Part 1

Introduction

Around 1505, Leonardo da Vinci made a drawing of the anatomy of the brachial plexus based on a post-mortem dissection. At that time, it was assumed that motor nerves were minute hollow tubes that guided the 'anima' from the central nervous system to the muscle, resulting in muscle contraction.

Chapter 1

General introduction to relevant aspects of nerve lesions, with special emphasis on OBPL

The re-growth of a severed nerve is a romance. It is, perhaps the most beautiful example of Nature's power of repair, but it cannot be hastened by artificial means. It is a very delicate process, easily arrested or retarded: it follows certain natural laws, which take a long time to work out. After the surgeon has removed the obstacles, which forbid Nature to begin her beneficent work, all that he can do is stand on guard to see that nothing shall be allowed to interfere with Nature.

Sir Robert Jones 1918

The obstetric brachial plexus lesion (OBPL) is a nerve traction injury to the brachial plexus. Its severity determines whether spontaneous recovery will occur or surgical therapy is warranted. In this chapter the pathophysiology of nerve lesions and principles of surgical repair will be outlined, with a discussion of the special mechanisms that characterize OBPL compared to brachial plexus lesions in adults.

Pathophysiology of nerve lesions

A nerve consists of numerous axons which conduct the action potential. The axons are surrounded by several layers of supporting connective tissue sheaths. In the context of regeneration, the crucial structure in these layers is that which directly surrounds the axon and Schwann cells, namely the basal lamina tube. Each basal lamina tube contains one axon. The nerve micro-architecture consists of a framework of endoneurial tissue around the basal lamina tubes. The endoneurium is in turn surrounded by a perineurial sheath collecting the fibres into fascicles or fascicle bundles. The fascicles are enclosed in a connective tissue packing: the epineurium. Along the entire length of the nerve, fascicles divide and unite thereby constituting intraneural fascicular plexuses.[1] (Figure 1)

A spinal nerve is formed by ventral and dorsal rootlets originating from the spinal cord, that contain motor (ventral) and sensory (dorsal) axons. The cell body of the motor fibres is located in the grey matter of the central cord. The cell body of the sensory axons is – conversely – located outside the spinal cord in the dorsal root ganglion. The dura extends around the spinal cord to form a sleeve that extends into the bony foramen, through which the spinal nerve emerges from the spinal canal.

Mechanical lesions of the nerve can be classified in several ways. The first distinction is between a sharp lesion and a traction lesion. Sharp lesions are beyond the scope of this thesis and will, therefore, not be discussed. In a traction lesion, the damage to the constituent parts of the peripheral nerve depends on the degree of traction. The severity of the lesion determines to which extent spontaneous recovery will take

Figure 1: **Architecture of a peripheral nerve**

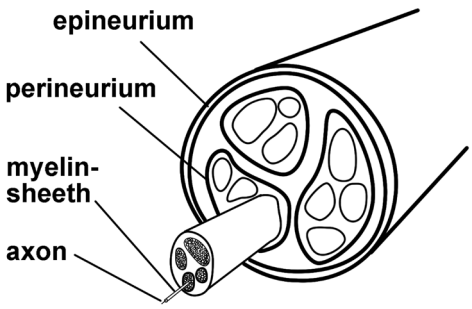


Table 1: **Seddon's classification**

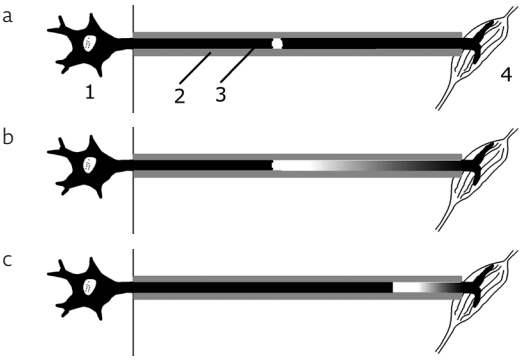
Neurapraxia	transient block: remarkably rapid recovery, which cannot possibly be explained in terms of axonal regeneration
Axonotmesis	complete interruption of nerve fibres with preservation of more or less of the supporting structure of the nerve
Neurotmesis	complete division of a nerve

place. In some cases the integrity of the distal part of the nerve will remain intact, but the interruption takes place at the junction of the central nervous system to the peripheral nerve: the nerve is ruptured from the spinal cord. This is referred to as root avulsion. The various aspects of these different lesion types will be discussed later in more detail.

