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Optoelectronic Pantography Diagnostics of Temporomandibular Disorders in Patients with Bruxism

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A B S T R A C T

Temporomandibular disorders (TMD) is a joint term that encompasses a number of clinical symptoms that involve the teeth, masticatory musculature and temporomandibular joints (TMJ). They are a frequent cause of orofacial medical conditions. The aetiology of disorders is complex and individual etiologic factors are not sufficiently defined. Bruxism, in its centric or eccentric form, is becoming a frequent problem for dentists. The purpose of this study is to show factors of the condyle leading in patients with bruxism by optoelectronic pantography, and to establish the possibility of using optoelectronic pantography in the diagnostic procedure of TMD. Patients were selected (N=42), with incomplete sets of teeth, without prosthodontic appliances and with traces and symptoms of TMD. After completing the history questionnaire a clinical check up and plaster cast analysis patients with bruxism were selected (N=22) and without bruxism (N=20). During the study optoelectronic String-condylocomp LR3, Dentron, D-Höchberg (software JAWS 30) was used. This study showed the possibility of applying optoelectronic pantography in TMD diagnostics and compares history, clinical and condylographic parameters in TMD patients with and without bruxism. Optoelectronic pantography enables us, by using relatively easy methods, to determine a more accurate diagnosis, highly important when choosing therapeutic methods and control of the aforementioned disorders.

Key words: *TMD, bruxism, diagnostics*

Introduction

Temporomandibular or craniomandibular dysfunction is a common term for disorder of one or more components of the stomatognathic system: teeth, masticatory muscles, temporomandibular joint and associated structures. According to the definition of the American Academy of Orofacial Pain (AAOP)[1,2] it is defined as a cluster of pathological changes which are manifested as a wide range of symptoms associated with the temporomandibular joint (TMJ) and masticatory muscles. It is classified as muscle-bone dysfunction of chronic character with considerable fluctuation of signs and symptoms during a certain period of time[3,4]. For easier recognition and understanding of temporomandibular dysfunction Kuttila et al.[5] differentiate symptoms (pain in masticatory muscle and TMJ area, limited possibility of opening the mouth) and signs (clacking, crepitation and deviation during jaw movement). De Boever et al.[6] explain the aetiology of TMD through five theories: mechanical dislocation, neuromuscular, psychosocial, muscular and psychological. Okeson[2] classifies etiological factors into endogenous (idiopathic, systemic, psychosomatic, psychological and pathophysiological) and exogenous, originating from trauma and occlusal dysfunction caused by prosthetic, conservative, surgical, orthodontic and periodontal therapy.

Occlusal dysfunction such as open bite, vertical overlap between 6–8 mm, difference between initial contact and maximal intercuspidation greater than 2 mm, five or

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more extracted teeth and unilateral occlusion can be related to the occurrence of TMD. The role of certain etiological factors of TMD is not fully explained and differs from case to case[7]. As somatization, depression and alexithymia are often associated with TMD it is hard to detect if the patient's psychological condition is the basis for TMD development or that TMD occurs as a response of the body to the painful condition[8]. In 1992 Dworkin and La Resche in their Research Diagnostic Criteria also specify psychosocial problems as the possible cause of depressive behaviour and emotional instability of the patient. Their biopsychosocial model for chronic pain includes diagnostic criteria for temporomandibular dysfunction, placed in the biaxial system (axis I and II)[3].

There are three basic diagnostic groups: Group 1 – muscular disorders, Group 2 – disk shift, Group 3 – arthralgia, arthritis and arthrosis[7,9]. Parafunctional activity becomes prominent when defensive reflexes weaken. They are characterised by strong occlusal contact forces, mostly of horizontal direction, relative occlusal instability and isometric muscular contractions[2,10]. AAOP defines bruxism as daily or nightly parafunctional activity, which includes (squeezing, scratching or teeth grinding[11]. Bruxism in sleep is defined by the American Sleep Disorders Association (ASDA) as stereotype dysfunction of motion characterised by (scratching, gnashing) and teeth squeezing during sleep[12,13]. Frequency of bruxism in the general population ranges from 8% to 21%. After clinical oral examination it is possible to detect it in a much higher frequency ranging between 48% and 50%[14]. According to some findings it can reach frequency of 90%[15]. There are two aetiological models: structural and functional. In the structural model the main role is played by occlusal disturbances or deviations in jaw to jaw height interrelationship[16,17]. In the functional model the most important role is played by emotional tension (anxiety) and personality traits[18,19]. Changes in activity of the limbic system, especially destruction of amygdale lead to behavioural changes similar to those such as bruxism. It allows the conclusion that such changes can play a role in the aetiology of such parafunctional activity[20]. Graber and associates[21] supported this theory. They described psychologically caused centric bruxism in individuals under intense and permanent stress. Vanderas[22] measured daily levels of catecholamine in the urine of 314 children, and observed an association between the level of epinephrine and dopamine with bruxism. Amir[23] also described cases of bruxism caused by beta-blockers and antipsychotic drugs.

