

## Obstetric Brachial Plexus Palsy. Diagnosis and Management Strategy

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### Introduction

Obstetric Brachial Plexus Palsy (OBPP) is not an uncommon condition. Yet it does not get the attention it deserves for various reasons. Multiple studies around the world show the incidence varies between 0.15 to 3: 1000 live births<sup>1</sup>. Smellie first described OBPP in 1764<sup>2</sup>. Duchenne in 1872 pointed to traction in the birth canal as the cause of the palsy. Erb described a similar palsy in adults in 1874 and suggested that traction or compression of the C5 and C6 roots could produce the injury<sup>3,4</sup>. Yet curiously the most common name for OBPP is Erb's palsy. In fact Erb's palsy only describes a C5,6 injury and OBPP in actual fact can encompass an entire range of injuries.

If one thinks about it, the incidence of OBPP is roughly similar to cleft lip and palate. Yet the number of therapeutic interventions done for the two conditions are markedly different. One of the reasons for the lack of attention is a dearth of surgeons interested in and trained for dealing with OBPP. However, more important is the conceptual problem. It is still firmly held by large numbers of obstetricians and paediatricians (the first line of care for an affected baby) that a conservative wait-and-watch approach along with physical therapy leads to good outcomes. While spontaneous recovery does occur in a significant proportion of babies it is not the norm - far from it - and those with more severe injuries often get delayed or no treatment leading to an inferior outcome. Reported incidence of spontaneous recovery varies from 30% to 90% in various series<sup>1,5-13</sup>.

The correct approach would be for a trained brachial plexus surgeon to see these babies early and to monitor their progress sequentially. This would enable timely and appropriate intervention and avoid suboptimal outcomes.

This article seeks to discuss a systematic approach to try and help prospective decision makers in making informed choices based on a critical review of worldwide literature.

### Aetiology

Aetiological factors are fetal and maternal as listed below;

#### Fetal Causes

1. Macrosomia
2. Abnormal presentation, especially breech
3. Especially breech

#### Maternal causes:

1. Small stature
2. Cephalopelvic disproportion
3. Prolonged 2<sup>nd</sup> stage of labour
4. Diabetes in pregnancy-leads to macrosomia
5. Primi or multiparity
6. Shoulder dystocia-this is more a result of the above, rather than a cause in itself

The most common mechanism is considered to be the abnormal pull exerted by the obstetrician in situations of shoulder dystocia. This can be manual, through vacuum extraction or with forceps. Either way it creates stress on the plexus due to a traction on the neck as the baby is being pulled out. It must be stressed here that shoulder dystocia cannot always be anticipated and when it does occur can be very dangerous to baby, mother or both. The obstetrician is not left with any choice but to pull. This should be borne in mind very carefully to avoid unfairly blaming the obstetrician for an event which is often not in their control. Obvious cephalo-pelvic disproportion can be diagnosed prior to delivery and treated with a caesarean section but, in its absence, a vaginal delivery is and ought to be the norm. In such circumstances it is impossible to predict a shoulder dystocia. Additionally, in our own experience about 40% of babies show OBPP without any history of shoulder dystocia and this too should be important to remember. There are

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many contrary arguments to this theory and medico legally it is a very difficult issue. Thatte et al have discussed the literature on alternative theories and objections in another paper in detail<sup>14</sup>.

**Pathology**

Nerve injury is caused by traction, whether by a manoeuvre by the obstetrician or intra uterine stress as the earlier narrative shows. Nerve injury has been traditionally classified on a pathologic basis, viz. Seddon’s and Sunderland’s classifications (see Table 1 below). However, a more clinically useful distinction is whether the injury is pre-ganglionic or post-ganglionic.

Essentially Pre-ganglionic injury is one that is proximal to the Dorsal Root Ganglion (DRG). Therefore, there are no nerves on the proximal or donor end of the injury. This type is also called avulsion. A post-ganglionic injury is a break at the level of the nerve roots, trunks or further distal to this level and therefore has intact donor nerves available for reconstruction. This type is also called as rupture. The treatment strategy and outcomes are very different for avulsion and rupture. This is truer of the adult injury; in infants good results can be obtained even with avulsions in selected cases.

