

## ISSUES IN SPINAL CORD PLASTICITY FOR THE MANIPULATIVE SCIENCES

Canadian Memorial Chiropractic College  
Convenor: Dr. Howard Vernon, DC, FCCS(C)  
June 8, 1991, Toronto

### Introduction

Spinal cord plasticity is the currently used term to describe changes which occur in the functional characteristics of spinal cord neuronal pools sufficient to alter behavioural mechanisms which are subserved by these neuronal pools. These changes can occur at all levels – single neuron, mono-synaptic reflex, poly-synaptic circuits, as well as in the peripheral afferents, dorsal root ganglion, dorsal root entry zone, dorsal horn and ventral horn cells.

Functional changes involve changes in behavioural characteristics as well as in the neurochemical mechanisms which subserve such mechanisms.

Historically, chiropractic scientists have held to a "neurobiological view" of the workings of human health. While Palmer spoke presciently, but ambiguously about "tone", one of the great contributions to this view arose out of the osteopathic research of Irwin Korr and his colleagues. The now-famous "facilitated state" concept is a primary example (perhaps a founding example) of spinal cord plasticity.

As such, the new terms "plasticity" should be highly relevant to modern-day chiropractors, and interest in it should be just as high as it was in earlier days for Korr's work. The fact is that, just as there has been modernization of Palmer's work, neuroscience has advanced well beyond Korr's work, and we as researchers and as general practitioners need to keep abreast of these developments. In fact, it has been a common experience that our unique chiropractic perspective when applied to basic science methods in this area, yields some distinct, important and clinically applicable research directions. As such, we should be proud of our past scientific perspectives, but we should be diligent in applying them now, to secure our place in their future shaping.

To be honest, chiropractors have had very little to do with what is viewed as the "chiropractic scientific perspective". We have borrowed mightily from others and we have created a patchwork of theories and ideas. Despite this, several "chiropractic" investigations are underway, and several researchers who are either chiropractors or are employed by chiropractic colleges are involved in this field currently.

On June 8, 1991, eight such experts met at the Canadian Memorial Chiropractic College under the sponsorship of the Chiropractic Foundation for Spinal Research, and presented original reports of theoretical work, methodological models and results of experiments. In the afternoon, three very lively round tables were formed on 1) Pain, 2) Autonomic Effects and 3) Motor Effects. Audience participation was solicited, and excellent discussions ensued. These resulted in a clearer focus on the issues directly relevant to chiropractic clinical science and a

clearer agenda for future investigations. Finally, a new camaraderie was developed between the experts which can only bode well for future developments in chiropractic neuroscience.

## SPINAL CORD PLASTICITY – HISTORY AND CONCEPTS

Howard Vernon, DC, FCCS(C)  
Canadian Memorial Chiropractic College  
1900 Bayview Avenue, Toronto

The phenomenon of referred pain from spinal origins has been described since the later 1800's with the work of McKenzie and Head. In the early 1900's, Goldthwait, as well as early chiropractic authors expanded on the concept. One of the prevailing theories advanced for this phenomenon involved "hyperactivity of the spinal cord". This theory culminated in the work of Korr et al. in the 1940's–50's, in the form of the concept of "central facilitation". Both osteopathic and chiropractic experts have adopted this as the model of "subluxation – induced" physiopathology.

Notwithstanding these developments, modern neuroscientists have only just begun to study the phenomenon of deep somatic pain (as distinguished from cutaneous pain), and only very recently, as with the work of Wall and Woolf in 1984, have the central effects of deep noxious pain on spinal dorsal horn neurones begun to be elucidated. Several findings of the recent decade compel us to consider a much greater degree of complexity of spinal cord processing of deep pain transmission. For instance, areas of experimentally-induced hyperalgesia remain resistant to peripheral analgesics for significant periods of time.Coderre et al. have found that mustard oil-induced pain injury creates signs of contralateral hyperalgesia (reduced latency for withdrawal reflex) which persists even when peripheral nerves on the injured side are transected. Wall and Woolf showed that dorsal horn neurones remained excited for much longer periods after C-fibre stimulation from muscle and joint nerves as compared to cutaneous nerves.

Woolf has proposed a theoretical model for these effects known as "central sensitization". These changes in the spinal dorsal horn are characteristic of neuroplastic changes in the presence of severe and/or prolonged noxious input from deep pain sources. This model is highly reminiscent of Korr's original concept.

The consensus of studies reporting this phenomenon includes the following phenomena: Wind-up and sensitization of dorsal horn neurones; lowering of their thresholds for activation; unmasking of latent, "silent" synaptic connections; facilitation of convergent inputs; expansion of their receptive fields; and long-lasting discharge activity (ectopic, oscillating) post-stimulus.

