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Technical notes & surgical techniques

Restoring the function in neuralgic shoulder amyotrophy by modified Somsak nerve transfer

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ABSTRACT

Background and Objectives: Neuralgic amyotrophy (NA), also known as Parsonage-Turner syndrome, is a rare and acute neuropathy of the shoulder and/or arm. It is characterized by sudden pain attacks, followed by patchy muscle paresis in the upper extremity. The frequency of remaining neurological deficits varies in the literature between 10% and 66%. In addition to other therapeutic strategies, nerve branch transposition might be a possible treatment option to improve muscle strength and reduce pain symptoms. Transposition of the first motor triceps branch to the axillary nerve was first described in 2003 to regain the deltoid muscle function in patients with C5 (C6) root injury.

To our knowledge, this is the first description of nerve transfer for treating permanent paresis in NA.

Methods: We present the case and the surgical treatment of a 16-year-old male with loss of axillary nerve function due to NA on both upper extremities. Neurological examination before surgery, 6 and 12 months after surgery included analysis of the upper extremity range of motion and muscle strength evaluated using the Medical Research Council Scale.

Results: Shoulder function and muscle strength improved significantly 12 months after surgery. The range of motion of arm abduction improved from 90° preoperatively, to as high as 120° at 6 months, and to 130° at 12 months. Muscle strength was strength grade 2/5 preoperatively, 3/5 at 6 months, and 4+/5 at 12 months. Subjectively, the patient also has a significantly improved quality of life.

Conclusion: Nerve transfer is a promising treatment method for patients suffering from NA.

1. Introduction

Neuralgic amyotrophy (NA) is a rare and acute neuropathy of the shoulder and/or arm. It has been described in the literature with various clinical manifestations since 1897 [1]. After the description by Personage and Turner in 1948, the term Personage-Turner syndrome has been used synonymously [2].

Recent literature suggests that NA has an incidence of 2–3/100000/year. It is more common in the male population and rare in the pediatric population [3–6]. The right side is affected more frequently; the involvement of both sides is a sporadic phenomenon [6,7]. Predisposing events and conditions have been identified in about 50% of cases. The disease is preceded by infections, vaccinations, surgery, the peripartum period, or unusually severe physical stress [8]. Interestingly, a concomitant hepatitis E virus (HEV) infection was present during the acute phase in 10% of the cases [9].

It affects the lower motor neurons of the brachial plexus or individual nerves and nerve branches of the upper extremities. It is characterized by severe, acute pain as the first symptom in 90% of patients, reaching levels of seven and more on the Visual Analogue Scale [2,10]. After the pain disappears, motor deficits, muscle wasting, and sensory disturbances follow. Paresis occurs in 85% of cases after days to weeks [8].

The diagnosis of NA is commonly based on typical clinical symptoms and its unique history. In addition, electromyography (EMG) substantiates the diagnosis by confirming muscle denervation. However, EMG is only applicable at least 4 weeks after the onset of NA because muscle denervation will not be fully apparent in EMG earlier [11]. Nothing less, EMG is essential to assess the course of muscle denervation

Abbreviations: EMG, Electromyography; HRUS, High-resolution ultrasound; MRI, Magnetic resonance imaging; NA, Neuralgic amyotrophy.

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and its recovery.

High-resolution peripheral nerve imaging, such as high-resolution ultrasound (HRUS) and magnetic resonance imaging (MRI), are valuable tools to confirm the diagnosis of NA. It presents as alterations in all clinically affected nerves, presenting as segmental swelling in HRUS and hyperintensity in MRI. [12] Furthermore, nerve constriction as a sign of NA can be detected on MRI. [13,14] In many cases, there is no satisfactory healing and improvement in motor function, especially in cases where the affected muscles are entirely denervated. There is divergent information on the long-term outcome. A recovery rate of 89% has been published as well as a poor neurological improvement with persistent paresis after years in around 66%. [15–19] One must consider that overall recovery is less favorable than usually assumed.