**Seddon’s Classification:**<sup>15</sup>

1. Neuropraxia
2. Axonotomesis
3. Neurotomesis

**Table 1:** Arrow shows increasing severity of injury correlating with microanatomy. A (+) sign indicates involvement at that level, (-) sign indicates no involvement.

Sunderland Grade	Only conduction	Axon	Endoneurium	Perineurium	Epineurium	Seddon Class
I	+	-	-	-	-	Neuropraxia
II	+	+	-	-	-	Axonotomesis
III	+	+	+	-	-	
IV	+	+	+	+	-	
V	+	+	+	+	+	Neurotomesis

**Diagnosis**

A diagnosis of OBPP remains by and large a clinical one. Under ideal circumstances the baby should be seen at 1 week, 4 weeks, 8 weeks and 12 weeks of

age by the treating surgeon. It is important to note the deficits and also the evolution of recovery. In our experience earlier the spontaneous recovery, better the long term prognosis. In case the child presents late (>6 months of age), the surgeon must carefully try to ascertain the recovery from the parents and give it a timeline. This has a bearing on prognosis and therefore on the management strategy.

Main types of injury are classified by Narakas:<sup>17</sup>

1. C5,C6 or upper trunk injury or Erb Duchenne palsy
2. C5,C6,C7 or extended Erb’s palsy
3. Global palsy C5-T1 without Horner’s sign
4. Global palsy C5-T1 with Horner’s sign

Grade 4 has the worst prognosis.

**Clinical Examination**

Sensory and motor examination is important. Sensory exam is done by mild nociceptive stimuli to points located on distinct dermatomes and seeing the reaction of the child including reflex movement, trying to withdraw the limb and grimacing with or without crying.

Motor examination is divided into movements at shoulder, elbow and hand with wrist.

Quick guide for motor:

1. C5,C6 shoulder abduction, external rotation and elbow flexion
2. C7. elbow and wrist extension (wrist can have C8 component and Triceps can have C6 component)
3. C8,T1. hand function, long flexors and Extensor Digitorum Communis (EDC) is usually C8 while Abductor Digiti Minimi (ADM) may have a C8 innervation

Quick guide for Sensory:

1. C5 skin over deltoid
2. C6 Thumb and index fingers
3. C7 middle finger
4. C8 Little finger
5. T1 medial forearm

**Investigations**

1. X ray chest to assess diaphragmatic involvement

2. Routine hematologic and urologic investigations for surgical fitness.
3. X-ray of clavicle or limb if a fracture is suspected
4. MRI
5. Electrophysiology (EDX)

Although an MRI can be very informative (especially MR Neurography in a 3 Tesla machine), We do not routinely use it in OBPP due to safety concerns with anaesthesia/sedation which is needed in infants. There are centres where it is done routinely and safely and if so it will certainly add information before surgery (if needed).

Electrophysiology or EDX is however routinely and regularly done in a sequential fashion and gives the following information:

1. Status of each root commenting on pre or post ganglionic injury
2. Re-innervation and its progress, if any
3. Status of important individual muscles
4. Compound motor action potential (CMAP) of recipient and donor nerves in cases involving distal nerve transfers
5. Documenting co-contractions, especially useful if Botulinum Toxin injections are being considered.

## Strategy for Treatment

### Background

If a child has a global plexus injury, it usually requires an exploration and repair with available roots. The so-called Erb's palsy or Narakas grade I injury mainly involving C5,C6 roots is the subject of huge debate and in the Indian Scenario there are two surgical strategies in vogue. The situation is a bit akin to the dilemma faced by the Prince of Denmark, Hamlet, when he says, 'To be or not to be, that is the question'; in this instance the question is 'to operate or not to operate'. To understand this question we need to understand the reasons behind it. The main reason is 'Spontaneous Recovery', which essentially means that a child looking paralysed initially starts showing recovery of function as the weeks go by. Alain Gilbert and his student Dr. Tassin published a study of natural history of OBPP where they showed that those children who did not regain an antigravity biceps function by 3 months were likely to show very poor recovery if

conserved<sup>18,19</sup>. Waters in a review has summarised the issue with the following observation: 'Infants who recover partial antigravity upper trunk muscle strength in the first 2 months of life should have a full and complete recovery over the first 1 to 2 years of life. Infants who do not recover antigravity biceps strength by 5 to 6 months of life should have microsurgical reconstruction of the brachial plexus, as successful surgery will result in a better outcome than natural history alone. Infants with partial recovery of C5-C6-C7 antigravity strength during months 3 through 6 of life will have permanent, progressive limitations of motion and strength; they also are at risk for the development of joint contractures in the affected limb'.

