Osteopathic manipulative treatment (OMT) effects on mandibular kinetics: kinesiographic study

A. MONACO*, V. COZZOLINO*, R. CATTANEO*,

T. CUTILLI**, A. SPADARO*

ABSTRACT. ***Aim*** *The aim of this study was to evaluate the effects of Osteopathic Manipulative Treatment (OMT) on mandibular kinematics in TMD patients.* ***Methods*** *The study was conduced on 28 children with non-specific TMD symptoms, limited mouth opening, history of trauma (delivery trauma, accident trauma). Patients were randomly divided into two groups: an OMT group (study group) and a no-intervention group (control group). All subjects underwent a first kinesiographic recording to evaluate the amplitude and velocity of maximal opening-closing movements. Study group patients underwent a second kinesiographic recording 2 months after OMT. Control group patients were submitted to a control kinesiographic recording six months after the first one. Kinesiographic tracings were acquired using the K7I system.* ***Results/Statistic*** *The kinesiographic data of the study group showed a moderate statistically significant difference (p<.07) of maximal mouth opening (MO) parameter and a high statistically significant difference (p<.03) of maximal mouth opening velocity (MOV) parameter. No statistically significative difference (null hypothesis confirmed) of kinesiographic parameters in the control group was observed.* ***Conclusion*** *The results of this study suggest that OMT can induce changes in the stomatognathic dynamics, offering a valid support in the clinical approach to TMD. Multifactorial genesis of chronic disorders is also confirmed.*

KEYWORDS: *Jaw movements; TMD; Kinesiography; Osteopathy; Sympathetic Nervous System.*

Introduction

Temporomandibular disorders (TMD) encompass a group of muscoloskeletal conditions that involve the temporomandibular joint (TMJ) or the masticatory musculature, or both. These conditions are typically characterised by pain at the preauricolar and other facial areas, usually aggravated by chewing or jaw function. TMD are often accompanied, singly or in combination, by limitation of jaw movement, joint sounds, muscular and fascial tenderness or joint soreness [Mohl et al., 1990].

** Department of Gnatology and Prosthetic Dentistry. School of Dentistry*
****Department of Maxillofacial Surgery. School of Dentistry*
University of L'Aquila-Italy 23-10-2007
e-mail: annalisamonaco@yahoo.it

Mandibular movement patterns have been commonly used by clinicians to investigate dysfunction of the masticatory system. Restricted maximal opening (normal values range of 45+/- 5mm) and deviations or deflections in the opening path have been reported as the third sign of the classic triad representing TMD [Dworkin and Le Resche, 1992]; the rate of freedom of jaw movements is also expression of musculoskeletal and fascial health [Greenman, 1998; Mitchell, 2000; Korr, 1995; Magoun, 1966].

Measurement techniques have included simple measurement devices, such as a millimetre ruler, to sophisticated electronic devices to record movements of the mandible using magnets or photodiode sensors. The Mandibular Kinesiograph (MKG) is an instrument designed for research and diagnosis of mandibular function/dysfunction. It electronically records mandibular incisor-point movements in three dimensions; measurement of vertical velocity is also provided by differentiating the vertical position signal [Jankelson, 1980].

TMD, as chronic disorder, appears to be multifactorial, potentially involving a complex interplay between anatomic structure, biomechanical function, environmental demands, and psychosocial responses, each capable of contributing to clinical manifestations and symptoms [Liebenson, 1992].

Some authors demonstrated a relationship between stomatognathic and postural systems.

Clark showed co-activation of sternomastoid and masseter muscles [Clark et al., 1993]. Trigeminal electrical and mechanical stimulation elicited sternomastoid inhibition showing functional coupling between mandible and neck-trunk system.

Ehrlich supported Clark's data stating that sternomastoid, trapezius, paravertebral and rectus abdominis muscles showed a significant increase in clenching SEMG compared to resting SEMG activity. [Ehrlich et al., 1999].

Solow found in 96 children a clear pattern of associations between head and neck posture and malocclusion, suggesting that sagittal development of dentoalveolar arches is impeded by increased dorsally-directed soft tissue pressure in individuals with extended craniocervical posture [Solow and Sonnesen, 1998].

On the other side head position is an important factor in determining the amount of vertical mandibular opening in healthy adults. Higbie stated that vertical mandibular opening ranged from 44 mm to 36.2 mm changing from forward to retracted head position [Higbie et al., 1999].

Body position affects jaw posture influencing swallowing function [Miralles et al., 2006; Ertekin et al., 2001].

