Managing back pain in general practice — is osteopathy the new paradigm?

NEFYN WILLIAMS

SUMMARY

Back pain is a common problem in general practice, and is of enormous economic importance. A recent report urges general practitioners (GPs) to refer early for manual therapies, such as osteopathy. The key concept to understanding osteopathic principles is somatic dysfunction. This is a disorder of function, rather than pathology, of the musculoskeletal and related systems. Its characteristic features are asymmetry of anatomical landmarks, asymmetry of joint movement, tissue texture changes, and tenderness. The scientific basis of the tissue texture changes and tenderness can be explained in terms of the 'facilitated segment', but the cause of movement asymmetry remains elusive. Randomized controlled trials provide some support for the use of osteopathic treatment in acute low back pain. It is proposed that somatic dysfunction is the new paradigm for non-specific back pain.

Keywords: musculoskeletal injuries; paradigms; osteopathy; randomized controlled trials.

Introduction

BACK pain accounts for 3–7% of all GP consultations and is the third most commonly reported symptom after headache and tiredness. There is a total of 14–15 million consultations for back pain each year in Britain, involving 5–7% of the adult population; it is of enormous economic importance, with an estimated 150 million days working incapacity due to back pain in Britain in 1993.[1] Despite this, the aetiology in the majority of cases remains uncertain, and is usually classified as 'non-specific back pain'. Perhaps the disciplines of osteopathy and chiropractic can provide some much needed insight. Recent guidelines on back pain management from the Clinical Standards Advisory Group[1] and the Royal College of General Practitioners,[2] based on the limited evidence available, have endorsed the use of such manual therapies. Indeed, 72% of GPs in the United Kingdom refer patients to complementary therapists,[3] most commonly for manipulation,[4] but is there much understanding among referring GPs of the underlying concepts behind these disciplines? In this discussion paper I will concentrate on osteopathic concepts: what are they? is there a scientific basis behind them? what is the rationale for manual therapy?

A definition of osteopathy

'Osteopathic practice is based on the concept that abnormal function of the musculoskeletal system, not dependent on structural pathological processes, is an important cause of disability and illness. In both diagnosis and treatment of this somatic dysfunction, osteopaths rely heavily on distinctive manual skills. Overall management involves assessing the relationship of dysfunction to body use, occupation, emotion, cognition, or any structural pathology.'[5]

Somatic dysfunction

The concept of somatic dysfunction is the key to understanding osteopathic principles. It has superseded previous descriptive terms, such as osteopathic lesion or chiropractic subluxation, and has been defined as:

> Impaired or altered function of related components of the somatic (body framework) system; skeletal, arthrodial and myofascial structures; and related vascular, lymphatic and neural elements.[6]

An important part of this concept is that symptoms and loss of function occur in the absence of pathological disease. The majority of back pain is often labelled 'non-specific' because of this absence of pathology; despite this, many medical authors pursue a reductionist, disease-centred search for some form of pathological change in tissues responsible for pain and loss of function.[7,8] In contrast, the concept of somatic dysfunction involves abnormal *functioning* of the neuromusculoskeletal system, sufficient to cause symptoms and disability *independently* of any structural pathology. The sources of nociception proposed are mechanical in stressed tissues and chemical in overactive muscles.

The difference between dysfunction and pathology can be contrasted in a number of ways: pathology can be defined by its localization and nature, and dysfunction is the result of the correlation and interplay of a whole chain of different structures in various locations. This is of fundamental importance because one cannot pinpoint function to any single structure or location. The diagnostic task in pathological diagnosis is to localize the lesion exactly and to determine its nature; in dysfunction, the task is to determine the pathogenetic chain and to assess the correlation and relevance of its individual links. In an analogy with computers, pathology involves a problem with the hardware, and dysfunction with the software of the motor system. Modern technology enables us to diagnose pathological conditions more effectively and with more objectivity. In dysfunction, technology is of little use and subjective clinical skills remain decisive.[9]

General practitioners are used to considering illness in terms of interacting physical, psychological, and social components. Perhaps this physical component should be further subdivided into pathology and dysfunction?