Permanent loss of active shoulder abduction due to neuropathy of the posterior fasciculus or axillary nerve means a severe functional limitation in everyday life. Patients need to change their profession or are unable to work in around 50%. [18] The shoulder joint instability and chronic pain syndrome that may develop due to muscle weakness also limit the quality of life. [19,20] Currently, there are no controlled studies about the natural course and treatment success of NA, and therapy varies. [21] Early oral or intravenous cortisone therapy or immunotherapy can lead to improvement. [3,21,22] A negative prognostic factor is the involvement of the inferior and contralateral plexus. [23].

The technique of transferring functional nerves or parts of nerves to replace a function was described as early as 1984. [24] The transposition of the first motor triceps branch to the axillary nerve according to Somsak Leechavengvongs (also known as “Somsak transfer”) was presented in 2003 in cases of traumatic avulsion injuries of the C5 and C6 nerve roots. [25].

Here, we present our experience in nerve transfer in a 16-year-old male with loss of axillary nerve function on both upper extremities due to NA. We use the modified Somsak transfer method, which uses the medial triceps branch instead of the branch to the long triceps head, to preserve innervation of the long triceps head, which is an important component of shoulder stability. [26].

2. Materials and Methods

2.1. Illustrative case

A 16-year-old male was transferred to our Neurosurgical department 18 months after the onset of devastating left shoulder pain and the first suspicion of NA. After increased physical activity, he reported acute, severe pain in the left shoulder and arm region. This was followed by weakened arm abduction and wrist and finger extension. He developed shoulder and arm pain on the other side six months later. The initial MRI shows an abnormality in the STIR sequence with suspected edema in the brachial plexus area. The axillary nerve was affected bilaterally, while the radial nerve was affected only on the left side. There was no relevant improvement of the symptoms under steroid treatment and intensive physiotherapy. The function of the triceps muscle recovered very quickly after symptom onset, while the function of the hand and finger extension recovered only in the long-term course. Unfortunately, there was a progressive loss of deltoid muscle function with an instability of the shoulder joint and paresis of arm abduction. Finally, the young patient had a complete axillary nerve palsy on both sides (Fig. 1a).

The regular follow-up and electromyographic examinations showed a progression of the denervation in both deltoid muscles and no volitional activity. It was impossible to identify a focal nerve lesion requiring local neurolysis or partial nerve grafting. We indicated nerve transfer surgery following the transfer according to Somsak Leechavengvongs for traumatic lesions of the C5 and C6 root approximately 12 months after the first symptoms appeared. Due to the more pronounced atrophy of the right deltoid muscle and to allow the left radial nerve more recovery time, we decided to initiate the nerve transfer on the right side first. Neurological examination, including the analysis of motor function, assessed using the Medical Research Council scale [27] the extent of motion (measured in degrees) and EMG, was performed preoperative, 6 and 12 months postoperative (Table 1).

2.2. Surgical treatment

The surgery is performed under general anesthesia in prone position with the right arm abducted on a small arm board. The superior and posterior shoulder, the axilla, and the entire arm are prepped as a surgical field.

A 15 cm longitudinal incision is made between the medial and the lateral head of triceps brachii extending into the axilla. The dissection is carried deeper to expose the quadrangular space that transmits the axillary nerve and a triangular space bounded by the teres major superiorly, the long head of the triceps medially, and the lateral head of the triceps laterally that contains the radial nerve and triceps nerve branches. The landmark and point of orientation is the teres major muscle with its transverse fascia. (Fig. 2 a) The axillary nerve is located above and the radial nerve below. The branch to the medial head of the triceps is located centrally and is shown first under microscopic view. It runs very close to the radial nerve. Under magnification, however, it is depicted to run separately and is easily identified with the help of electrical stimulation (mikrofork probe 45 mm straight, Inomed), corresponding to the motor response of the medial drive. Very low currents (0.2–0.5 mA) are used to prevent co-innervation of the other branches. The identified branch is microsurgically mobilized and sharply transected as far distally as possible. Subsequently, the axillary nerve is dissected proximally in the quadrangular space as far as possible. Electrical stimulation with currents of up to 5 mA helps to ensure that no muscular activity could be triggered in the deltoid muscle. Thereafter, the axillary nerve is sharply cut at the most proximal point, mobilized,

