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Research Article

Correction of Experimentally Induced Facial Paralysis with Great Auricular Nerve - Facial Nerve Coaptation*

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Abstract: The aim of the study was to investigate the possibility of using great auricular nerve - facial nerve (GAN-FN) coaptation in the correction of experimentally induced facial paralysis according to clinical, histological and histomorphometrical analysis in dogs. Five mongrel dogs of different age (range 2 to 6 years) and sex (2 male and 3 female) were the study material. Facial paralysis was induced by neurectomy at the side of the facial nerve trunk passing through the stylomastoid foramen. The great auricular nerve (GAN) was freed, neurectomized behind the furcations and transposed to the distal part of the neurectomized facial nerve trunk. Both nerve stumps were coapted by epiperineural sutures. All 5 dogs were kept under clinical control for 6 months. At the end of the observation period, each dog was anesthetized and the operated area exposed. Specimens were taken from the coapted area and about 1 cm proximal and distal of the coapted area. The remaining facial nerve stumps were repaired by nerve grafting and the animals were used for another study. The specimens were evaluated histologically and histomorphometrically with respect to axon counts, myelin area and nerve fiber diameter. Clinical signs of facial paralysis had improved, except in one dog, which had wound dehiscence. There were no significant differences between the proximal and distal parts of the coapted area with respect to axonal counts, nerve fiber diameter and myelin area. In conclusion, in facial paralysis, GAN-FN coaptation can be a good alternative treatment for intracranial facial nerve lesions that can not be treated and injuries located at a point after the nerve exits the stylomastoid foramen.

Key Words: Facial paralysis, Second cervical nerve, nerve regeneration, dog

Deneysel Fasial Paralizinin Nervus Aurikularis Magnus-Fasial Sinir Koapitasyonu ile Düzeltilmesi

Özet: Köpeklerde deneysel fasial paralizinin nervus aurikularis magnus – fasial sinir (NAM-FS) koapitasyonu ile düzeltilme olanağının klinik, histolojik ve histomorfometrik yönden incelenmesi amaçlandı. Çalışma materyalini farklı yaş (2-6 yaşlı) ve cinsiyette (2 erkek ve 3 dişi) 5 melez köpek oluşturdu. Fasial sinirin foramen stylomastoideus'tan çıktıktan sonraki ana trunkusunda nörektomi yapılarak fasial paralizi oluşturuldu. Nervus aurikularis magnus serbestleştirildi ve furkasyonun gerisinden kesilerek, fasial sinirin distal kısmına transpoze edildi. Her iki sinir ucu birbirine epiperinöral dikişlerle koapte edildi. Beş köpek 6 ay süresince klinik gözlem altında tutuldu. Gözlem süresi sonunda köpekler anesteziye alınarak operasyon bölgesi açığa çıkarıldı. Koaptasyon bölgesi, koaptasyon bölgesinin 1cm proksimal ve distalinden kesitler alındı. Kalan fasial sinir uçları greftlerle onarıldı ve hayvanlar başka bir çalışmada kullanıldı. Kesitler histolojik ve histomorfometrik (akson sayısı, miyelin alanı, sinir lifi çapı) yönünden incelendi. Yara iyileşmesi geciken bir olgu dışında fasial paralizinin klinik belirtilerinde düzelme oldu. Akson sayısı, miyelin alanı ve sinir lifi çapı yönünden distal ve proksimal kısımlar arasında anlamlı bir farklılık bulunamadı. Sonuç olarak fasial sinirin foramen stylomastoideus'tan çıktıktan sonraki kısmında oluşan yaralanmalarda ve sağaltılamayan intrakranial fasial sinir lezyonlarında, NAM-FS koapitasyonu iyi bir alternatif sağaltım seçeneği olabilir.

Anahtar Sözcükler: Fasial sinir, 2. servikal sinir, sinir rejenerasyonu, köpek

Introduction

Facial paralysis may be caused by brain stem inflammation, intracranial injury in conjunction with petrosal bone fracture, extracranial injuries, compression and erosion by neoplasia, idiopathic causes, otitis media-interna and iatrogenic trauma in dogs and cats (1,2).