Yamashita measured the vibration wave propagation in the body at teeth contact [Yamashita et al., 1998]. They found that the impact on teeth propagated to distal sites of the limbs through bones and soft tissues, influencing the whole body.

According to Gillies it's possible to consider a biomechanical model in which head and neck constitute an inverted pendulum stabilised by neuromuscular restoring forces [Gillies et al., 1998]. This model predicts that temporomandibular alterations could lead to perturbations of normal forces acting in head and neck. Authors concluded warning that the altered relationship could be expected to contribute to additional or accelerated degenerative effects on temporomandibular or postural system.

Osteopathic manipulative medicine (OMM) approach asserts that with sufficient diagnostic skills, osteopathic physicians are able to identify the exact anatomic region responsible for the pain or movement restriction [Kuchera, 2005].

Movement restriction in a local muscular-skeletal district could be solved treating the responsible primary anatomic region. Affected district and responsible anatomical region aren't necessarily the same. In conclusion same symptoms may require different treatments plans that focus on differing local, spinal, and supraspinal targets [Kuchera, 2005].

Findings demonstrated a relationship between stomatognathic and postural systems justifying the hypothesis that the musculoskeletal impairment in one system could affect the other.

The objective of our study is to analyse the possible relation between osteopathic manipulative treatment (OMT) and mandible kinematics in order to confirm the relationship between stomatognathic and postural systems.

Materials and methods

The study was performed at the Dental Centre of the University of L'Aquila; 28 TMD children (age average=12 years), selected among the patients referred to the Paediatric Dentistry Department, were submitted for the study. All patients presented: non-specific TMD symptoms, limited mouth opening, history of trauma (delivery trauma, accident trauma). The subjects were randomly assigned to the OMT group (study group) or the no-intervention group (control group), both composed of 14 subjects. Study group subjects were submitted to two kinesiographic (KNS) recordings: one at the first visit (T0) and a second one at two months from the end of OMT (T1). Control group subjects were also submitted to the same kinesiographic recordings: the first one at the first visit (T0) and the second one at six months (T1).

Kinesiographic tracings were acquired using K7I and positioning a magnetic sensor frame integral with the head and with the sensory field balanced on an artificial magnet fixed on the mucosa covering the roots of the mandibular incisors. Mandibular movement, changing the relative position between the magnet and the sensor, varied at magnetic field modifications, inducing the software tool to trace a line which connected the initial and final position.

During the recordings the patient was seated in a wooden chair with headrest in a comfortable stance and closed eyes to avoid enviromental stimulations.

Each kinesiographic trial provided two KNS recordings.

In the first recording (Scan1) the patient, starting from the teeth/contact position, perfomed 3 subsequent maximal opening/closing jaw movements, to obtain Maximal Opening (MO) parameter (KNS values expressed in mm for 10).

In the second recording (Scan2) the patient, starting from the teeth/contact position, performed 15 subsequent opening/closing jaw movements, as fast as possible, to obtain Maximal Opening Velocity (MOV), Maximal Closing Velocity (MCV), Opening Velocity Average (OVA) and Closing Velocity Average (CVA) parameters (KNS values expressed in mm for s-1). The opening movements were performed until the pain was reached. The timeframe between recording was set at 20 seconds. Patient was accepted if able to obtain four identical kinesiographic pattern; all patients complied with the requirement.

KNSs were examined by the same operator without knowledge of recording purpose.

Statistical analysis. A paired or impaired t-test as appropriate was performed using Stata statistics Package to obtain a comparison between mean and variance values of kinesiographic data between dependent or independent groups.

Study group to control group were compared at T0 (before Osteopathic Manipulative Treatment) in order to evaluate statistical differences in kinesiographic values.

In null hypothesis at T0 control and study group show no statistically significant differences. In this case control and study groups have similar values, therefore it is possible to evaluate T1 data (two months after Osteopathic Manipulative Treatment, T1) to assess by kinesiographic data the therapeutic effect of OMT.

In alternative hypothesis, at T0 study and control group show statistically significant differences. In this case an error in statistical sampling is possible: the two groups are not comparable.

Afterwards each group was compared by mean and variance at T0 and T1.

In null hypothesis kinesiographic values at T0 and T1 are not statistically significant showing no difference.

In alternative hypothesis T0 and T1 values show significant difference due to time for control group or due to osteopathic treatment in study group. Significant differences in the control group would invalidate the results obtained for the study group.

Null hypothesis in control group and alternative hypothesis in study group could suggest a positive treatment effect on kinesiographic data.