If the traction lesion causes an interruption of the axon, there is a loss of continuity of the distal part of the axon to the cell body in the spinal cord (motor nerve) or in the spinal ganglion (sensory nerve). This results in structural changes of the distal nerve, which were first described by August Waller in the mid-nineteenth century.[2] The *"various alterations, as seen under the microscope, which take place in the structure of the same nerves after their continuity with the brain has been interrupted"* are usually referred to as Wallerian degeneration. In short, distal to the lesion site the content of the basal lamina tube is phagocytosed, and the Schwann cells proliferate. The proximal axons, which are still in contact with the cell body, grow sprouts towards the distal end of the nerve. The outgrowing axonal sprouts are then surrounded by Schwann cells which are mandatory for regain of function. The speed of axonal outgrowth is around 1 mm/day.

Full functional recovery is only possible when outgrowing axons are guided by basal lamina tubes to their original target organ. In the case of diastasis of basal lamina

Figure 2: **Subsequent stages in axonotmesis**



a) rupture of the axon, intact basal lamina tube; b) Wallerian degeneration distal to the axon interruption; c) axonal outgrowth through the basal lamina tube; 1 – cell body in the spinal cord; 2 – basal lamina tube; 3 – axon; 4 – muscle; changes in the cell body and muscle are not drawn

tubes at the rupture site, outgrowing axons fail to find their way, resulting in neuroma formation. In these instances, the regenerative process can only take place after neuroma resection and bridging the interstump gap by interposing a graft.[1,3]

Classification of nerve injuries

Seddon's classification

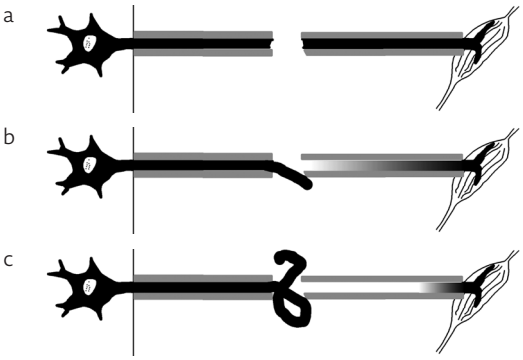
The first classification describing the severity of nerve lesions was proposed by Seddon in 1942. It is still widely used. Three levels of severity are defined: neurapraxia, axonotmesis and neurotmesis.[4] (Table 1)

This is quite a simplistic view of the different gradations of nerve lesions, which the author acknowledged himself: "*It is obvious that by no means all nerve injuries can be classified under these simple headings; the accidents that produce them are not uniform in their effects unless the violence is great. An injury may affect the fibres of a nerve in varying degree, and the clinical picture will then be more complex. A lesion may be made up by a combination of any of the following: (a) neurotmesis; (b) axonotmesis; (c) neurapraxia; (d) normal fibres.*"

In neurapraxia, the structure of the nerve has remained intact, no Wallerian degeneration will take place, and there will be complete spontaneous recovery. In axonotmesis, the severed axon will re-grow from proximal to distal, guided by the intact basal lamina tube. Eventually it will re-establish contact with its end-organ, and functional recovery will be regained. (Figure 2)

When not only the axon itself but also the basal lamina tube that guides axonal outgrowth is damaged, the outgrowing axon cannot reach the distal nerve stump, and neuroma formation occurs at the lesion site. (Figure 3)

Figure 3: **Subsequent stages in neurotmesis**

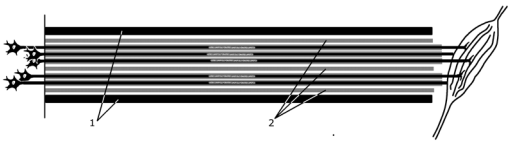


a) rupture of the axon and basal lamina tube; b) unguided axonal outgrowth; c) neuroma formation

