Depressed Mood Impedes Pain Treatment Response in Patients with Fibromyalgia

ARNSTEIN FINSET, SIGRID HØRVEN WIGERS, and K. GUNNAR GÖTESTAM

ABSTRACT. ***Objective.*** To investigate prognostic factors in the course of the fibromyalgia syndrome (FM) from baseline to post-treatment.

Methods. Fifty-seven patients with FM were examined in a randomized intervention study. Pre-treatment variables were entered into linear regression analyses: gender, age, duration of disease, allocation to treatment, pain distribution (based on a patient-made drawing), fatigue, sleep disturbance, and depressed mood (based on visual analog scores), with pain distribution at treatment completion as the dependent variable.

Results. Depressed mood at baseline was a significant predictor of sustained widespread pain at treatment completion.

Conclusion. The findings indicate a role for depressed mood as a predictive factor for treatment response. (J Rheumatol 2004;31:976–80)

Key Indexing Terms:
FIBROMYALGIA DEPRESSION TREATMENT

The fibromyalgia syndrome (FM) is defined as a chronic pain syndrome characterized by widespread musculoskeletal pain and the presence of at least 11 out of 18 specific tender points[1]. Moreover, a number of concurrent symptoms such as fatigue, psychological distress, disturbed sleep, stiffness, as well as headache, irritable bowel, and indications of autonomic dysfunction are commonly present[1-3]. About 2% of the general population, and 3.4% of the female population, have been reported to have FM[4,5].

The relationship between the defining clinical features (widespread pain and tender points) and the concurrent symptoms (fatigue, distress, etc.) in patients with FM have been addressed in a number of studies. Fatigue, sleep disturbances, and stiffness are reported by a large majority of FM patients in most studies[1,6], whereas the presence of psychological distress, specifically depression, varies in the literature[7-11]. Some authors consider depression to be so essential in FM that they characterize it as one of several affective spectrum disorders that share common causal factors[12-13]. Others have found that the prevalence of depression in FM is not higher than in other chronic illnesses[14,15]. Recent studies have tended to downplay the role of depression in the etiology of FM by emphasizing factors such as deficits in somatosensory perception[16] and central sensitization[17].

There is also disagreement on the relationship between depressed mood and pain in FM patients. Whereas Kurtze, *et al* found independent additive effects of both anxiety and depression on pain[18], Okifuji, *et al* reported recently that depressed and non-depressed FM patients did not differ in terms of pain severity and number of tender points[19].

Differences in both definition and measurement of depression and in characteristics of the study populations may contribute to these seemingly discrepant results. Thus, when exploring depression in patients with FM and other rheumatological disorders, Krag, *et al* showed that whether or not group differences emerged depended on the measure of depression being used[20]. The nature of the sample may also influence the prevalence of depression among FM patients. Aaron, *et al* have suggested that the strong association often found between FM and depression may partly be due to the fact that the subjects in most studies are patients who seek treatment for their condition[21], but an association between FM and depression has also been found in community samples[4]. However, the highest prevalence of depression in FM patients is reported among patients in tertiary care settings[10].

In the research literature there are few studies that investigate the prognostic factors that influence the course of FM over time, either in terms of the role of defining clinical features or of concurrent symptoms, including psychosocial factors[22]. Even if most followup studies find FM to be a relatively stable condition with few remissions[23-28], there are exceptions[29,30]. Granges, *et al* found that as many as 47% of

From Department of Behavioural Sciences in Medicine, University of Oslo, Oslo,; The Rehabilitation Centre Jeløy Kurbad, Moss; and the Department of Psychiatry and Behavioural Medicine, University of Trondheim, Trondheim, Norway.

Supported in part by The Research Council of Norway, Grant 101417/320.

A. Finset, PhD, Professor; S.H. Wigers, MD, PhD, Senior Consultant, Department of Behavioural Sciences in Medicine, University of Oslo; K.G. Götestam, MD, PhD, Professor, Department of Psychiatry and Behavioural Medicine, University of Trondheim.

Address reprint requests to Dr. A. Finset, Department of Behavioural Sciences in Medicine, University of Oslo, POB 1111, Blindern, NO-0317, Oslo, Norway. E-mail:arnstein.finset@basalmed.uio.no

Submitted December 30, 2002; revision accepted November 21, 2003.

FM patients no longer fulfilled the American College of Rheumatology 1990 (ACR-90) criteria[1] 2 years after diagnosis, and that there appeared to be a low correlation between mood levels and FM symptoms at followup[29].

