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Peripheral nerve conduction blocks caused by anatomical compression or trauma: Functional prognosis after late neurolysis

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Introduction:

Peripheral conduction blocks caused by compression or trauma are common pathologies but still their mechanism and the prognosis remain unclear. Many recent studies have provided new information about the underlying mechanisms influencing immediate and long-term prognosis. However, no publication made the link between new knowledge and clinical observations. Over the last years we have seen many clinical situations where clinical evolution and long-term prognosis did not correspond to what was generally recognized and expectable according to common surgical practice and literature. This paper aims to determine the underlying mechanisms that influence both the clinical status and prognosis of patients with compressive conduction blocks treated by neurolysis. We will also try to determine if the temporal window during which surgery improves the prognosis significantly, could be extended.

Methodology:

A search in the PubMed, Google Scholar and up-to-date was performed using, as key words nerve entrapment, nerve compression, peripheral neuropathy, late/ delayed neurolysis/ decompression, prognosis, carpal tunnel syndrome, treatment, outcomes, prognosis factor, ulnar nerve entrapment, peroneal nerve entrapment, carpal tunnel syndrome, cubital tunnel syndrome, Sudden classification, Sunderland classification, migraine surgery. References from retrieved papers were checked for any additional studies. Fifty-nine articles from 1992 to 2018 have been selected. Among these 16 were helpful for the question of surgical indication and 6 for the question of timing.
This work will also highlight three patients who showed favorable clinical outcome after neurolysis several years after the onset of their symptoms.

Epidemiology:

Carpal tunnel syndrome is the most common human entrapment neuropathy, affecting approximately one in every six adults. Cubital tunnel syndrome is the second most common compressive peripheral neuropathy, thirteen times less common than carpal tunnel syndrome. [1,2] In traumatic upper extremity peripheral nerve syndromes the most commonly injured nerve is the radial nerve followed by ulnar and median nerves.[3] In a population-based study in the Netherlands, clinical carpal tunnel syndrome was present in 3.4 percent of the population and was likely present (undiagnosticated) in an additional 5.8 percent.[4]
Lower limb peripheral nerve injuries are less common than upper extremity lesions. The sciatic is the most frequently injured nerve, followed by peroneal and even more rarely tibial or femoral nerves.[3]

Anatomy and Pathophysiology of nerve compression:

A peripheral nerve is composed of many structures. Within a nerve, fibers are organized in fascicles. Each fascicle is covered by the perineurium how is the main responsible to nerve tensile strength. All of the fascicles are surrounded by epineurium a connective tissue layer which encloses an individual nerve and runs between fascicles. Epineurium is a vascular tissue

whose function is to nourish and protect the fascicles. Inside the fascicles are grouped many axons (some myelinated and the rest non myelinated) responsible for the transmission of nerve impulse. [2,5,6]

Nerve compression usually occurs where nerves flow through fibrous tunnels near joints. The two most common examples are carpal tunnel syndrome and cubital tunnel syndrome. [2]
Under normal circumstances the tissue pressure within a compartment of a limb is 7 or 8 mmHg but for example in carpal tunnel syndrome, the pressure is often 30 mmHg and can reach 90 mmHg or more when the wrist is in flexion or in extension.[7] Pressure increase limits microvascular blood flow causes local ischemia. [7,8] Physiologic response after ischemic injury is conduction block witch is rapidly reversible when the pressure is released. [8]

Conduction blocks can be detected in electro-physiological study of nerve conduction. A conduction block is defined by a reduction in amplitude and / or surface of a motor response induce by a proximal nerve stimulation compared to one caused by an induced distal stimulation on the same nerve trunk.[9,10] Generally, it is called a conduction block when the amplitude and the surface of the response obtained by proximal stimulation is lower by more than 20% than those obtained by distal stimulation. [11]
There are several pathophysiological explanations to conduction block. First, demyelination causes the loss of the insulating ability of a nerve segment and thereby inducing a block. The slowing of nerve conduction is the key sign of demyelinating diseases.
A conduction block can also be caused by axonal dysfunction without damage to the myelin sheath. [10,11] This dysfunction may be due to an alteration of the sodium potassium pumps activity during transient ischemic compression, for example. Similarly, an activation defect of the sodium channel or an excess of the potassium channel function can also be causing the block.[11]
All these processes cause the loss of conduction capacities of axonal membrane.
Electrophysiological study is a diagnostic complement to clinical examination to confirm, localize and define the severity of the lesion. This information will help evaluate the prognosis and define therapeutic indications.[10] However, the absence of electrical response does not always mean that the continuity of the nerve is interrupted and conversely the response can be normalized after a week even if the nerve is completely severe.[12]

