



## NEUROPATHY LITERATURE

Clin Podiatr Med Surg. 2006 Jul;23(3):597-609.

### **Tarsal tunnel syndrome: a compression neuropathy involving four distinct tunnels.**

**Franson J, Baravarian B.**

Tarsal tunnel syndrome is a complex and often under-diagnosed or misdiagnosed condition that affects the foot and ankle. It is a compression neuropathy of the posterior tibial nerve as it passes in the anatomic tarsal tunnel in the medial ankle under flexor retinaculum. This article reviews diagnosis, conservative treatment, and surgical outcomes, which have dramatically improved with more comprehensive release of the foot nerves in addition to the tibial nerve. Internal neurolysis facilitates a second level of nerve decompression in needed cases. Physical therapy protocols have made it possible for patients to return to ambulation with limited long-term down time.

Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi. 2006 Apr;20(4):319-23.

### **Recent progress in diagnosis and treatment of the injury to the peripheral nerve**

**Zhu J.**

**OBJECTIVE:** To investigate the latest development in diagnosis and treatment of the injury to the peripheral nerve. **METHODS:** The literature at home and abroad was reviewed, and the research findings with clinical experience in diagnosis and treatment for the injury to the peripheral nerve were summarized. **RESULTS:** The treatment for the total brachial plexus avulsion injury was successfully performed by the extra-plexus nerve transfer. The avulsion of the brachial plexus could be directly repaired by the healthy C7 nerve root transfer through the anterior spinal approach. The forearm flexors could be reinforced by the neurovascularized gracilis transplantation. MRI and CTM were the best methods of early diagnosis for the brachial plexus injury. The pure upper or lower root avulsion of the brachial plexus injuries could be repaired by the intra-plexus nerve transfer, which involved a transfer of part of the ulnar nerve in the arm to the motor nerve of the biceps for C5-C6 avulsion of the brachial plexus, and a transfer of selective fascicles of the healthy C7 nerve root or brachial muscle branch to the flexors muscle fascicles of the median nerve or anterior interosseous nerve. The thoracic outlet syndrome always occurred in this position when the neck muscle fatigue occurred in the typists or the game-players after their longstanding looking forwards. The C5 and C6 roots were rolled by the tendinofibrotic tissue at the origin of the scalenus. After a procain block, the symptom subsided. Radiation neuropathy was a series of pathological changes caused by overdoses of the radiation therapy. The pathologic findings consisted of a series of vessel damages and final coagulation necrosis, which induced clusters of abnormally-dilated, thin-walled telangiectasias. Radiation neuropathy could be detected by MRI and could be differentiated from tumor. Once the diagnosis was established, the therapy should be begun early, including the systemic use of steroids, anticoagulation, and hyperbaric oxygen. **CONCLUSION:** Great progress has been made in diagnosis and treatment of the injury to the peripheral nerve, but a further study should be performed to promote regeneration of the nerves and reconstruction of the related functions.

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Pain Physician. 2005 Apr;8(2):157-61.

### **Effects of hyperbaric oxygen on skin blood flow and tissue morphology following sciatic nerve constriction.**

**Mychaskiw G 2nd, Pan J, Shah S, Zubkov A, Clower B, Badr A, Zhang JH.**

**BACKGROUND:** Constriction of the sciatic nerve by loose ligation produces an inflammatory neuropathic injury. This represents an animal model for peripheral mononeuropathy. Oxygen-derived free radicals are suspected to play an important role in the pathogenesis of ischemia/reperfusion injury, leading to neurogenic inflammation. Hyperbaric oxygen (HBO) has been used anecdotally to treat clinically similar conditions in humans, but specific effects on the animal model have not been well studied.

