Chapter 9

Electromyography, nerve action potential and compound motor action potentials in OBPL

Validation in the absence of a gold standard

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Objective Obstetric Brachial Plexus Lesions (OBPLs) are caused by traction to the brachial plexus during labour. Typically, in these lesions, the nerves are usually not completely ruptured, but form a "neuroma-in-continuity". Even in the most severe OBPL lesions, at least some axons will pass through this neuroma in continuity and reach the tubes distal to the lesion site. These axons may be particularly prone to abnormal branching and misrouting which may explain the typical feature of co-contraction. An additional factor that may reduce functional regeneration is that improper central motor programming may occur. Surgery should be restricted to severe cases in which spontaneous restoration of function will not occur, i.e., in neurotmesis or root avulsions. A major problem is how to predict whether function will be best after spontaneous nerve outgrowth or after nerve reconstructive surgery. When a decision has been made to perform an early surgical exploration, what to do with the neuroma-in-continuity can be a problem. The intraoperative appraisal is difficult and depends on experience, but even in experienced hands, misjudgment can be made.

Methods We performed an observational study to assess whether early electromyography (at the age of 1 month) is able to predict severe lesions. Additionally, the value of intraoperative nerve action potential and compound motor action potentials was investigated.

Results Severe cases of OBPL can be identified at 1 month of age on the basis of clinical findings and needle electromyography of the biceps. This outcome needs independent validation, which is currently in progress. Nerve action potential and compound motor action potential recordings show statistically significant differences on the group level between avulsion, neurotmesis, axonotmesis, and normal. For the individual patient, a clinically useful cutoff point could not be found. Intraoperative nerve action potential and compound motor action potential recordings do not add to the decision making during surgery.

Conclusion The absence of a "gold standard" for the assessment of the severity of the OBPL lesion makes prognostic studies of OBPL complex. The currently available assessment strategies used to obtain the best possible solutions are discussed.

Obstetric Brachial Plexus Lesions (OBPLs) are caused by traction to the brachial plexus during labour.[1,2] In the majority of cases delivery of the upper shoulder is blocked by the mother's symphysis pubis, i.e., shoulder dystocia. If additional traction is applied to the child's head, the angle between neck and shoulder is forcefully widened, overstretching the ipsilateral brachial plexus. The incidence of OBPL varies from 0.42 to 2.9 per 1000 births in prospective studies.[3-5]

The upper brachial plexus is most commonly affected, resulting in paresis of the shoulder and biceps muscles, as first described by Erb and Duchenne. Typically, in the C5, C6 lesion type, the affected arm, when lying in rest, is held in adduction, internal rotation and extension. The wrist and fingers are continuously flexed when C7 is damaged as well. Hand function is additionally impaired in approximately 15 % of patients.[3,6,7] Isolated injury to the lower plexus (Déjèrine – Klumpke's type) is very rare.[8] The severity of the traction injury may vary from neurapraxia or axonotmesis to neurotmesis and avulsion of rootlets from the spinal cord,[9] but this can only be assessed by evaluation of recovery in the course of time, because nerve lesions of different severity initially present with the same clinical features. Neurapraxia and axonotmesis eventually result in complete recovery. Neurotmesis and root avulsion, on the other hand, result in permanent loss of arm function, and in time development of skeletal malformations, cosmetic deformities, behavioural problems and socio-economic limitations.[10-14] The prognosis of OBPL is generally considered to be good, with complete or almost complete spontaneous recovery in more than 90% of patients.[15-20] However, this opinion is based on a limited number of series[21,22], which are cited indiscriminately, and without considering the methodological aspects of these studies. Having performed a systematic literature review, we discussed possible methodological problems in the available natural history studies.[23] We found that no study presented a prospective, population-based cohort that was scored with a proper scoring system with an adequate time of follow-up. In other words, there is no scientifically sound evidence to support the common belief that recovery will be complete. Instead, analysis of the most methodologically sound studies led us to estimate the percentage of children with residual deficits to be 20% to 30%.

