Dissertation Programme

BSc (Hons) Osteopathy Final Year Dissertation 2000

Title: A study to assess the quality of evidence for the existence of segmental somatic dysfunction: a systematic review.

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1.0 Acknowledgements

The author would like to acknowledge the help and support of the following individuals in alphabetical order:

- Damian Baskeyfield
- Helen Costello
- Jo Dear
- Elizabeth Oates
- Tim Oxbrow
- Will Podmore
- Stephen Sandler
- Stephen Tyreman

2.0 Abstract

The aim of this study is to assess the quality of evidence for the existence of segmental somatic dysfunction by subjecting suitable existing studies to the process of systematic review. Many consider that this concept is central to the philosophy and practice of osteopathy however, the full nature and scope of somatic dysfunction is not yet known nor how pervasive it is in rôles of disease and individually appropriate human function. To prevent osteopathy from stagnating it is therefore important to fully explore this area to understand the context in which somatic dysfunction should exist. The theories and history behind somatic dysfunction and the difficulties encountered in the terminology and definition are explored in order to provide a background for the enquiry. Eleven studies, six observational correlations and five experimental treatments, that fitted the inclusion criteria were retrieved from osteopathic and chiropractic literature and were subjected to a purposefully designed methodological scoring process which produced an overall score for each study. A statistically significant difference was found between the two types of study analysed with the experimental treatment protocol achieving better methodological scores. All the studies claimed a positive outcome except for one which returned an equivocal conclusion. The results of this systematic review suggest that all the studies were generally of a low methodological quality illustrating the confusion surrounding this area and suggesting the evidence for the existence of segmental somatic dysfunction to be poor. The potential difficulties in analysing and comparing studies involving different protocols and the difficulties in carrying out research in this area are demonstrated. No conclusions may be drawn from this review as to the reality of segmental somatic dysfunction however, it highlights the requirement for further studies of high methodological quality

3.0 Table of Contents

Word count (excluding tables and appendices): 10,900 words (with permission).

4.0 Introduction

4.1 Aim

The success of a systematic review is considered by de Bie (1996) to be governed by the posing of an explicit research question. Therefore, the aim of this study is to subject suitable modern research reports to the process of systematic review in an attempt to determine the quality of the evidence for the existence of segmental somatic dysfunction as reported by the included studies.

The null hypothesis is that there is no evidence for the existence of segmental somatic dysfunction, as revealed by analysis of the selected studies.

4.2 About defining and naming

The basic mechanism of research into any given phenomenon is to propose a theory then name, define, model, test, refine and retest that model until there is a comprehensive and satisfactory explanation of the phenomenon that comfortably exists within reality as it is understood at that time. If there is any subsequent change to the parameters of understanding of the named phenomenon or the reality surrounding it then the previously held model may fail and the cycle of test / retest begins again in order to construct a new satisfactory model. (Appendix 1, Note 1).

It is very difficult to explain a concept without using a definition. It is even harder if the concept has no name. What is more, if a definition and a name are used to describe two or more separate concepts that are mistakenly and unknowingly considered to be the same thing then confusion and frustration are bound to ensue.

Enter, with these notions, into the realms of medical research at the level of the individual whole organism with the furious and dynamic interplay of countless biological, mechanical, psychological, philosophical and spiritual domains and the problems facing researchers may be appreciated and may even render gold standard randomised clinical trials unsuitable for this kind of research.

This then, may be considered to be the situation surrounding the osteopathic lesion or somatic dysfunction (these names are, at this time, the most universally accepted to describe this phenomenon and so will be used for the time being in order to provide a working name. For closer discussion see later).

The concept of somatic dysfunction has been pervasive throughout the history of osteopathy from the early days of Andrew Taylor Still. It has existed in many guises being described, defined and named differently by different practitioners, researchers and professions. Many treatises, essays, studies and experiments have been carried out in its name and arguments have raged about its existence and nature and whether the researchers are actually describing the same phenomenon.

Certain issues are raised regarding the multiplicity of names and conditions:

- Do they describe the same phenomenon?
- Do they refer to a number of different phenomena that have been mistakenly consolidated and regarded as one?
- Do they refer to different aspects of the same phenomenon?
- Do they reflect the agreed clinical findings associated with this phenomenon?
- Are the associated clinical findings indeed correctly described and representative of the phenomenon?

To these questions there are no agreed or easy answers and in their pursuit a more philosophical route of inquiry becomes necessary.

However, the many modified concept has survived and has come to represent more than its original definitions as it provides a central basis for the osteopathic profession.

Given the above difficulties, an appropriate place to start would be with the description of the clinical findings associated with somatic dysfunction. To paraphrase Irvin Korr at the first UK osteopathic research conference, “The clinician raises questions for the researcher to answer”.

4.3 Clinical effects of somatic dysfunction

There have been many descriptions of the clinical findings associated with the identification of the somatic dysfunction, these classically being:

- Hyperalgaesia of the vertebral segment and paravertebral muscles.
- Hyperirritability displayed as altered states of muscular contraction.
- Changes in palpable tissue texture (skin, connective tissue and muscle).
- Local circulatory changes.
- Alteration in visceral and autonomic function.
- Decreased or altered range segmental range of movement.
- Segmental asymmetry.

The practitioner will assess the nature and quality of many of these characteristics within the scope of the subjective palpatory experience. Denslow considered Hyperalgaesia and abnormal tissue texture to be most important since they reflect the localised disturbance in homeostasis that precipitate, maintain and provide direct information as to the location and severity of the lesion. Anatomical asymmetry and joint movement may also regarded together due to their reciprocal nature (Denslow, 1975).

Korr, in his many experiments, pioneered the use of objective means to quantify these changes as outcome measures and gain insight into the neurological functioning of the dysfunctional segment. Many subsequent researchers have based their work on these techniques.

The physiological changes in tissue texture, tone and circulation observed may be measured by several means. Korr's investigations into the autonomic effects of sweat gland stimulation resulted in a quantifiable variation in electrical skin resistance and similar changes may be determined within muscle using electromyography (Korr *et al.,* 1947, 1958, 1962, 1964. Ellestad *et al.,* 1988). Local circulatory changes serve to create localised temperature differences which may be measured by thermography. Korr used thermocouples but thermal imaging

technology has superseded this (Kelso *et al*., 1982). Another method Korr employed was to utilise the change in the red response upon mechanical stimulation of the skin (Wright *et al.,* 1960) and changes in muscle tone have been measured using tissue compliance meters (Nansel *et al.,* 1993).

Therefore, the classic, localised clinical presentation of somatic dysfunction is a hyperalgaesic spinal segment of restricted mobility with surrounding abnormal tissue texture and tone. There may be a degree of asymmetry, localised sweating and erythema present.

The benefit of using quantitative measures over subjective is that the inter-practitioner (and intra-practitioner) reliability is difficult to establish. Examiner bias and variability together with biological variability serve to decrease the confidence in results achieved utilising palpation alone. This is not to say that this approach cannot be used and the errors mitigated by the imposition of suitable training and standardisation protocols. However, the effectiveness of these as compared to an objective means of measurement must be considered when assessing the quality of research.

4.4 Historical progression

Over the last century there has been a considerable number of studies carried out in this field. However, the bulk of this work was undertaken over forty years ago and there is a paucity of modern research either validating or building upon this.

The concept of the segmental somatic dysfunction goes back to the foundation of osteopathy when A.T. Still was establishing his principles at the turn of the century. Still considered that a disorganised structure lead to a dysfunction (particularly in blood flow) which in turn created illness. Still referred to such musculoskeletal manifestations as strains, slips, dislocations or subluxations and, with John Martin Littlejohn, these descriptions and their treatment developed into the Science of Adjustment which was concerned with returning the individual to harmony with his environment. Research at Kirksville during the early 1900s describes the appearance and effect of experimentally created lesions in animals. The reports illustrate the physiological changes observed during the acute and chronic phases. The term spinal or osteopathic lesion was subsequently used to describe these particular clinical findings.

During the 1940s and building on these early descriptions, the understanding was that spinal nerves had become compressed and therefore vital nerve factors were being prevented from flowing. The physiological basis of axonal flow both anterograde and retrograde had been investigated by Ramon Y Cajal, Waller and Schwann and was well established at this time as were the effects observed in its interruption. However, this *pinched nerve theory* did not provide a complete explanation for the observed clinical effects especially the immediacy of onset, the complexities of the chronic lesion and the apparent widespread response.

The pinched nerve theory gave way to the more sophisticated neurological model proposed by Littlejohn and then refined by Denslow and Korr. In the late 1930s, Denslow began investigating spinal dysfunction using pressure meters and electromyographs. He demonstrated that the lesioned areas could be prompted to produce muscular changes at much lower levels of stimulus than non-lesioned

areas of the spine. His efforts may be considered one of the first objective attempts at verification, through experimental means, of the osteopaths subjective claim of being able to detect areas of lesion through palpation alone.