The purpose of the present study was as follows:

- To establish the possibility of optoelectronic panthography use in the diagnosis of TMD
- To compare the symptomatics of TMD patients with and without bruxism.
- To demonstrate factors of condylar guidance in patients with bruxism by use of optoelectronic panthography.

Material and Methods

The present study comprised a selected group of subjects (N=42), aged from 18 to 65 years (72% females, 28% males). All subjects displayed disturbances of the temporomandibular joint and malocclusion caused by conservative, surgical, orthodontic therapy or loss of supporting zone. They did not wear prosthetic appliances. Examinees were divided into two groups: patients with bruxism (n=22), and patients without bruxism (n=20). The required data on subjects were obtained by means of:

- Specific questionnaires independently completed by examinees. The obtained values were assessed on a

TABLE 1
DIFFERENCES BETWEEN PATIENTS WITH BRUXISM AND PATIENTS WITHOUT BRUXISM IN ANAMNESTIC VARIABLES

Anamnestic variables	Patients with bruxism N=20	Controls (without bruxism) N=22	t-value	P-value
Teeth grinding (SD)	5.3 (2.6)	0.3 (1.3)	7.636	0.000
Teeth sensitivity (SD)	3.8 (3.2)	2.3 (2.6)	1.662	0.104
Mouth opening – decreased (SD)	1.9 (2.6)	2.3 (3.3)	–0.472	0.639
Painful masticatory muscles (SD)	4.5 (3.5)	4.6 (2.9)	–0.050	0.960
Masticatory pain and pain on jaw movement (SD)	1.7 (2.6)	3.7 (3.2)	–2.182	0.035
Jaw junction pain (SD)	2.9 (3.5)	5.9 (9.6)	–1.359	0.181
Jaw junction clacking (SD)	3.6 (3.9)	3.2 (3.6)	0.336	0.738
Painful neck and shoulder muscles (SD)	4.6 (3.3)	4.5 (3.5)	0.134	0.894
Painful neck spine (SD)	4.9 (3.3)	4.3 (3.5)	0.538	0.594
Ear buzzing, decreased hearing (SD)	2.3 (2.9)	2.5 (2.8)	–0.255	0.800
Stress (SD)	5.7 (3.4)	3.9 (2.6)	1.946	0.059
Depression (SD)	1.8 (2.0)	1.2 (1.4)	1.066	0.293
Helkim's index (SD)	7.0 (6.3)	8.9 (7.3)	–0.858	0.396

Data is shown as an average grade/degree and SD – standard deviation scale from 1 (without symptoms) to 10 (severe dysfunction). After collection of data Helkim's index was calculated.

- Patients with bruxism had grinding, stress and depression high on the scale and bruxofacets determined by a clinical examination and analysis of working models in the articulator.
- Clinical examination, muscular and joint palpation, opening measurement, deviations and deflexions, Bumann's manual functional diagnostics.
- Plaster cast analysis.
- Optoelectronic pantographic method via String-Condylocomp LR3 (Dentron, GmbH, D-Höchberg) device with JAWS 30 software. The hinge axis with accuracy of a hundredth millimetre was determined for each examinee. Condylar pathway was monitored at the same time and continuously in all three space levels from P1 (circle) which represent the centric relation (CR) to P2 reference point (cross) which represents habitual relation of the condyle (MI). Differences between CR and MI expressed in millimetres were compared by computer programme. The intermittent curve indicates uncoordinated movements and the position of the circle indicates distraction or compression of the joint. Occlusal distance was calculated by means of a special programme, as well as inclination of the condylar pathway and Benett's angle. (Computer print out – samples of CI from research are presented in Figures 1 to 4).

