The Relationships Among Depression, Pain, and Masticatory Functioning in Temporomandibular Disorder Patients

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Aims: *To evaluate the effect of comorbid depression and pain on an early biopsychosocial intervention for acute temporomandibular disorder (TMD) patients.* ***Methods:*** *Depressed (either current or lifetime; n = 32) or nondepressed (n = 31) acute TMD patients received a biopsychosocial intervention, and were evaluated at preintervention and again 12 months postintervention by Characteristic Pain Intensity, the Beck Depression Inventory, and a masticatory function test.* ***Results:*** *Findings revealed that both depressed and nondepressed patients reported comparable pain decreases at 12 months postintervention. Moreover, there were no significant differences between patient groups in masticatory function.* ***Conclusion:*** *With appropriate early biopsychosocial intervention, acute TMD patients, regardless of the presence or absence of vulnerability to depression symptomatology, can be effectively treated.* J OROFAC PAIN 2006;20:288–296

Key words: biopsychosocial, depression, intervention, masticatory function, pain, temporomandibular disorders

Gatchel[1] and Gremillion[2] have found that the most frequently cited reason for patients in the United States to seek medical or dental care is pain. Moreover, numerous studies have discovered a high rate of psychiatric disorders among chronic temporomandibular disorder (TMD) patients, with depression as the most commonly occurring.[3–8] It has been found that most cases of depression are treated in primary care settings,[9] as are a substantial majority of pain patients.[10] Although both depression and pain are known to be independently associated with a decrease in quality of life and increased somatic preoccupation,[11] there has been little research on the association between these conditions in the evaluation of clinical outcomes in an acute care arena.

The progression from acute to chronic pain syndromes such as TMD has been characterized by means of a 3-stage model.[12,13] In stage 1, referred to as the acute phase, normal emotional reactions, such as fear, anxiety, and worry, develop subsequent to the patient's perception of pain. If pain is not dealt with effectively at this stage, then it will progress into stage 2, where the pain begins to develop into a subacute condition in which psychological and behavioral problems are often exacerbated. Learned helplessness, anger, distress, and somatization are typical evolving symptoms for patients at this stage. Finally, stage 3, which represents the chronic phase of the model, is characterized by the progression to complex interactions of physical, psychological, and social processes. As the result of the chronic nature of the pain experience

and the stress that it creates, the patient's life begins to revolve around the pain and behaviors that maintain it. Turk and Monarch[14] have delineated the common affective factors associated with pain: depression/learned helplessness; anxiety/pain-related fear; and anger/frustration. Though these symptoms often initially surface during stage 2, they become more entrenched during this later stage.

Gatchel[1] has discussed and reviewed the significant advantages of early intervention for acute pain in order to prevent the development of these chronic pain and disability problems. Indeed, there have been a number of preliminary studies suggesting the success of such an approach.[1] One such study[15] has demonstrated both the treatment- and cost-effectiveness of early intervention for acute low back pain. Findings from that study demonstrated that acute low back pain patients who received early intervention displayed significantly fewer indications of chronic pain disability at a 1-year follow-up relative to those patients who did not receive early intervention. Moreover, there was a significant cost savings for the early intervention program. Thus, these results have major implications in terms of decreasing emotional distress and producing socioeconomic cost savings for the prevalent problem of acute low back pain disability. More research of this type, with other types of pain syndromes, is certainly needed because of the potential to save chronic pain patients both treatment costs and emotional distress.

Recent research has demonstrated that a combined biopsychosocial treatment, including muscle relaxation training, biofeedback, and cognitive-behavioral skills training, is effective with TMD patients.[16–18] More recently, a just-published randomized controlled trial[19] has also found that participation in a 6-session intervention aimed at increasing insight and awareness into the mind-body connection via education, instruction on self-regulating pain management techniques, stress management instruction, and communication skills training positively affected outcomes. At a 1-year post-intake evaluation, the intervention group fared far better in terms of pain and overall emotional functioning than a control group, thus demonstrating that a comprehensive, biopsychosocial approach can result in the most effective and successful long-term outcomes for TMD sufferers.