In one of the few studies of prognostic factors in FM, Forseth, *et al* studied 214 women at 2 assessments 5 years apart[31]. Among patients who did not have FM at baseline, one-fourth fulfilled FM criteria at the second assessment. Back pain at baseline predicted a development of FM, whereas tender points and neck pain did not. Moreover, having many associated symptoms, longstanding pain, and self-assessed depression were found to be predictors. Moreover, there are indications that the presence of depression not only represents a risk factor in developing FM, but may influence the efficacy of treatment as well. Ferraccioli, *et al* showed that depressed FM patients failed to benefit from the given treatment, in contrast to the non-depressed[32].

Given the fact that few studies have investigated predictors in the course of FM, we decided to reanalyze a sample of FM patients who had taken part in a clinical trial[33] in order to study the relationship between the defining clinical features of FM (widespread pain and pressure tenderness) on one hand and depressed mood and other concurrent symptoms (fatigue and sleep disturbances) on the other. Our intent was to investigate the role of these features and symptoms as prognostic factors for pain in the course of FM over the time span from baseline to post-treatment. As our measures of the concurrent symptoms were based on visual analog scale (VAS) scores, we applied the concept of depressed mood rather than depression in the reporting of our data.

MATERIALS AND METHODS

Subjects. Seventy-seven patients were recruited from the 4 following sources: local general practitioners, local rheumatologists, the Physical Medicine Outpatient Clinic at the University Hospital of Trondheim, and the local Fibromyalgia Patient Association. All patients were examined and diagnosed by the second author (SHW). Twelve patients did not meet the inclusion criteria of fulfilling both Smythe's[34] and Yunus'[2] diagnostic criteria for FM, and 5 refused to take part in the treatment study. Thus 60 patients, 11 from a tertiary referral center, 31 having consulted a rheumatologist, and 18 patients having seen general practitioners only, took part in the study. Retrospective investigation of the patient data revealed that the patients also fulfilled the ACR-90 diagnostic criteria[1]. Demographic data are given in Table 1. The patients were randomized into 3 treatment groups of 20, and went through a 14 week intervention period. Data from the 57 patients who completed both the baseline and treatment completion assessments are used in the present study.

Treatment. Group One was given aerobic exercise led by a physiotherapist, 45 minutes 3 times a week for 14 weeks. The exercise program involved the whole body, and tempo was gradually increased up to and down from 4 periods of high intensity training at 60-70% of maximum heart rate (altogether 18-20 minutes). The program started with a 23 minute session of music comprising warm-up and 2 high intensity peaks of 4 minutes each, and ended with 15 minutes of aerobic games, representing 2 high intensity periods of 5-6 minutes each. The program finished with warming down and thoroughly stretching out. Group Two was led by a clinical psychologist and received a cognitive-behavioral stress management package including applied relaxation as described by Öst[35] and an introduction to cognitive therapy in coping with psychological problems. The first 6 weeks patients met 90 minutes twice a week, and the last 8 weeks, only once a week. Each session consisted of equal portions of didactic presentations, group discussions, and relaxation training. Patients received an audiocassette containing the progressive and release only relaxation procedures and practiced relaxation techniques regularly at home. Group Three was a "treatment-as-usual" control, i.e., patients simply continued the treatments they had been using when included at baseline. These treatments included aquatic therapy (n = 3), psychomotor treatment (n = 1), tricyclic antidepressants (low evening doses), n = 8), and mostly when needed, low doses of: analgesics (n = 6), muscle relaxants (n = 3), hypnotics (n = 3), and tranquilizers (n = 2). Patients were excluded if new treatment regimens were initiated or new diseases were acquired in the intervention period. The treatment study has been described[33].

Measures. The following measures were made at baseline (T1) and at treatment completion (T2): Pain distribution was assessed by a patient-made drawing: the patients shaded the areas that had been painful the last 3 days on 2 small body maps representing the ventral and dorsal side of the body, respectively. No shading meant no pain. From this drawing, the percentage of total body area affected by pain was later estimated using Wallace's rule of 9 for assessing burns (head = 9%, one arm = 9%, one leg = 18%, one side of truncus = 18%, and genitalia = 1%)[36]. The present quantification of pain distribution has proven to be a reliable and appropriate measure in FM[37]

Pressure tenderness in 16 ACR-90 tender points (all except the low cervical ones) was measured using a hand-held, spring-loaded pressure dolorimeter (Pain Diagnostics and Thermography Inc., Great Neck, NY, USA). It was placed perpendicularly against the anatomically defined spots without prior palpation and with a rate of one kg per second. The value was recorded when the patient started to feel the pressure painful. The mean dolorimeter score from these 16 points represented the measure of pressure tenderness. All measurements were made by SHW, and neither she nor the patients had access to earlier recordings on any test occasion.

