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Facilitated Segments: a critical review

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The concept of spinal facilitated segments has dominated osteopathic neurophysiology for over half this century. This concept has been at the heart of osteopathic teachings and is often used both in clinical diagnosis and as part of the rationale of treating different musculo-skeletal and visceral conditions. Surprisingly, such an important subject has never been criticised: the existence of facilitated segments and their relevance to manual therapy or osteopathic medicine has never been questioned. This article re-examines the original studies of Korr, Denslow and their co-workers, aim to identify what has been demonstrated in these studies and to reinterpret their findings in the light of current knowledge of neurophysiology.

The spinal facilitation concept

In principle, the facilitated segment was described as a specific area of the spinal cord that was capable of organising disease processes. It was a very simplistic model: it had two input and two output routes. The input routes were sensory from musculo-skeletal and viscera. The output routes were the motor efferents to muscle and autonomic motor to sweat glands, blood vessels and viscera. Inside the spinal cord it was suggested that abnormal activity in one area of the spinal cord could spread to adjacent areas. The facilitation process would be initiated when aberrant sensory information from an area of damage or pathology (muscle or viscera) was conveyed via the afferents to the spinal cord. This would alter the neuronal activity at the same segmental level and might spread to adjacent areas of the spinal cord affecting spinal centres not directly related to the original injury. For example, a musculoskeletal injury could reach the spinal cord through its afferent connection causing spinal facilitation or sensitisation to take place. Because of the anatomical proximity of the motor and autonomic spinal centres, this spread of excitation would eventually involve these lateral centres. This in turn would alter the segmental autonomic activity leading to changes in vasomotor, sudomotor and visceral activity. The reverse could happen too: through the same neurological mechanisms a pathological condition in the viscera could end up affecting skeletal muscle activity.

Even before examining the original research, we can see that there are several problems with the facilitated segment model:

a. The descending influences from higher centres were not included in the model, although they have profound segmental influences. This omission is unrealistic - the spinal centres do not work in isolation from the higher centres. Both movement and autonomic activity are heavily organised from above the spinal cord (Sherrington, 1906; Folkow, 1956; Bard, 1960; Brown, 1968; Ganong, 1981; Schmidt, 1991).

b. Outside the spinal cord afferent and efferent connections are, anatomically, highly segmental. However once in the spinal cord all anatomical specificity is lost (Luscher & Clamann, 1992). Motoneurons of several muscles are intermingled within the ventral horn and distributed over several segments up or down from the point of exit (efferent peripheral nerve). Similarly afferents from one area or muscle, once in the spinal cord, tend to diverge up and down over several segments terminating on many different motoneurons and interneurons (Luscher & Clamann, 1992). For example, spindle afferents from one muscle connect with motoneurons of other muscle groups (Eccles et al, 1957; Eccles & Lundberg, 1958). This implies that if lateral spread of sensitisation does take place, it will not necessarily be segmentally specific.

c. The facilitation model creates a biological paradox which is not supported either by research or by clinical observations. If damage in muscle caused spinal facilitation and consequently visceral dysfunction, it would mean that each time we damage our muscles it would automatically result in some visceral dysfunction. In this scenario common conditions, such as delayed-onset muscle soreness which is associated with muscle damage (Bobbet et al, 1986; Ebbeling & Clarckson, 1989), would inevitably lead to visceral dysfunction. Yet, 'viscerally' speaking, most sports people are fairly healthy. They do not seem to develop visceral dysfunction in response to acute or chronic musculo-skeletal conditions.

The original research

Korr, Denslow and their co-workers were the first to describe the facilitated segments in osteopathic medicine. The original research consisted of several studies which were carried out on a large number of *normal* healthy subjects. They used different experimental set-ups and were able to demonstrate the following findings:

1. Varying motor thresholds - pressure over the spinous processes produced reflex muscle contraction at and close to the segment. In some segments this response was exaggerated (Denslow et al, 1947). Every person had an individual pattern of response.
2. Varying levels of skin conductivity - there were differences in the sweating pattern of the backs of all normal individuals (Korr et al, 1958). This suggests increased activity of the sweat gland, implying altered sympathetic activity.
3. Varying levels of vasomotor activity - using temperature and light sensors they were able to demonstrate that all normal subjects have individual vasomotor activity which is changed in different parts of the back (Wright et al, 1960).
4. Viscerosomatic changes - sometimes, known visceral pathologies manifested segmentally as increased skin conductivity (Korr et al, 1964).
5. Each individual had a unique thermal pattern with some common patterns shared by all normal subjects (Wright & Korr, 1965).