Another important quality of life measure that might be expected to be related with depression and pain in TMD patients is masticatory function. Mastication is the first stage of the digestive process, when foods are physically broken down into smaller particles. It is a complex task with many anatomical, physiological, and psychological determinants. The sum of all the determinants is the patient's ability to break down food (ie, masticatory performance). Masticatory performance is related to quality of life through its influence on dietary selections, quality of digestion, and the level of enjoyment experienced while eating. Smaller particle sizes facilitate enzymatic processing during later stages of digestion. Assessments of masticatory performance have been advanced by the development of standardized techniques, artificial foods, and mathematical tools that allow greater precision in measuring performance. Impairment of masticatory performance among the elderly with mutilated dentition has been linked with food choices, levels of enjoyment experienced, nutritional deficiencies, and gastrointestinal disturbances.[20,21]

While relationships between masticatory function and TMD have been well established, the effects of therapy on masticatory performance remain poorly understood. Range of motion, which is among the best studied functional measures, is typically reduced in subjects with TMD. Individuals with TMD also have decreased bite forces prior to treatment but may show improvements that closely approximate normal values following successful treatment.[22,23] The level of activity of the masticatory muscles may be higher than normal at rest[24] in TMD patients but lower than normal during maximal clenching.[25,26] Despite the fact that absolute bite forces and muscle activity levels may be lower, subjects with TMD have to use greater relative bite forces to masticate foods than normal subjects.[27] While it has been reported that pain associated with TMD results in reduced masticatory ability,[22,28] only a couple of studies have objectively shown that patients with TMD also have reduced ability to break down foods.[29,30] Tzakis and coworkers[29] showed an 11% improvement in masticatory efficiency among 12 patients after 1 month of therapy. More studies are needed to establish if and how treatment affects masticatory performance.

The major goal of the present study was to evaluate the relationship between self-reported pain and depression following treatment of acute TMD pain patients. This study was part of the larger investigation on early biopsychosocial intervention for TMD reviewed earlier.[19] A secondary goal was to evaluate changes on a functional chewing performance evaluation undertaken pre- and post-treatment. Individuals with pain and/or depression were expected to demonstrate compromised func-

tioning in a variety of areas. For example, a person with depression may exhibit psychomotor retardation, fatigue or loss of energy, and compromised concentration. Consequently, it was hypothesized that a person with depression may be susceptible to compromised chewing functioning as a manifestation of the psychomotor slowing that is frequently associated with depression. Likewise, TMD-related pain may also compromise chewing performance.

Materials and Methods

Subjects

As part of a larger, randomized controlled trial,[19] dentists and oral surgeons in the Dallas/Fort Worth area referred subjects to the TMD Clinical Treatment Program at the University of Texas Southwestern Medical Center at Dallas. Additionally, to recruit subjects, flyers were posted at local universities, and advertising was placed in local newspapers. Subjects were required to be adults between the ages of 18 and 70 who had had acute jaw pain for less than 6 months. Potential subjects were excluded if they had some other chronic, significant pain-exacerbating physical condition (such as cancer or fibromyalgia) or a past history of jaw pain. Sixty-three subjects with complaints of jaw pain or discomfort of less than 6 months' duration were assessed. All subjects signed a consent form in accordance with the University of Texas Southwestern Medical Center at Dallas Institutional Review Board guidelines. Of the subject population (n = 63), 79.4% were female and 20.6% were male. The average age of the female subjects was 36.84 years old, and the average age for the male subjects was 36.53 years old.

General Information Questionnaire. Each subject completed a general information form. Subjects reported information, including name, gender, address, home and work contact numbers, and 3 personal references to aid in contact at follow-up intervals. Socioeconomic information, such as educational level and occupation, were included, as well as marital status and referral source. Subjects were also asked to report any pending workers' compensation or personal injury claims. Questions concerning overall health and medication were addressed. Specifically, subjects reported TMD-related information such as onset of symptoms, assumed cause, and any previous treatment history.