In the 1940s Professor Irvin Korr joined Denslow who was already carrying out research in this area. This lead to his own interest and research. Together they demonstrated that, when a lesioned spinal segment was in this state of over-excitation, it could also be stimulated by indirect pressure, or irritation, from normal segments some distance above and below (Denslow *et al.,* 1947). However, when the lesioned segment was anaesthetised, it would only respond to the stimulus of segments above or below and not to the direct irritation. These over-excited, low-threshold spinal segments were termed *facilitated segments*. Korr reasoned that such direct pressure may be considered an unnatural form of stimulus and attempted to achieve the same results using a variety of other physical or psychological stimuli such as pain, loud noises and emotions. He found that the results were consistent and that the facilitated segments were the first to react and the last to relax when compared to non-lesioned segments. Korr encapsulated these findings in his theory of the *neurological lens* (Korr, 1947) which states that, according to the principles of reciprocity and convergence (where every nerve fibre connects with and can therefore influence every other), any nerve synapsing at the lesioned segment causes other fibres, especially efferent ones, to fire leading to an inappropriate or abnormal physiological response to an otherwise normal stimulus. In effect, the facilitated segment serves to focus neurological activity upon the skeletal (soma) and visceral structures innervated by the motor, sensory and autonomic neural outflow from that segment. This subsequently led to the formulation of the theories of somatovisceral and viscerosomatic reflexes. On this basis, in 1948, at an address to the American Osteopathic Association entitled, "The emerging concept of the osteopathic lesion", Korr recommended that the osteopathic lesion be, "conceived as a most important – and frequent – aetiological, predisposing, exacerbating and sustaining factor in disease".

Korr refined the neurological lens model to explain the direct and indirect effects of a spinal lesion on the functioning of the peripheral and central nervous systems. The direct effects (Korr's non-impulse based mechanism) operate at the level of

the intervertebral foramen and are due to compression irritation of the associated neural structures. It could be understood how these structures, responsible for motor, sensory and autonomic function, may become mechanically or chemically compromised. The indirect, or impulse based mechanism, describes the effects of persistent spinal pain and hypomobility on the reflex activities of an associated spinal level. Korr termed this *central facilitation* (Korr, 1975) and is now known as *central sensitisation*.

A modern concept leading to a more integrated model with the three areas of spinal segment, remote tissues and higher centres being involved, has been explored by Ward and Lederman. The previously held somatovisceral reflex model is divorced from the higher brain and occurs at the reflex arc of the relevant spinal level via the stimulation of segmental proprioceptors and acts upon the related autonomics and viscera. Lederman notes that there are many flaws with this model. Most importantly, he argues that the higher centres cannot be dismissed as an important source of autonomic control. Also, in experimental studies demonstrating somatovisceral reflexes, the test animals have often been subjected to trauma that is inappropriate to a real-life situation. The subsequent generation of reflexes often involves gross stimulus and Lederman points out that such conditions cannot be compared to the effects of a therapeutic manipulation. Furthermore, anatomical distinction between neurones within the spinal cord is lost and so, as Korr noted, there is a distribution over several segments both up and down. Lederman argues that this implies that sensitisation may not be entirely segmentally specific. In addition to these considerations, Lederman points out the existence of a *biological paradox* which suggests that, if the facilitation model were entirely accurate, then trauma sustained during the course of everyday activities would result in visceral dysfunction and this is clearly not the case.

In contrast to the somatovisceral theory, a new integrated model argues that manipulation of the area of dysfunction involves potent psychosomatic influences which serve to augment, or even nullify, the somatovisceral reflex alone. Such an influence has a non specific, generalised action that is not associated with particular spinal segments and is marshalled by the higher centres of the brain such as the limbic system (Lederman, 1997; 2000).

4.5 Terminology and definition

Understanding the rich history of somatic dysfunction illustrates why this phenomena has been repeatedly defined and named and this reflects the shift in the conceptual understanding from description in terms of positional derangement to one of disordered function. The osteopathic profession termed it the *osteopathic* or *spinal lesion* and later the *somatic dysfunction*. The chiropractors called it the *spinal subluxation* and manual medicine knows it as a *fixation, functional blockage* or simply a *hypomobility*.

Stephen Tyreman argues for a more accurate terminology of somatic dysfunction that places it firmly within context. Tyreman contends that the use of *somatic dysfunction* is, literally, inaccurate and refers to, "the inappropriate way in which the somatic (body framework) tissues function in response to environmental changes". He suggests the more accurate and descriptive *segmental somatic dysfunction* meaning, "specific local tissue change that occurs in the vertebral segment associated with excessive neural effects". (Tyreman, 1994).

The excessive neural effects may manifest in a local action, such as hyperaesthesia, hyperirritability, changes in tissue and local circulation, or in remote actions such as altered visceral and autonomic function (Korr, 1947).

Tracing the definitions of segmental somatic dysfunction the early notion of a positional emphasis may be understood. Gibbons and Tehan report Downing as saying in 1923, "Osteopathic lesions (spinal) are usually bony subluxations with ligamentous tension or shortening and muscular tension or contractions". Similarly, in 1935, Castillo said, "When a lesion exists it will usually be found that one or more vertebrae are not in normal relation to those above and below, but fixed relatively or absolutely in flexion, extension, rotation, sidebending or a combination of these positions" (Gibbons and Tehan, 2000).

In the mid 1900s the thinking moved away from a positional view to one of restricted mobility as exemplified in Wilson's 1955 definition (in Gibbons and

Tehan, 2000), "The mere position of a vertebra may not constitute a lesion. Loss of physiologic movement is a major diagnostic feature of the osteopathic spinal lesion". Stoddard, in 1959 (in Gibbons and Tehan, 2000), echoes this view, "An osteopathic lesion is a condition of impaired mobility in an intervertebral joint in which there may or may not be altered positional relations of adjacent vertebra…The moment when irreversible pathological changes take place in the joint, it ceases to be a purely osteopathic lesion. Similarly, when the altered position is such that articular facets are not in opposition, it is no longer an osteopathic lesion, it is a dislocation and this is outside the meaning of the term."

Considerations of mobility are part-way towards a functional appreciation of the segmental somatic dysfunction although, at this time, the mobility was seen more as a local phenomena and not viewed in terms of overall function.

The change from positional to functional understanding reflected the shift in the osteopathic treatment philosophy away from the restricted, localised lesion hunting of the mid 20th century towards the consideration of disorganised structure and the restoration of integrated function (and interestingly, a return to the original concepts of Still and Littlejohn). Aberrant position and mobility of the segment became features diagnostic of segmental dysfunction rather than its prime causes. This new emphasis on function led to a new definition, "Somatic dysfunction is an 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" (Greenman, 1989). This definition is used today by medical insurance companies in the United States of America and somatic dysfunction is registered as a classified disease.

However, a difficulty may be seen in describing a dysfunction (a general concept) in terms of a segmental somatic dysfunction (a specific condition) and therefore reducing the dysfunction to this level. Are they the same thing? Furthermore, is a disorganised structure leading to dysfunction wholly specific to segmental somatic dysfunction or do other considerations exist?

A major problem with any attempted definition is that there is a temptation to view the segmental somatic dysfunction in isolation from the mechanical, physiological and psychological context of the whole organism. Certainly, the key to understanding the nature of the segmental somatic dysfunction is that it is a disorder of function of the musculoskeletal and related systems causing, "symptoms and loss of function to occur in the absence of pathological disease" (Williams, 1997). A pathology may be described by its tissue location and disruptive action upon that tissue while a dysfunction must be considered, in a wider sense, as a breakdown of the correlation and interplay between various structures in different locations. This means that it is not necessarily possible to isolate a dysfunction to a specific tissue or location.

It is suggested that, with respect to the osteopathic model, this understanding still does not go far enough. It is the consideration of the distinct effects that the lesioned vertebral segment has upon the balanced physiology of body functions *and* an understanding of the fundamental interconnectedness of these body functions that provides an almost philosophical definition of the segmental somatic dysfunction and insight into the conceptualisation of osteopathy (Tyreman, 1994).

Other commentators have shied away from making a statement of definition, acknowledging its subjective nature and preferring instead to describe the phenomena in terms of its clinical presentation and effects (Denslow, 1975).

4.6 Mechanisms of segmental somatic dysfunction

Korr's proposed mechanism behind the facilitated segment and consequent neurological lens was that an increased input from an associated structure was holding the segment in a state of heightened excitation. The input could be from a segmentally related structure, such as sensory input from skeletal or visceral organs, or non segmentally related such as psycho-emotional stimuli. Korr particularly implicated the input from muscle spindles which might discharge continuously upon shortening (Korr, 1947; 1975). However, subsequent researchers, including Lederman, remain unconvinced by this as it has been demonstrated that *spindle silence* is common especially in non contracted muscle (Valbo, 1974; Lederman, 1997). Whatever the stimulus, the abnormal output from the facilitated segment was found to produce changes in autonomic function that could become stable in time (Korr *et al.,* 1947). Further research by Hix (1957) and Eble (1958) demonstrated the somatovisceral and viscerosomatic connections which could be responsible for communicating this activity in a two-way dialogue. Spinal lesion possibly leading to visceral dysfunction and visceral function possibly predisposing and maintaining a spinal lesion.