Making a diagnosis of somatic dysfunction

In the history, the patient may complain of pain arising after an awkward movement, postural insult, overuse, or on waking. It is often difficult to delineate the pain and it may be referred distally, often in a non-segmental pattern. It is aggravated by particular movements, postures, and activities, and is usually relieved by rest and lying down. There may be associated areas of paraesthesia and hyperaesthesia. Symptoms are often vague and difficult to describe. Patients may complain of odd sensations, such as temperature changes or peculiar gait disturbance. The dysfunction may be asymptomatic, but may become symptomatic when stressed by abnormal posture, overuse, etc. The characteristic features on examination are:

N Williams, MA, MRCGP, MLCOM, DM-SMed (Diploma of Musculoskeletal Medicine), general practitioner and osteopath, Health Centre, Llanfairfechan, North Wales.

Submitted: 3 December 1996; accepted: 17 March 1997.

Asymmetry of joint movement

This involves alteration in the range of motion of a joint, several joints, or a region of the musculoskeletal system. The alteration may be either restricted mobility or increased mobility, and is determined by observation and palpation using both active and passive movement.

Asymmetry of anatomical landmarks

This affects the pelvis and vertebral column, and is identified by palpation and observation. Structural asymmetry can be distinguished from that due to dysfunction by the fact that joint mobility is normal in structural asymmetry but is either hypomobile or hypermobile in dysfunction.

Tissue texture changes

These affect the skin, fascia, muscle, and ligaments, and are identified by observation and palpation. The affected areas of skin will show increased 'drag' when the examiner's fingers are run lightly along it; this is thought to be a result of locally increased sudomotor activity. The skin and subcutaneous tissues may be thickened, oedematous, and less compliant. Localized muscle hypertonus can be palpated as tender bands or nodules.

Tenderness

This affects deep or superficial tissues and is found at the site of dysfunction. However, it is also found at the point to which pain is referred, so should not be relied upon solely for locating the site of a dysfunction.

The barrier concept

The essential component of somatic dysfunction is abnormality of joint movement, commonly motion restriction. What is the nature of this restriction? The point beyond which a joint will not move is referred to as the barrier. A normal joint will move through a certain range of active motion. Beyond the end of this active range there is a small additional passive range. The limit of active motion is called the 'physiological' barrier, and that of passive motion is called the 'anatomical' barrier. If the anatomical barrier is exceeded, the joint will be disrupted. In a normal joint, motion between the physiological barriers is free and smooth — as passive motion is introduced after the end of the active range, the tension increases steadily until motion ceases as the barrier is reached. In joint movement restriction, motion is lost within this range. The barrier that prevents movement in the direction of motion lost is defined as the restrictive barrier; it is present in each plane of motion. The amount of active movement available on one side is limited by the normal physiological barrier, and on the other by the restrictive barrier. The goal of osteopathic intervention is to move the restrictive barrier as far into the direction of motion loss as possible.

In every joint there is a point of maximum ease. This so-called 'loose-packed' position is the point from which movement in either direction causes the soft tissues to become more tense, thus increasing bind. In a normal joint, this point is usually near the mid-point of the range. When there is a restrictive barrier, the point of ease will be found to have moved, usually to about the mid-point of the remaining range. As with the barrier, the point of ease exists in each plane of motion, but also around each axis.

Is there a scientific basis for the concepts of the barrier, ease, and bind? Could muscles be responsible? Muscles not only produce motion by their contraction, they also function as brakes. Abnormal muscle hypertonus could explain the phenomenon of steeply rising resistance to movement (or increasing bind) as the barrier is approached. However, movement restrictions can persist even when patients are anaesthetized and paralysed prior to surgery.[10] Articular phenomena, such as entrapment of a meniscoid at the edge of a joint space[10] and jamming of dimpled corrugated cartilaginous surfaces,[11] might explain this, but neither of these explain the phenomenon of increasing bind. Further research is needed into the scientific basis of these concepts.

Facilitated segments of the spinal cord

Scientific evidence is shedding light on the local tenderness and soft tissue changes of somatic dysfunction. Korr *et al* performed simple experiments on the paravertebral muscles and electrical skin resistance of subjects' backs, and demonstrated that the motor, pain, and sympathetic pathways, at segmental levels corresponding to somatic dysfunction, showed increased activity. They concluded that neurones in these spinal cord segments were maintained in a state of facilitation.[12,13,14]

What is the cause of these facilitated segments?