Physical Measures

Brief Jaw Pain Evaluation. Axis I Group Ia assessment items from the Research Diagnostic Criteria for TMD (TDC/TMD) were used to evaluate the subjects. The RDC/TMD, edited by Dworkin and LeResche,[4] was used to score both the TMD Examination Form and the History Questionnaire. The RDC/TMD are designed to determine whether there a muscle disorder or other joint condition is present. The RDC/TMD provides 2 additional scores: the Characteristic Pain Intensity (CPI) and the Graded Classification of Pain Score (GCPS). During this examination, the subjects' response to question 3 on the RDC/TMD history questionnaire ("Have you had pain in the face, jaw, temple, in front of the ear, or in the ear in the past month?") was used to determine the presence or absence of myofascial pain.[4]

Evaluation of Median Particle Size and Broadness of the Distribution. Standardized tablets (5 mm thick and 20 mm in diameter) of Cuttersil (Heraeus Kulzer, Hanau, Germany), a condensation silicone impression material, were formed using a Plexiglas template. After they had hardened for at least 1 hour, the tablets were cut into quarters. Five portions, containing 3 quarter-tablets each, were packaged for each subject.[31] Each subject was instructed to chew 3 of the quarter-tablets naturally for a total of 20 chews. The investigator counted the number of chews and timed each subject's chewing sequence. At the end of the 20th chew, subjects were instructed to stop chewing, expectorate the sample into a stainless-steel filter, and rinse with water until all particles were removed from the mouth. Rinsings were also collected in the filter. The procedure was repeated 4 times until approximately 10 g of Cuttersil had been chewed and expectorated into the filter. The subjects were instructed to rest between trials if they felt any fatigue.

The chewed samples were then air dried in filter paper over a stainless-steel colander. The samples were then separated using a series of 7 sieves, with mesh sizes 5.6 mm, 4.0 mm, 2.8 mm, 2.0 mm, 0.85 mm, 0.425 mm, and 0.25 mm, stacked on a mechanical shaker and vibrated for 2 minutes. Once the sample had been separated, the contents of each sieve were weighed to the nearest 0.01 g.

Cumulative weight percentages (defined by the amount of sample that could pass through each successive sieve) were calculated for each chewed sample. From these percentages, the median particle size (MPS) and broadness of particle distribution were estimated using the Rosin-Rammler

Table 1 No. of TMD Patients for Whom Data Were Collected by Group and Measure

Measure	Group	Intake	Last session of intervention	3 mo	6 mo	9 mo	12 mo	Final 12 mo no. analyzed†
CPI	Depressed	32	25	26	27	27	29	32
	Nondepressed	31	22	20	22	23	27	31
BDI*	Depressed	32	–	–	–	–	29	32
	Nondepressed	31	–	–	–	–	27	31
MPS	Depressed	29	–	–	–	–	16	29
	Nondepressed	27	–	–	–	–	16	27

*Beck Depression Inventory.[35]
†Last observation carried forward (LCOF) method for imputation of data used to allow for complete data set at 12-month follow-up.[36]
–No attempt was made to collect this data at this point.

equation.[32–34] It should be noted that, because of intermittent equipment problems, as well as the loss of patients to follow-up, complete pre- to posttreatment MPS data could not be obtained for all subjects. Three members of the depressed group and 4 members of the nondepressed group did not have pretreatment MPS data and therefore could not be imputed for subsequent analyses. This resulted in a reduced sample size for this measure (Table 1). There were no demographic differences between those who were analyzed versus those who were not.

