Biceps muscle trauma at birth with pseudotumour formation

A CAUSE OF POOR ELBOW FLEXION AND SUPINATION IN BIRTH LESIONS OF THE BRACHIAL PLEXUS

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We retrospectively studied the possibility that direct trauma to the biceps muscle might be the cause of poor elbow flexion and supination in 18 consecutive children with birth lesions of the brachial plexus who had delayed or impaired biceps recovery despite neurophysiological evidence of reinnervation. All had good shoulder and hand function at three months of age. Eight recovered a strong biceps after six months, but nine required a pectoralis minor to biceps transfer to augment elbow flexion and supination. One had a delayed but good recovery of the biceps after microsurgical reconstruction of the plexus. All had a clinical ‘pseudotumour’ in the biceps muscle, which was biopsied during pectoralis minor transfer in two patients and showed rupture and degeneration of muscle fibres with a fibro-fatty infiltrate, suggesting previous muscle trauma.

Direct muscle trauma is an uncommon but important cause of delayed or impaired biceps recovery after brachial plexus birth injuries. Surgery to reinnervate the biceps muscle will not work if substantial muscle damage is present when a suitable muscle transfer should be considered.

The recovery of biceps muscle function by three months of age is an important prognostic sign for good recovery in birth lesions of the brachial plexus. Many recommend exploration and reconstruction of the plexus if there is no recovery of elbow flexion by three months, but others do not, as delayed recovery of the biceps after three or even six months of age may be seen.

Instead of relying solely on the recovery of elbow flexion, we also use neurophysiological investigations (Table I) as a guide to prognosis in birth lesions of the brachial plexus when shoulder and elbow function are absent at three months of age. In one study, this method accurately predicted the severity of nerve damage in 78% of C5 lesions, 92% of C6, and 96% of C7. However, we observed that a small number of children failed to recover good elbow flexion and forearm supination despite a favourable neurophysiological investigation for C6, evidence of biceps reinnervation on serial electromyograms and good neurological recovery in the rest of the plexus.

In this retrospective study we investigated the possibility that not all cases of poor recovery of elbow flexion in birth lesions of the brachial plexus are due to poor reinnervation of the biceps, and that muscle trauma may be a factor in some cases.

Patients and Methods

Between 1997 and 2002, 720 children with birth lesions of the brachial plexus were seen at our unit. A total of 18 (2.5%) had an unexpectedly poor recovery of the biceps at three months of age, despite evidence of reinnervation on serial electromyograms and good recovery of shoulder and hand function. All were included in this study. The average birth weight was 4.6 kg (3.1 to 5.6). In 15 children there was documented evidence of a forceful delivery or shoulder dystocia. Fractures of the clavicle or humerus on the affected side were documented in four infants. There were 17 with favourable (type A or B1) (Table I) electrophysiological results for C6 and evidence of biceps reinnervation at three months of age were managed conservatively. These subsequently recovered good shoulder function with a modified Mallet score of >12 and good hand function without brachial plexus repair. One child had electrophysiological evidence of biceps reinnervation but delayed recovery of elbow flexion and supination following brachial plexus repair.

Shoulder and elbow function was assessed at a minimum age of three years (3 to 5). All data were obtained from the unit’s prospective birth lesions of the brachial plexus database. The patients were grouped according to the
classification described by Narakas:11 group I, C5-6 lesion; group II, C5-7 lesion; group III, C5-T1 lesion without Horner’s syndrome; group IV, C5-T1 lesion with Horner’s syndrome. Shoulder function was graded using Birch’s modification of the Mallet score, ranging from 4 (poor function) to 15 (near normal function).8 Elbow function was assessed using range of flexion and supination and strength of flexion according to the British Medical Research Council scale12 (Table II). All children were recalled for clinical examination of the biceps looking specifically for evidence of muscle damage and pseudotumour formation. Biopsies of the biceps muscle had been taken in two children during palliative muscle transfer for elbow flexion.

