



Temporomandibular Disorders: Association between Psychosocial Symptoms and Persistent Pain

Muhammad Wasim¹, Malik Mudasser Yasin², Shumaila Anjum³
University of Health Sciences, Lahore, Pakistan

Abstract: This longitudinal study of temporomandibular disorder (TMD) reports the association of psychosocial dysfunctions (depression, somatization without pain, somatization, and anxiety) with characteristic pain intensity (CPI), pain interference (PI), and number of disability days (DD) for subjects with TMD. Subjects (N=330) underwent a thorough series of assessments at baseline and follow-up (5-10 years later) to receive both Axis I and Axis II diagnoses per the Research Diagnostic Criteria for TMD (RDC/TMD). They reported their levels of CPI, PI, and DD at baseline and follow-up. Linear and log-binomial regression analyses were used to evaluate the change in CPI and PI and assess the risk of DD by baseline categories of psychosocial symptoms as measured by the Symptom Checklist (SCL-90). Linear regression analysis revealed that subjects with depression at baseline had higher PI at follow-up. Also, subjects with moderate to severe somatization with and without pain had higher CPI at follow-up than subjects without somatization. In conclusion, we found that psychosocial impairments (depression, somatization) were associated with increased characteristic of pain intensity, pain interference, and disability days at follow-up.

Keywords: Temporomandibular disorder, pain intensity, pain interference, number of disability days

Introduction

Temporomandibular disorder (TMD) is a term used to describe pain and dysfunction of the temporomandibular joint (TMJ) and muscles of mastication that control jaw motions (1). TMD consists of three subtypes and may affect one or more of these areas: myofascial (discomfort or pain in the muscles), internal derangement of the joint (displaced disc), and arthritis (degenerative/inflammatory joint disorders that affect TMJ) (2).

Pain is the primary characteristic of TMD and pain relief is the main reason for seeking treatment (3, 4). Other symptoms include disturbances in mandibular movement patterns, joints sound, and/or impairment in functional movement (2). TMD is considered to be one of the most common musculoskeletal conditions that cause pain and disability. Also, TMD pain is one of the three most prevalent types of chronic pain conditions globally after tension type headache and back pain (5, 6). TMD generally affects females more often than males, with ratios ranging from approximately 2:1 to 8:1 (7, 8). Most patients presenting with symptoms are between 20 and 50 years of age, but

frequency decreases after the age of 55 with an incidence rate of 4% across different populations (9, 10). The overall prevalence of TMD is reported to range from 4.6 to 15% (10, 11). Furthermore, a number of surveys have indicated that 20 to 25% of the United States (U.S.) population has experienced TMD-like pain; some estimates place the number of individuals suffering from TMD symptoms around 30 million, with 1 million cases diagnosed annually (10, 11, 12, 13).

Literature review

Etiology

The etiology of TMD is considered to be multidimensional, but the importance of individual factors is still unclear. These factors include, but are not limited to: biomechanical (occlusal overloading and parafunctions such as bruxism); biological (increased levels of estrogen hormones); medical conditions (back pain, fibromyalgia, sleep apnea, and rheumatoid arthritis); macro trauma (head trauma, old or recent motor vehicle accidents, sports injuries); and bio-psychosocial factors (stress, anxiety or depression) (14, 15, 16, 17, 18, 19, 20).

Treatment Options

The treatment of TMD is based on symptom management through conservative approaches, which show positive outcomes in the majority of patients.

Self-care: Self-care: includes muscle exercises, physical therapy, massage, and stretching; applying warm and cold pack on the affected muscles; eating soft foods and avoiding eating crunchy and “gummy” foods; and taking over-the-counter analgesics under a doctor’s supervision (21, 22, 23, 24,25). These are considered first-line treatments, and show high response rates, especially with patients who have no or low psychosocial aspects to their pain.

Oral Appliances (Splints): Different types of oral appliances exist, ranging from thin to thick, soft to hard, over-the-counter to lab-made, and those that reposition the mandible in one direction or another. The main purpose of such appliances is to reduce parafunctional jaw activities (clenching, bruxism), to protect teeth and reduce the working load on the condyles. The overall results of treatment with oral appliances are promising for the reduction of pain when used as adjunctive treatment along with self-



care (26, 27, 28). More comprehensive evidence-based reviews of splint therapy, however, have shown equivocal results (29, 30).