Quality of care of patients with OBPL requires concentration of severe cases in specialized centres, providing the opportunity to start appropriate and rigorous child-physiotherapy and/or early surgery, if necessary. However, a major problem is how to select the particular infants, shortly after birth, who will eventually comprise the aforementioned 20% to 30% with a poor prognosis. Referral of all cases to specialized centres is probably not feasible and would result in squandering of resources, as specialized treatment is not necessary in the majority of cases. Unfortunately, a satisfactory test for this selection is currently not available.

The second problem, also concerning the 20% to 30% with incomplete recovery, is how to predict whether function will be best after spontaneous nerve outgrowth or after nerve reconstructive surgery. Results achieved by surgery are claimed to be superior to the outcome in conservatively treated subjects with equally severe lesions.[24,24-26] However, this comparison has relied on historical controls,[27] and there

has been no randomized controlled study.[28,29] Stated bluntly, the question whether or not surgery improves functional outcome has, in fact, never been satisfactorily answered. In view of risks related to surgery and socio-economic factors, surgery should provide an uncontroversial benefit as compared with the natural course. Some may argue that the current standard of treatment does not necessitate such a prospective randomized trial. In general, it has been proven convincingly[30] that nerve surgery for neurotmetic lesions and root avulsions in which spontaneous restoration of function will not occur can offer functional recovery in the outflow of the reconstructed nerve elements, whereas there will be no natural recovery for these nerve elements. For each individual case, unfortunately, the functional recovery pursued by nerve reconstructive surgery cannot be guaranteed and the level of recovery is variable. This reasoning implies that surgery should be restricted to severe lesions (e.g. neurotmesis or root avulsions).[9] But, as previously noted, determining whether such lesions are present may only become apparent over time, but time cannot be wasted in OBPL, as the interval between nerve trauma and reconstruction is inversely related to the outcome of reconstruction. Early surgery is, therefore, preferred to delayed surgery. At present, the earliest accepted time at which severe lesions can be determined is 3 months of age. Paralysis of the biceps muscle at 3 months is associated with a poor prognosis[31] and is considered an indication for nerve surgery by some authors.[24,32-35] However, biceps paralysis at age 3 months does not preclude satisfactory spontaneous recovery.[25,36-38] Additionally, biceps muscle testing may not be reliable in infants.[38-40] Alternative tests[35,39,41] are complex or are done at an even later age.

When a decision has been made to perform a surgical exploration at an early age, a third problem arises, what to do with the typical finding during exploration: a neuroma-in-continuity of the superior trunk. As far as severity goes, this may represent a position intermediate between axonotmesis and neurotmesis. When the lesion is "mainly axonotmetic", surgical resection followed by nerve grafting is not indicated. But when the lesion is "mainly neurotmetic", a case may be made for resection and grafting. The differentiation between these 2 forms relies on an intraoperative appraisal. This process is difficult and depends on experience, but even in experienced hands, misjudgement can be made. Objective quantifiable parameters for an intraoperative assessment are, therefore, needed to assess the severity of a nerve lesion-in-continuity.

In the following sections we outline the difficulties that are encountered for the early selection for treatment and the intraoperative decision whether to resect a specific nerve element. A synopsis of our current understanding of the neuropathophysiology of the OBPL lesion serves as a basis. Our studies of preoperative electromyography (EMG) and of intraoperative nerve action potentials (NAPs) and compound motor action potentials (CMAPs), of which some parts have been published previously[42] are discussed. We focus on the impact of the absence of a "gold standard" for the assessment of the severity of the OBPL lesion and the currently available strategies used to obtain the best possible solutions.

