus nerve arises from the radial nerve and penetrates the supinator muscle. The nerve is most commonly compressed at the arcade of Frohse,
at the level of the cranial part of the supinator muscle resulting in muscle weakness (Bayramoglu 2004) and muscle denervation patterns in MRI (Andreisek et al. 2006). The compression of the median nerve in pronator syndrome and the ulnar nerve in cubital tunnel syndrome as well as anterior interosseous nerve entrapment may lead to high signal intensity changes in T2-weighted images and further to atrophy and intramuscular fatty degeneration (bright on T1 and T2) in the affected muscles (ibid.).

Imaging of the bony structures
MRI allows the evaluation of glenoid retroversion, possible subluxation/dislocation of the humeral head and cartilaginous structures in the younger child (van der Sluijs et al. 2001). Gudinchet et al. reported MRI findings of five children with BPBI-associated internal rotation contracture: a blunt posterior glenoid surface was visualized in all patients (Gudinchet et al. 1995). Gradient echo sequence allowed a good visualization of posterior thinning of the hyaline cartilage and fibrocartilage of the labrum both anteriorly and posteriorly (ibid.). Waters et al. evaluated in a prospective study the severity of the glenohumeral deformity associated with BPBI (Waters et al. 1998). They performed either MRI or computed tomography for 42 BPBI patients. The glenoscapular angle was measured according the technique described by Friedman et al. (Friedman et al. 1992). The degree of subluxation was graded by measuring the percentage of the humeral head located anterior to the line going through the medial tip of the scapula and middle of the glenoid fossa (Waters et al. 1998). They found progressive glenoscapular retroversion and posterior subluxation of the humeral head with increasing age (ibid.). Kozin stated that failure to maintain passive external rotation above the neutral position should be regarded as a probable sign of underlying joint deformity (Kozin 2004). MRI gives better evaluation of the glenoid cartilage and labrum and may be more sensitive in the early detection of glenoid deformity than arthrography (Pearl et al. 2003). Clarification of the shape of the posterior glenoid is essential for the planning of surgical interventions and for subsequent evaluation of postoperative outcome (Kon et al. 2004).

Magnetic resonance (MR) arthrography
MR arthrography with intra-articular contrast agent gives information especially about rotator cuff lesions (de Jesus et al. 2009), ligament, hyaline cartilage and labral structures (Chiavaras et al. 2010). The technique is invasive and is seldom needed in children with brachial plexus birth injury.
TREATMENT OF BPBI

Conservative treatment

Physical and occupational therapy is important in the treatment of brachial plexus birth injury. Exercises to maintain the passive range of motion of the affected upper limb are taught already to the parents of a newborn with BPBI. Active and passive range of motion exercises are useful in preventing development of contractures. Splints and orthoses are used in cases of contractures in order to prevent further deformity. Sensory training is recommended if sensory deficits exist. Children are encouraged to use the weak extremity in play, age appropriate hobbies and activities (Ramos and Zell 2000).

Brachial plexus reconstruction

In 1903 Kennedy first reported primary plexus reconstruction, done by resection of the proximal plexus neuroma and making primary suture repair (Kennedy 1903). Tassin regarded plexus surgery to be necessary if the biceps function had not appeared by three months of age (Tassin 1983). In Finland, Solonen et al. and Alanen et al. reported a few decades ago successful early primary reconstructions of the partially torn brachial plexus by either nerve grafting or direct neuroraphy (Solonen et al. 1981, Alanen et al. 1990).

Early primary exploration and microsurgical repair may improve long-term outcome. Gilbert and Tassin recommend make the decision about possible early brachial plexus surgery by evaluating biceps muscle function. They recognized that if the deltoid and biceps have not started functioning by at the age of 3 months, the outcome is poor without plexus reconstruction (Gilbert and Tassin 1984). Thus Gilbert et al. recommend plexus surgery if biceps function is completely absent at three months of age in C5-C6 palsies. They extended the criteria for operation to complete palsies with a flail arm and Horner syndrome after one month of age. After breech delivery, complete C5-C6 palsy with flail hand and a negative EMG without any signs of regeneration, are regarded as reason for surgery as well (Gilbert et al. 1988). Haerle and Gilbert got encouraging results in 75% of patients who underwent early exploration and repair of lower roots. They recommended early repair of the avulsed lower roots with nerve transfers in cases of extensive paralysis of the hand at 3 months of age with Horner’s syndrome (Haerle and Gilbert 2004). Some authors would prefer a longer follow-up time to select candidates for plexus surgery (Clarke and Curtis 1995). According to the thesis of Tassin, some recovery may occur spontaneously, but the surgical results are better.
This was confirmed by Waters, who also followed outcome of patients who had some recovery between the 4th and 6th months. Those patients who had plexus reconstruction done at six months of age because of absent biceps function had a better outcome than those who had some spontaneous recovery at the age of 5 months (Waters 1999). When biceps function is lacking at the age of three months, some recovery may occur spontaneously, but it is less satisfactory than that achieved with surgery, as found after a minimum two year follow-up (ibid.). When no recovery of the muscles occurs, possible cervical avulsions should be verified before plexus surgery.

Surgical planning demands appropriate differentiation between preganglionic lesions of the nerve rootlets and postganglionic injury. Preoperative myelography, EMG, the use of intraoperative SEP and advanced microsurgical techniques facilitate early surgical treatment (Alanen et al. 1990). Repair of upper roots is performed through a supraclavicular approach, while a transclavicular approach is needed to repair a complete lesion (Gilbert 1993). A nerve stimulator is needed during the operation. Depending on the type of injury, excision of the neuroma, end to end suture, grafting or neurotization are performed.

Surgical treatment of a preganglionic lesion usually consists of neurotization. In neurotization, an intact donor nerve is released from its end organ and attached to the distal part of the damaged nerve. For example, intercostal nerves, the hypoglossal nerve or the contralateral C7 nerve are possible candidates for nerve transfers in neurotization reconstruction (Bonnard and Anastakis 2001). When the phrenic nerve is used as an axon donor for neurotization of the musculocutaneous nerve, there is a danger of decreased diaphragm function and vital capacity (Luedemann et al. 2002). Recently Hsu et al. reported a new technique for repairing cervical root avulsions with a sural nerve graft (Hsu et al. 2004). They operated on eight patients, including one newborn baby with brachial plexus birth injury. In the first part of the two stage surgery, they implanted the sural nerve graft into the spinal cord using a posterior approach. The dura mater was incised longitudinally and a tiny hole was made to the pia mater, thereafter the graft was fixed with fibrin glue. The distal end of the nerve graft was protected with a Foley tube with a radiopaque clip and placed in the supraclavicular region. After one week, the distal anastomosis was made using an anterior approach. After a mean follow-up period of 8.87 months, they reported improvement in electrophysiological function and muscle power grading postoperatively. Due to laminectomy, this technique includes the risk of possible development of cervical spine deformity (Hsu et al. 2004).
In the case of a postganglionic lesion, the surgical planning is based on the possible continuity of the nerve or distance between the damaged nerve ends. Neurolysis and release of adhesions may be needed. The neuroma is excised if it decreases the nerve conductivity more than 50%. The sural nerve is the most commonly used graft. Boome and Kaye reported good recovery with effectively normal deltoid strength in 80% of the patients operated on using sural grafts (Boome and Kaye 1988). Nowadays, nerve grafts are glued instead of conventional suturing which minimizes damage to the nerve ends. Either end-to-end or end-to-side anastomoses are made with fibrin glue (Grossman 2000, Grossman et al. 2003).

**Botulinum toxin treatment**

Physiotherapy should be used to prevent or minimize the development of muscle and joint contractures. In addition to physiotherapy, botulinum toxin treatment may be used to diminish the dominating power of unaffected muscles while waiting for nerve regeneration (Rollnik et al. 2000). Especially the treatment of subscapular (Alfonso et al. 1998) and pectoralis major (Jellicoe and Parsons 2008, Price et al. 2007) muscles is performed with botulinum toxin. Botulinum toxin is the neurotoxin obtained from the anaerobic bacterium Clostridium botulinum in laboratory conditions. Botulinum toxin prevents nerve endings from releasing acetylcholine, which is needed as a neurotransmitter to reach the endplates of the muscle in order to cause muscle contraction. Botulinum toxin is injected into internal rotators to allow the weak antagonist muscles to strengthen. EMG guidance can be used but when palpation is used to identify muscles, injection into either the mid-belly or several sites of the muscle is recommended (Childers and Markert 2007). In the study performed by Hogendoorn et al., glenohumeral deformities were significantly greater with a C5-C6 (C7) lesion than with a total palsy in which the internal rotators were affected as well (Hogendoorn et al. 2010). The balance between agonist and antagonist muscles is needed to prevent the development of joint contractures and bony deformities. Early treatment with botulinum toxin may prevent retroversion of the glenoid and development of bony deformities by balancing the effect of the dominating muscle on immature bones (Ramachandran and Eastwood 2006). After botulinum boxin injection immobilization of the shoulder in a spica cast in external rotation has been used in treatment of subluxation of the humeral head (Ezaki et al. 2010). The effect of botulinum toxin lasts approximately 3-4 months, allowing the function of the affected muscles to be restored.
Secondary operative treatment

In spite of possible primary reconstruction surgery, incomplete recovery or residual dysfunction may occur, leading to secondary deformities, which need additional operative treatment. The surgical operation is tailored to the individual based on clinical and radiological findings. The goal of the operation is to restore the function of the shoulder, most commonly to improve the limited abduction and external rotation. Due to typical internal rotation contracture of the humeral head, patients lack the ability to place the hand on the neck and they need to put the hand in the so-called “trumpet position” (the arm is abducted as in blowing a trumpet) when lifting the hand to the mouth. Prior to the operation the status of the glenohumeral joint has to been assessed in order to make a choice between different types of operative treatment.

Secondary correction operations were performed already at the beginning of the last century by Fairbank with the technique of open exploration and reduction of the glenohumeral joint after liberation of the subscapularis tendon (Fairbank 1913). Sever developed the technique, and advised that opening the joint capsul should be avoided (Sever 1916). This technique was further developed by L’Episcopo, who carried out muscle transplantation of the teres major and later on also the latissimus dorsi muscle from the role as of internal rotators to assist weak external rotation (L’Episcopo 1939). Wickström et al. reported excellent results using the above mentioned combined technique in 10 out of 16 patients (Wickström et al. 1955). In a modification by Phipps and Hoffer the pectoralis major is released, teres major and latissimus dorsi are transferred near to the insertion of the infraspinatus into the rotator cuff (Phipps and Hoffer 1995).

Tendon surgery

Shoulder

When there is internal rotation contracture with a congruent glenohumeral joint, a soft-tissue operation alone should improve external rotation (Jellicoe and Parsons 2008). Release of tight anterior structures, tendon lengthening and muscle transfers have been used to improve poor external rotation (Narakas 1993). As part of the corrections, z-lengthening of the tendon of the subscapular muscle has in particular been advocated (van der Sluijs et al. 2004, Bertelli 2009). It has also been proposed that tendon transfers might halt the development of glenohumeral deformity (Waters and Bae 2005).
If retroversion of the glenoid, humeral head flattening and possible dislocation are already present, tendon transfers or other soft-tissue operations are not sufficient alone (Waters and Peljovich 1999, Waters and Bae 2006). Kozin et al. assessed MRI findings in a one year follow-up study after tendon transfers and detected no improvements in glenoid version or humeral head subluxation (Kozin et al. 2006). To prevent deterioration of the glenohumeral joint, Gilbert et al. regarded early release important if external rotation is less than 20 to 30 degrees (Gilbert et al. 1988). They propose trapezius transfer to improve abduction and latissimus dorsi transfer to restore abduction and external rotation (ibid). Many studies prefer latissimus dorsi and teres major transposition to the infraspinatus position in order to improve weak external rotation (Waters and Bae 2005) with a possible concomitant release of subscapular muscle (Aydin et al. 2004, Nath and Paizi 2007).