Results
In all patients repeat electrophysiological investigations were performed when biceps recovery was not seen at six months. These again showed favourable results for C6 and evidence of copious reinnervation of the biceps muscle on electromyography.

Of these 17 children, eight had delayed but useful recovery of elbow function after six months of age, but nine did not recover useful elbow function. One child had no recovery of biceps function, and in eight biceps recovery was poor (power M3 or less, with elbow flexion < 90° and < 50% of the active range of supination). Useful elbow function (power M3 or greater, elbow flexion > 90°, at least 50% active range of supination) was restored in nine children following a pectoralis minor to biceps transfer.13

One child with unfavourable electrophysiological investigations (type C) had ruptures of C5 and C6 on exploration of the plexus. Following microsurgical reconstruction with intraplexal grafting, he recovered good shoulder and elbow function but recovery of elbow function was delayed, appearing only one year after reconstruction.

All children had clinical evidence of a ‘biceps pseudotumour’ in the form of a firm, ovoid, non-tender, non-contractile swelling palpable in the belly of the biceps at the mid-arm level which became more prominent on attempting active elbow flexion (Fig. 1). This appearance may be mistaken for distal biceps tendon rupture, but the tendon was found to be intact in all patients when pectoralis minor transfer was performed. The pseudotumour was always detected later in life, from between six months and 97 months of age (mean, 36 months). In two children it was biopsied during the pectoralis minor transfer. The surrounding muscle fibres appeared healthy and innervated, but the swelling itself was pale and gritty. Microscopically, it consisted of disrupted and degenerate muscle fibres and fibro-fatty scar tissue (Fig. 2), consistent with previous muscle trauma.

Discussion
Our findings suggest that direct trauma to the biceps muscle accounts for some cases of delayed biceps recovery and for some cases of failure of nerve repair to restore biceps function. As a result, poor recovery of elbow flexion is not always an indicator of poor neurological recovery, nor is it always a reliable indication for exploration and reconstruction of the plexus. In patients where there is electrophysiological evidence of C6 recovery or biceps reinnervation but delayed or poor recovery of elbow flexion, direct muscle trauma should be considered. This occurred in 18 of 720 patients (2.5%) with birth lesions of the brachial plexus recorded in our general database between 1997 and 2002. As this study was retrospective it is possible that the incidence was higher and we may have missed cases that

<p>| Table I. Neurophysiological investigations for C6 in birth lesions of the brachial plexus (adapted from Bisinella et al9) |
|-------------------|--------------------|-----------------|-----------------|---------------------|</p>
<table>
<thead>
<tr>
<th>Type</th>
<th>Nerve action potential</th>
<th>Electromyography</th>
<th>Lesion predicted</th>
<th>Expected outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Normal</td>
<td>No spontaneous activity, reduced number of normal motor units, increasing firing rates</td>
<td>Conduction block</td>
<td>Normal biceps</td>
</tr>
<tr>
<td>B1</td>
<td>&gt; 50% amplitude of uninjured side</td>
<td>Relatively good motor unit recruitment, mixture of normal and polyphasic units suggesting collateral reinnervation</td>
<td>Axonotmesis</td>
<td>Useful biceps</td>
</tr>
<tr>
<td>B2</td>
<td>&lt; 50% amplitude of uninjured side</td>
<td>Few or absent normal motor units, collateral reinnervation</td>
<td>Neurotmesis</td>
<td>Weak biceps</td>
</tr>
<tr>
<td>C</td>
<td>Absent (present in preganglionic lesion)</td>
<td>Spontaneous activity, nascent units, poor recruitment</td>
<td>Complete rupture or avulsion</td>
<td>No spontaneous recovery</td>
</tr>
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| Table II. British Medical Research Council system for grading muscle power12 |
|-------------------|-----------------|-----------------|-----------------|---------------------|
| M0                 | No contraction                                         |
| M1                 | Flicker or trace or contraction                        |
| M2                 | Active movement, with gravity eliminated               |
| M3                 | Active movement against gravity                        |
| M4                 | Active movement against gravity and resistance         |
| M5                 | Normal power                                           |
did recover elbow flexion within three months, as we were not actively looking for this lesion at the time.