Intensity of pain, disturbed sleep, fatigue, and depressed mood were rated on a patient administered VAS scheme. The scheme consisted of 100 mm horizontal lines, each representing a symptom of its own. End descriptors were 0 = nothing and 100 = the worst you have ever experienced. Patients were asked to indicate the last 3 days' average values.

Dependent variable. Pain distribution on treatment completion was chosen as the dependent variable. Widespread pain is one of 2 main criteria of FM, and an improvement in pain distribution will therefore have reasonably high face validity. Moreover, we have found our pain distribution score to be strongly related to patients' own indications of global subjective improvement[37].

Statistical procedures. Pearson's product-moment correlations were employed to describe the associations between variables at T1 and the chosen dependent variable at T2, pain distribution.

Linear regression analysis was chosen to investigate what baseline variables would predict pain distribution at treatment completion. To assure that the dependent variable met the statistical assumptions for multiple regression we ran a scatter plot of the standardized residual against the standardized predicted value (which showed no patterns) and a normal probability plot (which showed a satisfactory cumulative probability) as well as a histogram of the standardized residual (which showed a satisfactory normal distribution).

Within-group differences in pain distribution from T1 to T2 were computed in paired sample t tests.

The Statistical Package for the Social Sciences (SPSS) was used for the statistical analyses.

RESULTS

Levels of defining features of FM and of concurrent symp-

Table 1. Patient characteristics in the total patient sample (n = 60) and in each treatment group (Group 1: aerobic exercise; Group 2: stress management; Group 3: treatment as usual).

Variable	Total	Group 1	Group 2	Group 3
n	60	20	20	20
Gender, F/M	55/5	18/2	18/2	18/1
Mean age, yrs (SD, range)	44 (10, 23–73)	43 (9, 23–62)	44 (12, 28–69)	46 (9, 29–73)
Mean symptom duration, yrs (SD, range)	10 (8, 1–42)	9 (5, 2–20)	11 (10, 1–40)	11 (9, 1–42)
Working full time, n (%)	13 (22)	4 (20)	5 (25)	4 (20)
Working part time, n (%)	14 (23)	7 (35)	5 (25)	2 (10)
Mean % pain distribution (SD)	68 (25)	71 (24)	62 (27)	71 (23)
VAS, mean (SD)				
Pain intensity	70 (18)	72 (19)	72 (18)	65 (17)
Fatigue	75 (23)	80 (20)	80 (22)	66 (27)
Disturbed sleep	57 (32)	60 (33)	65 (27)	47 (37)
Depression	39 (33)	34 (29)	44 (32)	40 (37)
Dropouts, n*	3	1	2	0

VAS: visual analog scale. * Transport problems and moving to another area.

toms (depressed mood, sleep, and fatigue) are given in Table 1.

There was a highly significant reduction in pain distribution from T1 to T2 in the sample as a whole from a mean of 68.2 (SD: 24.1) at T1 to 48.1 (SD: 30.0) at T2 ($t = 4.44$; $p < 0.001$).

The correlation matrix including variables at T1 (depressed mood, sleep, fatigue, pain distribution, pain intensity, gender, age, duration of illness, as well as a variable that indicated whether patients had been allocated to the treatment or control group) and the chosen dependent variable at T2, pain distribution, is given in Table 2. There were significant negative correlations at T1 between depressed mood and age as well as duration of illness. Pain intensity and pain distribution at T1 were significantly related to one another. Fatigue at T1 was significantly higher among patients allocated to one of the 2 treatment conditions compared to the treatment as usual group (Table 2).

The only 2 variables that were associated with pain distribution at T2 were depressed mood at T1 ($r = 0.26$; $p = 0.051$) and treatment allocation ($r = 0.26$; $p = 0.051$). These 2 variables were therefore chosen as independent variables in a linear regression analysis together with pain distribution at T1. Only the measure of depressed mood was significantly associated with pain distribution at treatment completion (standardized beta = 0.27, $p < 0.05$; Table 3).