Arthrocentesis & Arthroscopy: Arthrocentesis and arthroscopy are considered the least invasive TMD surgical procedures. Arthrocentesis is usually suggested for sudden-onset, restricted jaw opening in patients with no significant history of TMJ problems. Arthroscopy is performed for a variety of purposes including returning the disc to a normal relationship with the condyle. It is not widely used and is usually used in cases of recurrent and prolonged displacement of the disc. While arthroscopic surgery and arthrocentesis may be performed to lubricate joint surfaces and reduce inflammation, more research is needed to identify long-term outcomes, particularly in the absence of disc repositioning or replacement (31, 32, 33).

Joint replacement and condyloectomy: Joint replacement and condyloectomy are surgical procedures used to address severe structural damage to the joint (condyle and fossa) (34).

Treatment outcome modifiers: Most patients, regardless of the subtype of TMD describe a favourable natural course of the disease, with self-limiting and fluctuating symptoms that often seem to respond well to nonspecific treatments (35, 36, 37). Some patients with TMD however, develop chronic pain representing a challenge for pain clinicians, especially because of the concurrent presence of psychosocial disorders and their relationship with pain (38).

Psychosocial factors have been implicated in the initiation as well as in the permanence of TMD (39). Depression, somatic distress, and anxiety may be potential etiological risk factors for TMD-related pain (40). The role of psychosocial factors in different stages of TMD has been intensively investigated, with equivocal results. Multiple studies indicate that psychosocial factors such as depression, stress, and anxiety play a role in the initiation and continuation of TMD as well as in patients' response to treatment (41, 42). The role of those factors diverges in different cases according to the TMD diagnostic subgroup (43, 44). For instance, when the duration of pain increases, psychosocial factors may become more prominent. Even after reducing the pain, pain behavior and affect associated with it may continue and in some case may worsen (45).

Although depression is more prevalent in patients with chronic pain, data concerning its comorbidity, especially in the chronic stage of pain, is diverse (46, 47, 48). Moreover, the role of anxiety in chronic pain is controversial. For instance, anxiety levels in patients with migraine and facial

pain is positively related to muscle tenderness. However, multiple studies have failed to find a link between TMD and anxiety (49, 50, 51, 52, 53).

The main research question is whether the course of TMD pain and related interference with daily activities in participants with psychosocial symptoms differs over a period of 5-10 years compared to participants without psychosocial symptoms in terms of 1) characteristic pain intensity (CPI), 2) pain interference (PI), and 3) disability days (DD). The research hypothesis of this research is that participants with psychosocial symptoms at baseline would have less improvement with regard to their TMD-related pain at follow-up compared with subjects who do not have psychosocial symptoms at baseline.

Methodology

This longitudinal research included subjects from the Research Diagnosis Criteria for Temporomandibular Disorder (RDC/TMD) Validation (baseline) and Impact (follow-up) projects. These multisite projects involve University of Health Sciences, King Edward Medical College, Allama Iqbal Medical College at Lahore, Pakistan. The RDC/TMD provides a dual-axis biopsychosocial diagnostic approach assessing both physical (Axis I) and psychosocial (Axis II) diagnoses. While the Axis I diagnoses involve different muscle and joint disorders, diagnosing Axis II conditions involves instruments for the evaluation of different psychosocial aspects of pain. The research is described in detail elsewhere (2). Of those 705 subjects (case and control) who were included in RDC/TMD at baseline, 330 (case) were included in the study because they met primary criteria of having TMD at baseline and completing their Chronic Graded Pain Scale (CGPS) scores in both times.

