Mechanical Drainage of Nerves that Supply Tissues Derived from the Branchial Arches

by

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Ethical Standards:

Procedures followed in these studies were in accordance with the Helsinki Declaration of 1975.

Permission to publish was given by the case subject.

Abstract

Objective: To introduce the concept that a certain form of non-vascular edema can exist at various sites in the nervous system innervating tissue derived from the branchial arches.

Clinical Features: *The signs and symptoms will vary depending upon which nerves are affected. The patient in this case is suffering from osteoradionecrosis of the mandible and associated symptoms.*

Intervention and Outcome: The testing and treatment are mechanical. The affected nerves in the areas were stretched manually in the long axis direction causing the edema to be 'squeezed out' of the extracellular matrix. Outcomes were measured mainly by the patients' symptom abatement, his personal observations, and independent interprofessional observations and investigations.

Conclusion: The patient reported a marked improvement. By draining this edema a normal blood flow returns to the affected nerves. Practitioners in all fields should be aware of this phenomenon.

INTRODUCTION

A form of non-vascular edema can affect the nervous system at any level^{1,2,3,4,5,6}. This condition is characterized by an expanded extracellular matrix (ECM) surrounding the affected nerves, too high a concentration of macromolecules in this matrix, and a paucity of blood elements in the area^{2,3,5}. It is my hypothesis this edema raises the hydrostatic pressure around the involved nerves so that blood flow in the vasa nervorum is compromised^{7,8}. Blood flow only travels in the direction of lower pressure. If the pressure in these areas of non-vascular edema approaches that of the local capillary pressure at systole then the flow is diminished. Macromolecules such as proteoglycans and glycoproteins in the extra cellular matrix are one cause of this edema^{1,2,3,4,5,6,9,10,11,12,13,14,15,16,17,18,19,20,21}. Local nerves and their associated connective tissue excrete these macromolecules when under stress^{13,22,23,24,25,26}. Areas particularly affected seem to be where neurogenesis and angiogenesis are taking place^{12,14,15,16,27,28,29,30,31}. The patient's symptom picture will vary depending on which nerves are affected.

The goal of treatment is to mechanically drain the excess fluid so the hydrostatic pressure is decreased and blood flow returns.

The case presented here involves nerves supplying tissues derived from the branchial arches. Many conditions affecting these tissues may be caused by this condition and therefore may be helped by manual therapy. It behooves the practitioner to understand the embryonic development of the six pharyngeal (branchial) arches, their pouches and clefts. The interrelationship of these nerves ($CNV_{1,2,3}$, CNVII, CNIX, CNX, CNXI, CNXII, the cervical plexus) and their association with arch cartilage, blood vessels, muscles, fascia, mucosa, and mesenchyme give diagnostic clues for avenues of treatment. For example, Meckel's cartilage relates the incus and malleus to the sphenomandibular ligament. By end range loading this ligament perhaps small branches of $CNV_{2,3}$ would have a reflex effect in the middle ear. The same thinking is true for the caudal arches. Conditions such as dysphasia and dysphagia may have a beneficial effect by loading involved tissues. Please refer to the Fall 2010 edition of *Massage Matters*, the official magazine of the Massage Therapy Association of British Columbia (<u>http://www.massagetherapy.bc.ca</u>). In it you will find several other case studies regarding the head and neck.

CASE REPORT

A fifty-one year old male presented with symptoms resulting from radiation therapy for tongue cancer. The chief complaints were osteoradionecrosis of the mandible, decreased hearing, a sore and stiff tongue, dysphasia, gums and tongue that won't heal, stiffness in the jaw and neck. These symptoms were constant. He started cancer therapy in April, 2010. Physical examination on November 2, 2010 revealed the following: dystrophy of the skin on the left aspect of the throat, a small and hypomobile tongue, an open lesion in the gum and tongue, dysphasia, decreased hearing, hypertonic pharyngeal and masticatory muscles and a forward head posture. My tentative diagnosis was non-vascular edema affecting local nerves and was caused by stress to those nerves and associated connective tissue due to radiotherapy. The trial of therapy consisted of stretching the involved nerves and tissues. I repeated the procedures on 4 occasions over a period of 3 weeks.

My examination of him in late November revealed less hypertonicity in his pharynx and muscles of mastication. The lesions on his tongue and gums had diminished in size and depth. His voice was lower in tone and greater in amplitude.