Facial nerve injury can be repaired by direct coaptation of the cut nerves, nerve grafting and tubulation with nonneural structures if the injured area is suitable (3,4). For the treatment of lesions that can not be exposed properly, like intracranial disorders of the facial nerve or its nucleus and in severe hemifacial spasms, many operative procedures have been reported

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in humans (5-8). Direct neurotization can strengthen a weak muscle target without sacrificing its normal innervation (4). Nerve transfer techniques have been described, using hypoglossal, accessory and phrenic nerves as motor donors in clinical practice (7). Hypoglossal nerve transposition is used more extensively than the others but all of these techniques have some disadvantages such as adaptation difficulties of the patients and atrophy of the tongue (5,7). The other techniques are not complication free in correction of a paralyzed face (7).

It was thought that hypoglossal, accessory and phrenic nerve transposition techniques would not be suitable for dogs because of the adaptation and function problems. However, there are no studies about great auricular nerve-facial nerve (GAN-FN) coaptation in facial paralysis of the dog. It was hypothesized that the great auricular nerve (GAN), which innervates the skin of the neck, ear and the back of the head (9), could be sacrificed for the correction of facial paralysis. In pendulous ear dogs, there are no serious problems with denervation of the pinna. In erect pinna dogs, there is a risk of pinnal drooping. Despite the side effects of sacrificing the GAN, it was considered that this technique could be used for the correction of facial paralysis.

The effect of the coaptation of GAN – FN on the correction of experimentally induced facial paralysis in dogs by clinical, histological, and histomorphometrical analysis was evaluated in the present study.

Materials and Methods

Five adult mongrel dogs (2 male and 3 female), 2 - 6 years old, were used. The protocol was approved by the Ankara University Animal Rights Committee. The dogs were anesthetized and positioned in right lateral recumbency with the head elevated slightly. A para-auricular approach to the facial nerve and GAN was made. The GAN, a branch of the second cervical nerve, going to the ear, just under the fascia, was dissected, freed and neurectomized. Dissections were carried out in order to expose the facial nerve trunk as it exits the stylomastoid foramen to the facial muscles, between the proximal part of the parotid gland and sternomastoid muscle. Freeing the nerve trunk from the stylomastoid foramen towards furcations, which splits into cervical, dorsal buccal, ventral buccal and auriculopalpebral branches, was carried out with the dissection of the tissue around the nerve trunk. Neurectomy was performed on the main trunk of the facial nerve and the proximal stump was inserted into the sternomastoid muscle to prevent regeneration. Both nerve stumps were coapted epiperineurally with 10/0 propylene suture material using a 10X magnification operating microscope (TAKAGI OM 5). The dogs were followed for 6 months and at the end of this observation period, the dogs were anesthetized with the same anesthetic protocol. After the nerve specimens had been taken, the facial nerve was repaired with grafts and the dogs survived.

Specimens were taken from the proximal and distal parts of the coapted site and fixed separately in 2.5% gluteraldehyde solutions. The tissues were washed with cacodylate sodium in 1.1 molarity and they were fixed subsequently for 2 hours in osmium tetroxide solution. The specimens were washed with cacodylate sodium again and then were dehydrated in ethanol (30, 50, 70, 90 and 100 °). The specimens were transferred to a araldite (araldite CY 212 + araldite HY 964 + araldite DY 064 + Dibutyl fitalate) and propylene oxide mixture. They were embedded in araldite. The prepared blocks were hardened at 40°C for 24 h and 60°C for 24 h consecutively. Sections 1 µm thick were cut using an 8800 LKB ultramicrotome. Semithin sections were used for histomorphological evaluation.

Histomorphometrical evaluation of the sections stained with Toluidine Blue were performed with a video camera connected to a light microscope and computer (Leica Q 500) with an Image Analyzing Program (KS 400 software). Myelin sheath thickness and nerve fiber diameter were calculated at the inner and outer borders of myelin sheaths. They were randomly selected from 50 different areas (x100 magnification) and were drawn manually and calculated by computer. Axonal numbers of myelinated axons were estimated in at least 5 different areas of each section. Axons, which appeared to be rectangular or square, were not estimated.

$$\text{Total axonal numbers} = \frac{\text{Transected nerve area}}{\text{Evaluated area}} \times \text{Myelinated nerve numbers in evaluated area}$$

The outer diameter of myelinated axons was taken into account for the determination of nerve fiber diameter. An average value of each lesser and greater

diameter was calculated by computer to estimate nerve fiber diameter. Nerve materials taken from the coapted site were stained by Klüver Barrera methods (10).