Chronic compression cause perineural oedema and thickening, fibrosis, focal demyelination and finally nerve fiber degeneration.[2,8] Early stage changes are not fully characterized yet and better knowledge of them would help to determine the timeframe berfore the injury process becomes irreversible. That would also be helpfull for planning the treatment strategies.[2]
When a nerve undergoes trauma such as elongation or compression it results fibrous tissue formation (scar), which may be either perineural or endoneural. The scar increases the tension on the nerve fibres through the loss of the gliding system. This may reduce intra-neural blood supply, which can lead to prolonged ischemia of the nervous structures.[13]

When conservative treatment does not allow the resolution of symptoms, it is necessary to explore the lesion and practice a neurolysis to free nerve adhesions and remove scar tissue. Surgical treatment of these lesions begins with external neurolysis to free the nerve from extrinsic compression. [13,14] External neurolysis aims to release a nerve damaged by scar tissue or surrounding tissue, to allow regeneration. The endoneurolysis or internal neurolysis have to remove scar tissue within the nerve. [15]

Millesi et al. have described an anatomo-surgical classification of the intra and perineural scarring lesions. This classification matchs each subgroup of fibrotic lesions with specific types of neurolysis based on the severity of the scar. [13,15]
The compressions are divided into three subtypes. First those not involving the nerve itself, and therefore require only an external decompression.
When compression involves the nerve, it is necessary to practice an external neurolysis or even to transpose the nerve in a well vascularize bed if the compression has damaged a large portion of the gliding system. In type C fibrosis (fibrosis of endoneurium) or when the continuity of the nerve is lost, neurolysis is not effective and therefore it is necessary to practice a resection and then to restore the continuity of the nerve with a nerve graft. [15]

Fibrosis of type A	Epifascicular epineurium is involved
Fibrosis of type B	Fibrosis extends to the interfascicular tissue between the fascicles
Fibrosis of type C	Endoneurium has become fibrotic
Loss of fascicular pattern	Complete lost of the fascicular pattern

There are different ways to reduce the reformation of scar tissue. For example, early resumption of the movement allows sliding of the nerve and reduce adhesions.[16] The nerve may also be wrapped in different substances to reduce scarring (venous autograft, vascularized fascia or syntetical membrane). [16,17]

Classification of peripheral nerve damage and functional prognosis:

Nerve injury can be characterized by an injury of the myelin sheath, axonal injury or both. It causes sensitivity or motor disorders possibly leading to reduction/abolition of muscle strength. Functional recovery requires regenerating axon and myelin sheath with re innervation of targets (sensory receptors, neuromuscular junction). The time for functional resolution depends on the distance that the axon has to travel to reach its target.[12]
The potential recovery of peripheral nerves varies depending on the degree of involvement. The most used classifications to decide how to handle these lesions are those of Seddon and Sunderland. Although the classification of Seddon is easier to use, Sunderland is more often used for surgery to decide when and how to intervene.[12]

Sudden's classification uses three categories:

1. Neuropraxia, which is a lesion to the myelin sheath without axonal damage, with a quick spontaneous resolution.
2. Axonotmesis, axonal injury without loss of nerve continuity, connective tissue (perineurium and endoneurium) remaining intact.
3. Neurotmesis total loss of the nerve continuity. [18]

Sunderland refines this classification by dividing nerve damage in 5 grades:

1. Sunderland grade I corresponds to the neuropraxia.
2. The grade II axon is injured but endoneurium remains intact.
3. The axon and the endoneurium are harmed in the grade III.
4. The grade IV only the epineurium is preserved
5. The grade V is the neurotmesis in Seddon's system. [18]