The paper by Waters quoted above essential points to the same conclusion<sup>20</sup>. Many people thought the problem had been solved. However, there is increasing opinion around the world that one can wait more and 3 months is not necessarily the cut off point. Published evidence still supports the 3-month guideline and the author is in favour of it with exceptions as discussed ahead.

### Why biceps?

Recovery in the setting of an upper plexus (C5-C6) injury can be misleading. The C5C6 innervated functions are mainly shoulder abduction, shoulder external rotation and elbow flexion. The problem arises because there can be apparent recovery of some of these functions without true healing of the C5-C6 pathways. If for example there is a C5C6 rupture that is distal to the Erb's point - proximal to which the Suprascapular Nerve (SSN) arises -, then the Supraspinatus and Infraspinatus muscles supplied by that nerve will function and the shoulder can abduct and externally rotate. Similarly, Pectoralis Major which is C7 innervated can cause shoulder antepulsion, the elbow then flexes with gravity; thus giving the appearance of recovery. However, if the child does antigravity elbow flexion in elbow supination, the only muscle which can do it is the biceps (C5-C6). Positive elbow flexion means the entire neural pathway up to the entry of the Musculocutaneous nerve (MCN) into the Biceps is intact. It follows (although this is deductive logic and may not always follow) that the other pathways are intact too and therefore spontaneous recovery is likely. It must be noted that a strong Brachioradialis with intact C7 innervation can cause impressive elbow flexion especially in a baby. There are two ways to distinguish this from true biceps activity:

1. The Brachioradialis functions primarily in elbow pronation
2. If the Biceps is palpated while the baby is flexing it is not felt to be contracting.

### The Pathophysiology

Essentially what is happening is cases with neuropraxia (Sunderland Gr. I) causing palsy show early and good recovery. Cases with Sunderland Gr. II injury also are likely to show good recovery but later. It is therefore important to elicit from the parents when recovery was noted. Recovery at 3 weeks or less will result in almost complete return of function. 3 to 8 weeks also are fairly good. 8 to 12 weeks usually require secondary surgery in the form of reconstruction of shoulder function and pronosupination with later muscle transfers to address co-contractions which are higher in this group.

In cases with residual palsy at 12 weeks, full recovery in the authors' opinion is usually rare. If a contracting Biceps is not felt at 12 weeks it is time to operate. There are exceptions:

1. If a child shows a strong and visible Biceps contraction but simultaneously does not flex the elbow it can be a case of biceps-triceps co-contraction. This means the muscle is innervated well but does not function due to co-contraction with the triceps. In our institute we confirm this with the help of EDx where we document the Compound Muscle Action Potential (CMAP) of the two muscles and also document co-contraction with multichannel recording. Such cases deserve a trial of botulinum toxin to the triceps (typically also to Teres Major (TM) and Latissimus Dorsi (LD), which co-contract with the deltoid).
2. If a contracting biceps is felt with a reasonable CMAP but not yet antigravity at 3 months, for example MRC grade 2+ but not 3, then the author waits up to 6 months to see the improvement if any. Beyond 6 months it is not wise to wait in my opinion, although others may differ.

### Surgical Strategy

Surgery is of two types : Primary nerve repair and secondary surgery for subsequent deformities. This paper mainly discusses the primary surgery. Secondary issues though are mentioned briefly. Primary surgery is divided into

- a. Primary Intraplexal repair
- b. Distal nerve transfers

Intraplexal repair is still considered the standard of care. The accepted treatment in cases of rupture (post ganglionic injury) is resection of neuroma and reconstruction between healthy nerve ends with nerve grafts harvested from the Sural, Medial Cutaneous Nerve of Forearm and arm (MCNF/A), and in extremis the superficial radial nerve (in cases with 5 root rupture with good donors available even bilateral Sural plus MCNF/A is insufficient to satisfy the available nerve ends.) As mentioned earlier distal transfers are used in special circumstances.