Measurement: Symptom Check List 90 (SCL-90)

The SCL-90 is a questionnaire commonly used for self-report of psychosocial distress. The SCL-90 is validated for individuals aged 13 years and above. It contains 90 items and takes 12 - 15 minutes complete (55). Each item consists of a question to rate a specific complaint (e.g., "In the last month, how much have you been distressed by: Feeling easily annoyed or irritated"), and the response is scored on a five-point scale (0 = not at all, 1 = slight, 2 = somewhat, 3 = high, 4 = extremely), producing nine categories of primary symptom dimensions. Each primary symptom was categorized into normal, moderate, and severe. In this research, the three primary symptom dimensions evaluated were depression, somatization with and without pain, and anxiety. A number of studies have been conducted demonstrating the reliability, validity, and use of



this instrument (56, 57). Also, a subtype of somatization (without pain) was assessed in order to differentiate between subjects who have general somatic complaints in their body that do not involve pain.

Results

The total number of subjects who met the inclusion and exclusion criteria was 330. Subjects were mainly female (85.8%) with a mean (SD) age of 37.95 (12.8) years (Table 1). Follow-up time ranged

from 5.75 to 10.7 years (mean (SD) = 7.88 (0.78) years).

Duration of facial pain for 267 subjects reporting pain at baseline ranged from 0 to 40 years (mean (SD) = 9.8 (9.38) years). Among the 330 subjects at baseline, 32.1% (n=105) had moderate to severe depression; 26.1% (n=86) had moderate to severe somatization without pain; 70.9% (n=234) had moderate to severe somatization with pain; and 20% (n=66) had moderate to severe anxiety (Table 2).

Table 1. Sociodemographic characteristics of subjects

	Number	Percentage
Sex		
Male	47	14.2
Female	283	85.8
Education		
10-12 years	4	1.2
13-15 years	135	40.8
16-17 years	105	31.8
18-19 years	56	17
20 years	29	8.8
Marital status		
Single	85	25.8
Married	144	43.6
Separated/divorced	45	13.6
Widowed	4	1.2
Household income		
<10k	50	15.2
10-39k	101	30.6
40-79k	114	34.5
>=80k	62	18.8
General Health	General Health	General Health
Excellent	Excellent	Excellent
Very good	Very good	Very good
Good	Good	Good
Poor	Poor	Poor
Oral Health		
Excellent	39	11.8
Very good	126	38.2
Good	110	33.3
Fair	12	3.6
Poor	43	13

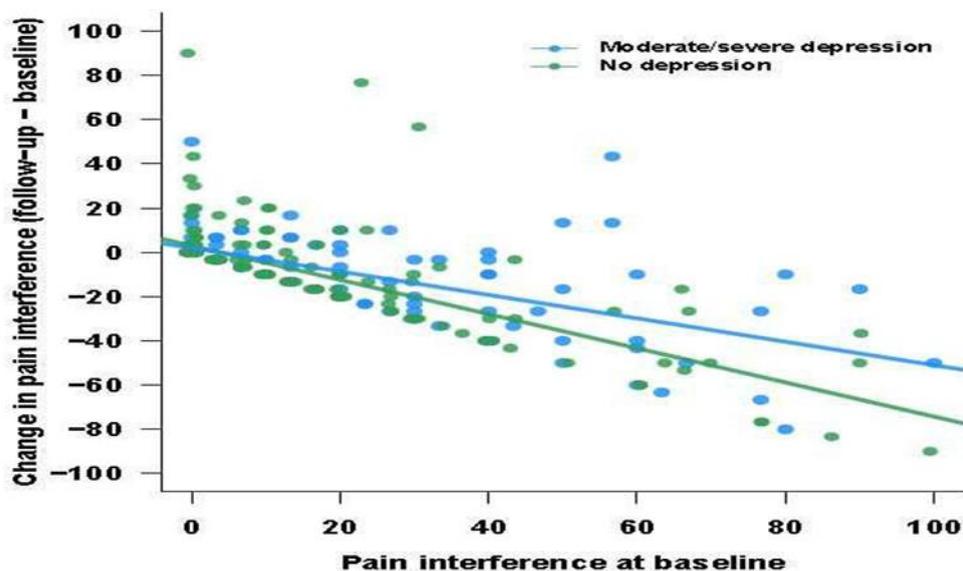
**Table 2.** Psychosocial symptoms at baseline