Figure 4: **Sunderland's classification[5]**

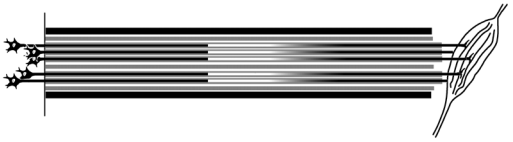
First degree injury

- conduction along the axon is interrupted at the site of the injury
- axonal continuity between the neurone and end-organ is preserved
- no Wallerian degeneration
- the disturbance is fully reversible



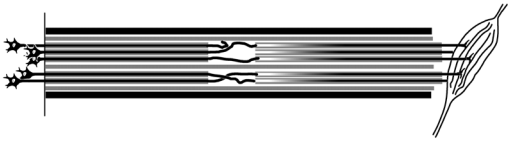
Second degree injury

- the axon is severed or axonal mechanisms are so disorganized that the axon fails to survive below the level of the injury
- the general arrangement of the axon sheaths and remaining structures is preserved
- the reaction which develops as the result of the injury and the ensuing Wallerian degeneration does not threaten the integrity of the endoneurial tubes



Third degree injury

- in addition to axonal disintegration and Wallerian degeneration, disorganization of the internal structure of the funiculi
- in the perineurium the general arrangement is retained so that the bundles remain in continuity
- a disorganization of the internal structure of the bundle in which endoneurial tube continuity is lost
- recovery in individual structures takes place more slowly and is usually incomplete, a paresis and/or sensory defect remaining



Fourth degree injury

- the entire funiculus is involved, all bundles being breached and so disorganized that they are no longer sharply demarcated from the epineurium in which they are embedded
- continuity of the nerve trunk is preserved but the involved segment is ultimately converted into a strand of tissue composed of a tangled mass of connective tissue, Schwann cells and regenerating axons which may be enlarged to form a neuroma.

Fifth degree injury

- loss of continuity of the nerve trunk which results in the complete loss of motor, sensory and sympathetic functions in the autonomous distribution of the severed nerve

1 – *epineurium*; 2 – *perineurium*

Sunderland's classification

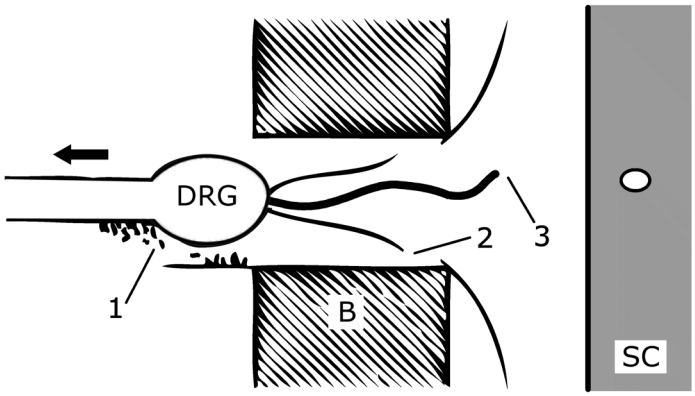
Sir Sydney Sunderland described five degrees of nerve lesion rather than three.[5] In his classification, the first degree injury corresponds to Seddon's neurapraxia and the fifth degree injury to neurotmesis. The continuum between axonotmesis and neurotmesis is defined in more detail by this classification. (Figure 4)

Root avulsion

Root avulsion occurs when the root filaments are torn out of the spinal cord at the junction of the central to the peripheral nervous system. Avulsion is also referred to as pre-ganglionic injury to distinguish it from the post-ganglionic lesions described before. A distinction between peripheral and central avulsion mechanism was suggested by Sunderland to explain different patterns of injury.[6] In the suggested peripheral mechanism, due to lateral traction to the spinal nerve, the damage occurs at a specific sequence of events. First the fibrous connections between the transverse process and the spinal root are torn. Next the dural sleeve ruptures, and finally the rootlets are torn from the spinal cord. (Figure 5)

The transverse processes of C4-6 present special features in the form of prominent bony gutters to which the corresponding spinal nerve is strongly bound.[7] This results in the clinical finding that at the level of spinal roots C5 and C6, a post-ganglionic lesion is encountered more frequently than a pre-ganglionic injury, because the nerve is firmly attached to the cervical spine. At lower levels, such tight connections between the spinal nerve and the transverse process are absent, and therefore, root avulsions are more common at these levels than post-ganglionic lesions.