Sudden	Sunderland	Injury	Nerve continuity	Recovery potential
Neuropraxia	I	Myelin sheat	Preserved	Quick and spontanous
Axonotmesis	II	Myelin and axone	Preserved	Complete
Axonotmesis	III	Myelin, axone and endoneurium	Preserved	Slow and incomplete
Axonotmesis	IV	Myelin, axone, endoneurium and perineurium	Preserved	Poor to zero
Neurotmesis	V	Total loss of the nerve continuity	Lost	Extremely low without surgery

Then Mackinnon and Dellon introduced an additional category (Grade VI) describing mixed lesions, combinations of grades II-IV. [12,18] The two systems try to correlate the degree of injury to clinical symptoms. Even with classifications, the nerve damage is a continuum and it's sometimes difficult to distinguish between the different grades. [19]

Each of these degrees of injury is associated with a prognosis of functional recovery. In the case of a neuropraxia or Sunderland grade I most patients spontaneously recover in two or three months when remyelination is completed and conduction block is resolved. [20] About axonotmesis (Sunderland grade II-IV) the prognosis of recovery varies depending on the axon ability to regenerate.[20] In a Sunderland grade II the recovery potential is complete, in a grade III it is usually slow (1mm / day) and incomplete and often poor to zero in a grade IV. [12,18,20] Finally in neurotmesis, without surgery the recovery chances are extremely low. [20]

Furthermore, the nerve lesions are in most cases mixed, composed of a combination of neurapraxia, axonotmesis and neurotmesis to varying degrees. The end result of recovery depends on the dominant pathophysiological characteristic. [21]

Regeneration in grade III lesions is often incomplete due to different reasons. First, retrograde damages to the cell body are more important and therefore the neurons are either destroyed or their recovery slowed. Then, the loss of endoneurium promotes intra fascicular fibrosis and disturbs axonal regrowth. Finally, the regeneration period being extended in these stages, the target organs undergo changes that do not allow full recovery (muscle atrophy and fibrosis). [17,18] Neurons and Schwann cells also gradually lose their ability to support axonal regrowth over time and distance. [18]

Also, it is important to distinguish the grades III lesions from those of grades IV and more because their care differs. Grades III are commonly supported medically and grades IV-V surgically. It is possible to distinguish them by setting a time limit of 3 to 6 months with no evidence of re innervation to electrophysiological examination. However this time to diagnosis significantly increases muscle atrophy and therefore causes poor functional recovery.[12]

This delay in diagnosis could be filled by the neurography by magnetic resonance (NMR) to assess the anatomy of healthy and pathological nerves with a good visualization of soft tissue and excellent spatial resolution. [12,22] It allows for example to distinguish the grades III and IV (VI: focal enlargement, loss of fascicular continuity. III: Homogeneous nerve, fascicular continuity preserved). Grades II and III are indistinguishable with this technique but the management is conservative in both cases. [12]

Surgical or non-surgical treatment:

Compression nerve injury can be managed in many different ways, surgical or not. There is many non-surgical treatment like oral AINS, corticoid injection, hand therapy, activity modification and splinting. [23–27]For the carpal tunnel syndrome the non-surgical modalities with the strongest evidence of benefit are bracing/splinting and steroid injections.[25,28]

Referring to the literature, the surgical approach seems to be the one with better outcomes.

Twelve articles on the 16 used to answer this question reported better result with surgery.

The results seem to vary depending on the type of nerve. With carpal tunnel sdr it seems clear that surgery gives better long term clinical outcomes.[23,24,27,29]

For the ulnar nerve indications for surgery are less clear. In patients suffering from severe neuropathy with motor deficits, muscle wasting and/or progressive sensory symptoms (McGowan type 3), surgery gives the best outcomes. Patients with milder symptoms (McGowan type 1 and 2) seems to have good clinical outcomes with conservative treatment. [30–33]

Finaly in cranial nerves causing migraine headache surgery seems to give very good results in patient how are not getting better with traditional therapy.[34–37]