TABLE 2
DIFFERENCES IN KINEMATIC CONDYLOGRAPHIC MEASUREMENT BETWEEN PATIENTS WITH BRUXISM AND WITHOUT BRUXISM

Average values (SD) of condylographic variables of jaw movement in mm		Patients with bruxism	Controls (Examinees without bruxism)	t-value	p-value
Jaw opening Na = 21 Nb = 18	RC – lateral shift	–0.02 (0.10)	0.02 (0.09)	–1.054	0.299
	RC – saggital shift	–0.04 (0.14)	0.04 (0.15)	–1.672	0.103
	RC – caudal shift	–0.05 (0.18)	0.08 (0.20)	0.622	0.538
	RC – total	0.19 (0.18)	0.23 (0.16)	–0.770	0.446
	LC – medial shift	0.02 (0.09)	0.01 (0.10)	1.233	0.225
	LC – saggital shift	–0.03 (0.18)	0.01 (0.23)	–0.605	0.549
	LC – caudal shift	–0.03 (0.14)	0.10 (0.18)	1.326	0.193
	LC – total	0.20 (0.14)	0.27 (0.16)	–1.336	0.189
Protrusion Na=22 Nb=20	RC – lateral shift	0.00 (0.16)	0.01 (0.11)	0.171	0.865
	RC –saggital shift	–0.11 (0.20)	0.04 (0.20)	–1.149	0.257
	RC –caudal shift	–0.04 (0.16)	0.16 (0.42)	1.221	0.229
	RC – total	0.22 (0.23)	51.12 (227.35)	–1.051	0.299
	LC – medial shift	0.01 (0.15)	0.01 (0.11)	–0.129	0.898
	LC – saggital shift	–0.10 (0.16)	0.10 (0.32)	–0.031	0.975
	LC – caudal shift	–0.10 (0.24)	0.23 (0.31)	1.509	0.139
	LC – total	4.79 (21.27)	0.33 (0.40)	0.936	0.355
Right mediotrusion Na=21 Nb=20	RC – lateral shift	–0.07 (0.21)	0.05 (0.19)	–0.277	0.783
	RC – saggital shift	–0.14 (0.27)	0.02 (0.38)	–1.888	0.067
	RC – caudal shift	–0.08 (0.37)	0.01 (0.59)	–0.486	0.629
	RC – total	0.34 (0.35)	0.48 (0.47)	–1.064	0.294
	LC – medial shift	0.08 (0.22)	0.05 (0.19)	0.448	0.656
	LC – saggital shift	–0.02 (0.21)	0.09 (0.39)	–1.066	0.293
	LC – caudal shift	0.01 (0.33)	0.00 (0.38)	0.055	0.956
	LC – total	0.28 (0.35)	0.41 (0.42)	–1.055	0.298
Left mediotrusion Na=20 Nb=20	RC – lateral shift	0.04 (0.29)	0.01 (0.10)	0.515	0.609
	RC – saggital shift	0.02 (0.72)	0.05 (0.28)	–0.139	0.889
	RC – caudal shift	0.10 (0.93)	0.01 (0.41)	0.361	0.720
	RC – total	0.50 (1.10)	0.36 (0.34)	0.531	0.599
	LC – medial shift	–0.04 (0.29)	0.02 (0.09)	–0.327	0.745
	LC – saggital shift	–0.09 (0.20)	0.10 (0.28)	0.098	0.922
	LC – caudal shift	–0.12 (0.24)	0.21 (0.34)	0.987	0.329
	LC – total	0.29 (0.35)	0.34 (0.38)	–0.396	0.694

Na – number of patients with bruxism, Nb – number of controls (examinees without bruxism); RC – right condyle; LC – left condyle

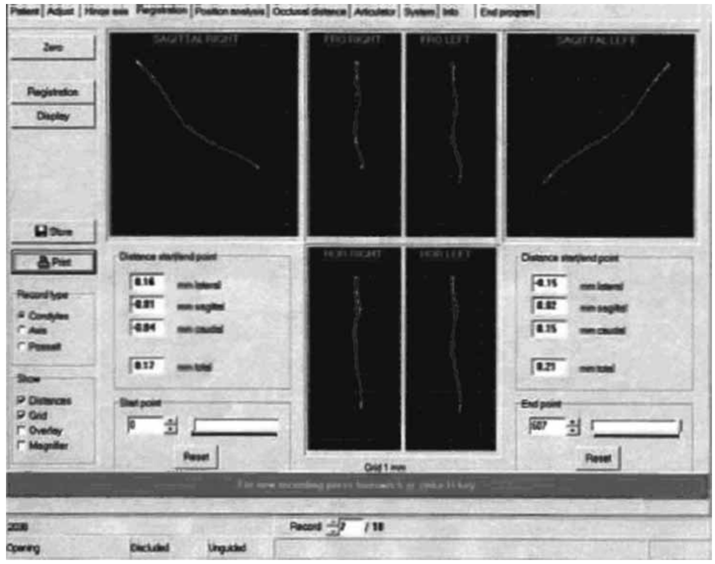

Fig. 1. Opening.

