RHEUMATOID ARTHRITIS: A PSYCHOLOGICAL INTERVENTION

DISSERTATION

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A psychological intervention involving relaxation training and biofeedback training for the control of peripheral skin temperature was investigated in this study with 27 female rheumatoid arthritics as participants. A two-group design was used with the only difference being the direction in which participants were instructed to alter their peripheral skin temperature. A temperature increase group was to use biofeedback to achieve an increase in peripheral skin temperature, while a temperature decrease group was to achieve a decrease. Both groups received identical relaxation training. Based on analysis of the temperature data, it was concluded that the biofeedback response was not learned. From electromyographic data, it was concluded that participants did learn to relax.

The hypothesis that the two treatment components would have beneficial affects on the physical, functional, and psychological aspects of rheumatoid arthritis was answered partially. No differential effects as a function of biofeedback training were found as the data for the temperature increase and temperature decrease groups were statistically combined in multiple analyses of variance for repeated

measures. Although no differential effects were obtained, numerous positive changes were found. Correlated with the relaxation training were decreases in reported subjective units of discomfort, percentage of time hurting, percentage of body hurting, and general severity of pain. Improved sleep patterns were reported as was increased performance of activities of daily living. Reductions were also found in psychological tension, and in the amount of time mood was influenced by the disease. Shifts were not found in imagery, locus of control, and other psychological dimensions. Constitutional improvements were also absent.

Relaxation training was recommended as an adjunctive therapy and its implications were discussed. Future research is suggested.

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RHEUMATOID ARTHRITIS: A PSYCHOLOGICAL INTERVENTION

Recent treatment strategies for many physical disorders reflect a trend toward an interdisciplinary health care approach (Williams & Gentry, 1977). It will be suggested that the treatment of arthritis, particularly rheumatoid arthritics, should include psychological intervention. A research review will be presented with special emphasis placed on the relationship of emotional factors, especially psychological stress, to the onset and progression of arthritis, as well as other biological disorders. A treatment strategy for rheumatoid arthritis will be described in which biofeedback and verbal relaxation training will be used to reduce the psychological stress and discomfort associated with that disease.

Clinical Description

Arthritis is estimated to be the chief cause of physical disability in 20-50 million Americans (Pelletier, 1977; Weiner, 1977), with approximately a quarter million new cases reported each year (Pelletier, 1977; Williams, 1974). Due to severe, chronic pain 17 million arthritics currently are receiving medical attention. Pelletier (1977) reports that the economic impact of arthritis is profound in terms of lost wages and the expense of chronic medical care. Medical costs directly attributable to this disease amount to about four billion dollars per year and are growing rapidly.

Several major types of arthritis may be diagnosed, the incidence of each varying with factors such as the age and sex of the afflicted individual. Most arthritics (85%) are 45 years of age or older, and of these 60% have osteoarthritis. Rheumatoid arthritis affects the majority of arthritics who are 45 years or younger. An estimated five million Americans fall into this latter diagnostic category, including approximately 200,000 children and over two million adolescents and young adults (Weiner, 1977). Significantly, rheumatoid arthritis afflicts approximately three times more women than men. Certainly, of all forms of arthritis, rheumatoid arthritis is the most crippling. It is this subtype of disorder which will be the concern in this paper.

Although the term "rheumatoid arthritis" was first used in the middle of the nineteenth century, a detailed description of the disease has only recently begun to emerge. Current clinical descriptions of rheumatoid arthritis characterize the disease as a generalized systemic illness (Williams, 1974). According to Williams, "multiple extra articular areas of involvement, the constitutional symptoms, and the interesting generalized prodromata often antedate the illness by years or months" (p. 31).

Apparently, rheumatoid arthritis begins slowly, usually in one or two joints at a time. Shoulders, elbows, hips, wrists, fingers, knees, ankles, and feet are most commonly involved. Temporomandibular and cricoartenoid joints may

also be involved and are of some diagnostic significance in that they are rarely affected by diseases other than rheumatoid arthritis. In some patients various prodromal symptoms of fatigue, diffuse muscle stiffness, dysthesias or paresthesias may occur. A symmetrical pattern of joint involvement is not unusual, although cases of nonsymmetrical involvement are also seen. The ultimate severity of the disease can not reliably be predicted by the presence or absence of prodromata nor by the acuity of onset. As the disease progresses, complaints of joint pain at rest and on moving, swelling of the involved joints and stiffness after inactivity, and a pronounced limitation of motion are typical. Soft tissue or periarticular swelling near involved joints is also common. Muscular atrophy occurs at an alarming rate and subcutaneous nodules form in approximately one-fifth of all patients. The severity of the symptoms may fluctuate over time. The most common complaints of the rheumatoid concern chronic pain, and the often dramatic reduction of mobility seen in the more advanced stages.

Underlying this symptomology there is also a predictable sequence of steps in the progression of the disease at the physiological level (Williams, 1974). Normally a joint interior is lined with a synovial membrane which secretes fluid as a lubricant. Rheumatoid arthritis affects the synovial cells causing them to multiply at an unnatural rate, thereby creating swelling. This tissue creeps into the joint, ultimately

packing it, destroying the cartilage, and covering the ends of the bone until erosion occurs, and the joint is rendered useless. In the most advanced stages joint deterioration may cause the formation of scar tissue which in turn produces a joint that is knobby, deformed, and completely immobilized. Peripheral manifestations such as vasomotor instability, exemplified by cold hands or excessive peripheral sweating are also common.

While this physiological progression is, for the most part, universally accepted, no single treatment regimen is so widely endorsed (Williams, 1974). Chemotherapy is the most typical intervention, but even still there is no generally accepted pharmaceutical agent. Instead there are currently five basic medication alternatives for rheumatoid arthritis (Carpenter & David, 1976), each using a drug agent for symptom relief. Aspirin is used most frequently. Dosages are set at a "maintenance" level, i.e., the largest possible dosage that does not produce counterproductive side effects. Steroids, gold, penicillamine, and cytotoxins follow as alternative treatment (Johansson & Sullivan, 1975; Weiner, 1977). Success rates, in terms of cure or stabilization within the five alternatives, vary but are generally quite low (Williams, 1974).

Alternatives to chemotherapy also are available (Silverman in Freedman, Kaplan, & Sadock, 1975). A comprehensive intervention often requires a therapy team, which in addition to

a physician, involves a physiotherapist, physical therapist, occupational therapist, social worker, psychiatric nurse, and a psychiatrist or psychologist.

The involvement of a psychologist in the treatment program may be extremely important since chronic pain and the loss of mobility may create serious problems of psychological functioning, including depression, frustration, apathy, and a helpless outlook (Pelletier, 1977; Weiner, 1977). These psychological symptoms may act to undermine compliance to the treatment regimen, blocking any intervention strategy. Further, psychological distress may antedate or exacerbate certain diseases, including rheumatoid arthritis (Pelletier, 1977; Soloman & Moos, 1964; Williams, 1974; Wolff, 1968). Therefore, treatment must involve a process of ever-changing decisions and goals based on the patient's constantly shifting status of physical and psychosocial functioning (Katy, Vignos, & Moskowitz, 1968). The psychologist should minimize maladaptive emotional reactions, and provide an adjunctive treatment to insure compliance to a medical regime and hopefully aid the patient in the management of his or her pain. Unfortunately, psychologists are in no more agreement as to what to include in their treatment strategy than are their physician counterparts.

One major problem for both psychologists and physicians is that criteria used in diagnosing the illness are many and varied, and often of a dysjunctive nature (Bennett & Burch,

1967; Kellgren, 1968; Ropes, Bennett, Cobb, Jacox, & Jesser, 1958; Weiner, 1977). As a result, research reports are inconsistent, as are data obtained from etiology and pathogenesis investigations. Theories adhering to an epedimiological, physiological, genetic, or psychological causal basis are equally frequent, and often contradictory (King, 1955; Spergel, 1972; Weiner, 1977; Williams, 1968; Wolff, 1968).

Although areas of psychological investigation have varied, historically the interest in the illness has been of a traditional nature. Relationships between rheumatoid arthritis and personality or traits, defense mechanisms and conflicts are among the most frequently researched areas. Excellent critical reviews of psychological research methodology in this area have been offered by King (1955), Moos (1964), and Scotch and Geiger (1962).

More directly germane to the current paradigm, however, is the well-documented relationship between psychological stress and rheumatoid arthritis. Pellitier (1977) and others (Cobb, 1959; Cormier & Wittkower, 1957; Crown, Crown, & Fleming, 1974; Meyerowitz, 1971; Weiner, 1977) report that the illness may begin, or exacerbations may occur, in association with conscious worry, grief, depression, or with exposure to various life events labeled by the patient as stressful. A review of Table 1 summarizes and leads to the conclusion that psychological stress may play some role in initiation or aggravation of symptomology.

Table 1

The Relationship Between Stress and Rheumatoid Arthritis

Author/Date	Sample	Stressor	Outcome Measure	Results
Bourestom & Howard (1965)	94 rheumatoids 74 multiple sclerosis patients 100 spinal cord injuries	Various personal pathology patterns	Frequency of ego-dystonic pathology patterns among rheumatoids	Found the most common pathology for male rheumatoids was depression; for females somatic concerns and inhibited emotional expression
Cobb, Schull, Harburg, & Kasl (1969)	49 families with at least 1 rheumatoid	An arbitrary, severe, unreasonable, and controlling mother	Frequency with which rheumatoids described their mothers as arbitrary, severe, unreasonable, and controlling	Found a significant trend for rheumatoids to describe mothers in this way
Dewind & Payne (1976)	230 jejunoilcal bypass surgery patients	The bypass surgery	Type and frequency of complications occurring during 2 years following surgery	9% of men and 19% of women reported onset of arthritis greatly exceeding baserates for the general population
Heisel (1972)	34 rheumatoids 68 controls	Life change events as measured by questionnaire	Frequency of onset of rheumatoid arthritis	For year preceding onset, rheumatoids had life change scores averaging 166.98 versus 82.78 for controls ($p < .0005$).

Table 1--Continued

Author/Date	Sample	Stressor	Outcome Measure	Results
Moos & Solomon (1964)	14 rheumatoids	Intense athletic competition	Level of rheumatoid factor in the blood	Rheumatoid factor level significantly increased after hard-fought game, especially in those athletes who were most anxious and upset prior to competition
Pipineli-Potamianoa (1976)	11 rheumatoids	Overwhelming anxiety about aggressive impulse (various other dynamic conflicts as measured in two face association interviews of 1½ hours each	Frequency of onset of rheumatoid arthritis	Psychoanalytic interpretation suggests that central to all rheumatoids were castration anxiety-inducing factors
Rimon (1969)	100 rheumatoids 100 matched controls	Family mental illness, broken-home background, sexual problems, personal mental illness, various major life crises	Frequency of onset and exacerbation of rheumatoid arthritis	Found significant relationship between identified stressors/ rheumatoid arthritis in patients reporting acute initial onset. Those with insidious onset did not manifest such a relationship

Table 1--Continued

Author/Date	Sample	Stressor	Outcome Measure	Results
Schochet, Lisansky, Schubart, Fiocco, Kurland, & Pope (1969)	12 rheumatoids	Life-change events as measured by interview and questionnaire	Frequency of onset and exacerbation of rheumatoid arthritis	Onset and exacerbation were found to be significantly related to the occurrence of life crises, especially loss of loved ones
Short (1957)	293 rheumatoids	Various categories of stressful life events	Frequency of onset or exacerbation of rheumatoid arthritis	Found 50% of the sample reported one or more life events preceded the attack. Events were distributed as follows: Mental and/or physical strain . . . . . .27.3% Infection. . . . .16.7% Exposure to cold or dampness . . . . .10.6% Surgical operation . . . . 5.5% Trauma . . . . . 5.1% Pregnancy 2.1%

The consistent reports of stress as an antedater to flare-ups of rheumatoid arthritis are rare exceptions to the usually contradictory evidence reported in the arthritis literature. Unfortunately, a hypothesis of arthritic pathegenesis connecting psychological with physiological functioning must await elucidation of the exact site at which the process begins. Clarification of the interaction would also require discovery of a pathogenetic agent. Currently a variety of hypotheses exist, all attempting to describe the physiological initiating agent. Major hypotheses include infection by virus (Kilroy, 1970; Phillips & Christian, 1970; Warren, Marmor, Liebes, & Hollins, 1969) or bacteria (Duthie, Brown, Knox, & Thompson, 1975; Sharp, 1971), immunopathology, i.e., rheumatoid factors (specifically antibodies directed against the body's own healthy blood cells)(Kellgren & Ball, 1959; Lawrence, Valkenburg, Tuxford, & Collard, 1971), and vascular lesions (Schumacher, 1975).