Differences with a value of $p < .05$ and $< .07$ were respectively regarded as significant and moderately significant.

A paired t-test for dependent samples was performed to obtain a comparison between mean and variance values of kinesiographic data.

Results

Table 1 shows mean values and standard deviation (in parenthesis) of kinesiographic data of study and control group at T0. No significant differences were found in all parameters evaluated. In this case the null hypothesis was confirmed.

Table 2 shows mean values and standard deviation (in parenthesis) of kinesiographic data of study group

Parameter (m.u.)	Condition	Mean (S.D.)	
MO (mm x 10)	CG	375,1	(54,3)
	SG	380,7	(84,4)
	Diff.	NS	
MOV (mm x s-1)	CG	398,5	(84,1)
	SG	261,6	(118,3)
	Diff.	NS	
MCV (mm x s-1)	CG	416,7	(101,3)
	SG	310,0	(110,4)
	Diff.	NS	
OVA (mm x s-1)	CG	236,3	(56,6)
	SG	154,7	(74,9)
	Diff.	NS	
CVA (mm x s-1)	CG	266,8	(69,3)
	SG	178,1	(84,2)
	Diff.	NS	

MO: Maximal Opening; MOV: Maximal Opening Velocity; MCV: Maximal Closing Velocity; OVA: Opening Velocity Average; CVA: Closing Velocity Average.
** Moderately statistically significant*
*** Highly statistically significant*
NS: Not significant

TABLE 1 - *Mean values and Standard Deviation (in parenthesis) of Study Group (14 subjects) and control Group (14 subjects) at T0.*

Parameter (m.u.)	Condition	Mean (S.D.)	
MO (mm x 10)	T0	380,7	(84,4)
	T1	426,3	(28,2)
	Diff.	.07	*
MOV (mm x s-1)	T0	261,6	(118,3)
	T1	316,4	(93,1)
	Diff.	.03	**
MCV (mm x s-1)	T0	310,0	(110,4)
	T1	330,0	(82,3)
	Diff.	NS	
OVA (mm x s-1)	T0	154,7	(74,9)
	T1	175,3	(49,0)
	Diff.	NS	
CVA (mm x s-1)	T0	178,1	(84,2)
	T1	195,0	(62,4)
	Diff.	NS	

MO: Maximal Opening; MOV: Maximal Opening Velocity
MCV: Maximal Closing Velocity; OVA: Opening Velocity Average
CVA: Closing Velocity Average
** Moderately statistically significant*
*** Highly statistically significant*
NS: Not significant

TABLE 2 - *Mean values and Standard Deviation (in parenthesis) of Study Group (n=14) before Osteopathic Manipulative Treatment (T0) and two months after Osteopathic Manipulative Treatment (T1).*

Parameter (m.u.)	Condition	Mean (S.D.)	
MO (mm x 10)	T0	375,1	(54,3)
	T1	374,0	(57,8)
	Diff.	NS	
MOV (mm x s-1)	T0	398,5	(84,1)
	T1	399,9	(91,2)
	Diff.	NS	
MCV (mm x s-1)	T0	416,7	(101,3)
	T1	409,3	(109,1)
	Diff.	NS	
OVA (mm x s-1)	T0	236,3	(56,6)
	T1	243,6	(47,4)
	Diff.	NS	
CVA (mm x s-1)	T0	266,8	(69,3)
	T1	260,8	(58,7)
	Diff.	NS	

MO: Maximal Opening; MOV: Maximal Opening Velocity
MCV: Maximal Closing Velocity; OVA: Opening Velocity Average
CVA: Closing Velocity Average
** Moderately statistically significant*
*** Highly statistically significant*
NS: Not significant

TABLE 3 - *Mean values and Standard Deviation (in parenthesis) of Control Group (n=14) at first visit (T0) and six months after (T1).*

at T0 and T1. A statistically significant difference was observed for MOV (p< .03); a moderate statistically significant difference was observed for MO (p<. 07). No statistical difference (null hypothesis confirmed) for the other KNS parameters was observed.

Table 3 shows mean values and standard deviation (in parenthesis) of kinesiographic data of control group at T0 and T1. No statistical difference for all the KNS parameters was observed.

Discussion

In our study patients who received osteopathic manipulative treatment of the extra-stomatognathic area showed low significant increase in vertical mandible maximal opening (MO) and high significant increase in maximal opening velocity (MOV).

Control group did not show significant changes in mandibular kinetics.