In a long-term follow-up study Pagnotta et al. reported that after latissimus dorsi transfer active external rotation was better preserved than shoulder abduction. The function of the muscle transfers was better maintained in patients who took part in a physiotherapy program or engaged in physical activities, especially swimming (Pagnotta et al. 2004). Kirkos et al. published a report on a follow-up study, conducted a mean of 30 years (range 25 to 42) after anterior release, teres major and latissimus dorsi transfers of 10 patients with BPBI (Kirkos et al. 2005). They observed a deterioration in early successful results by the end of the long follow-up period. This may be due to degeneration of the glenohumeral joint, transferred muscles and surrounding soft tissues (ibid).

Elbow and forearm
To decrease limitations in forearm rotation, Zancolli has developed Z-lengthening of the biceps tendon. The technique includes rerouting of the biceps tendon around the radius to produce pronation. Interosseous membrane is also released if needed (Zancolli 1967). Surgical procedures are tailored on the basis of different elbow problems including elbow flexion contracture, possible dislocations of the radius or ulna, weak flexion and supination contracture (Hoffer and Phipps 2000). When there is elbow flexor deficiency, Steindler flexorplasty may be performed for elbow flexion reconstruction. In this technique, described by Arthur Steindler in 1918, increased flexion is achieved by transferring the pronator teres, flexor carpi radialis, palmaris longus, flexor carpi ulnaris and flexor digitorum superficialis arising from the medial epicondyle of the humerus to a more proximal position (Leffert 1998). With the above-mentioned technique, Carrol and Cartland achieved good results in 56%, fair results in 25% and poor results in 19% of 28 cases operated on because of poliomyelitis, or BPBI or other trauma to the brachial plexus (Carrol and Cartland
In a series described by Al–Qattan, good results were obtained in 89% of BPBI cases (8 out of 9) operated on using Steindler flexorplasty (Al-Qattan 2005).

Relocation of the humeral head

If muscle imbalance leads to posterior subluxation or dislocation of the glenohumeral joint, relocation of the humeral head is needed. The glenoid surface will be better preserved if the patient is operated on before 4 year of age (El Gammal et al. 2006). Goddard in turn stated that deformity of the humeral head is detected increasingly among children operated on after 4 years of age (Goddard 1993). Thus, prolonged conservative treatment may lead to a failed outcome after a relocation operation, because the remodeling capacity of the growth area diminishes after 4 years of age. In a prospective study by Birch et al. 100 children underwent primary plexus surgery at a median age of 4 months (Birch et al. 2005). Posterior dislocation of the shoulder was observed in 30 of these children and the shoulders were relocated successfully in all of them after the age of one year. Dislocation of the shoulder had an adverse effect on functional outcome. Even patients with successful relocation of the humeral head had lower Mallet scores than those without dislocation (ibid.). Torode and Donnan presented results of 12 children with BPBI-associated posterior dislocation of the humeral head who had undergone open reduction via an anterior approach at a mean age of 2 years and 3 months. Postoperative immobilization was carried out with a shoulder spica for 6 weeks and an additional 6 weeks in an orthosis. The dislocation and relocation of the humeral head was confirmed using CT. No redislocations were detected over a minimal follow-up of 12 months (Torode and Donnan 1998). Hui and Torode reported that open reduction with tendon lengthening decreased glenoid version by a mean of 31% over a mean follow-up period of 3 years and 7 months for 23 operated patients (mean age at surgery 2 years 5 months) (Hui and Torode 2003).

Kambhampati et al. performed a relocation operation for 183 patients with posterior subluxation or dislocation of the numeral head (Kambhampati et al. 2006). The anterior capsule was preserved as much as possible. For 70 patients, the degree of retroversion was over 40° or the humeral head was particularly unstable after relocation, and so an additional derotation osteotomy was done. The majority of the patients maintained a reduction during the 5 year follow-up period. There were only 20 failures with recurrent malpositioning of the humeral head. The mean Mallet score improved from 9.4 to 13. Remodeling of the humeral head was evident in most cases after successful relocation. Remodeling of the glenoid was difficult to measure because only plain radiographs were
taken in most cases. The successful relocation was done at a median age of 2 years 2 months, whereas patients with recurrent relocation were operated on a median age of 4 years 5 months (*ibid*).

**Osteotomy of the humerus**

In cases of internal rotation contracture with advanced glenohumeral deformity and an already flattened humeral head, Wickström et al. recommended rotation osteotomy of the humerus above the insertion of the deltoid. The goal of the external rotation osteotomy is to improve the functional arc of the shoulder rotation. An amount of external rotation sufficient to allow the hand to be brought to the mouth with the elbow in flexion and the arm adducted was selected during the operation (*Wickström 1955*). Goddard and Fixen performed rotation osteotomy using the above mentioned technique for 10 patients with good results. According to their experience sufficient lateral rotation of the distal part of the humerus is 30° beyond the neutral position. This correction improves the supination enough to allow the palm to be brought to the mouth instead the dorsum of the hand (*Goddard and Fixen 1984*). Bae and Waters propose 60° to 90° as a desirable external rotation. During the operation the hand has to be placed at the midline in order to avoid overcorrection before fixation with plate and screws (*Bae and Waters 2007*). Kirkos and Papadopoulos performed rotational osteotomy of the proximal part of the humerus on 22 patients with an average age of 10 years and 3 months because of internal rotation contracture or limited active external rotation of the shoulder. The average increase of 25 degrees external rotation and 27 degrees in active abduction allowed the patients to better wash themselves and eat. Given this good result, they recommended rotational osteotomy for late treatment of deformity in BPBI (*Kirkos and Papadopoulos 1988*). Several reports support rotational osteotomy as a late deformity correction method in BPBI (*Al-Qattan 2002, Waters and Bae 2006*). Al-Qattan performed derotational osteotomy of the humerus in 15 children, whose hand-to-neck Mallet score values increased from 2.2 to 4 after an average three years follow-up (*Al-Qattan 2002*).
AIMS OF THE STUDY

I: US screening for GH joint instability in BPBI
To assess prospectively the use and optimal timing of routine ultrasound (US) screening in order to detect shoulder subluxation in brachial plexus birth injury (BPBI) among infants.

II: MRI for detecting rotator cuff muscle changes related to GH joint pathology in BPBI
To assess rotator cuff muscles and the glenohumeral (GH) joint in brachial plexus birth injury (BPBI) with MRI and to investigate whether or not any correlation exists between muscular pathology and the deformity of the glenohumeral joint.

III: Muscle changes in BPBI with elbow flexion contracture
To investigate whether there is a correlation between specific patterns of muscular pathology and limited range of motion of the elbow in brachial plexus birth injury.

IV: MRI in evaluation of the surgically treated shoulder girdle in BPBI
To evaluate the outcome of shoulder operations in BPBI by MRI and to define the indications for different shoulder operations in correcting GH-subluxation and improving the range of motion of the joint.
MATERIALS AND METHODS

Patients

The study was approved by the ethics committee of the Hospital for Children and Adolescents, Helsinki University Hospital. When clinically indicated, MR imaging was performed under anesthesia in young patients.

US screening for GH joint instability in BPBI (I)

All neonates born in Women’s Hospital, Helsinki from 2003 to 2006 with evident or suspected BPBI as diagnosed by a pediatrician in a routine examination performed on all infants at 2 days of age were included in the prospective study. All BPBI patients born during the same 4-year-long study period who were referred to Children’s Hospital, Helsinki University Hospital maximum of 1 year of age from 8 other obstetric hospitals within the catchment area were also enrolled in the study. Neonates who had only a birth fracture of the clavicle without sufficient findings to support a diagnosis of BPBI during clinical examinations at 2-days, 2-weeks, and 1-month of age were excluded from the study. Patients that had a full recovery were also excluded from further follow-up and from the study.

MRI of rotator cuff muscle changes related to GH joint pathology in BPBI (II)

Of the 42 consecutive BPBI patients who were referred to the Hospital for Children and Adolescents, Helsinki University Central Hospital, 22 were boys and 20 girls. In the period March 2002 through April 2003, all patients underwent MRI because of internal rotation contracture, lack of active external rotation of the glenohumeral joint or possible posterior subluxation of the humeral head. Three patients (2 boys, 1 girl) were excluded from the study because MRI could not be completed for technical reasons, leaving 39 patients younger than 16 years in the prospective study. The mean age of the patients was 7.7 years (range 2.0-15.7 years). The right shoulder was affected in 23 patients (59%) and the left shoulder in 16 (41%). Seven patients (18%) had undergone brachial plexus reconstruction and 6 (15%) a subscapular release operation.

Muscle changes in BPBI with elbow flexion contracture (III)

Study III included 15 consecutive BPBI patients (9 boys, 6 girls) with elbow flexion contracture who were referred to Children’s Hospital, Helsinki. The mean age of the patients was 11 years (range 3-18 years). The injury involved the upper roots (C5-C6) in six (40%), the C5-C7 roots in five (33%) and the entire plexus (C5-T1) in four patients (27%) according clinical and ENMG
findings. The right side was affected in 10 patients (67%) and the left in five (33%). Two patients had had obstetric brachial plexus surgery at 3 and 5 months of age.

**MRI evaluation of surgically treated shoulder girdle in BPBI (IV)**

During the period March 2002 through December 2005, 34 permanent BPBI patients (14 girls, 20 boys) underwent surgery of the shoulder region in the Hospital for Children and Adolescents, Helsinki University Central Hospital. The final study group consisted of 31 patients. Three patients were excluded from the study because two of the patients failed to participate in the preoperative MRI study and one patient underwent a subscapular release operation by a surgeon not participated in the study. BPBI extended to the upper plexus (C5-C6) in 7 patients, to roots C5-C7 in 15 and to the entire plexus (C5-Th1) in 9 patients. Primary plexus reconstruction had been performed for 9 patients (29%).

**Healthy children**

Three healthy neonates (1 girl, 2 boys) participated in three ultrasound examinations for both shoulders performed by three pediatric radiologists at 2-day intervals over the course of one week for intra- and interobserver measurements in study I.

**Methods**

**US screening for GH joint instability in BPBI (I)**

*Clinical protocol*

A physiotherapist performed the examination at 2 weeks of age. Individualized passive range of motion exercises, which patients were unable to do actively, were taught to the parents. Repeat examinations were performed by a pediatric orthopedic surgeon or a hand surgeon at 1, 3, 6, and 12 months of age or until function of the upper extremities was clinically normal and symmetrical. Stability of the shoulders and range of motion of the upper limb joints were recorded during all examinations. Possible other birth injuries and stability and range of motion of all joints were assessed during the clinical examination at 1 month of age. The extent of the injury was assessed clinically according to the following scheme: C5, weak shoulder; C5-6, weak shoulder and elbow; C5-7, weak shoulder, elbow, and wrist; C5-8, weak shoulder, elbow, wrist, and hand; C5-T1, flail hand. The prevalence of permanent palsy at 1 year of age was calculated for all neonates with BPBI born in Helsinki during the 2003-2006 period. After the 1 year follow-up period, classification as
temporary or permanent BPBI was made. Patients with temporary BPBI had a full recovery over the 1 year follow-up period, whereas patients with permanent BPBI had persisting pathological findings.