Without a full understanding of the nature of the interaction between the psychological and biological aspects of rheumatoid arthritis, effective treatment and prevention is unlikely. However, psychological stress is believed to be an important piece of the elusive puzzle, and reduction of that stress a significant treatment adjunct.

Stress and Other Diseases

The relationship of psychological stress to somatic disease has been recognized for over 2000 years. In the

fourth century B.C., Hippocrates prescribed rest and relaxation for both physiological and psychological complaints (Silverman, in Freedman et al., 1975). Persian texts written in the twelth century A.D. have noted the effects of inhibited aggression, grief, sorrow, shock, and general emotional stress on the course of disease (Shafii, 1973). These early revelations anticipated development of the more recent study of "psychosomatic" (American Psychiatric Association, DSM-II, 1968), and "behavioral" (Williams & Gentry, 1977) medicine.

In behavioral medicine, the relation of a patient's attitudes and behavior to the progression of his or her disease is emphasized. Innovative uses of traditional psychological principles are directed toward the elimination or reduction of nonproductive emotionality with the belief that the control of stress will increase the probability of successful somatic recovery.

It seems logical that stress reduction would be of benefit to some physiological complaints more than others. However, predicting the diseases which would be the most responsive to psychological intervention has been a very speculative venture (Alexander, 1950; Freedman et al., 1975; French & Alexander, 1941; Pelletier, 1977; Williams, 1968; Williams & Gentry, 1977).

Although results are often inconsistent or contradictory, some findings follow an identifiable pattern. Most notably, there appears to be considerably more evidence that

psychological stress may exacerbate a patient's disease rather than cause or antedate the initial acquisition of that disease (Weiner, 1977; Wolff, 1968).

Accordingly, Wolff maintains that disease processes should not be considered to be psychogenic simply because of a hypothesized origin based on psychological conflict. Instead the individual's "way of life" may be an exacerbating factor without specifically being considered causal. He further hypothesized genetic influences to be of primary importance, with the individual's attitudes and emotional life, in part, determining penetrance. However, attempts to achieve greater specificity by delineating personality profiles for each disorder have failed (Williams, 1968; Wolff, 1968; Spergel, 1972). Of more direct relevance to the current project is Spergel's explanation of a patient's response to his or her disease. He maintains this response is largely dependent on the premorbid manner in which an individual may have handled a variety of life problems. Unfortunately, efforts to differentially predict the onset of, or reaction to, a specific disease, based on prior behavioral patterns elicited by stress, have met with little success (Spergel, 1972; Williams, 1968). These failures concern specific prediction, however, and do not obscure the importance of psychological stress to the progression of a disease, whatever its nature. The following studies will serve to empirically

demonstrate the existance of this important, although incompletely understood, relationship.

Holmes and Rahe (1967) constructed a "social readjustment scale" which could be used to quantify and predict the effects of psychological stress on disease. An economic and cultural cross-section of several hundred people were recruited to assess the stressfulness of 43 common "life events." The participants assigned each item a stress value based on the predicted amount of adjustment needed to cope with that event. These tabulated stress values (labeled as Life Change Units) ranged from a high of 100 (death of spouse) to a low of 11 (minor violations of the law). The authors found that an individual who had accumulated 200 or more life change units in a single year was later more likely than a similar person with fewer life changes to succumb to myocardial disorder. These results were interpreted as clearly supporting the relationship between psychological stress and onset of disease. Although the Holmes and Rahe study is correlational, with an alternate interpretation of the data being that early and undiagnosed psychiatric or physiological disturbances may themselves lead to stressful life changes, a caution to therapists is recommended: Treatment programs should be avoided which might elevate an individual above the 200 unit level.

While the work of Holmes and Rahe began by focusing on psychological stressors and subsequently monitoring the associated incident of disease, others have begun by first looking

at a particular disease and then searching retrospectively for a premorbid personality configuration, or the presence of certain conflicts which might consistently antedate that disease. Treuting (1962) tried to delineate such a profile for patients with diabetes mellitus. Theorizing that emotional stresses could precipitate the disease, Treuting hypothesized that diabetes would be disproportionately represented among highly stressed populations, such as soldiers in wartime. However, the data did not support this belief (Hinkle & Wolff, 1952) and Treuting therefore theorized that perhaps only certain personality types would succumb to diabetes mellitus when under stress. Attempts to delineate a premorbid personality specific to the diabetic, however, were also unsuccessful (Treuting, 1962).

Although Treuting's data did not verify the hypothesized relationship between personality type, stress, and the onset of diabetes mellitus, more positive results have been found regarding the effect of stress on those already afflicted. Schless and von Laveren (1964) confirmed earlier findings by Rosen and Lidz (1949), that stress can aggravate diabetes, either through physiological change or by leading the patient to neglect the proper management of his or her disease. Hinkle et al. (1952) have demonstrated that a stressful interview designed to threaten the dependency, affectional, and emotional needs of a diabetic, elevate blood ketosis. A similar stress-produced metabolic shoft also is found in nondiabetics.

If such data are accurate, then a treatment strategy designed to alleviate the stress reaction in favor of a more homostatic, relaxed state could have a positive effect on the diabetic's symtomatology. Fowler, Budzynski, and Vendenbergh (1976) supported such an observation by using electromyographic biofeedback relaxation training, and verbal relaxation tapes with a 20-year-old chronic diabetic. Decreased levels of maintenance medication and fewer episodes of ketosis resulted. The average dose of insulin needed for normal functioning was dramatically reduced (approximately 50%) and the patient described herself as decreasing in emotionality and in diabetic fluctuations. Such findings can be interpreted as suggesting that not only does stress play an important role in the progression or symptomatology of diabetes, but also that its impact may be effectively controlled via psychological intervention.

Similar results have been obtained for other endocrine disorders as well. For example, Koran and Hamburge (in Freedman et al., 1975) report the presence of high levels of psychological stress in more than 50% of the patients being treated for Cushing's syndrome. Mason's (1968a) review of numerous human and animal studies summarizes reports of consistently high levels of adrenal production of relevant corticosteroids by organisms exposed to stressful situations (e.g., novel, unpredictable, or emotionally arousing). Additionally, psychological stress seems to directly influence

certain psychological processes which in turn may exacerbate the Cushing's syndrome (Gifford & Gunderson, 1970). However, consistent with research on other diseases is the failure to delineate a specific personality configuration for sufferers of Cushing's disease.

Research on the other endocrine disorders shows a similar pattern. Psychological stress seems to be related to Addison's disease (Michael & Gibbons, 1963), hypoglycemia (Marks & Rose, 1965), and amenorrhea (Rakoff, 1968). Yet, again the relationship of stress to a patient's personality for these diseases has not been demonstrated reliably.

Although biological predispositioning may be the single most important factor for expression of coronary or cardiovascular disorders (Medalie & Goldbourt, 1976), evidence of the association of stress and these diseases has been provided by Szklo, Tonasciand, and Gordis (1976). Specifically, Bennett, Hoskins, and Hampton (1976) have shown that mental stress can evoke tachycardia and vasodilatation in the majority of subjects tested.

Stress research, with results similar to those already reported, has also been conducted with gastrointestinal disorders (Alexander, 1950; Engel, 1975; Weiss, 1972), insomnia (Dement, 1975), allergic and skin disorders (Engels & Wittkower, 1975), asthma (Creer & Renne, in press; Knappe, 1969; Stein & Schiari, 1975), and rheumatoid arthritis

(Carpenter & David, 1976; Cobb, 1959; Hartfall, 1955; Wickramasekera, Truong, Bush, & Orr, 1976).

In summarizing the literature on the relationship of stress and physiological complaints, three trends emerge. First, when stress factors antedate or aggravate a disease, they do not reliably seem to be associated with the assessed personality of the patient. Second, efforts to demonstrate that psychological stress can act as a primary precipitant of a given disease have yielded equivocal results. Third, psychological stress can have an exacerbating effect on the majority of the diseases investigated. Although it is tempting to speculate that these findings could be replicated with any physiological disorder (Freedman et al., 1975; Spergel, 1972), it is important to note that most of the relevant published literature deals only with complaints which can be categorized as psychosomatic or psychophysiological as a function of a psychogenic etiology. Perhaps only these types of illness are prone to exacerbation by psychological stress.

Alexander's (1977) conceptualization of chronic asthma is in disagreement with such a criticism. Clearly, several disorders (e.g., peptic ulcers) can be triggered by psychological stress. However, Alexander claims that research on asthma, categorized as a psychophysiological disorder (DSM-II, 1968), suggests that psychological factors can influence the actual biological pathology, i.e., hypersenitive airways. He rejects labeling asthma as a psychophysiological disorder.

He points out, however, that psychological stress can result from, and contribute to, the progression of asthma in general and specifically affectsthe frequency and severity of bronchospasms. In concert with other investigators, Alexander views psychological stress as an exacerbator rather than a precipitor of both somatic as well as psychosomatic diseases.

Apparently the assessment and treatment of subjective stress is a potentially important component of planned intervention for any physical disorder. For rheumatoid arthritics, pain is a major source of salient stress with possibly autoexacerbating effects. An effective treatment program for arthritis should therefore minimize subjective distress whether of an internal (pain) or external origin. Several possible intervention components will be suggested.

Proposed Treatment Components

An individual's response to stress is said to be as variable as the situations which produce it (Wolff, 1968). Regardless of the manner of expression, all stress reactions are characterized by physiological arousal (Selye, 1950; Williams & Gentry, 1977; Wolff, 1968). A detailed description of the autonomic biological mechanisms which underlie arousal will be omitted in favor of a experientially oriented description of the phenomenon.

Obviously, short-term physiological arousal would seem to provide an organism with an adaptive advantage in that there is a mobilization of biological defenses, such as "fight

or flight" (Selye, 1950). Increased muscle strength, respiration, heart rate, and vigilance are typically observed in states of arousal (Coleman, 1968; Williams & Gentry, 1977; Wolff, 1968). However, extreme and/or long-term arousal not only may be nonadaptive, it actually may be counterproductive.

The psychologist's most effective means of combating inappropriate, maladaptive, physical stress is by training deep muscle relaxation as a response incompatable to arousal (Suinn, 1977; Suinn & Richardson, 1971). Although several indirect approaches, such as jogging, listening to music, reading, alcohol, etc., yield a degree of relaxation, clinical applications of direct relaxation techniques allow greater control. These alternatives include physical massage, relaxation imagery, meditational instruction, biofeedback, and verbal relaxation strategies. Of these, biofeedback and verbal relaxation are the most central to the treatment programs proposed.

Biofeedback. Several researchers have reported biofeedback training to be effective in the control of psychological stress. Essentially, in biofeedback therapy a preselected biological response is mechanically or electrically monitored and transmitted, in amplified form, to the respondent via visual and/or auditory displays. Using the external feedback as a guide, the subject tries to alter his or her physiological state in a specified direction (Morris, 1976).

Love, Montgomery, and Moeller (1977) used biofeedback in an attempt to alleviate the exacerbating influence of stress on hypertension. It was hypothesized that patients trained to relax by electromyographic (EMG) feedback subsequently would show a reduction in blood pressure. The feedback procedure was continued for four weeks, with only one or two training sessions per week. At the conclusion of therapy, the subjects who received electromyographic relaxation training had significantly lower systolic and diastolic pressures than did a nontreated control group. In a later paper (Montegomery, Love, & Moeller, 1977), 23 of the original subjects were reexamined. Blood pressure readings indicated that earlier progress had been maintained or improved.

Patel (1977) used biofeedback training with three chronically hypertensive subjects and reported results similar to those found by Love et al. However, Patel suggested that benefits from relaxation therapy depended on the patient's daily practicing of the relaxation response for approximately a year.

Biofeedback also has been applied successfully to Raynaud's disease, using skin temperature as the target response. Jacobson, Hackett, Surman, and Silverberg (1973) reported a case study involving a 31-year-old male with a 3-year history of Raynaud's. After failing to affect symptomology or peripheral skin temperature with three session of hypnosis and autohypnosis, skin temperature feedback was added to the

treatment protocol. The patient was able to elevate skin temperature up to 4.3°C above a baseline measure, and color changes in both hands were observed. When retested 7 months later, the ability to control skin temperature had been maintained.

A 28-year-old woman with a chronic case of Raynaud's disease was successfully treated by Blanchard and Haynes (1975). After initial training with skin temperature biofeedback, the woman was retested, 2, 4, and 7 months later. The clinical problem of Raynaud's disease had abated.

Biofeedback also has been used in the specific treatment of the chronic pain that accompanies a variety of disorders. Physicians and physical therapists generally accept that muscle tension leads to immobility which then increases the subjective experience of pain (Fowler et al., 1975; Gentry & Bernal, 1977). Treatment in these fields often includes massage, heat, traction, medication, and ultra-sound (Williams & Gentry, 1977). Sifnificantly, these techniques do not require the patient to learn relaxation skills which could be used outside of the clinical setting. Compromising generalization of effect, on the other hand, biofeedback training teaches the patient a method of breaking the pain-tension-pain cycle. By providing information about a physiological system that covaries with tension, such as electrical activity in muscles or skin temperature, the patient is trained to recognize and modify deviant states which might exacerbate pain. Relaxation

is still the goal, as it is considered incompatible with physiological tension.

