



COMPARATIVE STUDY

# Osteopathic manual therapy versus conventional conservative therapy in the treatment of temporomandibular disorders: A randomized controlled trial

A.M. Cuccia<sup>a,b,\*</sup>, C. Caradonna<sup>a,b</sup>, V. Annunziata<sup>b</sup>, D. Caradonna<sup>a,b</sup>

<sup>a</sup> Department of Dental Sciences "G. Messina", University of Palermo, Via del Vespro 129, 90128 Palermo, Italy

<sup>b</sup> School of Specialization in Orthodontics, University of Palermo, Via del Vespro 129, 90128 Palermo, Italy

Received 10 April 2009; received in revised form 1 August 2009; accepted 12 August 2009

## KEYWORDS

OMT;  
Physical therapy;  
Stomatognathic system;  
Occlusal splint;  
Masticatory muscle

**Summary Objective:** Temporomandibular disorders (TMD) is a term reflecting chronic, painful, craniofacial conditions usually of unclear etiology with impaired jaw function. The effect of osteopathic manual therapy (OMT) in patients with TMD is largely unknown, and its use in such patients is controversial. Nevertheless, empiric evidence suggests that OMT might be effective in alleviating symptoms. A randomized controlled clinical trial of efficacy was performed to test this hypothesis.

**Methods:** We performed a randomized, controlled trial that involved adult patients who had TMD. Patients were randomly divided into two groups: an OMT group (25 patients, 12 males and 13 females, age  $40.6 \pm 11.03$ ) and a conventional conservative therapy (CCT) group (25 patients, 10 males and 15 females, age  $38.4 \pm 15.33$ ).

At the first visit (T0), at the end of treatment (after six months, T1) and two months after the end of treatment (T2), all patients were subjected to clinical evaluation. Assessments were performed by subjective pain intensity (visual analogue pain scale, VAS), clinical evaluation (Temporomandibular index) and measurements of the range of maximal mouth opening and lateral movement of the head around its axis.

**Results:** Patients in both groups improved during the six months. The OMT group required significantly less medication (non-steroidal medication and muscle relaxants) ( $P < 0.001$ ).

\* Corresponding author at: Department of Oral Sciences, University of Palermo, Via del Vespro 129, 90128 Palermo, Italy. Tel.: +39 091 6552296/6811287; fax: +39 091 214637.

E-mail address: [cucciaam@odonto.unipa.it](mailto:cucciaam@odonto.unipa.it) (A.M. Cuccia).

**Conclusions:** The two therapeutic modalities had similar clinical results in patients with TMD, even if the use of medication was greater in CCT group. Our findings suggest that OMT is a valid option for the treatment of TMD.

© 2009 Elsevier Ltd. All rights reserved.

## Introduction

Temporomandibular disorders (TMD) are a collective term that includes disorders of the temporomandibular joint (TMJ), of the masticatory muscles and their associated structures in the absence of other visceral pathology (for example ear disorder, pharyngeal tumour, or dental abscess). It is characterized by pain, joint sounds, and restricted mandibular movement (De Bont et al., 1997).

The pathogenesis of the TMD, however, is unclear. Physical (trauma, muscles spasms, chronic malocclusion, bruxism causing grinding or clenching of teeth), biochemical (vitamin inadequacy), and physiological factors (anxiety, stress and depression) may all play a role (Levy and Gortin, 1953; Haskin et al., 1995). Upledger (1987) stated that TMD may originate from sacral dysfunction (Upledger, 1987).

Several types of treatment have been proposed in the literature by dentists, orthodontists, psychologists, physical therapists, and physicians, although with highly disparate results among the published studies (Cascos-Romero et al., 2009).

non-invasive therapies should be attempted before pursuing invasive, semipermanent or permanent treatments (such as orthodontics or surgery) that have the potential to cause irreparable harm.