The results were analyzed using to the Chi-square test.

Results

Clinical Findings: Clinical signs of facial paralysis, such as drooling saliva from the affected side of the mouth, flaccidity of the lips, deviation of the nose to the healthy side, and absence of blink reflex, were seen in the first week of the postoperative period. There were no problems with wound healing except for one case in which seroma formation occurred and the wound healed with persistent fibrous tissue. After one month, all the clinical signs of facial paralysis were still present in each dog. Improvement of clinical dysfunction was evident at the end of three months in the four uncomplicated cases. These dogs had nearly normal appearance during resting, barking, eating and drinking at the end of the six months. An ear drooped in an erect ear dog. However, in the case with the wound healing problems, facial paralysis did not resolve.

Histological Findings: There was no fibrous tissue reaction around the surgical site except in the dog that had wound dehiscence. In that dog there were capillary vascularization, dense collagen fibers and neuroma formation (Figures 1-2). The rest of the cases' sections had an orderly fascicular pattern and well myelinated nerve fiber. However, some axons had a granular appearance in some sections (Figures 3-4).

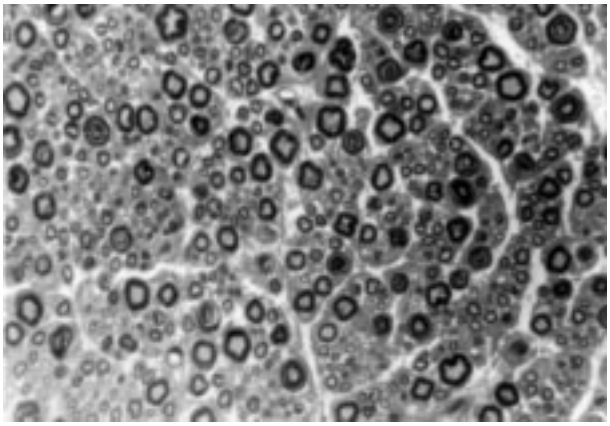

Figure 1. Well myelinized and uniform structure of the axons groups at the proximal part of the coapted site of the clinically unhealed case Toludine Blue x 160.

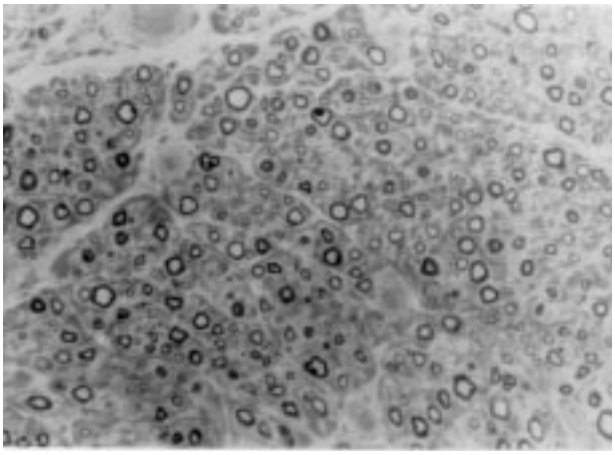

Figure 2. Irregular structure and different size of the axons in the distal part of coapted site of the clinically unhealed case Toludine Blue x 160.

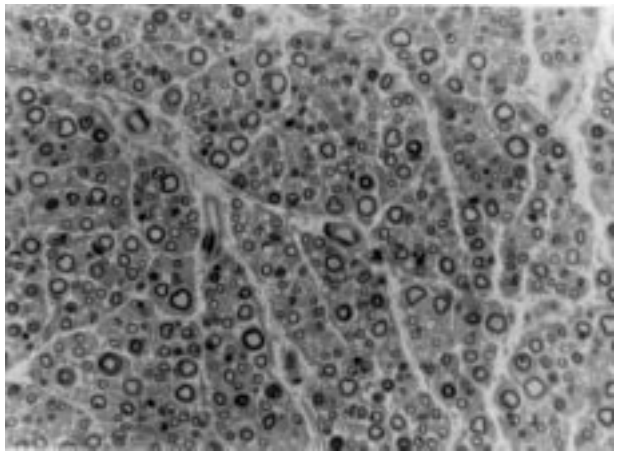

Figure 3. The well myelinization and uniformity of axons at the proximal part of coapted site of the clinically healed case toludine Blue x 160.