Gentry and Bernal (in Williams & Gentry, 1977) have reported two case studies which illustrate the use of biofeedback-facilitated relaxation as a treatment for chronic pain. Case One involved a 42-year-old man who complained of lower back pain. On a scale of 0 (no pain) to 6 (severe pain) the patient rated himself at an average of 4.82 over a 2-week pretreatment baseline. After the session of electromyographic relaxation training, the muscle tension in his subject had fallen from an average of 7.0 μV to an average of 4.1 μV. Self ratings of pain fell to an average of 3.62. When reexamined 6 weeks after training, the patient's progress had been maintained.

A 39-year-old woman who complained of neck and shoulder pain aggravated by phlebitis was involved in the second case. At the initial treatment session, subjective self ratings of pain averaged 4.73 on a 7-point scale. Her electromyographic baseline averaged 10.8 μV. By the end of the first session the average electromyograph had decreased over 54% when compared to the baseline level. Subjective ratings of pain had decreased by 31%.

Controlled, experimental evidence showing the efficiency of biofeedback as a treatment for chronic pain is also available. Peck and Kraft (1977) is exemplary. Eighteen patients with frequent tension headaches, eight with back and shoulder

pain, and six with temporomandibular joint pain were treated. All 32 patients learned to relax via electromyographic biofeedback. Pain correspondingly declined significantly in 12 of 18 patients with tension headaches, and one of eight patients with back pain complaints. Additionally, three headache patients, three back and shoulder pain patients, and two temporomandibular joint pain patients reported slight relief. While efficacy varied as a function of the disorder, these results support electromyographic biofeedback as a viable treatment for tension-related pain (Hutchings & Reinking, 1976; Reeves, 1976).

Additional support is found in the results of Budzynski, Stoyva, Adler, and Mullaney (1973). Treating pain from tension headaches, 18 patients underwent a 2-week baseline period during which self-report headache evaluations and two electromyographic baselines were taken daily. Then, patients were assigned randomly to three groups. Group One received electromyographic feedback twice a week and was encouraged to practice the relaxation response at home. Group Two also was seen twice a week, but received no feedback training and only was encouraged to relax during the session. Taped auditory feedback generated by the first group's responding was played, not as sham feedback but with the explanation that it would help keep out intrusive thoughts. The third group received no treatment but was required to keep daily headache data. Data from the 8 weeks of treatment and from a

reexamination 3 months later revealed Group One patients reduced electromyographic base levels below those of controls and significantly reduced the intensity of reported headaches. These and other results (Wickramasekera, 1973) seem to justify the current enthusiasm for biofeedback-facilitated relaxation as a primary treatment for tension headache.

More debilitating than simple tension headaches, the migraine headache is another source of severe pain to which biofeedback therapy has been applied. Reading and Mohr (1976) trained six patients to voluntarily elevate hand temperature. It was hypothesized that increased skin temperature would correlate with muscle relaxation, and lead to a decrease in severity of the migraines reported by the participants. All patients learned to raise hand temperature, and data analysis yielded statistically and clinically significant improvement on several indices of migraine activity. Later examination attested to the stability of these findings. Similar results are reported by numerous investigators using both skin temperature and electromyographic level as target physiological responses (Morris, 1976).

Sargent, Watters, and Green (1973) also used temperature training for treating migraines. Seventy-five patients suffering from migraine pain recorded daily self-rating inventories for one month. Following this, weekly biofeedback temperature control training sessions were begun. After all patients could increase hand temperature with feedback, the

thermistors were withdrawn and the patients were asked to practice the response without equipment. Five months after the beginning of training, 80% of those participants diagnosed as true migraine patients reported at least some headache relief.

Although migraine-related pain seems responsive to skin temperature training techniques, the exact curative elements of the training are uncertain. Of course, this is crucial in understanding the disorder and the treatment, but the clinical practitioner must be concerned more with the demonstrated efficacy of skin temperature biofeedback in reducing the frequency and the pain of severe migraine headaches.

In general, the use of biofeedback in the treatment of physiological disorders has distinct advantages relative to the more traditional medical approaches. It may prove to be a valuable alternative to long-term chemotherapy (with its attendant counterproductive side-effects) for disorders such as Raynaud's disease. The active participation of the patient in the treatment of his or her illness is probably another important aspect; patients with a "type A," high-achiever personalities, or perhaps, with rheumatoid arthritis, possibly experience an exacerbation of symptomotology when relegated to an unaccustomed role of passivity in treatment. Finally, biofeedback therapy probably elicits from the patients an increased sense of responsibility and motivation. Feelings of frustration and helplessness are minimized. For

these reasons biofeedback training was included as a major component of the treatment program proposed for rheumatoid arthritis.

Verbal relaxation. A treatment strategy often coupled with biofeedback training is verbally induced relaxation (Goldfried & Davison, 1976; Rimm & Masters, 1974; Williams & Gentry, 1977). Broadly conceived, such techniques as Jacobson's (1948) progressive muscular relaxation, Schutzes and Luthe's (1969) autogenic training with imagery, and Benson's (1975) meditation to elicit the "relaxation response" are all verbally induced relaxation procedures. It has been demonstrated repeatedly that voluntary muscular relaxation markedly reduces subjective stress and anxiety (Bernstein & Borkovec, 1973; Goldfried & Trier, 1974; Jacobson, 1948; Lang, Melamed, & Hart, 1970; Paul, 1969b). Also of importance, the vocal instructions for relaxation can be tape recorded and used in clinical training sessions and in home practice (Achterberg, 1978; Lant et al., 1970). Based on an impressive pool of positive results and on the simplicity of the treatment procedure, verbal relaxation (as well as biofeedback) has been used as least as an adjunctive treatment of numerous physical disorders (Williams & Gentry, 1977). In Table 2, a brief summary of representative studies demonstrating the efficacy of verbal relaxation training is presented. In that the already reviewed literature on biofeedback training substantiates the effectiveness of inducing

Table 2

Data on the Efficacy of Verbal Relaxation Training*

Author/Date	Target Behavior	Treatment Method(s)	Results
Alexander, Miklich, & Hershkoff (1972)	Expiratory flow in asthmatic children	Systematic verbal relaxation training as compared to quiet rest	Patients receiving verbal relaxation training achieved a significant mean increase in expiratory flow versus a nonsignificant decrease for patients receiving rest only. (All patients in relaxation group were able to achieve the relaxed state)
Bassett, Blanchard & Estes (1977)	Anxiety as measured by questionnaire	Verbal relaxation training with high, medium, or low expectancy set versus soothing music with same three expectancy sets	All subjects receiving verbal relaxation training achieved significant reductions in anxiety independent of expectancy set.
Craighead (1973)	Snake fear	Systematic desensitization with and without relaxation with childhood scenes, and verbal relaxation	All subjects receiving relaxation training mastered the response, and all groups significantly reduced their fear of snakes. Treatments were equivalent.
Freeling & Shamberg (1970)	Test anxiety	Systematic desensitization with and without relaxation, and verbal relaxation	Subjects in systematic desensitization and verbal relaxation groups mastered the relaxation response. These groups were equal and both exceeded systematic desensitization without relaxation.

Table 2--Continued

Author/Date	Target Behavior	Treatment Method(s)	Results
Gershmand & Clouser (1974)	Insomnia	Systematic desensitization and verbal relaxation	All subjects mastered the relaxation response and treatments were equally effective.
McGlynn (1971)	Mouse fear	Systematic desensitization with imagery (pleasant scenes)	All subjects mastered the relaxation response and made significant progress in overcoming mouse fear. The two treatments did not differ.
Mitchell & White (1977)	Predormital insomnia	Progressive verbal mental and muscle relaxation, and cognitive control procedures	All subjects in relaxation phases mastered the response and reduced presleep tension. Intrusive cognitions were controlled by cognitive intervention.
Ratliff & Stein (1968)	Scratching associated with neurodermatis	Aversion therapy and verbal relaxation	The subjects mastered the relaxation response. Aversion therapy controlled scratching in session and the relaxation response was employed as a competing response when an urge to scratch occurred between sessions.
Tasto & Chesney (1974)	Dysmenorrhea	Verbal relaxation training with imagery of reduced menstrual pain	All patients mastered the relaxation response and reported success in obtaining relevant images. Results were positive.
Taylor (1972)	Polakuria	Verbal relaxation training and systematic desensitization	A case study in which the patient mastered the relaxation response (continued on page 29)

Table 2--Continued

Author/Date	Target Behavior	Treatment Method(s)	Results
Taylor (1972) (continued)			allowing progression through the hierarchy and ultimate control of the target behavior.
Zeisset (1968)	Interview anxiety	Verbal relaxation training versus systematic desensitization	All subjects were able to master the relaxation response and reductions in anxiety were significant in comparison to no-treatment controls. Verbal relaxation and systematic desensitization groups did not differ.

*In that verbal relaxation is an integral facet of systematic desensitization, studies investigating the effects of systematic desensitization on various disorders are considered appropriate and are included in this table.

a relaxed state in the treatment of many physical disorders, and since Table 2 documents that the same state may be induced with verbal instruction, detailed examples of verbally induced relaxation as a treatment for physical disease will not be discussed. However, it should be noted that verbally trained relaxation is an especially effective intervention strategy in disorders where immobility or muscular tension are involved. Rheumatoid arthritis is such a disorder. Thus, in the treatment program proposed, verbally trained relaxation was the second major component used to manage that disease.

Psychological Aspects of Adult Rheumatoid Arthritis

Before reviewing the arthritis literature, it should be reemphasized that research conducted on arthritic patients is fraught with methodological flaws in design and intepretation (King, 1955; Moos, 1964; Scotch & Geiger, 1962; Weiner, 1977). These contribute to the inconsistent findings often reported. Analysis of published results must be attempted cautiously, with an awareness that in many studies of rheumatoid arthritis scientific rigor was not always maintained.

As described earlier, most psychological investigators historically have taken a typological or trait-oriented approach in researching rheumatoid arthritis. Many have dealt, therefore, with delineating an "arthritic personality" or at least identifying configurations of traits, demographic variables, defense mechanisms, or conflicts unique to the population. The emphasis in this portion of the research has

been on identifying high-risk groups and predicting the likelihood of rheumatoid arthritis onset. As previously shown in Table 1, a second large segment of the literature includes reports on the patient's reaction to certain life events, including arthritis, as it affects the progression of the illness.

Among the earliest typological research was that by Halliday (1941, 1942). Believing he had identified a rheumatoid personality, Halliday described a small sample of female rheumatoids as consistently self-restricted, emotionally calm, detached, and possessing marked compulsive traits. Also, according to Halliday, most were independent and self-sufficient; strict parental discipline was common in childhood, and most lived a self-sacrificing, conscientious, quiet life. The patients reported few intensive friendships, and often exhibited a domineering personality. Unfortunately, Halliday's description of these traits was incomplete and often vague. Nonetheless Johnson, Shapiro, and Alexander (1947) reported findings which supported the compulsive aspect of the proposed configuration. Johnson et al. additionally identified a proclivity for arthritic patients to report that they had been vigorous and physically competitive as children. Supposedly this activity had been an expression of suppressed rebellious resentment against parental dominance.

Research interest in rheumatoid arthritis accelerated throughout the 1950s and early 1960s, with each investigator

suggesting additional descriptive terms and speculations about childhood dynamics. For instance, Geist (1966, 1969) maintained that rheumatoid arthritics repressed hostility and thus were intrapunitive. These results coincided with and added to a multitude of earlier studies (Cobb, 1959; Cormier, 1957; Ludwig, 1954, 1962; Mueller & Lefkowitz, 1956) characterizing rheumatoids as latently hostile, experiencing interpersonal difficulties, and as having unemotional mothers and authoritarian fathers. Eventually, the descriptors became so numerous that personality measures grew useless as diagnostic predictors of arthritis. The utility of these early reports also is minimized by the frequent omission of control procedures by the researchers, and insufficient objectivity in the definition of crucial terms (Spergel, 1972).