Non-invasive therapies may include pharmacological treatment (non-steroidal anti-inflammatory drugs, muscle relaxants, antidepressants and corticosteroids), oral appliances, home care procedures, cognitive-behavioral information program, acupuncture, and dry needling, chiropractic, physical therapy, osteopathy, relaxation and meditation (Carlson et al., 2001; DeBar et al., 2003; Alcantara et al., 2002; Buescher, 2007).

Physical therapy is intended to relieve musculoskeletal pain, reduce inflammation, and restore oral motor function. The American Academy of Craniomandibular Disorders and the Minnesota Dental Association have cited physical therapy (electrophysical modalities, therapeutic exercises, manual therapy techniques) as an important treatment to relieve musculoskeletal pain, reduce inflammation and restore oral motor function (Sturdivant and Fricton, 1991).

Numerous physical therapy interventions are potentially effective in managing TMD. These therapies include: electrophysical modalities (shortwave diathermy, ultrasound, biofeedback, microwave, laser therapy and transcutaneous electrical nerve stimulation), acupuncture, therapeutic exercises for the masticatory or cervical muscles and manual therapy techniques. These interventions are commonly used to reduce pain and to improve mandibular range of motion (McNeely et al., 2006).

Osteopathic treatment is a physical therapy intervention, characterized by fine manipulative techniques, less invasive than other interventions, individually adapted to

tissue quality, in order to maintain or restore the circulation of body fluids (Magoun, 1976).

Osteopathic treatment is utilized by many practitioners of neuromusculoskeletal medicine and osteopathic manipulative medicine in many countries, including the United States of America, Australia, South Africa and the United Kingdom. The evidence regarding treatments is from clinical reports, patient outcomes and is largely anecdotal.

Only a few studies evaluated the effect of osteopathic treatment in TMD (Larsen, 1976; Royder, 1981). Monaco et al., (2008) suggested that OMT can induce changes in the stomatognathic dynamics, offering a valid support in the clinical approach to TMD.

The purpose of the current case-control study was to study the effects of OMT in adult subjects with TMD.

## Methods

The subjects in this study were recruited from among the patients with TMD who attended the Department of Orthodontics and Gnathology, University of Palermo, Italy, during a six-month period from September 2008 to February 2009. A total of 50 consecutive patients, aged 18–50 years, diagnosed with TMD were selected for the study. The subjects were randomly assigned to the OMT group (25 patients, 12 males and 13 females, age  $40.6 \pm 11.03$ ) and a conventional conservative treatments (CCT group, 25 patients, 10 males and 15 females, age  $38.4 \pm 15.33$ ).

A standardized TMD examination was executed in all patients: joint pain, crepitation, uncoordinated movements of the head of the mandibular condyles during opening or closing the mouth were investigated by lateral and posterior palpation of each TMJ with both index fingers.

Subjects were included if they had a temporomandibular index (TMI) reference value of  $\geq 0.08 \pm 0.10$ , and a minimum pain intensity of 40 mm on a visual analogue scale (VAS). The TMI is a clinical measure used to determine the severity of the disorder. It is composed of a total index (TMI) with three sub-indices: function index (FI), muscle index (MI) and joint index (JI). The FI includes 12 items related to the range of motion of the mandible. The MI measures pain associated with bilateral digital palpation of selected masticatory muscles at a total 20 sites. The JI measures pain evoked by digital palpation of 2 sites for each TMJ and the incidence of noise in each TMJ. The FI, MI and JI are calculated by dividing the sum of positive findings for each subindex by the total number of items examined (respectively 12 for FI, 20 for MI and 8 for JI). The scores of all indices ranged from 0 to 1, with 1 being the highest score possible. The overall TMI score is the average of the scores for the FI, MI, and JI. (Pehling et al., 2002).

The intensity of jaw pain was recorded on the visual Analogue (VAS) pain scale of 1–10 with 1 indicating mild pain, 5 moderate pain and 10 unbearable pain (Huskisson, 1974).

In addition, assessment of the range of maximal mouth opening (MOV) and lateral movement of the head around its axis were examined (ROM).