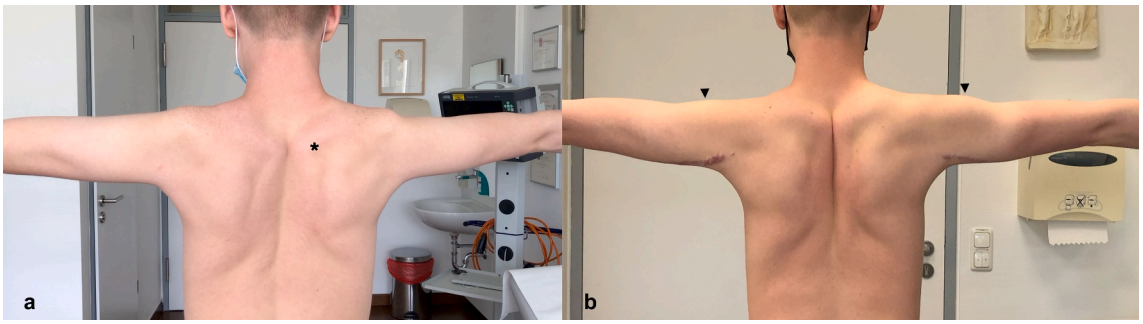

Fig. 1. (a) Dorsal view with marked atrophy of the right deltoid muscle and slightly less atrophy of the left deltoid muscle (black arrowheads). The excessive tension in the trapezius muscle to compensate for the lifting weakness is also obvious (*). (b) Postoperative course after12 months with clear improvement of the abduction strength on both sides and increasingly harmonious muscle silhouette of both deltoid muscles (▾).

Table 1
Functional and electrophysiological changes of the deltoid muscle preoperatively, 6 - and 12 months postoperatively.

	MRC (arm elevation)	Range of motion (arm elevation)	Electrodiagnostics EMG	Electrodiagnostics NCV
before surgery	2/5	90°	left m. deltoideus: no safe volitional innervationPSA (pathological spontanous activity) +++ left m. triceps brachii + left m. brachioradialis: safe volitional innervation PSA + Complete regeneration of voluntary motor function of nervus radialis innervated characteristic muscles compared to one year preoperatively.	left + right nervus radialis (motoric): no stimulus response left + right nervus radialis (sensitive): no pathology right nervus radialis:CMAP (compound muscle action potential) normalSNAP (sensory nerve action potential) normal left nervus radialis: CMAP not available SNAP greatly reduced amplitudes
6 month after surgery	3/5	120°	not possible due to pain	left nervus radialis (motoric): pathologicalleft nervus radialis (sensitive) : pathologicalleft nervus radialis (motoric) : normalright nervus radialis (sensitive) : normal
12 month after surgery	4/5	130°	left + right m. deltoideus: good volitional innervation reinnervation detectable signs of acute denervation and chronic neurogenic remodeling right: PSA ++ left: no PSA	left nervus radialis (motoric): pathologicalleft nervus radialis (sensitive) : pathologicalright nervus radialis (motoric) : pathologicalright nervus radialis (sensitive) : normal