In the mid 1960s there was a proliferation of more adequately controlled, but still nondefinitive, studies. Several researchers attempted to delineate an arthritic personality profile with the relatively objective Minnesota Multiphasic Personality Inventory (MMPI) replacing the less reliable projective personality tests (Bourestom & Howard, 1965; Moos & Soloman, 1964, 1965a, 1965b; Nalven & O'Brien, 1968). Although many descriptors were again generated, the most consistent finding was a neurotic pattern characterized by a high degree of bodily concern, depression, and somatization. However, these findings were significant only in comparison to a normal population and were of little use in terms of differential

diagnosis when contrasted to patients with other chronic diseases (Spergel, 1972). As yet no personality profile specific to rheumatoid arthritis or any other severe disease has been found (Weiner, 1977), although there does appear to be a profile characteristic of individuals suffering from chronic disease in general. According to reviews by Spergel (1972) and Moos and Soloman (1964) this profile is characterized by intrapunitiveness, latent hostility, familial and interpersonal difficulty, shyness, rigidity, self-consciousness, an inability to express anger, masochism, and a perfectionistic standard of self-evaluation. Emotionally, moderate depression and a tendency to somatisize are not uncommon. Personality configurations of rheumatoids (although highly variable) overlap somewhat with this general chronic disease profile.

It is evident that attempts to find the arthritic personality generated much attention to childhood conflicts. Alexander (1950) and Johnson et al. (1947) hypothesized that a specific childhood psychological conflict might predispose an individual to contract rheumatoid arthritis, provided that the person also possessed a certain (unspecified) physiological substrate. Based on clinical findings that were derived primarily from studies with women, Alexander and his associates (Alexander, 1950; Alexander, French, & Pallock, 1968; Alexander, Stewart, & Duthie, 1968) currently postulate that the core conflict in rheumatoid arthritis has its genesis in

the restrictive parental attitudes experienced by the patient in childhood. The child supposedly rebels, but due to an excessive dependency on the punitive parent (typically the mother) represses the rebellion for fear of rejection. For girls, "tomboyish" behavior supposedly provides an outlet for the repressed emotions of anger and hostility. Later in life the rebellion is, according to Alexander, transferred to men and involves rejection of the feminine role in favor of aggressiveness in sports, work, and environmental control. Any guilt which may be experienced is alleviated through serving others in some way. The disease purportedly has its onset when the patient can no longer discharge her hostility by dominating others or relieve her guilt by periodically serving them.

Although Alexander's data are used to support his theory, research by other investigators has been equivocal. For example, Booth (1939), Cleveland and Fisher (196), Cobb (1959), and Meyerowitz, Jacox, and Hess (1968) support the results showing an active, competitive lifestyle, while Moos and Soloman (1965a, 1965b) and Rimon (1969) find just the opposite. Equivocal results not withstanding, the conflict specificity theory is frequently used to explain the psychogenesis of rheumatoid arthritis.

Other investigators adhere to a nonspecific conflict etiology theory (Blom & Nichols, 1953; Cobb et al., 1965; Ludwig, 1954; Robinson, 1957; Schochet et al., 1969).

Although the advocates of the nonspecific conflict theory accept hostility, resentment, overcontrol, and inhibited expression as centrally important, they do not tie these traits to definite childhood conflicts. This theory is more easily supported and more difficult to empirically refute. To illustrate, Rimmon (1969) conducted a study with female rheumatoid patients for whom no explicit environmental or physiological antecedents of the disease could be found. These sugjects were significantly more inhibited in their expression of hostility and aggression than were patients who had a clear precipitator of arthritis. They were also less aware of negative emotions. These data support a relationship between repressed emotionality and the onset of the disorder in the absence of salient antecedent conditions.

It is not surprising that those interested in identifying the defense mechanisms most common to rheumatoid arthritics consistently find denial and avoidance to be paramount (Cobb, 1959, 1965; Ludwig, 1954; Schochet et al., 1969). Reaction formation, isolation, intellectualization and undoing also were common. In related research, Gregg (1939), King (1955), Nissen and Spencer (1936), Pilkington (1956), Rothermilch and Phillips (1963), and Trevaham and Tatum (1954) have reported a lower incidence of psychosis among rheumatoid arthritics than would be expected in the general population. Nissen and Spencer (1936), for instance, did not find one case of rheumatoid arthritis among 2,200

schizophrenic patients, nor did Gregg (1939) in 3,000 autopsied psychotics. Since subjects in these and other cited studies were inpatients, it has been suggested that the protective atmosphere of the hospital may account for the absence of the disorder. Such explanations must be considered speculative due to the lack of carefully controlled research in this area.

Researchers in an extensive segment of the relevant literature have dealt with the effect psychological factors may have on the progress of the established disease, rather than describing the relationships of the etiology of rheumatoid arthritis to personality variables. As has already been demonstrated, considerable data exist which support the conclusion that stress, emanating from numerous sources, can have an exacerbating effect on rheumatoid arthritis, and that this effect is independent of the specific origin of the stress. Worry about financial matters, job absentism, anger, major surgery, divorce, death of a loved one, anxiety about prognosis or incapacitation, and intense competition have been shown to be related to a worsened symptom pattern (Spergel, 1972; Williams, 1968; Wyatt, 1969). A review of several selected studies should help illustrate this association.

Moos and Soloman (1964) examined rheumatoid arthritics under conditions of intense athletic competition. A rheumatoid factor count was determined from blood drawn before and

after participation in a highly competitive physical sport. The subjects who were distressed by the pending competition had a higher precontest rheumatoid count than those subjects who displayed little distress, and all participants showed an increase following their involvement in the very arousing contest.

Schochet, Lisansky, Schubart, Fiocco, Kurland, and Pope (1969), in a study of 12 subjects, found a strong relationship between the occurrence of major life crises (e.g., separation from a loved person) and the temporary exacerbation of arthritic symptoms. Similarly, Rimon (1969) uncovered an unexpected amount of psychological distress in the life-histories of 100 female patients diagnosed as having rheumatoid arthritis. The families of 25% of the patients had members with psychiatric disturbances, and 37% of the patients had come from homes where the parents had separated or divorced. Marital discord and/or sexual problems in the years immediately preceding the onset, or serious exacerbation, of the disorder were reported by 23%. Over half (55%) reported major life conflicts preceding onset and of these, 65% reported additional exacerbation related to a significant life crisis. Certainly, it would seem that the frequency with which stress precedes onset or exacerbation of rheumatoid arthritis far exceeds that which would be expected by chance. Although no unequivocal evidence exists on how this psychological stress interacts with the physiology of

rheumatoid arthritis, it is widely accepted that at least one mediator is probably increased muscle tension (Alexander, 1950; Barchiton, 1963; Gentry & Bernal, 1977; Weiner, 1977; Wyatt, 1969). This seems plausible as it is congruent with physiological models of the stress reaction (Cannon, 1929; Selye, 1950), as well as clinical observations that muscular tension often precedes sudden arthritic outbreaks (Alexander, 1968; Barchiton, 1963; Wyatt, 1969). It is well documented that muscle tension can be produced by psychological stress (Barchiton, 1963; Freidman, 1975; Moos & Engle, 1962; Morrison, Short, Ludwig, & Schwab, 1974; Rodnan, 1973; Selye, 1950; Wolff, 1968). Demonstrations of the translation of stress into muscle tension have been achieved through the electromyographic monitoring of muscle activity while intermittently presenting stressful stimuli (Moos & Engel, 1962; Southworth, 1958). Based on these findings, it may be suggested that psychological conflicts, such as those espoused by the dynamic theorists as being central to the onset and/or exacerbation of illness, are simply nonspecific sources of stress which lead to muscle tension and a subsequent aggravation of inflammed joints. A stress-muscle tension-exacerbation cycle seems plausible. As part of the present study, an attempt was made to interupt this stress cycle by training patients to respond to stressful stimuli with a relaxation response rather than by tension. Unfortunately, too little is known about the physiological components responsible for arthritic outbreaks

to warrant a high degree of specificity in the design of the study. Skin temperature biofeedback was used, and since the curative mechanisms of this technique are uncertain, it would seem presumptuous to predict that increases in peripheral skin temperature would be more beneficial than decreases. Although it is believed that the systematic induction of relaxation will be helpful in controlling arthritis, and that increases in skin temperature generally coincide with relaxation, Achterberg (1978b) reports that some patients may actually experience a decrease in skin temperature when relaxed. Additionally, although physical therapists traditionally have endorsed the application of "hot packs" to diseased joints (Jivoff, 1975-76), many therapists presently are reporting the application of ice packs (called cryotherapy) to be successful treatment techniques (Achterberg, 1978b). It is also possible that skin temperature may retard the activity of leukocytes in the area of the affected joint, thus reducing the local inflammation. Therefore, since it is uncertain whether raising the skin temperature of arthritic patients would be more beneficial than lowering it, both techniques were investigated. To better quantify whether or not muscular relaxation was actually occurring in either or both treatment groups, electromyographic measures were taken, in addition to self report. Solely a dependent variable, no feedback as to changes in muscular activity was available to patients.

Two groups of patients suffering from rheumatoid arthritis were treated. All participants received identical verbal relaxation training, but one group was taught, via biofeedback, to raise peripheral skin temperature, and the other was taught to lower it. Treatment-related changes in the patients were measured on physical, functional, and psychological dimensions. It was hypothesized that the systematic induction of relaxation and changes in peripheral skin temperature would influence the symptomology of the subjects. It was further hypothesized that effects on symptomology would differ as a function of which biofeedback training was received.

Method

Subjects

A total of 24 female rheumatoid arthritic patients participated in this study. The women were recruited from several sources in Dallas and Wichita Falls, Texas. These included the arthritic clinic at the Southwestern Medical School (Parkland), referral by private physicians in Dallas, and by physician referral and voluntary self-referral in Wichita Falls. Criteria for inclusion in the sample were: (a) a medical diagnosis of rheumatoid arthritis, (b) involvement (defined as the clinical observation of inflammation, pain, and a restricted range of motion for a given joint) in at least two joints, (c) an ongoing "maintenance" level of medication, and (d) a minimum of 1-year history of the disease. Half of the subjects (seven from Wichita Falls,

five from Dallas) were randomly assigned to a condition in which increased peripheral skin temperature training would be provided. The remaining subjects (eight from Wichita Falls and four from Dallas) were assigned to a program in which they would be taught to decrease skin temperature.

When potential participants were initially contacted they were given a brief letter of explanation about the proposed treatment programs (see Appendix A). Later, the women were called on the telephone, and those expressing interest were scheduled for an interview in which a detailed explanation of the experimental program was given. Those who agreed to participate signed a form on which they certified their consent (see Appendix B). Following this, the volunteers were interviewed about themselves, and their medical records, to determine if the volunteers met the criteria for participation. For each subject who qualified, the examiner determined and recorded a subjective evaluation about the severity of the disease.

Five dollars per visit was paid to some of the more indigent participants to help defray travel expenses. All participants remained under the care of their personal physicians throughout the study.

Apparatus

Psychosocial assessment tools. The lifestyle, familial relationships, modes of emotional expression, and other aspects of psychosocial development for the patients were

explored with a structured social-history interview (see Appendix C). To assess a patient's tendency to accept or reject responsibility for life happenings, a scale designed by Levinson (1973) to measure locus of control was also administered. The Levinson instrument was chosen because of demonstrated high reliability and validity. Additionally, it provides information about the degree to which a respondent is likely to attribute the cause of life happenings to themselves, to powerful others, or to chance. To facilitate administration, the scale was retyped so as to provide larger spaces for marking answers (see Appendix D). A health locus of control (Wallston, Wallston, Kaplan, & Maides, 1976) was also administered and is included in Appendix E. The health locus of control scale consists of 11 items worded either "internally" or "externally." The scale yields a score indicating a tendency to accept (internal) or assign (external) responsibility for current health status. The Profile of Moods State (Educational and Industrial Testing Service) was additionally given and yields a score on six dimensions of current mood state and a sample form is included as Appendix F.

Two other diagnostic instruments were used to evaluate each patient's perceptions about her disease. A questionnaire labled Image A was patterned after a questionnaire used by Achterberg and Lawlis (1978) with cancer patients (see Appendix G). As a second assessment procedure, patients were

instructed to draw an image of a healthy and a diseased joint, as well as their treatment and disease process, as they invisioned them.

Physical/Functional assessment tools. To assess physical and functional abilities, two protocols for evaluation were designed. Range-of-motion and strength of affected joints were considered in accordance with the Physical Therapy Evaluation (see Appendix H). A format was also designed to measure the performance of daily activities. Ratings of 1 (always can), 2 (sometimes can), or 3 (never can) were assigned to each of certain tasks in the areas of personal hygiene, dressing, eating, household chores, locomotion, and communication. This form was entitled Functional Evaluation of Rheumatoid Arthritis (see Appendix I).

Equipment. A Biofeedback Technology (BFT), Model 302, was used to monitor skin temperature and to provide visual and auditory feedback to patients. Muscle tension was measured and integrated by a BFT 401, an electromyograph, and a BFT 215 respectively.