The neuropathophysiological basis of the problem

In the majority of OBPL infants, those with a C5, C6 spinal nerve lesion, the damaged nerves are usually not completely ruptured in the sense that there is a gap between 2 stumps. This is most likely caused by the gradual exertion of traction forces over a small distance that acts during a relatively long period. In contrast, true rupture of nerves occurs frequently in adult traumatic brachial plexus lesions owing to the high impact of kinetic energy in a fraction of time. The two crucial factors that determine good functional recovery are: 1) the number of damaged axons that successfully elongate past the lesion site, and 2) their routing. Axonal outgrowth and restoration of connections with their original motor or sensory end-organs can take place only when the basal laminal tubes surrounding the axons – which are in this context the crucial anatomic structures – remain intact. The lesion is then qualified as being axonotmetic. The distance from the lesion site, which in OBPL almost always concerns the root and trunk levels, to the end organ determines the duration from lesion to recovery. Recovery of predominantly axonotmetic OBPLs is usually seen within the first 3 to 4 months of life. When the traction lesion is more severe, the basal laminal tubes are ruptured, but the perineurium and epineurium remain more or less intact. Outgrowing axons will then not end up directly in any tube. The lesion is then qualified as neurotmetic. Typically in OBPLs, the stretched and damaged nerve forms a neuroma-in-continuity, i.e., a tangled mass of connective scar-tissue and outgrowing, branching axons. The local environment encountered by the axonal growth cone may impede outgrowth and may ultimately block the restoration of axonal continuity.

Even in the most severe OBPL lesions, at least some axons will pass through the neuroma-in-continuity and reach the tubes in the distal to the lesion site. This may be partially attributable to the superior ability of the peripheral nervous system in infants to regenerate,[43] as compared with that in adults. The number of axons that will not pass the lesion site depends on the severity of the lesion, which is determined by the magnitude and angle of the exerted traction forces. There is a minimum in the number of axons that should reconnect with an end-organ to regain function. In addition, to regain function a minimum number of axons should find the way to their intended goal. We presume that axons in a neuroma-in-continuity in OBPL are particularly prone to abnormal branching and misrouting. Since the direction of outgrowth after severe lesions is essentially random[44], outgrowing axons growing through a neuroma-in-continuity are likely to end up in the wrong tube. Branching and misrouting can also explain co-contraction,[45] a typical feature of OBPL at a later age, in which shoulder abduction and elbow flexion, or elbow flexion and extension become irreversibly linked.

An additional factor to the inadequate number of outgrowing axons and misrouting that may reduce functional regeneration is that improper central motor programming may occur.[46] There are various reasons why the formation of motor programs may fail in OBPL. First, OBPL causes deafferentation as well as weakness; many functions in the central nervous system depend on afferent input in a specific time

window or else they are not formed correctly. Secondly, aberrant outgrowth of motor axons may present the central nervous system with conflicting information. A motor command for shoulder abduction may, for instance, cause elbow flexion in addition to abduction through misrouted motor axons. The resulting feedback may well hamper the formation of a selective abduction program, as there is probably no way for the central nervous system to identify the "misbehaving" motor units.[47,48] Thirdly, sensory axons may also be prone to misrouting, compounding the problem. A final hurdle for the central nervous system may be the severity of paresis. In such cases the only way to effect certain movements may be through "trick movements" (such as scapular rotation instead of glenohumeral rotation), which then represent a functional adaptation. Each OBPL case is unique on the axonal level in the sense that the number of ruptured axons and basal laminal tubes differ for each intraplexal involved nerve element. This subsequently leads to the wide variety in level of functional recovery that can be found in individual cases.