Maximal mouth opening was measured using calibrated caliper with a 1 mm accuracy, as the maximal inter-incisal distance added to the vertical overlap of the incisors. Patients were asked to open their mouth as wide as possible to the point of pain, and were measured with their heads supported in a neutral position (Figure 1).

The Cervical Range of Motion instrument (Performance Attainment Associates, 958 Lydia Drive, Roseville, MN 55113) was used in order to measure the rotation of the cervical spine on the transverse plane. This instrument consists of an eyeglass-shaped plastic frame with inclinometers. For the rotation measures (degrees), the inclinometer is magnetic and moves along the transversal plane (Neiva and Kirkwood, 2007).

The exclusion criteria were: history of adverse effects with osteopathic treatment, being under orthodontic treatment or under treatment for TMD, previous treatment for TMD, making regular use of analgesic or anti-inflammatory drugs, use of dental prosthesis, presence of any other oro-facial pain condition, neurological or psychiatric disorders and systemic inflammatory disorder.

The OMT group received osteopathic manipulation by a doctor of osteopathy (VA). Treatments lasted 15–25 min, and were gentle techniques such as myofascial release, balanced membranous tension, muscle energy, myofascial release, joint articulation, high-velocity, low-amplitude thrust and cranial-sacral therapy (Greenman, 2003; Magoun, 1976; Géhin, 2007; Winkel et al., 1997). Treatment was directed to the cervical and TMJ regions.

In particular, the specific manipulative procedures performed by the osteopath were designed both to reduce the dysfunction (pain and restriction) of the ligaments of the TMJ (stylo-mandibular and speno-mandibular ligaments, lateral collateral ligament) and to retrain the involuntary neuromuscular, reflexive control of posture and balance.



**Figure 1** Clinical measurement of maximal active mouth opening using calibrated caliper.

The CCT was provided by a gnathology specialist. Gnathology is the study of the masticatory system, including its physiology, functional disturbances and treatment.

The treatment included use of an oral appliance, physical therapy (gentle muscle stretching and relaxing exercises), therapies such as hot or cold packs (or both), transcutaneous electrical nerve stimulation.

Both groups could take a non-steroidal medication (anti-inflammatory medication and analgesics) and/or muscle relaxants, when prescribed by their medical practitioner.

The therapeutic protocol specified treatments at intervals of two weeks in both groups. At 24 (T1) and 32 (T2) weeks, the patients were assessed by an evaluator who was blinded to the treatment assignments.

The Ethics Committee of the University Palermo approved the protocol. Written informed consent was obtained from each subject after a full explanation of the experiment.

### Statistical method

Chi-square tests were used to compare the age and sex of OMT and CCT groups. Scores of TMI, FI, MI JI, and VAS, age and range of MOV and ROM (mm) were presented as the means  $\pm$  standard deviation (sd).

The *t* of Student was applied to compare the data between OMT patients and control group.

The two-way mixed analysis of variance (ANOVA) with the Tukey Post test was performed in order to verify whether the differences in the measurements of VAS, MOV, ROM and TMI at T0, T1, and T2 between OMT and CCT groups were statistically significant.

Data were analyzed using Primer of Biostatistics for Windows (version 4.02, McGraw-Hill Companies, New York) (Glantz, 2002). Significance for all statistical tests was set at  $P < 0.05$ .