Surgical or non-surgical treatment: n=16

Autors	Date	Type of injury	Recovery		N= x
			Surgical treatment	Non-surgical treatment	
Gerritsen and al.	2002	Compression/ Carpal tunnel (TC)	80%	54%	176
Hui and al.	2005	Compression/ TC	75%	16%	50
Jarvik and al.	2009	Compression/ TC	46%	27%	116
Wiley and al. (Cochrane)	2003	Compression/ TC	significantly better relieves of symptoms		198
Graham and al.	2003	Compression/ TC		10%	105
AAOS (US Guidlines)	2016	Compression/ TC	Strong evidence supports that surgical treatment of carpal tunnel syndrome should have a greater treatment benefit at 6 and 12 months as compared to splinting, NSAIDs/therapy, and a single steroid injection.		
Staples and al.	2017	Compression/Trauma Ulnar nerve (UN)	For sever injury	For mild and moderate injury	litteratur
Shah and al.	2013	Compression/ UN		88%	25
Dellon and al.	1993	Compression/ UN		Mild: 89% Moderate:67% Severe: 37%	164
McKee and al.	1998	Trauma/ NU	80%		21
Lauretti and al.	2017	Compression/NU	78%		50
Gfrerer and al.	2018	Compression/ Migrain Headhach (MH)	82%		83
Kung and al.	2011	Compression/ MH	67-95%		346
Guyuron and al.	2002	Compression/ MH	95.5%		22
Poggi and al.	2008	Compression/MH	67%		18
Stevan J. Anselami	2006	Compression/ Peroneal nerve	86 %		21

Functional recovery after nerve injuries:

Mechanisms of peripheral nerve recovery include resolution of conduction block (in neurapraxic lesions), distal axonal sprouting (in axonotmetic lesions) and axonal regeneration (in axonotmetic and neurotmetic lesions). [21]

The main barriers to early recovery are the Wallerian degeneration, the speed of axonal regrowth and adverse effects of denervation on muscle target. The research focuses on treatment that can reduce the impact of these barriers. For example by improving axonal regrowth (growth factors), reducing the time of denervation (electrical stimulation of the target) and delaying or avoiding Wallerian degeneration with biocompatible glue.[18] Glue allows to keep intact nerve architecture, better fascicular alignment, and less scarring (less tissue trauma) compared to micro-sutures. Polyethylene glycol hydrogel (PEG) is a nontoxic and biocompatible glue that avoid Wallerian degeneration after neurotmesis injury by reconnecting the proximal and distal axon ends. [18] Preliminary studies have been promising demonstrating that PEG-fusion rapidly restores physiological and morphological axonal continuity (measured by the restored conduction of compound action potentials and the intra-axonal diffusion of fluorescent dye across the lesion site) and improves a faster motor recovery. [38]

The likelihood of functional recovery is also influenced by the extent of demyelination, thus the level of nerve conduction slowing, as well as the amount of axonal loss and the distance between the lesion and the muscle. More the lesion is close to the targeted muscles better is the recovery prognosis. [20]

A study about the outcome and the prognostic value of clinical, sonographic, and electrophysiologic features in patients with ulnar neuropathy shows that more pronounced ulnar nerve thickening at the time of the diagnosis is associated with poor outcome while electrodiagnostic signs of demyelination on testing indicate favorable outcome. This study showed no association between clinical features at baseline and outcomes at follow-up. [39]

Other studies showed that clinical examination may also be useful for determining the prognosis after late decompression, for example, muscle impairment represents a negative prognostic factor in ulnar nerve neuropathy. [40] A study about ulnar nerve duct syndrome at the elbow showed age, severity of nerve involvement (evaluated with the McGowan Classification), and long-term symptom progression (beyond one year) as poor prognosis factors after surgical management. [41]

Grade 1	Grade 2a	Grade 2b	Grade 3b
Subjective symptoms	Objective symptoms without intrinsic muscular atrophy of the hand	Objective symptoms with intrinsic muscular atrophy of the hand	Sensory and motor dysfunction with paralysis of the intrinsic muscles of the hand

Modified McGowan classification for ulnar nerve compression at the elbow.