Electrophysiology in OBPL – known problems

EMG and prognosis

Until now, ancillary testing, in particular EMG, has not been considered reliable enough for prognostication of OBPL.[47,49] A needle EMG might seem a useful tool in this respect, but at present, its role is debated. A main reason for this is that EMG findings may be discordant with clinical findings at 3 months of age at which time the biceps test is performed.[24] In a paralytic biceps brachii muscle, the expected findings are absence of Motor Unit Potentials (MUPs) and presence of positive sharp waves and/or fibrillation potentials (so-called "denervation activity"). However, in a typical OBPL case, MUPs are present and denervation is absent in a paralytic biceps muscle at 3 months of age. This confusing finding has been noted by others,[50,51] and may have contributed to the opinion that the EMG is not useful in OBPL.[19,52] We previously outlined several possible explanations for "inactive MUPs", i.e., MUPs in a paralytic muscle.[47] These suggest that the presence of inactive MUPs may depend on time after injury, as they reflect incomplete outgrowth of damaged axons and the formation of motor programs in the central nervous system. In one study, 20 of 28 infants who had no biceps function at 3 months had developed biceps contraction at 6 months.[37] In addition, spontaneous recovery of useful extremity function has been observed in a carefully selected subset of patients without elbow flexion at three months of age.[38] Together with our findings[53] that MUPs can almost always be found in the biceps muscle at 3 months, this strongly suggests that the age of 3 months does not represent a stable state in OBPL. In fact, the outgrowing axons may well have only just arrived in the various muscles, and the central nervous system may not yet have learned to cope with the situation. In nerve lesions in adults, one may expect all motor programs to be ready and waiting for the restoration of peripheral connections. In OBPL, axonal outgrowth may only be the starting point for restoration of function, as formation of central nervous system motor programs may only commence after enough axons have arrived to start exerting force. At the same time, forming such central motor

programs may be more difficult and thus take longer than in healthy children, as the central nervous system must somehow take aberrant outgrowth and the confusing feedback it causes into account. Faced with a degree of inescapable co-contraction, it may not be easy to program effective elbow flexion, abduction or rotation. In this hypothetical view, the age of 3 months may well be the very worst period imaginable to correlate the EMG with clinical findings: it is late enough to show evidence of axonal outgrowth, but too early for the brain to control contraction efficiently. This leaves the role of the EMG for prognosis at 3 months undetermined at present.

Intra-operative NAP and CMAP recording

In adults, intraoperative recording of NAPs and CMAPs is advocated to objectively distinguish between axonotmetic and neurotmetic lesions.[54,55] It has been shown that the presence of a NAP across the lesion site requires at least 3000 to 4000 nerve fibres with a diameter of more than 5 µm. The presence of these fibres in a recovering nerve indicates that spontaneous functional recovery will take place and that, therefore, resection and grafting is not indicated.[56] In contrast to testing in adults, little is known about intraoperative neurophysiological assessment in infants, and the use of this technique is not widespread. In one study of 10 patients, a lesion-in-continuity was not resected in 5 patients, because NAP was considered to be indicative for neural continuity across the neuroma.[57] These patients, however, did not recover well, so NAPs were considered too optimistic.[57] Other authors only mentioned the use of intraoperative neurophysiological evaluation as a potential assessment tool,[58-60] but neither their methods nor the results were detailed.

We have performed 2 studies to explore the use of EMG and intraoperative NAP and CMAP measurements in patients with OBPL. The results have been published in part elsewhere.[42,53] The most important drawback of the EMG and NAP and CMAP studies we have undertaken is that a gold standard to assess the severity of the nerve lesion in OBPL does not exist. The consequences of the absence of a gold standard will be discussed to improve our understanding of the unique features of OBPL and to put the interpretation of the results of our studies in perspective.

Gold Standard

The criteria to define an OBPL lesion as neurotmesis or avulsion are missing. The ideal study design to assess the predictive values of EMG, NAP, and CMAP would theoretically be as follows. Clinical and EMG parameters should be measured in a consecutive series of all OBPL infants born during a preset period of time, in a specified area or country, at a specific age (i.e., at 1 month). Subsequently, the brachial plexus should be explored in all of these infants and NAP and CMAP values should be recorded. After collection of NAP and CMAP data, the wound should be closed without reconstruction of the damaged brachial plexus nerve elements. The spontaneous recovery of upper limb function in all of these infants should then be followed and documented until the end stage of recovery, growth and adaptation has been reached. This might

be at 16 or even 20 years of age. A thorough functional and neurological examination should then be performed.

