Binocular motility system and temporomandibular joint internal derangement: A study in adults

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Introduction: Ocular convergence defects have been confirmed in adults with temporomandibular joint (TMJ) dysfunction, but few studies of the relationship of the oculomotor apparatus to TMJ disc displacement have been reported. The purpose of this study was to examine the impact of disc displacement on the oculomotor capacity of the eyes in adults. **Methods:** Fifty symptomatic patients with bilateral TMJ disc displacement (13 men, 37 women; mean age, 28.84 ± 8.22 years; range, 18-40 years) were compared with the same number of asymptomatic volunteers with normal disc position (14 men, 36 women; mean age, 29.96 ± 5.04 years; range, 18-37 years). All subjects underwent standardized clinical examinations, bilateral TMJ magnetic resonance imaging, and sensorial and motor orthoptic tests by an orthoptist. **Results:** Subjects with TMJ disc displacement had alterations in binocular function, including reductions in convergence ($P < 0.023$) and positive fusional vergence (break point [$P < 0.046$] and recovery point [$P < 0.045$]) compared with those with normal disc positions. **Conclusions:** Significant alternations in binocular function were seen in patients with TMJ disc displacement compared with healthy control subjects. (Am J Orthod Dentofacial Orthop 2008;133:640.e15-640.e20)

Temporomandibular joint disorder (TMD) is a collective term for a condition that can include clinical problems involving the masticatory muscles (MM), the temporomandibular joint (TMJ) and its associated structures, or both.[1] Internal derangement (ID) of the TMJ is the most common type of TMD, characterized by an abnormal relationship of the articular disc to the mandibular condyle, the fossa, and the articular eminence with progressive disc displacement (DD). This condition can lead to TMJ clicking, crepitus, and, in some cases, pain and limitation of jaw movement.[2] In DD with reduction (DDR), the disc is reduced on mouth opening; in DD without reduction (DDNR), there is no reduction of the anteriorly displaced disc on mouth opening.

In addition to anatomical evidence of connections between the oculomotor apparatus and the trigeminal system shown in several studies,[3-9] some researchers have examined the clinical relationship between dental occlusion and the oculomotor system,[10,11] and showed that there might be an association between TMD and oculomotor function.[12,13] Ocular convergence defects have been found in adults with TMD, limited maximal opening, myofascial pain, and pain in the neck and shoulders area.[12]

Our aim in this case-control study was to study the impact of DD on the oculomotor capacity of the eyes in adults.

MATERIAL AND METHODS

The exclusion criteria for this study were systemic disease, vestibular or equilibrium problems, hearing abnormalities, clinical signs of oculomotor system dysfunction, and other ophthalmic symptoms such as orbital pain, asthenopia, or anisocoria. Subjects with missing posterior teeth, except for the third molars, were excluded.

Fifty symptomatic patients (13 men, 37 women; average age, 28.84 ± 8.22 years; range, 18-40 years) with bilateral DD, determined by magnetic resonance imaging (MRI) (27 DDR patients and 23 DDNR), were selected from a pool of 126 consecutive patients with mandibular dysfunction who were referred to the Department of Orthodontics and Gnathology, University of Palermo in Italy. Subjects were included if they had a temporomandibular index (TMI) reference value of ≥0.08 ± 0.10. The TMI is a clinical measure used to determine the severity of the disorder. It includes 3 subindexes: function index (FI), muscle index (MI), and joint index (JI). The scores of all indexes ranged from 0 to 1.[14] Twenty-three (46%) of the subjects wore glasses. Of the 126 patients screened, 76 were excluded because they were over 40 years old (n = 16) or did not have bilateral DD (n = 60).