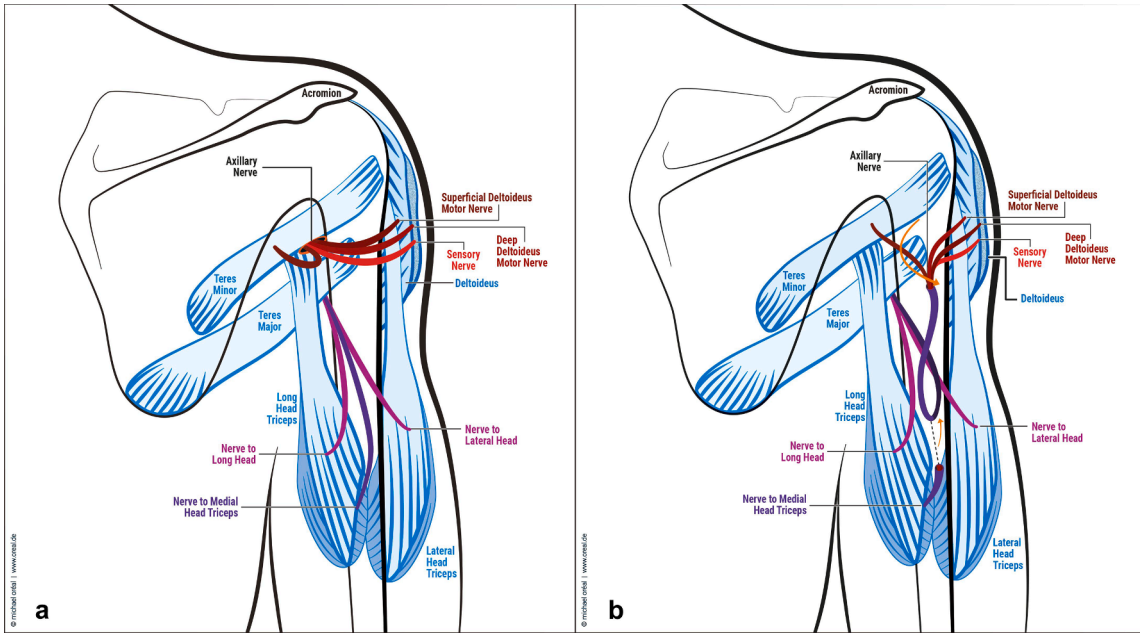

Fig. 2. The posterior approach to the quadrangular space is shown with the axillary and radial nerve branches (a) and the transfer (b). (a) Axillary nerve in the area of the quadrangular space. The greater motor branch to the deltoid muscle forms the deep aspect of the nerve. The nerve branch to the medial head of the triceps sits deep in the area between the long and lateral heads of the triceps muscle. (b) The branch to the medial head of the triceps was cut distally and the proximal segment transferred to the distal segment of the axillary nerve. The axillary nerve was transected as proximally as possible. (Illustrator: Michael Oreal).

and turned distally with the cut end. Subsequently, the distal end of the branch is moved proximally so that both nerve endings are adjacent to each other without tension. (Fig. 2 b) Both nerve endings are directly connected microsurgically with 10.0 nylon single sutures. (Fig. 3) As additional suture security, a small amount of fibrin glue is applied to the coadaptation sites. The wound is closed using a multi-layer technique without a wound drain. The patient is repositioned in the supine position with careful fixation of the operated arm. A shoulder orthosis is applied, which prevents the mobility of the shoulder joint for 14 days.

3. Results

3.1. Postoperative course

The patient's postoperative course was without complications. The elbow joint was carefully mobilized after one week to prevent stiffening. After two weeks, mobilization of the shoulder joint and intensive ergotherapeutic therapy began. Intensive active and passive treatment with physiotherapy and electrostimulation was initiated after the wound healing. Electrostimulation was done with a parse tuner, which is a versatile single-channel simulator with 4 pulse shapes. The

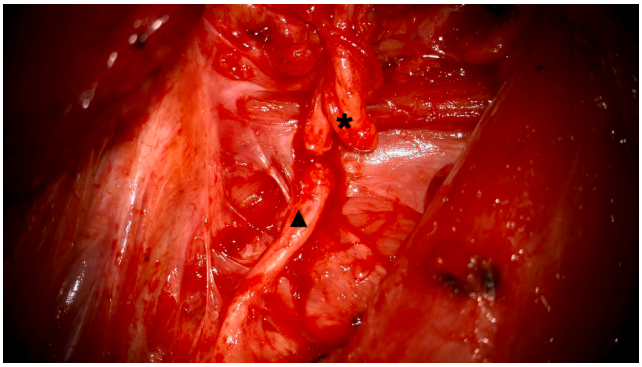

Fig. 3. Microscopic view of the coadaptation site between the axillary and radial nerves. It is mandatory to adapt the proximally transected axillary nerve (*) and the distally transected branch of the radial nerve (▲) without traction.