Osteopathic manipulative medicine (OMM) is a component of osteopathic medicine's approach to total patient care. It emphasises application of osteopathic philosophy and integrates recognised healing approaches known as osteopathic manipulative treatment (OMT).

Osteopathic philosophy is based on three key principles [Seffinger et al., 2003]:

1) the body is a unit;
2) the body possess self-regulatory mechanism;
3) structure and function are reciprocally interrelated.

Irvin demonstrated that chronic complaints throughout the body could be attributed to an unlevelled sacral base and that establishing postural homeostasis it was possible to remove most of these symptoms [Irvin, 1997].

Direct and indirect sympathetic control could affect some musculoskeletal symptoms, including restricted range of active and passive movement or pain.

Various coupling and regulating mechanisms have been proposed to explain the homeostatic influence on physiologic processes responsible for maintaining restricted range of movement and pain [Sterling and Eyer, 1988; Seeman et al., 1997; McEwen, 1998]. Homeostasis may be altered through simpathetic, biochemical or neuroendocrine mechanisms affecting specific structures or target receptors, or both.

Modulation of sympathetic tonus, enhancing healing rates, has been linked to improvement of visceral and, in the light of our study, somatic functions [Korr, 1995].

As claimed by osteopathic literature, osteopathic lesion responsible of movement restriction, is referred to impairment of sympathetic transmission. According to this hypothesis manipulative treatment enhancing balance in sympathetic nervous system could improve movement restriction.

Somatomotor system and sympathetic nervous system (SNS) are intimately correlated. SNS supplies motor performance by modifying vegetative function parameters to meet the varying metabolic requirements of the active muscle [Passatore and Roatta, 2007; Thomas and Segal, 2004]. Increase in SNS outflow affects motor function through actions exerted at the muscle level.

Our data on increase of MO and MOV agree with those of Passatore [Passatore and Roatta, 2007], who stated that sympathetic nervous system controls both muscle blood flow and intracellular contractile mechanism and may affect motor function by modulating afferent activity from muscle spindles that are highly concentrated in jaw-closing muscles.

Recent immunohystochemical data on masseter muscle confirmed the presence of non vascular sympathetic innervation on muscle spindles in close association with intrafusal muscle fibres. These data suggest that the sympathetic nervous system could modulate the spindle afferent discharge by altering intrafusal fibre mechanics [Bombardi et al., 2006].

Increase in SNS outflow may act by:

- decreasing muscle blood perfusion, which is an inseparable factor of muscle pain;
- enhancing contractile force in fast-contracting muscle, while exerting a fatiguing action on slow-contracting ones;
- reducing the quality of proprioceptive information.

The latter is likely to worsen different aspects of motor control increasing co-contraction of antagonist muscles aimed at recovering movement precision by increasing joint stiffness. This effect has been studied in *in vitro* and *in vivo* and seems to be particularly powerful in jaw closing muscles [Grassi et al., 1993].

Our data on MOV could be explained with the findings of a study about physiology of jaw muscles.

Koolstra demonstrated in open jaw movement that passive forces produced by jaw-closing muscles were remarkably stronger than those produced by the jaw-opening muscles in close jaw movement [Koolstra and Van Eijden, 1997].

The mentioned findings could explain the changes observed in the dynamic of mandibular jaw opening-closing movements after OMT in our study.

Osteopathic treatment, by reducing hypersympathicotonia as a consequence of general postural stress, could improve the range of freedom of jaw muscle activity and viscoelasticity (increase of MO values), particularly reducing the jaw closing passive forces that seem to have a great influence on the jaw-opening dynamics (the highly significant statistical improvement of MOV can only be explained as an expression of this physiological relation).

The results of this study suggest that OMT can offer a valid support in the clinical approach to TMD, confirming the multifactorial genesis of these chronic disorders.

Conclusions

This preliminary study compared OMT effects on two groups of subject affected by TMD: study group and no-intervention group. The study group showed a significant improvement of maximal mouth opening and maximal mouth opening velocity compared with no- intervention group.

Our preliminary findings also indicated that manipulative treatment of non stomathognatic areas was related to changes in the KNG activity of the mandible. Future investigation will be aimed at evaluating these variables in a follow-up study, in order to clarify the stability of results and the pathogenesis of some TMDs signs and symptoms.

References

Bombardi C, Grandis A, Chiocchetti R, Bertolani R, Johansson H, Lucchi ML. Immunohistochemical localization of alpha 1a-adrenoceptors in muscle spindles of rabbit masseter muscle. Tissue Cell 2006;38:121-5.