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One hundred one asymptomatic volunteers, matched for age and sex, and with scores on the TMI within the reference values for asymptomatic subjects (0.08 ± 0.10),[14] were examined. Each subject signed a consent form permitting the investigation and had a standardized TMJ clinical examination and a bilateral MRI. Joint pain, crepitation, and uncoordinated movements of the head of each mandibular condyle during opening or closing of the mouth were investigated by lateral and posterior palpation of each TMJ with the index fingers. In addition, the range of mandibular vertical and horizontal movement was assessed. Subjects older than 40 years (n = 10) and with any of the following signs and symptoms (n = 41) were excused: localized pain in the TMJ or ear, pain on mandibular movement, headaches aggravated by jaw movement, joint sounds, limited mandibular range of vertical opening (<40 mm) and horizontal (<5 mm) movements, deviation on mandibular opening (>2 mm), history of locking, trauma, and TMD treatment.[15] Fifty control subjects (14 men, 36 women; average age, 29.96 ± 5.04 years; range, 18-37 years) without ID, as determined by MRI, were enrolled. Twenty-two (44%) subjects wore glasses.

Each person in this study received a bilateral, high resolution, surface-coil MRI with a 1.5-T magnet (Magnetom 63; Siemens, Erlangen, Germany). TMJ disc positions were obtained from consecutive sagittal MRI slices for both opened and closed mouth positions (produced from 3-mm-thick volume slices, 4-6 slices per joint), with the procedure described by Nebbe et al.[16] A radiologist experienced with MRIs of the TMJ interpreted the images.

The same orthoptist did sensorial and motor orthoptic tests and was blinded to the clinical characteristics of the ID patients and healthy subjects. The following tests were done: Maddox, alternating cover, ocular motility, convergence, and near positive fusional vergence.

The Maddox test subjectively estimates heterophorias (axial defects). The subject was asked to observe a light spot, set up at 33 cm with a high-power lens (500 diopters, focal 2 mm, Maddox lens) in front of the right eye. The lens presents particular cylinders and transforms a punctiform image into a linear image, orthogonal to the cylinders. We estimated the imbalances on the horizontal plane, having placed the cylinders of the lens horizontally (the image of light spot appears vertical). A Berens prism was placed in front of the left eye. The right eye saw a red light streak, and the left eye saw the light spot. The subject was asked to report the position of the red light streak relative to the light spot. A crossed position indicated exophoria, and an uncrossed position indicated esophoria. Base-in and base-out prisms were used, respectively, to measure the exophoria and esophoria in prism diopters (Δ).[17]

The alternating cover test is an objective test for heterophoria. The directions of movements (esophoria, exophoria, and orthophoria) were recorded.[18]

The test of ocular motility estimates the excursion of the eye by the analysis of the boundaries of sight (cardinal points). Normally, such movements are symmetric, simultaneous, and regular; otherwise, ocular motility is considered pathologic.[19]

The convergence test was used as a main method of diagnosing convergence insufficiency and estimating the equilibrium of the extrinsic musculature of the eye. The operator moves a stick toward the subject's nose at the height of the eyes. Normally, the convergence of the eyes is symmetrical and simultaneous. Convergence is measured with a ruler supported at the temporal orbital margin, allowing an estimate of the distance by which the 2 eyes diverged; 3 to 4 cm was considered normal, 5 to 7 cm was sufficient, and more than 7 was insufficient.[18]

A test for near positive fusional vergence was used to estimate the positive fusional vergence/reserve of the eyes. The patient observed a card (letters of Snellen) set up at 40 cm. A base-out prism bar was placed in front of the subject, increasing the demand until diplopia was reached. That was the value in prism diopters at which fusion broke (break point). Then the prism was decreased until a single image was seen, and that value was recorded to represent fusion recovery (recovery point).[20]

Statistical analysis

To compare the sex and the number of orthoptic tests altered in the ID patients vs the control group, the chi-square test was used.

Scores of TMI, FI, MI, JI, age, and prism diopters were calculated as means and standard deviations. To compare these data between the ID patients and the control group, the Student *t* test was used.

Analysis of variance (ANOVA) for positive fusional reserve at near (break and recovery points) was used to compare the DDR and DDNR patients.

Chi-square and Student *t* tests were also used in comparing all orthoptic tests between the ID patients (DDR and DDNR) and the control group.

Data were analyzed by using Primer of Biostatistics for Windows (version 4.02; McGraw-Hill, New York, NY).[21]

P values <0.05 were considered statistically significant.