Psychosocial dysfunction	Moderate N (%)	Severe N (%)	Total N (%)
depression	73 (22.4)	32 (9.7)	105 (32.1)
somatization without pain	50 (15.2)	36 (10.9)	86 (26.1)
somatization with pain	92 (27.9)	142 (43)	234 (70.9)
anxiety	48 (14.5)	18 (5.5)	66 (20)

Depression

Depression was not associated with CPI at follow-up after adjusting for baseline pain intensity, or adjusting for gender, age, and follow-up duration (Table 3). The linear regression for depression, identified a significant interaction between depression and baseline PI (interaction estimate [SE] = 0.24 [0.08]; $p = .0014$) (Table 3). On average subjects had lower levels of PI at follow-up than baseline, but the reduction was smaller for subjects with moderate to severe depression than subjects without depression among subjects with elevated levels of PI at follow-up. For example,

among subjects with a baseline PI one standard deviation above the mean ($PI = 37.2$), subjects with moderate to severe depression had a significantly smaller reduction in PI at follow-up than subjects without depression (estimate [SE] = 7.65 [2.28]; $p = .0009$). Whereas among subjects with a baseline PI one standard deviation below the mean ($PI = 0$), there was no difference by depression in the reduction of PI (estimate [SE] = -1.35 [2.20]; $p = .54$) (Figure 1).

Figure 1. Interaction effect of baseline PI and depression on the change in PI

Subjects with moderate to severe depression were more likely to have 1 or more disability days at follow-up than subjects without depression (RR = 2.18; CI 95% 1.39 to 3.41; $p = .0006$). However, the increased risk was no longer statistically significant after controlling for baseline disability days, gender, age, and follow-up time (adjusted RR = 1.50; 95% CI 0.96 to 2.33; $p = .071$) (Table 4).

Somatization without Pain

For somatization without pain, subjects with moderate to severe somatization had higher CPI at follow-up than subjects without somatization without pain (normal), after adjusting for baseline somatization, gender, age, and follow-up time [estimate (SE) = 5.27 (2.28); $p = .022$] (Table 3). For subjects with baseline CPI one standard deviation below the sample mean there was no significant interactions $p = 0.85$. Somatization was not associated with pain interference at follow-up



after adjusting for baseline pain interference, or adjusting for gender, age, and follow-up duration.

There was a significant interaction between somatization without pain and having any disability days at baseline ($p = .029$). Among subjects without any disability days at baseline, subjects with moderate to severe somatization without pain were more likely to have 1 or more disability days at follow-up (RR(95%CI) = 2.94 (1.45, 5.92) p -value = .0026). In contrast, for subjects with 1 or more disability days at baseline, the risk of 1 or more disability days at follow-up was similar for subjects with and without somatization without pain (RR (95% CI) = 1.14 (0.67, 1.90); $p = .63$) (Tables 4)

Somatization with Pain

For somatization with pain, subjects with moderate to severe somatization had higher pain intensity at follow-up than subjects without somatization (normal), after adjusting for baseline somatization, gender, age, and follow-up time [estimate (SE) = 4.55 (2.4), $p = .059$]. Somatization was not associated with pain interference or disability days at follow-up (Tables 3 and 4).

Anxiety

Anxiety at baseline was not predictive of CPI [estimate (SE) = -0.32 (2.5), $p = .90$], PI [estimate (SE) = -0.94 (2.07), $p = .65$], and DD (RR (95%CI) = 1.34 (0.82, 2.16), $p = .23$) at follow-up (Tables 3 and 4).

Table 3. Linear regression results for CPI and PI scores (Model 4)

Variables	depression		somatization without pain		somatization with pain		anxiety	
	Estimate (SE)	P-value	Estimate (SE)	P-value	Estimate (SE)	P-value	Estimate (SE)	P-value
Characteristic								
Pain Intensity								
Psychosocial variables	1.38 (2.33)	.53	5.27 (2.28)	0.021	4.55 (2.62)	.059	-0.32 (2.5)	.90
Baseline CPI	-0.60 (0.04)	<.0001	-0.61 (0.04)	<.0001	-0.63 (0.04)	<.0001	-0.60 (0.4)	<.0001
PI Score								
Psychosocial	2.96 (1.78)	.096*	2.34 (1.88)	.22	1.95 (1.87)	.30	-0.94 (2.07)	.65
Baseline PI	-0.68 (0.04)	<.0001	-0.67 (0.04)	<.0001	-0.68 (0.04)	<.0001	-0.66 (0.04)	<.0001

*There was significant interaction between baseline depression and baseline PI, $p = .0014$.