We are compelled to ask if this model, as an outgrowth of the earlier one proposed by Korr, is not applicable in explaining subluxation-induced effects? If so, is this model comprehensive



in its inclusion of motor and autonomic effects as was the earlier model? The answers to these and other questions pose the great challenges to our current researchers.

# **SPINAL FIXATION: A BASIS FOR THE EFFECTS OF MANIPULATION AND ADJUSTMENT THERAPIES**

Michael M. Patterson, PhD  
College of Osteopathic Medicine  
Ohio University, Athens, Ohio 45701

One of the basic tenants of both osteopathy and chiropractic is that the body is an adaptive mechanism, which adjusts to the stresses and demands of everyday life in complex and often subtle ways. Thus, change in body function is in reality the basis of homeostasis – the continuing struggle of the body to maintain the internal milieu. Without the ability to change its function to meet the continually changing demands of the environment and indeed of the body's own demands upon itself, homeostasis cannot be properly maintained and the seeds of disease are planted. What factors could decrease the body's ability to respond to change and hence to maintain health? It is to this I wish to address my remarks.

In the normal joint, there are certain numbers of joint receptors which are active, reporting the joint's activity to the central nervous system. When joint inflammation occurs, the number of active joint receptors increases dramatically, perhaps by a factor of 10 (Schaible and Schmidt, 1988). This dramatic increase in input to the spinal cord and higher centers of the central nervous system due to joint malfunction may seem inconsequential until one begins to examine the potential effects of such changes in sensory input on spinal reflex function and long-term excitability, and of the tremendous interactions between somatic sensory inputs and autonomic outflows from the cord. It has been shown that up to 80% of spinal interneurons which receive input from somatic afferents also receive input from visceral afferents. Thus a tremendous increase in somatic input could not help but disrupt the normal autonomic outflow patterns, by itself disrupting normal body function and hence homeostasis. However, such dramatic input changes can have other, longer-lasting effects on the reflex circuits of the spinal cord.

For many years, the processes of habituation and sensitization have been studied in spinal reflexes. Habituation is the process whereby the response of a reflex circuit to a steady input decreases over time, with sensitization being the opposite, the increase of output to a continued input (Groves and Thompson, 1970). Both of these appear to be transient effects. In 1929, Di Giorgio described a phenomenon which suggested that sensitization could be maintained for long periods rather than rapidly disappearing after the initiating stimulus was discontinued. She demonstrated that in anesthetized animals, a cerebellar lesion would cause a limb flexion which could be maintained for hours. While this is perhaps not surprising, the remarkable thing

was that after the spinal cord was severed in such an animal, the hindlimb which was flexed remained to some degree flexed rather than returning to a flaccid paralysis, which would have been expected following the spinal transection. The spinal motor reflex pathways had been altered, retaining the increased excitability caused by the heavy descending influences of the injured cerebellum. This phenomenon became known as "spinal fixation" and was treated as an unimportant oddity for years. However, Chamberlain, Halick and Gerard (1963) showed that fixation could occur with only about 45 minutes of sustained spinal bombardment (see Patterson, 1976 for a review). We began to look at the process of spinal fixation in the early 1980s and have shown that it can occur with sensory input from the skin of the hindlimb, and is not dependent on cerebellar outflows. The resultant heightened spinal reflex excitability can last for days, and is truly a spinal phenomenon, not a change in muscle mechanics or some other peripheral change (Patterson and Steinmetz, 1986).

We have more recently shown that the spinal reflex excitability increases resulting from heavy doses of peripheral input can occur in a few minutes, and can be augmented by stress and other factors (e.g., Steinmetz, Beggs, Molea and Patterson, 1985; unpublished data). It seems quite likely that the basis of the fixation is a sensitization which becomes "fixed" in the spinal circuits. Such an alteration of spinal excitability would disrupt the normal input-output relationships at that level of the cord. Thus, the resetting of the spinal circuits to a higher level of excitability would restrict the ability of these circuits to respond normally to maintain the constant milieu of the body, and hence to maintain health.

It should be obvious that manipulation and adjustment treatments can have a beneficial effect on all phases of this process, first by restoring the joint to its normal structural relationships, thus reducing the barrage of sensory inputs to the cord, and second by restoring proper movement characteristics in the joint, thus reducing the continued and repeated improper tensions around the joint. These measures will also result in reduced inflammation, thus further decreasing sensory input to the cord. The immediate effect of the treatment or adjustment is then reduced sensory input, thus reducing sensitization, which usually begins to abate within seconds. The secondary reduction in inflammation allows the reflex excitabilities to further subside with time. The underlying longer-term excitability increase, or fixation, will take longer to subside. Indeed, it may never return to normal levels, thus accounting for the increased susceptibility, often for days, months or years, of somatic areas which have been in distress, to recurrence of the problem. Thus, the sooner a problem is treated to relieve overactive afferent inputs, the less severe should be the aftermath.