### Results

The findings indicated that the OMT and CCT groups did not demonstrate any significant difference. The use of medication was greater in the CCT group than in the OMT group, with significant differences for non-steroidal anti-inflammatory drugs ( $\chi^2 = 4.083$ ,  $P < 0.001$ ) and muscle relaxants ( $\chi^2 = 4.878$ ,  $P < 0.001$ ). Non-steroidal medication was prescribed to 14 patients of the CCT group vs 6 patients of the OMT group. In addition, a muscle relaxant was prescribed in 8 patients in the CCT group and to 1 patient in the OMT group (Table 1). There were no differences in the mean pre-test values of VAS, MOV and ROM between OMT and CCT groups. When the two groups were compared at T1 and T2, the best results were obtained in the OMT group: only the VAS value at T2 was not statistically significantly different between two groups ( $3.80 \pm 1.26$  vs  $4.40 \pm 1.75$ ,  $P > 0.05$ ) (Table 2). Improvement in values of VAS, MOV and ROM in both groups was observed at T1 and at T2 than at T0. A statistically significant difference was observed in the OMT group between T1 values and T2 values for the VAS ( $1.5 \pm 0.85$  vs  $3.8 \pm 1.26$ ,  $F = 184.44$ ,  $P < 0.000$ ) and MOV ( $46 \pm 4.78$  vs  $42.9 \pm 2.69$ ,  $F = 48.19$ ,  $P < 0.000$ ), and in CCT group for the VAS ( $2.6 \pm 0.7$  vs  $4.4 \pm 1.75$ ,  $F = 48.66$ ,

**Table 1** The ratio and number of distribution for sex, age and patients who took medication.

Age y	OMT	CCT	<i>t</i>	<i>P</i>
Mean ± SD	40.6 ± 11.03	38.4 ± 15.33		NS
Range	30–63	29–62		NS
<b>Age group (years), <i>n</i> (%)</b>			<b>χ<sup>2</sup></b>	<b><i>P</i></b>
Women < 45	15 (60)	16 (64)		NS
Men ≥ 45	10 (40)	9 (36)		NS
<b>Sex</b>			<b>χ<sup>2</sup></b>	<b><i>P</i></b>
Women	17 (68)	15 (60)		NS
Men	8 (32)	10 (40)		NS
<b>Medications <i>n</i> (%)</b>			<b>χ<sup>2</sup></b>	<b><i>P</i></b>
Non-steroidal medication	6 (24)	14 (56)	4.083	0.001
Muscle relaxants	1(4)	8(28)	4.878	0.001
Total number of medications used	7 (28)	22 (88)	13.718	0.000

$P < 0.000$ ). These higher values at T2 indicate moderate worsening of symptoms and signs after 2 months (Table 3). Improvement in values of FI ( $F = 3.72$ ,  $P < 0.005$ ) and MI ( $F = 4.43$ ,  $P < 0.015$ ) was observed at T1 compared to T0 in OMT group (Table 4).

## Discussion

Osteopathic treatment is a form of manual medicine first applied by Still (1902). His principles and philosophy are based on an appreciation of human beings' triune unity (body, mind, and spirit), the interrelationship between structure and function, and the body's ability to heal itself (Ward et al., 2003). Still hypothesized that manipulative treatment stimulated the production of endogenous compounds that promoted homeostasis and healing. A study by Licciardone et al. (2005) indicated that OMT significantly reduces low back pain. The level of pain reduction was greater than expected from placebo effects alone and persisted for at least three months. OMT has been utilized not only in musculoskeletal disorders (e.g. low back pain,

fibromyalgia), but also in several pathologies such as recurrent acute otitis media, cerebral palsy, learning disorders, neurologic deficits, asthma, pneumonia, bronchiolitis, gastrointestinal disorders and headaches (Andersson et al., 1999; Mill et al., 2003; Duncan et al., 2004; Frymann, 1966, 1976; Degenhardt and Kuchera, 2006).

It is likely that the benefits of osteopathic interventions in these conditions could extend to other pain conditions such as TMD.

Results of this study suggest that reduction in pain and improved range of motion were reported after six months, suggesting that OMT and CCT provide relief for TMD related conditions. However, in the OMT group it was observed that the best values were for VAS, MOV and ROM at T1 and T2, and the reduction of FI and MI and the use of medications.

Even if at T2 there was a mild worsening of MOV, ROM and VAS than at T1, MOV and ROM values remained within the normal range of motion, and the reduction of VAS was noteworthy in that, and however there was an improvement at T2 when compared to T0.