Table 4. Log-binomial results for any disability days (Model 4)

Variable	depression			somatization w/o pain			somatization with pain			anxiety		
	RR	95% CI	P-value	RR	95% CI	P-value	RR	95% CI	P-value	RR	95% CI	P-value
Any disability days												
Psychosocial	1.5	(0.96, 2.33)	.071	1.70	(1.06, 2.71)	.027	2.05	(0.87, 4.82)	.097	1.34	(0.82, 2.16)	.23
Any DD at baseline	3.1	(1.97, 5.0)	<.001	3.04	(1.87, 4.93)	<.001	2.94	(1.84, 4.67)	<.001	3.21	(1.98, 5.21)	<.0001

*There was significant interaction between baseline somatization without pain and any disability days at baseline, $p = .029$.



The associations between depression and somatization with psychosocial symptoms remained significant after adjusting for the other psychosocial symptoms (Tables 5 and 6). Interestingly, anxiety at baseline was not predictive of pain intensity, pain interference or disability days at follow-up (Tables 3 and 4), but after adjusting for depression and somatization without

pain, there was a significant interaction between anxiety and baseline CPI ($p = .035$) with an unexpected effect. Among subjects with a baseline CPI one standard deviation above the mean (CPI = 65.9), subjects with moderate to severe anxiety had a significantly greater reduction in CPI at follow-up than subjects without anxiety [estimate (SE) = -8.77 (3.77); $p = .021$].

Table 5. Linear regression results with all 3 psychosocial variables included in the regression model (Model 4)

Variable	Characteristic pain intensity		Pain interference		Any disability days	
	Estimate (SE)	P-value	Estimate (SE)	P-value	RR (95% CI)	P-value
Baseline	-0.61 (0.04)	<.0001	-0.67 (0.04)	<.0001	3.33 (2.04, 5.40)	<.000
depression	0.21 (2.63)	.94	4.03 (2.18)	.065 ³	1.27 (0.73, 2.21)	.40
somatization (without pain items)	6.71 (2.66)	.012 ¹	2.26 (2.20)	.31	1.79 (1.05, 3.04)	.031
anxiety	-3.71 (3.03)	.22 ²	-4.54 (2.52)	.073	0.89 (0.50, 1.58)	.69

*There was significant interaction between baseline somatization and baseline CPI, $p = .044$.

*There was significant interaction between baseline anxiety and baseline CPI, $p = .035$

*There was significant interaction between baseline depression and baseline PI, $p = .0006$

Table 6. Regression results with all 3 psychosocial variables included in the regression model (Model 4)

Variable	Characteristic pain intensity		Pain interference		Any disability days	
	Estimate (SE)	P-value	Estimate (SE)	P-value	RR (95% CI)	P-value
Baseline	-0.63 (0.04)	<.0001	-0.68 (0.04)	<.0001	2.80 (1.72, 4.52)	<.0001
depression	1.53 (2.56)	.55	4.43 (2.11)	.037	1.35 (0.78, 2.33)	.28
somatization (with pain items)	4.56 (2.46)	.064	1.60 (1.92)	.41	1.86 (0.78, 4.42)	.16
anxiety	-2.01 (2.92)	.49	-3.40 (2.43)	.10	1.04 (0.58, 1.83)	.90

*There was significant interaction between baseline depression and baseline PI, $p = .0003$

Discussion

The aim of this paper was to determine if psychosocial symptoms at baseline were associated with a change in the course of pain and its associated disability from baseline to follow-up. This prospective research reports the prediction of pain outcomes by using baseline psychosocial dysfunctions (depression, somatization, anxiety) and Chronic Graded Pain Scale (CGPS) in patients in three different demographic areas (WA, NY,

MN) who were diagnosed with Temporomandibular Disorder (TMD) in terms of characteristics of pain intensity (CPI), pain interference (PI), and disability days (DD).