Figure 5: **Root avulsion (peripheral type according to Sunderland)[6]**



In subsequent order rupture takes place at (1) the fibrous connections between nerve and foramen, (2) the dura and (3) the rootlets. DRG – *dorsal root ganglion;* B – *bony foramen;* SC – *spinal cord.*

Table 2: **Birch's classification of root avulsions**

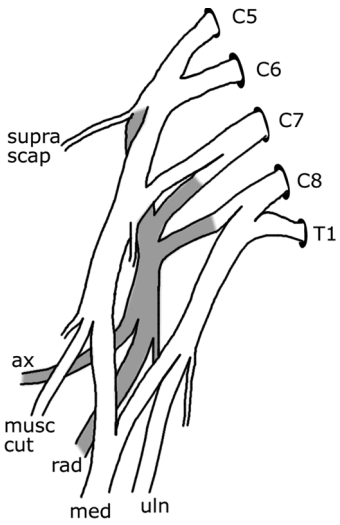
Type A:	roots torn central to the transitional zone, true avulsion
Type B:	roots torn distal to the transitional zone
	B1 dorsal root ganglion (DRG) displaced into the neck, dura torn within spinal canal
	B2 DRG more or less displaced, dural sleeve torn at the mouth of the foramen
	B3 rupture of roots within the spinal canal, without displacement of the DRG or rupture of the dura
	B4 avulsion confined to dorsal or ventral root, DRG not displaced

A central-type avulsion mechanism is supposed to result from bending of the cervical spine and the spinal cord away from the spinal nerve. Such a traction injury results in an avulsion of the spinal nerve without a lesion of the dural sleeve.[6]

An alternative classification for avulsion type lesions was proposed by Birch.[8] Avulsions are divided into types A end B depending on whether the lesion occurs proximally or distally to the transition of central to peripheral nervous system. A true avulsion (type A) takes place inside the spinal cord, proximal to this transitional zone. B type avulsions are ruptures of the rootlets that form the spinal nerve and these types are further classified according to the displacement of the dorsal root ganglion. (Table 2)

Neither classification for root avulsions is widely used. The clinical relevance of both systems is to explain that a root avulsion can exist with or without rupture of the dural sleeve. This is important, as on radiological images a dural rupture presents as a pseudo-meningocoele. The absence of a pseudo-meningocoele, – whether documented by plain myelography, CT-myelography or MRI – does not, therefore, exclude rupture of the spinal rootlets. The target of radiological investigation should be the visualisation

Figure 6: **Anatomy of the brachial plexus**



suprascap – suprascapular nerve; ax – axillary nerve; musc cut – musculocutaneous nerve; rad – radial nerve; med – median nerve; uln – ulnar nerve

of the rootlets and not only the presence or absence of pseudo-meningocoeles.[9] In addition, it is important to realize that surgical exposure is limited to the exterior osseous boundaries of the neuroforamen. A seemingly intact proximal nerve inside the foramen does not exclude the presence of a root avulsion type B3 and B4.

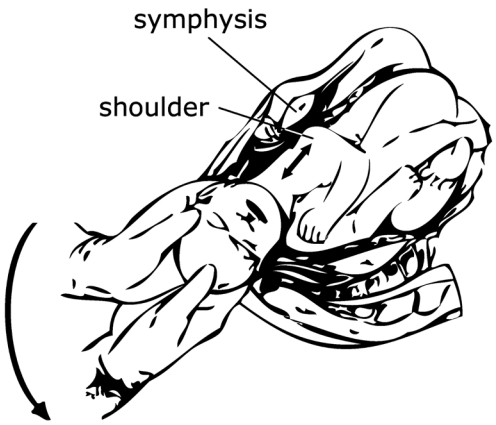
The brachial plexus lesion

The brachial plexus is a complex network of nerves extending from the spinal cord to the nerves innervating the upper limb. It usually consists of spinal nerves C5 through T1. The gross anatomical concept involves these roots joining to form three trunks, each of which divides into an anterior and posterior division. These divisions in turn form the lateral, medial and posterior cord from which all major nerves to the arm subsequently arise. (Figure 6)

The location of most traction, the direction of the traction and amount of force on the brachial plexus determines which elements of the brachial plexus are affected and to what extent. In adults, traumatic lesions are encountered in the entire course of the brachial plexus or the major nerves. Forceful widening of the angle between the head and the shoulder usually results in a supra-clavicular traction lesion, while fracture or dislocation of the gleno-humeral joint leads to an infraclavicular lesion. Blunt trauma to the chest may lead to a costo-clavicular crush lesion. Distraction due to humeral fracture usually affects the radial nerve, and luxation of the elbow can result in ulnar or median nerve injury.