Numerous mechanisms have been considered as sources of muscle and articular pain: local factors (microtrauma, local ischemia or hypoperfusion) can produce structural or functional consequences due to release of endogenous algescic substances (glutamate, histamine and others) from tissue cells and afferent nerve fibres leading to excitation or sensitization of nociceptors; central processes involving neuroendocrine factors (endogenous and exogenous hormones) as well as neurophysiological mechanisms (peripheral and central sensitization) also play a role in the pathophysiology of muscular pain (Sessle, 1999; Svensson and Graven-Nielsen, 2001).

Researchers suggest that massage and manipulation trigger a release of neuropeptides in patients and have studied the relationship between OMT and the endocannabinoid system (Christian et al., 1988). The endocannabinoid system, like the better-known endorphin system, consists of receptors in the brain, nervous system and elsewhere (cannabinoid receptors) and their endogenous ligands (endocannabinoids). McPartland et al. (2005) inferred that the endocannabinoid system may be elicited by OMT, with sedative, anxiolytic, analgesic and hemodynamic effects (McPartland et al., 2005).

There is also low evidence from a single case study that massage therapy and strain-counterstrain technique

**Table 2** Comparison of the VAS, MOV and ROM values between OMT and CCT groups ( $n = 25$ ) at T0, T1 and T2.

		OMT		CCT	<i>t</i>	<i>P</i>	
T0	VAS <sup>a</sup>	6.9	±0.88	6.40	±1.42	NS	
	MOV <sup>b</sup>	35.1	±4.36	34.9	±34.5	NS	
	ROM <sup>c</sup>	62.4	±10.67	64.5	±9.55	NS	
T1	VAS <sup>a</sup>	1.5	±0.85	2.6	±0.7	-4.995	0.000
	MOV <sup>b</sup>	46.0	±4.78	41.3	±4.52	3.572	0.000
	ROM <sup>c</sup>	81.9	±10.31	71.9	±9.05	3.654	0.000
T2	VAS <sup>a</sup>	3.8	±1.26	4.4	±1.75		NS
	MOV <sup>b</sup>	42.9	±2.69	40.4	±2.41	3.461	0.001
	ROM <sup>c</sup>	80.5	±5.44	72.4	±2.95	6.545	0.000

<sup>a</sup> The visual analogue pain scale was scored from 0 to 10.

<sup>b</sup> Measure in millimeters.

<sup>c</sup> Measure in degrees.

**Table 3** Average values and SD of the VAS, MOV and ROM values, ANOVA for repeated measures and Tukey Post test results.

Group	T0		T1		T2		F	P	Tukey Post test
	Mean	SD	Mean	SD	Mean	SD			
<i>OMT</i>									
VAS <sup>a</sup>	6.9	±0.88	1.5	±0.85	3.8	±1.26	184.88	0.000	TO vs T1, TO vs T2, T1 vs T2
MOV <sup>b</sup>	35.1	±4.36	46.0	±4.78	42.9	±2.69	48.19	0.000	TO vs T1, TO vs T2, T1 vs T2
ROM <sup>c</sup>	62.4	±10.67	81.9	±10.31	80.5	±5.44	35.53	0.000	TO vs T1, TO vs T2,
<i>CCT</i>									
VAS <sup>a</sup>	6.40	±1.42	2.6	±0.7	4.4	±1.75	48.66	0.000	TO vs T1, TO vs T2, T1 vs T2
MOV <sup>b</sup>	34.9	±34.5	41.3	±4.52	40.4	±2.41	23.6	0.000	TO vs T1, TO vs T2
ROM <sup>c</sup>	64.5	±9.55	71.9	±9.05	72.4	±2.95	8.07	0.000	TO vs T1, TO vs T2

<sup>a</sup> The visual analogue pain scale was scored from 0 to 10.

<sup>b</sup> Measure in millimeters.

<sup>c</sup> Measure in degrees. The mean values of VAS, MOV and ROM at T0, T1, and T2 in the OMT and CCT. For each parameter the Post test results are reported. If  $P < 0.05$ , difference between treatment at T0, T1 and T2 is statistically significant.