**Ultrasound study**

A pediatric radiologist performed the scheduled US of both shoulders for all patients at 1, 3, 6, and 12 months of age. When there was full clinical recovery, the patient was removed from the US study.

The US study was carried out using an 8 MHz linear transducer (Acuson, Sequoia 512, Acuson Corporation, Mountain View, CA, USA or ATL HDI 5000, ATL Ultrasound, Bothell, WA, USA) and stability of the glenohumeral joint was assessed according to the modified method of Hunter et al. (*Hunter et al. 1998*). Sizes of the humeral head and the humeral ossification center were measured in transverse and longitudinal views. Possible humeral fractures or physeal injuries were recorded. A posterior axial approach with the upper arm of the patient adducted and the elbow flexed to 90° was used both in the static evaluation of joint congruency in maximal internal and external rotation and in the dynamic part of the study. In dynamic evaluation, the shoulder was scanned during the full range of internal to external rotation and the possible posterior subluxation of the humeral head was assessed visually. Posterior scanning also revealed the shape of the posterior glenoid. During the US examination young infants were lying on their side, and 1-year-olds were seated. A parent supported the child and held the studied hand in the proper position.

**Figure 5.**

US image of the left affected glenohumeral joint of a 1-month old girl with BPBI. a) Without tracing. b) With tracing. c) α-angle (20°) is within normal limits at the age of 1 month and ossified nucleus of the humeral head is located ventral to posterior scapular line. CG = cartilaginous glenoid, OC = ossification center of humeral head.
Ultrasound measurements

Measurement of the $\alpha$-angle was performed as described by Vathana et al. in order to evaluate possible posterior subluxation in addition to the visual assessment (Vathana et al. 2007). The angle between the posterior margin of the scapula and the line drawn tangentially to the humeral head and posterior edge of the glenoid is called the $\alpha$-angle (the normal value is $\leq 30^\circ$, Figure 5). Normally the humeral ossification center is located anterior to the posterior margin of the scapula (the posterior scapular line). In subluxation the posterior displacement of the humeral head gradually increases relative to the above described line.

Intra-and interobserver variations were evaluated by doing the measurements on healthy children. Three pediatric radiologists independently performed a total of 18 US examinations each. Both shoulders of 3 newborns were examined by all three observers three times at two-day intervals over the course of one week.

MRI of rotator cuff muscle changes related to GH joint pathology in BPBI (II)

Physical examination

A senior orthopaedic surgeon assessed the range of motion of the shoulder, glenohumeral (GH) congruence and possible subluxation of the GH joint by inspection and palpation during passive movements of the arm. The degree of internal contracture was measured by passive external rotation (PER) with the arm in adduction. PER and GH joint subluxation were used to appraise degree of internal rotation contracture and GH joint incongruence.

Magnetic resonance imaging

Both shoulders were imaged using a surface coil in a 1.5 T MR imager (Siemens, Erlangen, Germany). T1-weighted spin echo images (TR 817 ms, TE 20 ms, TA 6 min 2 sec, matrix 220 x 256) in axial, oblique coronal, and oblique sagittal planes were obtained. In addition T2*-weighted 2 D gradient echo (MEDIC) images (TR 944.3 ms, TE 25.8 ms, flip angle 30 $^\circ$, TA 4 min 2 sec, matrix 256 x 256) were obtained in the axial plane. The field of view was 160 mm x 138 mm in T1-weighted images and 220 mm x 220 mm in 2 D gradient echo images. A 4.0 mm slice thickness was used in all sequences and the scans were archived in PACS (IMPAX, Agva-Gaevert N.V., Mortsel, Belgium).

A senior radiologist, blinded to the clinical situation, evaluated all MR images using the IMPAX 4.5 for Orthopedics software tool. The glenoscapular angle (GSA; degree of version of the glenoid)
was measured by drawing two bisecting lines according to the technique described by Friedman et al. (Friedman et al. 1992). The first line was drawn between anterior and posterior corners of the glenoid cartilage. A second, bisecting line was drawn from the medial tip of the scapula to the middle point of the glenoid. The angle between the first line going through the posterior margin of the glenoid and the line along the axis of the scapular (posteromedial quadrant) was measured. The GSA was obtained by subtracting 90° from the posteromedial quadrant angle (Figure 6). The glenoid configuration was classified using the scheme of Pearl et al., as concentric (the humeral head centered in a glenoid with a matching curve), flat, biconcave (the humeral head articulated with a posterior surface of the glenoid), or pseudoglenoid (the posterior articular surface of the glenoid retroverted in relationship to the original glenoid). In the pseudoglenoid case, the angle of version was measured along the posteriorly retroverted glenoid (Pearl et al. 2003).

The percentage of humeral head anterior to the middle of the glenoid fossa (PHHA) was measured as described in Waters et al. (Waters et al. 1998): The anterior aspect of the humeral head (anterior to the scapular line described above) is divided by the transverse diameter of the humeral head, and then multiplied by 100 (Figure 6). Normally, half of the humeral head is located anterior to the scapular line and the PHHA value is around 50%. The more severe is the posterior subluxation, the lower is the PHHA value.

In order to evaluate the degree of rotator cuff muscle atrophy, the greatest thicknesses of the subscapular, infraspinous, and supraspinous muscles were measured from the oblique sagittal plane in millimeters (Figure 7). To exclude the effect of age variation, the muscle ratio between the affected side and the normal contralateral side was calculated for every muscle. Muscle atrophy was considered mild for a ratio of 0.9 – 0.75, moderate for a ratio of 0.75 – 0.5, and severe for a ratio of < 0.5. The amount of fatty infiltration was assessed visually from the deltoid muscle without measurements in diameters.
Figure 6.

Figure 7.
Muscle changes in BPBI with elbow flexion contracture (III)

Physical examination

An experienced orthopedic surgeon measured both passive and active range of motion of the elbow and of the forearm using a goniometer in order to assess the congruency of the elbow joint. Normal range of motion of the elbow is rated from 0° (full extension) to 150° (full flexion). Pronation and supination were measured with the upper arm in adduction and the elbow at 90° flexion, with normal values measuring 90°–0°–90° (total active motion, TAM, 180°).

Magnetic resonance imaging

The prospective MRI studies were performed with a 1.5-T Siemens Sonata or a Siemens Vision imager (Siemens, Erlangen, Germany) using a surface coil, with the patient supine. Imaging studies were carried out between April 2004 and September 2005. T1-weighted axial spin echo (SE) images (TR 760 ms, TE 20 ms, matrix 256 x 256, field of view 160 x 138 mm, 3.0-mm slice thickness, an approximate acquisition time of 6 minutes) were obtained. The interslice gap and the number of slices were adjusted to cover the arm, elbow, and forearm. Congruency of the elbow joint was assessed with following MR sequences: sagittal T1-weighted flash 2 D (TR 500 ms, TE 10 ms, flip angle 90°, matrix 256 x 256, field of view 150 x 138 mm, and 3.0 mm slice thickness with an approximate acquisition time of 4 minutes), coronal T2-weighted (TR 3000 ms, TE 90 ms, matrix 256 x 256, field of view 150 x 138 mm), and PD-weighted (TR 3000 ms, TE 16 ms, matrix 256 x 256, field of view 150 x 138 mm) with 3.0-mm slice thickness and an approximate acquisition time of 5 minutes. Both the affected and the contralateral upper limb were imaged with the same protocol. For technical reasons, one patient underwent the MR study only for the elbow region. All the remaining 14 patients underwent the entire MR study.

The images were interpreted by consensus, by two radiologists with 3 and 5 years experiences in musculoskeletal radiology blinded to both clinical history and findings, using ordinary clinical workstations (Agfa Impax DS3000, Agfa-Gevaert Group, Mortsel, Belgium). The congruency of the elbow joint was evaluated. The degree of muscle atrophy was assessed visually based on the presence of an abnormal amount of fatty infiltration or reduction in size of the muscle compared to the healthy contralateral side. The amount of muscle fat content was semi-quantitatively graded on a four-point scale (F0-F3), and the reduction in muscle size was assessed using a three-grade scale (SR 0-SR 2), both adapted from Mahjneh et al. in the following way: (F0) normal muscle signal intensity: homogenous hypointense signal, contrasting sharply with subcutaneous and intermuscular fat; (F1) hyperintense: patchy but not confluent intramuscular signal; (F2) hyperintense; patchy and...
confluent intramuscular signal, involving <50% of individual muscle volume; (F3) homogenously hyperintense: confluent intramuscular signal, involving >50% of individual muscle volume; (SR 0) normal size; (SR 1) moderate atrophy corresponding to a slight (<50%) reduction in muscle size; (SR 2) severe atrophy (>50%) of the muscle (Mahjneh et al. 2004).

MRI evaluation of surgically treated shoulder girdle in BPBI (IV)

Clinical examination

A physiotherapist measured with a goniometer external rotation in adduction and abduction as well as elevation of the arm pre- and postoperatively. Range of motion of the upper limb was assessed and Mallet classification (Figure 1) was used for functional assessment of the shoulder (Mallet 1972).

The most important denominator in selection of the operation method was congruency of the GHJ. The relocation operation (n=13) was performed for young patients (age 0.9–7.7 years) with posterior shoulder subluxation and deformity (GSA -60º to -20º, PHHA 4-40%). An additional operation had been performed for four relocation patients: teres major transposition for three patients and internal rotation osteotomy of the humerus for one patient. The coracoid process was shortened in addition to subscapular tendon lengthening and coracohumeral ligament recession in 10 of these 13 patients. Two of the patients had a biconcave glenoid and eleven a pseudoglenoid.

Older patients (age 5.7–14 years) with a deformed GHJ (GSA -60º to -35º, PHHA range -10% to 21%) were operated on using external rotation osteotomy (n=5) above the deltoid tuberosity. In this operation the distal humerus was externally rotated (30º –40º) and osteosynthesis was performed with a plate.

Children (n=5) without significant shoulder joint deformity (internal rotation contracture: limited passive external rotation, range -20º to 0º) were treated by subscapular tendon lengthening.

Children (n=8) with poor active elevation without significant shoulder joint deformity (limited active abduction, range 90º to 180º) were treated by teres major transposition; for 3 of these patients (internal rotation contracture with limited active abduction) subscapular lengthening was also done. The algorithm of the treatment protocol is shown in Figure 8.
After relocation surgery the upper extremity was immobilized with a thoracobrachial cast or soft cast for 3 to 7 weeks (mean 5 weeks). The immobilization time was 3 to 4 weeks after humeral osteotomy, 3 to 4 weeks after subscapular tendon lengthening and 4 to 5 weeks after teres major transposition. Possible postoperative complications were registered and the opinion of the patient (above 7 years) or parents (children under 7 years of age) was obtained regarding the usefulness of the operation.