Clark GT, Browne PA, Nakano M, Yang Q. Co-activation of sternocleidomastoid muscles durin maximum clenching. J Dent Res 1993 Nov;72(11):1499-502.

Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomand Disord Facial Oral Pain 1992;6:301-55

Ehrlich R, Garlick D, Ninio M. The effect of jaw clenching on the electromyographic activity of 2 neck and trunk muscles. J Orofac Pain 1999 Spring;13(2):115-20.

Ertekin C, Keskin A, Kijlioglu N, Kirazli Y, Yagiz On A, Tarlaci S, et al. The effect of head and neck position on oropharyngeal swallowing: a clinical and electrophysiologic study. Arch Phys Med Rehabil 2001;Sept 82:1255-60.

Gillies GT, Broaddus WC, Stenger JM, Taylor AG. A biomechanical model of craniomandibular complex and cervical spine based on the inverted pendulum. J Med Eng Technol 1998 Nov-Dec;22(6):263-9.

Grassi C, Deniu F, Artusio E, Passatore M. Modulation of the jaw jerk reflex by the sympathetic nervous system. Arch Ital Biol 1993;131:213-26.

Greenman P: Principles of manual medicine, Baltimore , Md: Williams & Wilkins; 1998. p. 39-44.

Higbie EJ, Seidel-Cobb D, Taylor LF, Cummings GS. J Orthop Sports Phys Ther 1999 Feb;29(2):127-30.

Irvin RE. Suboptimal posture: the origin of the majority of idiopathic pain of the musculoskeletal system. In: Vleeming A, Mooney V, Snijders C, Stoeckert R eds. Movement, Stability and Low Back Pain. The Essential Role of the Pelvis. New York, NY: Chuechill Livingstone; 1997. p. 133-155.

Jankelson B: Measurement accuracy of the mandibular kinesiograph-a computerized study. J Prosthet Dent 1980;44(6):656-665.

Koolstra JH, Van Eijden TMGJ. Dynamics of the human masticatory muscles during a jaw open-close movement. J Biomechanic 1997;30(9):883-9.

Korr IM. The collected papers of I.M. Korr. Indianapolis: American Academy of Osteopathy; 1995. p. 119-228.

Kuchera ML. Osteopathic manipulative medicine considerations in patients with chronic pain. JAOA 2005; 105(9): 529-536.

Liebenson CS. Pathogenesis of chronic back pain. J Manipulative Physiol Ther 1992;15:299-308.

Magoun HI. Osteopathy in the cranial field, Northwest Printing, Inc. Boys. Id; 1966. p. 267-306.

McEwen BS. Protective and damaging effects of stress mediators. N Engl J Med 1998;338:178-9.

Miralles R, Gutierrez C, Zucchino G, Cavada G, Carvajal R, Valenzuela, et al. Body position and jaw posture effects on supra- and infrahyoid electromiographic activity in humans. J Craniomandib Pract 2006 April;24(2):98-103.

Mitchell FL Jr. The muscle energy manual. Vol.2. Michigan: Met/Press East Cancing; 2000. p. 1-14.

Mohl ND, McCall WD, Lund JP, Plesh O. Devices for the diagnosis and treatment of temporomandibular disorders. Part I: Introduction, scientific evidence, and jaw tracking. J Prosthet Dent 1990; 63:198-201.

Passatore M, Roatta S. Modulation operated by the Sympathetic Nervous System on jaw reflexes and masticatory movement. Arch Oral Biol 2007;52:343-6.

Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation-allostatic load and its health consequences: McArthur studies of successful aging. J Clin Endocrinol Metab 1997;157:2259-2268.

Seffinger MA, King HH, Ward RC, Jones JM, Rogers FJ, Patterson MM. Osteopathic philosophy. In: Ward RC ed. Foundations for Osteopathic Medicine. 2nd ed. Philadelphia: Lippincot Williams & Wilkins; 2003. p. 3-18.

Solow B, Sonnesen L. Head posture and malocclusions. Eur J Orthod 1998 Dec;20(6):685-93.

Sterling P, Eyer J. Allostassis: a new paradigm to explain arousal pathology. In: Fisher S, Reason J eds. Handbook of of Life Stress, Cognition and Health. New York: John Wiley & Sons; 1988. p. 629-649.

Thomas GD, Segal SS. Neural control of muscle blood flow during exercise. Appl Pysiol 2004;98:731-8.

Yamaschita R, Suenaga H, Yamabe Y, Torisu T, Fujii H. Propagation of various tooth impacts in human body. J Oral Rehabil 1998 Oct;25(10):785-91.