The results of this research support the importance of psychological screening of TMD patients in order to evaluate the risks of depression and somatization that may influence treatment (58). Our findings indicate that a psychosocial assessment of TMD patients may be important as a



physical evaluation and may be more important when predicting pain outcomes. Our findings suggest that the clinical relevance of measuring psychosocial dysfunction cannot be underestimated (57).

The present research performed by means of a prospective analysis of RDC/TMD findings, suggests that there might be a significant

correlation between psychosocial impairments (depression, somatization with and without pain) and characteristic pain intensity, pain interference, and disability days at baseline. We found that psychosocial impairments (depression, somatization with and without pain) were associated with increased of characteristic pain intensity, pain interference, and disability days.

References

1. TMJ Disorders - NIDCR Home. National institute of dental & craniofacial research. <http://nidcr.nih.gov/oralhealth/topics/tmj/tmjdisorders.htm>. Accessed October 23, 2016.
2. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: Recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Group†. *Journal of Oral & Facial Pain and Headache*. 2014;28(1):6-27. doi:10.11607/jop.1151.
3. Visscher CM, Lobbezoo F, de Boer W, van derMeulen M, Naeije M. Psychological distress in chronic craniomandibular and cervical spinal pain patients. *Eur J Oral Sci*. 2001; 109:165–171.
4. Dworkin, S.F. and LeResche, L., Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique, *J. Craniomandib. Disord. Fac. Oral Pain*, 6 (1992) 301–355
5. National Institute of Dental and Craniofacial Research [7/28/2013]; Facial Pain. <http://www.nidcr.nih.gov/DataStatistics/FindDataByTopic/FacialPain/> [Ref list]
6. Maísa Soares G, Rizzatti-Barbosa CM. Chronicity factors of temporomandibular disorders: A critical review of the literature. *Braz Oral Res*. 2015;29: pii: S1806-83242015000100300
7. Prevalence of mandibular dysfunction in young adults. Solberg WK, Woo MW, Houston JBJ *Am Dent Assoc*. 1979 Jan; 98(1):25-34
8. Martins-Junior RL, Palma AJ, Marquardt EJ, Gondin TM, de Kerber FC. Temporomandibular disorders: a report of 124 patients. *J Contemp Dent Pract*. 2010; 11:071–8.
9. Van Loon JP, de Bont LG, Stegenga B, Spijkervet FK, Verkerke GJ. Groningen temporomandibular joint prosthesis. Development and first clinical application. *Int J Oral Maxillofac Surg*. 2002;31:44–52.
10. Isong U, Gansky SA, Plesh O. Temporomandibular joint and muscle disorder-type pain in U.S. adults: the National Health Interview Survey. *J Orofac Pain* 2008; 22: 317–322.
11. Solberg WK, Woo MW, Houston JB. Prevalence of mandibular dysfunction in young adults. *J Am Dent Assoc*. 1979;98:25–34.
12. Carlsson GE, LeResche L. Epidemiology of temporomandibular disorders. In: Sessle BJ, Bryant P, Dionne R, editors. *Temporomandibular disorders and related pain conditions*. Seattle: IASP Press; 1995. pp. 497–506
13. Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. *J Am Dent Assoc*. 1993;124:115–21.
14. Kirveskari P, Alanen P, Jämsä T. Association between craniomandibular disorders and occlusal interferences in children. *J Prosthet Dent*. 1992;67:692–6.
15. Oral K, Bal Küçük B, Ebeoglu B, Dinçer S. Etiology of temporomandibular disorder pain. *Agri*.2009;21:89–94.
16. Furquim BD, Flamengui LM, Conti PC. TMD and chronic pain: A current view. *Dental Press J Orthod*.2015;20:127–33.
17. Kolbinson DA, Epstein JB, Burgess JA, Senthilselvan A. Temporomandibular disorders, headaches, and neck pain after motor vehicle accidents: A pilot investigation of persistence and litigation effects. *J Prosthet Dent*. 1997;77:46–53.
18. Grzesiak RC. Psychologic considerations in temporomandibular dysfunction. A biopsychosocial view of symptom formation. *Dent Clin North Am*. 1991;35:209–26.