rehabilitation program includes muscle strengthening with emphasis on muscle balance, cortical mapping, re-education and normal movement patterns. Re-learning mainly involves contracting the unaffected side firstly the donor muscle (triceps) and also the receiving muscle (deltoid). In parallel, the same is then performed on the affected arm. After that, only the affected side. Here, both sides were affected, so after the first surgery he started with the unoperated arm as the unaffected arm. As soon as the patient has developed sufficient strength in the receiving muscle (deltoid), the target action is disconnected from the donor muscle. Visual and also auditory biofeedback is important for relearning. With the help of the surface electrodes, the antagonizing muscle can also be addressed via a 4-channel biofeedback unit. As the tension in the re-innervated muscle increases, the antagonist can then be minimized. [26].

Three weeks later, we could perform the same procedure on the left side without complication. During the postoperative examinations, we were able to detect a slight weakness in the triceps function, which the patient was able to quickly compensate for through muscle training.

A gradual increase in muscle strength and function was seen after 4 months. A noticeable successive strengthening in both deltoid muscles was present at 6 months' follow-up. A significant improvement in the muscular strength of the left (grade 0 to grade 4) and right (grade 0 to grade 4) deltoid muscle was seen after 12 months (Table 1). A complete abduction of the shoulder of more than 90°and the ability to maintain this position for a longer period of time and against resistance was bilaterally possible (Fig. 1b). After 6 month the left deltoid muscle already shows a significant improvement in function. Sensitivity in the hand has already improved significantly. Otherwise, he is able to bear weight well and does strength training. Mobility is clearly improved. Nevertheless, there are deficits in the area of gross and fine motor skills. After 12 months, there is a clear subjective improvement compared to 6 months postoperative. In the course of time there is now a pleasing improvement of the shoulder function. The scapula is symmetrical and strong when lifting the arms, both repetitive and strong. The patient is satisfied with the progress and continues to be unrestricted and independent in everyday life. There are still slight deficits in fine motor skills with limited extension of the left thumb and spreading of the fingers on the left, but he mentioned no more problems with writing or grasping. The electromyographic examination demonstrated good voluntary innervation of the muscles on both sides.

4. Discussion

Neuralgic amyotrophy is an acute neuropathy of the shoulder/arm nerves and can lead to permanent neurological deficits and pain. According to the literature and the published studies, the incidence of these permanent deficits ranges from 10% to 66%. [8,10,18,19,23] The etiology of this disease remains unclear. An autoimmune reaction still seems likely, especially due to the frequent occurrence after viral infections. Some authors have included NA in the spectrum of nonsystemic vasculitic neuropathies. This group of inflammatory peripheral nerve diseases is characterised by histopathological signs of vasculitis (i.e. vessel wall inflammation and vessel damage) without signs of systemic vasculitis. [28,29] Ultra-selective imaging of nerves using MR-neurography and HRUS has made it possible to visualize pathological changes in affected nerves. Here, segmental swellings and torsions are described in particular. [12] The high frequency of these focal pathological changes substantiates the assumption that it is a focal inflammation rather than a generalized autoimmune disease of the motor neuron. This theory justifies a treatment concept based on focal inspection, neurolysis, or segmental nerve replacement.

Conservative therapy is mandatory at the beginning of symptom presentation. There is no specific therapy for Parsonage Turner syndrome yet. The primary goal initially is pain management. There is also evidence in the literature of an effect of acupuncture and especially transcutaneous electrical nerve stimulation (TENS). Oral steroids are also most commonly used initially, although the literature is not entirely clear on this. Recently, immunotherapies have also been used.[3] To date, there are no randomized controlled trials demonstrating specific therapy, but there are studies suggesting that early corticosteroid therapy may have a positive impact on pain and speed recovery in some patients. [21] Gstoettner et al. presented a surgical treatment algorithm based on a literature review. Conservative treatment should be performed for three months and be continued if a neurological improvement is present. In cases of insufficient regeneration, surgery should be performed if MRI or HRUS revealed signs of nerve/fascicle constrictions. Intrafascicular neurolysis or nerve grafting are possible treatment options. [11].