Psychosocial Measures

For purposes of this study, the subjects completed the Structured Clinical Interview for the Diagnostic and Statistical Manual (DSM)-IV Axis I and Axis II Disorders (SCID-I[37]) and the BDI-II.[35]

SCID-I. This methodology is designed to determine the presence or absence of DSM-IV Axis I or Axis II disorders.[37] Disorders such as substance dependence, somatization, depression, and anxiety are diagnosed on Axis I. Disorders that are considered long-standing characteristics and traits that are maladaptive or disruptive interpersonally, such as personality disorders, are diagnosed on Axis II. The SCID-I and SCID-II are administered orally in the form of an in-depth flowchart that leads to diagnoses of particular Axis I and/or Axis II disorders, based on the examinee's item responses. It takes approximately 1 to 2 hours to administer both the SCID-I and SCID-II. Research has found the interrater reliability to be satisfactory, with an 86% agreement for generalized anxiety disorder and 82% for major depressive disorders.[38] The test-retest reliability of the SCID has also been shown to be good, with coefficients for current and lifetime diagnoses exceeding 0.60.[39] It should also be noted that, in administering the SCID, it is extremely important to have well-qualified psychologists involved in the process, as well as careful monitoring of the fidelity of its administration. This was accomplished by careful training of 2 interviewers, as well as regular monitoring (via audiotape) of these assessments, accompanied by regular case conferences to ensure reliability. Quality was assured by re-evaluating randomly selected cases throughout the study. This resulted in the maintenance of high diagnostic reliability, with kappa coefficients for Axis I disorders ranging from 0.905 to 1.00.

The BDI-II is a substantially revised version of the original measure.[35,40] It is a self-report inventory that provides a quantitative assessment of the current severity of depression in adults and adolescents aged 13 years and older. Each of the 21 items is measured on a 4-point scale from 0 to 3. An aggregate score of 13 or less indicates minimal depression, 14 to 19 indicates mild to moderate depression, 20 to 28 indicates moderate to severe depression, and 29 to 63 indicates severe depression. The validity is satisfactory for the BDI, with a correlation greater than 0.71 with the Hamilton Psychiatric Rating for Depression, and a correlation of 0.68 with the Beck Hopelessness Scale. The reliability is also good, as coefficients exceed 0.92 in outpatient samples.[35]

Procedure

Clinical psychology research personnel (psychologists and masters-level counselors) explained the purpose and procedures of the study prior to obtaining informed consent. All subjects then completed the self-report measures described, in addition to a number of other measures not used for this analysis: the Multidimensional Pain Inventory (MPI),[41] the Schedule for Nonadaptive and Adaptive Personality (SNAP),[42] Ways of Coping—Revised (WOC),[43] and Profile of Mood States (POMS).[44] The results of these measures have been

Table 2 Demographic Characteristics of Sample

Variables	Depressed (n = 32) n	Depressed (n = 32) %	Nondepressed (n = 31) n	Nondepressed (n = 31) %	*P*
Gender (%)					.095
Male	4	12.5	9	29.0	
Female	28	87.5	22	71.0	
Race (%)					.164*
Caucasian	26	81.3	20	64.5	
Non-Caucasian	6	18.8	11	35.5	
African American	2	6.3	4	12.9	
Latino	1	3.9	3	9.7	
Asian	2	6.3	2	6.5	
Other	1	3.5	2	6.5	
Marital status (%)					.362
Single	7	21.9	9	29.0	
Married/living together	21	65.6	21	67.7	
Divorced or separated	4	12.5	1	3.2	
Employment status (%)					.890
Full-time work, school or self-employment	20	32.5	18	58.1	
Part-time work	3	9.4	4	12.9	
Not working	9	28.1	9	29.0	
Mean education (y) (SD)	15.0	2.0	15.4	2.0	.525
Mean age (y) (SD)	36.0	11.3	37.6	11.4	.576
Mean days of pain (SD)	102.0	50.6	94.9	48.5	.570

Data given as number and percentage of patients except where noted.
* Chi-square was run only on the Caucasians and non-Caucasians due to power limitations resulting from lack of variability in the sample.

published elsewhere.[19] A brief jaw pain evaluation was then administered according to the RDC/TMD examination form. The evaluators were regularly recalibrated by the study's supervising dentist to ensure accuracy of the jaw evaluation. All subjects also completed a chewing performance evaluation in which they chewed and expectorated the artificial food substance. The assessment took approximately 2.5 hours. Each subject was compensated $70 for his/her participation.