This brief account of fixation and overlying sensitization of spinal circuits as accounting for much of the distress seen with many subluxations and somatic dysfunctions can serve as a basis for further analysis of the far-flung effects of joint dysfunction of body function. The spinal cord is an active partici-



pant in the appearance of abnormal reflex patterns which restrict the ability of the body to maintain homeostasis. Manipulation and adjustment therapies are uniquely positioned to restore the body's ability to meet demands of constant change, and hence to restore and maintain health.

## References

- Chamberlain TJ, Halick P, Gerard RW. Fixation of experience in the rat spinal cord. *J Neurophysiol* 1963; 26:662-673.
- Di Giorgio AM. Persistenza nell'animale spinale, di asimmetrie posturali e motorie di origine cerebellare. *Nota I-III Archivio di Fisiologia* 1929; 27:19-42.
- Groves PM, Thompson RF. Habituation: A dual-process theory. *Psychological Review* 1970; 77(5):419-450.
- Patterson MM. Mechanisms of classical conditioning and fixation in spinal mammals. In: Reisen AH, Thompson RF. (eds.). *Advances in Psychobiology*, vol. 3. New York: Wiley, 1975.
- Patterson MM, Steinmetz JE. Long-lasting alterations of spinal reflexes: A potential basis for somatic dysfunction. *Manual Medicine* 1986; 2:38-42.
- Steinmetz JE, Beggs AL, Molea D, Patterson MM. Long-term retention of a peripherally induced flexor alteration in rats. *Brain Research* 1985; 327:312-315.
- Schaible HG, Schmidt RF. Direct observation of the sensitization of articular afferents during an experimental arthritis. *Proceedings of the Vth World Congress on Pain*. Elsevier 1988: 44-50.

## MECHANISMS OF SPINAL CORD PLASTICITY IN THE TRIGEMINAL SYSTEM

Barry J. Sessle

Faculty of Dentistry, University of Toronto  
Toronto, Canada M5G 1G6

Recent research has identified nociceptive neurones in the V brainstem nucleus that have features indicating their crucial role in our ability to localize an acute orofacial pain and sense its intensity and duration. Many of the nociceptive neurones however receive convergent inputs from afferents supplying not only face or mouth, but also muscle, TMJ or even the neck. These features may be involved in mechanisms underlying the spread and referral of pain and the manifestation of chronic pain. Some of the neurones for example show neuroplastic changes as a result of inflammatory and deafferentation conditions affecting peripheral nerves. Injection into masticatory muscles of the inflammatory irritant mustard oil, a C-fibre stimulant, produces in V brainstem nociceptive neurones increases in their cutaneous receptive field and excitability that may be involved in the spread of pain and hyperalgesia that often result from trauma and inflammation to deep craniofacial tissues. Of further clinical significance are findings that V brainstem neurones can also be altered by therapeutic procedures or trauma associated with deafferentation: endodontic therapy produces pulp deafferentation resulting in expansion of

neuronal receptive fields and increased incidence of spontaneous activity and abnormal responses to orofacial stimuli. Since a number of clinical problems manifesting chronic pain may be linked to deafferentation, such changes might be involved in the development of several chronic orofacial pain conditions.

## DORSAL HORN PLASTICITY RELATING TO LOW BACK PAIN

WJ Roberts, RG Gillette and RC Kramis

Dow Neurological Sciences Institute  
\* Good Samaritan Hospital and Medical Center  
Portland, Oregon, 97212, USA

Acute and chronic low back pain are major medical problems in the industrialized world. However, the origins of these pains are poorly understood, and few studies of nociceptive systems have been directed specifically toward the lumbar region.

We have begun studies of somatosensory systems serving the low back in pentobarbital anesthetized cats and have found specific differences in afferent projections into the dorsal horn and in the characteristics of dorsal horn neurons subserving proximal regions (including the lumbar region, hip, and/or proximal hindlimb) compared to the analogous elements serving the distal hindlimb.

The most distinctive difference observed was in the complexity of afferent input onto dorsal horn neurons. Most (72% of 118) of those having proximal receptive fields received excitatory input from both dorsal and ventral compartments, including skin, and paraspinal or proximal hindlimb muscles, and other deep tissues such as zygapophysial joint capsules, tendons, periosteum, intervertebral discs, and dura. Fewer neurons had receptive fields restricted to just skin (23%) and very few to just deep tissues (5%). Most previous studies of neurons with distal receptive fields reported a predominance of cutaneous input.

The receptive fields of neurons having proximal receptive fields were much larger than those reported for neurons with distal fields, typically including part of the low back, and hip and proximal hindlimb. Many (82%) had receptive fields that crossed the midline, unlike the strictly ipsilateral receptive fields of neurons with distal fields.