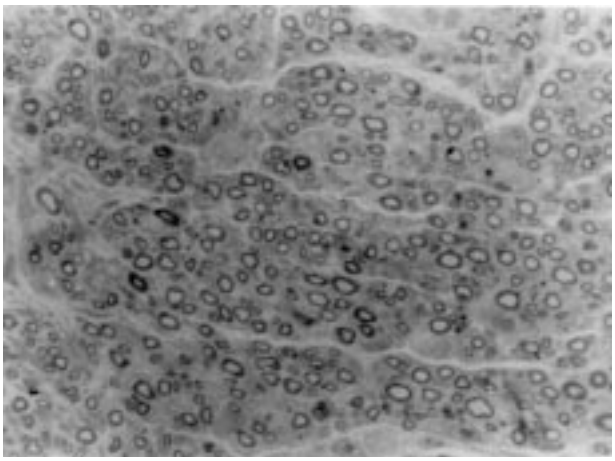

Figure 4. Well myelinized but irregular separation of axons at the distal part of coapted site of the clinically healed case Toludine Blue x 160.

Histomorphometry: The histomorphometrical evaluation of the myelin area, axon number and nerve fiber diameter demonstrated that there were no significant differences between the proximal and distal parts of the repaired site (P>0.05) (Figure 5). However, in the dog with wound dehiscence, measurements looked smaller in the distal part than in the proximal part of the repaired site of the nerve.

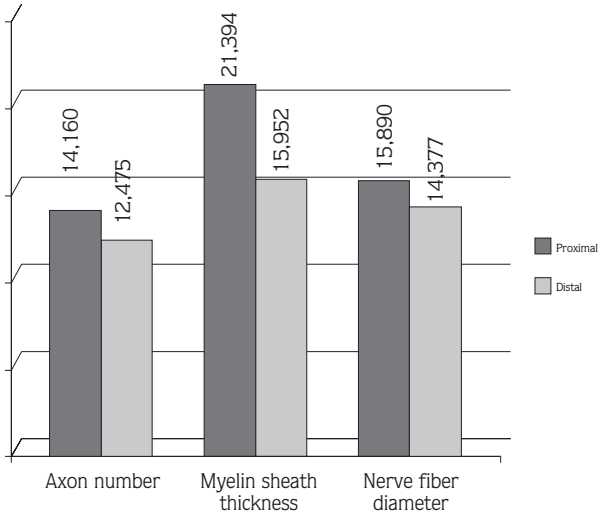

Figure 5. Histomorphometrical values of the cases. There are no significant differences between proximal and distal part of the coapted site (AN: Axon Number, MST: Myelin sheath thickness, NFD: Nerve Fiber Diameter).

Discussion

Traumatic, iatrogenic and idiopathic facial paralysis is seen frequently in dogs (1,2,11,12). Specific treatment methods suggested for dogs and surgical correction of the paralyzed face have not been studied enough in veterinary medicine. Direct repair or grafting, direct neurotization, muscle transfer, cross facial nerve grafting, and transposition of the hypoglossal, accessory or phrenic nerves have been utilized for surgical correction of facial paralysis in humans (7,8). In hypoglossal nerve transposition, paralysis and atrophy of the ipsilateral tongue muscle, difficulty in swallowing because of catching food in the vestibule on the affected side and sometimes difficulty in readaptation in using the tongue are the major disadvantages (5,7). In spinal accessory nerve transposition, the shoulder needing to be elevated to produce facial movement, and a drooping shoulder are disadvantages. Phrenic nerve transposition is complicated by facial twitches and asymmetry of the face during coughing and laughing (7).

The three nerve transposition techniques used in humans seemed to be unsuitable for dogs because of their disadvantages. These would cause more serious problems in dogs like biting the tongue, motor dysfunction of the thoracic limb, and difficulty in adaptation of facial movements during breathing. These techniques are also more complicated surgically. The results of the grafting and tubulation and direct neurotization techniques (3,4,7) are speculative. These techniques can not be used properly in some circumstances, like donor site morbidity, the need for multiple lengths of nerve, exposure e.g., GAN innervates the skin of the neck, the ear and the back of the head. When it is sacrificed, the pinna will droop (13). It was hypothetized that if GAN-FN coaptation could be performed, reinnervation of the facial muscle would be accomplished.