**Magnetic resonance imaging**

Pre- and postoperative MRI of the affected shoulder was performed for all patients at a mean MRI follow-up time of 3.8 (SD1.2) years (range 1.7–6.8 years). Imaging studies were done with a 1.5 T MRI imager (Magnetom Vision, Siemens, Erlangen, Germany or Achieva, Philips Medical Systems, Best, the Netherlands) with a surface coil. During the MR examination the patient was in the supine position on the table with the arm along the side of the body. The MR imaging protocol consisted of the following sequences for the Siemens imager: T1-weighted spin echo images in axial, oblique coronal, and oblique sagittal planes (TR 817 ms, TE 20 ms, TA 6 min 2 sec, matrix 220 x 256, FoV 160 x 138 mm) and T2*-weighted 2D gradient echo images in the axial plane (TR 944.3 ms, TE 25.8 ms, flip angle 30 °, TA 4 min 2 sec, matrix 256 x 256, FoV 220 x 220 mm). For the Philips MRI imager, the corresponding parameters were: T1-weighted spin echo images in axial, oblique coronal, and oblique sagittal planes (TR 500 ms, TE 15 ms, TA 4 min, matrix 240 x 240, FoV 160 x 144 mm) and 3D WATSc sequence in the axial plane (TR 20 ms, TE 7.7 ms, flip angle 25 °, TA 5 min 47 sec, matrix 288 x 232, FoV 160 x 129 mm). Slice thickness was 4.0 mm in all the sequences, except 2mm in the Philips 3D WATSc- sequence. The measurements were performed using the IMPAX for Orthopedics (Agfa-Gevaert Group, Mortsel, Belgium) software tool.

The glenoscapular angle (GSA), according to the method defined by Friedman et al. (*Friedman et al. 1992*), and PHHA, according Waters et al., were measured as described in the methods section of study II (*Waters et al. 1998*). The shape of the glenoid was classified as either normal concentric, flat, biconcave or pseudoglenoid according Pearl et al. (*Pearl et al. 2003*). The glenohumeral joint was classified as congruent, subluxated, or badly deformed pseudoglenoid when the humeral head was articulating to the deformed posteriorly retroverted glenoid which was located in a different plane than the normal glenoid.
Figure 8. Treatment flowchart for brachial plexus birth injury with poor active external rotation. GHJ = glenohumeral joint.
Statistical analysis

Study I: US screening for GH joint instability in BPBI
Analysis of variance (NCSS and PASS; Number Cruncher Statistical Systems, Kaysville, Utah) was used for statistical analysis. A difference with $P=0.05$ was considered significant, and 95% confidence intervals were calculated for the measurements (Figures 10,11) using spreadsheet software (Microsoft Office Excel 2003 for Windows; Microsoft, Redmond, Wash).

Study II: MRI of rotator cuff muscle changes related to GH joint pathology in BPBI
The Mann-Whitney test and Spearman’s rank correlation test were used for statistical analysis (SPSS for Windows, version 13.0, SPSS, Chicago, IL, USA). A $P$-value < 0.05 was considered statistically significant.

Study III: Muscle changes in BPBI with elbow flexion contracture
The Wilcoxon signed-rank test was used, as appropriate, for statistical analysis. P-values of < 0.05, <0.01 and < 0.001 were considered to indicate significant, very significant and highly significant differences, respectively. Correlations were calculated using Spearman’s two-tailed correlation test (SPSS for Windows, version 13.0, SPSS, Chicago, IL, USA), where a $P$-value < 0.05 was considered statistically significant.

Study IV: MRI evaluation of surgically treated shoulder girdle in BPBI
Values of ROM are presented as medians (range) with SE. Mallet score sums were expressed with mean values and standard deviations. The Wilcoxon test was used to compare pre- and postoperative Mallet and GSA- values in different operation groups. $P< 0.05$ was considered significant.
RESULTS

US screening for GH join instability in BPBI (I)
Of the 41 980 live births during the years 2003-2006 at Women’s Hospital 187 neonates with suspected BPBI were referred to Children’s Hospital. A clavicle fracture was diagnosed in 75 of these neonates, of whom 55 were excluded from the study because they had insufficient clinical findings to support BPBI, leaving 132 patients with BPBI (73 girls, 59 boys; right side 73, left side 59). The incidence of BPBI in Helsinki during the study period was 3.1 BPBI per 1000 live births. There were 131 cephalic and 1 breech presentation, all vaginally delivered with a mean birth weight of 4172g (range 2930 – 5850g). In 31 cases (23%) vacuum cup assistance was needed during delivery.

Additionally 21 BPBI patients from eight different obstetric hospitals within the tertiary catchment area from among 48 994 births were also referred to Children’s Hospital. These BPBI patients (11 girls, 10 boys; right side 14, left side 6, bilateral 1) were referred at 1-12 months of age. All of these patients were vaginally delivered; vacuum cup assistance was needed in 8 cases with cephalic presentation. The mean birth weight was 4389 g (range 3605 – 5310 g).

Clinical findings. The extension of affected roots are presented in Table 3. BPBI healed during the first year of life in 105 cases (80%) out of 132 palsies, leaving 27 cases for whom BPBI was considered to be permanent (incidence 0.64 per 1000 live births). Primary brachial plexus surgery was performed for one (0.76%) of 132 BPBI patients and one (4%) of 27 permanent palsies. Only two of the referred patients from the tertiary catchment area had temporary palsy and two (9.5%) of these referred 21 patients had had obstetric brachial plexus surgery.
### Table 3. Affected roots in BPBI.

<table>
<thead>
<tr>
<th>Roots</th>
<th>Women’s Hospital</th>
<th>Referred patients from tertiary catchment area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Extent of BPBI at 2 days</td>
<td>Extent of BPBI at 1 year of age</td>
</tr>
<tr>
<td></td>
<td>Temporary</td>
<td>Permanent</td>
</tr>
<tr>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1 (C5)</td>
<td>17</td>
<td>-</td>
</tr>
<tr>
<td>2 (C5-6)</td>
<td>51</td>
<td>8</td>
</tr>
<tr>
<td>3 (C5-7)</td>
<td>30</td>
<td>9</td>
</tr>
<tr>
<td>4 (C5-8)</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>5 (C5-Th1)</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Sum</td>
<td>105</td>
<td>27</td>
</tr>
</tbody>
</table>

### Table 4. Primary diagnosis of posterior subluxation.

<table>
<thead>
<tr>
<th>Months</th>
<th>Women’s Hospital</th>
<th>Referred patients from tertiary catchment area</th>
<th>All patients combined</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinically US</td>
<td>(\alpha)-angle</td>
<td>Clinically US</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>5</td>
<td>47° (40°-62°)</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>3</td>
<td>47° (40°-60°)</td>
</tr>
<tr>
<td>12</td>
<td>1</td>
<td>1*</td>
<td>50°</td>
</tr>
<tr>
<td>Sum</td>
<td>8</td>
<td>9</td>
<td>8</td>
</tr>
</tbody>
</table>

Mean \(\alpha\)-angle measurements are given with ranges. * No earlier US studies done. # 6-month US study was normal for one patient, 12-month US study was the first for another patient.
Eighteen of the 27 patients with permanent palsy from Women’s Hospital had restricted passive external rotation of the shoulder at a mean age of 2.5 months (median 1 month, range 0.5-12 months). Clinically evident instability of the shoulder was recorded at a mean age of 6 months (median 3 months, range 3 months to 3 years) in 9 (50%) of these 18 patients. At the time of diagnosis of posterior subluxation of the humeral head the mean passive external rotation was 52° (range 0°-80°). None of the patients with temporary palsy developed shoulder instability.

Thirteen of the 19 patients with permanent palsy from the tertiary catchment area had restricted passive external rotation of the shoulder at a mean age of six months (median 6.5 months, range 3-11 months). Nine (69%) of these 13 patients had clinically diagnosed posterior subluxation of the humerus at a mean age of four months (median 3 months, range 2-9 months). At the time of diagnosis of posterior subluxation of the humeral head the mean passive external rotation was 48° (range 0°-80°).

**Ultrasound findings.** A total of 194 US studies were performed for 96 BPBI patients during the study period (Figure 9). Twenty-four children were examined at 1, 3, 6, and 12 months of age. Growth of the cartilaginous humeral head and ossification center during the first year of life is presented in Figure 10, where error bars show 95% confidence intervals. On the affected side the widths of the cartilaginous humeral head and the ossified nucleus were smaller than on the healthy side in all patients with permanent palsy. Patients with posterior subluxation of the humeral head had the largest discrepancy (Figure 11). A conjoint fracture of the humerus was detected in two patients, but no physeal injuries were seen. At 1 month of age glenohumeral congruency and shape of the glenoid were symmetrical in all scanned children. Nine (6.8%) of the 132 BPBI patients born at Women’s Hospital and 10 (48%) of the 21 BPBI patients referred from the tertiary catchment area developed US-verified posterior subluxation of the humeral head (Table 4). US study was not done for two patients with clinically detected posterior subluxation. 15 patients with clinically and ultrasonographically verified posterior subluxation had rounded posterior margins of the glenoid. Posterior subluxation (α-angles 38°-53°) was detected on US in three patients who were clinically estimated to have a congruent glenohumeral joint. The results for the subgroup of patients who participated in the complete US follow-up program are presented in Figure 10b and Figure 11b. In this group there were 10 posterior subluxations, all of which were permanent palsies. Seven of these subluxations were diagnosed by US at 3 months of age (Figure 12) and three at 6 month.
Figure 9. Flowchart providing information about the number and timing of US studies conducted on neonates referred to Children’s Hospital from a) Women’s Hospital of Helsinki.
BPBI patients referred to Children’s Hospital from tertiary catchment area outside of Helsinki (n=21)

1 month

Ultrasound study (n=7)

Ultrasound study not done (n=1)
• full recovery before 3 months of age

3 months

Ultrasound study (n=6)

Ultrasound study (n=7)

6 months

Ultrasound study (n=5)

Ultrasound study (n=3)

Ultrasound study (n=3)

12 months

(n=3)
• not done n=1

(n=1)
• not done n=2

(n=1)
• not done n=3

(n=1)
• not done n=2

(n=1)
• not done n=3

Figure 9. Flowchart providing information about the number and timing of US studies conducted on neonates referred to Children’s Hospital from b) tertiary catchment area outside Helsinki.
Size of the humeral head (mm) on the healthy size

Figure 10 a. Ultrasound (US) findings of a healthy humeral head performed on 82 patients with brachial plexus birth injury (BPBI) at 1 month of age. Thirty-nine US examinations were done at 3 months of age, 38 at 6 months of age, and 35 at 1 year of age. Error bars show 95% confidence intervals. cw=cartilage width, ch=cartilage height, ow=osseus width, oh=osseus height.

Figure 10 b. Ultrasound (US) findings of a healthy humeral head in the 24 BPBI patients who participated in US studies at all time points. Error bars show 95% confidence intervals. cw=cartilage width, ch=cartilage height, ow=osseus width, oh=osseus height.
Figure 11. Effect BPBI on humeral head growth analyzed by calculating the ratio of the humeral head width between the affected and the healthy side. Error bars show 95% confidence intervals. ct= cartilage temporary palsy, cpc= cartilage permanent BPBI congruent GHJ, cps=cartilage permanent BPBI subluxation of the humeral head, ot= osseus temporary palsy, opc= osseus permanent BPBI congruent GHJ, ops= osseus permanent BPBI subluxation of the humeral head.
a)Ultrasound measurements were performed on 51 patients with temporary BPBI at 1 month of age, on 9 patients at 3 months of age, and on 8 patients at both 6 and 12 months of age. Nineteen of the 44 patients with permanent BPBI had posterior subluxation of the humeral head (top graph).