Zancolli proposed a theory of direct muscle damage to the subscapularis impairing the recovery of shoulder function in birth lesions of the brachial plexus, but this has never been proven histologically. We now have clinical and histological evidence that this occurs in the biceps muscle. It is possible that it also occurs in other muscles, and may account for unusual patterns of selective recovery of muscles innervated by a spinal nerve.

It would be reasonable to expect some degree of upper limb muscle trauma following a traumatic birth. In our series, 15 of 18 patients had a traumatic or difficult delivery. There was documented shoulder dystocia in 11, seven were assisted deliveries by forceps or Ventouse, and four had fractures of the clavicle or humerus. Unfortunately, these factors overlap with the main risk factors for birth lesions of the brachial plexus, namely high birth weight (> 4 kg), shoulder dystocia and assisted delivery, making it difficult specifically to predict biceps trauma at birth.

A muscle pseudotumour indicates muscle damage and scarring. This is seen more commonly in the sternocleidomastoid, where it may cause muscle shortening and torticollis. With a biceps pseudotumour, a flexion contracture is not seen at the elbow as the biceps is flaccid and easily overcome by gravity and a functioning triceps. The pseudotumour was initially not apparent in the atrophied and denervated muscle, but became more obvious when neurological recovery and reinnervation increased the surrounding muscle bulk and tone. In younger infants before sufficient reinnervation of the biceps muscle occurs, an ultrasound or MRI investigation might demonstrate the lesion. The pseudotumour was diagnosed clinically in all our patients. It was diagnosed very late in several in whom the diagnosis was only made when the children were recalled for this study. We think it would have been detected earlier if we had looked for it specifically, both clinically and with ultrasound and MRI.

Table III shows a suggested grading system for biceps muscle trauma. We postulate that in those with less significant trauma, spontaneous recovery of strong elbow flexion and supination will eventually occur, albeit delayed, as a greater portion of the muscle fibres are undamaged (grade I). In those with more significant trauma involving a larger volume of the muscle (grades II and III) there is poor recovery. However, this grading system was constructed retrospectively based on the elbow function before muscle transfers. A better system would be one based on objective evidence that predicts the functional outcome, such as measurement of the pseudotumour, or of the volume of muscle damage relative to undamaged muscle by ultrasound or MRI. Unfortunately, this was not undertaken in any of the children in this study, and further investigation into the use of ultrasound or MRI as a diagnostic and prognostic tool is necessary.
In conclusion, we stress that a ‘biceps pseudotumour’ is an uncommon cause of poor recovery of elbow flexion in birth lesions of the brachial plexus. We suggest that electrophysiological studies should be performed in these cases where biceps muscle recovery is absent at three months of age, or six months after plexus repair. If these show signs of reinnervation, but biceps recovery lags behind the rest of the upper limb, then the possibility of direct muscle trauma should be considered. Reinnervation of the biceps is futile if substantial muscle trauma is confirmed by the presence of a pseudotumour on clinical examination, ultrasound or MRI. If sufficient elbow flexion and supination do not recover spontaneously, a healthy muscle should be transferred to restore or augment this.

Supplementary material

A table showing details of the 18 cases of biceps pseudotumour is included with the electronic version of this article on our website at www.jbjs.org.uk

The authors thank Professor R. Birch, FRCS, of the Peripheral Nerve Injuries Unit, Royal National Orthopaedic Hospital, who contributed all the cases in this study and provided expert guidance throughout. We thank Dr J. Pringle, histopathologist, formerly of the Royal National Orthopaedic Hospital, for her help in preparing and interpreting the histological specimens. We also thank Mrs M. Taggart, Mrs D. Shah and Mr S. Patel for setting up and maintaining the PNI unit’s prospective database of patients with birth lesions of the brachial plexus, without which this study would not have been possible.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References