The following step is to define 2 subgroups: one with good functional recovery and one with poor function. We realize that there is no common agreement to define a clear cutoff point between these 2 subgroups. It may even be difficult to set parameters for definition of good and poor function, given the gradual differences between the OBPL lesions of each individual that are normally encountered. Anyhow, subsequently, correlations between the preoperatively collected items at 1 month of age and the subgroups with a good or poor outcome should then be assessed with logistic regression. The predictive value of each item or the stepwise combination of items can then be used to develop a test for prognostication at the age of one month. This test can then be prospectively used to select those infants at 1 month in whom a poor outcome is predicted at 20 years of age.

The next question that needs to be addressed is whether nerve reconstructive surgery can significantly improve the function of the arm of the selected subgroup with predicted poor outcome. To answer this question, the selected group with poor outcome should be prospectively randomized in 2 groups: one that should undergo nerve surgery and reconstruction and one that should be treated conservatively. It is obvious that this ideal design is unworkable, for ethical as well as practical reasons. Be that as it may, which tests can serve as the second best alternative?

The "Leiden Gold Standard" for Validation of EMG and NAP/CMAP

Selection for surgery

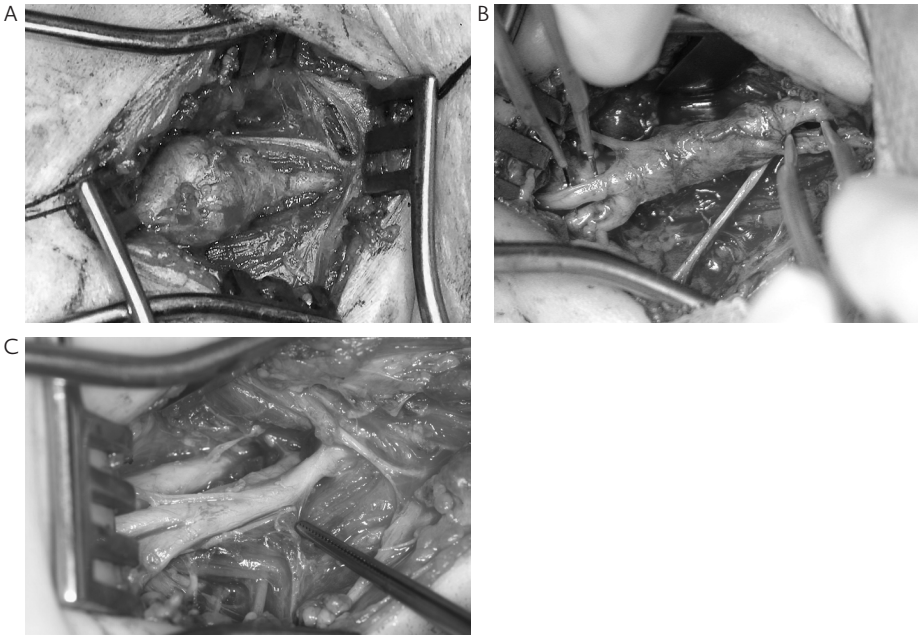
In the Leiden University Medical Center, surgery for OBPL is rarely performed before 3 months of age, mainly for anaesthesiological reasons, but it is almost always done before the age of 7 months. In selecting infants for surgery, we seek to identify all cases of neurotmesis or avulsion. Infants are selected for surgery when external shoulder rotation and elbow flexion with supination remain paralytic after a 3 to 4-months period to await spontaneous recovery. Impaired hand function is an absolute indication for nerve surgery as soon as the infant turns 3 months old.[61] If there is doubt about the quality of shoulder and elbow joint movements, surgical exploration is performed in the hope that errors would consist of not finding neurotmesis or avulsion during surgery rather than letting such lesions go without surgical treatment. Preoperative ancillary investigations in all patients consist of ultrasound of diaphragm excursions to assess phrenic nerve function and computed tomography (CT)-myelography under general anaesthesia to detect root avulsions.[62,63]