The distribution of depressed mood at T1 was bimodal, with no individual scores between 31 and 40. The sample was therefore broken down into subgroups of low depressed mood scores (n = 31; mean: 12.9, SD: 11.1) and high depressed mood scores (n= 26; mean: 70.4, SD: 19.3) at T1 (based on a cutoff score of 35). In the low depressed mood group there was a highly significant improvement in pain distribution from a mean score of 68.1 (SD: 25.0) to a mean of 43.1 (SD: 28.5) from T1 to T2 ($t = 4.67$; $p < 0.001$), whereas the trend towards improvement among patients with high depressed mood scores [from a mean of 68.4 (SD: 23.5) to 54.0 (SD: 31.1)] did not reach statistical significance.

Table 2. Correlation matrix.

Variable	Depressed Mood at T1	Disturbed Sleep at T1	Fatigue at T1	Pain Intensity at T1	Pain Distribution at T1	Pain Distribution at T2
Gender	0.24#	0.07	0.15	–0.01	–0.06	–0.10
Age	–0.40**	–0.07	–0.17	0.06	0.00	–0.11
Disease duration	–0.26*	–0.08	0.02	0.13	0.04	0.03
Treatment allocation	–0.03	0.21	0.27*	0.17	–0.09	–0.26#
Depressed mood at T1	—					0.26#
Distrubed sleep at T1	0.06	—				–0.01
Fatigue at T1	0.10	0.12	—			0.10
Pain intensity (VAS) at T1	–0.18	0.21	0.24#	—		0.11
Pain distribution at T1	–0.06	0.08	–0.07	0.30*	—	0.21

male: 0; female: 2; treatment as usual: 0; allocated to one of 2 experimental conditions: 1. # $p < 0.10$. * $p < 0.05$. ** $p < 0.01$.

Table 3. Linear regression analysis with pain distribution at treatment completion (T2) as dependent variable.

Independent Variables	Standardized Beta	p
Pain distribution at T1	0.21	NS
Depressed mood at T1	0.27	< 0.05
Allocation to treatment	–0.23	0.07

DISCUSSION

We investigated the relationship between widespread pain as a defining clinical feature in FM and concurrent subjective symptoms in a sample of FM patients who had participated in a treatment study. We found that the presence of depressed mood was associated with a failure to recover from widespread pain over a 4-month period, indicating that depressed patients failed to display a recovery in pain scores from T1 to T2. In a linear regression analysis, depressed mood at baseline was a significant predictor of sustained widespread pain at treatment completion. Depression or depressed mood may be based on self-report in a number of different ways. One approach is a to apply a self-rating scale with several dimensions, as anhedonia (loss of pleasure), depressed mood, including somatic symptoms, and behavioral symptoms (slowness/retardation), and somatic or more unspecific symptoms as fatigue, sleeplessness, and lack of initiative. This gives a multifaceted picture of the depression, including both specific and unspecific depressive symptoms. Another approach, which was our choice, is to assess the affective component of depression, which we have labeled depressed mood, with a VAS or visual analog mood scale[38]. Such a VAS is also amenable to repetitive assessments[39], and has shown satisfactory validity and reliability[40-44]. When results are going to be compared between studies, it is important to have in mind what type or spectrum of depressive symptoms the scales in question are comprising. A positive association between depression and pain is more likely to be seen when an instrument including somatic aspects of depression is applied than when the assessment is limited to the affective component, depressed mood, as in our study.

Okifuji, *et al*[19] conclude in their study of FM patients that do and do not satisfy depression criteria that depression may be independent of the cardinal features of FM in terms of widespread pain and tender points. They did, however, find that maladaptive thoughts and subjective functional limitations were more frequent and severe among the depressed patients. The failure to respond to treatment and improve their pain scores seen among depressed patients in our study could well be related to cognitive factors associated with depression such as catastrophizing and negative expectations.

A major limitation of our study is the small sample size. We have therefore chosen to divide the linear regression analyses into 3 separate analyses. Still, the findings from the multivariate analyses should be interpreted with caution. The findings would need to be replicated in a larger sample. As most clinical studies on FM have been reported from tertiary medical centers, probably including patients with more severe illness than community cases, etc., trials enrolling a patient population independent of a specific treatment context have been called for[45]. Our study meets this call by recruiting patients from all levels of the health care system. This was achieved by recruiting patients from the local patient association, as nearly all FM patients in Trondheim were members of the local patient organization in 1988 during patient recruitment, even those that were recruited through the University Hospital Outpatient Clinic. In later years the attitude of the patient organization changed, and today members of the same organization probably would represent a selected sample of FM patients, but it was not so in the beginning. As the present study includes patients from all levels of the health care system with a sex distribution similar to the general FM population[5], with about 90% women, the sample is judged to be representative for the general FM population in Trondheim.