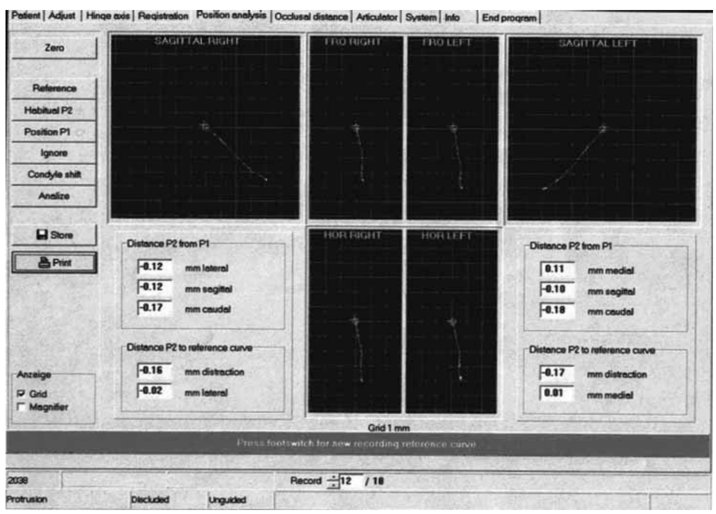

Fig. 2. CPA – condyle position analysis.

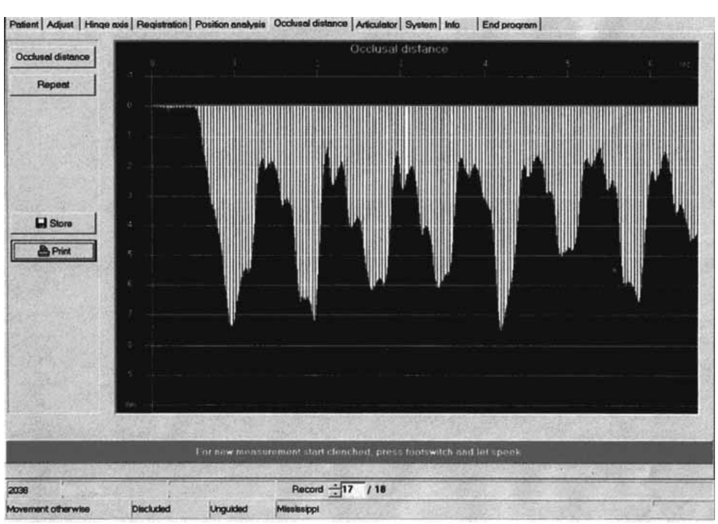

Fig. 3. Ocllusal distance.

TABLE 3
DIFFERENCES IN CONDYLOGRAPHIC MEASUREMENTS BETWEEN PATIENTS WITH BRUXISM AND WITHOUT BRUXISM

Average values (SD) of condylographic jaw movement variables in mm		Patients with bruxism N=22	Patients without bruxism N=20	t-value	p-value
CPA	RC – lateral shift	–0.05(0.12)	–0.01(0.07)	–1.375	0.177
	RC – saggital shift	–0.08(0.14)	–0.01(0.12)	–1.796	0.080
	RC – caudal shift	–0.16(0.13)	–0.19(0.15)	0.632	0.531
	RC – distraction	–0.16(0.12)	–0.16(0.11)	0.119	0.906
	RC – lateral shift	–0.02(0.12)	–0.01(0.09)	–0.371	0.713
	LC – medial shift	0.04(0.11)	0.01(0.07)	1.283	0.207
	LC – saggital shift	–0.08(0.18)	–0.04(0.17)	–0.705	0.485
	LC – caudal shift	–0.20(0.22)	0.20(0.14)	–0.054	0.957
	LC – distraction	–0.20(0.21)	0.15(0.10)	–0.929	0.358
	LC – medial shift	0.05(0.17)	0.01(0.13)	1.248	0.219

CPA – condyle position analysis; RC – right condyle; LC – left condyle

TABLE 4
DIFFERENCES IN INCLINATION OF CONDYLE PATH AND BENETT'S ANGLE BETWEEN PATIENTS WITH BRUXISM AND WITHOUT BRUXISM

Average values (SD) of other condylographic variables	Patients with bruxism N=21	Patients without bruxism N=20	t-value	p-value
Occlusal distance	3.67 (1.88)	2.05 (2.44)	2.385 *	0.022
Inclination of condyle path – left	39.38 (13.62)	41.55 (16.63)	0.458 n.s.	0.649
Inclination of condyle path – right	36.38 (9.30)	40.45 (14.78)	1.061 n.s.	0.295
Benett's angle – left	5.43 (6.13)	6.45 (6.91)	0.501 n.s.	0.619
Benett's angle – right	2.81 (3.86)	4.30 (4.84)	1.094 n.s.	0.281

* $P<0.05$

Statistical analysis of obtained data was performed by statistical package Statistica for Windows, Kernel release 5.5 A[24]. For comparison of continuous variables between groups Student t-test was used. Kolmogorov-Smirnov test was applied when the required conditions for Student's test were not fulfilled. Continuous variables were analysed by application of analysis of variance. Comparison of discrete variables between groups was performed by χ^2-test. Association of individual variables (discrete and continuous) was assessed by univariante and multivariate logistic regression analysis.