Figure 11 b) Ultrasound measurements of the 24 patients in the subgroup who participated in US examinations at all time points. Among these patients BPBI was permanent in 20 patients, of whom 10 had posterior subluxation of the humeral head (bottom graph).
Figure 12.
US images of a 3-month-old girl with permanent brachial plexus birth injury on right side. 

a) Both glenohumeral joints: normal finding on the left unaffected side and posterior subluxation of the right humeral head ($\alpha$-angle $40^\circ$)(upper images). 

b) Posterior subluxation of the right humeral head in internal rotation (IR) and relocation in external rotation (ER) (lower images).
MRI of rotator cuff muscle atrophy related to GH joint incongruence in BPBI (II)

Posterior subluxation of the humeral head was clinically detected in ten (25%) patients and the mean PER was 34° (range 20° to 85°) in physical examination. In MRI the shape of the glenoid was concentric in 18, flat in six, biconcave in two and pseudoglenoid in 13 children. The mean GSA was -20.1° ± 18.2° (range -60° to 0°) and the average PHHA was 30.2% (range -36% to 54%) on the affected side (Figure 13). Mean values of GSA and PHHA measurements are presented in Table 5.

**Figure 13.** Axial T1-W images of a) normal right shoulder and b) the affected left shoulder of a 8-year-old boy with BPBI (C5-7). Retroversion of the left glenoid with an abnormal GSA of -45°, PHHA 18%. On the normal right side GSA is -2° and PHHA 47%.

Table 5. Mean values of glenoscapular angle (GSA) and percentage of humeral head anterior to the middle of glenoid fossa (PHHA) measurements in affected (BPBI) and contralateral (normal) side. GSA=Glenoscapular angle, PHHA=Percentage of humeral head anterior to the middle of glenoid fossa. N=number of patients.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>N</th>
<th>GSA BPBI</th>
<th>GSA normal</th>
<th>PHHA BPBI</th>
<th>PHHA normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.0-4.0</td>
<td>11</td>
<td>-29.7</td>
<td>-2.9</td>
<td>29.5</td>
<td>46.7</td>
</tr>
<tr>
<td>4.1-8.0</td>
<td>12</td>
<td>-19.6</td>
<td>-2.3</td>
<td>31</td>
<td>46.3</td>
</tr>
<tr>
<td>8.1-12.0</td>
<td>6</td>
<td>-10.3</td>
<td>-2</td>
<td>41.7</td>
<td>45.7</td>
</tr>
<tr>
<td>12.1-14</td>
<td>10</td>
<td>-16.1</td>
<td>-2.4</td>
<td>23</td>
<td>46.4</td>
</tr>
</tbody>
</table>
Muscle atrophy was noted in all rotator cuff muscles (Figure 14); affected/healthy muscle ratios are presented in Table 6 and correlations in Table 7. Muscle atrophy of the supraspinous muscle was classified as mild in 17, moderate in 11 and severe in one patients. The corresponding values for infraspinous and subscapular muscles were 5, 26, 5 and 13, 16, 7, respectively. The difference between the affected side and the healthy side was statistically significant for all three muscles: supraspinous (p=0.01), infraspinous (p<0.001), and subscapular (p<0.001). On the affected side every patient had also intramuscular fatty degeneration of the deltoid muscle.

Table 6. Affected/healthy muscle ratio showing muscle atrophy.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>No prior subscapular release (n=33)</th>
<th>Prior subscapular release (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supraspinous</td>
<td>81.9 % (range 49-100%)</td>
<td>73.15% (range 49-100%)</td>
</tr>
<tr>
<td>Infraspinous</td>
<td>69.1% (range 38-100%)</td>
<td>56.96% (range 29-71%)</td>
</tr>
<tr>
<td>Subscapular</td>
<td>69.4% (range 15-100%)</td>
<td>60.8% (range 32-96%)</td>
</tr>
</tbody>
</table>

Figure 14. Oblique sagittal T1-W images of the a) affected right shoulder and b) the normal left shoulder of a 6-year-old girl with BPBI (C5-7). Muscle atrophy was observed in the supraspinous, infraspinous and subscapular muscles.
Table 7. Correlations of various anatomical and functional measurements.

<table>
<thead>
<tr>
<th>Correlations</th>
<th>$r_s$</th>
<th>P</th>
<th>$r_s$ (ratio)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSA/PHHA</td>
<td>0.80</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>supras/infras</td>
<td>0.81</td>
<td>0.01</td>
<td>0.54</td>
<td>0.01</td>
</tr>
<tr>
<td>supras/subsc</td>
<td>0.67</td>
<td>0.01</td>
<td>0.36</td>
<td>0.05</td>
</tr>
<tr>
<td>infra/subsc</td>
<td>0.66</td>
<td>0.01</td>
<td>0.35</td>
<td>0.05</td>
</tr>
<tr>
<td>GSA/supras</td>
<td>0.32</td>
<td>0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSA/infras</td>
<td>0.37</td>
<td>0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSA/subsc</td>
<td>0.56</td>
<td>0.01</td>
<td>0.47</td>
<td>0.01</td>
</tr>
<tr>
<td>PHHA/supras</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHHA/infras</td>
<td></td>
<td></td>
<td>0.35</td>
<td>0.05</td>
</tr>
<tr>
<td>PHHA/subsc</td>
<td>0.45</td>
<td>0.01</td>
<td>0.51</td>
<td>0.01</td>
</tr>
<tr>
<td>PER/GSA</td>
<td>0.41</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PER/PHHA</td>
<td>0.48</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PER/supras</td>
<td></td>
<td></td>
<td>0.32</td>
<td>0.05</td>
</tr>
<tr>
<td>PER/infras</td>
<td>0.38</td>
<td>0.05</td>
<td>0.41</td>
<td>0.01</td>
</tr>
<tr>
<td>PER/subsc</td>
<td>0.53</td>
<td>0.01</td>
<td>0.43</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Note: GSA = glenoscapular angle, PHHA = the percentage of humeral head anterior to the middle of the glenoid fossa, supras = supraspinous muscle, infras = infraspinous muscle, subsc = subscapular muscle, PER = passive external rotation, $r_s$ = correlation coefficient, $r_s$ (ratio) = correlation of the diameters of the affected muscle/ the contralateral healthy muscle, P = statistical significance.

Muscle changes in BPBI with elbow flexion contracture (III)

TAM and passive motion of the elbow ranged between 50° and 140° (mean 113°) resulting in a mean elbow extension deficit of 31° (10° to 90°). Mean active pronation was 57° (5° to 90°) and mean active supination 34° (range 0° to 80°). Passive pronation was better than active in five children (mean 14°, range 10° to 30°) and passive supination better than active in eight children (mean 39°, range 10° to 75°). Radial head dislocation occurred in one patient (patient 7), and subluxation of the humeroulnar joint in one (patient 14) both detected in physical examination and in MR imaging. In MR imaging, congruency of the elbow joint was found to be normal in 12 patients and the radiohumeral joint was subluxated in one additional patient (patient 9) without clinically evident instability. Atrophy was visible especially in the supinator, brachial, and brachioradial muscles (Table 8). The summary of clinical and MR imaging findings is presented in Table 9.

TAM of the elbow correlated with TAM of the forearm ($r_s=0.67$, p=0.01). Elbow TAM correlated negatively with size reduction in the brachioradial muscle ($r_s=−0.62$, p=0.05), with wrist and finger flexors ($r_s=−0.74$, p=0.01), and with patient age ($r_s=−0.52$, p=0.05). Forearm TAM correlated
negatively with the extent of injury (C5–6 vs. C5–7 vs. total) ($r_s = -0.72, p=0.01$), with fatty infiltration ($r_s = -0.66, p=0.01$) and size reduction ($r_s = -0.62, p=0.05$) in the pronator teres muscle. The extent of injury correlated with the summed grades of fatty atrophy and size reduction in the muscles ($r_s=0.52, p=0.05$) and with the extent of pathological findings on MRI in the ten muscles examined ($r_s=0.76, p=0.01$).

Table 8. Changes of the muscles in BPBI.

<table>
<thead>
<tr>
<th>Muscle (root)</th>
<th>F</th>
<th>SR</th>
<th>F/SR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deltoid (C5-6)</td>
<td>0 (0-2)*</td>
<td>1 (0-1)*</td>
<td>7</td>
</tr>
<tr>
<td>Biceps (C5-6)</td>
<td>0 (0-1)*</td>
<td>0 (0-1)</td>
<td>7</td>
</tr>
<tr>
<td>Brachial (C5-6)</td>
<td>1 (0-2)**</td>
<td>1 (0-2)**</td>
<td>15</td>
</tr>
<tr>
<td>Brachioradial (C5-6)</td>
<td>1 (0-2)**</td>
<td>1 (0-1)**</td>
<td>11</td>
</tr>
<tr>
<td>Supinator (C5-6)</td>
<td>2 (1-3)***</td>
<td>1 (1-2)***</td>
<td>15</td>
</tr>
<tr>
<td>Protanor teres (C6-8)</td>
<td>0 (0-1)*</td>
<td>0 (0-1)**</td>
<td>7</td>
</tr>
<tr>
<td>Anconeus (C7-8)</td>
<td>0 (0-3)</td>
<td>0 (0-2)*</td>
<td>7</td>
</tr>
<tr>
<td>Triceps (C6-Th1)</td>
<td>0 (0-2)</td>
<td>1 (0-2)**</td>
<td>10</td>
</tr>
<tr>
<td>Extensors (C6-Th1)</td>
<td>0 (0-1)</td>
<td>1 (0-1)**</td>
<td>11</td>
</tr>
<tr>
<td>Flexors (C6-Th1)</td>
<td>0 (0-2)</td>
<td>0 (0-1)**</td>
<td>7</td>
</tr>
</tbody>
</table>

Median (range) amount of fatty infiltration (F, 0-3) of affected side and median (range) reduction in size of muscle (SR, 0-2) are given. Numbers of the patients who have fatty infiltration and/or size reduction (F/SR) of the studied muscles are provided. Extensors =extensor muscles of wrist and fingers, Flexors = flexor muscles of wrist and fingers. The nerve roots innervating studied muscles are also given in the parentheses. Statistically significant differences between the affected and healthy muscles are marked as follows: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. 
Table 9. Total active motion (TAM) of the elbow and forearm and muscle atrophy.

<table>
<thead>
<tr>
<th>Patient</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>6</td>
<td>9</td>
<td>8</td>
<td>16</td>
<td>8</td>
<td>3</td>
<td>12</td>
<td>12</td>
<td>18</td>
<td>8</td>
<td>13</td>
<td>13</td>
<td>16</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Extent of injury</td>
<td>C5-6</td>
<td>C5-7</td>
<td>C5-7</td>
<td>C5-6</td>
<td>C5-6</td>
<td>Total</td>
<td>C5-7</td>
<td>C5-6</td>
<td>C5-6</td>
<td>C5-7</td>
<td>C5-6</td>
<td>Total</td>
<td>Total</td>
<td>Total</td>
<td>C5-7</td>
</tr>
<tr>
<td>TAM of elbow</td>
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<td>135°</td>
<td>135°</td>
<td>130°</td>
<td>125°</td>
<td>125°</td>
<td>120°</td>
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<td>115°</td>
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<td>105°</td>
<td>105°</td>
<td>90°</td>
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<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
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<td>1</td>
<td>1</td>
<td>2</td>
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</tr>
<tr>
<td>Brachio-radial</td>
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<td>1</td>
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<td>Triceps</td>
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<td>0</td>
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<td>0</td>
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F=fat, SR=size reduction in muscle. Flexion-extension and active prosupination movements are given in degrees. Explanations of the additional data: □= flexor carpi ulnaris (FCU) pro extensor digitorum communis transposition, + = plexus reconstruction, #= radiohumeral incongruence, *= humeroulnar incongruence, • = anterior elbow flexor release, N/A not available.
MRI evaluation of surgically treated shoulder girdle in BPBI (IV)

All patients or parents were satisfied to the result of the operation except one patient who found no improvement from a subscapular tendon lengthening operation. Table 10 presents the data on number of patients with different operation types, age at the time of surgery, postoperative GSA, PHHA, type of glenoid, congruency of the GH joint, external rotation in adduction, and elevation measurements. Pre- and postoperative mean values and standard deviations of Mallet score sums are shown in Figure 15. The only complication was a deep wound infection in one patient who had undergone a relocation operation.