In brachial plexus lesions occurring at birth, the site of injury is consistently the supraclavicular brachial plexus. In the majority of cases delivery of the upper shoulder is blocked by the mother's symphysis, which is called 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. (Figure 7) This trauma

Figure 7: **Mechanism of obstetric lesion**



mechanism was demonstrated in historical papers describing mechanical experiments with stillborn foetuses.[10-12] They described that increasing the lateral traction to the head with fixed shoulders resulted in a typical pattern of traction injury of the brachial plexus. First of all, the upper part of the brachial plexus was injured; then with increasing traction, the middle and lower parts were also injured. The superior trunk (the junction of C5 and C6) is almost always affected, resulting in the typical clinical picture of a paralysis of the shoulder and elbow flexion. As the severity of the traction increases, C7, C8 and T1 are affected subsequently, with neurological deficit in the corresponding muscles.

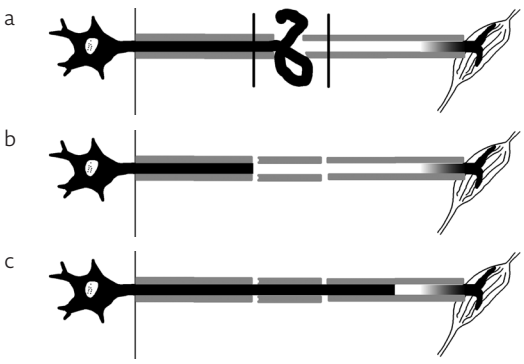
As OBPL is a closed traction injury, the extent of nerve damage can only be assessed by evaluating recovery in the course of time, because nerve lesions of different severity initially present with the same clinical features, i.e. function loss of the affected nerve.

It is worth mentioning that the phrase "***Obstetric*** brachial plexus lesion" in this thesis is not meant to imply that the *obstetrician* is to blame for the occurrence of the nerve lesion. The discussion about responsibility is a matter of continuous dispute in many papers in the obstetric literature. The aim of certain papers is clearly to acquit the obstetrician from liability and related litigation claims. Examples of synonyms used to avoid such sensitivity are perinatal brachial plexus injury, birth brachial plexus palsy, neonatal brachial plexus injury, Erb's palsy and congenital brachial plexus palsy. The last term is somewhat misleading, as it suggests that the lesion is in fact a developmental anomaly, whereas in our opinion, the large majority result from nerve traction injury during delivery.

Nerve surgery

Neurapraxia / Sunderland grade I lesions will recover completely within a matter of minutes, hours or days. Axonotmesis / Sunderland grade II lesions will show complete or almost complete spontaneous recovery after a delay of several months to allow for

Figure 8: **The principle of nerve grafting**

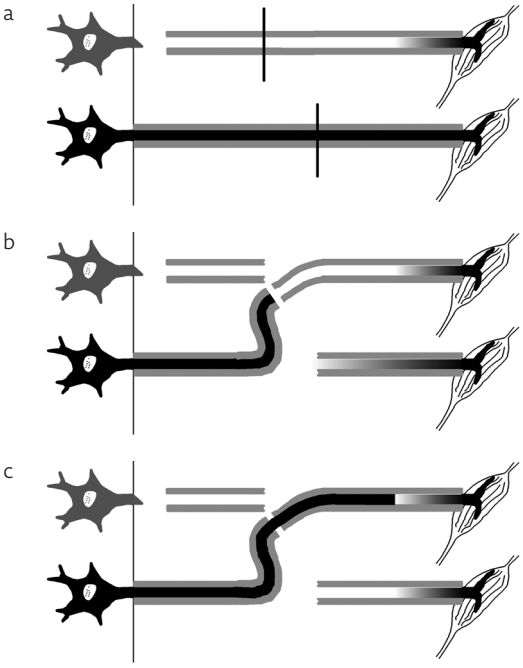


a – resection of neuroma; b – interposition of a nerve graft; c – axonal outgrowth through the graft

