Combined effect of vestibular and craniomandibular disorders on postural behaviour

Effetti combinati dei disordini vestibolari e cranio-mandibolari sulla postura

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Summary

A correlation has been reported in the dental literature between temporomandibular disorders and musculoskeletal abnormalities, however, the question whether they modify body postural sway remains controversial. In the present investigation, the Craniomandibular Index was used to evaluate the clinical extension of temporomandibular joint dysfunction and related problems in 40 patients with normal vestibular function and in 42 patients with peripheral vestibular disorders. Balance function was assessed by static posturography and body sway area was measured in two conditions: i) eye open, and g) eye closed. Data were compared to those of 40 healthy subjects. Postural control showed a significantly different behaviour between groups with an increase in average body sway in patients with craniomandibular disorders as opposed to controls ($p<0.005$). Although the involvement of the stomatognathic apparatus was not quantitatively different in the two groups of patients, those also presenting a peripheral vestibular disorder exhibited greater average body sway than patients with only craniomandibular disorders ($p<0.005$). The latter showed a greater average body sway than controls only in the trial with eyes closed ($p<0.05$). The results demonstrated that craniomandibular alterations could produce moderate postural instability in patients with a normal vestibular function. Conversely, their association with peripheral vestibular disorders becomes a real challenge to the upright quiet stance probably due to a negative effect of somatosensory origin on the vestibulo-spinal reflex impairment.

Riassunto

Sebbene l'esistenza di una correlazione tra i disordini dell'articolazione temporo-mandibolare e le alterazioni dell'apparato muscolo-scheletrico sia stata più volte riportata in Letteratura in campo odontostomatologico, la questione se esista una correlazione tra questi disordini e le modificazioni della postura è ancora da definire. In questo studio il Cranio Mandibular Index è stato utilizzato per valutare il grado di disfunzione temporo-mandibolare ed i problemi ed essa corrrelati in 40 pazienti con funzionalità vestibolare nella norma, ed in 42 pazienti affetti da disordini vestibolari periferici. La valutazione dell'equilibrio è stata eseguita utilizzando la posturografia statica e la "body sway area" misurata in due condizioni: ad occhi aperti e ad occhi chiusi. Questi dati sono stati successivamente confrontati con quelli ottenuti nei 40 soggetti sani. Il controllo posturale ha evidenziato un comportamento significativamente differente tra i due gruppi con un aumento del "body sway" medio nei pazienti con disordini cranio-mandibolari rispetto ai controlli ($p<0,005$). Sebbene il coinvolgimento dell'apparato stomatognatico non risulti quantitativamente differente nei due campioni patologici, i pazienti affetti anche da sofferenza vestibolare periferica hanno presentato un "body sway" medio maggiore rispetto ai pazienti affetti solo da disordini craniomandibolari ($p<0,005$). Questi ultimi hanno presentato un "body sway" medio maggiore dei controlli solo nelle prove ad occhi chiusi ($p<0,05$). I risultati di questo studio hanno dimostrato che le alterazioni craniomandibolari possono essere alla base di una moderata instabilità posturale nei pazienti con funzionalità vestibolare nella norma. Al contrario tali alterazioni, se associate a disordini vestibolari periferici, possono determinare problemi nel mantenimento della stazione eretta, probabilmente a causa dell'effetto negativo di origine somatosensoriale sui riflessi vestibolo-spinali.

Introduction

While abnormal postural stability related to primary neck dysfunction has been extensively discussed in the medical literature [1 2] there is lack of information concerning the role possibly played by craniomandibular disorders (CMDs) in the co-ordination of whole body posture and balance function. These disorders mainly affect the function and the morphology of the mandible with respect to the bilateral glenoid fossae of the skull (temporomandibular joint, TMJ), and its neuromuscular apparatus. The most frequent symptoms are joint noise, limited mandibular movements, painful reaction of the muscles, earache and cephalalgia. The aetiology of CMD still remains to be fully elucidated, however, there is general agreement that tempomandibular arthropathy, functional disturbances of the masticatory system (such as bruxism) and, above all, abnormal dental occlusion [3] are variously involved. A recent study [4] has shown that, in normal sub-

jects, experimental occlusal interference produces electrical variations in the antigravity muscles in a very short time and it is also known that vibration applied to these extensor muscles causes proprioceptive 'misinformation' which produces an illusion of movement and a consequent body shift in upright stance [5]. Moreover, it has been observed that neurologically intact patients with temporomandibular disorders exhibit abnormal balance and coordination functions in response to extreme mandible positions [6].