Korr considered these reflexes to occur at a purely spinal level with the spinal cord acting as a passive mediator in the process. Patterson subsequently argued that the spinal cord has a more active rôle in preventing, predisposing and maintaining segmental facilitation. Non traumatic, background, afferent input within the set normal range produced by everyday activities results in habituation at the spinal segment and is therefore effectively filtered or damped out. This being the case, then the segmentally innervated structures are buffered from this spurious influence and remain under the control of descending pathways from higher centres (Patterson, 1976).

If an input level were to rise above the upper threshold of the normal range, for instance by sensory input from the tissues of a joint that has been traumatically taken out of its physiological range, then a potentiating, feedback loop to the injured structure might be set up, in other words the segment becomes sensitised.

By the principles of reciprocity and convergence any other structures under this segmental influence would also begin to receive inappropriate stimulation. This abnormal input may essentially drown out control from higher centres and render the structures dysfunctional. Patterson, like Lederman, suggests that psycho-emotional input has as much a role to play as skeletal and visceral input in the formation of sensitisation (Patterson, 1976).

Once the sensitised state has become established Patterson describes a mechanism for it acquiring neurological stability and becoming entrenched. Through a process of, what Patterson terms *spinal learning* the occurrence of normal, threshold input and abnormal, sensitised neural traffic that are approximated closely in time may lead to conditioning. Thus the weaker, normal input acts to potentiate, and so stabilise, the stronger aberrant neurological signals (Patterson, 1976). Subsequent experiments in rats have shown that repeated stimulation of the spinal reflex pathways may lead to *spinal fixation* which increases the neural excitability for a considerable time post stimulation (Patterson and Steinmetz, 1986).

Van Buskirk considers nociceptors to be the source of the repeated efferent input. He suggests that pain is the commonality between all the involved structures, it is mediated by higher centres and aggravated by movement. The consequent physiological responses, such as muscular guarding, fluid disruption and chemical inflammatory mediator release, together with the potent psychological effects encountered during the perception of pain, may provide the necessary levels of input needed to facilitate and sensitise the spinal segment (Van Buskirk, 1990).

So far, in considering the nature of the segmental somatic dysfunction, this study has concentrated only on the neurological issues. Other models for dysfunction have been presented and these have mostly concentrated on mechanical issues. These ideas may involve physical entrapment of tissue such as the posterior aspect of the intervertebral disc or at the zygapophyseal joint meniscoid, both of which are innervated by nociceptors (Bogduk *et al.,* 1981; 1985). Suggestions of spinal intrinsic muscle spasm have been made to account for the hypomobility demonstrated (Buerger, 1983) as have the formation of scarring and adhesions due

to previous injury together or separate from degenerative changes and adaptation (Arkuszewski, 1988).

Other possible contributory mechanisms include a fluid circulation failure (Zink, 1977) and connective tissue shortening (Burns, 1948) causing restricted musculoskeletal movement.

However, while these models undoubtedly have a rôle to play in the aetiology of spinal dysfunction and cannot be dismissed, these mechanisms cannot fully explain the nature and clinical presentation and treatment of segmental somatic dysfunction.

4.7 Segmental somatic dysfunction as a factor in disease

The osteopathic lesion has long been considered and described as an aetiological factor in disease although firm evidence has yet to be discovered. MacDonald and Hargrave-Wilson (1935) wrote much on the nature of the osteopathic lesion and went as far as identifying primary and secondary lesions. They described primary lesions as those caused by direct trauma or other impingement upon the musculoskeletal system. Secondary lesions were defined as those being caused indirectly by a primary source outside the musculoskeletal system and acting via viscerosomatic and somatovisceral pathways.

Korr carried out experiments designed to measure the various neurological responses of the segmental somatic dysfunction. His investigations suggested that if a mechanical dysfunction occurred in a spinal segment it may cause exaggerated somatic and autonomic neurological activity (the neurological lens) which could act to disrupt the finely balanced homeostatic processes that maintain health thus becoming a factor in the aetiology of disease. This theory was termed the reflex based somatovisceral theory of disease or somatic dysfunction.

In recent years there has been much discussion and debate emanating from the chiropractic, osteopathic and physiotherapy professions as to whether this somatovisceral theory of disease is actually a causative factor in frank visceral disease or whether it simply serves to mimic the symptom sets of frank disease that has its aetiology elsewhere (Nansel and Szlazak, 1995).

Nansel and Szlazak offer the same argument as Lederman's more recent biological paradox and suggest the *simulated visceral disease model* which details the confusion experienced by higher centres when afferent nociceptive signals from somatic structures converge upon the same common spinal neuronal pools as independent visceral afferent nociceptor fibres. This produces, "common sets of indistinguishable perceptive, somatic, autonomic and neuroendocrine responses which leads to equally indistinguishable sets of signs and symptoms" thus mimicking visceral disease (Nansel and Szlazak, 1995).

4.8 Systematic reviews

A systematic method of review exists within the realms of evidence based medicine. Evidence based medicine (EBM) is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients (Sackett *et al.,* 1996). Practising evidence based medicine requires the dynamic combination of individual clinical experience with leading external evidence gathered through effective research and systematic review. It is designed to bridge the gap between research evidence and clinical practice in order to prevent costly, inefficient and potentially harmful practitioner decision making.

To work properly, evidence based medicine must exist at all levels and represent all aspects of the modern medical framework. Thus the term *evidence based medicine* may be considered the name for this whole process. The components of EBM are therefore evidence based diagnosis leading to an evidence based treatment decision based upon evidence based research.

There are five basic stages in the practice of clinically applied EBM which bind the individual components together (after Rosenberg and Donald, 1995):

1) The formulation of clear clinical questions from the patient's presenting problems.
2) A search of literature to secure relevant clinical articles on the best available evidence. (Appendix 1, Note 2).
3) Evaluate (critically appraise) the evidence for it's validity and applicability.
4) Implement the useful findings in the treatment of the patient.
5) The willingness and ability to critically and reflectively self-evaluate performance.

In the context of this study we are clearly only concerned with components 2 and 3 although it can now be seen how the systematic review fits into whole procedure.

Dove (1977), in his address to the Conference on Back Pain, reports Dr. Gregg, director of medical sciences at the Rockerfeller Foundation, giving advice to the American Osteopathic Profession in 1938. Dr. Gregg said that osteopaths should, "Attempt to substantiate osteopathic observation by research in the basic sciences of anatomy, physiology, biochemistry and pharmacology for three reasons: Firstly, the great advances in medicine in the future would be through the biologic sciences. Secondly, straightforward and clear-cut answers can be attained through studying health and disease mechanisms in the basic sciences in contrast to the enormously complicated studies which are involved in clinical research. Lastly, many of the inter-professional conflicts might be avoided by working with basic scientists." Even so research in the United Kingdom was non-existent until 1976 and the publication of Burton's OAGB project. (Appendix 1, Note 3).

Gibbons and Tehan (1997) recognise the requirement for active research in osteopathy as an essential stage in the development of osteopathy as a primary health care resource. There are increasing budget pressures on all forms of health care and therefore a drive to prove that individual treatments actually achieve the efficacy they claim. Furthermore, if the osteopathic profession is seeking acceptance from the established medical model then valid research is vital.

Evidence based research means clinically relevant basic research which may take the form of mechanistic studies in the medical sciences or patient-centred research into the accuracy and power of many elements such as diagnosis, prognostic markers and the efficacy and safety of therapeutic, rehabilitative and preventative practices. Classically there have been proponents for each type of research, especially where research funds are limited, Bogduk and Mercer (1995) maintain that it is, "more valuable to demonstrate the efficacy of a therapy before one explores its mechanism". The reality may be that both are needed and in fact compliment each other to provide evidence that a positive outcome has been generated by a documented and understood biological and physiological mechanism. Then, and only then can properly conducted, valid and reproducible research fully legitimise any therapy.

But, more than this, the vast amount of research that exists and that is being produced varies vastly in quality of methodology and accuracy. The systematic review seeks to reduce the time the busy clinician requires to find and digest relevant literature so that he can find the best clinical evidence in order to develop a treatment plan. The systematic review aspires to present this best evidence, based on analysis of collated existing studies, in a form that is accurate and succinct.

There are many problems and difficulties in medical research, osteopathic medicine being no exception, which make the utopian view of valid, relevant and reproducible research very hard to achieve. Among these difficulties lie such problems as inter-practitioner reliability, lack of standardised terminology, a lack of accurate systems for reporting subjective observations (whether they be clinician observation or patient pain) and a realisation that the understanding of the aetiology of many presenting complaints (such as low back pain) has defied previous research attempts. Coupling this with the need to direct scarce funding effectively one begins to appreciate the problems facing the osteopathic profession.

