

Update 16

Loss of Joint Nerve Supply Impairs Healing & Promotes DJD

The neurological component of the Vertebral Subluxation (VS) has traditionally been the cornerstone of chiropractic theory. Chiropractors viewed the nervous system as the mediator of vitality to the individual organs and tissues. Moreover, in the original 'bone on nerve' model of VS, it was hypothesised that the nervous system played the central role in mediating all the deleterious effects of VS's and the benefits of chiropractor's adjustments.

However, during the later part of the twentieth century biomedical researchers increasingly focused on the biomechanical effects of joint dysfunction. Because much of the chiropractic community relied on medical basic science research to understand the effects of spinal adjustment, chiropractic researchers and practitioners seemed to become more focused on chiropractic as a means of improving joint dysfunction as opposed to optimising nervous system function.

Interestingly, more recent biomedical research has begun to focus on the nervous system and supports the fundamental observation of our profession's forefathers, that the nervous system is the key mediator of vitality and health to the body's individual organs and tissues.

Take for example the research into degenerative joint disease (DJD) over the last 20 years. The focus of that research has very much been on mechanical failure, and biochemical properties, of ligaments, discs, capsules and articular cartilage. Much less interest has been shown toward the relationship between the nervous system and DJD.

However that has started to change. Up until the mid 1990's existing models of how nervous system compromise might contribute to the development of DJD were limited by the technical difficulties of obtaining either highly selective or complete joint denervation in a minimally invasive fashion. Then in 1997 researchers from the University of Toronto, Canada, undertook experiments to determine the feasibility of using the newly described method of selective neuronal lesioning with injected immunotoxin as a means of creating a more tractable model of so called neuropathic arthritis. Retrograde tracing revealed that the knee joint of Wistar rats is supplied by 581 joint afferents. Injection of rat knee joints with an immunotoxin resulted in the selective ablation of an average of 88% of the joint afferents that are normally found in the ipsilateral L3 and L4 ganglia. (1)

In a further study the same authors made histological examinations of knee joints of mice at various ages and showed that loss of joint innervation always preceded histological changes of cartilage degeneration. The mice usually developed a mild form of osteoarthritis, but surgical ablation of joint innervation caused the development of severe patellofemoral osteoarthritis. The authors conclude that their findings are consistent with the hypothesis that a loss of joint innervation may contribute to the development of DJD. (2)

In another study the authors hypothesized that a loss of joint afferents is involved in the pathogenesis of osteoarthritis. To test their hypothesis, they denervated knee joints of 16 rats at age 2 months, by intra-articular injection of the immunotoxin. The immunotoxin killed neurons after retrograde axonal transport to the cell body. At 16 or 24 months follow-up, each joint was histologically assessed and assigned an osteoarthritis score. At follow-up, the number of joint afferents had spontaneously decreased by 40% in control knees and 70% in denervated knees. They found that control knees developed arthritic changes with aging. However, denervated knees had far more severe changes, as evidenced by a 54% higher average DJD score than control knees ($P = 0.0016$, both groups 16 knees). The authors propose that their results suggest a loss of afferents predisposes a joint to DJD. To explain the mechanism causing DJD, they suggest denervation permits aberrant joint loading, either by disturbing neuromuscular joint control, or by inducing joint laxity after neurogenic loss of tissue homeostasis. (3)

In another study researchers used high-resolution nuclear magnetic resonance (NMR) techniques to compare the effects of unilateral knee joint denervation on the biochemical profiles of synovial fluid in a bilateral canine model of osteoarthritis. Paired synovial fluid samples were obtained from seven dogs all of which had previously undergone bilateral anterior cruciate ligament transection, unilateral knee denervation and contralateral sham nerve exposure. All synovial fluid samples were then analyzed using NMR Spectroscopy to assess differences in endogenous metabolite levels between the paired fluids. The results indicate statistically significant differences in the biochemical profiles of the synovial fluids from denervated with respect to control knees. This study lends support to the principle of neurogenic acceleration of DJD in that the observed differences in metabolite concentrations found in the denervated knee fluids seem to correlate with metabolic changes resulting from aggravation of the DJD process caused by joint denervation. (4)

The lead author of most of the above body of research, Dr. Paul Salo recently commented on his similar research with rabbits that has not yet been published,

“We found that denervation markedly impairs the healing of ligament injuries in the rabbit model. Denervated joints showed dramatically lower blood flow, reduced angiogenesis and inferior mechanical properties when compared to normally innervated joints.” (5)

Interestingly, the above biomedical research has focused on the relationship between the nervous system and articular health. It supports the fundamental observation of our profession’s forefathers, that the nervous system is a key mediator of vitality and health for the body’s individual tissues.

References:

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2. Salo PT, Seeratten RA, Erwin WM, Bray RC. Evidence for a neuropathic contribution to the development of spontaneous knee osteoarthrosis in a mouse model. *Acta Orthop Scand* 2002;73:77-84.
3. Salo PT, Hogervorst T, Seerattan RA, Rucker D, Bray RC. Selective joint denervation promotes knee osteoarthritis in the aging rat. *J Orthop Res* 2002;20:1256-64.
4. Damyanovich AZ, Staples JR, Marshall KW. ¹H NMR investigation of changes in the metabolic profile of synovial fluid in bilateral canine osteoarthritis with unilateral joint denervation. *Osteoarthritis Cartilage* 1999;7:165-72.
5. Paul T. Salo, M.D., FRCSC. Associate Professor, Department of Surgery, Faculty of Medicine, University of Calgary. <http://www.boneandjoint-training.ca/salo.htm>