RESULTS

The TMI scores of the DDR and DDNR patients did not differ statisticaly. Similary the FI, MI, and JI scores

Table I. Temporomandibular index and the associated subindex mean values (SD) for 2 groups of ID patients and the control group

Index/subindex	*DDR patients (n = 27)*	*DDNR patients (n = 23)*	*Control group (n = 50)*	*Student t test (P value)**
Function index	0.40 (0.12)	0.44 (0.12)	0.10 (0.05)	0.000
Muscle index	0.54 (0.23)	0.64 (0.27)	0.11 (0.12)	0.000
Joint index	0.43 (0.28)	0.45 (0.24)	0.02 (0.07)	0.000
Temporomandibular index	0.46 (0.21)	0.51 (0.21)	0.08 (0.08)	0.000

*DDR patients vs control group and DDNR patients vs control group.

for the 2 ID groups were not statistically different. However, all scores of TMI, FI, MI, and JI of the DDR and DDNR patients were significantly different from the TMI, FI, MI, and JI scores of the control group ($P < 0.000$).

A significant difference was found for the convergence test; it was altered in 24 (48%) ID patients and in 8 (16%) control subjects ($P < 0.023$).

For mean age, sex, presence of correction of the visus through the use of glasses, Maddox test, alternating cover test, and ocular motility test, no statistical differences were found between the ID patients and the control group.

Significant variations in break and recovery points of near positive fusional vergence (expressed in Δ) were found between the ID patients and the control group (24.04 ± 9.20 vs 27.2 ± 8.56, $P < 0.046$; 16.5 ± 7.25 vs 19.5 ± 7.52, $P < 0.045$, respectively).

For the comparisons between the ID subgroups for positive fusional reserve at near, a smaller value of break point in the DDNR patients (27.61 ± 8.89 vs 22 ± 9.62, $P < 0.037$) was found.

In the comparisons between the ID subgroups and the control group, the subjects with DDNR had most of the convergence insufficiency ($P < .002$) and worse values of break and recovery points ($P < .023$ and $P < 0.043$, respectively).

Data are shown in Tables I through VI.

Table II. Results of convergence, cover, and ocular motility tests in ID patients vs control group (number of patients compared with chi-square test)

ID patients	*control group*		
Convergence test			$P = 0.023$
Normal (3-4 cm)	26	42	
Altered	24	8	
Sufficient (5-7 cm)	10	6	
Insufficient (>7 cm)	11	2	
No convergence	3	—	
Cover tests			NS
Esophoria	4	5	
Exophoria	8	6	
Orthophoria	38	39	
Ocular motility test			NS
Normal	46	48	
Altered	4	2	

NS, Not significant.

DISCUSSION

The sensory innervation of the TMJ is entrusted to the trigeminal nerve, and the cell bodies of the afferents connecting the receptor of the TMJ (free nerve endings, Ruffini endings, Golgi organs, and Vater-Pacini corpuscles) to the central nervous system were found to be located in the trigeminal ganglion.[22]

Anatomic connections were shown between the trigeminal and oculomotor systems. In the mesencephalic nucleus of the trigeminus, which extends from the dorsal portion of the spinal trigeminal nucleus to the caudal part of the superior colliculus, in addition to the primary afferent neurons associated with the MM, tooth pulp, periodontal ligaments, also connections associated with extraocular muscles were present.[3,4]

Buisseret-Delmas and Buiseret[7] used a peroxidase injection into the oculomotor muscles as a marker that diffuses in the Gasser node, in the pars interpolaris and caudalis of the spinal trigeminal nucleus, in the paratrigeminal nucleus, and in the dorsal horn of the cervical spinal cord.

A connection was also demonstrated between the main nucleus of the trigeminus and the oral, interpolar, and caudal portions of the spinal trigeminal nucleus with the vestibular nucleus and the prepositus nucleus of the hypoglossus.[8] This nucleus is part of a small group of nervous cells adjacent to the nucleus of the hypoglossus, but it does not connect with it; it is also an important center of calculation of the position and movements of the eyes, because of its strict relationship with the vestibular nuclei, the cerebellum, and the oculomotor nuclei. These results suggest that portions of the trigeminal system strongly influence the coordination of posture and sight.[9]

The mesencephalic nucleus of the trigeminus also had connections with medial, inferior, lateral, and

Table III. Means and standard deviations for vergence system in ID patients and control group

	ID patients	*Control group*	*Student* t *test*
Positive fusional reserve at near (break point, in Δ)	24.04 ± 9.20	27.2 ± 8.56	$t = -2.026$, $P = 0.046$
Positive fusional reserve at near (recovery point, in Δ)	16.5 ± 7.25	19.5 ± 7.52	$t = -2.031$, $P = 0.045$
Heterophoria at near (in Δ)	0.5 ± 2.05	1.06 ± 3.48	NS

Δ, Prism diopter; *NS,* not significant.