A relocation operation of the humeral head was performed for 13 patients. After successful relocation (10/13) the postoperative median value for passive external rotation was 48° (range 10° to 80°, SE 8). The improved postoperative measurements were as follows: active external rotation among 10 patients in adduction (median increase 38°, range 10° to 100°, SE 9) and in abduction (median 48°, range 15° to 80°), and in elevation among 6 patients (median 40°, range 30° to 70°). After the successful relocation operation the mean increase in Mallet score sum (Figure 15) was 5.5 (SD 3.0). The postoperative mean improvement in GSA was 33° (SD 14) and PHHA 25% (SD 10) among 10 patients. In the relocation group the difference between pre-and postoperative GSA values was statistically significant (p<0.05). Postoperatively the shape of the glenoid improved in 10 patients (flat 9, concentric 1). Three failed procedures resulted in resubluxation of the humeral head. Two of these patients were over 6 years old at the time of the relocation operation and the third patient had an immobilization time that was too short (4 weeks).

Humeral osteotomy was performed for five patients at a mean age of 9.4 (SD 3.2) years. The postoperative median passive external rotation was 30° (range 0° to 60°, SE 10). In this group the median increase in active external rotation in adduction was 25° (range 20° to 40°, SE 3.7) and in abduction 30° (range 25° to 45°). Elevation improved in two patients postoperatively. One patient had an increased PHHA of 16%. GSA values or the shape of the glenoid did not change after humeral osteotomy.

The median passive external rotation was 45° (range -20° to 80°, SE 17) after subscapular tendon lengthening operation. For four patients the subscapular tendon lengthening operation resulted in better active external rotation in adduction (median increase 20°, range 20° to 35°, SE 3.7) and in abduction (median 55°, range 45° to 75°, SE 6.6). For three patients elevation improved (median 20°, range 20° to 40°, SE 6.6) as well. In retrospect, one of these five patients should have been
treated with osteotomy. GSA, PHHA, and the shape of the glenoid did not improve after subscapular tendon lengthening.

A teres major to infraspinatus transposition operation was done for eight patients, three of whom also had subscapular tendon lengthening. The median active external rotation in abduction was 73° (range 45° to 80°, SE 6.0). Postoperatively, five of these eight patients showed improved active external rotation in adduction (median 40°, range 10° to 45°, SE 6.8), whereas elevation (median 25°, range 10° to 90°, SE 13) improved in six patients. This operation type did not improve GSA, PHHA, or shape of the glenoid.

**Figure 15.** Pre- and postoperative mean values and standard deviations of Mallet score sums for different operation types.
Table 10. Patient data before and after surgery

<table>
<thead>
<tr>
<th></th>
<th>Relocation (n = 13)</th>
<th>Osteotomy (n = 5)</th>
<th>ST lengthening (n = 5)</th>
<th>TM transposition (n = 8)</th>
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<tr>
<td><strong>Add. oper (n)</strong></td>
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<td></td>
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<td><strong>PHHA (%)</strong></td>
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<tr>
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Median (range) values of age, number of roots injured, degrees of GSA, ER and elevation, percentage of PHHA are given for all operated patients. ST lengthening = subscapular tendon lengthening, TM transposition= teres major transposition, Plex. rec.=plexus reconstruction, GSA=glenoscapular angle, PHHA=percentage of humeral head anterior to the middle of the glenoid fossa, ER=active external rotation in adduction, Glenoid=type of glenoid, Congruency=congruency of the glenohumeral joint, Additional operations performed later: * = teres major transposition, ● = osteotomy, # = subscapular lengthening, n= number of patients.
DISCUSSION

Fetal macrosomia, shoulder dystocia, breech extraction and instrumental deliveries are known to be risk factors for BPBI (Soni et al. 1985). Nerve root injury most often affects the upper roots but may affect the entire plexus. The majority of patients have temporary palsy and recover within the first years of life (Jackson et al. 1988). In permanent palsy nerve injury results in a limited range of motion of the shoulder, elbow and hand as well as anatomical changes of the affected upper limb. Most authors believe that BPBI-associated shoulder pathology develops gradually after birth and is not caused by direct joint trauma (Gilbert 1993, Waters et al. 1998). The discussion has been quite extensive in the literature concerning BPBI-associated bony deformities imaged with plain radiographs (Polloc and Reed 1989), CT (Hernandez and Dias 1988, Friedman et al. 1992, Waters and Peljovich 1999, Hui and Torode 2003) and MRI (Gudinchet et al. 1995, Waters et al. 1998, Pearl et al. 2003, Kozin 2004). Discussion concerning imaging findings of the pathological muscle changes in BPBI has intensified lately.

This prospective study included a population-based section where the incidence and early detection of posterior subluxation were defined. Further, the prospective part also included studies on MRI findings of muscle and articular changes in the shoulder and elbow areas in late sequelae of permanent BPBI and additionally imaging findings after operative treatment of shoulder sequelae in BPBI.

The incidence of BPBI has been reported to vary between 0.42 and 3.8 per 1000 in different studies (Hardy 1981, Levine et al. 1984, Jackson et al. 1988, Bhat et al. 1995, Evans-Jones et al. 2003, Dahlin et al. 2007). Previously full recovery in BPBI has been reported in 13-80% of cases (Wickström et al. 1955, Gordon et al. 1973, Evans-Jones et al. 2003). In our population-based data, the incidence of BPBI was found to be 3.1 per 1000 births, which is in line with previous Scandinavian studies (Sjöberg et al. 1988, Walle and Hartikainen-Sorri 1993, Dahlin et al. 2007). Full clinical recovery took place within the first year of life in 80% of our patients. Thus, the incidence of permanent BPBI in Helsinki is 0.64 per 1000.

Early detection of posterior subluxation of the humeral head

In brachial plexus birth injury, the imbalance of the developing muscle leads to progressive posterior subluxation. A humeral head that is too dorsally located further limits external rotation and reduces
the function of the glenohumeral joint. The glenoid surface becomes first flat and then slopes posteriorly, resulting in progressive glenohumeral deformity (Pearl and Edgerton 1998). However, the exact timing of this process has not been studied in depth and the age at which pathological changes of the shoulder occur is unknown.

In evaluating and screening instability of the hips, US is a commonly used method (Holen et al. 1994, Holen et al. 1999, Harcke and Grissom 1990). Despite shoulder problems being the most common permanent sequelae in BPBI, US screening of shoulder joints in BPBI patients is infrequently performed.

Hunter et al. used the posterior approach with US to detect posterior instability in BPBI patients (Hunter et al. 1998). Moukoko et al. changed their technique to a posterior US approach after realizing that the ossification center obscured the view of the glenoid in the lateral US approach technique (Moukoko et al. 2004). We studied in a 4-year prospective study the time frame of development of shoulder pathology in BPBI using posterior approach US in the City of Helsinki and in BPBI patients referred from the tertiary catchment area.

In a North American study, posterior shoulder dislocation was detected by US at a mean age of 6 months (range 3-10 months) (Moukoko et al. 2004). Among Swedish BPBI children posterior shoulder dislocation was clinically observed between 4 and 12 months of age (median 6.5 months) (Dahlin et al. 2007). We believe, that shoulder instability can be detected earlier using US than by clinical means only. Shoulder instability can be detected earlier with ultrasound since over half of our patients with posterior subluxation were diagnosed already at 3 months of age.

Moukoko et al. calculated the risk of posterior dislocation of the humeral head during the first year of life in patients born with BPBI to be 8% in North America (Moukoko et al. 2004). In a population-based study Dahlin et al. found a 7.3% early incidence of posterior shoulder dislocation in BPBI in Sweden (Dahlin et al. 2007). Our population-based study in Helsinki gave very similar findings: 6.8% of all neonates born with BPBI had sonographically-verified posterior subluxation of the humeral head during the first year of age. Shoulder instability developed only in patients with permanent BPBI, affecting one-third of these children. Among patients referred from the tertiary catchment area to our hospital, the rate of posterior subluxation was even higher. 19 of 21 referred patients still had BPBI symptoms at the age of 1 year; in other words, they had permanent BPBI. 10 patients of these 19 (53%) had posterior subluxation, but this is because only the severe
cases were sent to our hospital. The referred patients were not included in the incidence
calculation, so they did not bias the result.

The best clinical indicator for posterior shoulder subluxation is the loss of external rotation of the
shoulder (Moukoko et al. 2004). External rotation of the GH joint was restricted in all our patients
with posterior subluxation of the humeral head. 10 of the 31 patients with restricted passive
external rotation maintained glenohumeral congruency during the study period. US was a useful
screening method that allowed detection of subluxation of the humeral head in 3 out of 19 patients,
not depicted clinically. Based on our results US should be performed only if BPBI symptoms
persist because patients with temporary palsy did not develop shoulder instability.

Hypoplasia of the humeral head and shoulder deformities result from permanent BPBI due to
growth disturbances (Pollock and Reed 1989). In our study, the size of the affected humeral head
was smaller in all patients with permanent BPBI. Mean size difference was most marked in
patients with instability of the shoulder. The size of the humeral head was 7% smaller and the size
of the ossification center 18% smaller on the affected side at an age of 1 year. The bar graphs
(Figures 10b and 11b) for the patients who, participated in all the scheduled US studies show that
the excluded patients did not bias the results because the bar graphs do not differ substantially
from the graphs with all the studied patients included. Most common cause not participate in all
US studies was exclusion due to full clinical recovery (= 93 patients).

The etiology of displacement of the humeral head in BPBI has been widely discussed. The theory
that the humeral head dislocates during delivery at the time that the brachial plexus injury occurs,
and then muscle imbalance maintains the dislocation is supported by Dunkerton (Dunkerton 1989).
Gilbert and Waters et al. presented that the muscle imbalance after BPBI leads to posterior
subluxation or dislocation of the humeral head (Gilbert 1993, Waters et al. 1998). According to
our findings, US examination of the glenohumeral joint was normal at the age of 1 month in all
patients evaluated. This supports the hypothesis that muscle imbalance instead of birth trauma
underlies glenoid retroversion and posterior subluxation.

US is a fast and reliable tool for diagnosing posterior subluxation of the humeral head. In
permanent BPBI there is a high risk for shoulder instability during the first year of life. Over half
of the posterior subluxations were detected at 3 months of age and 89% were detected at 6 months
of age in our population-based study. In our whole data set, 58% of posterior subluxations were
diagnosed with US by 3 months of age and 84% by 6 months. The timing of these screening exams resembles recommendations for US screening for hip dysplasia. Early detection of posterior subluxation allows the possibility of early treatment with botulinum toxin and planning of operative shoulder relocation if needed.

**MRI of muscle and articular changes in late sequelae in permanent BPBI**

In permanent BPBI neural damage causes changes in the affected muscle anatomy and function (Zancolli and Zancolli 2000). Muscle dysfunction and imbalance results in progressive loss of external rotation and abduction and allows malformation of the bony structures. Bony deformities with increased glenoid retroversion allow posterior subluxation of the humeral head (Waters et al. 1998). However, the underlying muscle pathology in BPBI has not been properly evaluated using modern imaging. MRI is the method of choice for evaluating denervated muscles (Fleckenstein et al. 1993), it can provide precious information about the type and distribution of the primary neural injury. So far reports of imaging findings have concentrated on the injury in osseous and cartilaginous structures in the shoulder girdle. To our knowledge, BPBI retarded muscle pathology of the arm or forearm has not been elucidated previously with MRI.