axonal outgrowth. In the case of Sunderland grade III lesion, some spontaneous recovery will take place, but it will remain incomplete. The most severe post-ganglionic lesion types (Neurotmesis / Sunderland IV and V) will not show clinically significant recovery. Surgical therapy consists of resection of the scar tissue and neuroma formation and the interposition of a conduit to guide the outgrowing axons from the proximal to the distal stump. (Figure 8) In the present clinical situation, an autologous nerve graft is used, harvested from a non-injured location. Usually the sural nerve is used, because its sole function is sensory, thus only sacrificing the sensation of the lateral side of the foot and ankle, a loss which is well tolerated.[13] Alternatively, the sensory supraclavicular nerves or the sensory branch of the radial nerve is used. A mixed motor and sensory nerve is seldom employed as a graft. Unfortunately, the development of artificial nerve grafts is still inadequate for widespread clinical application.[14]

In some cases an alternative technique is preferred (to nerve grafting): nerve transfer. The technique consists of cutting a healthy neighbouring nerve (the donor nerve) and coapting it to a denervated distal brachial plexus nerve (the recipient or target nerve). (Figure 9) Nerve transfers can be employed if a proximal stump is unavailable due to avulsions or intraforaminal rupture, or alternatively when the expected functional recovery of a nerve grafting procedure is low. This is the case when the defect to be bridged by nerve grafts is very long, and / or a long time has elapsed since the moment

Figure 9: **The principle of nerve transfer**



a – cutting the donor and recipient nerve; b – coaptation of the stumps; c – axonal outgrowth from the donor into the recipient

of the injury. It has been shown that a long interval between injury and repair results in inferior results of nerve repair.[15,16]

A prerequisite for nerve transfer is that the function innervated by the donor is of less value than the function to be restored. After nerve transfer, cerebral control must change to adapt to the new function.[17]

Various extraplexal nerves are used as donor. Most frequently the spinal accessory nerve or the intercostal nerves are connected to intraplexal targets: the suprascapular nerve or the musculocutaneous nerve respectively. Intraplexal transfer means the redirection of an element within the brachial plexus towards the distal part of a damaged recipient nerve. An example of such a transfer is the medial pectoral nerve that is coapted to the musculocutaneous nerve to reinnervate the biceps muscle at the expense of partial denervation of the pectoral muscle.

A recently developed concept in nerve surgery is the transfer of only one fascicle of a healthy nerve to restore function of a denervated nerve. The most frequently employed reconstruction is the transfer of one fascicle of the median or ulnar nerve selectively to the motor branch of the biceps or brachialis muscle.[18] The sacrifice of one ulnar or median nerve fascicle does not affect the function of the donor nerve, but the re-directed axons produce reinnervation of the target nerve sufficiently powerful for clinical function. The advantages of such a transfer are 1) a single coaptation site (compared to two coaptation sites in the case of nerve grafting) and 2) a more distal repair, which results in earlier reconnection and thus a shorter denervation period.

An alternative technique is end-to-side repair: the proximal stump of the recipient nerve is attached to the side of an intact donor nerve.[19] It is hypothesized that the attached denervated recipient nerve stimulates collateral sprouting of the axons of the intact donor nerve. From the axons of the intact donor nerve a supplementary branch will grow into the recipient nerve, which leads to recovery of its function, but without any negative effect on the integrity and function of the donor nerve. In laboratory settings, end-to-side coaptation has shown promising results. The clinical results of end-to-side repair in OBPL will be discussed in Chapter 13.