Two standardized cassette tapes were used in the verbal relaxation portion of the experiment. These tapes were part of the Perceptual Program in Relaxation and Guided Imagery (Achterberg, 1978a). One tape (Pre-Biofeedback Relaxation) was intended for general use with biofeedback patients, and the other (Arthritis) was designed specifically for arthritics. The Perceptual Program in Relaxation tape provided

instructions on how to achieve the relaxation response. The emphasis was on reducing tension in major muscle groups. Relaxing sounds such as an "ocean surf" were included. The Arthritis tape repeated relaxation instructions and provided specific information about the arthritic process. Listeners were given a construct system for conceptualizing their disease, e.g., white blood corpuscles wrongly attacking other blood cells and causing inflammation. This tape was included in order to provide all patients with a minimal standard explanation of their disease. Copies of both tapes are available from the author.

The treatment area was a room measuring approximately 2.5 X 3 meters. It contained a standard hospital bed and a recliner, and a small desk and chair for the therapist.

Procedure

After all subjects had been recruited and assigned to one of the two experimental conditions, the program began. It consisted of four phases--pretreatment assessment, treatment, and mid- and posttreatment assessment. In the first session, each participant met with a trained physical therapist who evaluated her on the severity of her arthritis, using estimations of range of motion, and strength and number of effected joints, as guides. The patient also provided social-history information during rest periods throughout the examination. Specifically, data were elicited concerning: sleep patterns; work, leisure and physical activity

levels; general and specific experiences of pain; mood levels; percentage of time hurting; and portion of body hurting. These reports were illicited pre-, mid-, and posttreatment. Finally, the physical therapist completed the Functional Evaluation for Rheumatoid Arthritis form, including a timed 50-foot walk.

Next, the patient listened to the Arthritis tape after being told:

> Arthritis is a mysterious disease. We do not even know what causes it. But the real mystery lies in the different ways it affects each individual. No one knows better than you how it affects you. First of all, I am going to ask you to listen to a tape recording. On the tape will first be some relaxation instructions. The purpose of the relaxation is that we have found we can think better about our bodies when we are relaxed because our minds are actually more alert to those things. Then, the tape will give you some information on arthritis and how it is treated.

Patients then were asked to complete the Image A and to draw a normal joint, an arthritic joint, a picture of her disease, and an image of her treatment process. Specifically, the patient received the following instructions.

> Some things are difficult to describe in words, so I want you to draw for me. It doesn't matter whatsoever how well you draw, but just that you get some

> ideas on paper. First, draw the worst joint that has arthritis in it. Do this any way you want--real or fantasy--as long as it makes sense to you. Next, show on paper how your disease works inside your body. Then, draw a picture of a normal joint as it might look after the treatment had been effective or you had gotten well for some other reason. Finally, draw your treatment process as you picture it in your mind's eye.

At the conclusion of the first session, each participant was given Levinson's locus of control scale, the mood scale, and the Wallston et al. health locus of control scale and asked to complete them at home prior to returning for the treatment phase.

Patients were then treated individually in 12 30-minute sessions which occurred over a 4- to 6-week period. With the exception of the biofeedback manipulation, patients in both experimental groups received identical instructions and training.

Immediately upon entering the treatment room, the patient was asked to rate the severity of her pain on a scale ranging from 1 (minor, occasional, hardly noticeable pain) to 10 (major, constant, debilitating pain). Upon completion of the report, the patient was asked to lie down and become comfortable. Three surface electromyographic electrodes (two active, one ground) were placed on the under side of the participant's

nondominant arm and connected to the electromyographic amplifier. No attempt was made to position these over specific muscle groups. A thermistor probe was attached to the tip of an index finger and the monitoring unit was positioned so that the patient could view it easily. Since the biofeedback dial was masked, and the tone generator turned off, information about skin temperature was not available to the subject at this time.

Five minutes elapsed, after which the experimenter recorded the average integrated muscle tension in microvolts, and the terminal skin temperature. These scores constituted a baseline level against which later measurements would be compared. The peak skin temperature for the baseline period was also recorded.

Next, the taped prebiofeedback-relaxation instructions were played for the patient. At the conclusion of the tape, a second electromyograph and skin temperature measurement was recorded.

The remainder of the session was devoted to skin temperature training. The mask was removed from the monitor, and the auditory feedback generator was turned on. The following instructions were used for both experimental groups with the words in parenthesis omitted in the group trained to raise skin temperature or substituted for the immediately preceding word in the group trying to lower skin temperature.

Psychologists have recently learned that patients can control certain body processes in a way that may be helpful in overcoming various illnesses. For example, we believe that skin temperature may be related to arthritis, and that increasing (decreasing) it from time to time could lessen the severity of your symptoms. This machine you are connected to measures your skin temperature and the needle and tone will tell you whether it is staying the same or going up or down. For instance, if your skin temperature starts to go up, the tone will go down, and the needle will move to the right. If your skin temperature starts going down, the tone will go up and the needle will move to the left. Just relax and concentrate on changing the tone and/or the dial. This will produce the best results. You are to make the tone and the dial go up (down). You may listen to the tone and watch the dial or you may wish to attend to only one or the other. Please do not pinch the probe on your finger or press it against your body. Are there any question? Okay, let's begin.

At the conclusion of 20 minutes, the therapist recorded the patient's terminal skin temperature, her most extreme temperature during the training, and her posttreatment muscle

tension (averaged over 1 minute). Finally, the patient again was asked to rate the severity of her pain on the 10-point scale. After six sessions, the Levinson scale, the health locus of control scale, and the mood scale were administered.

Upon completion of the last training session, every patient was reevaluated by both the psychologist and the physical therapist. This evaluation was identical to the pretreatment assessment, with the exception that portions of the social-history interview were omitted.

Results

To summarize the effectiveness of the biofeedback training, an analysis of variance source table for the data from Sessions 1-4, 5-8, and 9-12 is presented in Table 3. These data show that while significant skin temperature changes are found, there are no differential effects relative to the treatment received. Instead, the differences are basically in the warming direction regardless of whether the subjects were in the increase or decrease temperature group during biofeedback training. One possible exception is seen in the data obtained from Sessions 9-12. The decrease temperature group shows an average drop of 3.3 degrees Fahrenheit, which according to Fisher's T test reaches significance ($p < .05$).

A review of Figure 1 graphically confirms that the majority of the change in temperature for both groups took place between the baseline and postrelaxation measures. It then appears that the biofeedback training is not a

Table 3

Analysis of Variance on Peripheral Skin Temperature for Increase and Decrease Training Groups

Source	df	MS	F
Block 1--Sessions 1-4			
Between Subjects	26		
Increase vs. Decrease (A)	1	53.33	< 1
Error B	25	65.25	
Within Subjects	54		
Temperature Changes within Blocks (B)	2	56.66	13.85**
A X B	2	6.56	1.59
Error W	25	4.09	
Block 2--Sessions 5-8			
Between Subjects	25		
Increase vs. Decrease (A)	1	.09	< 1
Error B	24	53.17	
Within Subjects	52		
Temperature Changes within Blocks (B)	2	82.75	19.85**
A X B	2	10.88	2.61
Error W	28	4.17	

Table 3--Continued

Source	df	MS	F
Block 3--Sessions 9-12			
Between Subjects	25		
Increase vs. Decrease (A)	1	3.44	< 1
Error B	24	41.20	
Within Subjects	52		
Temperature Changes within Blocks (B)	2	145.84	24.66**
A X B	2	23.04	3.89*
Error W	48	5.91	

*$p < .05$.

**$p < .01$.

significant factor in the control of the peripheral skin temperature.

That all subjects learned the relaxation response, independent of the treatment employed, is apparent from the electromyographic data analysis summarized for Sessions 1-4, 5-8, and 9-12 in Table 4. Figure 2 is a graphic representation of the linear decline of muscle tension in both groups. These data, in combination with the skin temperature data, show that while the relaxation response was learned, and thus a likely contributor to variance on other dimensions,

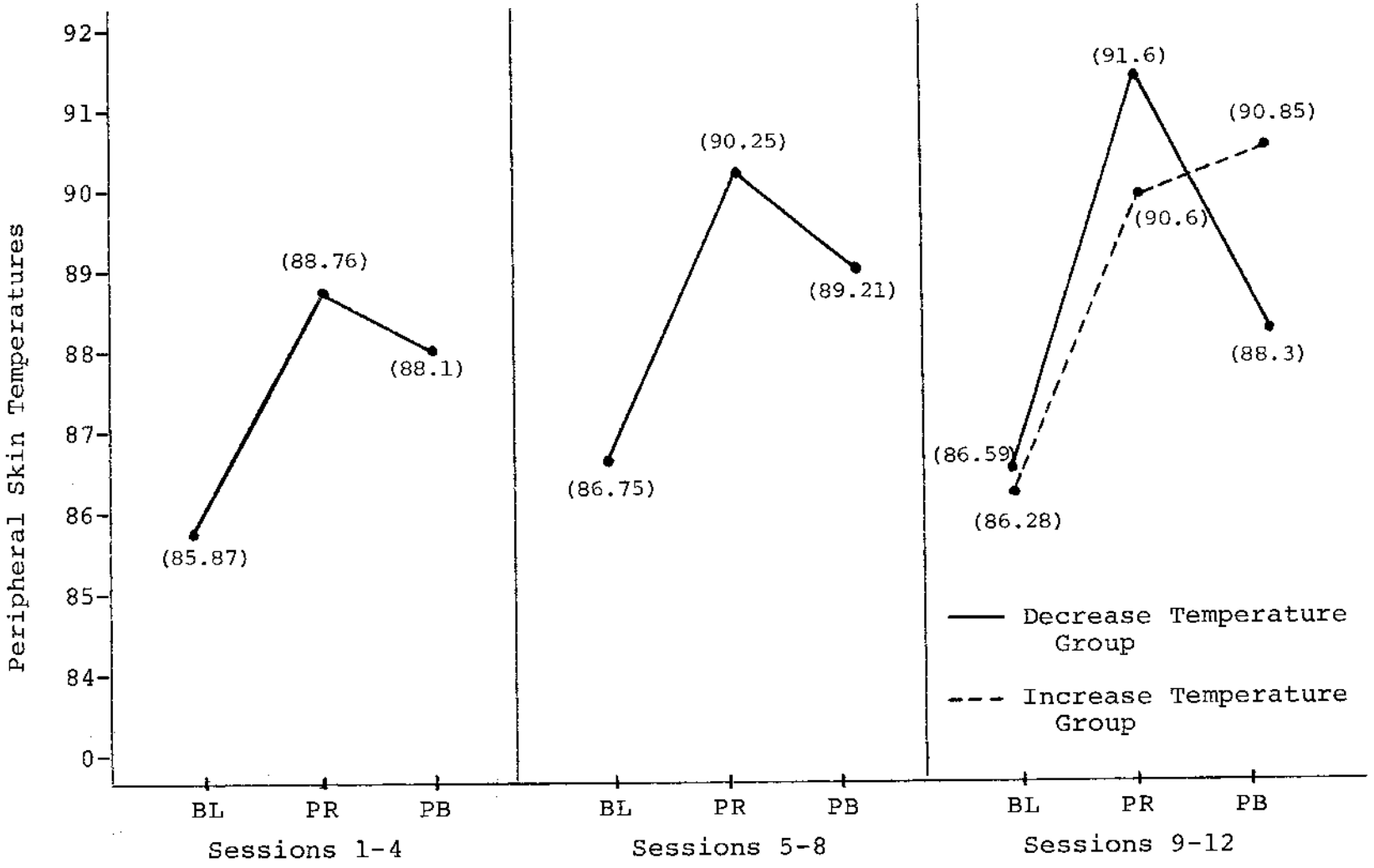

Figure 1. Peripheral skin temperature data for combined groups in Sessions 1-4 and 5-9, and plotted separately for Sessions 9-12. (BL = Baseline; PR = Postrelaxation; PB = Postbiofeedback)

the biofeedback response was not learned. Further, the learning of the relaxation response appears to be independent of the type biofeedback training received.

Table 4

Analysis of Variance for Electromyographic Data Across Blocks

Source	df	MS	F
Block 1--Sessions 1-4			
Between Subjects	19		
Increase vs. Decrease (B)	1	8.29	< 1
Error B	18	33.60	
Within Subjects	40		
EMG Changes within Blocks (A)	2	5.23	7.21**
A X B	2	1.56	2.14
Error W	36	.72	
Block 2--Sessions 5-8			
Between Subjects	19		
Increase vs. Decrease (B)	1	9.60	< 1
Error B	18	32.31	
Within Subjects	40		
EMG Changes within Blocks (A)	2	11.14	13.46**

Table 4--Continued

Source	df	MS	F
A X B	2	1.82	2.19
Error W	36	.83	
Block 3--Sessions 9-12			
Between Subjects	20		
Increase vs. Decrease (B)	1	14.72	< 1
Error B	19	27.83	
Within Subjects	42		
EMG Changes within Blocks (A)	2	7.10	8.63**
A X B	2	.49	< 1
Error W	38	.82	

*$p < .05$.