(positional release), stimulated parasympathetic activity, and reduced neuromuscular activity. These activities would engage the relaxation response and, in turn, reduce stress and anxiety associated with TMD (Eisensmith, 2007).

In the current study, the positive therapy effect on TMD may be explained by neural plasticity which could have been induced by these therapeutic interventions. Plasticity is a property of a self-organizing central nervous system (changes in network, synaptic or cell intrinsic properties) that is continually optimizing its own performance.

Therapies targeting the masticatory system (occlusal splints, physiotherapy, osteopathic manipulation and others) may have significant neurologic implication via sensorimotor integration with the brainstem, subcortical and cortical centers, cervical region, proprioception and body posture. If therapeutic approaches induce appropriate neural plasticity, then it is possible that considerable neurologic improvement of the patient may be achieved (Yin et al., 2007).

It is also possible that manual therapies may influence myofascial tone. Thus, increased or decreased myofascial

tone or diminished neuromuscular coordination may have been associated with improvement of TMD in this study (Schleip et al., 2005).

The favourable cost benefit ratio of physiotherapy over other treatment modalities seems to indicate that physiotherapy, in general, can be regarded as a first choice approach in selected TMD patients. OMT, in particular, had a positive effect on physical symptoms of TMD, and it is recommendable as an effective treatment in patients suffering from TMD.

Further studies need to be conducted to evaluate whether the findings are reproducible, and if positive long-term outcomes can be achieved. If the findings of this study are reinforced by future research, OMT would prove to be a non-invasive solution for managing TMD, either alone or together with other therapies and/or medication as part of an overall treatment plan.

In this regard, it would be desirable that in the management of disorders involving the TMJ and related musculoskeletal structures, dentist should work in close collaboration with osteopaths and physical therapists.

**Table 4** Average values and SD of the temporomandibular index and the associated subindex in OMT group and the CCT group, ANOVA for repeated measures and Tukey Post test results.

Group	T0		T1		T2		F	P	Tukey's test $P < 0.05$
	Mean	SD	Mean	SD	Mean	SD			
<i>OMT</i>									
Function index	0.45 (0.12)	0.34 (0.12)	0.39 (0.15)	0.45 (0.12)	0.34 (0.12)	0.39 (0.15)	3.72	0.005	TO vs T1
Muscle index	0.64 (0.23)	0.44 (0.27)	0.51 (0.22)	0.64 (0.23)	0.44 (0.27)	0.51 (0.22)	4.43	0.015	TO vs T1
Joint index	0.46 (0.28)	0.38 (0.24)	0.43 (0.17)	0.46 (0.28)	0.38 (0.24)	0.43 (0.17)			NS
Temporomandibular index	0.52 (0.21)	0.39 (0.21)	0.44 (0.18)	0.52 (0.21)	0.39 (0.21)	0.44 (0.18)			NS
<i>CCT</i>									
Function index	0.47 (0.23)	0.35 (0.14)	0.40 (0.17)	0.47 (0.23)	0.35 (0.14)	0.40 (0.17)			NS
Muscle index	0.62 (0.41)	0.45 (0.19)	0.52 (0.18)	0.62 (0.41)	0.45 (0.19)	0.52 (0.18)			NS
Joint index	0.47 (0.36)	0.41 (0.37)	0.44 (0.09)	0.47 (0.36)	0.41 (0.37)	0.44 (0.09)			NS
Temporomandibular index	0.52 (0.33)	0.40 (0.23)	0.45 (0.15)	0.52 (0.33)	0.40 (0.23)	0.45 (0.15)			NS

The mean values of FI, MI, JI and TMI at T0, T1, and T2 in the OMT and CCT. For each parameter the Post test results are reported. If  $P < 0.05$ , difference between treatment at T0, T1 and T2 is statistically significant.

## Acknowledgements

The authors would like to thank Patrizia Rovere Querini, M.D., Ph.D. San Raffaele Scientific Institute, for her contribution to the design and development of this study.