The author believes that in order for osteopathy to be able to "take it's rightful place" the osteopathic profession must be seen to be justifying, developing and reflecting upon and validating its historical claims in light of modern science. The research that has been carried out must be rationalised and presented to the established medical community in standardised terminology in a way that is understood. Only by doing this can a platform for historical work to be built upon be created allowing osteopathy to progress to become an equal part of the establishment. The rules of performing 'good science' have been laid out and accepted and so osteopaths must acknowledge this, however, this must be done in such a way as to ensure that the questioning nature and philosophy of osteopathy not be eclipsed.

5.0 Methodology

In order to carry out a meaningful systematic review the author must closely identify the studies that are relevant to the research question. This may only be achieved if the inclusion and exclusion criteria are well delineated and a thorough and comprehensive search strategy is employed.

For studies to be included in this systematic review they must:

- Examine and provide evidence for the existence of segmental somatic dysfunction.
- The studies must be experimental or multi-case observational (single case reports to be excluded).
- Be published and written in the English language. (Abstracts and unpublished articles (grey literature) will be excluded from the study although not discounted as a source of information leading to the identifying of relevant papers).
- Be published in the last 20 years.

Advice was sought from tutors and lecturers to identify the broad parameters of inquiry. The search strategy employed a first level computer survey for relevant literature in the MEDLINE and AMED databases using the search terms, described in Appendix 2 Section 1, singly and in combination. Searches were made using both UK-English and American-English spelling. Online searches using general internet search engines such as Infoseek, Lycos and Magellan, as well as specific medical resources such as Medscape, Bandolier and the Cochrane collaboration were used with similar search terms. This initial IT search enabled the identification of:

- Specific studies.
- Researchers relevant to the field of study.
- Candidate journals for relevant publication.

Having identified firm and possible candidates for inclusion, these papers were retrieved and their references scrutinised for further relevance. Using the experience gained, a second level computer search was then carried out using refined search terminology. Hand-searches were then made through the indexes of the identified journals. Papers were retrieved from the BSO library, by interlibrary loan or directly from the Science Reference Library.

It was discovered that certain issues of the Journal of the American Osteopathic Association (JAOA) were not sent to England. Consequently, it proved impossible to obtain studies that were published in these issues through the conventional channels. In order to try and trace these papers the authors were contacted and asked to supply copies. Contact details were obtained from the abstract or from the authors' institution website personnel directory. Requests were also made to the American Osteopathic Association. These requests were made by e-mail, but unfortunately not all of these were acknowledged or any papers received (Appendix 2, Section 2).

In all, eleven studies were identified as relevant and retrieved, consisting of six observational correlations and five experimental treatments (Appendix 3).

In order to assess and assign value to the studies, scoring criteria were devised which represented aspects of the study design, implementation and analysis that were considered important for this area of investigation. The scoring criteria were based upon and adapted from previously conducted systematic reviews (Ter Riet *et al*., 1990; Koes *et al*., 1991; Anderson *et al*., 1992; Koes *et al*., 1993; Koes and van den Hoogen, 1994; Van der Heijden *et al*., 1995).

There is no consensus as to what factors determine trial quality, or indeed if there is a minimum or maximum criteria list (de Bie, 1996). Therefore, the determination of scoring criteria depends on the area and type of study being reviewed, with the assignment of a value to each criterion a subjective exercise. After a period of trial and error the scoring criteria were refined and the weighting of the scores considered suitably sensitive to reveal the differences in methodological quality between the studies. The final scoring criteria and their weightings are shown in

Table 1 and detailed in Appendix 4. There were three variations in the scoring criteria: Firstly, a binary criterion received an all-or-nothing score depending on its presence or absence. Secondly, a multiple-choice criterion had levels of award reflecting their different quality assessment. Lastly, the subjective criteria were assessed by the author on the basis of individual merit and in relation to the overall study and were awarded a score from a narrow range. Where a single criterion was stated, absence of compliance with that criterion scored zero. The criteria were applied independently of each other and in cases of their doubt or conflict punative marking was applied. The maximum score for each study was 100 points. It was considered that consistency of scoring would serve to help mitigate any error due to subjectivity, as each study would be similarly exposed.

The criteria were grouped together into five sections reflecting different aspects of the studies methodology, execution and conclusions: Study design, study population, interventions, effect and data presentation, results and analysis.

5.1 Study Design

Criterion A examines whether the authors have approached their study from a point of established research. This is not to say that novel ideas were penalised for challenging existing ideas but that the study question shows evidence of considered and appropriate background research.

Criterion B serves to make the distinction between the two types of study being subjected to systematic review. This criterion weights in favour of the experimental approach over the observational, with the arguably judgemental reasoning that an experimental study is more objective. The accepted ‘gold standard’ for clinical research is considered to be the randomised, clinical trial (RCT). However, it was decided to keep this criterion binary and the merits of RCT considered and marked in subsequent criteria. This allows for a more sensitive scoring scheme. Another reason is that there is a paucity of contemporary experimental research in this field and to restrict entry to RCT’s would mean the rejection of most if not all papers. An extra mark was awarded if there was evidence of a pilot or previous study on

the basis that this allows for the identification and solution of procedural errors and difficulties.

Randomisation issues and procedures concerned with the assignment of subjects to either the intervention or the control group are dealt with by criterion C. Marks were awarded for evidence of randomisation, explicit description of the procedure used and a randomisation process that excludes selection bias in this context. For example, were subjects personally assigned to a group or was this achieved by use of an objective method such as a random number generator? The process of randomisation allows a more equal dissemination of potential errors and confounding variables and helps mitigate any possibility of subject preference. Randomisation is also important in the initial recruitment and selection of subjects and this is considered later under criterion E - Homogeneity.

Criterion D awards marks for the explicit description of the control procedures utilised. It was decided that because control procedures are a central methodological component of an acceptable study, this criterion would be scored on an all-or-nothing basis. In studies involving manipulative interventions it is acknowledged that the administration of a control placebo is difficult. Evidence of satisfactory control would be the use of a 'sham' manipulation or procedure that conferred the control subjects no apparent benefit (Appendix 1, Note 4).

5.2 Study Population

The level of homogeneity within the study population is considered under criterion E. This involves the examination of recruitment comparability between the subjects and marks were awarded for the statement and definition of subject inclusion and exclusion criteria. Further marks were available if there was evidence of an attempt to limit recruitment or comparison to subjects of similar test variable similarity. For instance Cox *et al*., (1983), in their study into musculoskeletal findings in coronary artery disease, group their subjects into severity of coronary disease with 'significant coronary disease' being defined as, *"One or more vessels being 50 percent or more stenosed"* when assessed by angiography. Similarly,

another mark was given if there had been efforts to provide randomised or unbiased recruitment. *"In population based studies random sampling is the ideal method of avoiding selection bias and producing a sample typical of the study population"*, Fowkes and Fulton (1991). However, this last criterion is difficult to comprehend and enforce due to the occurrence of an operational conflict; if subjects with a particular baseline condition are being sought then it stands to reason that the best place to recruit is from a relevant clinic list, for instance cardiac patients from a cardiac department. However, this may be considered a biased selection procedure (referral bias). This criterion is therefore seeking to identify a degree of randomness or attempt to mitigate subjective recruitment decisions that may be operative in this procedure. Therefore, scoring this category is in itself highly subjective.

Distinct from the concept of population homogeneity, criterion F addresses the fundamental comparability of selected subjects in an attempt to provide a uniform environment across which the study may be executed and conclusions drawn. The scenario surrounding the research question was analysed and factors considered threatening to this comparability identified.

Criteria G and H reflect the size of the sample and report on the number of dropouts respectively. The larger study populations were rewarded, as were those studies that either had no dropouts or report their dropouts faithfully. Accurate reporting is necessary because it is possible that a high dropout rate may lead to an incomparable study population and cause a skew in the overall result. Therefore, the result would have to be viewed with this fact in mind.

5.3 Interventions

Criterion I confers marks for full description of the experimental assessment or treatment procedure used and extra points were available if the position of the subject during intervention was described together with the acknowledgement of the number of assessment or treatment sessions. Criterion J rewards statement of operator experience or qualification.

The standardisation of the intervention procedure was examined under criterion K. Maximum marks were awarded to those studies which maintained a consistent operator. Studies that described attempts to standardise interventions between different operators scored less whilst those that made no mention or effort to standardise operator or procedure were penalised with a zero score.

Criterion L presents a subjective marking range addressing the strategies of coping with cointerventions. Cointerventions are extrinsic factors that may be beyond the control of the investigators such as change in subject lifestyle (exercise, diet etc), self medication or treatment and change to a previously prescribed drug regimen.

5.4 Effect

Blinding is an important aspect of any quality experimental intervention as it constitutes a significant systematic bias that may arise in the unconscious (or conscious) desire of the subject or operator to achieve a particular outcome. Both subject and operator (observer) blinding was considered in criterion M and scored equally highly. However, blinding involving manipulative procedures is impossible from the operator's point of view and only a subject naïve to manipulative treatment may be considered blinded although the elements of preconception and prior knowledge in the subject cannot fully be accounted for.