19. Shaefer JR, Holland N, Whelan JS, Velly AM. Pain and temporomandibular disorders: A pharmaco-gender dilemma. *Dent Clin North Am.* 2013;57:233–62.
20. Christensen L, Luther F. Adults seeking orthodontic treatment: Expectations, periodontal and TMD issues. *Br Dent J.* 2015;218:111–7.
21. McNeely ML, Armijo Olivo S, Magee DJ. A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. *Phys Ther.* 2006;86:710–25.
22. Treacy K. Awareness/relaxation training and transcutaneous electrical neural stimulation in the treatment of bruxism. *J Oral Rehabil.* 1999;26:280–7.
23. Friction JR. Management of masticatory myofascial pain. *Semin Orthod.* 1995;1:229–43.
24. Dimitroulis G, Gremillion HA, Dolwick MF, Walter JH. Temporomandibular disorders. 2. Non-surgical treatment. *Aust Dent J.* 1995;40:372–6.
25. Dionne RA. Pharmacologic treatments for temporomandibular disorders. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997;83:134–142.
26. Kalamir, A., H. Pollard, A. L. Vitiello, and R. Bonello. TMD and the problem of bruxism—a review. *J. Bodywork Mov. Ther.* 11:183–193, 2007. doi:10.1016/j.jbmt.2006.11.006.
27. Glaros, A. G., Z. Owais, and L. Lausten. Reduction in parafunctional activity: a potential mechanism for the effectiveness of splint therapy. *J. Oral Rehabil.* 34:97–104, 2007. doi:10.1111/j.1365-2842.2006.01660.x.
28. Glass, E. G., A. G. Glaros, and F. D. McGlynn. Myofascial pain dysfunction: treatments used by ADA members. *Cranio* 11:25–29, 1993.
29. Forssell, H., and E. Kalso. Application of principles of evidence-based medicine to occlusal treatment for temporomandibular disorders: are there lessons to be learned? *J. Orofac. Pain* 18:9–22, 2004; discussion 23–32.
30. Kreiner, M., E. Betancor, and G. T. Clark. Occlusal stabilization appliances. Evidence of their efficacy. *J. Am. Dent. Assoc.* 132:770–777, 2001.
31. Nitzan DW, Price A. The use of arthrocentesis for the treatment of osteoarthritic temporomandibular joints. *J Oral Maxillofac Surg.* 2001;59:1154–9. discussion 1160.
32. Holmlund A, Hellsing G, Bang G. Arthroscopy of the rabbit temporomandibular joint. *Int J Oral Maxillofac Surg.* 1986;15:170–5.
33. Holmlund A. Diagnostic TMJ arthroscopy. *Oral Surg Oral Diagn.* 1992;3:13–8.
34. Dolwick MF. The role of temporomandibular joint surgery in the treatment of patients with internal derangement. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997;83:150–5.
35. Rammelsberg P, Leresche L, Dworkin SF, Mancl L. Longitudinal outcome of temporomandibular disorders: a 5-year epidemiologic study of muscle disorders defined by research diagnostic criteria for temporomandibular disorders. *J Orofac Pain* 2003;17: 9–20.
36. Kalaykova S, Lobbezoo F, Naeije M. Twoyear natural course of anterior disc displacement with reduction. *J Orofac Pain* 2010;24:373–8.
37. Manfredini D. Fundamentals of TMD management. In: Manfredini D, editor. *Current concepts on temporomandibular disorders.* Berlin: Quintessence Publishing; 2010 . p. 305– 18.
38. Dworkin SF, Massoth DL. Temporomandibular disorders and chronic pain: disease or illness? *J Prosthet Dent* 1994;72:29–38.
39. Yap AUJ, Tan KBC, Chua EK, Tan HH. depression and somatization in patients with temporomandibular disorders. *J Prost Dent.* 2002;88:479–484. doi: 10.1067/mpr.2002.129375
40. Zakrzewska JM, Harrison SH. Assessment and management of orofacial pain. *Pain research and clinical management.* Amsterdam. Elsevier; 2002
41. Sipila K, Veijola J, Jokelainen J, Jarvelin MR, Oikarinen KS, Raustia AM, et al. Association between symptoms of temporomandibular disorders and depression: an epidemiological study of the Northern Finland 1966 Birth Cohort. *Cranio* 2001;19:183–7.
42. Glaros AG, Lumley MA. Alexithymia and pain in temporomandibular disorder. *Journal of Psychosomatic Research* 2005;59:85–8.