Table IV. Comparisons between the positive fusional reserve at near (break and recovery points in Δ) of patients with DDR and DDNR

Patients with DDR (n = 27)	*Patients with DDNR (n = 23)*	*ANOVA*
Positive fusional reserve at near (break point, in Δ)		
27.61 ± 8.89	22 ± 9.62	F = 4.59, $P = 0.037$
Positive fusional reserve at near (recovery point, in Δ)		
17.92 ± 5.64	15.51 ± 8.02	NS

NS, Not significant.

Table V. Convergence defect in patients with ID vs control group (number of patients compared uwith chi-square test)

	Patients with DDR (n = 27)	*Control group (n = 50)*	
Normal (3-4 cm)	21	42	NS
Altered	6	8	
Sufficient (5-7 cm)	6	6	
Insufficient (>7 cm)	—	2	
No convergence	—	—	
	Patients with DDNR (n = 23)	*Control group (n = 50)*	
Normal (3-4 cm)	5	42	$P = 0.002$
Altered	18	8	
Sufficient (5-7 cm)	4	6	
Insufficient (>7 cm)	11	2	
No convergence	3	—	

NS, Not significant.

superior vestibular nuclei, with lobes IX and X of the cerebellum,[5] and the superior colliculus (SC),[6] and this connection suggested that neurons of the mesencephalic nucleus of the trigeminus can influence vestibular control of eye and head movements.

We analyzed the behavior of various parameters used to evaluate binocular function (motility and vergence system) in subjects aged 18 to 40 with and without ID of the TMJ. The 2 groups were comparable with regard to sex, numbers of teeth, and wearing of glasses.

Evidence of the correlation between the eyes and the dental occlusion was noticed in the use of appliances which, on modifying mandibular position, changed visual focusing.[10]

Gangloff et al[11] showed the relationship between dental occlusion, proprioception, and visual stabilization.

Recently, some authors suggested a much higher prevalence of ocular convergence defects in adults with TMD and limited maximal opening, myofascial pain, and pain in the neck and shoulders,[12] and in children with functional mandibular laterodeviation.[13]

The oculomotor system, moving the eyes in all directions, functions to stabilize the images on the retina to maintain the binocular vision on the fovea centralis during active and passive movement of the body and the head in space. Ocular movements are normally conjugate: each eye completes a movement of the same amplitude in the same direction.[23]

Ocular convergence is a disconjugate movement: the eyes move in several directions, and also the amplitude of the movements can vary. These disconjugate movements are generated by the vergence system, allowing the image of the object that is approached to remain aligned on the fovea.

Ocular convergence is checked by a group of cells in the midline of the oculomotor nuclear complex (Pelia's nucleus) that are under the control of the SC. [24]

Convergence insufficiency is a condition in which the patient cannot sustain sufficient convergence for comfortable near vision; it is a common cause of ocular discomfort.[25]

In this study, the ID patients showed reduced convergence compared with the healthy subjects, particularly the DDNR patients.

Moreover, a positive fusional convergence reduction was found in the ID patients. The normal alignment of the eyes is maintained by their fusional movements. The reflex is driven by retinal image disparity. In normal conditions, retinal image disparity produces diplopia. Fusional movements then trigger a vergency response to align the images of the object in regard to the 2 foveas. Near positive fusional vergence measures the extent to

Table VI. Means and standard deviations for vergence system in patients with ID (DDR and DDNR) vs control group