Differences between adult brachial plexus lesions and OBPL

As early as 1852, August Waller noticed that nerve lesions in younger animals had a different recovery potential than those in adult. *"In the preceding experiments, I have always used animals in a state of maturity. If, on the contrary, the experiments are made upon the young frog, we find a considerable difference in the period of disorganisation, and in its appearance. (...) The regeneration of the nerves, which takes place very rapidly on the young animal, does not appear to meet with such difficulties as with the fullgrown frog, and the restoration of the nervous influence is also much more complete."*[20]

The differences between adult brachial plexus lesions and OBPL can be found at different levels: peripheral nerve, spinal cord and motor cortex. (Table 3)

Peripheral nerve lesion and regeneration

The trauma mechanism of a birth trauma differs from an adult lesion. During delivery, the stress on the nerves is applied with less velocity (compared to a motor vehicle accident), but is probably longer in duration. As a result, a true rupture of the nerves (Sunderland V) is seldom seen, but during surgical exploration a neuroma-in-continuity is usually found (Sunderland III-IV lesion). The forces on the nerves are strong enough, however, to cause root avulsions. Especially in delivery of infants presenting in breech position, root avulsions of the upper nerves are common.[21,22]

Contradictory findings have been published on the level of recovery of peripheral nerves in young animals or humans. First, in neonate animals, axotomy results in substantial neuronal death.[23,24] The immature neurones seem to have an increased vulnerability especially in newborn animals aged only a few weeks. This would decrease the corresponding motor neurone pool resulting in inferior regeneration.[24] In contrast, axonal outgrowth was shown to be superior in young sheep (1 week) compared to adult specimens (1 year) in a repair model of experimentally induced brachial plexus avulsions.[25] In general, nerve regeneration in younger animals is superior compared to that in older animals.[23]

Histological analysis of neuroma and axon counting was performed in 28 surgically treated infants with OBPL.[26] The characteristic lesion shows a neuroma-in-continuity, in which a surprisingly large number of axons were shown to have grown across the lesion site, although these infants showed no clinical recovery at a mean age of 6 months. In the same study, the authors quantified the neuronal cell death by counting the number of fibres proximal to the lesion compared to 23 control juvenile plexuses. In the OBPL infants the median axon count was 27,500, compared to 34,000 in healthy controls.[26]

The alleged superior axonal outgrowth through the neuroma might explain the lack of sustained atrophy in OBPL, which is a typical feature of adult brachial plexus lesions. A better outgrowth towards the target organ might be in part due to a shorter regeneration distance between the level of injury and the target organ.

Nonetheless, the outgrowing axons are not per se destined for their original target organs, but can grow to another target. This phenomenon is called misrouting, and may occur in different ways. A topographic specificity in the regrowth of axons towards their original end-organ does not seem to exist.[27] This means that an outgrowing axon that was originally connected to muscle A, does not preferentially grow back to muscle A, but instead grows in a random way, being equally likely to grow to muscle A as to muscle B. A second form of misrouting is that a motor axon grows through a sensory pathway (and vice versa), as so-called preferential motor reinnervation does not seem to exist.[28] The re-growth of a motor axon to a sensory target does not lead to function. A third form of misrouting is that during the outgrowth process different sprouts of the same axon may find their way to different muscles: after a crush lesion or graft repair, 2.5 to 6% double-labelled motor neurones were found in the spinal cord.[29] This may lead to double connections, and so an action potential in one nerve could lead to contractions in two muscles.

All these factors may occur both in younger and in adult animals, although an animal study (carried out in rats) showed that misrouting occurs in young more often than in adult animals. A suggested explanation is that axons of peripheral nerves in younger animals have a more penetrable basal lamina tube encasing them than those in older rats.[30]

Another influential factor may be axonal cross-excitation; this means that in the neuroma, there is a tendency for an action potential to co-activate a neighbouring axon. In such circumstances, stimulation of nerve fibres from C5 may lead to cross-excitation of nerve fibres from C6 in the neuroma, which may result in simultaneous contractions of more than one muscle.[31]

Due to misrouting, double connections and ephaptic cross-excitation the action potential to muscle A might lead to contraction of muscle B, or to a contraction of both muscles at the same time. Thus, misrouting obviously diminishes the voluntary control of the targeted muscle. Two clinical examples will be mentioned. First, co-contractions between biceps and triceps were described: biceps and triceps muscles contract at the same time. As these muscles counteract movement of the elbow joint, the resulting elbow-flexion is weak or absent. One treatment option is to weaken the triceps muscle with botulinum toxin, so that the pre-existing force of the biceps muscle is more efficient in providing elbow flexion.[32] Second, outgrowing axons from C4, originally destined for the diaphragm, were found to have grown towards the muscles of the arm, which led to the observation of the "breathing arm".[33]