In early January, 2011 the patient reported the following. His jaw and pharyngeal muscles are much more relaxed and his TMJ has a much greater range of movement. His speech, swallowing and hearing have also improved. Some lesions in his oral cavity have almost disappeared. The severity and area of dystrophic skin on his lower neck has lessened.

Both his oral surgeon and his radio oncologist noted a rapid improvement since starting care here.

Table 1 Summary of results.

	Date of Onset	Frequency, Severity and Duration of symptoms	Intervention and Date(s)	Frequency, Severity and Duration of Symptoms
Case I Radiation	April, 2010	Constant	Nerve Stretching Nov.03, 09, 10, 15, 2010	Since late November a 75% abatement of his symptoms.

DISCUSSION

In cases of this type of non-vascular edema involving nerves in the periphery, traction of those nerves will cause excess fluid around them to be removed. Nerves are viscoelastic and stretching them will decrease their internal volume and increase the pressure within³³. The increased pressure from the loading procedure causes the fluid to leave the area. It is important for the traction to be held for several seconds and then repeated, allowing the excess fluid enough time to flow out. This is like 'wringing-out' a damp cloth. These kinds of procedures were used in treating this patient.



Fig. 1. This patient felt a warm and tingling sensation into his TMJ area immediately after the treatment. I believe this sensation was due to the blood flowing into the affected nerves.



Fig. 2 This patient felt a warming in his right ear afterwards.

I have been a practicing chiropractor for thirty-three years and first stumbled onto this phenomenon in 1998. Only in 2009 did I realize the entire nervous system may be affected by non-vascular edema and not just the occasional peripheral nerve. This more universal hypothesis motivated me to write this paper.

Over the last ten years I have had the opportunity to 'stretch' affected peripheral nerves on many occasions. Here, the nerves to the tissues associated with the viscerocranium is one case. Other examples include the primary ventral rami of the spinal nerves, including their plexi and end nerves, the primary dorsal rami of the spinal nerves, some cranial nerves and some visceral nerves. Obviously not all chronic mechanical problems have this pathology but those that do will respond quickly.

CONCLUSION

The subject experienced significant improvement. These results along with my experiences over the last ten years have allowed me to draw certain conclusions. The condition of non-vascular edema is real and this treatment is effective. Perhaps in the future microcirculation to the affected nerves will actually be measured.

This case represents a novel diagnosis and treatment protocol for many conditions seen in the dental surgeon's office. Temporomandibular dysfunction, chronic odontalgia, and tinnitus are some examples. The treatment described in this paper falls well within your scope of practice.

More generally, the affected nerves could be anywhere. They may include those to a tendon resulting in a tendinopathy^{3,5,6,9,10,11,20,21}, those to a disc resulting in an internal disc disruption syndrome^{27,34,35,36,37,38}, or those to the intestine as in an irritable bowel syndrome (Auerbach's and Meissner's plexi). Other nerves could include those of the brain and central nervous system resulting in conditions such as anosmia, migraines, tinnitus, post concussive syndrome, slow recovery from an ischemic stroke, and various psychiatric disorders^{1,2,12,13,14,15,16,17,25,32,39,40}.

Thousands of articles have been written about chronic problems affecting the musculoskeletal system, the viscera and the central nervous system. What is being presented in this paper is a new way to observe, test and treat many of those conditions. The testing for and treatment of non-vascular edema are both parts of the same process. If the patient responds, you have a good diagnosis. If there is no effect from the testing then the condition is due to something else and no harm was done.

Hopefully this paper will represent a starting point for future research.

REFERENCES

1) Chodobski A, Chung I, Kozniewska E, Ivanenko T, Chang W, Harrington JF, et al. Early neutrophilic expression of vascular endothelial growth factor after traumatic brain injury. Neuroscience. 2003; 122(4): 853-67.

2) Engel DC, Mies G, Terpolilli NA, Trabold R, Loch A, De Zeeuw CI, et al. Changes of cerebral blood flow during the secondary expansion of a cortical contusion assessed by 14C-iodoantipyrine autoradiography in mice using a non-invasive protocol. J Neurotrauma. 2008 Jul; 25(7): 739-53.

3) Mooney V. Overuse syndromes of the upper extremity: Rational and effective treatment. The Journal of Musculoskeletal Medicine. 1998 August; 11-15.