**$p < .01$.

Table 5 presents the analysis for numerical ratings of subjective units of discomfort pre- and posttreatment for increase and decrease groups across blocks. Although the groups do not differ significantly regarding initial discomfort ratings, the combined groups appear to significantly decline in initial levels across the 12 sessions. Table 5 also shows a similar pattern for posttreatment ratings with reported discomfort declining across the 12 sessions.

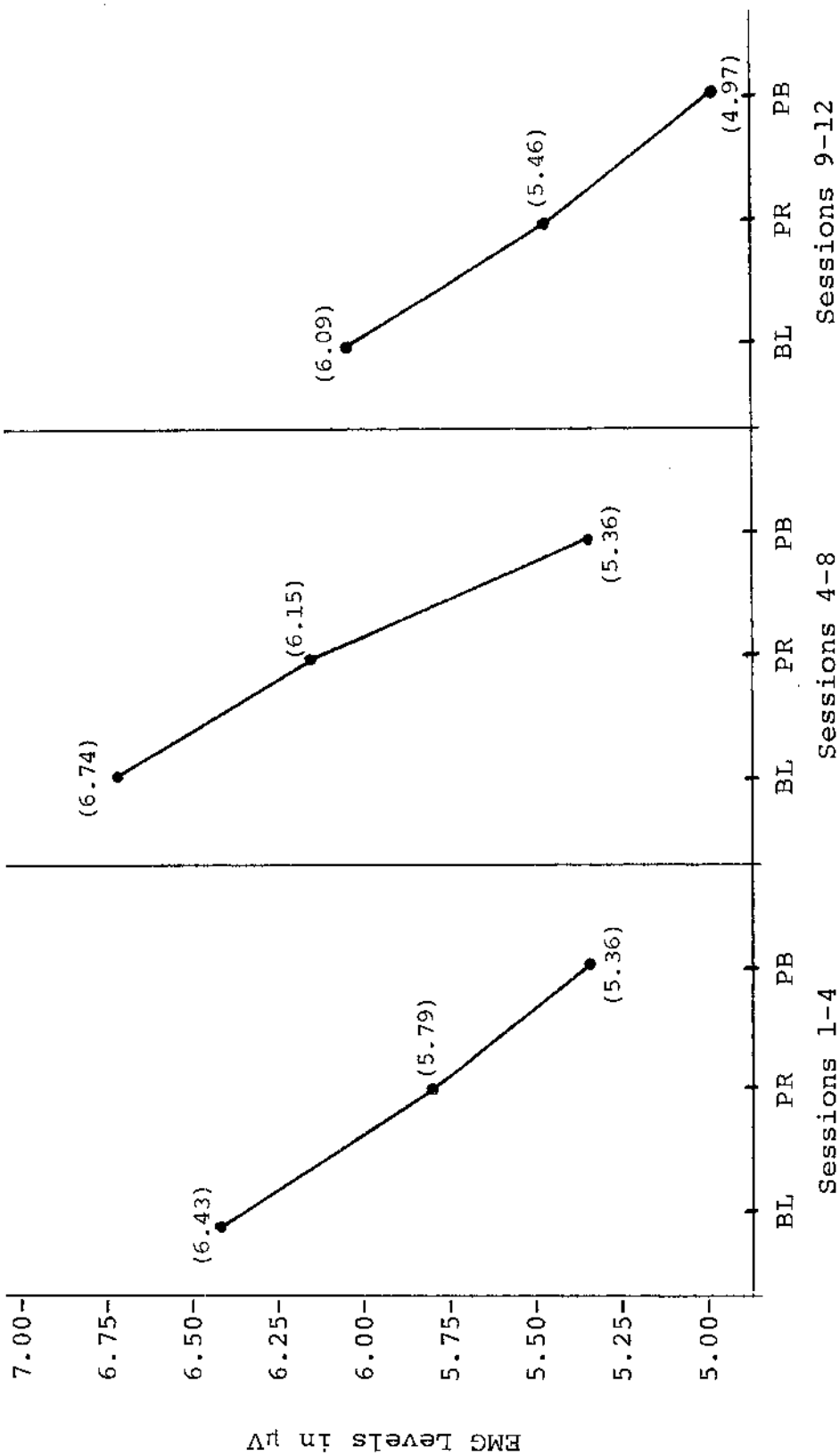

Figure 2. Electromyographic (EMG) data for combined groups. (BL = Baseline; PR = Postrelaxation; PB = Postbiofeedback)

Table 5

Analysis of Variance on Pre- and Posttreatment Discomfort Ratings for Increase and Decrease Groups Across Blocks

Source	df	MS	F
Pretreatment Discomfort Ratings			
Between Subjects	24		
Increase vs. Decrease (B)	1	309.19	< 1
Error B	23	659.97	
Within Subjects	50		
Discomfort Changes within Blocks (A)	2	998.85	14.92**
A X B	2	80.69	1.16
Error W	46	69.24	
Posttreatment Discomfort Ratings			
Between Subjects	25		
Increase vs. Decrease (B)	1	24.10	< 1
Error B	24	430.39	
Within Subjects	52		
Discomfort Changes within Blocks (A)	2	291.96	6.50**
A X B	2	96.09	2.14
Error W	48	44.90	

*$p < .05$.

**$p < .01$.

Figure 3 graphically represents the linear decline for pre- and posttreatment discomfort reports. A comparison of pre- to posttreatment measures reveals that both increase temperature (t [35] = 8.60, p .01) and decrease temperature (t [41] = 7.87, p .01) training groups report significantly less discomfort after treatment than before.

Table 6 presents the analysis of reported percentage of time during which disease-related pain was experienced and recorded from all subjects pre-, mid- and posttreatment.

Table 6

Analysis of Variance on Reported Percentage of Time Hurting Across Treatment

Source	df	MS	F
Between Subjects	17		
Increase vs. Decrease (B)	1	146.68	< 1
Error B	16	3708.00	
Within Subjects	36		
Percent Changes for Combined Groups (A)	2	414.02	9.47**
A X B	2	23.57	< 1
Error W	32	43.69	

*p < .05.

**p < .01.

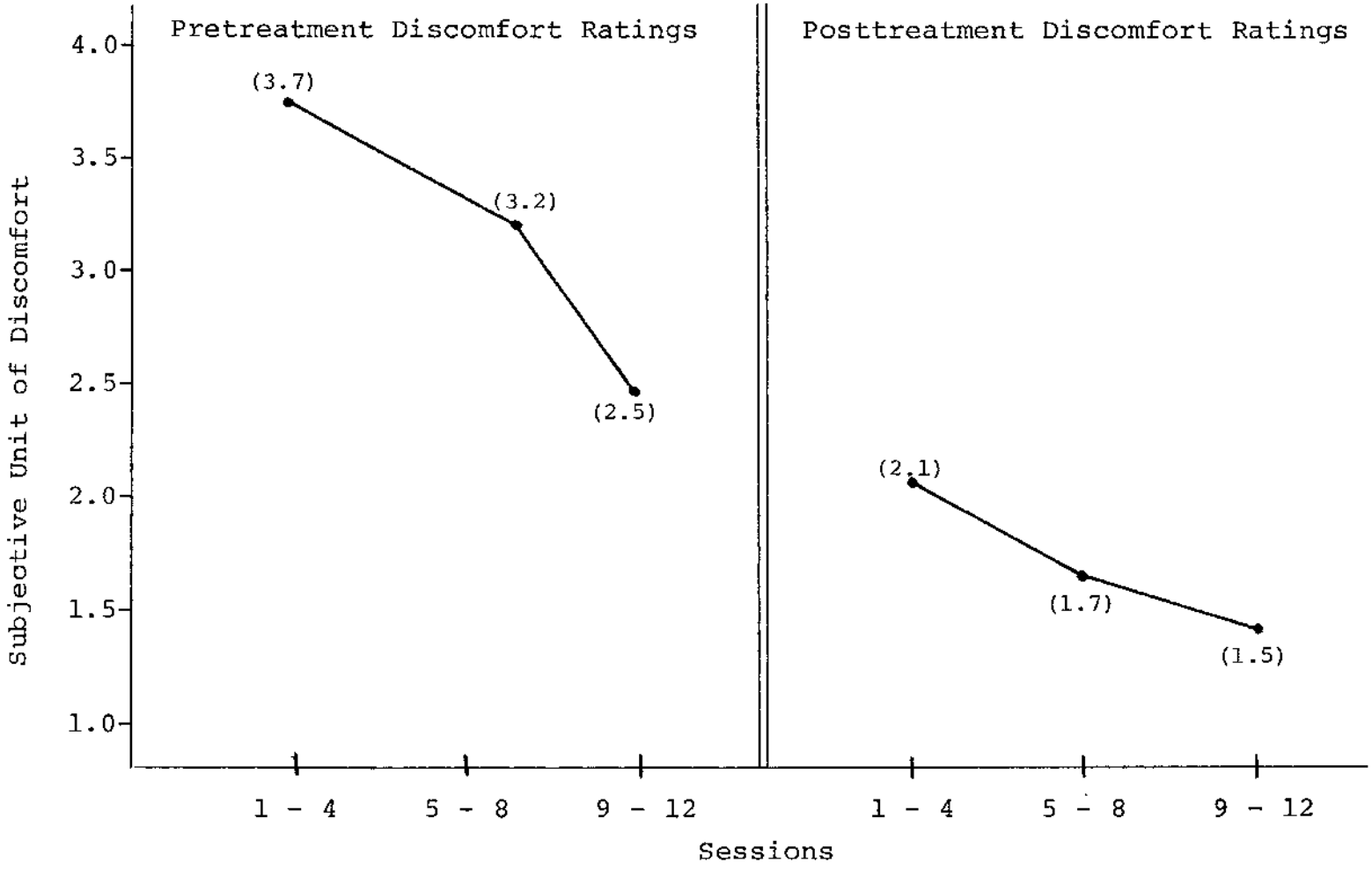

Figure 3. Pre- and posttreatment Discomfort ratings for combined groups.

While there are no differential biofeedback training effects, there is a general decline in the percentage of time during which disease-related pain is experienced across sessions when the two groups are pooled. As shown in Figure 4, the majority of the improvement occured during the second half of treatment.

Table 7 presents the analysis of numerical reports of percentage of the body hurting as a result of rheumatoid arthritis. Significant differences are indicated both between and within groups for pre-, mid-, and posttreatment intervals.

Table 7

Analysis of Variance on Reported Percentage of Body Hurting Across Treatment

Source	df	MS	F
Between Subjects	17		
Increase vs. Decrease (B)	1	13632.66	4.74*
Error (B)	16	2873.81	
Within Subjects	36		
Percent Changes for Combined Groups (A)	2	684.57	7.66**
A X B	2	29.05	< 1
Error W	32	89.29	

*$p < .05$.

**$p < .01$.

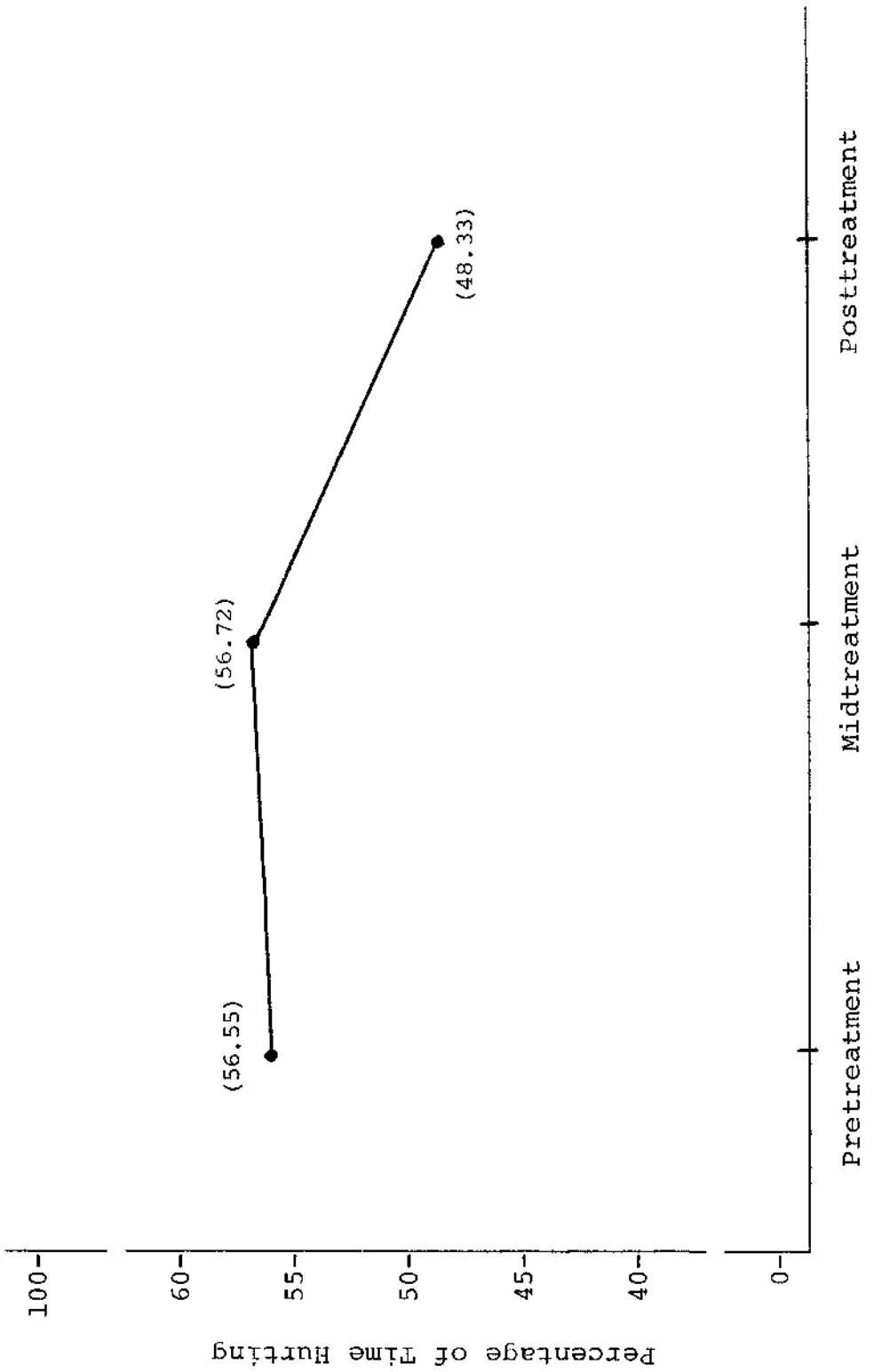

Figure 4. Percentage of time during which disease-related pain is experienced for combined groups.