Epidemiological studies have demonstrated that impaired balance, vertigo and other otoneurological symptoms are frequently associated in such patients [7] thus suggesting the possibility that CMD could produce changes in postural and gait control by inducing perturbation of the vestibulo-spinal reflexes. This hypothesis is supported by experimental observations that visual, vestibular and proprioceptive end-organs interact with each other and, on the whole, contribute to postural stability [8] and by the evidence of convergence of labyrinthine and trigeminal afferent fibres in the vestibular nuclei [9 10]. Moreover, it has been shown that vestibular signals and proprioceptive inputs from the neck region converge not only in the central nervous system but also in the central cervical nucleus of the upper spinal cord which is also thought to receive sensitive signals of temporomandibular origin via the trigeminal nuclei [11]. Despite experimental evidence for a possible interaction between the stomatognathic apparatus and vestibulo-spinal reflexes, most of the previous investigations evaluating body postural responses to CMD, unfortunately failed to assess the vestibular function in appropriate clinical settings [12-14].

The purpose of the present study was, therefore, twofold: the first was to determine whether the CMD induce adverse effects on body sway during active upright stance and the second to test this feature in a population of patients presenting also peripheral vestibular diseases.

Patients and methods

Patients

From February 1998 to March 2001, 82 patients were selected from new patients coming for treatment at the Dental and Maxillo-Surgery Department, University of Modena and Reggio Emilia. This sample comprised 29 males (35.4%) and 53 females (64.6%) of similar age (males: 23-40, mean 34.3 years; females: 24-43 years, mean 35.4). Inclusion criteria were as follows:

- complaint of one or more episodes of acute vertigo (i.e., a rotatory illusion of movement) and/or disequilibrium in the six months prior to examination;
- presence of at least two of the following clinical signs and symptoms: TMJ sounds, tenderness to palpation of the TMJ and the masticatory muscles, or painful limitations of mandibular movements;
- presence of a full permanent dentition, regardless both of first permanent molar and canine relationships and previous orthodontic treatment or cusp coverages and restorations.

Exclusion criteria were:

- previous craniocervical trauma;
- musculoskeletal diseases and previous bone fractures of the spinal cord and lower limbs;
- neurological diseases;
- use of neuroleptic drugs.

Patients were compared with 40 healthy volunteers (controls) well-matched according to sex (10 males, 33.4 %, 30 females, 66.6%) and age (males: 24-40 years, mean 33.6; females: 22-41 years, mean 33.7). Healthy subjects were recruited among hospital staff personnel and students on the bases of a normal vestibular examination and a full permanent dentition that included the second molars, with bilateral Angle I first permanent molar and canine relationships, no previous and/or current orthodontic treatment, no signs or symptoms of CMD and no reported history of jaw dysfunction and balance disturbances.

The experimental protocol conformed with the Declaration of Helsinki for Human Experimentation and informed consent was obtained from each individual taking part in the study before examination.

Experimental protocol

To clinically establish the extent of the CMD, all patients and controls were evaluated according to the Craniomandibular Index (CMI) [15]. A list comprised 62 items grouped into two composite scores which are the Dysfunction Index (DI) and the Palpation Index (PI) from which an overall score (i.e., craniomandibular index) is calculated according to the formula (DI + PI)/2. The DI is a list of items scored as a positive, 1, or negative, 0, clinical finding in mandibular movement (MM) and the intracapsular TMJ noise (TN) while the PI includes items scored as positive, 1, or negative, 0, in relation to tenderness to palpation of the intraoral- and extraoral masticatory muscles (IM and EM), the superficial neck muscles (NM) and TMJ capsule palpation (TP). Both Indices are calculated by using the sum of the positive responses divided by the total number of items. The CMI is, then, the sum of DI and PI divided by 2 (Table I). The complete list of items and the methodological guidelines have been published elsewhere [15].