**OBJECTIVE:** This study in a rat model examined the effects of hyperbaric oxygen on skin blood flow and tissue morphology by light and electron microscopy following sciatic nerve constriction. **DESIGN:** A scientific investigation in a rat model. **METHODS:** In this study, the neuropathic injury was established by loose ligation of the rat sciatic nerve. The animals were divided into three groups, sham (S, n=8), ligation but no treatment (LN, n=8) and ligation and treatment with hyperbaric oxygen (LT, n=8). The treatment group (n=8) received hyperbaric oxygen treatment immediately following the injury and daily for four additional days at the same time interval. One hundred percent O<sub>2</sub> at 3 atmospheres absolute pressure (66 feet sea water) was administered for two hours. The hindpaws of the rats were observed by light microscopy, electron microscopy, laser Doppler flowmetry (LDF), and clinically for the presence of edema. **RESULTS:** Untreated animals demonstrated marked tissue edema following sciatic constriction, whereas animals that received hyperbaric oxygen had minimal to no edema. The sham group demonstrated normal histology. The group not treated with hyperbaric oxygen demonstrated swollen mitochondria (2-3 times), with loss of cellular integrity, multiple vacuole formation in both nerve and muscle tissue, widened sarcomeres in muscle, and degenerative changes in the nerve myelin sheaths. The group treated with hyperbaric oxygen demonstrated preservation of cellular structure including mitochondrial integrity, no vacuole formation, and maintenance of normal, easily identifiable nerve structure. The sham group had no change of skin blood flow. Skin blood flow of LT group was decreased immediately after ligation (p<0.05) and recovered to baseline level before ligation on Day 5 after four hyperbaric oxygen treatments. Skin blood flow of LN group was decreased immediately after ligation (p<0.01) and did not recover (p<0.01). **CONCLUSION:** This study evaluated tissue changes after nerve injury caused by loose ligation of the sciatic nerve in rats. Hyperbaric oxygen treatment following sciatic nerve injury reduced tissue edema, improved skin blood flow, and preserved muscle and neuronal ultrastructural integrity.

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Microsurgery. 2004;24(3):255-61.

### **Effect of hyperbaric oxygen therapy on nerve regeneration in early diabetes.**

**Aydin A, Ozden BC, Karamürsel S, Solakoğlu S, Aktaş S, Erer M.**

Nerve regeneration in diabetes is essential for reversal of neuropathy as well as the recovery of nerves from injury due to acute nerve compression and entrapment. Endoneural hypoxia due to hyperglycemia-induced blood flow reductions is observed early in the course of diabetes, and the resultant ischemia plays a role in the diminished neural regeneration. Hyperbaric oxygen therapy is capable of producing tissue hyperoxia by raising oxygen tensions in ischemic tissues, and was shown to be beneficial in the reversal of experimental ischemic neuropathy. In this study, an experimental diabetes model was used to evaluate the functional and histomorphological effects of hyperbaric oxygen therapy on early diabetic nerve regeneration. Our results indicate that there is significant histomorphological impairment of nerve regeneration, even in very early stages of diabetes. However, no beneficial effects of hyperbaric oxygen therapy could be demonstrated at this stage.

Dis Colon Rectum. 2003 Nov;46(11):1549-54.

### **Hyperbaric oxygen in the treatment of fecal incontinence secondary to pudendal neuropathy.**

**Cundall JD, Gardiner A, Chin K, Laden G, Grout P, Duthie GS.**

PURPOSE: Hyperbaric oxygen therapy has several physiologic effects on damaged nerves in animal models, which lead to an improvement in neurologic function. Idiopathic fecal incontinence secondary to pudendal neuropathy is usually treated with biofeedback, which shows improvement in only 50 percent of patients. METHODS: Thirteen patients (12 females, age range, 40-75 years) with chronic pudendal neuropathy and fecal incontinence were identified. They received 30 treatments of hyperbaric oxygen during a period of 6 weeks. Each treatment was at 2.4 atmospheres breathing pure oxygen for 90 minutes. Pudendal latencies were performed sequentially throughout the treatment and one and six months after it had finished. Questionnaires were used to assess improvements in symptoms and quality of life (Wexner fecal incontinence quality of life score). RESULTS: All patients completed the treatment without major complications. There was a consistent improvement of the latencies (on the left 2.36 msec initially, reduced to 2.08 msec at 6-month follow-up and on the right 2.23 msec, on the left reduced to 2.07 msec at 6 months). These improvements were significant (Wilcoxon's two-tailed, asymptomatic significance, comparing pretreatment to 6-month follow-up, left 0.005, right 0.003). Incontinence sores also improved (12.08 initially to 11.64 at the end of treatment, 10.55 at 1-month follow-up, and 10.45 at 6-month follow-up). Using the same test, the improvement in incontinence scores also was significant when comparing pre-end (0.05) and pre-one month (0.011) but not pre-six month (0.054). CONCLUSIONS: Hyperbaric oxygen therapy has improved pudendal nerve function and continence in this group of patients. The cause for this improvement in latencies is unclear at present but may be because of a direct effect on the nerve or an improvement in blood flow to the nerve through angiogenesis. However, these results are good enough to schedule further trials.