The between-group differences are considered to be of only minor importance in that significant variance appears to have been contributed by pretreatment differences between the groups. Figure 5 represents the decline in the reported percentage of body hurting across the treatment sessions.

Table 8 shows pre-, mid-, and posttreatment analysis of numerical report regarding the general severity of pain experienced.

Table 8

Analysis of Variance of Reported General Severity of Pain

Source	df	MS	F
Between Subjects	18		
Increase vs. Decrease	1	7.79	1.84
Error B	17	4.22	
Within Groups	38		
Severity Changes for Combined Group (A)	2	1.75	6.23**
A X B	2	.20	.72
Error W	34	.28	

*$p < .05$.

**$p < .01$.

Again no significant differences are found between treatment groups, while a general decline in the average severity of

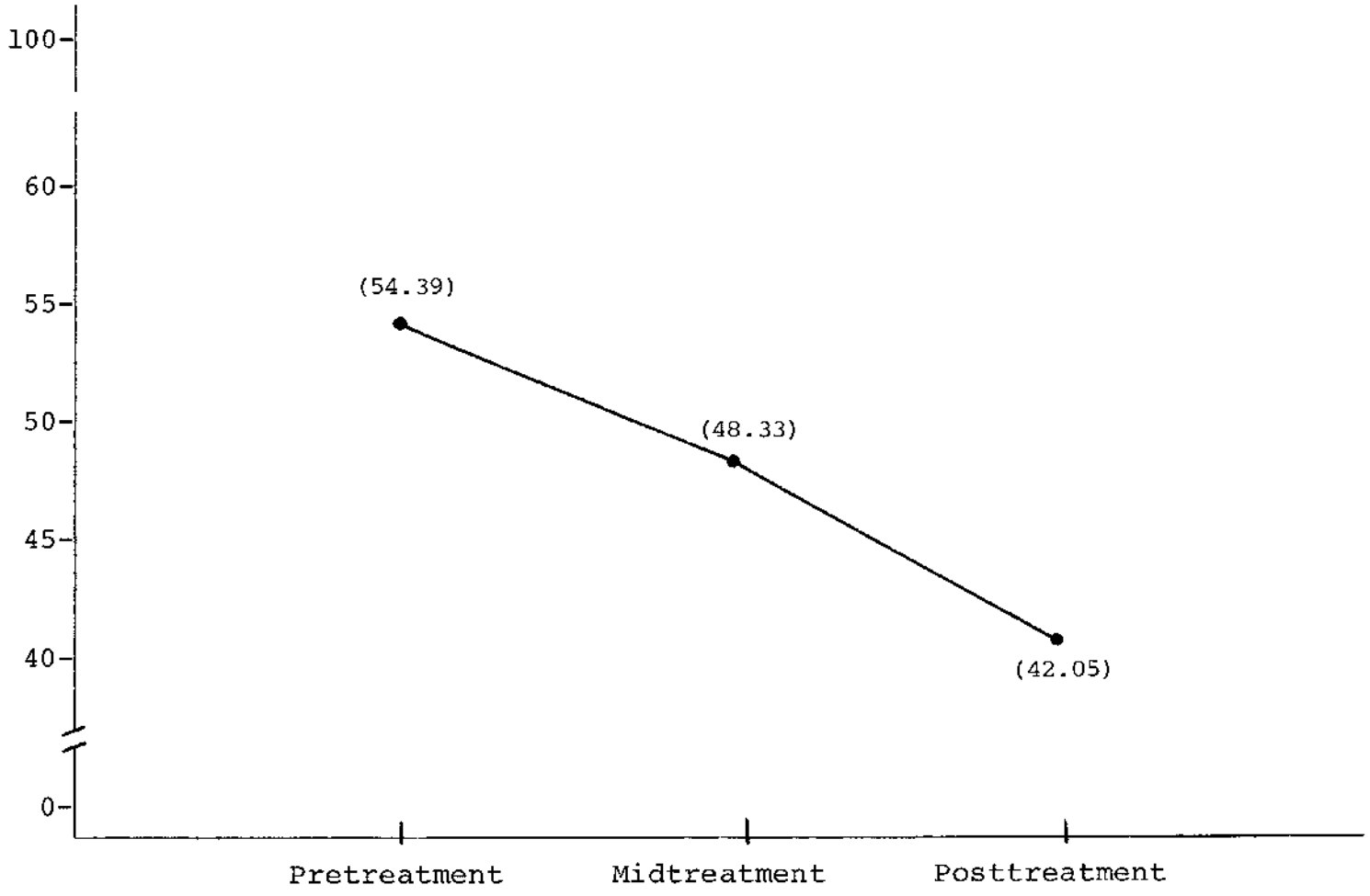

Figure 5. Percentage of the body experiencing disease-related pain for combined groups.

pain experienced is seen across sessions for the combined group. Figure 6 is a graphic representation of the pattern.

The data analysis summarizing self-report of specific (the actual time of the questionning) pain severity yields no significant results and is presented in Table 9.

Table 9

Analysis of Variance of Reported Specific Pain Severity

Source	df	MS	F
Between Subjects	17		
Increase vs. Decrease (B)	1	2386.68	< 1
Error B	16	2907.21	
Within Subjects	36		
Severity Changes for Combined Groups (A)	2	364.52	2.40
A X B	2	173.40	1.14
Error W	32	151.46	

*$p < .05$.

**$p < .01$.

Table 10 presents an analysis of the data from the Levinson locus of control scale. No significant differences were found.

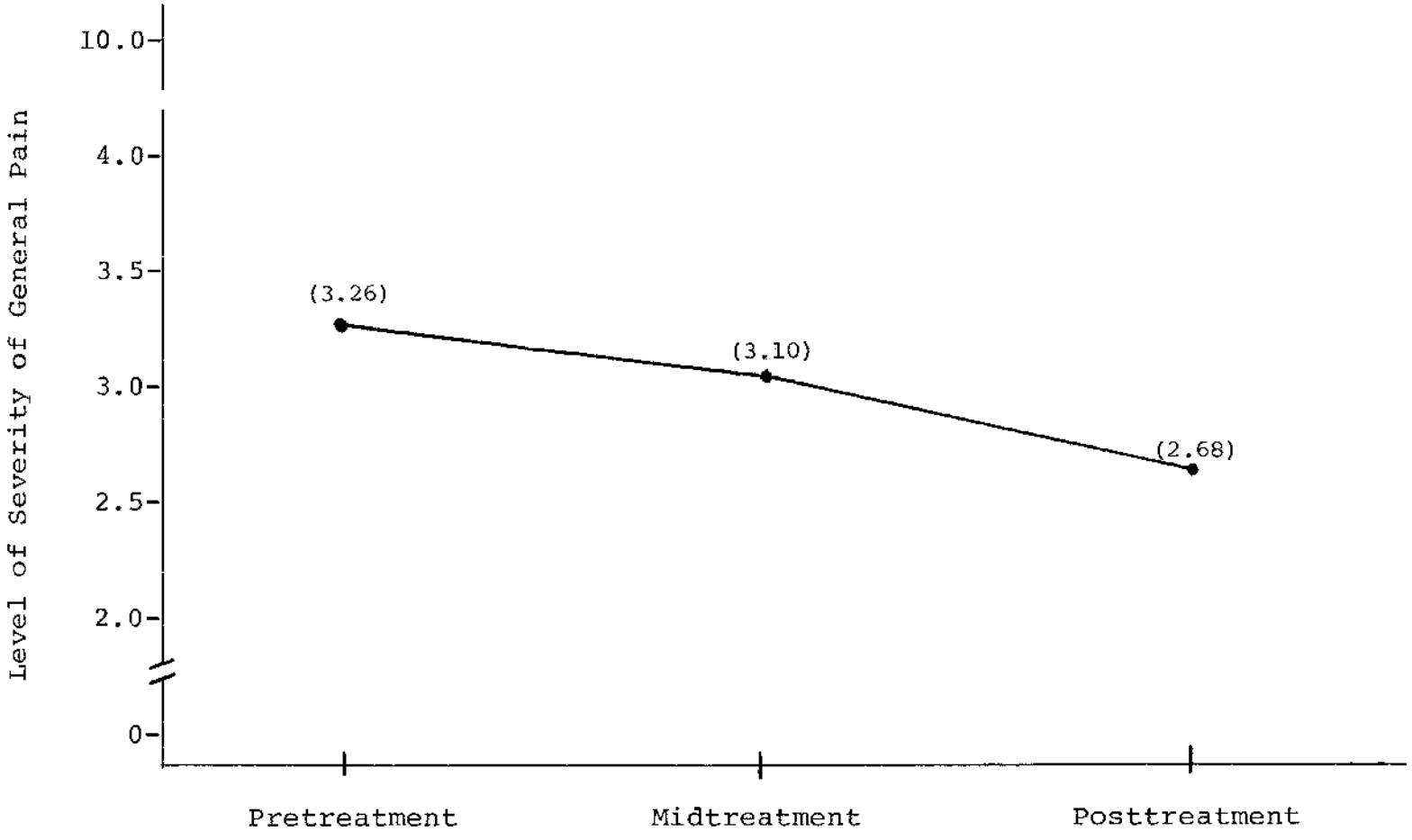

Figure 6. Level of severity of pain generally being experienced across treatment course, rated on a 0-10 scale, for combined groups.

Table 10

Analysis of Variance of Data from Levinson's Locus of Control Scales

Source	df	MS	F
Internal Scale			
Between Subjects	26		1.12
Increase vs. Decrease (B)	1	245.00	
Error B	25	219.43	
Within Subjects	54		
Dimensional Shifts for Combined Groups (A)	2	68.92	< 1
A X B	2	36.92	< 1
Error W	50	93.60	
Powerful Others Scale			
Between Subjects	26		< 1
Increase vs. Decrease (B)	1	10.60	
Error B	25	216.72	
Within Subjects	54		
Dimensional Shifts for Combined Groups (A)	2	28.53	< 1
A X B	2	17.20	< 1
Error W	50	46.32	

Table 10--Continued

Source	df	MS	F
Chance Scale			
Between Subjects	22		
Increase vs. Decrease (B)	1	371.71	2.27
Error B	21	163.58	
Within Subjects	46		
Dimensional Shifts for Combined Groups (A)	2	24.18	1.16
A X B	2	12.70	< 1
Error W	42	20.76	

The analysis of the data from the Walston et al. scale is presented in Table 11. No significant differences were found.