43. Auerbach SM, Laskin DM, Frantsve LM, Orr T. depression, pain, exposure to stressful life events, and long-term outcomes in temporomandibular disorder patients. *Journal of Oral and Maxillofacial Surgery* 2001;59:628–33. (discussion 34).
44. Lindroth JE, Schmidt JE, Carlson CR. A comparison between masticatory muscle pain patients and intracapsular pain patients on behavioral and psychosocial domains. *Journal of Orofacial Pain* 2002;16:277–83.
45. Okeson JP. *Bell's orofacial pains*. Sixth. Chigago: Quintessence Publishing; 2005.
46. Turner JA, Dworkin SF. Screening for psychosocial risk factors in patients with chronic orofacial pain: recent advances. *Journal of the American Dental Association* 2004;135:1119– 25. (quiz 64–5).
47. Korszun A, Hinderstein B, Wong M. Comorbidity of depression with chronic facial pain and temporomandibular disorders. *Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontics* 1996;82:496–500.
48. Gerschman JA, Wright JL, Hall WD, Reade PC, Burrows GD, Holwill BJ. Comparisons of psychological and social factors in patients with chronic oro-facial pain and dental phobic disorders. *Australian Dental Journal* 1987;32:331–5.
49. Mongini F, Ciccone G, Deregibus A, Ferrero L, Mongini T. Muscle tenderness in different headache types and its relation to anxiety and depression. *Pain* 2004;112:59–64.
50. Mongini F, Ciccone G, Ceccarelli M, Baldi I, Ferrero L. Muscle tenderness in different types of facial pain and its relation to anxiety and depression: a cross-sectional study on 649 patients. *Pain* 2007;131:106–11.
51. Wright AR, Gatchel RJ, Wildenstein L, Riggs R, Buschang P, Ellis 3rd E. Biopsychosocial differences between high-risk and low-risk patients with acute TMD-related pain. *Journal of the American Dental Association* 2004;135:474–83.
52. Vassend O, Krogstad BS, Dahl BL. Negative affectivity, somatic complaints, and symptoms of temporomandibular disorders. *Journal of Psychosomatic Research* 1995;39:889–99.
53. Friction JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. *Oral Surgery Oral Medicine and Oral Pathology* 1985;60:615–23.
54. Korff MV, Dworkin SF, Resche LL. Graded chronic pain status: an epidemiologic evaluation. *Pain*. 1990;40(3):279-291. doi:10.1016/0304-3959(90)91125-3.
55. Derogatis LR, Cleary PA. Confirmation of the dimensional structure of the scl-90: A study in construct validation. *Journal of Clinical Psychology J Clin Psychol*. 1977;33(4):981-989.doi:10.1002/1097-4679(197710)33-4
56. Arrindell W, Ettema J. *Handleiding bij een multidisciplinaire psychopathologie-indicator*. Lisse: Swets & Zeitlinger; 1986.
57. Bech P, Bille J, Møller S, Hellström L, Østergaard S. Psychometric validation of the Hopkins Symptom Checklist (SCL-90) subscales for depression, anxiety, and interpersonal sensitivity. *Journal of Affective Disorders*. 2014;160:98-103.
58. Dworkin SF, Von Korff M, LeResche L. Multiple pains and psychiatric disturbance. An epidemiological investigation. *Arch Gen Psychiatry* 1990; 47:239-44.