Criterion N addresses the definition, number and type of outcome measured in each study. There are many, well documented, changes that are observable and associated with segmental somatic dysfunction. Studies scored well if the authors had taken time to describe the particular indicators used. A further point was awarded for each different outcome measurement taken, therefore rewarding those studies that used multiple indicators. The changes may be objective or subjective and more marks were available if there was amplification by use of instrumentative measurement, eg the use of a tissue compliance meter to assess muscle hypertonicity (Nansel *et al*., 1993) or electromyography (Ellestad *et al*., 1988). Subjective changes may similarly be increased in reliability with the use of visual

analogue scales or questionnaires (Walsh and Polus, 1999). Indirect outcome methods may be observed in the alteration of disease severity or symptomology which may involve a change in reported subject wellbeing or variation in objective clinical findings such as endoscopic evaluation of duodenal ulcer progression (Pikalov and Kharin, 1994).

Criteria O and P are respectively concerned with the time of measurement and whether a follow up assessment was carried out. Time of measurement is important and immediate outcome measurement was rewarded over delayed. A follow up assessment would be valued, as it would demonstrate the level of stability in the findings and identify any delayed effects.

5.5 Data presentation, results and analysis

Points were allocated for the clear and adequate presentation of the most relevant outcome measures under criterion Q.

Criterion R awards more points if the statistical analysis involves the application of specific tests rather than inferred or descriptive reporting. The description and relevance of analysis is assessed and rewarded by criterion S.

A study was considered positive and scored, if the authors demonstrated or concluded that there was a significant difference between the groups studied. Depending on the type of study, this might be a positive correlation of segmental somatic dysfunction to a baseline condition or a positive outcome after treatment intervention. If a study was deemed equivocal, no change or no significant difference detected or a negative result returned, then no marks were awarded under criterion T.

Finally, criterion U offers a range of marks for the identification of bias, errors and confounding factors. A range of marks were awarded at the discretion of the reviewer depending on whether it was felt that the study authors had suitably addressed these issues. It was decided to score in this manner due to the fact that,

because the studies were different in protocol, it would be easier and more equitable to score on the basis on individual relevance and merit.

In order to apply these scoring criteria to the selected studies a scoring table was drawn up (Appendix 2, Section 3) so that a comprehensive description of each result could be recorded. After this was done the scoring system was applied and a total score achieved.

In order to mitigate single observer subjectivity, the eleven studies were subjected to a second and independent scoring process. The detailed scoring criteria were non-specifically explained to a colleague who was then blinded to study author, publication, date and journal. The colleague then carried out the same scoring process. In the case of disagreement over scoring close discussion resulted in consensus, which then provided a final agreed total score for each study.

Table 1: Scoring criteria (see Appendix 4 – Criteria breakdown):

STUDY DESIGN		SCORE
A:	Discussion of research question / hypothesis	1
B:	Type of study	4
C:	Randomisation	8
D:	Control Procedure	3
		16
STUDY POPULATION		
E:	Homogeneity	6
F:	Comparability of relevant baseline characteristics	11
G:	Study population size	3
H:	Withdrawals	3
		23
INTERVENTIONS		
I:	Explicit description of experimental assessment or treatment procedure	5
J:	Experience and qualifications of operator stated	3
K:	Standardisation of procedure	2
L:	Cointerventions avoided, comparable, neutralised or acknowledged	3
		13
EFFECT		
M:	Blinding performed and described	6
N:	Outcome measures	22
O:	Time of measurement	3
P:	Follow up performed	3
		34
DATA PRESENTATION, RESULTS AND ANALYSIS		
Q:	Data presented for most important outcome measures	3
R:	Statistical analysis	3
S:	Statistical tests described and relevant	2
T:	Significant difference or positive outcome demonstrated	3
U:	Discussion of bias, errors and confounding factors	3
		14

6.0 Results

At this time, eleven studies fulfilled the inclusion criteria, six of which were observational correlative studies (case controlled trials) and the remainder experimental treatments (clinical trials). Table 2 and Appendix 3 provide a study summary and methodological quality score while Table 3 shows the breakdown of quality scoring by criteria as previously discussed and detailed in Appendix 4.

The methodological study scores range from 36% (Beal and Morlock, 1984) to 63% (Ellestad *et al*., 1988). The average percentage scores (adjusted to the nearest whole number) were 42% for the six observational correlative studies and 54% for the five experimental treatment studies which displayed overall higher scores.

It was decided to statistically analyse the two study type populations in order to determine any difference in methodological quality. The null hypothesis was that there was no difference between the methodological quality of the observation correlative studies and the experimental treatments. A related T-test was applied which gave a probability of 0.015 at 9 degrees of freedom (Appendix 5). This was below the 0.05 confidence level and therefore the null hypothesis was rejected.

It may therefore be accepted that there appears to be a significant difference in methodological quality between a study involving observational correlation of segmental somatic dysfunction and a study involving an experimental treatment protocol. The studies utilising an experimental treatment procedure attaining significantly higher methodological scores.

Two studies assessed segmental somatic dysfunction with reference to coronary artery disease, two studies examined hypertension, the other studies assessed pulmonary disease, pneumonia, premenstrual syndrome, dysmenorrhoea, duodenal ulcer, lumbar paraspinal muscle tone and electromyography with skin resistance. These demonstrating conditions where segmental somatic dysfunction may be found and easily observed. Three of these studies were found in chiropractic research and the remainder from osteopathic research.

Several of the studies were described in their abstracts as being a 'randomised clinical trial' (Walsh and Polus, 1999) or a 'double blind study' (Cox *et al*., 1983). Although these studies displayed elements of these criteria, no study demonstrated a methodology that could be described as a 'double blind randomised clinical trial' in the form that is accepted as the gold standard for clinical research.

Of the six observational correlations, where subjects with a diagnosed baseline condition were screened for the presence of segmental somatic dysfunction, two studies were restricted to the cervicothoracic spine (Johnston and Hill, 1981; Johnston *et al*., 1982) the remainder observed the whole spine. All studies declared the operator's qualifications and all described the assessment procedure except for one (Beal and Morlock, 1984). Three studies maintained consistent operators, one study (Cox *et al*., 1983) explicitly described interoperator standardisation and two studies made no attempt to maintain consistent operators or describe a standardisation procedure (Johnston *et al*., 1982; Walsh and Polus, 1999). Only one study (Walsh and Polus, 1999) used a control procedure.

Of the five experimental treatment studies, all study interventions consisted of a range of techniques that included high-velocity thrust (HVT) but only two demonstrated operator qualification and adequate standardisation (Boesler *et al*., 1993; Noll *et al*., 1999). All of these studies described a control procedure but only two implemented a randomised subject allocation (Ellestad *et al*., 1988 and Nansel *et al*., 1993). Of these control procedures one study (Nansel *et al*., 1993) delivered a sham manipulation whilst the control group of Boesler *et al*., (1993) received no intervention. The remainder of the studies used conventional therapy as control. One experimental study employed operator blinding (Nansel *et al*., 1993) and one study blinded the subjects (Noll *et al*., 1999).

The outcome measures used varied in number and type with four studies utilising more than one outcome measure. Two studies used only range of movement (Johnston and Hill, 1981; Johnston *et al*., 1982), one study utilised electromyography (Boesler *et al*., 1993), another study measured electrical skin resistance (Ellestad *et al*., 1988), and another investigated muscle tone (Nansel *et*

al., 1993). Two studies assessed the objective and subjective change in symptomology of a diagnosed baseline condition (Pikalov and Kharin, 1994; Noll *et al*., 1999).

All studies were deemed to have positive outcomes by their researchers except for one (Noll *et al*., 1999) which was deemed equivocal.