Positive fusional reserve at near (break point, in Δ), Student *t* test		
DDR patients (n = 27)	Control group (n = 50)	NS
27.61 ± 8.89	27.2 ± 8.56	
DDNR patients (n = 23)	Control group (n = 50)	$t = 2.318$, $P = 0.023$
22 ± 9.62	27.2 ± 8.56	
Positive fusional reserve at near (recovery point, in Δ), Student *t* test		
DDR patients (n = 27)	Control group (n = 50)	NS
17.92 ± 5.64	19.5 ± 7.52	
DDNR patients (n = 23)	Control group (n = 50)	$t = 2.062$, $P = 0.043$
15.51 ± 8.02	19.5 ± 7.52	

Δ, Prism diopter; *NS,* not significant.

which a person can maintain fusion with gradually increasing vergence demands. The clinical purpose of this test is to provide information about a patient's ability to maintain comfortable binocular single vision.[26] A lower value of the break point in ID patients is the same as a smaller range of binocular vision, whereas a larger value of the recovery point shows quicker reinstatement of binocular vision in healthy subjects.

Also, in this case, these disorders are more frequent in patients with DDNR.

These findings can be explained by destruction of the innervate articular structures (fibrous capsule, peripheral fibrous layers of the disc where it is attached to the joint capsule) that can cause pain and inflammation with greater stimulation of the central nervous system.[27]

Degenerative joint diseases were found in only 17% of the DDR patients and in 95% of the DDNR patients. Most DDNR patients had joints with discs that were not reparable.[28]

Moreover, in the DDNR patients, there was greater muscular involvement. DDNR can cause spastic activity in the temporalis and masseter muscles on the same side as a joint with anterior disc displacement, and hinder or inhibit the condylar movement necessary to achieve reduction.[29]

According to the TMI scores, the FI and JI scores were similar for DDR and the DDNR patients, whereas the DDNR patients had MI scores of 0.64, and the DDR patients had scores of 0.54, indicating (although no statistical differences was found between these means) that the disorder in the ID group was predominantly muscular.

In particular, pain of the TMJ and the MM could play an important role in the ocular motor findings.

Pain from TMD is usually variable and can be described as dull or throbbing, lasting minutes to hours. The pain (bilateral or unilateral) is typically triggered by jaw movements or palpation of the TMJ or the MM, and can be located especially in the preauricular region and sometimes radiates to the temples or the neck.[30]

Noxious stimuli and tissue inflammation of the TMJ and the MM can produce pain hypersensitivy. The crucial mechanisms underlying the increased excitability of nociceptive neurons of the spinal trigeminal nucleus, singly or together, are central sensitization (increased excitability of brainstem nociceptive neurons), temporal summation (increase in the experienced intensity of pain from repeated stimulation), and glial cell (microglia and astrocytes) activation.[31]

We hypothesize that altered ocular movements might be related to dysfunction at a subcortical level, particularly in SC.

The SC is a relay center in the midbrain receiving visual, somesthetic, and proprioceptive afferent fibers and is involved in postural motor and gait control as well as gaze movements. Bilateral trigemino-collicular connections have been described in pigs, rats, and cats, and this connection is of interest particularly for the principal trigeminal nucleus.[32,33]

The maintenance of a correct movement of vergence, while trigeminal nociceptive afferent stimulus of altered intensity and frequency come (trigeminotectal pathways), can produce an increasing sensitivity, interpreted as possible "central fatigue," characterized by progressive reduction of motoneuron recruitment and capacity of synthesizing neurotransmitter, with loss of muscular force.[34] This central fatigue can be greater in TMD patients than in healthy subjects.

Future studies should ascertain, with stimuli of different intensity and duration, the threshold for oculomotor trouble and how treatment for TMD influences the oculomotor findings.

CONCLUSIONS

TMD is a complex series of disorders often caused by trauma and characterized by pain and musculoskeletal symptoms.

The results of this study show that patients with ID can have alterations in binocular function. Thus, the clinical TMJ examination should include the extraocular musculature, estimating its behavior during gnathologic therapy. Probably, altered afferences carried by the trigeminal system, resulting from lesions in the MM or the TMJ, can disturb binocular function, because of the functional relationship between the trigeminal and oculomotor systems.

Even if the oculomotor function cannot be considered as serious as the TMD, since the visual function is the most important afferent pathway in posture, the clinician should be aware of these possibilities, especially when treating patients with a problem in the postural scheme.

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