Surgical exposure and assessment of the severity of the lesion

In the vast majority of OBPL cases, supraclavicular exposure alone will suffice for a proper exposure and reconstruction. The brachial plexus is exposed in the lateral neck triangle through a straight incision parallel to the clavicle. The severity of the lesion of

each clinically involved spinal nerve is subsequently assessed. A distinction is made between axonotmesis, neurotmesis and root avulsion on the basis of: 1) inspection of the status of nerve continuity at the intraforaminal level in combination with presence or absence of root filaments on CT-myelography; 2) the extent and location of neuroma formation; and 3) selective electrical stimulation of all of the involved spinal nerves using a bipolar forceps in combination with a 2.5-Hz pulse generator with increasing voltage (maximum 6 V). (Figure 1)

Figure 1: **Supraclavicular exploration of the left brachial plexus in 3 different infants with obstetric brachial plexus lesions**



Preoperatively, the infants presented with an identical neurological picture of the left arm befitting a C5 and C6 spinal nerve lesion. Surgery was performed at the age of approximately 5 months. **A**, *diagnosis: neurotmesis superior trunk. Reconstruction: nerve grafting from C5 to suprascapular nerve, C5 to posterior division superior trunk, C6 to anterior division superior trunk.* **B**, *diagnosis: neurotmesis C5, partial neurotmesis/ axonotmesis C6, neurotmesis phrenic nerve. Reconstruction: nerve grafting from C5 to suprascapular nerve, C5 to posterior division superior trunk, C5 to C5 contribution of anterior division superior trunk, phrenic nerve to phrenic nerve. Spinal nerve C6 outflow was neurolyzed. Note the nerve action potential recording.* **C**, *breech delivery. Pseudomeningoceles of C5 and C6 on CT-myelography. Diagnosis: root avulsion of C5 and C6. Reconstruction: transfer accessory nerve to suprascapular nerve end medial pectoral nerve to half of the cross sectional area of musculocutaneous nerve. C5 and C6 spinal nerves were left in situ.*

A spinal nerve root is considered *avulsed* when the nerve at the intraforaminal and juxtaforaminal level exhibits root filaments, the dorsal root ganglion is visible, neuroma formation is absent, and there are no muscle contractions after direct stimulation. In the majority of spinal nerves, these findings correspond with the absence of root filaments, as demonstrated by CT-myelography. Avulsed roots are cut as proximally as possible. When the dorsal root ganglion can be morphologically identified, it is dissected from the ventral root and removed. After confirmation by frozen section of the presence of ganglion cells, it is certain that the distal stump consists only of the ventral root. This ventral root can be the target for nerve grafting, or the ventral root can be attached to a qualitatively good nerve stump directly without a nerve graft.

A spinal nerve is considered *neurotmetic* when the following features are present: a normal appearance at the intraforaminal level, a clear increase of the cross-sectional diameter at the juxtaforaminal level, abundant epineurial fibrosis, loss of fascicular continuity, and increased consistency and increase of the length of the nerve elements with concomitant distal displacement of the trunk divisions. Electrical stimulation of the spinal nerve proximal to the neuroma may cause weak muscle contractions that are detectable with palpation but are not strong enough to move the limb. Resection of neurotmetic tissue is performed, and the proximal and distal stumps are prepared for nerve reconstruction.

A spinal nerve is considered *axonotmetic* when neurolysis reveals no substantial increase of the cross sectional diameter, only limited epineurial fibrosis, and intact fascicular continuity. Furthermore, on C5 stimulation, abduction with movement of the limb and some external rotation should be present, and on C6 stimulation, elbow flexion against gravity with supination should be found. Axonotmetic nerves are left in situ because spontaneous nerve regeneration is in process, although as yet clinically not clearly apparent. Axonotmesis is confirmed by the occurrence of good spontaneous recovery after at least 2 years of follow-up.