Surgical treatment should be recommended if the conservative therapy shows no effect. The optimal timing for surgical intervention is still under debate. While some authors advocate surgical treatment as early as three months after symptom onset, following the treatment strategy for traumatic nerve lesions, others have reported improvements as late as 6 or 12 months and therefore do not recommend early surgical intervention. [11] From the time course, electrophysiological examinations were performed 1, 2, 6 and 10 months after the onset of symptoms. The first two examnination only with motor and sensory nerve conduction velocity, the later examinations also including EMG. In this particular case, we waited a relatively long time for spontaneous progression with steady improvement of radial function. A time limit of 9–12 months is recommended in the literature.[30] Especially in children, a good functional improvement can be expected even after such a period. Due to painfulness needle EMG examinations are not performed as a standard procedure in children. Nerve transfer should be considered as a therapeutic option in those cases without a focal lesion and ineffective conservative therapy. The presented nerve transfer of the radial nerve branch to the medial head of the triceps muscle to the axillary nerve is a promising technique for re-innervation of the deltoid muscle. It can be applied in traumatic lesions and cases of persistent paralysis due to neuropathy of the upper brachial plexus. After one year, this substantial functional improvement supports complex neurotization techniques as a viable treatment option. Patients must be carefully selected for this surgery. Close clinical and electrophysiological monitoring should be performed to prevent ongoing deterioration of the affected nerve.

The study's main limitation is that no other case has been described in the literature so far. It remains unclear how other patients with NA recover after the nerve transfer. On the other hand, the surgical technique has been described for different indications and is widely used with good results. Therefore, we encourage other surgeons to publish their results after similar interventions to gather larger patient numbers. Nerve transfer is a promising treatment method for patients suffering from NA.

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Ethics approval and consent to participate

Written consent was obtained from the patient. The ethics commission waived further ethical approval.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Authorship

All authors gave substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work.

All were involved in drafting the work or revising it critically for important intellectual content.

