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CHIROPRACTIC (GENERAL)

A Fresh Look at the Meric System and Modern Neuroscience

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The use of manipulation in the management of biomechanical disorders of the spine may now be regarded as a mainstream approach across health care. However, the management of visceral disorders through treatment of the spine remains controversial, and the association of particular spinal regions with specific visceral disorders, a core precept of the meric system, is regarded with considerable scepticism outside of chiropractic.

It is, of course, appropriate to critique and thereby refine our understanding of how the human body works in health and disease. Unfortunately, in all health care disciplines, ideas may come into and go out of vogue without much reference to the available evidence. Chiropractic's meric system is a good example of this. As such, it is appropriate to take a fresh look at the diagnostic rationale of the meric system in light of what is currently known about the neurobiological basis of spinovisceral interactions.

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The Meric System

The meric system had its origins in D.D. Palmer's observations that the adjustment of specific spinal segments reliably gave relief of particular disorders.[1] The formalization of specific associations into a diagnostic system – for example, that subluxation of the ninth thoracic vertebra would be associated with adrenal disease – is credited to B.J. Palmer and James Wishart circa 1910.[2] The early formulations of chiropractic theories were influenced by, among other factors, clinicians' own observations and the basic scientific knowledge of the day.

In particular, early chiropractors were aware of the segmental innervation of the viscera and likely understood that the sympathetic nervous system had a role in both the development and ongoing regulation of dependent organs. Indeed, D.D. Palmer made frequent reference to the function of the sympathetic nervous system in his seminal text.[1]

Thus, early expressions of the meric system held that subluxation of a vertebra would impinge on the spinal nerve(s) originating at the subluxated segment, thereby interfering with sympathetic output to organs that received innervation from that spinal level. From the point of view of neuroanatomy, the system is, in broad strokes, quite logical.

The Concept of Impingement

Certain elements of the meric model, however, remain in contention. In fact, the very concept of subluxation as a bone out of place and impinging on a nerve remains a subject of debate, even within the chiropractic profession. If, for purposes of discussion, we begin with the Palmer model

of the subluxation, it has been argued that this lesion is unlikely in general to cause nerve root impingement because of the large amount of free space in the intervertebral foramen.[3] Other authors, however, point out that extraforaminal ligaments, which naturally tether the spinal nerves at the foramena, may also contribute to impingement with subluxation,[4] and that structural changes of aging are associated with, for example, reduced free space in the lateral recess.[5]

Indeed, lateral stenosis with symptoms of nerve root impingement is a relatively common phenomenon,[6] with radiculopathy attributed to ischemic compression of nerve roots.[7] Further, spinal manipulation and strategies to alter spinal posture and movement appear to have some positive clinical effects.[8] Thus, while it is difficult to say that in any particular patient a subluxation is impinging on a given spinal nerve, the general concept of vertebral subluxation causing nerve root or spinal nerve impingement in some proportion of patients has validity.

Somatotopic Relationships

A separate issue with the meric system is the matter of somatotopy: the proposition that subluxation at certain segmental levels is linked to disease in specific organs. On first blush, this seems like a reasonable idea, given the geometric anatomical distribution of the peripheral sympathetic nerves. Thus, one can imagine impingement of one spinal nerve compromising sympathetic outflow to one or a small set of organs, causing disease in that – and only that – organ or set of organs.

This rationale becomes uncertain, however, when one begins to look at the microscopic structure and the physiology of the autonomic nervous system. Regarding the structure of the peripheral sympathetic nervous system, while indeed it seems that particular viscera are, across the population, served by the same one or two peripheral spinal nerves, microscopic examination paints a somewhat different picture.

Hence, by way of example, the adrenal gland seems to be served overwhelmingly by sympathetic efferents from the ninth thoracic nerve. However, when one actually traces the individual axons terminating on an adrenal gland, one finds that they arise from a half-dozen or more spinal segments, converging in one or two paraspinal ganglia (or even more peripherally) well away from the intervertebral foramena.[9] Therefore, a subluxation of the ninth thoracic vertebra would, at most, impinge on only a minority of nerve fibres to the adrenal gland – not a very convincing mechanism of disease.

Beyond Impingement Models

Early iterations of the meric model were based on interference with nerve outflow, ignoring the role of afferent information coming in from the periphery. This limited point of view is surprising in the historical context – the basic principles of reflex physiology had not yet been laid down when chiropractic was being founded.

Through the first half of the 20th century, however, chiropractic and osteopathic researchers began to speculate that reflex mechanisms might also be responsible for the effects of spinal lesions. Thus, aberrant information from dysfunction at a single spinal level would reflexly alter the behaviour of spinal motor neurons, particularly sympathetic preganglionic neurons, at that level (and perhaps one or two adjacent levels). By extrapolation from what was known about somato-somatic reflexes, this mechanism was seen as a good candidate for producing localized responses in viscera. To expand on the analogy to somato-somatic reflexes, when you tap a patellar tendon,

the leg on that side jumps, but you don't expect to see a response in the contralateral leg or in the arms.

Animal research supports this model of the localized reflex, with the classical text by Prof. Akio Sato citing approximately 800 experimental studies demonstrating somatovisceral reflexes.[10] Further, in the relatively few studies looking at spinal pain, it was indeed found that stimulation at any given spinal level was preferentially associated with changes in the behaviour of viscera which received their sympathetic innervation from that level.

These results, however, come with a caveat that may resonate with chiropractors in particular – spinovisceral reflexes tend to be dampened or even completely masked when communication between the brain and spinal cord is intact; it is only by compromising descending inhibitory influences from the brain that these spinovisceral reflexes are fully liberated.

A Synthesis of the Neurological Evidence

Common sense tells us that every biomechanical dysfunction of the spine cannot cause visceral disease. If our species were that fragile, we would have become extinct long ago. On the other hand, all of us who have been in practice for any length of time have seen instances which convinced us that spinal dysfunction had provoked visceral disorders in some patients. The modern neurosciences help us to address important questions about these phenomena, including 1) Are the perceived somatotopic relationships real? and 2) Why some patients and not others?

On balance, the evidence is that certain aspects of the meric model are quite robust. On the basis of the microscopic anatomy and the physiology of the sympathetic nervous system, it is rational that there should be some kind of somatotopic relationship – lesions in one region of the spine should preferentially affect a particular set of visceral organs. The relationship should not be as digitally precise as early proponents believed, but the relationship should exist in broad terms.

Furthermore, the research predicts that most people should not develop visceral disease from spinal dysfunction most of the time. It would appear that the pathological effects of spinal dysfunction are most likely to express themselves when there is a loss of moderating influences from higher centres. The exemplar for this model is the sympathetic dysreflexia seen in patients with high spinal-cord injuries; in these patients, relatively trivial stimulation below the level of injury can provoke severe – even fatal – paroxysmal hypertension.

Thus, there is a growing body of neurobiological evidence in support of the use of chiropractic strategies in the management of some visceral problems, as for some musculoskeletal problems. This article has not examined the clinical research, but most practitioners will understand that there is a need for more (and more robust) clinical studies of chiropractic care for visceral disorders, as for musculoskeletal disorders.

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