Results

Statistical significant difference was established between examinee groups with and without bruxism according to the anamnestic questionnaire for teeth grinding variable ($p<0.001$), while other variables and Helkim's index were not statistically significantly different between groups (Table 1). In the examined groups no statistical significant difference was determined in all three space levels, for condylographic measurement values of kinematic movements of the mandible (Table 2) (expressed in millimetres) and also for the difference between the centric relation of the condyle and habitual relation of the right and left condyle (Table 3).

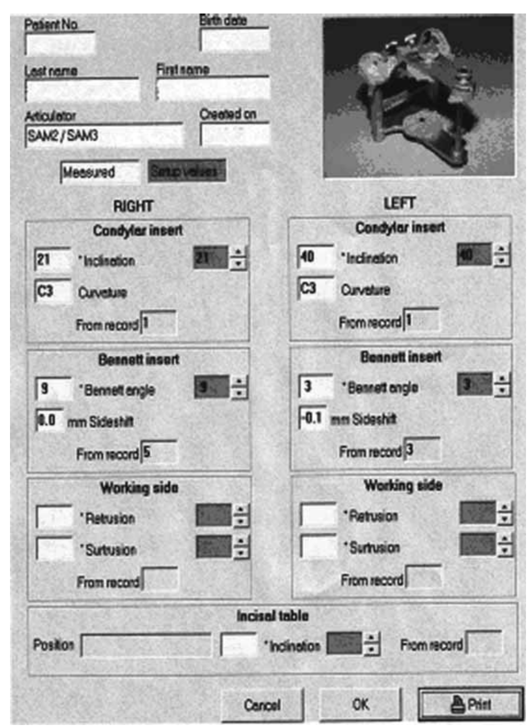

Fig. 4. Condyle inclination and Benett's angle.

TABLE 5
ANALYSIS OF POSSIBLE DENTAL TREATMENT IN RELATION TO SOME CONDYLOGRAPHIC VARIABLES

Etiologic TMD factors					Variance analysis	
Variables	Restorative dentistry	Orthodontics	Surgery	Loss of supporting zone	F	P
Opening – l – caudal (SD)	0.065 (0.163)	–0.197 (0.204)	–0.128 (0.065)	–0.039 (0.137)	3.161 *	0.037
D.mediotr.-r (SD)	0.198 (0.620)	–0.486 (0.543)	0.530 (0.680)	–0.119 (0.266)	5.963 ***	0.002
D.mediotr.-l (SD)	0.268 (0.545)	–0.254 (0.379)	0.252 (0.333)	–0.044 (0.258)	3.339 *	0.029
Cpa -r-lat (SD)	0.026 (0.107)	0.100 (0.113)	–0.044 (0.088)	–0.028 (0.096)	3.015 *	0.042
Cpa -l-dis (SD)	–0.35 (0.356)	–0.247 (0.149)	–0.134 (0.136)	–0.138 (0.093)	3.156 *	0.036
Inclination –condyle (SD)	49.250 (5.909)	53.333 (24.271)	43.400 (16.682)	35.538 (10.723)	3.463 *	0.026

Computer print of condyle path (issue from this research)
* P<0.05
*** P<0.01

Occlusal distance variable can differentiate the examined groups (p=0.022) and suggests that it can be significant diagnostic criterion for participation of TMD in bruxism patients. Differences between the two groups in inclination of the condyle path and Benett's angle were not statistically significant (Table 4). Only slight biological correlations between basic anamnestic and condylographic variables were observed (r<0.44; <19.3%).

No significant difference was observed between the examined groups with regard to palpatory examination of muscles, deviation and deflection of the jaw opening. Although slightly more frequent occurrence of intensive pain on palpation in patients with bruxism was noted. Bilaminare zone test showed a positive result (pain) in 90% of examinees in both groups. No statistically significant difference was established among the groups with regard to distribution of primary diagnosis according to muscle condition (χ^2=2.369, p=0.795). Diagnosis of neuromuscular dysfunction and myofascial pain dominated in both examinee groups. Neuromuscular dysfunction was more frequently found in patients with bruxism than in patients without bruxism.

Significantly more frequent disorders associated with disk condition were established in patients without bruxism (χ^2=11.072, p=0.049).