The involvement of the stomatognathic apparatus was further investigated by means of surface electromyography of the masticatory muscles, mandibular kinesiography and T-scan of the temporomandibular joint. These investigations allowed us to classify patients, according to the criteria of the American Academy of Craniomandibular Disorders [16], as suffering from pure masticatory muscle disorders (myofascial pain and

Table I. Craniomandibular index score.

Scales		Method	Range
Mandibular Movements	MM	No. positive responses	0-16
TMJ Noise	TN	No. positive responses	0-4
TMJ capsule palpation	TP	No. positive responses	0-6
Dysfunction Index	DI	DI=(MM+TN+TP)/26	0-1
Extra-oral jaw muscle palpation	EP	No. positive responses	0-18
Intra-oral jaw muscle palpation	IP	No. positive responses	0-6
Neck muscle palpation	NP	No. positive responses	0-12
Palpation Index	PI	PI=(EP+IP+NP)/36	0-1
Craniomandibular Index	CMI	CMI=(DI+PI)/2	0-1

contracture) in 32 cases (39%), from temporomandibular joint disorders (deviation in form, disc displacement with and without reduction) in 20 cases (24%) and from both muscular and articular disorders in 30 cases (37%).

All patients and controls were also submitted to audiological and vestibular examinations (Computerised electrooculography Toennies Pro System, Erich Jaeger GmbH & Co. KG, Wurzburg, Germany, 1994). The electrooculographic battery included tests for exploring oculomotor (saccades and smooth-pursuit) and optokinetic functions, spontaneous and gaze nystagmus, three cycles of sinusoidal rotation testing with a maximum speed of 60°/sec for vestibulo-oculomotor reflex and bithermal irrigation (33°C and 44°C) of both ear canals for labyrinthine activity. The vestibular paresis formula of Jongkees et al. [17] was used: {[(R 30°C + R 44°C) – (L 30°C + L 44°C)/ [(R 30°C + R 44°C + L 30°C + L 44°C)]} x 100 where, for example, R 30°C is the maximum slow phase velocity of nystagmus induced by the caloric irrigation of the right ear canal with 30°C warm water. Vestibular paresis was defined as more than a 25% asymmetry between the right-side and left-side responses [18]. This asymmetry was the minimum requirement for a significant labyrinthine hypofunction. The vestibular examination was completed by cerebral magnetic resonance in all cases. Patients with signs and symptoms suggesting an involvement of the central vestibular system were excluded.

Static posturography was performed in all subjects by means of a stable force-plate sensitive to vertical force. The force-plate was mounted on three strain-gauge force transducers which are positioned at the vertices of an equilateral triangle, providing description of body sway in terms of displacement of the centre of gravity of the patients (i.e., approximately the projection of the centre of mass to the ground). Stabilometric recordings were performed in standardised conditions: healthy participants and patients were all requested to maintain a relaxed, motionless upright stance, stand bare foot with feet at an angle of 30°, with a natural head-neck posture and habitual occlusion, both arms hanging beside the trunk, under two different conditions: 1) gazing at a steady, vertical light bar at a distance of 150 cm (EO), 2) with eyes closed (EC), in total darkness. The duration of each test was 52 seconds.

Stabilometric parameters

Stabilometric data were sampled at 5 Hz in each test. Postural sway (S) was computed by continuously detecting the body's centre of gravity and calculating an elliptic area which corresponds to 90% of the positions of the centre of gravity over time. This procedure is designed to eliminate 10% of the more extreme positions which could be due to involuntary perturbations of quiet stance. The mean magnitude of S (expressed in square millimetres) was computed in both visual conditions (EO and EC) and a stabilometric Romberg's quotient was calculated using the following formula:

Romberg's quotient = [(S score EC/S score EO) x 100]

which is designed to evaluate the sensorial weight of visual cues in the multisensory control of postural sway.