General practitioners are familiar with patients with low pain thresholds who complain of pain after minimal provocation. It is now known that neural pathways are not hard-wired, but can be sensitized or habituated following repetitive stimulation. After tissue inflammation or nervous system damage, acute pain can be altered in the following ways: pain may occur in the absence of any apparent stimulus (spontaneous pain); response to suprathreshold stimuli may be exaggerated in either amplitude or duration (hyperalgesia); the threshold for eliciting pain may decrease to a level such that normally innocuous stimuli begin to elicit pain (allodynia).[15] These phenomena can be partly explained by sensitization of the nervous system centrally and peripherally.[15] The sympathetic nervous system is increasingly recognized as an important mechanism in the development of such sensitization.[16] It can be postulated that tissue injury from trauma, inflammation, or postural stress markedly alters the sensory input from articular and periarticular structures; this initiates aberrant motor and sympathetic responses, which cause the segmental facilitation seen in somatic dysfunction.

Why does pain persist in our patients, long after the original insult has disappeared?

Korr[12] reported that facilitated motor and sympathetic regions may endure for months or years. This suggests that there is long-lasting alteration in neural pathways, so that a residual hyperexcitability remains despite the eventual cessation of the initial painful stimulus. There is some research to suggest that the spine can 'learn', and retains a 'memory' of previous injuries. It has been demonstrated in experimental animal preparations that long-term alterations of spinal reflex patterns can be induced by noxious, repetitive peripheral stimulation.[17] It is unclear whether the effects of such long-term facilitation of spinal interneurones can be completely reversed. Korr's facilitated segments may be the clinical manifestations of such areas of spinal neuronal reflex hyperexcitability, which may be secondary to previous noxious conditioning stimuli, such as earlier trauma.

These ideas of the facilitated segment, neuroplasticity, and spinal learning can help to explain the pain, tenderness, and soft tissue changes seen in somatic dysfunction.

Rationale for manual therapy

The aim of manual treatment methods is to reverse dysfunction in the neuromusculoskeletal system. Osteopathic treatments are

conventionally classified into soft tissue techniques, articulation, and mobilization.

- Soft tissue techniques mechanically stretch the skin, fascia, and muscle tissues to enhance their motion and pliability, either as a specific therapeutic goal or in preparation for other procedures.
- Articulation consists of repetitive, oscillatory movements engaging a restrictive barrier up to its end point before backing away. Its purpose is to restore range of movement, and to stretch out connective tissue surrounding a restricted articulation.
- Mobilization involves engaging the restrictive barrier of the joint needing treatment, followed by a high velocity and low amplitude thrust through the barrier, which briefly separates the joint surfaces and commonly produces a cavitation sound.

Successful treatment will restore the range of movement, improve symmetry, reduce muscle hypertonicity, and possibly restore normal joint receptor activity. This might improve somatic neural input to the spinal cord and allow reflex activity in facilitated spinal segments to return towards normal.

What evidence exists from randomized controlled trials?
Most trials have examined the efficacy of high-velocity thrusts to patients with back pain. Unfortunately, most are of poor quality.[18,19] One review concluded that 'spinal manipulation is of short-term benefit in some patients, particularly those with uncomplicated acute low back pain.'[19] Several new randomized controlled trials have been published, and an updated review of these is awaited.[20]

Time for a paradigm shift

There is some evidence supporting the concept of somatic dysfunction. Korr's work on the facilitated segment, and more recent ideas about neuroplasticity and spinal learning, are consistent with the tissue texture changes and tenderness found in areas of dysfunction; however, how these changes relate to the positional asymmetry and joint movement abnormality is not known. Similarly, the underlying basis of joint bind, ease, and blockage remains uncertain. Randomized controlled trials have demonstrated short-term benefits after spinal mobilization, but most are of poor quality.

In his book *The structure of scientific revolutions,* Kuhn[21] explains his ideas of how science develops. In what he calls 'normal science', research is firmly based upon one or more past scientific achievements that the scientific community acknowledges as supplying the foundation of its further practice. This accepted model or pattern he calls a 'paradigm'. Normal scientific research is directed at the articulation of the phenomena and theories that the paradigm supplies. Arguably, the paradigm dominant in contemporary medicine is that illness is caused by pathological disease. If the illness is investigated in progressively finer detail, eventually the relevant pathology will be uncovered.