Significant differences between the groups of examinees with respect to primary diagnosis of the joint condition were established (χ^2=3.680, p=0.451). Dorsal and dorsocranial compression was present in both joints equally. In the group of patients with bruxism a statistically higher percentage of disturbances was established.

In the group without bruxism the values of certain condylographic variables were significantly different in relation to etiological factors of TMD (Table 5). Patients undergoing orthodontic treatment did not have a high incidence of bruxism but it was statistically insignificant.

Discriminante analysis of teeth grinding variable indicates that this variable discriminated the two examined groups. Arbitrary margin from 3 and more on a scale to 10, which represents the intensity of grinding, discriminated the two groups with accuracy of 85.7%, sensitivity of 77% and specificity of 95% (F=58.31; p<0.0001). Thus, anamnestic information on teeth grinding intensity represents a crucial index in diagnosis. When this is added to anamnestic information, grinding and depression evaluation, accuracy increases up to 88% (F=31.160, p<0.0001).

Multivariate discriminate analysis with gradient comprehension of new variables, isolates variables (digastric muscle, precisely their rear belly) in the palpatory clinical examination with other condylographic factors in combination with: *opn r sa* – saggital shift of the right condyle on opening movement, *opn l to* – overall shift of the left condyle on opening movement, *prot r s* – saggital shift of the right condyle on protrusion movement, *prot l c* – caudal shift of the left condyle on protrusion movement, *r med l s* – saggital shift of the left condyle on right mediotrusion which show statistical significance for classifying patients with bruxism from patients without bruxism (F=7.7281, p<0.0001). These factors are extracted in a model which shows accuracy of 89.5%, sensitivity of 90% and specificity of 89% for bruxism diagnosis in patients with TMD, which indicates the quality diagnostic compound factor. By a combination of more parameters, including clinical and condylographic parameters, significant diagnostic value was obtained despite an earlier analysis which showed statistically significant difference for only two variables between the examined groups.

Discussion

Criteria irregularity for evaluation of clinical signs and symptoms complicates diagnosis and TMD therapy. Subjective patient sensation, established via an anamnestic questionnaire on the existence and intensity of signs and symptoms is without question an important index of disorders. It must be followed by a clinical examination, particularly muscle palpation and manual functional analysis[25] and usage of instrumental registration systems (mechanical and electrical, magnetic, optoelectronic and ultrasonic). The significance of the aforementioned for TMD diagnosis is still disputable[26] or its efficacy is equal to standard techniques of manual clinical examination[27]. Osawa and Tanne[28] concluded that instrumental analysis, especially mechanical devices, is not *per se* a reliable index of precise TMD diagnosis, particularly of chronic or converted disorders than can serve as completion. The optoelectronic system functions without contact of both jaws. String Condylocomp LR3 tri-dimensionally and simultaneously shows the paths of both condyles, analyses their position (CPA), Benett's angle and other factors, although via software JAWS 30 (it can programme totally adaptive articulator CAR (Dentron))[29]. This determination of clinical centric is of exceptional therapeutic significance during treatment of the prosthodontic patient.

Correlation between bruxism and TMD has been established without doubt, although the entire mechanism is still unclear. Yamada and associates[30] showed that a large number of parafunctional habits indicates greater risk of condylar osseous changes (56.4%) and disk shift (59.6%). Molina and associates[31] carried out a similar study to the present study in a group of examinees with and without bruxism and established greater incidence of synovitis, retrodiskal pain and disk attachment pain in patients with TMD and bruxism and consequently they indicate close correlation bruxism and TMD ($p<0.05$). The greatest bruxism frequency was determined in patients with the same diagnoses as in the present study. Gonzalez[32] assumes that bruxism is more closely related to muscle disorders than to disk shifting and juncture disorders. It is unclear as to whether TMD patients in which a high level of bruxism occurs have more expressed signs and symptoms of TMD and teeth than patients with TMD in which a lower level of bruxism occurs. Pergamalian et al.[33] concluded that teeth abrasion is the same in patients with and without bruxism, and that the quantity of bruxistic activity is not connected with the severity of the muscular pain, but is connected with TMJ pain induced by palpation. In a study by Lobbezzo-Schooltea et al.[34] the incidence of disk shifting without reduction in patients with TMD is 6%, while Linde and associates found 36%[35]. The results of this study show incidence of anteromedial dislocation of the disk with repositioning of the right and left juncture in 45% of the TMD cases in patients without bruxism and 23% of those with bruxism. Also, the different incidence of dorsal and dorsocranial compression of the right and left junction in this study can be explained through the variations between the examined populations and diagnostic criteria which defines a certain disorder.