Statistics

One-way analysis (ANOVA) of data was used to test the null hypothesis that the means of the scores of CMI, DI, PI and summary items, stabilometric data and Romberg's quotient are equal in the samples. Post-hoc analysis was performed with the Bonferroni test. Finally, the Pearson correlation coefficient was calculated to measure the association between CMI and its composite scores and stabilometric parameters. The statistical significant level was set at 0.05 in all procedures.

Table II. Number, sex and diagnostic categories of patients with peripheral vestibular diseases (group B).

	Vestibular neuritis	Meniérè's disease	Perilymphatic fistula	Acoustic neuroma	Total
Males	11	3	2	–	16
Females	15	10	–	1	26

Table III. Mean values of craniomandibular index and the composite scores in patients (groups A and B) and controls. Degree of freedom (df) and p values (two-tail) are reported.

	Control		Group A		Group B			
	M	SD	M	SD	M	SD	Df	p
MM	1.65	1.15	8.10	3.07	9.54	3.23	2	0.000
TN	0.12	0.32	2.21	1.32	2.34	1.11	2	0.000
TP	0.93	0.77	2.00	1.53	1.78	1.26	2	0.000
DI	0.10	0.70	0.47	0.14	0.52	0.14	2	0.000
EP	1.63	1.70	12.3	4.00	13.5	1.48	2	0.000
IP	1.26	1.20	3.12	1.06	3.02	0.88	2	0.000
NP	2.02	1.12	1.95	1.15	1.90	0.77	2	0.853
PI	0.14	0.11	0.65	0.19	0.68	0.09	2	0.000
CMI	0.12	0.06	0.47	0.11	0.51	0.07	2	0.000

Results

After collecting a full history and complete neurotological examination, craniomandibular symptomatic patients were divided into two groups: those with no vestibular dysfunction (group A: 40 patients) and those with a unilateral vestibular hypofunction (group B: 42 patients) (Table II).

Table IV. Post-hoc analysis of mean value of CMI total scores and the composite scores between controls, CMD patients (group A) and patients suffering from both CMD and peripheral vestibular disorders (group B).

Variables	Group	Group	Difference of means	p
	A	Control	0.37	0.000
DI	B	Control	0.42	0.000
	B	A	0.05	1.0
	A	Control	0.51	0.000
PI	B	Control	0.54	0.000
	B	A	0.03	1.0
	A	Control	0.43	0.000
CI	B	Control	0.47	0.000
	B	A	0.04	0.273

As expected, the mean scores of the CMI and both the composite scores (Dysfunction and Palpation Index) were higher in the two patient groups than in controls ($p < 0.000$). Almost all the items provided the sources of variation ($p < 0.000$) with the exception of the outcome from neck palpation (NP) the mean value of which was not statistically different ($p > 0.05$) (Table III). Post-hoc analysis revealed that CMI and its composite scores were higher in both the patient groups than in controls but no difference was found between groups A and B ($p > 0.05$) (Table IV).
Stabilometric parameters varied greatly between groups ($p < 0.000$) (Tables V-VI). Mean body sway areas, in each visual condition, showed a greater increase in group B than in group A and controls ($p < 0.000$), while post-hoc analysis showed that patients with CMD exhibited a larger body sway in the EC condition than controls ($p < 0.05$) but no difference in EO ($p > 0.05$).
The stabilometric Romberg's quotient was greater in both patient groups than in controls ($p < 0.005$), with no statistical difference between the former ($p > 0.05$). These data indicate that the visual stabilisation of posture is greater in the patient groups than in controls even if the high standard deviation confirms a marked interindividual variability [19].
A linear association ($p < 0.005$) was found between postural body sway in both visual conditions and the CMI and its composite scores, with the exclusion of neck palpation ($p > 0.05$). Similar correlations were

Table V. Means and standard deviations of the stabilometric parameter (body sway surface) in the two visual conditions (eye open and eye closed) and stabilometric Romberg quotient (RQ).

	Controls		Group A		Group B			
	m	SD	m	SD	m	SD	df	p
S (EO)	131.3	53.8	135.6	77.3	407.7	242.9	2	0.000
S (EC)	163.9	78.0	366.4	170.8	881.8	479.3	2	0.000
RQ	134.0	57.6	225.0	133.7	277.1	105.3	2	0.000

Table VI. Post-hoc analysis (Bonferroni test) of the means of body sway (S) in both visual conditions (EO and EC) and the stabilometric Romberg quotient.