All authors gave final approval of the version to be published and agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] J. Feinberg, Fall von Erb-Klumpke scher Lahmung nach influenza Central Bl 1897 (16): p. 588–637.
[2] M.J. Parsonage, J.W. Turner, Neuralgic amyotrophy; the shoulder-girdle syndrome, Lancet 1 (6513) (1948) 973–978.
[3] J.H. Feinberg, J. Radecki, Parsonage-turner syndrome, HSS J. 6 (2) (2010) 199–205.
[4] E. Beghi, et al., Brachial plexus neuropathy in the population of Rochester, Minnesota, 1970–1981, Ann. Neurol. 18 (3) (1985) 320–323.
[5] B.K. MacDonald, et al., The incidence and lifetime prevalence of neurological disorders in a prospective community-based study in the UK, Brain 123 (Pt 4) (2000) 665–676.
[6] E. Rotondo, et al., Clinico-diagnostic features of neuralgic amyotrophy in childhood, Neurol. Sci. 41 (7) (2020) 1735–1740.
[7] G.D. Rix, E.H. Rothman, A.W. Robinson, Idiopathic neuralgic amyotrophy: an illustrative case report, J. Manipulat. Physiol. Ther. 29 (1) (2006) 52–59.
[8] J.J. Van Eijk, J.T. Groothuis, N. Van Alfen, Neuralgic amyotrophy: An update on diagnosis, pathophysiology, and treatment, Muscle Nerve 53 (3) (2016) 337–350.
[9] J.J. van Eijk, et al., Neuralgic amyotrophy and hepatitis E virus infection, Neurology 82 (6) (2014) 498–503.
[10] N. van Alfen, et al., Incidence of neuralgic amyotrophy (Parsonage Turner syndrome) in a primary care setting–a prospective cohort study, PLoS One 10 (5) (2015) e0128361.
[11] C. Gstoettner, et al., Neuralgic amyotrophy: a paradigm shift in diagnosis and treatment, J. Neurol. Neurosurg. Psychiatry 91 (8) (2020) 879–888.
[12] D. Lieba-Samal, et al., Neuroimaging of classic neuralgic amyotrophy, Muscle Nerve 54 (6) (2016) 1079–1085.
[13] D.B. Sneag, et al., MRI bullseye sign: An indicator of peripheral nerve constriction in parsonage-turner syndrome, Muscle Nerve 56 (1) (2017) 99–106.
[14] D.B. Sneag, et al., Brachial plexitis or neuritis? MRI features of lesion distribution in Parsonage-Turner syndrome, Muscle Nerve 58 (3) (2018) 359–366.
[15] Tsairis, P., P.J. Dyck, and D.W. Mulder, *Natural history of brachial plexus neuropathy. Report on 99 patients.* Arch Neurol, 1972. **27**(2): p. 109-17.
[16] J.D. England, The variations of neuralgic amyotrophy, Muscle Nerve 22 (4) (1999) 435–436.
[17] Geertzen, J.H., et al., *Brachial plexus neuropathy. A long-term outcome study.* J Hand Surg Br, 2000. **25**(5): p. 461-4.
[18] N. van Alfen, B.G. van Engelen, The clinical spectrum of neuralgic amyotrophy in 246 cases, Brain 129 (Pt 2) (2006) 438–450.
[19] E.H. Cup, et al., Residual complaints after neuralgic amyotrophy, Arch Phys Med Rehabil 94 (1) (2013) 67–73.
[20] N. van Alfen, The neuralgic amyotrophy consultation, J. Neurol. 254 (6) (2007) 695–704.
[21] van Alfen, N., B.G. van Engelen, and R.A. Hughes, *Treatment for idiopathic and hereditary neuralgic amyotrophy (brachial neuritis).* Cochrane Database Syst Rev, 2009(3): p. CD006976.
[22] D.P. Smith, J.A. Elliott, J.H. Helzberg, Intravenous corticosteroid therapy for bilateral parsonage-turner syndrome: a case report and review of the literature, Reg Anesth Pain Med 39 (3) (2014) 243–247.
[23] N. van Alfen, S.P. van der Werf, B.G. van Engelen, Long-term pain, fatigue, and impairment in neuralgic amyotrophy, Arch Phys Med Rehabil 90 (3) (2009) 435–439.
[24] G. Brunelli, L. Monini, Neurotization of avulsed roots of brachial plexus by means of anterior nerves of cervical plexus, Clin Plast Surg 11 (1) (1984) 149–152.
[25] K. Witoonchart, et al., Nerve transfer to deltoid muscle using the nerve to the long head of the triceps, part I: an anatomic feasibility study, J Hand Surg Am 28 (4) (2003) 628–632.
[26] B. Dympep, et al., Long head function preserving modification of Somsak's nerve transfer for the reanimation of shoulder abduction in the adult upper partial brachial plexus injuries: a cadaver and clinical study, Eur. J. Plast. Surg. 44 (2021).
[27] Compston, A., Aids to the investigation of peripheral nerve injuries. Medical Research Council: Nerve Injuries Research Committee. His Majesty's Stationery Office: 1942; pp. 48 (iii) and 74 figures and 7 diagrams; with aids to the examination of the peripheral nervous system. By Michael O'Brien for the Guarantors of Brain. Saunders Elsevier: 2010; pp. [8] 64 and 94 Figures. Brain, 2010. **133**(10): p. 2838-44.
[28] M.P. Collins, R.D. Hadden, The nonsystemic vasculitic neuropathies, Nat. Rev. Neurol. 13 (5) (2017) 302–316.
[29] M.P. Collins, et al., Peripheral Nerve Society Guideline on the classification, diagnosis, investigation, and immunosuppressive therapy of non-systemic vasculitic neuropathy: executive summary, J Peripher Nerv Syst 15 (3) (2010) 176–184.
[30] D. Power, et al., Rewiring the upper limb: Motor nerve transfer surgery in the reconstruction of paralysis, Journal of musculoskeletal surgery and research. (2019).

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