**Shoulder girdle**

In a study of a healthy pediatric population Mintzer et al. imaged 111 normal shoulders in order to verify the values for normal glenoid version. The retroversion of the glenoid was biggest during the first 2 years of life (-6.3° ± 6.5°), and decreased to 2.1° ± 5.9° among children after 2 years and to −1.7° ± 6.4° among children older than 8 years (Minzer et al 1996).

Insufficient recovery of the damaged nerves results in permanent muscle changes including increased muscle atrophy and intramuscular fatty degeneration (Fleckenstein et al. 1993). To our knowledge, our study was the first to show that atrophy of the subscapular muscle was most evident and correlated with increased posterior subluxation of the GH joint. After a subscapular release operation, these changes became more evident, obviously due to permanent damage to the muscle. Degeneration of the subscapular muscle seems to contribute to the development of glenoscapular deformity. Distinct atrophy of the infraspinous muscle was also seen. Kozin emphasized the denervation of infraspinous and teres minor muscles (Kozin 2004). However, our study shows that all the rotator cuff muscles were affected. The normal function of the subscapular and infraspinous muscles as agonist/antagonist is disturbed due to muscle imbalance, resulting in a restricted range of motion of the GH joint. Additionally atrophy of the deltoid muscle was observed. It could not be
quantified in the present study because the greatest cross-sectional area of the muscle was not visualized on the images focused on the GH joint.

In our study, there was also a correlation between GSA and thickness of the studied muscles. The correlation was most obvious between GSA and subscapular muscle and its ratio (Table 7). Our results are confirmed in a recent study by Hogendoorn et al. who reported that BPBI-associated muscle degeneration was most prominent in subscapular muscle. They measured diameters of the affected muscle and compared them to the unaffected side (Hogendoorn et al. 2010). Additionally, they scored the deltoid, infraspinous, supraspinous and subscapular muscle atrophy with a 3-point visual scale. In 55% of their 102 patients subscapular muscle was atrophic with fatty degeneration or fibrosis, and in 87% of the studied children the subscapular muscle was either atrophic or atrophic with fatty degeneration (ibid).

In the present study, the thickness of the affected subscapular muscle was 69.4% of the unaffected muscles among patients without prior subscapular release. In all studied patients the subscapular muscle atrophy was most often classified as severe. A clear correlation was seen between the GSA and PHHA, showing the degree of subluxation. GSA ranged widely on the affected side (-60º to 0º) while little variability was seen (-9º to 1º) at the healthy side. An accurate level for the image positioning is essential in order to obtain exact measurements of the glenoscapular angle.

Our results are in line with newly published study by van Gelein Vitringa et al. (van Gelein Vitringa et al. 2010). In a retrospective study they reported MRI findings of 36 infants with BPBI at less than 12 months of age (mean age 4.8 months). They measured the thicknesses and segmental volumes of subscapular, infraspinous and deltoid muscles and analysed the relation between muscle ratios and GSA, subluxation and PER. They expected to find atrophy especially in the infraspinous muscle and to a lesser extent in the subscapular muscle, but the study found the most severe atrophy for the subscapular muscle. The volume of the affected subscapular muscle was only 64% and its thickness only 79% of the corresponding values on the unaffected muscle. For the infraspinatus muscle the corresponding values were 74% and 83%, respectively. They observed subluxation to correlate to and GSA as well as PER (van Gelein Vitringa et al. 2010).

Our results are interesting, because subscapular muscle is, at first clinically thought to be the least affected muscle, while paralyzed external rotators keep the arm internally rotated. Einarsson et al. have recently reported increased mechanical stiffness and a reduction in a sarcomere length in
muscle biopsies they took during open surgery of BPBI children with internal rotation contracture (Einarsson et al. 2008). They concluded that insufficient passive stretching of the muscle probably leads to changes in the extracellular matrix resulting in a dynamic feedback system to the sarcomere level (ibid).

In our study PER correlated with ratios of all the studied muscle thicknesses. In contrast to our study, van Gelein Vitringa et al. found no relation between muscle atrophy and external rotation (van Gelein Vitringa et al. 2010). They suggested that inaccurate measurement of the PER for the non-sedated infants in their study could be explanation. A more probable explanation is that contractures need time to develop and in their study all the children were younger than 12 months, while in our study the average age of the patients was 7.7 years.

In an earlier prospective study van Gelein Viringa et al. confirmed our findings that glenoid deformity was related to severe infraspinatus atrophy and subluxation of the humeral head was related to both infraspinatus and subscapularis atrophy (van Gelein Vitringa et al. 2009). Our study was the first to show the relation between imaging findings of muscle atrophy and glenohumeral deformity associated with BPBI in children with a mean age of 7.7 years. Van Gelein Vitringa et al. showed that that this relation already existed at a mean age of 3.3 years in 24 studied children. In this study they measured the thickness and segmental volume of subscapular, infraspinous and deltoid muscles. They did not find any relation between PER and muscle atrophy either (ibid). In the study performed by Kozin, progressive loss of external rotation beyond neutral correlated both with increased glenoid retroversion and posterior subluxation of the humeral head (Kozin 2004).

In the two studies published by van Gelein Vitringa et al. both shoulders were imaged at the same time in axial plane, there after thickness and the segmental volume of the studied muscles were measured from axial images (van Gelein Vitringa et al. 2009 and 2010). In our study both shoulders were imaged separately with a surface coil and the thickness measurements were performed from oblique sagittal images. When shoulders are imaged separately, the imaging planes can be adjusted to the possible three dimensional rotation of the scapula allowing more precise measurements. It is not possible when both shoulders are imaged simultaneously. On the other hand, among our patients the fatty infiltration of the muscles made the margins of the muscle very lacy, so thickness measurement was chosen to simplify the measurement. Although measurement of the area and calculation of the segmental volume gives a more precise approximation of the muscles it does not
completely characterize whole muscle either. Further studies are needed to get a complete assessment of muscle atrophy and related features.

Glenoid deformity with increased retroversion leads to posterior subluxation of the humeral head, followed by a diminished range of motion of the GH joint because of abnormal momentum of the muscles of the rotator cuff. The multiplanar imaging capability of MRI allowed evaluation of both GH joint deformity and atrophy of the muscles around the shoulder joint. The present study provides new information about the degree of muscle atrophy in BPBI and verified its correlation with the development of secondary deformation of the GH joint. Defining muscle changes associated with shoulder deformation will help to understand pathogenesis. A thorough assessment of the shoulder joint is mandatory preoperatively before any operative treatment aimed to improve shoulder function in BPBI. Correctly timed operative treatment may prevent development of more severe sequelae.

*Elbow and forearm*

Elbow flexion contracture may be caused by deformation of the elbow joint or muscle imbalance or combination of these two. It is a common finding in permanent BPBI (*Hoffer and Phipps 2000*). One obvious reason for extension deficit of elbow in our study was elbow joint incongruency detected clinically or on MRI in 4 patients out of 15. There was a discrepancy between clinical findings and interpretation of the MRI in two patients with radial head subluxation. A possible explanation for MR to miss subluxation is that MR imaging captures a static view of the joint while clinical diagnosis of the subluxation of the radial head is made when moving the elbow. Elbow flexion contracture is most probably caused by atrophy of brachial muscle. Brachial and supinator muscles were affected in all our patients. The biceps brachii was better preserved, only seven patients showed degenerative changes in MRI. The further distally situated motor points for supinator and brachial muscles may demand longer time for reinnervation than for biceps brachii (*Kawai 2000*). Hoeksma et al. had similar results and reported that supination was the last movement to recover (*Hoeksma et al. 2004*).

Permanent injury of the entire plexus results in significant deterioration of forearm function. However, in our study fatty degeneration and size reduction of the supinator muscle were also seen in all patients with upper plexus lesions and extension deficits of the elbow. This may suggest that the supinator muscle, which is the most distal target muscle receiving innervation from C5 and C6 roots, is substantially affected in all patients with permanent BPBI. The longer the reinnervation
time of the muscle is, the worse the pathological changes are in the muscle. Irreversible atrophy of the supinator muscle seems to take place before the muscle is reinnervated. An unaffected pronator teres warranted better preserved TAM of the forearm. This underlines the significance of the balanced function of agonist and antagonist muscles in maintaining an optimal range of motion. The supinator function of the biceps brachii can probably compensate for the deficient supinator muscle in BPBI patients with an unaffected pronator teres, and thus preserve a sufficient range of prosupination.

Supinator atrophy is rarely seen in the pediatric population. In the present study every BPBI patient with elbow flexion contracture had significant atrophy of the supinator muscle. TAM did not correlate with supinator pathology or brachial pathology, because all patients had changes in the above-mentioned muscles innervated by C5-6 roots. Swelling and edema of the supinator muscle detected by MRI has been reported in radial tunnel syndrome (Bordalo-Rodriques and Rosenberg 2004). In cases of early or subacute denervation, hyperintensity is seen on STIR images due to increased water content of the muscle (Fleckenstein et al. 1993). These findings may disappear, in contrast to our irreversible findings when end-stage muscle changes have already occurred. When there is chronic denervation of the muscles, fatty infiltration and size reduction are visible on T1-weighted images.

Based on our findings we present a suggested pathomechanism for BPBI. The neural injury launches the cascade: changes in target muscles lead to diminished muscle amplitude followed by muscle function imbalance of the upper limb, resulting in permanent joint deformity with incongruence and instability. In this suggested pathomechanism, there can be a feedback cycle in which diminished muscle amplitude increases changes in target muscle structure and the already developed permanent joint deformity and joint incongruence/instability enhance each other.

Previously, upper limb pathology in BPBI have been characterized principally by clinical and radiographic methods. In addition to the anatomy of the growing joints, MRI can be used to reliably visualize muscular pathology. Permanent muscular changes are often found behind diminished range of elbow and forearm motion in BPBI. MR imaging can visualize these muscle changes which lead to functional impairment of the elbow and forearm in permanent BPBI. MRI is a good diagnostic tool for revealing fatty infiltration and atrophy of the supinator muscle in permanent BPBI patients. MRI studies could thus serve as objective medicolegal tools.
Imaging findings after treatment of BPBI

Patients in this study have great variability in the extent of their injuries (C5-C6, C5-C7, C5-T1) and they have undergone various secondary surgical treatments. We wanted to examine this complex field to help to choose the best of the different operation techniques for future patients.

Maintenance or restoration of glenohumeral congruency is the goal of the shoulder sequelae treatment in BPBI. The remodeling capacity of the growth plates has an influence on the final outcome after the relocation operation. In a recent study El Gammal et al. have suggested that glenohumeral remodeling capacity decreases after 4 years of age (El Gammal et al. 2006). This observation is in line with the correction of acetabular dysplasia. After 4 years of age, the remodeling capacity of the treated congenital dislocation of the hip is clearly diminished (Kasser et al. 1985, Lalonde et al. 2002). The above suggestion is supported by our study. Two of our patients who were at age of 6.9 and 7.7 years at the time of their operations, failed to preserve the relocation and developed resubluxation without glenohumeral remodeling. These patients should have been treated by osteotomy, evaluating the situation retrospectively. The immobilization time for our third patient with resubluxation was too short.