Contrary to commonly held belief, they did not demonstrate the following:
1. They did not show facilitation - most of the studies were carried out on normal healthy subjects. In all subjects they found varying levels of neurological activity at different segmental levels. This is a complex situation to begin with: if the subjects were healthy how come they all displayed a supposedly neuropathological state of facilitation? If the biological norm is that healthy subjects all show signs of facilitation, it implies that the regional changes observed probably represent the normal variability of a highly complex system rather than a facilitation phenomenon. Such variability can be demonstrated anywhere in the body. For example, if you prod different parts of your own leg, you will find some areas are more tender, with the muscles feeling stiffer, and if you press hard enough you may make the muscle contract to evade pain and discomfort. When this procedure was applied to the spine, as Denslow et al (1947) did, it was very attractive to view it as segmental facilitation.
Spinal facilitation does occur and can be seen following musculoskeletal injuries. It is well established that inflammation produces both peripheral sensitisation of the afferents (such as free nerve endings) and central sensitisation within the spinal cord (Dunbar & Ruda 1992, Hylden et al 1989, Cook et al 1987, Woolf & Walters 1991). This sensitisation means that the threshold of different neurons is reduced, so they respond to mechanical stimuli to which they were impervious before injury. This process tends to spread laterally in the spinal cord *but in a selective way; not all neurons are sensitised*. The selectivity of the spread seems to be functional in character supporting the process in some way. For example, lateral sensitisation has been shown to spread to the motoneurons which supply the muscles in the affected area (He et al, 1988). This may have a functional role in the muscle guarding often observed at the site of damage. It is very difficult to imagine what would be the functional role of a lateral spread to autonomic-visceral centres in musculo-skeletal damage. It should also be noted that the sensitisation process seems only to take place when nociceptors are excited by pain or inflammation and not when proprioceptors are stimulated, such as during a manual treatment.
A similar sensitisation phenomenon was demonstrated by Korr and his co-workers (1962), by introducing chemical insults to different spinal structures. They demonstrated that this lateral spread could alter sympathetic activity to the segmental sweat glands. This change only took place when pain was inflicted. However, the spread was not always 'neatly' segmental; some of the changes were general or remote from the segmental distribution. This finding is not surprising in the light of what has been discussed about afferent divergence within the spinal cord. These changes in sympathetic activity may not necessarily have been due to facilitation. They may arise as a secondary functional physiological process, e.g. to support changes in muscle activity or the inflammation process at the site of damage. Furthermore, such sympathetic changes in sudomotor activity have no clinical relevance to osteopathic practice. More important clinically are changes in motoneuron threshold by spinal sensitisation as described by He et al (1988).
When Korr et al (1962) introduced postural insults, such as heel lifts on one side, or having the subjects sit on a tilted chair, they observed changes in the pattern of sweating. The changes were general but sometimes more noticeable as an exaggeration of the pattern observed before the insult. Again, they concluded that these changes were due to facilitation. However this is also doubtful: the changes were

probably due to whole body adjustments to changes in posture rather than a locally organised change in the spinal cord. Here too the leg can be used as an example. If you ask a subject to stand on one leg there will be considerable differences in the muscle activities of the two legs. Naturally the blood supply and sweat gland activity will also vary considerably between the two legs with an increase in activity in the balancing leg. These are whole body postural adjustments incorporating complex patterns of neuromuscular and supportive autonomic changes. These patterns of recruitment are organised within the whole system rather then segmentally by the limited and local processes of facilitation.

All the changes that were demonstrated were during separate studies on different individuals: one study showed that in normal subjects there may be a variable pattern of muscle response to pressure (Denslow, 1947). Another showed changes in skin conductivity (Price & Korr, 1957), and a third showed variability of vasomotor tone (Wright & Korr, 1960). They never took the logical step of examining all three phenomena of facilitation in the same group of subjects! This is equivalent to seeing three different patients, one with joint pain, one with conjunctivitis and another with urethritis, and diagnosing them all as having Reiter's Syndrome! Eventually they did examine the three manifestations of facilitation in a group of subjects with musculoskeletal injuries. However, for some reason not all subjects had the full test procedure, e.g. some had skin conductivity but not EMG examination. In this study they claimed that "frequently" the exaggerated patterns were segmentally related to the site of injury. This suggests that the nervous system does not respond in a stereotypic manner to injury. Unfortunately no statistical analysis was carried out on the data and their use of terminology such as "frequently" is not very helpful; does it mean 10% or 90% of subjects? Furthermore they never compared the findings of this study (subjects with musculoskeletal injuries) to the extensive control group of the previous studies (normal subjects). Interestingly, when one compares the photographs of skin conductivity of subjects with injury (Korr et al, 1964, pages 68-70) to those of normal subjects (Korr et al, 1958, pages 35-37), they don't seem to be different. The results in this study could be interpreted like the results of their other studies - they demonstrated individual variability rather than facilitation.Overall, given that the studies did not exclud the influences of higher centres and made no direct recordings from the spinal cord it can be argued that all the changes observed in the studies were not due to local segmental facilitation but were in fact organised by the total nervous system (with the prominent role of supra spinal centres).