The patterns of responding to cutaneous stimuli in neurons with proximal receptive fields were qualitatively similar to those of neurons with distal receptive fields. WDR (multireceptive units (69%) typically had an area of low mechanical threshold superimposed on a larger high-threshold area. However, responding to mechanical stimulation of muscles, joint capsules and other deep tissues appeared to be continuously graded over a wide range of stimulus intensities. Other neurons responded maximally to low intensity stimuli (8%) or were initially nociceptive specific (22%).

Noxious stimulation (bradykinin or 6% saline injections, or 60s skin pinch) were used as a conditioning stimulus in testing



of 34 units. Most (65%) showed persistent increases in responding to non-noxious mechanical stimuli and expansions of both low-threshold and high-threshold receptive fields. In some, the high-threshold field expanded from the hip region to include the entire hindquarter and low back. Significantly, expansions of deep-tissue fields were also noted. The expanded receptive fields retracted slowly over several hours in some units. Five neurons that were initially nociceptive-specific became clearly responsive to non-noxious stimulation after noxious conditioning. Injection of algogens onto a zygapophysial joint was, for many units, the most effective conditioning stimulus.

In summary, most nociceptive neurons recorded from the lateral dorsal horn in this study had very complex receptive fields in both skin and deep tissues of the low back, hip and proximal hindlimb. These neurons showed marked increases in responding and expansion of receptive fields following brief noxious conditioning stimuli. Such neurons are well suited to subserve diffuse low back pain, which can occur after focal injury to any one of several lumbar tissues.

#### ELECTROMYOGRAPHIC (EMG) RESPONSES OF RAT NECK AND JAW MUSCLES TO INFLAMMATORY IRRITANT

BJ Sessle, X-M Yu, JW Hu, H Vernon\*, DA Haas  
Faculty of Dentistry, University of Toronto,  
Toronto, Canada M5G 1G6

\* Canadian Memorial Chiropractic College  
Toronto, Ontario, Canada M5G 1G6

An EMG study was carried out in 9 anesthetized rats to determine if the activity of neck and jaw muscles could be influenced by the injection of the inflammatory irritant mustard oil into the deep tissues surrounding the C2 vertebra. EMG baseline activity was recorded bilaterally in the deep neck muscles and ipsilaterally in trapezius, masseter and digastric muscles. Vehicle injection (20  $\mu$ l, mineral oil) usually induced a transient (1.5 min) increase in EMG activities of all 5 muscles. In contrast, injection of mustard oil (20  $\mu$ l, 20% allyl isothiocyanate) produced an EMG increase in all 5 muscles that was significantly greater in magnitude (50%,  $P < 0.05$ ) and longer in duration (3 min,  $P < 0.05$ ). Furthermore, some muscles often showed a second phase of activation which appeared 3.5–20 min after the mustard oil injection and lasted 15–20 min. These effects suggest that inflammatory irritants injected in deep periarticular tissues of the neck may reflexly enhance the activity of muscles distant from the injection site. Such effects may be related to increased muscle activity associated with trauma to deep tissues. Supported by NIH grants DE04786 and DE09559, and by the National Institute for Chiropractic Research.

#### PRODUCTION OF VERTEBRAL LESIONS AS A MIMIC OF SUBLUXATION IN RABBITS AND THEIR PHYSIOLOGICAL CHARACTERIZATION

KF DeBoer

Palmer College of Chiropractic  
Institute of Graduate Studies and Research  
Davenport, Iowa

Manual and surgical models of subluxations in rabbits, and the physical response to them has been the focus of our laboratory for the past 10 years. While one method of inducing "subluxations" using a Grostic type gun yielded a minimally satisfactory vertebral lesion (VL), later work using manipulation by hands only, a la the early osteopathic work of Burns and others, proved unsatisfactory. However, we have been successful to a marked degree in inducing an acute vertebral lesion by momentary manual rotatory and anterior pressure on the thoracic spine of normal rabbits. We found an immediate and dramatic decrease in gastric motility, which returned to normal upon release of the pressure. Recent work has focused on a surgical model, in which 3 adjacent vertebral segments are misaligned slightly and also fixated by means of an implanted steel bar. Palpatory, visual, X-ray, biochemical, biomechanical and histological data have been obtained from induction of VL by this means.

Biochemical analysis of the locally lesioned vertebral motor unit structures has also been preliminarily made, most extensively of ascorbate free radical concentrations from the intervertebral foramen (IVF), spinal cord and intervertebral disc (IVD). Ascorbate radicals were elevated in tissues known to be inflamed and the first few animals analyzed showed elevations in the lesioned areas. This mechanical model should serve as a useful first generation model to evaluate some of the "subluxation's" major characteristics.



# EPILEPSY

## Think Epilepsy. Think Research.

Epilepsy Canada  
1470 Peel St.  
Suite 745  
Mtl., Qc. H3A 1T1  
Tel: (514) 845-7855  
Fax: (514) 845-7866

Give  
generously.