### Available Roots and Choices

Exposure is done by our standard approach described elsewhere, however we have combined the two incisions as shown in Fig.1 and Fig.2<sup>21</sup>. The author has published on the subject before and quoting from that article the strategy is<sup>14</sup>:

Upper Plexus C5, C6 Strategy:

Both Roots available: Typically C6 to Anterior Division of upper trunk and C5 to Posterior Division of Upper Trunk.

One Root Available: Root to anterior division of upper trunk (or both divisions if really good quality with plenty of healthy axons) and XIth Nerve to SSN. Intercostal nerves (ICNs) can be used for additional neurotisation of Axillary Nerve (AXN) depending on situation and fitness<sup>22</sup>.

Global Palsy Strategy:

Four or five roots available-very rare-just direct them to respective trunks / cords.

Three Roots available: One each to medial, lateral and posterior cords. XIth to SSN depending on the patho-anatomy of the upper trunk lesion. In OBPP hand is the priority and the best root must be given to the lower trunk/medial cord

Two Roots available: Root 1 to Lower trunk /medial cord, Root 2 to Lateral cord(or shared between lateral and posterior cord) and XIth to SSN. Some authorities would prefer Root 2 to posterior division of upper trunk/posterior cord and 2 or 3 Intercostal nerves (ICN's) to biceps. Their point is getting a strong triceps and a stable shoulder with deltoid and triceps as well as the rotator cuff is important to balance a well-recovered biceps. This is logical, however, the addition

of ICN's adds to surgical time, blood loss and increases surgical risks and morbidity and needs to be considered carefully before attempting. In well-equipped centres with blood bank support, ICU for children and experienced anaesthesiologists it can be done if the child is fit.

One Root Available: Root to lower trunk/medial cord + XI<sup>th</sup> to SSN, 2 or 3 ICN's to biceps. Shoulder will need secondary transfers if possible.

Zero roots [-rare total avulsion: Opp. C7 root(Full or Posterior division) to lower trunk/Medial cord and XI<sup>th</sup> to MCN or XI<sup>th</sup> to SSN and ICN's to MCN plus one ICN to Long Head of triceps (If child fit-or stagger in a second session in a couple of months if not too fit)

In our experience OBPP is one of the indications for the use of Contralateral C7 (CC7) root to reinnervate the lower trunk with most encouraging results as per our as yet unpublished data of over 15 cases. Typically, these are children with multiple avulsions and the ruptured root appears to be very poor quality.



**Fig. 1** Original incision described by author



**Fig. 2** Modification of original incision

Available roots and XI<sup>th</sup> nerve are used for shoulder and elbow and CC7 for C8T1.

### Distal Nerve Transfers

In the Indian and South Asian scenario, especially in Narakas Gr. I the shoulder motion noted due to Pectoralis Major and the visible triceps function creates a problem when one talks of neuroma excision and counsels parents about temporary loss of visible but poor quality function. Many parents refuse permission if guarantees are not offered (they cannot be offered). In such situations the author has changed strategy to do primary distal nerve transfers<sup>23</sup>. This is perhaps the first such paper for primary distal transfers. Other papers in the field have done them either for late presentation or dissociated recovery where shoulder recovers but elbow flexion does not, etc. our experience with primary distal nerve transfers is encouraging but given a choice I will still prefer an intra plexal repair because it leaves distal transfer as a fall back option in case recovery does not progress after an intra plexal repair.

The other indication for primary distal nerve transfers is of course avulsion injury where intra plexal repair is not possible.

Typical transfers are:

1. Oberlin's transfer ulnar to Biceps<sup>24</sup>.
2. XI<sup>th</sup> nerve to Suprascapular Nerve (SSN)
3. Nerve to triceps long head to Axillary Nerve (AXN) described by Somsak from Bangkok<sup>25</sup>.

### Co-Constrictions in OBPP

Co-contractions between opposing groups of muscles remains one of the most vexed problems in OBPP with no clear cut answer. It is noted both in spontaneous recovery as well as in intra plexal repair. Basically what happens is nerves cross over during the advance of the axon repair and regeneration cone and result in simultaneous firing of opposing muscle groups.