4) Negrini D, Passi A, Moriondo A. The role of proteoglycans in pulmonary edema development. Intensive Care Medicine. 2008 April; 34(4): 610-618.

5) Scott A, Lian O, Roberts CR, Cook JL, Handley CJ, Bahr R, et al. Increased versican content is associated with tendinosis pathology in the patellar tendon of athletes with jumper's knee. Scand J Med Sci Sports. Epub 2007 Dec 7.

6) Xu Y, Murrell GAC. The Basic Science of Tendinopathy. Clinical Orthopedics and Related Research. 2008 July; 466(7):1528-1538.

7) Abele H, Pieper KS, Herrmann M. Morphological investigations of connective tissue structures in the region of the nervus occipitalis major. Funct Neurol. 1999 Jul-Sept;14(3): 167-170.

8) Reina MA, Lopez A, Villanueva MC, de Andres JA, Leon GI. Morphology of peripheral nerves, their sheaths, and their vascularization. Rev Esp Anestesiol Reanim. 2000 Dec;47(10): 464-75.

9) Corps AN, Jones GC, et. al. The regulation of aggrecanase ADAMTS-4 expression in human Achilles tendon and tendon-derived cells. Matrix Biol. 2008 June;27(5):393-401. Epub 2008 Apr 2.

10) Corps AN, Robinson AH, Movin T, Costa ML, Hazleman BL, Riley GP. Increased expression of aggrecan and biglycan mRNA in Achilles tendinopathy. Rheumatology (Oxford). 2006 Mar;45(3): 291-4. Epub 2005 Oct 11.

11) Fu SC, Chan KM, Rolf CG. Increased deposition of sulfated glycosaminoglycans in human patellar tendinopathy. Clin J Sport Med. 2007 Mar;17(2):129-34.

12) Galtrey CM, Fawcett JW. The role of chondroitin sulfate proteoglycans in regeneration and plasticity in the central nervous system. J.Brain Res Rev. 2007; 1-18.

13) Haddock G, Cross AK et al. Brevican and phosphacan expression and localization following transient middle cerebral artery occlusion in the rat. Biochem. Soc. Trans. 2007 Aug; 35(Pt.4):692-694.

14) Hai J, Li ST, Lin Q., Pan QG, Gao F, Ding MX. Vascular endothelial growth factor expression and angiogenesis induced by chronic cerebral hypoperfusion in rat brain. Neurosurgery. 2003 Oct; 53(4):963-70; discussion 970-2.

15) Harris NG, Carmichael ST, Hovada DA, Sutton RL. Traumatic brain injury results in disparate regions of chondroitin sulfate proteoglycan expression that are temporally limited. J Neurosci Res. 2009 May 12; Epub 2009.

16) Jones LL, Margolis RU, Tuszynski MH. The chondroitin sulfate proteoglycans neurocan, brevican, phoshacan, and versican are differentially regulated following spinal cord injury. Exp. Neurol. 2003 Aug;182(2):399-411.

17) Kwok JC, Afshari F, Garcia-Alias G, Fawcett JW. Proteoglycans in the central nervous system: plasticity, regeneration and their stimulation with chondroitinase ABC. Restor Neurol Neurosci. 2008; 26(2-3):131-45.

18) Matsui F, Oohira A. Proteoglycans and injury of the central nervous system. J of Congenital Anomalies. 2004 Dec; 44(4): 181-188.

19) Properzi F, Lin R, Kwok J, Naidu M, van Kuppervelt TH, Ten Dam GB, et al. Heparan sulphate proteoglycans in glia and in the normal and injured CNS: expression of sulphotransferases and changes in sulphation. Eur J Neurosci. 2008 Feb; 27(3):593-604.

20) Riley GP, Harrall RL, Constant CR, Cawston TE, Hazleman BL. Prevalence and possible pathological significance of calcium phosphate salt accumulation in tendon matrix degeneration. Ann Rheum Dis. 1996 Feb;55 (2): 109-15.

21) Tom S, Parkinson J, Ilic MZ, Cook J, Feller JA, Handley CJ. Changes in the composition of the extracellular matrix in patellar tendinopathy. Matrix Biol. Epub 2009 Apr 14.