Table 2 - Results
Methodological quality scores and study summary

Study	Methodological quality score	Type of study	Investigated condition	Result
Walsh and Polus. (1999).	52	Observational correlation	Premenstrual syndrome	Positive
Beal and Kleiber. (1985).	44	Observational correlation	Coronary artery disease	Positive
Beal and Morlock. (1984).	36	Observational correlation	Pulmonary disease	Positive
Cox *et al.* (1983).	44	Observational correlation	Coronary artery disease	Positive
Johnston, *et al.* (1982).	38	Observational correlation	Hypertension	Positive
Johnston and Hill. (1981)	37	Observational correlation	Hypertension	Positive
Noll, *et al.* (1999).	60	Experimental treatment	Pneumonia	Equivocal
Pikalov and Kharin. (1994).	44	Experimental treatment	Duodenal ulcer	Positive
Boesler, *et al.* (1993).	50	Experimental treatment	Dysmenorrhoea	Positive
Nansel, *et al.* (1993).	54	Experimental treatment	Lumbar paraspinal muscle tone	Positive
Ellestad, *et al.* (1988).	63	Experimental treatment	Electromyography and skin resistance	Positive

Table 3 – Quality scoring breakdown by criteria

Study	Criteria (Section maximum score)																				Total (percentage)
	A 1	B 4	C 8	D 3	E 6	F 11	G 3	H 3	I 5	J 3	K 2	L 3	M 6	N 22	O 3	P 3	Q 3	R 3	S 2	T 3	U 3	
Observational correlation (case controlled trial)																						
Walsh and Polus (1999).	1	2	0	3	4	7	3	2	3	3	0	0	0	7	3	0	3	3	2	3	3	52%
Beal and Kleiber (1985).	1	3	0	0	4	3	3	2	4	3	2	0	3	3	3	0	3	1	0	3	3	44%
Cox et al. (1983)	1	3	0	0	5	1	3	2	3	3	1	0	3	4	2	0	3	3	2	3	2	44%
Johnston, *et al.* (1982).	0	3	0	0	1	0	3	3	4	3	0	0	3	1	3	0	3	3	2	3	3	38%
Johnston and Hill (1981).	1	2	0	0	1	1	3	2	5	3	2	0	0	4	3	0	3	1	0	3	3	37%
Beal and Morlock (1984).	1	2	0	0	0	3	3	1	1	3	2	0	0	7	3	0	3	1	0	3	3	36%
Experimental treatment (clinical trial)																						
Ellestad, *et al.* (1988).	1	3	8	3	5	5	3	1	5	3	0	2	0	4	3	3	3	3	2	3	3	63%
Noll, *et al.* (1999).	1	3	0	3	5	4	3	1	5	3	1	3	3	8	3	3	3	3	2	0	3	60%
Nansel, *et al.* (1993).	1	4	3	3	1	4	3	2	5	0	2	0	3	7	3	0	3	3	2	3	2	54%
Boesler, *et al.* (1993).	1	3	0	3	3	4	3	1	5	3	2	1	0	6	3	0	3	3	2	3	1	50%
Pikalov and Kharin (1994).	1	3	0	3	5	2	3	1	1	3	0	1	0	5	2	0	3	3	2	3	3	44%

7.0 Discussion

The scoring criteria were designed to reflect the authors' conception of a notionally ideal research protocol. The choice of criteria, although rooted in accepted thinking, is still, essentially, a highly subjective process which is compounded by the subjective process of scoring. Although this is mitigated, to a certain extent, by a check scorer a consensus is still reached implying a degree of latitude in the scoring procedure. This *subjectivity error* is effectively magnified and built upon any subjectivity introduced by the authors of the original studies.

Each study therefore receives an overall score whose only value is in relation to other studies scored in exactly the same manner. However, what score is considered the cut-off between good and bad methodology? This author believes that it must be considered that any score below 100% represents, in the ambit of this study, a flawed methodology which would imply that the studies are to be judged on a sliding scale. If this is not the case then an arbitrarily chosen pass score must be taken which is again introducing an element of subjectivity.

A possible problem here might be that, even with a carefully designed set of criteria, the assessment may never be sensitive enough to adequately differentiate between the subtle variations in each study. It may be that, because there are many criteria to assess, most of which are only relevant to a few studies, the overall scores are diluted by irrelevant, low-scoring criteria. The ultimate in sensitivity would be to evaluate each paper individually upon its own merits but, clearly, this would be of limited value when trying to compare studies especially within the scope of a systematic review.

For example, if a methodology score of 50% is chosen as the cut-off between a good and bad methodology and that score used to make a decision as to the validity of the outcome of that study regardless of the conclusions, then we may observe the following situation. Study A passes with a score above 50% and Study B fails with a score below. Study A triumphs because it achieved many smaller weighted scores for minor elements of methodology but in the absence of the presence of significant criteria. Even with carefully considered weighting this may be possible due to the vagaries of study

design and the impossibility of being able to account for all design elements. Study B, however, possesses significant features and achieves good scores in these but lacks the many small refinements of Study A. On this basis, Study B fails to achieve the pass mark and by that token is considered to possess a bad methodology.

There is also a problem in trying to analyse something that is inherently difficult to define. As has been discussed, the definition of segmental somatic dysfunction is complicated by the many different concepts and domains that are involved and confused by the lack of clarity surrounding their interactions. If what is being investigated is not fully definable then how can it be known with any certainty that what is being observed is a reflection of the whole process. Through this confusion, can it be fully determined that the study under analysis is accurately focused upon segmental somatic dysfunction?

Similarly, in all the studies, somatic dysfunction is being indirectly assessed by the measurement of a manifestation, whether it be the course of a disease process or a local effect such as change in muscle tone. A difficulty arises when an inference as to the existence of a complex phenomena is attempted from studies that may only be concerned with a small aspect of the whole. Can it be said with any certainty that the outcome measurement is actually related to the segmental somatic dysfunction and, if so, is this an accurate relationship or is it so masked by remoteness or complex interaction as to be an inaccurate measure?

Looking at the individual study conclusions we see that all profess to demonstrate a positive outcome except Noll *et al.* (1999) who suggest that caution should be taken when interpreting and drawing conclusions from their results. Given the above arguments, this author feels that caution should be exercised in interpreting the results of this systematic review. Placing this into context, the total methodological scores for the eleven included studies are generally low implying poor quality studies which therefore questions the value of their conclusions. The methodological quality of each study suffers from the problems associated with research of this type for example, difficulty in blinding, difficulty in administering placebo, the vagaries of the human subject and the innumerable possible confounding factors in operation. Furthermore, the studies each deal with narrow and limited aspects of the effects of segmental

somatic dysfunction and therefore extrapolation of results would be of questionable value especially given the ambitious and far-reaching nature of the posed research question.

Dove, reporting Littlejohn, points out that the great problem in understanding osteopathic principles is, "over the distinction between the vertebral lesion and the osteopathic concept, the vertebral lesion being only a part of the osteopathic concept". (Dove, 1965). In this he is warning the reader against the dangers of viewing the somatic dysfunction as the totality of osteopathy and forgetting that the osteopathic concept is much wider than any single component. The surrounding ambiguity has been previously explored and it has been shown that the local and remote effects of spinal dysfunction are complex and wide ranging affecting or involving nearly all domains in their genesis and maintenance. Considering this, the difficulty in delineating the boundaries of existence, ramification and involvement of segmental somatic dysfunction may be appreciated and the paucity of research, especially research of good methodological quality, does little to redress this lack of clarity.

Is it therefore relevant to describe the concept of segmental somatic dysfunction as holistic? By this it is meant that if segmental somatic dysfunction is merely a small part of the osteopathic whole then it may be considered false to view it in isolation as representative of osteopathy within a holistic framework. However, if the emphasis of importance is expanded away from this almost reductionist view and it is seen within the context of an osteopathic philosophy then it may be argued that an holistic view of segmental somatic dysfunction is being adopted rather than considering it to be holistic in itself. This interpretation might encompass the relation of the existence of any dysfunction to its effects upon the integrated and individually appropriate functioning of a person.

If indeed the nature of somatic dysfunction is highly individualistic is it possible to compare subjects within studies let alone between studies? How can it be ensured that what is being investigated is the same phenomenon at least in terms of what segmental somatic dysfunction means within the context of each individual being studied. This brings us back to the questions surrounding definition.

This author considers that the true characterisation and nature of segmental somatic dysfunction cannot be found in a single description but different aspects are reflected in the various definitions. This stance may be considered by some to be a failure but, as has been noted by researchers many times, the expression of segmental somatic dysfunction varies by individual and so, although close and complete definition is valid and worthwhile, it seems unlikely that a single unified theory will be sufficient. Instead, as Denslow and Tyreman have suggested, definition lies in the comprehensive understanding of the surrounding concepts and recognition of the clinical presentation.

The issues surrounding this area may be seen to underpin and be central to the concepts, philosophies and practice of osteopathic medicine. Answers have been sought since the early days of osteopathy to such questions as: What is the origin of somatic dysfunction? How is it associated with the local physical findings? What are the remote effects of somatic dysfunctions and their response to treatment? How does manipulative treatment change the local condition, provide relief and restore health? (Ward, 1997).

The spinal manipulation is commonly assumed to be a hallmark of the osteopath yet a complete, cohesive model of the mechanisms behind this treatment intervention are poorly understood. It may be considered to be of paramount importance to investigate this area as fully as possible in order to define the footing upon which osteopathy is to continue to establish itself.

Because segmental somatic dysfunction is suggested to confer both local and remote effects in the body, the issues and questions raised are varied and important. Aside from the local treatment of a dysfunctional area of spine, a central question asks whether there is a segmental somatic component to frank visceral disease or is it simply mimicry of true pathology? Within this exists the question as to the definition of a ‘true pathology’. The possibility of mistaken diagnosis caused by this and therefore two distinct routes to the same diagnosis.

It can therefore be appreciated that such issues and questions would appear fundamental to the development of modern osteopathy. If the theories of segmental somatic dysfunction are correct then it will herald a revolution in the understanding and practice of medicine and prove important for all manual therapies. Clarification of these

questions is not only important for ourselves as osteopaths but also, in these days of accountability and evidence based medical practice, the osteopathic profession must be seen to be actively reflective and willing to submit to scrutiny.