For a congruent glenohumeral joint with an internal rotation contracture of the shoulder, soft-tissue procedures alone should be sufficient to improve external rotation (Jellicoe and Parsons 2008). We reached the same conclusion: in our study, absent passive external rotation of the shoulder was treated with subscapular tendon lengthening in congruent joints. Poor active external rotation in shoulder abduction or poor active abduction were indications for TM transposition in order to improve hand to neck movement in patients with a congruent glenohumeral joint.

Tendon transfers have been suggested for preventing the development of shoulder deformity. After latissimus dorsi and teres major tendon transfers to the rotator cuff combined with appropriate extra-articular musculotendinous lengthenings, Waters et al. have shown scarce improvement in glenoid version and glenohumeral congruency (Waters et al. 2005). Mintzer et al concluded that glenoid version may diminish about 6° in childhood during the 10 first years (Mintzer et al. 1996). Thus minor changes in glenoid version in patients who have undergone corrective soft-tissue procedures may be due to normal growth. In a 1-year follow-up study performed by Kozin et al. functional outcome improved without improvement of glenoid version or congruency of the GHJ in 23 children after latissimus dorsi and teres major tendon transfers, determined both clinically and
with MRI study (Kozin et al. 2006). Our results are in line with these findings; retroversion of the glenoid improved only after relocation of the humeral head.

Clinically it is not easy to determine when decreased external rotation is a result of muscle-related internal rotation contracture or GHJ incongruence. In the study of Kon et al. internal rotation contracture correlated with intraoperative arthrography findings of consistent patterns of deformity of the GHJ in BPBI (Kon et al. 2004). They proposed arthrography or MRI for the assessment of glenoid deformity in order to help in surgical planning. Torode and Donnan recommend CT scanning to detect potential posterior dislocation for all children with BPBI and restricted external rotation (Torode and Donnan 1998). The preoperative investigations are essential in planning an optimal surgical strategy, but we advocate MRI instead of CT; MRI has an exceptional capacity to visualize cartilage, muscle, and bony structures without radiation. Waters et al. emphasize the role of MRI assessment in determination of the need for and timing of surgical treatment, because no significant correlation existed between physical examination and glenohumeral deformity in their study of 74 BPBI children with an age range of 0.6 to 6.2 years (Waters et al. 2009).

The choice of the surgical technique is based on the congruency of the shoulder joint, age of the patient, presence of internal rotation contracture and strength of external rotators and abductors of the shoulder. According to our results, glenohumeral congruence can be improved only after a properly performed and timed relocation of the humeral head.

**Role of imaging in BPBI**

In the diagnosis and follow-up of BPBI and its sequelae, several imaging methods have proved to be important. US reveals the posterior subluxation of the humeral head at an early stage before permanent deformities have developed and enables early treatment. MRI using 3D heavily T2 weighted sequences is superior in detecting preganglionic root avulsions, valuable information needed for surgical planning (Vargas et al. 2010). Furthermore, MRI is needed for preoperative planning of the shoulder girdle. Selection of different surgical operation types is based on the congruence of the glenohumeral joint, degree of glenoid version and the condition of the muscles. This information is often difficult to obtain clinically. MR can differentiate glenoid retroversion, posterior subluxation of the humeral head, joint deformity, or muscle atrophy as possible factors resulting in decreased external rotation. Elbow joint incongruence, muscle atrophy and intramuscular fatty infiltration are detectable with MRI when clarifying the reason for elbow flexion contracture.
Possible remodelation of the glenoid can be visualized with MRI during postoperative follow up. MRI has definite potential in the evaluation of brachial plexus injuries in the future. Diffusion-weighted (DW) neurography and fiber tracking (tractography) have shown promise in imaging of the brachial plexus (Vargas et al. 2010, Tagliafico et al. 2011).

Methodological considerations
As this was a clinical study, a prospective observational approach was chosen. Due to the clinical setting it was impossible to use a double-blind study design in the dynamic US study. The clinical setting resulted in other limitations to this prospective study as well. Some neonates missed the scheduled US study either because of infections or insufficient co-operation of the guardians, especially patients with a long journey from the tertiary catchment area. However, patients were excluded mostly because of full recovery.

The study design also included patients referred from the tertiary catchment area for US analysis. Although this patient group was not included in the incidence calculation, they did not disturb the diagnostic methodology or the primary goal of determining the usefulness of US as a diagnostic tool. These patients were included because they did not influence to diagnostic accuracy of the US.

In the US study the healthy humeral head served as a healthy control, decreasing the influence of interindividual variation.

The two-day interval between US studies was regarded to be sufficient to exclude growth of the ossification center between measurements done in order to calculate intra- and interobserver variation.

The same pediatric radiologist performed most of the US studies, but because of the slow learning curve in this relatively rare disorder, this is probably not a disadvantage. US findings were not confirmed with any other imaging modality. However, it would have been unethical to expose children to the radiation of CT study, or the sedation necessary for MRI.

In our study concerning muscle pathology of the arm and forearm the group size was very small. Operative procedures on the elbow region in BPBI are relatively seldom performed and as this was a clinical study the patients were referred to imaging in order to assess the need for possible
surgical treatment. Although the group was small, it gives a good understanding of the imaging findings of 15 consecutive patients with extension deficit of the elbow. Two radiologists with extensive experience in musculoskeletal radiology blinded to clinical history and findings interpreted the MRI images by consensus.

There are relatively small numbers of patients in different groups in our prospective follow-up study after surgical treatment. Nevertheless, several secondary surgical approaches are necessary in this complex field of late sequelae after nerve injury. We believe that all possible evidence on sequelae is valuable for the care of the patients with BPBI.

**Implications for possibly future studies**

During recent years MRI sequences have advanced a lot, reaching 0.5 mm slice thickness with a good visualization of nerve roots. Further studies are needed to establish the role of MRI in the imaging of the nerve roots.

When the US studies were carried out, information about botulinum toxin treatment was also collected. The aim of the botulinum toxin treatment is to achieve equilibrium between the power of affected and unaffected muscles while waiting for recovery of the affected muscles (Rollnik et al. 2000) and thus inhibit the development of the posterior subluxation of the humeral head. Further research is warranted to determine the outcome for BPBI patients with botulinum toxin-treated posterior subluxation.

The mean follow-up time of 3.8 years after operational treatment is relatively short. There is concern about the loss of clinical improvements during a longer follow-up: loss of abduction in a longer term follow-up after latissimus dorsi transfer was reported by Pagnotta et al. (Pagnotta et al. 2004). Kirkos et al. reported in a mean 30 year-long follow-up study loss of operatively-gained active external rotation, which had been achieved with anterior release and tendon transfer of teres major and latissimus dorsi (Kirkos et al. 2005). A long term follow-up of our patients, at least to the end of the growing period, could provide more information to scientific data to test their results.
CONCLUSIONS

I: US screening for GH joint instability in BPBI
In permanent BPBI the risk for shoulder instability is 30% during the first year of life. US of the glenohumeral joint should be performed at 3 and 6 months of age in infants with persisting symptoms of BPBI.

II: MRI of rotator cuff muscle changes related to GH joint pathology in BPBI
All rotator cuff muscles, especially the subscapular muscle were atrophic in BPBI patients with internal rotation contracture or a lack of active external rotation of the shoulder joint. Muscle imbalance of the shoulder muscles results in progressive glenoid retroversion, subluxation of the humeral head and internal rotation contracture.

III: Muscle changes in BPBI with elbow flexion contracture
Elbow flexion contracture is mainly caused by brachialis muscle pathology. Fatty infiltration and size reduction of the supinator muscle occurred in all of the studied patients. Prosupination of the forearm is better preserved when the pronator teres is not severely affected.

IV: MRI evaluation of surgically treated shoulder girdle in BPBI
Remodelling of the glenohumeral joint can be achieved after successful relocation of the humeral head in patients younger than 5 years. Functional improvement of the shoulder was also achieved with rotation ostetomy of the humerus, subscapular tendon lengthening and teres major transposition operations.
ACKNOWLEDGEMENTS

This study was carried out during the years 2002-2010 in the Department of Pediatric Radiology, at Helsinki Medical Imaging Center, University of Helsinki, in cooperation with the Department of Surgery, Hospital for Children and Adolescents, Helsinki University Central Hospital.

I would like to express my gratitude to Professor Emerita Leena Kivisaari, acting Professor Taina Autti, acting Professor Nina Lundbom, Professor Risto Rintala, Docent Pekka Tervahartiala, Medical Director of the Helsinki Medical Imaging Center, Jyrki Putkonen, CEO of Helsinki Medical Imaging Center, Docent Jari Petäjä, Head of the Hospital for Children and Adolescents for providing excellent research facilities.

I am most grateful to my supervisor Docent Antti Lamminen for sharing his extensive knowledge in the field of musculoskeletal radiology and for gentle guidance and encouragement during this project. I owe my warm gratitude to my supervisor Docent Jari Peltonen for enthusiastic and constant support during the years of research.

My deepest thanks go to Docent Yrjänä Nietosvaara, who taught me the principles of hand surgery in BPBI and contributed to all levels of this study: study design, clinical studies, data acquisition and interpretation, manuscript drafting and revision for intellectual content as well as surgical procedures performed. This study would not have started nor would it have ever been finished without him. It has been a privilege and a great pleasure to work with him.

I am sincerely grateful to all my co-authors: PhD Mikko Kirjavainen, Docent Mika Koivikko, Docent Ville Remes and PT Patrick Willamo.

I wish to thank the official reviewers, Docent Kimmo Mattila and Docent Timo Hurme for their valuable comments and constructive advice after their thorough evaluation of the manuscript.

I have the honor to work with the best pediatric radiologists. I wish to express my warm thanks to my nearest chief, Docent Kirsi Lauerma, for creating a positive atmosphere and allowing the opportunity to take the time necessary to do the research. I thank my colleagues Anna Föhr, Miia Holmström, Teija Kalajoki-Helmiö, Reetta Kivisaari, Laura Martelius, Liisa Mäkinen, Kaija
Niskanen, Sakari Mikkola, Raija Seuri and Sanna Toiviainen-Salo from the Department of Pediatric Radiology for their kind collaboration. Kiri and Kaija are also acknowledged for kindly taking part in the intra-and interobserver measurements.

I wish to extend my warm thanks to Eila Syrjänen and Birgitta Palomäki-Salminen from the Department of Surgery for their excellent collaboration, Raija Äkerblom from the library of the Hospital for Children and Adolescents for help in searching for old articles, Helena Lustig for advice with computer problems, and the staff of the Pediatric Radiology Department for their positive attitude towards the present work.

Mothers who voluntarily brought their newborns for repeated ultrasound measurements for the sake of science are greatly acknowledged.

I thank all my friends for their friendship, sharing and refreshing moments: those who have been there for decades, and those whom I have come to know during the recent busy years. I am also delighted with the companionship of all the gorgeous women in Women-web.

My whole-hearted thanks go to my mother and late father for lifelong love and caring and to my precious brother and his family for being there.

I owe this book to Docent Reino Pöyhiä, my Love and Best Friend. As a husband you have always encouraged and supported me. With your energy, sense of humour, music and gourmet food you have kept my spirits high. I am also grateful for your help with the computer and statistics whenever needed. We are blessed with two brilliant children: Aino-Maria and Sakari – my beloved ones– you are my joy, you brighten my days!

Financial support by the Pehr Oscar Klingendahl Foundation, the Maud Kuistila Foundation, the Radiological Society of Finland, the Foundation for Pediatric Research, Päivikki and Sakari Sohlberg Foundation, The Finnish Medical Society Duodecim, HUS Röntgen EVO and EVO of the Hospital District (HUS) is greatly appreciated.

Helsinki, April 2011, SDG
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