A study of TMD frequency in psychiatric patients[36], confirms the results of this study and indicates depression frequency in patients with bruxism. Depression variable was one of the most significant variables, apart from gnashing, which differentiated the two examined groups. In patients with myofascial pain depression is more dominant in patients with TMD pathology[37]. Velly et al.[40], while excluding psychological factors, suggest a connection between disk shifting and teeth grinding.

The significance of occlusal factors, particularly of attenuated vertical dimension of more than 2 mm[41], resulting in the loss of supporting zones[7], was previously established in TMD diagnostics[42], and was confirmed in this study. It is unclear as to why TMD is more frequent in females and whether it is due to a combination of biological (estrogens), psychological (explicit stress) or social factors including lower pain threshold[25]. It was also confirmed that patients with bruxism have more frequent muscular disorders (neuromuscular discoordination), while those without bruxism have more frequent disorders of the disk-condyle complex (dorsal, dorsocranial compression and anteromedial dislocation without repositioning) and finally differences exist in the symptomatics of TMD patients with and without bruxism.

Conclusions

1. Teeth grinding is a significant parameter in diagnosis of bruxism.
2. Helkimo index is not a relevant parameter in diagnosis of bruxism.
3. Statistically significant difference was not established between the groups of examinees with and without bruxism for condylographic measurement values of jaw movement (opening, protrusion, right and left mediotrusion).
4. The variable žocclusal distance' statistically significantly differentiated the examined groups and is significant diagnostic criteria for participation of TMD in bruxism patients.
5. Inclination of the condyle path and Benett's angle does not indicate statistically significant difference in dividing/differentiating the two examined groups.
6. Muscle palpation, deviation, deflexion and jaw opening did not show statistically significant difference between the examined groups, although a moderate increase was noticed in the examined group with bruxism.
7. In both examined groups a diagnosis of neuromuscular discoordination was dominant, although more in the bruxism group.
8. Dorsal and dorsocranial compression in both junctions was the same in both examined groups, with a higher percentage in examinees without bruxism, although this was not statistically significant.
9. Patients with bruxism more frequently do not have disk disorders.