As noted earlier, the current subject pool was taken from a larger ongoing study.[19] Of the 147 subjects in the original study, 63 subjects were eligible for inclusion in this present smaller sample, as they received the early biopsychosocial intervention described in our earlier publication, whereas 78 subjects did not receive the intervention due to group assignment, and 6 subjects were deemed ineligible for the study during the intake evaluation (eg, because of chronic pain).[19] Of the 63 included subjects, 56 completed the 1-year follow-up (48 completed 6 of 6 therapy visits, 3 completed 5 visits, 2 completed 4 visits, 1 completed 3 visits, and 2 completed 1 visit). The reasons for noncompletion of all visits, as well as the reasons given by the 7 subjects who did not complete the 1-year follow-up visit, were similar, and included reasons such as family and work scheduling issues, moving out of the country, and unrelated sickness. As a result, an intent-to-treat statistical method was used to calculate the projected 1-year follow-up results for those individuals on whom some data were missing. The method used to manage missing data was the last observation carried forward (LOCF) approach, where missing values are replaced with the last previous nonmissing value. Table 1 summarizes the number of patients for whom data were collected for each measure and time period.

Subsequently, based upon the results from the subjects' initial evaluation, subjects were divided into 2 groups: the depressed group (n = 32) and the nondepressed group (n = 31). The intervention phase included 6 1-hour sessions that provided education, instruction in muscle relaxation, biofeedback, stress management and coping skills. All subjects had 3-, 6-, and 9-month postintake telephone follow-ups and a 12-month evaluation in person. The telephone follow-ups assessed pain with the CPI and evaluated subjects' healthcare utilization and symptoms. The 12-month postintervention evaluation consisted of re-evaluation with all intake psychosocial and functional measures.

Table 3 Mean CPI, MPS, and BDI Scores for the Depressed and Nondepressed Groups

	Depressed group			Nondepressed group		
	Mean	SD	n	Mean	SD	n
CPI						
Intake	56.84	13.41	32	58.26	10.97	31
12-month	22.77	17.54	32	24.94	18.78	31
MPS						
Intake	3.68	1.23	29*	3.71	1.28	27*
12-month	3.54	1.29	29*	3.87	1.35	27*
BDI-II						
Intake	11.97	12.04	32	5.84	4.91	31
12-month	7.25	8.60	32	2.87	3.25	31

*It was not possible to impute data in cases where intake values were missing.

Statistical Analysis

For all continuous variables (eg, the CPI and BDI), *t* tests and analysis of variance (ANOVA) were conducted to evaluate differences between depressed and nondepressed subjects as well as differences between evaluation points. Mann-Whitney tests were used when assumptions of normality of these data were not met. For dichotomous variables (eg, gender, ethnicity) chi-square analyses were conducted. Finally, a logistic regression analysis was conducted to evaluate what combination of variables best predicted depression.

Results

Demographic Characteristics

Descriptive analyses and frequency distributions were performed on the study sample and are presented in Table 2. Of the subject population, 32 subjects (50.8%) were considered depressed according to their responses to the SCID I at intake. The depressed group comprised subjects whose responses indicated current (3.2%), current and lifetime (15.9%), or lifetime (31.7%) incidences of depression. Therefore, about 19.1% of the 63 subjects were considered "currently" depressed. It was decided to include all 3 categories because patients with a lifetime incidence of depression often have minor recurrences that may not meet all criteria but nevertheless are diatheses or predispositions that can easily be exacerbated.[45] This is 1 reason why the BDI-II was also administered. Independent *t* tests (for continuous variables) and chi-square analyses (for categorical variables) revealed no significant differences between the depressed and nondepressed groups with regard to gender, ethnicity, age, education, duration of pain, marital status, or employment status.