2. They did not demonstrate somatovisceral reflexes - These early studies did not show that abnormal muscle activity or skeletal abnormalities will spread to affect the viscera by the process of facilitation. This is a very important point: they assumed (along with many generations of osteopaths) that sympathetic changes to sweat glands of the skin mean that the whole segmental autonomic system has been affected including the autonomic centres controlling visceral activity (Korr, 1948; Korr et al, 1962; Korr 1978). This conclusion is a fantastic hypothetical leap, one which was never demonstrated in humans with intact nervous systems. Furthermore, they did not show that stimulation of mechanoreceptors (proprioceptors) would cause a change in visceral activity. They simply observed the triad of muscle tone, local tenderness and local sympathetic changes (skin conductivity and vasomotor). The generally held belief that stimulation of different groups of proprioceptors can alter visceral activity was never demonstrated in these studies. They have demonstrated the reverse: that sometimes, known visceral pathologies manifested segmentally as increased skin conductivity. However, that does not mean that the reverse is true, i.e. that stimulation of the soma will alter the activity in the viscera. This would be comparable to suggesting that since we reflexively close our eyes during sneezing, we would sneeze each time we close our eyes.

3. They failed to demonstrate relevance to osteopathic manual therapy - Another interesting point is that there is no mention in all these studies of which form of manual technique could bring about autonomic changes. The logical next step of these studies was never taken and was totally side-stepped in the articles, i.e., testing the effect of different forms of manual techniques on spinal facilitation. Without discussing techniques, the concept of facilitated segment has no meaning to an osteopath. The osteopath needs to know how to change the activity of the facilitated segment. So many generations of osteopaths have assumed that high velocity thrusts (HVT) are the most appropriate form of manipulation for normalising or resetting the facilitated segment.

Recent studies into the effects of manual techniques on neuromuscular activity have strongly suggested that passive manual techniques are unlikely to affect this system (Sullivan et al, 1991; Kukulka et al, 1986; Leone & Kukulka 1988; Belanger et al, 1989; Goldberg, 1992; Sullivan et al, 1993; Lederman, 1997; Newham & Lederman, 1997). They only produce a transient artefact event that has no permanent influence on, or ability to bring about functional changes in overall motor processes. Even if one accepts the possibility of facilitated segments as described by Korr, Denslow and their co-workers, it is extremely doubtful that passive stimulation of the soma would result in the resetting of neurological activity

(Lederman, 1997). All neuromuscular activity is organised centrally to spread centrifugally to the periphery (Schmidt, 1991). The peripheral receptors (proprioceptors / mechanoreceptors) provide feedback rather than control the motor system.

Fascinating segments

The criticism in this article is not about the quality of the research but the interpretations of the results and the far-reaching conclusions that were drawn. Overall in their studies, Korr, Denslow and their co-workers did not demonstrate the facilitation phenomenon. In the light of our current understanding of neurophysiology it is doubtful whether the facilitated segment model as described by Korr, Denslow and their co-workers has any neurological basis or clinical application.

An interesting question arises: what was and still is so attractive in the concept of facilitated segments? The answer I believe lies in the high velocity thrust (HVT) and segmental adjustments. The concept of the facilitated segment provides the justification for performing a very accurate HVT on particular segments. It gives the HVT a physiological depth beyond the biomechanical structural fixing of the spine. The osteopath is now able to reach deep into the interior of the patient to affect visceral pathologies. This was done at a great cost to osteopathy - osteopathic understanding of neurophysiology has starts and ended at the facilitated segment. In my view, the principle of the facilitated segment has stifled the development of osteopathic neurophysiology for the last 50 years. Important issues such as neuromuscular rehabilitation following musculoskeletal injuries, central nervous damage, posture and movement guidance, the psychodynamics of touch and psychophysiological processes and pain management have never been addressed in depth. Some of these issues and their relevance to osteopathy and manual therapy have been discussed in detail by Lederman (1997).

The way forward

There is a need in osteopathy to develop a better understanding of neurophysiology; to see the wider picture rather than concentrate on a single fraction of the total system / person. There also needs to be a better understanding of how osteopathic manual approaches can be developed to become effective therapeutic processes for treating the nervous system. This is essential for working with a wide range of clinical conditions which have a neurophysiological element in them. Some of these are common clinical conditions that osteopaths see in daily practice such as postural and movement changes, neuromuscular changes following musculoskeletal injuries and the neurophysiological aspects of pain.

In order to influence the nervous system treatment should imitate natural processes that bring about changes in the nervous system. Most important is the use of cognition, volition and repetition and avoiding the use of reflexive type treatments that have been demonstrated to have no long term effects (Lederman 97, Newham & Lederman 97). Lederman (1997), has discussed in detail how these elements of neurophysiology can be incorporated into and expand osteopathic practice enabling the treatment of a wider range of conditions. Some of these points will be discussed in future articles.

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