Typical examples are:

1. Deltoid with Biceps leading to Trumpet Sign
2. Biceps with Triceps leading to lack of adequate elbow flexion
3. Deltoid with Teres Major, Pectoralis Major and Latissimus Dorsi leading to inadequate shoulder abduction

#### 4. Infraspinatus with Teres Major, Latissimus Dorsi and Pectoralis Major

These may occur singly or in combination. Co-contractions result in the typical shoulder deformity and disability consisting of:

1. Inadequate abduction
2. Inadequate External rotation
3. Internal rotation contracture

This if left unchecked leads to:

1. Glenoid retroversion
2. Posterior subluxation of Humeral head from the glenoid
3. Persistent pronation at elbow
4. Eventual Radial head dislocation with forearm deformity

Strategy consists of prevention. A separate XI<sup>th</sup> to SSN coaptation in the primary repair is very useful, as is distal transfer for biceps. If, however co-contraction is present the following strategy is used:

1. Close monitoring from infancy for early detection
2. Passive mobilisation of shoulder; both gleno humeral abduction and external rotation with shoulder adducted by the mother/father/care giver
3. Judicious use of botulinum toxin if the co contraction is unrelenting and leading to internal rotation contracture.
4. Monitoring with MRI shoulder for assessing the glenoid and Humeral head
5. If botulinum toxin fails to reverse the process, then early shoulder surgery to reverse the changes.

### Discussion

OBPP remains one of the most neglected/undertreated problems in our system. This has multiple causes. Primarily it is due to lack of diagnosis and appropriate knowledge amongst the primary care physicians. The author personally has tried to address this by presenting in meetings of Paediatricians and Obstetricians at district, state and national level. The response has been very encouraging in terms of early referral. This allows the appropriate person to evaluate the progress and step in at the right moment with no loss of time. The other issues are lack of trained surgeons who can deal

with this problem, lack of infrastructure in paediatrics and anaesthesia departments to handle very small babies undergoing major surgery and of course resistance from parents to treat surgically. Physical therapy while very vital to rehabilitation cannot substitute damaged nerves. Unfortunately parents in denial resort to continued therapy in the presence of clear cut indication for surgery and delay the repair.

The question of timing does not have a clear cut answer. There is insufficient data/evidence to support the hypothesis of wait-and-watch beyond 3-6 months. The papers by Gilbert and Waters both support this conclusion. The point is not whether the biceps will come back adequately after waiting for 9 months but whether in the long run, the limb will be a strong and useful limb reaching out in space adequately in multiple dimensions. The jury is still out on that in the absence of adequate long term data from opposing viewpoints. In the authors' opinion the 3 month and 6 month cut-offs are useful and ought to be followed based on current evidence and offered as such with full discussion of all pros and cons. In case of more severe especially global lesions, it is most desirable to do early surgery and controversy does not really exist.

A detailed strategy to reverse and treat the shoulder issues encompassing a large series is published by the author for further reference<sup>26</sup>.

Other unresolved or partially resolved problems are:

1. Radial head progressive dislocation in proximal radioulnar joint
2. Persistent pronation deformity
3. Supination deformity
4. Co-contraction of forearm muscles leading to decreased hand and wrist function, especially on the extensor aspect-tendon transfers can be attempted but give mixed results compared to those for isolated nerve palsy.
5. Treatment of the neglected plexus child presenting late with poor musculature

### Conclusions

1. Obstetric Brachial Plexus injury is eminently treatable.
2. Early referral is paramount.
3. It is desirable to follow an algorithm for deciding the need for primary surgery.
4. Delaying surgery leads to worse outcomes in

general and is preferably avoided unless the parents decline permission/consent.

5. Secondary deformities are possible and should be looked for early and treated appropriately.

### Algorithm for treatment of the new-born with OBPP

History and Clinical Examination to establish the diagnosis

Note findings serially, rapid return of various muscle groups is likely to lead to spontaneous recovery.

At 4 weeks try and slot into a Narakas grade

At 4 weeks do an electrophysiology test

If global and hand not moving start preparing for surgical repair, typically at 12 weeks with 5Kg weight and good Haemoglobin

If Narakas grade I or II then observe till 12 weeks.

Points in observation are:

- a. Antigravity biceps function
- b. Shoulder abduction
- c. Shoulder external rotation
- d. Triceps anti-gravity function

If antigravity biceps is missing repeat electrophysiology to see CMAP on biceps and document co-contractions.

Reasonable CMAP and well felt biceps, continue therapy and observe till 6 months

Poor CMAP take decision to operate the plexus

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