Outcome Measures

Table 3 presents the CPI, MPS, and BDI-II measures at the initial preintervention intake and at the 12-month follow-up for the depressed and nondepressed groups.

CPI. At intake, the average CPI score among depressed subjects was 56.84, while the average CPI score among nondepressed was 58.26; the difference was statistically nonsignificant (independent *t* test). Additionally, there were no significant differences for any other time interval (6th session, 3, 6, and 9 months). ANOVA of the difference in mean CPI between depressed and nondepressed subjects from preintervention to 12-month follow-up yielded a significant time effect, $F(1, 61) = 144.68$ ($P < .001$) but not a significant group effect. There was no interaction effect. Thus, the early intervention produced positive CPI changes in both groups.

MPS. The average MPS at intake among depressed subjects was 3.68, while the average MPS at intake among nondepressed subjects was 3.71 (Table 3). A *t* test revealed no significant differences for mean MPS at intake between depressed and nondepressed subjects. Further, ANOVA revealed no significant differences between groups at the 12-month follow-up (ie, no time or interaction effects).

MPS data were also analyzed by gender. At intake, a Mann-Whitney test yielded a significant difference in MPS between female and male subjects ($P = .006$), with male subjects performing better. These differences were also evident at the 12-month follow-up ($P = .02$), with men continuing to perform better. Based on the results of a chi-square test, there was no significant difference in the distribution of depression between men and women.

BDI. An ANOVA revealed significant decreases in BDI-assessed depression for both groups from preintake to 12-month follow-up, $F(1, 61) = 16.21$, $P = .001$. As expected, a nonparametric Mann-Whitney test also found significance differences between the groups at intake, ($U = 336.5$, $P = .03$) and at the 12-month follow-up ($U = 321.00$, $P = .02$). Moreover, as expected because of the initial SCID diagnosis of presence/absence of depression, there was an overall group effect, $F(1, 61) = 8.72$, $P < .004$. There was no interaction effect.

Multivariate Analyses of Data

In order to more comprehensively evaluate all the variables, the following were each considered as predictive variables in a bivariate logistic regression for modeling depression: gender, CPI (intake and 12 months), BDI (intake and 12 months), and MPS (intake and 12 months). Each variable was examined univariately and multivariately using a stepwise selection procedure that included 2-way interactions. The only variable to reach statistical significance ($P = .027$) in any selection procedure was the BDI at intake, which resulted in an odds ratio of 1.1 (95% CI: 1.01 to 1.20); however, this odds ratio is clinically unimportant because of the small sample size.

Discussion

As reviewed earlier, research indicates that psychosocial factors play a key role in TMD, especially when it becomes more chronic in nature. Additionally, research suggests that psychosocial factors can intensify both pain behavior and the amount of pain.[46] The present study was the first 1-year prospective study designed to examine whether already-existing depression (as assessed by the SCID) in acute TMD subjects affects pain and performance before and after intervention. Some investigators have hypothesized that depressed subjects would experience their pain more severely than nondepressed subjects due to increased vulnerability toward negatively distorted cognitions and a heightened fear of susceptibility to illness.[47,48] Despite this, the present findings revealed that those diagnosed with depression did not report significantly different levels of pain at intake (as determined by the CPI) when compared to nondepressed subjects. Although purely speculative, one conclusion that can be drawn from these results is that persons with either a history of depression or current depression may have grown accustomed to increased feelings of negativity and vulnerability and, therefore, considered heightened pain as normal. Both the depressed and nondepressed groups received intervention and reported pain decreases from intake to the 12-month postintervention follow-up. Moreover, there was no significant difference in change in CPI scores (ie, improvement) between the 2 groups. Another conclusion that can be drawn from these results is that the intervention, which was biopsychosocial in nature, addressed the psychosocial needs and issues of the subjects. Consequently, the results seem to show that both groups, regardless of preexisting depression, benefited equally from such interventions. Finally, one must also consider the fact that any additional comorbid depression developed as the result of suffering with chronic TMD would have a greater exacerbation effect on pain because of this "double dose" of negative affect. This would again highlight the importance of early intervention to prevent greater exacerbation.