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J Natl Med Assoc. 1998 Jun;90(6):355-8.

### **The effectiveness of intermittent hyperbaric oxygen in relieving drug-induced HIV-associated neuropathy.**

**Jordan WC.**

This 3-month study evaluated the effects of hyperbaric oxygen on drug-induced neuropathies in 22 patients with human immunodeficiency virus. All patients included in the study had been taking an antiretroviral medication for at least 12 months and had subjective symptoms of numbness or tingling, lethargy, and a decrease in deep tendon reflex. Patients with an active substance abuse history or Kaposi's sarcoma were excluded. Of the 20 patients who completed the series, 17 had significant improvement, 2 had a demyelinating disorder that may have affected the outcome, and 1 had no change.

Nippon Ganka Gakkai Zasshi. 1997 Sep;101(9):730-3.

### **The effect of hyperbaric oxygen on ophthalmic artery blood velocity in patients with diabetic neuropathy**

**Okamoto N, Nishimura Y, Goami K, Harino S.**

We investigated changes in the blood velocity of the ophthalmic artery to observe its relationship to the complication of diabetes mellitus before and after hyperbaric oxygen therapy (HBO). Color Doppler imaging (Toshiba, SSA-260A) was used. In this study, there were 7 diabetic neuropathy patients, 3 diabetes patients without neuropathy, and 7 normal subjects for control. These patients were examined before and after HBO. The patients were made to breathe 100% oxygen at 2.0 atmosphere absolute (ATA) for one hour. HBO is sometimes used for treatment of diabetic neuropathy. HBO produced an average decrease in blood velocity of 15.0 +/- 9.0 (mean +/- standard deviation) % in normal subjects, 10.7 +/- 8.6% in diabetes patients without neuropathy, and returned to the baseline level 4 hours after the conclusion of HBO. On the other hand, the blood velocity increased by about 20.6 +/- 9.5% in diabetic neuropathy patients regardless the degree of severity of diabetic retinopathy. These results suggest that the increase in the blood velocity of the ophthalmic artery after HBO in diabetic neuropathy patients could be attributed to an imbalance in autonomic nervous function.



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Ann Neurol. 1995 Jan;37(1):89-94.

### **Experimental ischemic neuropathy: salvage with hyperbaric oxygenation.**

**Kihara M, McManis PG, Schmelzer JD, Kihara Y, Low PA.**

Hyperbaric oxygenation is effective in augmenting the delivery of oxygen to tissue, but also causes oxidative stress. As part of our focus on improving peripheral nerve salvage from ischemic fiber degeneration, we evaluated whether hyperbaric oxygenation rescues peripheral nerve, rendered ischemic by microembolization, from ischemic fiber degeneration. The supplying arteries of rat sciatic nerve were embolized with microspheres of 14 microns diameter at moderate ( $2 \times 10^6$ ) and high ( $5.6 \times 10^6$ ) doses. Rats were randomized to receive hyperbaric oxygenation treatment (2.5 atm 100% oxygen for 2 hours/day for 7 days beginning within 30 minutes of ischemia), or room air. End points for the embolized limb were (1) behavioral scores (0-11 in increasing levels of limb function), (2) nerve action potential of sciatic-tibial nerve, (3) nerve blood flow, and (4) histological grade as percentage of fibers undergoing ischemic fiber degeneration (0 = < 5%; 1 = 5-25%; 2 = 26-50%; 3 = 51-75%; 4 = > 76%). Nerve blood flow and nerve action potential were uniformly absent and more than 90% of fibers had degenerated in both control and treatment groups receiving high doses. Control and treatment groups receiving moderate doses were well matched by level of ischemia ( $8.5 \pm 0.3$  [N = 18] vs  $7.7 \pm 0.4$  ml/100 gm/min [N = 18],  $p > 0.05$ ) but were significantly different by behavior score ( $5.6 \pm 0.7$  vs  $9.2 \pm 0.5$  [N = 19],  $p < 0.001$ ), nerve action potential ( $1.4 \pm 1.0$  vs  $3.9 \pm 0.5$  [N = 6],  $p < 0.05$ ), and histology ( $2.4 \pm 0.4$  [N = 5] vs  $0.8 \pm 0.5$  [N = 4],  $p < 0.05$ ). On single teased fiber evaluation, the predominant abnormality was E (axonal degeneration). We conclude that hyperbaric oxygenation will effectively rescue fibers from ischemic fiber degeneration, providing the ischemia is not extreme.