The Leiden EMG and NAP/CMAP studies

We recently prospectively studied 48 infants with OBPL with a minimum follow-up of 2 years.[53] Clinical items and needle EMG of the deltoid, biceps and triceps muscles were gathered at 1 week, 1 month and 3 months of age. A poor outcome was defined as the absence of flexion and supination at 6 months of age on clinical examination and the presence of a neurotmetic lesion or avulsion of C5 and C6 at surgical exploration, irrespective of any C7-T1 lesion. A good outcome was defined as the presence of active elbow flexion and supination at 6 months of age either at clinical examination or on direct intra-operative nerve stimulation. Patients with a good outcome showed spontaneous restoration at 2 years of age of a subtotal range of elbow flexion, supination and subnormal glenohumeral abduction befitting a predominantly axonotmetic lesion of C5 and C6. Predictors for a severe lesion were identified using logistic regression analysis. We showed that severe cases of OBPL can be identified at 1 month of age on the basis of clinical findings and needle EMG of the biceps.[53] The outcome of our

EMG study is currently being validated by an independent group because of the lack of a gold standard.

Indeed, the key features in our study that eventually determined the severity of the lesion, namely the clinical picture, the results of CT-myelography and the surgical findings, are the same factors that define the pre-test probability of a severe lesion in the normal clinical decision process. The consequence of the absence of a gold standard is that the diagnosis in our studies and, therefore, the calculated correlations, may not have been correct. Independent validation is, therefore, required to evaluate the predictive value of the test.

In an additional study, we analyzed the results of intraoperative NAP and CMAP recordings in a consecutive series of 95 patients with OBPL (mean age 175 days) to assess the predictive values for the diagnosis of axonotmesis, neurotmesis, avulsion or normal spinal nerves.[42] Intraoperative NAPs and CMAPs were systematically recorded from damaged nerves and control nerves of the upper brachial plexus. A total of 599 intra-operative NAPs and 832 CMAPs were analyzed. The severity of the nerve lesion was scored for each involved spinal nerve as normal, axonotmesis, neurotmesis or root avulsion. The correlations of NAP and CMAP with the severity of the lesion were assessed. We found statistically significant differences between diagnosis groups. For the individual patient, however, a clinically useful cut-off point for NAP and CMAP recordings to differentiate between avulsion, neurotmesis, axonotmesis, and normal could not be found. The sensitivity for an absent NAP or CMAP was too low for clinical use. We concluded, therefore, that intraoperative NAP and CMAP recordings do not add to the decision making during surgery in infants with OBPL.

Conclusion

An obstetric brachial plexus lesion does not necessarily have a good prognosis: 20% to 30% of infants with this condition will have a functional disability for the rest of their lives. The level of impairment depends on the extent of the nerve lesion. Selection for nerve surgical treatment is difficult and requires experience, as does nerve reconstructive surgery. Good results with nerve reconstructive surgery have been obtained significantly improving the functional level of the arm to a level that could presumably not have been reached through spontaneous regeneration and conservative treatment. Specialized centres with a multidisciplinary approach are probably best suited for the treatment of these infants. Selection for referral to these centres and selection of lesions with neurotmesis or avulsion with the ultimate consequence of surgical treatment is difficult. In the absence of a gold standard for the assessment of neurotmetic lesions or avulsions, we apply a second-best standard that is based on surgical, clinical, histological, and radiographic criteria. In our series, histological examination of resected tissue confirmed abundant neuroma formation in all samples of assumed neurotmetic lesions. Obviously, we did not obtain histological samples from the cases of axonotmesis to serve as a control. These children were followed clinically and all showed good spontaneous recovery of elbow flexion and shoulder abduction.

In studying the value of EMG, NAP and CMAP, the consequence of the absence of a gold standard is that the key features that eventually determine the severity of the lesion (the clinical picture, the results of CT-myelography, and the surgical findings) are the same factors that define the pre-test probability of a severe lesion in the normal clinical decision process. In this aspect, our studies differ from the clinical decision process, in which the neurological examination, CT-myelography, and surgical findings are known. Ancillary investigations are expected to provide additional information.

Our studies support our current view that severe cases of OBPL can be identified at 1 month of age based on clinical findings and needle EMG of the biceps and that intraoperative NAP and CMAP recordings do not add to the decision making during surgery in these infants.

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