This study underscores the differences between acute and chronic pain. Specifically, these findings appear to support the 3-stage model of pain posited by Gatchel,[12,13] which outlines the transition from acute to chronic pain. This comprehensive model incorporates the psychological and physiological factors associated with pain. The subjects in the present study would be categorized as stage 2 because potential subjects who had experienced pain for more than 6 months were excluded from the study. The present results suggest that intervention during stage 2 may thwart the development of chronic pain in patients, regardless of depression history or symptomatology, by providing coping skills and ancillary resources. Of course, it should be noted that TMD is known to have a fluctuating nature; thus, the patients might have presented similar results with no treatment at all. However, as pointed out earlier, this present study was part of a larger randomized controlled trial in which the biopsychosocial intervention resulted in significantly greater therapeutic gains relative to nonintervention.

This study also investigated whether depression resulted in a significant difference in functional

performance. It was thought that if a person was vulnerable to depression, he or she would be more susceptible to compromised chewing functioning. Therefore, it was hypothesized that the depressed group would demonstrate lower levels of chewing performance on the functional measure (MPS) than the nondepressed group. However, no significant difference was found to exist between depressed and nondepressed subjects on this measure at intake; furthermore, no significant differences in chewing performance were found between the 2 groups at the 12-month follow-up. One speculative conclusion that can be drawn from these results is that, just as depressed subjects acclimate to increased pain, it appears that they may also accommodate pain on more "rote," ingrained activities such as chewing. Thus, chewing performance does not appear more compromised for depressed subjects than nondepressed subjects. After intervention, both the depressed and nondepressed subjects may benefit from a biopsychosocial approach to such an extent that they improve chewing performance. Of course, one may also view these findings as another example of the often low degree of concordance among the 3 major components of behavior (self-report, overt motor function, and physiology). Thus, even though self-reported pain is a characteristic of TMD, the motor or functional implications of the complaints may be only perceptual in nature, especially during the acute phase of the disorder.

In conclusion, the present investigation demonstrated that both depressed and nondepressed patients benefited from biopsychosocial treatment. Specifically, prompt interdisciplinary intervention addressing both physical and psychosocial needs of patients appears to be significantly effective in preventing the development of chronic pain in TMD patients.

Of course, any study of this type has potential limitations, and some recommendations for future research should be noted. For example, most of the subjects in the present study were Caucasian and female. Although consistent with clinical population norms for TMD patients, the lack of diversity in the sample evaluated may have affected the results of the study. One could also argue that only a diagnosis of current depression, rather than current or lifetime incidence, should have been used to classify the depression group. However, as discussed by Gatchel and Dersh,[45] many patients with a lifetime incidence of depression have minor recurrences that may not meet the full DSM criteria. Such patients frequently meet subthreshold levels (lacking only 1 of the criteria). This diathesis or predisposition may be exacerbated during a period of stress (such as the onset of a pain episode).

In spite of these issues, the present study adequately addressed the very important, but relatively unexplored, issue of the clinical burden of comorbid depression and pain and its management in the acute care setting. As results clearly showed, with an appropriate biopsychosocial intervention, acute TMD patients can be effectively treated, regardless of the presence or absence of depression. Such results are important because comorbid conditions are usually the norm rather than the exception among patients with depressive disorders. Unfortunately, depression is often overlooked in primary care settings. Use of a biopsychosocial approach in clinical practice will remedy this current deficiency in primary care for a pain syndrome such as TMD.

Acknowledgments

This research was supported by grant nos. 2R01 DE10713 and K05 MH01107 from the National Institutes of Health.

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