## Supplementary data

The supplementary data associated with this article can be found in the on-line version, at doi:[10.1016/j.jbmt.2009.08.02](https://doi.org/10.1016/j.jbmt.2009.08.02)

## References

- Alcantara, J., Plaughter, G., Klemp, D.D., Salem, C., 2002. Chiropractic care of a patient with temporomandibular disorder and atlas subluxation. *J. Manipulative Physiol. Ther.* 25, 63–70.
- Andersson, G.B.J., Lucente, T., Davis, A.M., Kappler, R.E., Lipton, J.A., Leurgans, S., 1999. A comparison of osteopathic spinal manipulation with standard care for patients with low back pain. *N. Engl. J. Med.* 341, 1426–1431.
- Buescher, J.J., 2007. Temporomandibular joint disorders. *Am. Fam. Physician* 15 (76), 1477–1482.
- Carlson, C.R., Bertrand, P.M., Ehrlich, A.D., Maxwell, A.W., Burton, R.G., 2001. Physical self-regulation training for the management of temporomandibular disorders. *J. Orofac. Pain* 15, 47–55.
- Cascos-Romero, J., Vázquez-Delgado, E., Vázquez-Rodríguez, E., Gay-Escoda, C., 2009. The use of tricyclic antidepressants in the treatment of temporomandibular joint disorders: systematic review of the literature of the last 20 years. *Med. Oral Patol. Oral Cir. Bucal.* 14, E3–E7.
- Christian, G.F., Stanton, G.J., Sissons, D., How, H.Y., Jamison, J., Alder, B., Fullerton, M., Funder, J.W., 1988. Immunoreactive ACTH,  $\beta$ -endorphin, and cortisol levels in plasma following spinal manipulative therapy. *Spine* 13, 1411–1417.
- De Bont, L.G., Dijkgraaf, L.C., Stegenga, B., 1997. Epidemiology and natural progression of articular temporomandibular disorders. *Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod.* 83, 72–76.
- DeBar, L.L., Vuckovic, N., Schneider, J., Ritenbaugh, C., 2003. Use of complementary and alternative medicine for temporomandibular disorders. *J. Orofac. Pain* 17, 224–236.
- Degenhardt, B.F., Kuchera, M.L., 2006. Osteopathic evaluation and manipulative treatment in reducing the morbidity of otitis media: a pilot study. *J. Am. Osteopath. Assoc.* 106, 327–334.
- Duncan, B., Barton, L., Edmonds, D., Blashill, B.M., 2004. Parental perceptions of the therapeutic effect from osteopathic manipulation or acupuncture in children with spastic cerebral palsy. *Clin. Pediatr.* 43, 349–353.
- Eisensmith, L.P., 2007. Massage therapy decreases frequency and intensity of symptoms related to temporomandibular joint syndrome in one case study. *J. Bodyw. Mov. Ther.* 11, 223–230.
- Frymann, V.M., 1966. Relation of disturbances of craniosacral mechanisms to symptomatology of the newborn: study of 1250 infants. *J. Am. Osteopath. Assoc.* 65, 1059–1075.
- Frymann, V.M., 1976. Learning difficulties of children viewed in the light of the osteopathic concept. *J. Am. Osteopath. Assoc.* 76, 46–61.
- Géhin, A., 2007. *Cranial Osteopathic Biomechanics, Pathomechanics and Diagnostics for Practitioners*. Churchill Livingstone Elsevier, Philadelphia.
- Glantz, S.A., 2002. *Primer of Biostatistics*, fifth ed. McGraw-Hill Companies, New York.
- Greenman, P.E., 2003. *Principles of Manual Medicine*, third ed. Lippincott Williams & Wilkins, Philadelphia.
- Haskin, C.L., Milam, S.B., Cameron, I.L., 1995. Pathogenesis of degenerative joint disease in the human temporomandibular joint. *Crit. Rev. Oral. Biol. Med.* 6, 248–277.