Variables	Group	Group	Difference of means	p
S (EO)	A	Control	4.2	1.0
	B	Control	276.3	0.000
	B	A	272.1	0.000
S (EC)	A	Control	202.5	0.027
	B	Control	717.9	0.000
	B	A	515.4	0.000
RQ	A	Control	91.1	0.013
	B	Control	143.1	0.000
	B	A	52.0	0.309

seen for the stabilometric Romberg's quotient, suggesting that the stabilising effect of visual cues in postural control is largely conditioned by the totality of craniomandibular signs and symptoms, but not by the cervical myofascial pain and tenderness (Table VII). No apparent difference was found between the articular and the muscular dysfunction of the masticatory system as far as the correlation with postural stability is concerned, however, it should be pointed out that this investigation was not specifically designed to address this issue.

Discussion

Recent studies in humans [20] have shown a close correlation between temporomandibular disorders and impaired orthostatic posture in particular with regard to abnormal position of the shoulder and pelvis line. Furthermore, a correlation between malocclusion and forward head and body posture [21] has been observed. Despite these results, this study has demonstrated that postural stability is only slightly affected by the presence of CMDs since a moderate increase in body sway area is triggered only by eye closure (i.e., no visual information for postural control available). A possible explanation why these somatic bases do not cause marked postural imbalance is that intact vestibular and visual systems adapt balance reflexes in patients with musculoskeletal misalignment related to CMDs. In fact visual, vestibular and somatosensory systems are mutually interactive in postural control and provide redundant information thus partially compensating each other's deficiencies. As confirmation, the stabilising role of visual cues on postural body sway, as demonstrated by the stabilometric Romberg's quotient, was greater in both patient groups than in controls and showed a close correlation with the CMI. On the contrary, it was confirmed that patients with both craniomandibular and peripheral vestibular disorders manifested a greater average postural sway as compared to patients with CMDs alone [22]. It is feasible to suggest that an abnormal so-

Table VII. Measurements of association between stabilometric parameters and CMI, the composite scores and main items. Pearson's coefficient and probability levels are reported.

Pearson's correlation	MM	TN	TP	DI	EP	EM	NP	PI	CI
S (EO)	0.274	0.255	0.228	0.281	0.302	0.230	0.078	0.281	0.287
Sig. 2-tails	0.002	0.004	0.002	0.001	0.001	0.010	0.383	0.001	0.001
S (EC)	0.460	0.295	0.301	0.369	0.415	0.296	0.017	0.369	0.418
Sig. 2-tails	0.000	0.001	0.002	0.000	0.000	0.001	0.852	0.000	0.000
RQ	0.472	0.379	0.296	0.340	0.383	0.305	0.101	0.390	0.414
Sig. 2-tails	0.000	0.000	0.001	0.000	0.000	0.001	0.260	0.000	0.000

matosensory input of stomatognathic origin might produce an additional negative effect on the altered vestibulo-spinal reflexes which are equally affected by the abnormal neuromuscular proprioception of other areas [23]. A valid physiological basis for this hypothesis is evidence from previous investigations [24] for a hyperactivity of the masticatory muscles with the mandible at rest and their reduced functional activity in patients with craniomandibular dysfunction.
Although some reports [25] have suggested a high incidence of cervicovertebral dysfunction in patients with temporomandibular disorders, we found neither a significant neck muscle involvement in our patient population nor a correlation with increased postural sway. This finding suggests that even if comorbidity between malocclusion, temporomandibular disorders and the cervical spine might be suspected, the destabilising effect of abnormal neck somatosensory input could be elicited only by specific neck positions such as head extended backward [26] or by an extrinsic vibratory perturbation of cervical muscles [1 2].
It was, therefore, concluded that craniomandibular alterations may modify body postural sway in upright stance and further investigations are needed to clarify the role played by these disorders whereas patients with peripheral vestibular lesions exhibit persistent abnormal postural control or complaints with recurrent subjective postural instability.

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