Many other factors affect the prognosis of these patients. For example, age, general health status, comorbidities (eg diabetes), smoking, social circumstances, economic and cultural status, mechanism and location of the lesion, injuries of vascular structures and adjacent soft tissues and the time between the injury and the reconstruction can be included. [18,42–45] According to a study by R. Maalla outcomes of neurolysis are less favorable in patients with polyneuropathy, many of whom have co-morbidities, such as diabetes and/or chronic alcohol abuse. [46]

The type of surgery also seems to influence the prognosis of patient with nerve injury. A study about outcomes of secondary reconstruction of ulnar nerve lesions demonstrate the role of the type of surgical repair performed on clinical improvement (both motor and sensory). Three techniques were compared (neurolysis, secondary suture repair, or graft repair). The patients who had undergone neurolysis compared with suture repair or nerve grafting show better outcomes for both motor and sensory outcomes. [47]

Some studies show that in cases of idiopathic ulnar nerve compression at the elbow or cubital tunnel syndrome, both simple neurolysis and submuscular transposition after neurolysis, improve clinical outcome. There seems to be no statistically significant advantage of one technique over the other.[48,49] However the transposition appears to be associated with a higher number of complications particularly infectious. [49]

A study which compares simple neurectomy versus neurectomy with intramuscular implantation for interdigital neuroma shows that intramuscular implantation might offer superior pain relief with comparable complications and functional outcomes than the simple neurectomy technique. [50]

The physiological rate of axonal regrowth varies between 1 and 2 mm/day without any possibilities to accelerate the regrowth. On the other hand there is a maximum interval of 12 to 18 months to reach the target muscle and if it is not the case there is deterioration of the neuromuscular junction and muscle, which do not allows more functional recovery.[18] Despite this, some authors have reported cases of recovery after 26 months post-injury and reconstruction. [5,18]

Although early surgical management generally seems to achieve better results in peripheral nerve injury, some studies have shown that even after a delayed treatment (greater than one year post-lesion) functional improvement remains possible. [51,52]

In a study by Gezercan and al the EMG follow-up results of patients who received late-term surgical treatment for peripheral nerve lesions caused by penetrating injuries showed apparent electrophysiological improvement. [51]

A prospective study was carried out by Rochkind and Alon in patients suffering from old peripheral nerve and brachial plexus injuries (from 1,2 to 50 years after injury) to validate that functional improvement still was possible with a good functional outcome in 57% of 35 patients with old peripheral nerve injuries. This study showed that 19 of 35 patients suffering from old peripheral-nerve injuries have increased their amplitude of compound muscle action potential (CMAPs) and decreased their latency after external/ interfascicular neurolysis. Clinical

improvement was also observed with 57 percent of old peripheral-nerve-injured patients who showed functional motor improvement up to M4 (active movement against gravity and slight resistance). Furthermore intraoperative electrophysiological findings like presence of response (CMAPs) during neurolysis which indicates axonal continuity through the lesion, implies that the viability of an injured peripheral nerve is of longer duration than previously supposed, and can be considered as a prognostic factor in cases of old injuries. This study suggests that the use of microsurgical techniques results in the functional improvement of patients suffering from old injuries of peripheral nerve.[52]

M0	No mouvement observed
M1	Only a trace or flicker of movement is seen or felt in the muscle, or fasciculations are observed in the muscle
M2	Muscle can move only if the resistance of gravity is removed
M3	The joint can be moved only against gravity with the examiner's resistance completely removed
M4	Muscle strength is reduced, but muscle contraction can still move joint against resistance
M5	Muscle contracts normally against full resistance

Medical Research Council's grading system for muscle strength. The patient's muscular effort is graded on a scale of 0 to 5. [14]

Peripheral nerve injury can also cause neuropathic pain. A study carried in Lausanne by Dr Decrouy-Duruz about patient with chronic neuropathic pain shows a successful treatment of the pain in 80 % of patient, without influence of the delay between injury and intervention, of the type of nerve, the surgical method and the characteristics of the patient like age or gender. In this study the mean time between the injury and the surgical treatment was about 4 years. [53]