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REFERENCES

1. American Academy of Orofacial Pain: Temporomandibular disorders: guidelines for classification, assessment and management.(Quintessence Publishing Co., Chicago, 1993). — 2. OKESON JP, Orofacial pain: guidelines for assessment, diagnosis and management. (Quintessence, Chicago 1996). — 3. DWORKIN SF, LE RESCHE L, J Craniomandib Disord, 6 (1992) 301. — 4. SELLIGMAN DA, PULLINGER AG, J Craniomandib Disord, 5 (1991) 96. — 5. KUTTILA M, LEBELLY P, ALENEN P, Acta Odontol Scand, 54 (1996) 332. — 6. DE BOEVER JA, ADRIANES PA, J Oral Rehabil, 10 (1983) 1. — 7. DULČIĆ N, PANDURIĆ J, KRALJEVIĆ S, Coll Antropol, 27 (2003) 61. — 8. RUGH JD, HARLAN J, J Adv Neurol, 49 (1988) 329. — 9. DWORKIN SF, J Craniomandib Disord, 6 (1992) 301. — 10. BADEL T, PANDURIĆ J, KRALJEVIĆ S, MAROTTI M, KOCIJAN L, ŠUTALO J, Acta Stomatol Croat, 37 (2003) 306. — 11. BASIĆ V, MEHULIĆ K, Acta Stomatol Croat, 38 (2004) 93. — 12. HANSSON T, NORDSTROM B, Acta Odontol Scand, 35 (1977) 281. — 13. THORPY MJ, J Clin Neurophysiol, 7(1990)67. — 14. SELIGMAN DA, PULINGER AG, J Craniomandib Disord Oral Facial Pain, 3 (1989) 227 — 15. BARTHLEN GM, STACY C, Mt Sinai J Med, 61 (1994) 139. — 16. DROUKAS B, LINDEE C, CARLSSON GE, J Prosthet Dent, 53 (1985) 402. — 17. RAMFJORD SP, ASH MM, Occlusion (WB Saunders, Philadelphia, 1972). — 18. BIONDI M, PICIARDI A, Psychother Psychosom, 59 (1993) 84. — 19. GOLA R, CHROSSEGROS C, ORTHLIEB JD, Sindrome algo-disfunzionale dell´apparato masticatorio (Masson, Milano, 1996). — 20. DAVIS M, Annu Rev Neurosci, 15 (1992) 333. — 21. GRABER G, Analisi funzionale della masticazione e terapia. In: SCHWENZER N (Ed) Protesica e scienza dei materiali.(Saronno (VA),1996). — 22. VANDERAS AP, MENENAKOU M, KOUIMTZIS T, J Oral Rehabil, 26 (1999) 103. — 23. AMIR I, HERMESH H, GAVIS A, Clin Neuropharmacol, 20 (1997) 86. — 24. Statistica for Windows, Kernel release 5.5 A (StatSoft, Inc. Tulsa, OK) (StatSoft, Inc. (2000). STATISTICA for Windows [Computer program manual], URL: http://www.statsoft.com). — 25. ČELIĆ R, Istraživanje temporomandibularnih poremećaja primjenom dijagnostičkih kriterija DKI/TMP protokola. MS Thesis. In Croat. (University of Zagreb, Zagreb 2003). — 26. LUND JP, WIDMER CG, FEINE JS, J Dent Res, 73 (1994) 1157. — 27. LANDES C, WALENDZIK H, KLEIN C, J Maxillofac Surg, 28 (2000) 352. — 28. OSAWA S, TANNE K, J Orofac Pain, 11 (1997) 222. — 29. KLETT R, Der Frei Zahnärzt, 3 (1996) 28. — 30. YAMADA K, HANADA K, FUKUI T, SATOU Y, OCHY K, HAYSHI T, Oral Surg Oral Med Oral Pathol Oral Radiol Endod, 92 (2001) 265. — 31. MOLINA OF, DOS SANTOS JUNIOR J, Cranio, 17 (1999) 17. — 32. GONZALEZ B, Int J Orthod Milwaukee, 18 (2007) 17. — 33. PERGAMALIAN A, RUDY TE, ZAKI HS, J Prosthet Dent, 90 (2003) 194. — 34. LOBBEZOO-SCHOLTE AM, DE LEEUW JRJ, STEENKS MH, BOSMAN F, BUCHNER R, OLTHOFF LW, J Orofac Pain, 9 (1995) 24. — 35. LINDE C, ISACSSON G, J Craniomandib Disord, 4 (1990) 197. — 36. KANEHIRA H, AGARIGUCHI A, KATO H, YOSHIMINE S, INOVE H, Nihon Hotetsu Shika Gakkai Zasshi, 52 (2008) 375. — 37. EVERSOLE LR, STONE CE, MATHESON D, Oral Surg Oral Med Oral Pathol, 60 (1985) 269. — 38. YAP AUJ, TAN KBC, CHUA EK, J Prosthet Dent, 88 (2002) 479. — 39. RUDY TE, TURK DC, KUBINSKI JA, J Orofac Pain, 61 (1995) 103. — 40. VELLY AM, GORNITSKY M, PHILIPPE P, J Oral Rehabil, 29 (2002) 408. — 41. SONNESEN L, SVENSSON P, Eur J Orthod, 30 (2008) 621. — 42. ĆELIĆ R, JEROLIMOV V, PANDURIĆ J, Int J Prosthodont, 15 (2002) 43.

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DIJAGNOSTIKA TEMPOROMANDIBULARNIH DISFUNKCIJA KOD PACIJENATA S BRUKSIZMOM OPTOELEKTRONIČKOM PANTOGRAFIJOM

SAŽETAK

Temporomandibularna disfunkcija je pojam koji obuhvaća mnogobrojne kliničke simptome koji zahvaćaju zube, žvačnu muskulaturu i čeljusne zglobove. Čest su uzrok orofacijalnih bolnih stanja. Etiologija disfunkcija je složena, a udio i značenje pojedinih etioloških čimbenika nedovoljno definirano. Bruksizam, bilo njegova centrična ili ekscentrična forma, sve je prisutniji problem u stomatološkoj praksi. Svrha ovog istraživanja je optoelektroničkom pantografijom prikazati čimbenike kondilarnog vođenja kod pacijenata s bruksizmom te utvrditi mogućnost uporabe optoelektroničke pantografije u dijagnostici TMD. U istraživanju su sudjelovali ciljano odabrani pacijenti (N=42); oba spola, nepotpunih zubnih nizova, bez protetskih radova, a sa znakovima i simptomima TMD-a. Nakon ispunjenog anamnestičkog upitnika te kliničkog pregleda i analize modela odabrani su pacijenti s bruksizmom (N=22) i oni bez bruksizma (N=20). U istraživanju se koristio optoelektronički String-condylocomp LR3, Dentron, D-Höchberg (software JAWS 30). Ovim radom je potvrđena mogućnost uporabe optoelektroničke pantografije u dijagnostici TMD-a, te uspoređuje anamnestičke, kliničke i kondilografske parametre kod TMD pacijenata s i bez bruksizma. Optoelektronička pantografija omogućava primjenom relativno jednostavne metode, precizniju i lakšu dijagnostiku navedenog poremećaja što može biti od izuzetne važnosti za odabir terapijskog postupka i kontrolu efikasnosti terapije.