Numerous studies had been done for repairing peripheral nerve injuries, but most were for the nerves of the limbs (14-16). When compared to peripheral nerves of limbs, the difficulty of exposure, the sensibility of cranial nerves to trauma and the thinness of epineurium make repair difficult (17). Cranial nerve grafting is not as easy as grafting of the peripheral nerves of the limbs. Regenerating axons have to pass through two barriers because of the presence of distal and proximal suture lines at the grafting site. To avoid this disadvantage, GAN-FN coaptation might be an alternative technique. A complex microsurgical approach was not needed to expose the GAN or coapt the facial nerve.

In acute facial paralysis, the muzzle deviates towards the healthy side because of the active muscle contraction. In chronic cases, deviation towards the paralyzed side is seen because of muscle atrophy and fibrous tissue contraction, which occurs in the late stages of paralysis (1,17). Asymmetric appearance of the face and deviation of the muzzle towards the healthy side were seen in all dogs in this study. Deviation to the paralyzed side was seen at the end of the first month. Wound dehiscence and the condition of the grafting bed were considered to be responsible for the persistent facial paralysis in the unsuccessful case.

Some abnormal movements and undesired results during feeding, speaking or breathing were seen humans who were treated with transposition of the hypoglossus, accessory and phrenic nerves (7). These symptoms are related to the function of the transposed nerve. There were no signs related to the GAN during these necessary

activities in dogs. However, pinnal drooping was seen in a dog with normally erect ears. It was considered that this technique had less disadvantages compared with the studies done in humans.

Neuroma formation and adhesions are common problems when repairing nerves (18). In one dog, wound dehiscence occurred in the postoperative period. In this dog, neuroma formation was also seen histologically. The unsatisfactory clinical and histological results in this case could be attributed to the wound dehiscence.

Regenerated axons can branch into as many as 20 distal fibers, and the axonal count in the distal segment may be 150% greater than that in the proximal segment (19). In experimental studies, the fastest conduction is seen in the nerve fibers with the largest diameters. In addition, nerve fiber diameters are greater in the proximal part of the grafted sites than both in grafted sites (20) and the distal part of it in the peripheral nerve of the extremity (21,22). In a study about grafting facial nerves after neurectomy, the axonal diameter and myelin area in the grafted site were reported to be smaller than in the distal part of the grafted sites (4). Functionally, the myelin sheath serves as an insulator controlling leakage of current. The myelin area would increase according to the nerve fiber diameter. Myelination begins with the regeneration phase when growing axons reach the distal tube, and increases distally over time (19,23). It is reported that regenerated and myelinated nerve fibers have thinner sheaths than normal (7).

In the present study there were no significant differences ($p>0.05$) between the proximal and distal parts of the coapted area according to nerve fiber diameter, myelin area and axonal counts. These results indicated that the second cervical nerve could be regenerated in the facial nerve tube easily. This regeneration was found to be sufficient for resolving signs of facial paralysis in dogs. Paralyzed facial muscles could be reinnervated with this technique.

In conclusion, this study has shown that an injury of the facial nerve can be repaired by GAN-FN transposition. The techniques can also be considered in severe hemifacial spasms as described in humans.