- Huskisson, E.C., 1974. Measurement of pain. *Lancet* 2, 1127–1131.
- Larsen, N.J., 1976. Osteopathic manipulative contribution to treatment of TMJ syndrome. *Int. J. Osteopath. Med.* 3, 15–27.
- Levy, B.M., Gorlin, R.J., 1953. The temporomandibular joint in vitamin C deficiency. *J. Dent. Res.* 32, 622–625.
- Licciardone, J.C., Brimhall, A.K., King, L.N., 2005. Osteopathic manipulative treatment for low back pain: a systematic review and meta-analysis of randomized controlled trials. *BMC Musculoskelet. Disord.* 6, 43.
- Magoun, H.I., 1976. *Osteopathy in the Cranial Field*, third ed. Northwest Printing Inc, Boise.
- McNeely, M.L., Armijo Olivo, S., Magee, D.J., 2006. Therapy interventions for temporomandibular disorders. *Phys. Ther.* 86, 710–725.
- McPartland, J.M., Giuffrida, A., King, J., Skinner, E., Scotter, J., Musty, R.E., 2005. Cannabimimetic effects of osteopathic manipulative treatment. *J. Am. Osteopath. Assoc.* 105, 283–291.
- Mill, M.V., Henley, C.E., Barnes, L.L.B., Carreiro, J.E., Degenhardt, B.F., 2003. The use of osteopathic manipulative treatment as adjuvant therapy in children with recurrent acute otitis media. *Arch. Pediatr. Adolesc. Med.* 157, 861–866.
- Monaco, A., Cozzolino, V., Cattaneo, R., Cutilli, T., Spadaro, 2008. Osteopathic manipulative treatment (OMT) effects on mandibular kinetics: kinesiographic study. *Eur. J. Paediatr. Dent.* 9, 37–42.
- Neiva, P.D., Kirkwood, R.N., 2007. Measurement of neck range of motion among mouth-breathing children. *Rev. Bras. Fisioter.* 11, 355–360.
- Pehling, J., Schiffman, E., Look, J., Shaefer, J., Lenton, P., Friction, J., 2002. Interexaminer reliability and clinical validity of the temporomandibular index: a new outcome measure for temporomandibular disorders. *J. Orofac. Pain* 16, 296–304.
- Royder, J.O., 1981. Structural influences in temporomandibular joint pain and dysfunction. *J. Am. Osteopath. Assoc.* 80, 460–467.
- Schleip, R., Klingler, W., Lehmann-Horn, F., 2005. Active fascial contractility: fascia may be able to contract in a smooth muscle-like manner and thereby influence musculoskeletal dynamics. *Med. Hypotheses* 65, 273–277.
- Sessle, B.J., 1999. The neural basis of temporomandibular joint and masticatory muscle pain. *J. Orofac. Pain* 13, 238–245.
- Still, A.T., 1902. *The Philosophy and Mechanical Principles of Osteopathy*. Hudson-Kimberly Publication Co., Kansas City, MO.
- Sturdivant, J., Friction, J.R., 1991. Physical therapy for temporomandibular disorders and orofacial pain. *Curr. Opin. Dent.* 32, 485–496.
- Svensson, P., Graven-Nielsen, T., 2001. Craniofacial muscle pain: review of mechanisms and clinical manifestations. *J. Orofac. Pain* 15, 117–145.
- Upledger, J.E., 1987. *Craniosacral Therapy II: Beyond the Dura*. Eastman Press, Seattle.
- Ward, R.C., et al. (Eds.), 2003. *Foundations for Osteopathic Medicine*, second ed. Lippincott Williams & Wilkins, Philadelphia.
- Winkel, D., Matthijs, O., Phelps, V., 1997. *Diagnosis and Treatment of the Spine*. Aspen Publishers, Gaithersburg.
- Yin, C.S., Lee, Y.J., Lee, Y.J., 2007. Neurological influences of the temporomandibular joint. *J. Bodyw. Mov. Ther.* 11, 285–294.