A majority of the patients in our study of patients with FM had relatively low depression scores as measured with a VAS. Depressed mood was not significantly related to pain and fatigue at baseline, but patients who scored high on depressed mood responded significantly less to treatment in terms of reduction in pain distribution scores. The findings indicate a role of depression as a predictive factor for treatment response. More research is needed to determine the aspects of depression that represent an obstacle for recovery from widespread pain in FM.

REFERENCES

1. Wolfe F, Smythe HA, Yunus MB, et al. The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis Rheum 1990;33:160-72.
2. Yunus M, Masi AT, Calabro JJ, Miller KA, Feigenbaum SL. Primary fibromyalgia (fibrositis): clinical study of 50 patients with matched normal controls. Semin Arthritis Rheum 1981;11:151-71.
3. Jacobsen S. Chronic widespread musculoskeletal pain: the fibromyalgia syndrome. Danish Med Bull 1994;41:541-64.
4. Wolfe F, Ross K, Anderson J, Russell IJ, Hebert L. The prevalence and characteristics of fibromyalgia in the general population. Arthritis Rheum 1995;38:19-28.
5. Wolfe F. The epidemiology of fibromyalgia. J Musculoskeletal Pain 1993;1:137-48.
6. Moldofsky H, Scarisbrick P, England R, Smythe H. Musculosketal symptoms and non-REM sleep disturbance in patients with "fibrositis syndrome" and healthy subjects. Psychosom Med 1975;37:341-51.
7. Yunus MB. Psychological aspects of fibromyalgia syndrome: a component of the dysfunctional spectrum syndrome. Baillieres Clin Rheum 1994;8:811-37.
8. Hudson JI, Hudson MS, Pliner LF, Goldenberg DL, Pope HG Jr. Fibromyalgia and major affective disorder: a controlled phenomenology and family history study. Am J Psychiatry