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References

1. Kern, T.J. and Erb, H.N.: Facial Neuropathy in Dogs and Cats: 95 Cases (1975 - 1985). Journal of American Veterinary Medical Association 1987; 191, (12): 1604 –1609.
2. Wright, J.A.: Ultrastructural Findings in Idiopathic Facial Paralysis in the Dog. J. Comp. Path. 1988; 98: 111-115.
3. Kitahara, A.K., Nishimura, Y., Shimizu, Y., Endo, K.: Facial Nerve Repair Accomplished by the Interposition of a Collagen Nerve Guide. J. Neurosurgery 2000; 93: 113-120.
4. Thanos, P.K., Terzis, J.K.: A Histomorphometric Analysis of The Cross-Facial Nerve Graft in the Treatment of Facial Paralysis. Journal of Reconstructive Microsurgery 1996; 19, (6): 375 – 382.
5. Conley, J., Baken D.J.: Hypoglossal - Facial Nerve Anastomosis For Reinnervation of the Paralyzed Face. Plastic & Reconstructive Surgery 1979; 63, (1): 63 – 72.
6. Freilinger, G.: A New Technique to Correct Facial Paralysis. Plastic & Reconstructive Surgery 1975; 56, (1): 44 - 48.
7. Laeken, N.V., Manktelow, R.T.: Facial Paralysis: Principles of Treatment. Textbook of Maxillofacial and Reconstructive Surgery, Philadelphia, 581-595, 1992.
8. McCabe, B.F.: Facial Nerve Grafting. Plastic & Reconstructive Surgery 1970; 45, (1): 70 – 75.
9. Evans, H.E., DeLahunta, A.: Millers Guide to the Dissection of the Dog. Philadelphia, W.B. Saunders Co., 101-102, 1971.
10. Luna, L.G.: Manual of Histologic Staining Methods. 3rd ed. New York, McGraw-Hill Book Company, 203 – 204, 1968..
11. Parker, A.J., Cusick, P.K., Park, R.D., Small, E.: Hemifacial Spasms in a Dog. Veterinary Record 1973; 93: 514 – 516.
12. Robert, S.R., Vainisi, S.J.: Hemifacial Spasm in Dogs. Journal of American Veterinary Medical Association 1967; 150, (4): 381–385.
13. Whalen, L.R., Kitchell, R.L.: Electrophysiologic and Behavioral Studies of the Cutaneous Nerves of the Concave Surface of the Pinna and the External Ear Canal of the Dog. American Journal of Veterinary Research 1983; 44,(4): 628 - 634.
14. Jacobsen, A., Schrader, S.C.: Peripheral Nerve Injury Associated with Fracture or Fracture - Dislocation of the Pelvis in Dogs and Cats: 34 Cases (1978-1982). Journal of American Veterinary Medical Association 1987; 190, (5): 569 - 572.
15. Kanje, M., Lundborg, G., Edstrom, A.: A New Method for Studies of the Effects of Locally Applied Drugs on Peripheral Nerve Regeneration in vivo. Brain Research, 1988; 439: 116-121.
16. Lipton, R., Mccaffrey, T.V., Ellis, J.: The Beneficial Effect of Triamcinalone Acetonide on Nerve Repair with Autogenous Grafts. Otolaryngology -Head and Neck Surgery 1986; 94,(3):310-315.

17. Van Beek, A.L., Biffe P. Principles of Microneurosurgery. Textbook of Plastic Maxillofacial and Reconstructive Surgery, 2nd ed (Georgida, G.S. et al). pp.1041-1054.1992.

18. Lal, A.P., Joseph, T., Chandi, S.M., Pant, B.: Facial Neurorraphy with Autologous Surral Nerve Graft Using the CO_2 Laser in a Primate Model. Neurosurgery, 1993; 32, (6): 1011 -1014.

19. Chiu, D.T.W., Janecka, I., Krizek, T.J., Wolf, M., Lovelace R.E.: Autogenous Vein Graft as a Conduit for Nerve Regeneration. Surgery, 1982; 97, (2): 226 - 233.

20. Lee, H.K., Chung, M.S., Kim, H.J.: A Comparison of the Passage of Regenerating Axons Through Old Degenerated Nerve Autografts and Fresh Nerve Autografts in Rats. International Orthopaedics (SICOT), 1993; 17: 193 –197.

21. Braund, K.G., Mehta, J.R., Amling, K.A., Toivio-Kinnucan, M.: Morphologic and Morphometric Study of the Facial Nerve in Clinically Normal Adult Dogs. American Journal of Veterinary Research 1991; 52, 11: 1879 – 1882.

22. Shibata, M., Tsai, T.M., Firell, J., Breidenbach, W.C.: Experimental Comparison of Vascularized and Nonvascularized Nerve Grafting. The Journal of Hand Surgery 1988; 13A, (3): 358 - 365.

23. Terzis J.K. and Smith K.L. Repair and Grafting of the Peripheral Nerve. Plastic Surgery, General Principles, Vol. 1, (Joseph, G., McCarthy, MD) W.B. Saunders Company; 631-697. 1990.