Table 3: **Special characteristics of OBPL**

peripheral nerve lesion and regeneration	
clin [26]	no rupture, but neuroma-in-continuity
anim [23,25]	better axonal sprouting in younger animals
hist [26]	many axons passing the neuroma without the presence of clinical function
anim [30]	increased misrouting in younger animals
phy [31]	ephaptic cross-excitation
clin [32]	co-contractions between biceps – triceps
clin [33]	breathing arm
clin	no atrophy
spinal cord	
anim [23,24]	increased motor neurone death in the spinal cord
anim [24] / phy [34]	increased recruitment of neighbouring motor pool
clin [35]	no deafferentation pain
brain	
clin [37]	developmental apraxia, due to absent motor control and sensory feedback during the time of development of motor programmes
clin [35]	greater plasticity, resulting in normal sensation, even in the most severe lesions
clin [39]	shift from right hand dominance to left hand dominance

arranged on the levels of the peripheral nerve, the spinal cord, and the brain. anim – animal study; clin – clinical finding / clinical study; hist – histological study in OBPL patients; phy – neurophysiological phenomenon

Spinal cord

There is re-organization at the level of the spinal cord. As mentioned before, axotomy in newborns results in motor neurone death, the degree being four times greater than in adult animals.[24] This is, however, compensated for by an increase in the neighbouring motor neurone pool. In an animal model of obstetric C5-C6 injury, a larger spinal cord C7 motor neurone pool was found compared to adult animals.[24] In electrophysiological studies performed in infants with OBPL during surgery, a contribution of spinal nerve C7 to biceps innervation was supposed, while normally only C5 and C6 contribute significantly to biceps innervation.[34] Equally, the sensory pathways in the spinal cord may show enhanced adaptive properties: adult brachial plexus avulsion injuries in adults often result in chronic deafferentation pain; this phenomenon is absent in OBPL infants.[35]

Brain

An evenly or more important form of central nervous system compensatory adaptation probably lies in the motor cortex and supplementary motor cortex.[36] If an axon grows towards a different end organ than the original target organ, cortical control must change accordingly. In this respect, misrouted sensory axons may have an important role equivalent to that of motor axons, as sensory axons provide feed-back to the developing motor schemes. In fact, the plasticity in the case of a nerve lesion in adults might be less demanding than in OBPL, as existing motor programmes may be able to drive the motor neurones as soon as the muscle fibres are re-innervated. In OBPL, on the other hand, function is also dependent on the formation of motor programmes in addition to recovery of the peripheral nerve lesion. The peripheral nerve paralysis coincides with the critical time in infant development of motor programmes. This development is probably hampered severely by aberrant outgrowth of both motor and sensory axons, possibly leading to erroneous learning of voluntary control; this has been named developmental apraxia.[37]

A frequent, clinical observation is that an older OBPL child can have fairly good control over the affected limb, but whenever he/she starts running, the limb is held in adduction-flexion to the trunk and does not swing with the running movement as the unaffected arm does.

To illustrate that the occurrence of an OBPL leads to different functional development of the brain apart from motor programming, development of language dominance in OBPL infants was investigated. In a functional MRI study a correlation was found between the severity of right-sided brachial plexus injury and language lateralization. The peripheral right-sided limb injury is suggested to prevent the usage of the right upper limb; therefore, the left upper limb is preferred, which, in turn, may cause a shift of language lateralization from the left to the right hemisphere.[38] In fact the existence of an OBPL might lead to a change from right hand to left hand dominance.[39]

A greater plastic adaptive capacity of the young brain is generally presumed[36], which might make it easier to modify central control. For instance, the cortical representation of hand sensibility is usually gravely disturbed after nerve trauma and sur-

gical treatment of adult nerve lesions.[40] In infants with OBPL, a normal sensation was concluded after surgical rerouting of the normal sensory pathways from the lower roots to the upper roots of the brachial plexus.[35]

Summary

Spontaneous recovery after a severe nerve lesion in OBPL is dependent on a large number of factors. Lesion severity determines the extent and quality of axonal outgrowth. The central nervous system control over these axons is also dependent on plasticity and learning processes.

If spontaneous recovery does not occur following a severe lesion (i.e. neurotmesis and root-avulsion), surgical intervention is required to regain axonal continuity to the end-organ.

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