1985;142:441-6.
9. Hudson JI, Pope HG, Jr. The relationship between fibromyalgia and major depressive disorder. Rheum Dis Clin N Am 1996;22:285-303.
10. Walker EA, Keegan D, Gardner G, Sullivan M, Katon WJ, Bernstein D. Psychosocial factors in fibromyalgia compared with rheumatoid arthritis: I. Psychiatric diagnoses and functional disability. Psychosom Med 1997;59:565-71.
11. Yunus MB, Ahles TA, Aldag JC, Masi AT. Relationship of clinical features with psychological status in primary fibromyalgia. Arthritis Rheum 1991;34:15-21.
12. Hudson JI, Pope HG, Jr. Fibromyalgia and psychopathology: is fibromyalgia a form of "affective spectrum disorder"? J Rheumatol 1989;19 Suppl:15-22.
13. Alfici S, Sigal M, Landau M. Primary fibromyalgia syndrome: a variant of depressive disorder? Psychother Psychosom 1989;51:156-61.
14. Kirmayer LJ, Robbins JM, Kapusta MA. Somatization and depression in fibromyalgia syndrome. Am J Psychiatry 1988;145:950-4.
15. Ahles TA, Khan SA, Yunus MB, Spiegel DA, Masi AT. Psychiatric status of patients with primary fibromyalgia, patients with rheumatoid arthritis, and subjects without pain: a blind comparison of DSM-III diagnoses. Am J Psychiatry 1991;148:1721-6.
16. Sorensen J, Graven-Nielsen T, Henriksson KG, Bengtsson M, Arendt-Nielsen L. Hyperexcitability in fibromyalgia. J Rheumatol 1998;25:152-5.
17. Staud R, Smitherman ML. Peripheral and central sensitization in fibromyalgia: pathogenic role. Curr Pain Headache Rep 2002;6:259-66.
18. Kurtze N, Gundersen KT, Svebak S. The role of anxiety and depression in fatigue and patterns of pain among subgroups of fibromyalgia patients. Br J Med Psychol 1998;71:185-94.
19. Okifuji A, Turk DC, Sherman JJ. Evaluation of the relationship between depression and fibromyalgia syndrome: why aren't all patients depressed? J Rheumatol 2000;27:212-9.
20. Krag NJ, Norregaard J, Larsen JK, Danneskiold-Samsoe B. A blinded, controlled evaluation of anxiety and depressive symptoms in patients with fibromyalgia, as measured by standardized psychometric interview scales. Acta Psychiat Scand 1994;89:370-5.
21. Aaron LA, Bradley LA, Alarcon GS, et al. Psychiatric diagnoses in patients with fibromyalgia are related to health care-seeking behavior rather than to illness. Arthritis Rheum 1996;39:436-45.
22. Eich W, Hartmann M, Muller A, Fischer H. The role of psychosocial factors in fibromyalgia syndrome. Scand J Rheumatol 2000;S113:310-31.
23. Felson DT, Goldenberg DL. The natural history of fibromyalgia. Arthritis Rheum 1986;29:1522-6.
24. Hawley DJ, Wolfe F, Cathey MA. Pain, functional disability, and psychological status: a 12-month study of severity in fibromyalgia. J Rheumatol 1988;15:1551-6.
25. Norregaard J, Bulow PM, Prescott E, Jacobsen S, Danneskiold-Samsoe B. A four-year follow-up study in fibromyalgia. Relationship to chronic fatigue syndrome. Scand J Rheumatol 1993;22:35-8.
26. Ledingham J, Doherty S, Doherty M. Primary fibromyalgia syndrome: an outcome study. Br J Rheumatol 1993;32:139-42.
27. Henriksson CM. Longterm effects of fibromyalgia on everyday life. A study of 56 patients. Scand J Rheumatol 1994;23:36-41.
28. Bengtsson A, Bäckmann E, Lindblom B, Skogh T. Long term follow-up of fibromyalgia patients: clinical symptoms, muscular function, laboratory tests — an eight years comparison study. J Musculoskeletal Pain 1994;2:67-80.
29. Granges G, Zilko P, Littlejohn GO. Fibromyalgia syndrome: assessment of the severity of the condition 2 years after diagnosis. J Rheumatol 1994;21:523-9.
30. Fitzcharles MA, Costa DD, Poyhia R. A study of standard care in fibromyalgia syndrome: a favorable outcome. J Rheumatol 2003;30:154-9.
31. Forseth KO, Husby G, Gran, JT, Førre O. Prognostic factors for the development of fibromyalgia in women with self-reported musculoskeletal pain. A prospective study. J Rheumatol 1999;26:2458-67.
32. Ferraccioli G, Ghirelli L, Scita F, et al. EMG-biofeedback training in fibromyalgia syndrome. J Rheumatol 1987;14:820-5.
33. Wigers SH, Stiles TC, Vogel PA. Effects of aerobic exercise versus stress management treatment in fibromyalgia. A 4.5 year prospective study. Scand J Rheumatol 1996;25:77-86.
34. Smythe HA. Non-articular rheumatism and psychogenic musculoskelatal syndromes. In: McCarty DJ, editor. Arthritis and allied conditions. Philadelphia: Lea and Febiger; 1979:881-91.
35. Ost LG. Applied relaxation: description of a coping technique and review of controlled studies. Behav Res Ther 1987;25:397-409.
36. Masterton JP. Burns. In: Dudley HAF, editor. Hamilton Bailey's emergency surgery 11th edition. Bristol: John Wright & Son; 1986;68-76.
37. Wigers SH, Skrondal A, Finset A, Gøtestam KG. Measuring change in fibromyalgic pain: the relevance of pain distribution. J Musculoskeletal Pain 1997;5:29-41.
38. Aitken RC. Measurement of feelings using visual analogue scales. Proc R Soc Med 1969;62:989-93.
39. Litte JC, McPhail NI. Measures of depressed mood at monthly intervals. Br J Psychiatry 1973;122:447-52.
40. Folstein MF, Luria R. Reliability, validity, and clinical application of the visual analogue mood scale. Psychol Med 1973;3:479-86.
41. Davies B, Burrows G, Poynton C. A comparative study of four depression rating scales. Aust N Z J Psychiatry 1975;9:21-4.
42. Luria RE. The validity and reliability of the visual analogue mood scale. J Psychiatr Res 1975;12:51-7.
43. Luria RE. The use of the visual analogue mood and alert scales in diagnosing hospitalized affective psychoses. Psychol Med 1979;9:155-64.
44. Wewers ME, Lowe NK. A critical review of visual analogue scales in the measurement of clinical phenomena. Res Nurs Health 1990;13:227-36.
45. Goldenberg DL. Treatment programs. J Musculoskeletal Pain 1993;1:71-81.