But why is the issue of facilitation relevant to osteopaths at all? Would osteopathy continue to exist if the existence of segmental somatic dysfunction was categorically disproven? Has it become the sum total of osteopathy or is it that this concept has become an icon for the osteopathic profession to cling to representing something that cannot be adequately articulated? Lederman sees the answer to these questions in that the concept of the facilitated segment and subsequent somatic dysfunction reinforces and justifies the use of high-velocity thrust (HVT) as a therapeutic measure. It provides a greater "physiological depth" for the HVT and is considered to allow the osteopath to affect the patient on many more levels (Lederman, 2000). Lederman bemoans the fact that, as he sees it, this, possibly dogmatic view, has caused the osteopathic profession to stand still with respect to research. Fearful to find out whether the theories are right or wrong osteopaths are paralysed by the concept of somatic dysfunction. If wrong, does osteopathy cease to exist? If right, will osteopaths be capable of coping with the ramifications, after all, if true where does the theory of segmental somatic dysfunction begin and end?

By this token, the issue here is not why individual studies achieved low methodological scores but what this small sample of representative research suggests about a phenomenon that is hailed as being important.

The thinking that has been expended in this area is vast and yet, in the spirit of holistic, patient-centred, investigations, can the close experimental examination of single aspects provide a comprehensive and integrated solution? This is the nature of purely scientific enquiry which, although valid and necessary, may only hope to clarify on a small level as compared to the whole. A more complete understanding must occur when scientific inquiry and philosophical thought combine to produce a better, mutually complimentary model.

However, for the time, the world of scientific research is rooted in reductionist orthodoxy and there is much that can be learnt in this field by employing these

techniques. For surely, to fully comprehend a part of the whole is a step along the road to understanding the whole.

8.0 Conclusion

Through the process of this systematic review, it was demonstrated that the methodological quality of the eleven included studies was generally poor. On the basis of this study it would appear that there is inconclusive evidence to provide a full explanation and account of the existence of segmental somatic dysfunction.

The methodological analysis showed that there was a statistically significant difference in methodological quality between studies with an observational correlative protocol and those with an experimental treatment protocol. The latter proving more methodologically sound.

The confusion over terminology and definition, the difficulty in assessment and measurement and the issues surrounding the boundaries of segmental somatic dysfunction have been explored.

The difficulties surrounding the applicability, mechanics and execution of a systematic review in this area, with particular relevance to the subjectivity inherent to this process, have also been discussed.

In summary, the evidence presented is inconclusive about the existence of segmental somatic dysfunction and the author believes that it is a false conclusion to extrapolate beyond this study for the following reasons.

- The low number of studies available.
- The poor methodological quality of the studies.
- The question over how representative of segmental somatic dysfunction the studies are.
- The difficulties and confusion surrounding research in this field.
- The difficulties inherent in the process of systematic review and its applicability to this area.

It would appear rather strange that this, supposedly, central concept has attracted so little research. Throughout this history many eminent osteopaths and chiropractors have discussed and theorised on somatic dysfunction however, there has been no concerted or orchestrated effort to elucidate its nature. A reason for this may be the tenuous unease that is felt by practitioners when confronted with the possibility that suitable research into this area may remove what is considered to be the major factor in their continuing justification and practice. However, this author contests the notion that osteopathy is defined and constrained by the segmental somatic dysfunction. While it is necessary to understand this phenomenon, osteopathy cannot allow its development and progress to be ransomed by it.

In order to comprehend somatic dysfunction the domains involved must be identified and their dynamic interplay understood. In this manner the boundaries surrounding somatic dysfunction (however large or small) may be felt and it may be then possible to appreciate the entirety of the phenomenon. Once this is realised then there is potential to discover better and more efficient means of interacting and treating the dysfunction with the ability to scale intervention up or down depending on the domain type and level chosen for therapeutic entry.

From this study it is clear that there is a definite requirement for further research in this area to be carried out with sound methodological protocols. However, although quality scientific enquiry is essential, a philosophical exploration and the methodology of intuition will be also required in order to fully understand all aspects.

9.0 Appendices

9.1 APPENDIX 1 – Notes to text

Note 1:

This is assuming that reality is constant and able to be defined. If it is not then, the scientific models used are constantly invalid and represent nothing more than a ‘best fit’.

Note 2:

Component 2 in the stages of clinically applied EBM makes the assumption that the best available evidence actually exists. It may also be arguable as to what constitutes 'relevant and best'.

Note 3:

The first part of the quote by Dr. Gregg makes an enormous assumption that the future great advances in medicine will indeed be made through the biological sciences. His second part may also be questionable in that do studies into the health and disease mechanisms necessarily lead to 'straightforward and clear-cut' answers and there is a potential problem with the actual definitions of 'health' and 'disease'. These issues of validation are continued in the last part of his quote which presumes that 'basic scientists' represent the epitomy of research objectivity and are consequently beyond criticism. So, what types of inquiry are relevant for this area of study? Should it be wholly scientific or should humanitarian, philosophical, psychological or sociological elements be included?

Note 4:

It is arguable as to what manipulative procedures constitute treatment and by what domain, model or level treatment is being described and explained. The neurological model may describe the neurological effects of high-velocity-amplitude thrust but a spiritual or affective model may talk in terms of ‘laying on hands’. This is all very well, but when attempts are made to isolate these potentially conflicting effects, with the

intention of studying them individually, problems are faced. How do researchers know that their control placebo sham manipulation is not serving as a valid therapeutic intervention via some other mechanism?

9.2 APPENDIX 2 – Methodology

9.2.1 Section 1: Search terms used

Database: Medline <1996 to January 2000>
Medline <1993 to 1995>

Set	Search	Results
1	Osteopathic lesion.tw	0
2	Somatic dysfunction.tw	15
3	Segmental dysfunction.tw	8
4	Segmental facilitation.tw	0
5	Facilitated segment.tw	2
6	Segmental sensitisation.tw	0
7	Segmental sensitization.tw	0
8	Segmental habituation.tw	0
9	Habituated segment.tw	0
10	Sensitised segment.tw	0
11	Sensitized segment.tw	0
12	Segmental.tw	3585
13	Facilitated.tw	4402
14	Sensitised.tw	142
15	Sensitized.tw	2585
16	Habituated.tw	129
17	Osteopathy.tw	89
18	Osteopathic.tw	146
19	17 or 18 (Osteopathy or Osteopathic)	229
20	12 and 19 (Segmental and Osteopathy or Osteopathic)	1
21	13 and 19 (Facilitated and Osteopathy or Osteopathic)	1
22	15 and 19 (Sensitized and Osteopathy or Osteopathic)	0
23	Segmental.tw	3583
24	23 and 19 (Segmental and Osteopathy or Osteopathic)	1

25	Dysfunction.tw	19062
26	25 and 19 (Dysfunction and Osteopathy or Osteopathic)	7
27	Facilitation.tw	1927
28	27 and 19 (Facilitation and Osteopathy or Osteopathic)	0
29	Sensitisation.tw	228
30	29 and 19 (Sensitisation and Osteopathy or Osteopathic)	0
31	Sensitization.tw	3293
32	31 and 19 (Sensitization and Osteopathy or Osteopathic)	0
33	Habituation.tw	572
34	33 and 19 (Habituation and Osteopathy or Osteopathic)	0
35	Subluxation.tw	547
36	Chiropractic.tw	301
37	35 and 36 (Subluxation and Chiropractic)	8
38	Subluxed.tw	21
39	36 and 38 (Chiropractic and Subluxed)	0
40	35 and 19 (Subluxation and Osteopathy or Osteopathic)	0
41	38 and 19 (Subluxed and Osteopathy or Osteopathic)	0
42	23 and 35 (Segmental and Subluxation)	5
43	25 and 36 (Dysfunction and Chiropractic)	14
44	12 and 36 (Segmental and Chiropractic)	6
45	14 or 15 (Sensitised or Sensitized)	2724
46	36 and 45 (Chiropractic and Sensitised or Sensitized)	0
47	29 or 31 (Sensitisation or Sensitization)	3516
48	36 and 47 (Chiropractic and Sensitisation or Sensitization)	0
49	16 or 33 (Habituated or Habituation)	657
50	36 and 49 (Chiropractic and Habituated or Habituation)	0
51	13 or 27 (Facilitated or Facilitation)	6068
52	36 and 51 (Chiropractic and Facilitated or Facilitation)	0
53	15 and 19 (Sensitised and Osteopathy or Osteopathic)	0

Searches were made using both UK-English and American-English spelling.

Literature search complete at time of submission.

9.2.2 Section 2: Example E-Mail requesting a copy of an unavailable study

E-Mail requests were made directly to the author and to the American Osteopathic Association. E-Mail addresses were determined from published abstracts or from the authors institution website personnel directory.