Such a reductionist approach has failed to explain the majority of cases of back pain. Kuhn suggests that, as awareness of the inadequacies of the current paradigm grows, a crisis develops. The only way forward is for a new paradigm to emerge. There is much supportive scientific evidence underlying some, but not all, of the basic concepts of somatic dysfunctions. Kuhn argues that, to be accepted as a new paradigm, a theory must seem better than the old paradigm, but it does not, and never can, explain all the facts with which it can be confronted. Somatic dysfunction is the new paradigm for non-specific back pain.

Manual therapies used to be practised by many GPs, but these skills have been neglected. Indeed, such therapies are largely ignored in both undergraduate and postgraduate teaching. However, practising osteopathy within general practice is more than just providing manipulation for back pain sufferers, it offers new insights into diagnosing many common pain syndromes seen daily in our surgeries. The introduction of osteopathic concepts, such as somatic dysfunction, into postgraduate and undergraduate education, is long overdue.

References

1. Clinical Standards Advisory Group. *Back pain.* London: HMSO, 1994.
2. Wadell G, Feder G, McInotosh, *et al. Low back pain evidence review.* London: RCGP, 1996.
3. Warton R, Lewith G. Complementary medicine and the general practitioner. *BMJ* 1986; **292:** 1498-1500.
4. Anderson E, Anderson P. General practitioners and alternative medicine. *J R Coll Gen Pract* 1987; **37:** 52-55.
5. MacDonald RS. Introductory lecture in the membership course of the London College of Osteopathic Medicine, 1995.
6. Greenman PE. *Principles of manual medicine.* Baltimore, MA: Williams and Wilkins, 1989.
7. Jayson M. Mechanisms underlying chronic back pain. *BMJ* 1994; **309:** 681-682.
8. Bogduk N. Sources of low back pain. In: Jayson M (ed.). *Lumbar spine and back pain.* 4th edn. Edinburgh: Churchill Livingstone, 1992.
9. Lewit K. The functional approach. *J Orth Med* 1994; **16:** 73-74.
10. Lewit K. *Manipulative therapy in rehabilitation of the motor system.* (2nd edition.) Oxford: Butterworth Heinemann, 1991.
11. Laxton AH. Practical approaches to the normalization of muscle tension. *J Manual Med* 1990; **5:** 115-120.
12. Korr I. Proprioceptors and somatic dysfunction. *J Amer Osteopath Assoc* 1975; **74:** 638.
13. Denslow JS, Korr IM, Krems AD. Quantitative studies of chronic facilitation in human motoneuron pools. *Amer J Physiol* 1947; **150:** 229-238.
14. Korr IM, Wright HM, Thomas PE. Effects of experimental myofascial insults on cutaneous patterns of sympathetic activity in man. *J Neural Transmission* 1962; **23:** 330-355.
15. Woolf CJ. Recent advances in the pathophysiology of acute pain. *Br J Anaesth* 1989; **63:** 139-146.
16. Kidd B, Cruwys S, Mapp PI, Blake DR. Role of sympathetic nervous system in chronic joint pain and inflammation. *Ann Rheum Dis* 1992; **51:** 1188-1191.
17. Slosberg M. Spinal learning: central modulation of pain processing and long-term alteration of interneuronal excitability as a result of nociceptive peripheral input. *Journal of Manipulation & Physiological Therapy* 1990; **13:** 326-336.
18. Koes BW, Assendelft WJ, van der Heijden GJ, *et al.* Spinal manipulation and mobilization for back and neck pain: a blinded review. *BMJ* 1991; **303:** 1298-1303.
19. Shekelle PG. Spinal manipulation for low back pain. *Ann Intern Med.* 1992; **117:** 590-598.
20. Assendelft WJ, Shekelle PG, Koes BW. Spinal manipulation for low back pain. In: Bombardier C, Nachemson A, Deyo R, *et al.* (eds) CMSG back module of *The Cochrane database of systematic reviews.* Oxford: Update Software, 1997.
21. Kuhn TS. *The structure of scientific revolutions.* Chicago, IL: University of Chicago Press, 1970.

Acknowledgements

This paper was adapted from an essay written for the membership course of the London College of Osteopathic Medicine. I would like to thank Clare Wilkinson for her critical reading and help in editing this paper, and Jane England and Jayne Westmoreland for their help in its production.

Address for correspondence

Dr Nefyn Williams, Health Centre, Village Road, Llanfairfechan, North Wales LL33 0NH.