Referring to the literature, the functional improvement remains possible even after delayed surgery. Three studies had good result with 57% to 82% of improvement with surgery from 1,2 to 50 years after the beginning of symptoms.[34,52,53] One study about iatrogenic lesions conclude that iatrogenic injuries should be corrected in a timely fashion way what means less than 6 months after the first operation.[54]

Timing for surgery : n=6

Autors	date	Injury	Delay	Recovery	N=x
Grinsell and al.	2014	Trauma	12-18 month for Muscles, Longer for sentitiv receptor		0
Rochkind and Alon	2000	Trauma/ Compression	1,2-50 y	57%	50
Gfrerer and al.	2018	Compression / MH	+/- 20 y	82%	83
Decrouzy-Duruz and al.	2017	Trauma/ Compression	Pain relief did not vary in a statistically signifcant way with time between trauma and surgery.	80%	231
Kretschmer and al.	2001	Iatrogenic	< 6 monthes	70%	97

In our daily clinical practice we also observe the ability of peripheral nerves to recover quickly when a compression that has been evolving for several years is corrected by neurolysis. Take the case of Mr DK. 54-year-old patient in usual good health who had a typical ulnar nerve compression at the right elbow and a carpal tunnel syndrome also on his right arm. Both had been evolving for several years. The patient was treated with neurolysis and anterior transposition of the ulnar nerve of the right elbow on 3.10.17 and decompression of the median nerve at the wrist on 15.11.17. Following the two interventions immediate disappearance (the first night post op) of the neurological signs of nerve compression without post-surgery complications.

Another example is Mr M.P 60 years old, known for arthropathy, diffuse osteoporosis and overweight suffering from bilateral neuropathy of the median nerve for more than 10 years. Clinically, all signs of severe compression of the median nerve are present. Following the release of the median nerve at the right carpal tunnel, the patient presents a very good evolution with immediate disappearance of paresthesia and complete recovery of sensitivity at 2 weeks post-surgery. The operation on the left side has been done in January with the same outcome.

The type of nerve also seems to influence the operative window. The timeframe for reinnervation of sensory receptors appears to be much longer than for motor nerves although early repair shows better sensory outcomes. [5] Sensory receptors can be reinnervated years after injury but the maximum timeframe remains uncertain. [18] Some nerves seem to have a worse prognosis than others. Injuries of the ulnar nerve in upper extremities and the peroneal nerve in lower extremities show less favorable outcomes. [51]

Practically, nerve healing is a complex process, which is mainly influenced by the site and type of lesion, the structures involved, the surgical method and the individual possibilities of recovery. (19)

Rehabilitation (post-surgery physiotherapy) also plays an important role for functional recovery. [21]

Chronic Migraine Headhaches caused by compression of craniofacial nerves:

Another situation in which peripheral nerves late decompressive surgery has shown a beneficial effect is the treatment of migraines headhaches (MH). Compression of craniofacial nerves can be a peripheral MH origin. Usual pain localization of patient with migraine headache are forehead, temple, or occiput. These sites correlate with specific migraine triggers who involves peripheral nerves compressed by craniofacial muscles and anatomical structures. [36] Theses triggers can be deactivated either with botulinum toxin type A (BT-A) injections, local anesthesia or with surgery.[55]

To illustrate the effect of decompressive surgery on patients suffering from chronic migraine headhaches we will take the case of Mrs. A.M, 61 years old. The patient suffers of chronic periorbital and temporal bilateral migraine headhaches. Following surgical management by neurolysis of the supraorbital and supratrochlear nerves by bilateral blepharoplasty approach and neurolysis and sectioning of the bilateral temporal nerve branches, the patient presents a reduction of pain from 8 /10 to 4/10. Occipital pain was persisting, which is the reason we decide to do a neurolysis of the two large occipital nerves with ligature and resection of the occipital artery and occipital vein branches. The outcome was good with no residual occipital pain.