From: Baskeyfield@holism.co.uk
To: craigwax@mem.po.com
Subject: Chest pain and the role of somatic dysfunction
Dear Sir,
Please forgive the unsolicited E-Mail. I am a final year student of Osteopathy at the British School of Osteopathy, London, UK.
To fulfil the requirements for graduation, final year students must undertake a dissertation. I am currently performing a systematic review that is attempting to assess the quality of evidence for the existence of segmental somatic dysfunction.
In order to do this I have identified candidate studies to include and, from the abstract, your paper, "Chest pain and the role of somatic dysfunction", appears to meet my inclusion criteria.
However, copies of your paper are not available in the United Kingdom. It seems as though certain issues of the Journal of the American Osteopathic Association were never delivered to England. The British Library does not hold them and attempts to trace copies have been frustrated.
I was therefore wondering if you would be kind enough to send me / E-Mail me a copy of this paper. It would greatly aid my dissertation and also help to improve the holding in our school library.
I thank you in advance for your time and effort.
Yours sincerely,
David JH Baskeyfield
British School of Osteopathy
275 Borough High Street
London, SE1 1JE
United Kingdom
Baskeyfield@holism.co.uk

9.2.3 Section 3: Criteria scoring table

Study title	
A: Discussion of research Q / hypothesis	
B: Type of study	
C: Randomisation	
D: Control procedure	
E: Homogeneity	
F: Comparability of baseline characteristics	
G: Study population size	
H: Withdrawals	
I: Description of experimental assessment or TTT procedure	
J: Operator qualification	
K:Standardisation	
L:Cointerventions	
M: Blinding	
N: Outcome measures	
O: Time of measurement	
P: Follow up performed	
Q: Data presented for important outcomes	
R: Statistical analysis	
S: Statistical tests described and relevant	
T: Significant difference or positive outcome	
U: Discuss bias, error & confounding variables	

9.3 Appendix 3 - Included studies

Reference	Title	Type of study
Walsh, M.J. and Polus, B.I. (1999). *JMPT,* Vol.**22**, No.4,. p.216-220.	The frequency of positive common spinal clinical examination findings in a sample of premenstrual syndrome sufferers.	Observational correlation
Beal, M.C. and Kleiber, G.E. (1985). *JAOA,* Vol.**85**, No.5, p.302-307.	Somatic dysfunction as a predictor of coronary artery disease.	Observational correlation
Beal, M.C. and Morlock, J.W. (1984). *JAOA,* Vol.**84**, No.2, p.179-183.	Somatic dysfunction associated with pulmonary disease.	Observational correlation
Cox, J.M. *et al*. (1983). *JAOA,* Vol.**82**, No.11, p.832-836.	Palpable musculoskeletal findings in coronary artery disease: Results of a double blind study.	Observational correlation
Johnston, W.L. *et al.* (1982). *JAOA,* Vol.**81**, No.12, p.830-836.	Identification of stable somatic findings in hypertensive subjects by trained examiners using palpatory examination.	Observational correlation
Johnston, W.L. and Hill, J.L. (1981). *JAOA,* Vol.**81**, No.1, p.22-28.	Spinal segmental dysfunction: Incidence in cervicothoracic region.	Observational correlation
Noll, D.R. *et al.* (1999). *JAOA,* Vol.**99**, No.3, p.143-152.	Adjunctive osteopathic manipulative treatment in the elderly hospitalised with pneumonia: A pilot study.	Experimental treatment
Pikalov, A.A. and Kharin, V.V. (1994). *JMPT,* Vol.**17**, No.5, p.310-313.	Use of spinal manipulative therapy in the treatment of duodenal ulcer: A pilot study.	Experimental treatment
Boesler, D. *et al.* (1993). *JAOA,* Vol.**93**, No.2, p.203-214.	Efficacy of high-velocity low-amplitude manipulative technique in subjects with low-back pain during menstrual cramping.	Experimental treatment
Nansel, D.D. *et al.* (1993). *JMPT,* Vol.**16**, No.2, p.91-95.	Effect of cervical spinal adjustments on lumbar paraspinal muscle tone: Evidence for facilitation of intersegmental tonic neck reflexes.	Experimental treatment
Ellestad, .SM. *et al.* (1988). *JAOA,* Vol.**88**, No.8, p.991-997.	Electromyographic and skin resistance responses to osteopathic manipulative treatment for low-back pain.	Experimental treatment

9.4 Appendix 4 – Criteria breakdown

9.4.1 Study Design

A: Discussion of research question / hypothesis (evidence of background research) (1)

B: Type of study:

Experimental (3)

or

Observational (2)

Pilot or previous study carried out (1)

C: Randomisation:

Random assessment / treatment and control group allocation (3)

Randomisation procedure explicitly described (3)

Randomisation procedure that excludes bias (2)

D: Control Procedure:

Control procedure explicitly described (3)

9.4.2 Study Population

E: Homogeneity:

Inclusion / exclusion criteria stated (3)

Restriction to a homogenous population of severity (1)

Evidence of randomised or unbiased recruitment (1)

F: Comparability of relevant baseline characteristics:

Comparable age (subjects within 10 years) or restriction to a specific age group (1)

Psychological profile assessed and stable (1)

Patient knowledge and expectations assessed and comparable (1)

Confirmed previous diagnoses / assessment of baseline condition (1)

Comparable diagnoses (type and number of pathologies) (1)

Activities / trauma accounted for (1)

Comparable chronicity of baseline condition (1)

Prior manipulative treatment (1)

Male : Female ratio to within 10 subjects (1)

Handedness accounted for (1)

Subject stabilisation before and during study (1)

Comparability of specific characteristics relevant to the study (1)

G: Study population size:

>100 (3)

or

>50 (2)

or

<50 (1)

H: Withdrawals:

No dropouts or number of dropouts stated (3)

9.4.3 Interventions

I: Explicit description of experimental assessment or treatment procedure (3)

Subject position (1)

Number of treatment / assessment sessions described (1)

J: Experience and qualifications of operator stated (3)

K: Standardisation of procedure:

Operator consistent (2)

or

Explicit description of standardisation method between operators (1)

or

No standardisation or no explicit description (0)

L: Cointerventions avoided, comparable, neutralised or acknowledged (eg change in prescribed medicine, concurrent physical treatment) (3) (2) (1) (0)

9.4.4 Effect

M: Blinding performed and described:

Of subjects (3)

Of operator / observer (3)

N: Outcome measures:

Indicators of outcome measurements defined (3)

Decreased ROM (1)

Erythema (1)

Skin temperature change (1)

Localised sweating (1)

Asymmetry (1)

Hyperaesthesia (1)

Change in skin texture (1)

Muscle hypertonicity (1)

Electrical activity (EMG / Resistance) (1)

Subjective scale (eg visual analogue scale or questionnaire) (2)

Instrumentative qualitative, objective measurement (3)

Subjective change in disease symptomology (2)

Objective change in disease symptomology (3)

O: Time of measurement:

Immediately after assessment / treatment (3)

or

Delayed after assessment / treatment (2)

P: Follow up performed (3)

9.4.5 Data Presentation, Results and Analysis

Q: Data presented for most important outcome measures (3)

R: Statistical analysis:

Applied (3)

or

Inferential (1)

S: Statistical tests described and relevant (2)

T: Significant difference or positive outcome demonstrated (3)

U: Discussion of bias, errors and confounding factors (3) (2) (1) (0)

Where single criteria stated: Absence of compliance with that criteria will score 0.
Criteria applied independently.
In cases where there is doubt or conflict punative marking is applied.
In some cases (eg in the discussion of bias, errors and confounding factors) the author has subjectively awarded a score from a range.

9.5 Appendix 5 - T-test calculation

Observational	Experimental
52	63
44	60
44	54
38	50
37	44
36	

n_1 =	6	n_2 =	5
Σx_1 =	251	Σx_2 =	271
Σx_1^2 =	10685	Σx_2^2 =	14921
$(_1$ =	41.83	$(_2$ =	54.2

$\Sigma x_1^2 = \Sigma x_1^2 - (\Sigma x_1)^2 / n_1 = 10685 - 251^2 / 6$

$= 10685 - 10500.17$

$(n_1 - 1)s_1^2 = 184.83$

$\Sigma x_2^2 = \Sigma x_2^2 - (\Sigma x_2)^2 / n_2 = 14921 - 271^2 / 5$

$= 14921 - 14688.20$

$(n_2 - 1)s_2^2 = 232.80$

$s^2 = \Sigma x_1^2 + \Sigma x_2^2 / (n_1 - 1) + (n_2 - 1) = 184.83 + 232.80 / 5 + 4$

$= 417.63 / 9$

$= 46.40$

$df = (n_1 - 1) + (n_2 - 1) = 9$

$sd = \sqrt{s^2 (n_1 + n_2) / n_1 n_2} = \sqrt{46.40 (6 + 5) / 30}$

$= \sqrt{17.01}$

$= 4.12$

$t = d / sd = 41.83 - 54.20 / 4.12$

$= -3.00$ with 9 degrees of freedom

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