22) Leadbeater WE, Gonzalez AM, Logaras N, Berry M, Turnbull JE, Logan A. Intracellular trafficking in neurons and glia of fibroblast growth factor-2, fibroblast growth factor receptor1 and heparan sulphate proteoglycans in the injured adult rat cerebral cortex. J Neurochem. 2006 Feb;96(4): 1189-200. Epub 2006 Jan 17. 23) Ong WY, Levine JM. A light and electron microscope study of NG2 chondroitin sulfate proteoglycan-positive oligodendrocyte precursor cells in the normal and kainite-lesioned rat hippocampus. Neuroscience. 1999;92 (1): 83-95.

24) Pankonin MS, Sohi J, Kamholz J, Loeb JA. Differential distribution of neuregulin in human brain and spinal fluid. Brain Res. 2009 Mar 3; 1258: 1-11. Epub 2008 Dec 29.

25) Shimizu H, Ghazizadeh M, Sato S, Oguro T, Kawanami O. Interaction between beta-amyloid protein and heparan sulfate proteoglycans from the cerebral capillary basement membrane in Alzheimer's disease. J Clin Neurosci. 2009 Feb; 16(2):277-82. Epub 2008 Dec 16.

26) Thallmair M, Ray J, Stallcup WB, Gage FH. Functional and morphological effects of NG2 proteoglycan deletion on hippocampal neurogenesis. Exp Neurol. 2006 Nov; 202 (1): 167—78. Epub 2006 June 30.

27) Freemont AJ, Jeziorska M, Hoyland JA, Rooney P, Kumar S. Mast cells in the pathogenesis of chronic back pain: a hypothesis. The Journal of Pathology. July 2002; 197(3):281-285(5).

28) Kirpatrick ND, Andreou S, Hoying JB, Utzinger U. Live imaging of collagen remodeling during angiogenesis. Am J Physiol Heart Circ Physiol. 2007 Jun; 292(6):H3198-206. Epub 2007 Feb 16.

29) Krishnan L, Underwood CJ, Maas S, Ellis BJ, Kode TC, Hoying JB, et al. Effect of mechanical boundary conditions on orientation of angiogenic microvessels. Cardiovasc Res. 2008 May 1; 78(2):324-32. Epub 2008 Feb 28.

30) Kuslich S, Ulstrom C, Michael C. The tissue origin of low back pain and sciatica. Orthopedic Clinics of North America. 1991 Apr;22(2):181-187.

31) Mammoto T, Seerattan RA, Paulson KD, Leonard CA, Bray RC, Salo PT. Nerve growth factor improves ligament healing. J Orthop Res. 2008 Jul; 26(7): 957-64.

32) Bigler E D. Neuropsychology and clinical neuroscience of persistent postconcussive syndrome. Journal of the International Neuropsychological Society. 2008; 14, 1-22.

33) Millesi H, Zoch G, Reihsner R. Mechanical properties of peripheral nerves. Clin Orthop.1995 May;(314):76-83.

34) Koike Y, Uzuki M, Kokubun S, Sawai T. Angiogenesis and inflammatory cell infiltration in lumbar disc herniation. Spine. 2003 Sep 1; 28(17):1928-33.

35) Palmgren T, Gronblad M, Virri J, Kaapa E, Karaharju E. An immunohistochemical study of nerve structures in the annulus fibrosis of human normal lumbar intervertebral discs. Spine 1999; 24(20): 2075-2079.

36) Roberts S, Evans H, Trivedi J, Menage J. Histology and pathology of the human intervertebral disc. Bone Joint Surg Am. 2006 Apr; 88 (Suppl 2):10-4.

37) Simon BR, Wu JS, Carlton MW, Evans JH, Kazarian LE. Structural models for human spinal motion segments based on a poroelastic view of the intervertebral disc. J Biomech Eng. 1985 Nov; 107(4):327-35.

38) Yeung AT, Yeung CA. In-vivo endoscopic visualization of patho-anatomy in painful degenerative conditions of the lumbar spine. Surg Technol Int. 2006; 15:243-56.

39) Orre K, Wennstrom M, Tingstrom A. Chronic lithium treatment decreases NG2 cell proliferation in rat dentate hilus, amygdala and corpus callosum. Prog Neuropsychopharmacol Biol Psychiatry. 2009 Apr 30; 33(3): 503-10. Epub 2009 Feb 6.

40) Wang A, He BP. Characteristics and functions of NG2 cells in normal brain and neuropathology. Neurol Res. 2009 Mar; 31 (2): 144-50.