A decompressive surgery is indicated when the patients remain symptomatic following a conservative management including the avoidance of environmental trigger and pharmacologic therapies or for those who are unable to take medications. Surgery is also indicated when improvement of the pain following the injection of BT-A or local anesthesia to specific anatomical locations is objectified or when physical examination indicates a potential trigger site. [36,55,56] The BT-A is useful as significant predictive factor of migraine surgery success and to identify potential migraine surgery trigger sites.[55]

The four most common peripheral trigger sites identified are:

Trigger I: Irritation of the supratrochlear and supraorbital nerves by glabellar muscles group and arteries. Cured with glabellar muscle group resection in the frontal region.

Trigger II: Irritation of zygomaticotemporal branch of the trigeminal nerve by the temporalis muscle, adjacent vessels, and tight deep temporal fascia opening. Surgical treatment involves decompression or avulsion of the nerve zygomaticotemporal branch of the trigeminal nerve

Trigger III: Irritation of the trigeminal end branches induced by intranasal abnormalities. Irritation of septoplasty, turbinectomy in the nasal region.

Trigger IV: Irritation of the greater occipital nerve by semispinalis capitis muscle, trapezius muscle, occipital artery, or surrounding fascial bands. Treated by greater occipital nerve decompression in the occipital region and/or removal of vessels.

Injection of BT-A and/or local anesthesia is a powerful prognosticator for triggers I, II and IV but not for trigger III. During a pain episode, nerve blocks with local anesthetics during a pain episode can also be used as a diagnostic and positive prognostic factor for surgery.[55,56]

BT-A action on MH his action on reducing peripheral nerves irritations caused by external forces applied by craniofacial muscles contraction. This result in reducing the occurrence of MH by decreasing peripheral nerve inflammation and excitability. Blocking the presynaptic release of the neurotransmitter acetylcholine caused the relaxation of muscles and thereby BT-A is used in many spastic disorders and in our case helps to eliminate migraines triggers that consist of compressed nerves by surrounding muscles. [36,57] *A study by* Dodick et al. on 172 migraine headache patients who were not taking preventive medications shows good results with significant decreased in headache frequency and severity compared with placebo. [58]

Effects of the toxin are temporary, the blocking is reversible and generally lasts from 3 to 6 months depending the pathology being treated. [36,59] Also its use has risks as diplopia, injection-site pain, blepharoptosis or atrophy of the injected muscles. Furthermore surgery shows more permanent and effective results. [55] Therefore Botulinum toxin should be used as screening test rather than as usual treatment modality and those patients who are responders may then benefit surgical decompression. [36]

Several studies have investigated the potential for migraine surgery, with conclusion that surgery is effective in MH treatment. [36] For example a prospective study carried out by Guyuron about the role of removal of corrugator supercilii muscles, transection of the zygomaticotemporal branch of the trigeminal nerve, and temple soft-tissue repositioning in the treatment of migraine headaches shows a significant benefit in 21 of 22 patients involved. The study also shows that injection of Botox is an extremely reliable predictor of surgical outcome. [37] Another study by Poggi shows a reduction in duration, intensity, and frequency of MH after surgical decompression of the supraorbital, supratrochlear, zygomaticotemporal, and greater occipital nerves. [35]

Conclusion:

In patients with typical symptoms of peripheral conduction blocks caused by compression or trauma evolving over many years, surgery should be considered. Always for carpal tunnel syndrome and only in severe ulnar neuropathy. Although early surgical management generally seems to achieve better results in peripheral nerve injury, some studies and our surgical practice have shown that even after a delayed treatment functional improvement remains possible. [18,34,51–53]

The prognosis factor remains unclear for patients with compressive conduction blocks treated by neurolysis but some factors have already been shown to influence the clinical outcome of these patients. Axonal continuity, presence of CMAPs during neurolysis or electrodiagnostic signs of demyelinisation seems indicate favorable outcome. On the other hand muscle impairment, sonographic features like pronounced nerve thickening, age and comorbidities like diabetes, polyneuropathy, chronic alcohol abuse or smoking seems to have poor outcomes. Furthermore some studies showed that clinical examination may also be useful for determining the prognosis.
The type of nerve also seems to influence the operative window and type of treatment we should undertake. Sensory receptors have a bigger timeframe to be reinnervated than the neuromuscular junction.[18]

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