Table 11

Analysis of Variance of Data from the Walston et al. Locus of Control Internal and External Scales

Source	df	MS	F
Scale I (Internal)			
Between Subjects	16		
Increase vs. Decrease (B)	1	20.12	< 1
Error B	15	70.92	

Table 11--Continued

Source	df	MS	F
Within Subjects	34		
Dimensional Shifts for Combined Groups (A)	2	10.76	1
A X B	2	18.09	1.15
Error W	30	15.70	
Scale E (External)			
Between Subjects	16		
Increase vs. Decrease (B)	1	86.65	1.03
Error B	15	81.45	
Within Subjects	34		
Dimensional Shifts for Combined Groups (A)	2	30.02	1.85
A X B	2	25.24	1.56
Error W	30	16.19	

Data regarding measured images of the disease, on both the Image A questionnaire and on the subjects' drawings, are equivocal. Two independent raters evaluated each participant in the study and Section 1 of Table 12 contains a summary of the analysis of the data derived from Rater 1 which shows no change. Data generated from a second rater's evaluation are contained in Section 2 of Table 12 and, while showing no

differential effects, a shift in imagery for the combined groups is evident. Due to the disagreement of the independent raters, however, the data are considered unreliable.

Table 12

Analysis of Variance on Image A
Data from Raters 1 and 2

Source	Rater 1			Rater 2		
	df	MS	F	df	MS	F
Between Subjects	22			21		
Increase vs. Decrease (B)	1	36.07	< 1	1	15.36	< 1
Error B	21	44.30		20	46.56	
Within Subjects	23			22		
Image Shifts for Combined Groups (A)	1	7.68	1.21	1	209.45	15.44**
A X B	1	.38	< 1	1	2.27	< 1
Error W	21	6.35		20	13.56	

*$p < .05$.

**$p < .01$.

The six different measures of physical functioning are noteworthy. Two of the six measures involve sleep patterns. Table 13 is a summary of the analysis of the data derived from the subjects' self-reports about the number of hours spent sleeping each night during pre-, mid-, and posttreatment.

Table 13

Analysis of Variance on the Reported Number of Hours Slept

Source	df	MS	F
Between subjects	17		
Increase vs. Decrease (B)	1	3.13	< 1
Error B	16	5.40	
Within Subjects	36		
Shifts in Hours Slept for Combined Groups (A)	2	1.46	3.41*
A X B	2	1.68	3.93*
Error W	32	.43	

*$p < .05$.

**$p < .01$.

As seen in the table, significant differences are shown for the combined groups and the significant interaction indicates the patterns are nonparallel. Figure 7 is a graphic representation of the reported sleep habits for the two groups across the 12 treatment sessions, and shows that the increase temperature group reports a disruption in the number of hours slept at midtreatment. While the combined groups show a significant increase in the number of hours slept each night, no differential effects are seen.

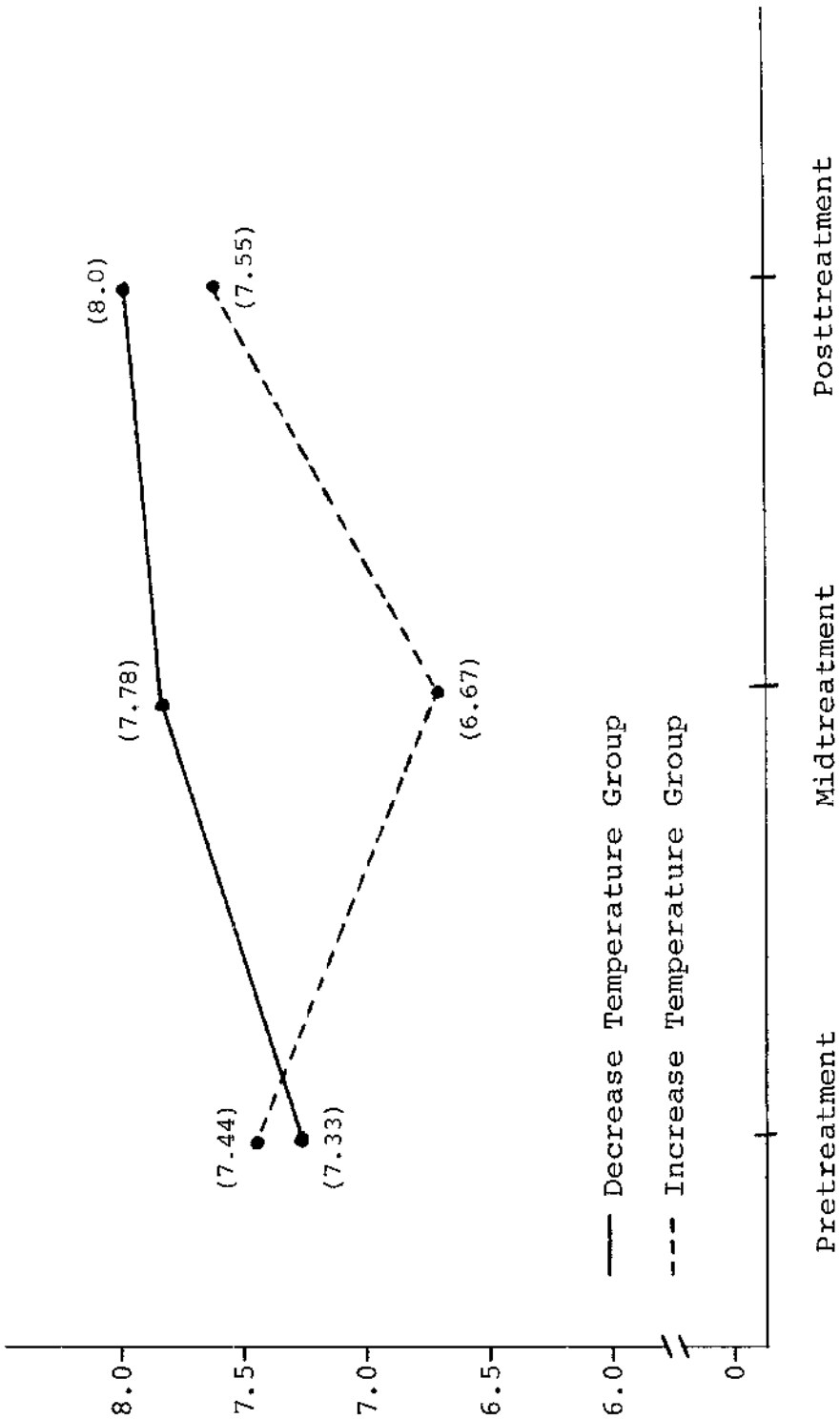

Figure 7. Hours of sleep per night for combined groups.

A second sleep-related measure involves the subjects' self-reports of the number of times they awoke during a typical night. Table 14 is a summary of the analysis of the data.

Table 14

Analysis of Variance on Reported Number of Times Awakened Per Night

Source	df	MS	F
Between Subjects	18		
Increase vs. Decrease (B)	1	23.88	1.69
Error B	17	14.13	
Within Subjects	38		
Times Awake for Combined Group (A)	2	2.73	3.43*
A X B	2	.06	< 1
Error W	34	.79	

*$p < .05$.

A significant change for the combined groups is shown in Figure 8. Again, while there is a positive and significant change for the combined group, no differential effects are seen.

A third measure involves the subjects' self-reported level of participation in work-related activities. Table 15

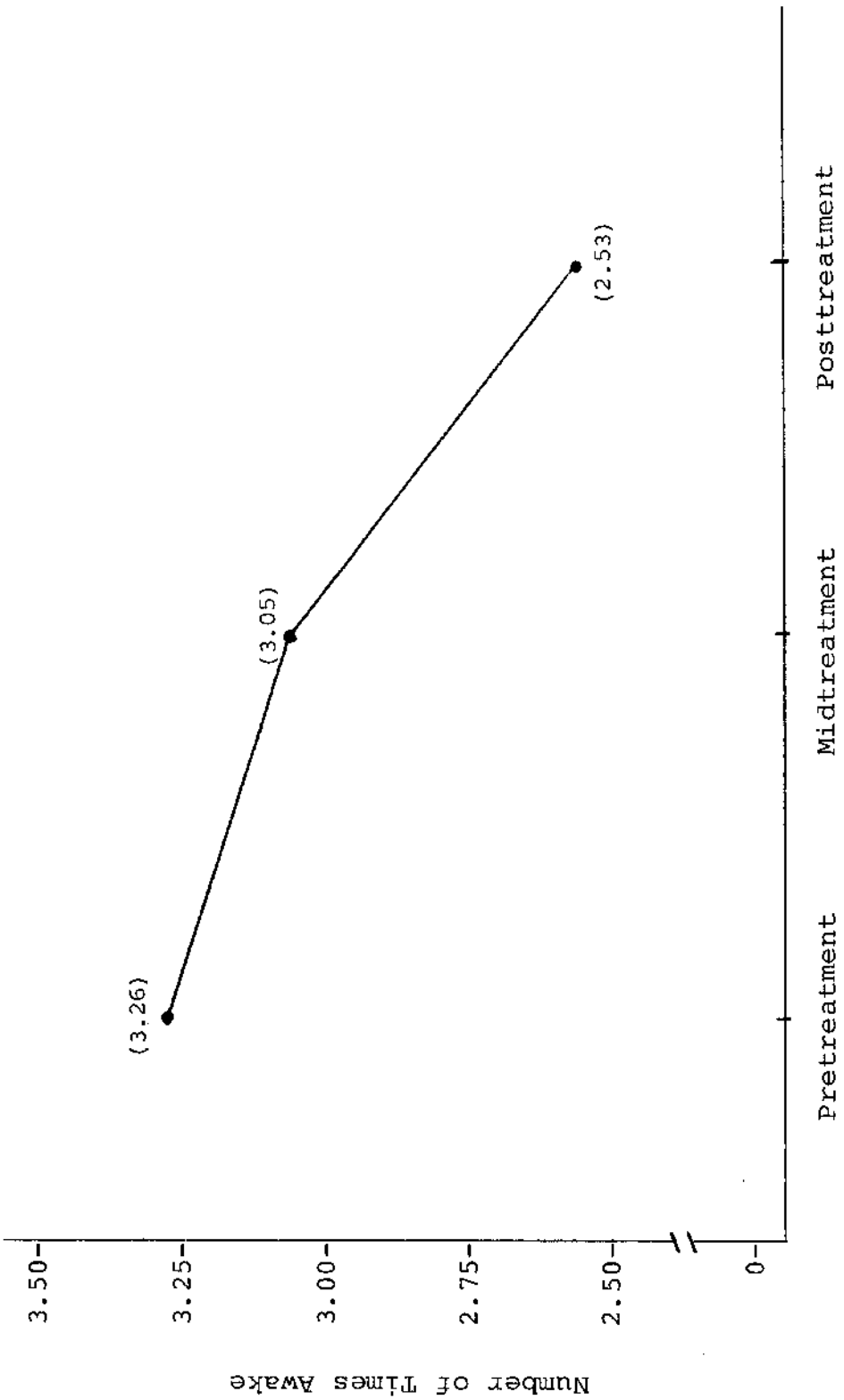

Figure 8. Number of times awake per night for combined groups.

is a summary of the analysis of these data. No significant changes across treatment sessions are in evidence.

Table 15

Analysis of Variance on Reported Changes in Work-Related Activities

Source	df	MS	F
Between Subjects	18		
Increase vs. Decrease (B)	1	.55	< 1
Error B	17	3.43	
Within Subjects	38		
Changes for Combined Groups (A)	2	.22	1.45
A X B	2	.15	< 1
Error W	34	.15	

*$p < .05$.

**$p < .01$.

Table 16 presents the analysis for numerically rated changes in subjects' reported participation in leisure-related activities. No significant changes occurred on this dimension throughout the course of treatment.

Table 16

Analysis of Variance on Reported Changes in Leisure-Related Activities

Source	df	MS	F
Between Subjects	18		
Increase vs. Decrease (B)	1	8.27	3.42
Error B	17	2.42	
Within Subjects	38		
Changes for Combined Groups (A)	2	.23	< 1
A X B	2	.02	< 1
Error W	34	.24	

Table 17 presents the analysis for numerically rated changes in physical activities. No significant changes are evident.

Table 17

Analysis of Variance on Reported Changes in Physical Activities

Source	df	MS	F
Between Subjects	17		
Increase vs. Decrease (B)	1	27.63	< 1
Error B	16	1101.16	
Within Subjects	36		
Changes for Combined Groups (A)	2	17.13	< 1
A X B	2	19.90	< 1
Error W	53	74.77	

Table 18 is an analysis of times required to walk a distance of 50 feet. No significant differences are apparent within or between groups.

Table 18

Analysis of Variance on Measured Changes in Walking Time

Source	df	MS	F
Between Subjects	22		
Increase vs. Decrease (B)	1	10.13	< 1
Error B	21	20.75	
Within Subjects	23		
Changes for Combined Groups (A)	1	9.02	1.67
A X B	1	1.71	< 1
Error W	21	5.41	

*$p < .05$.

**$p < .01$.

Table 19 presents the analysis for numerical ratings of functional performance pre-, and posttreatment. While no differences between groups are evident, there is a significant change in functional performance across the duration of treatment when the two groups are pooled. Figure 9 shows the positive direction of change, i.e., functional performance improved from pretreatment to posttreatment.

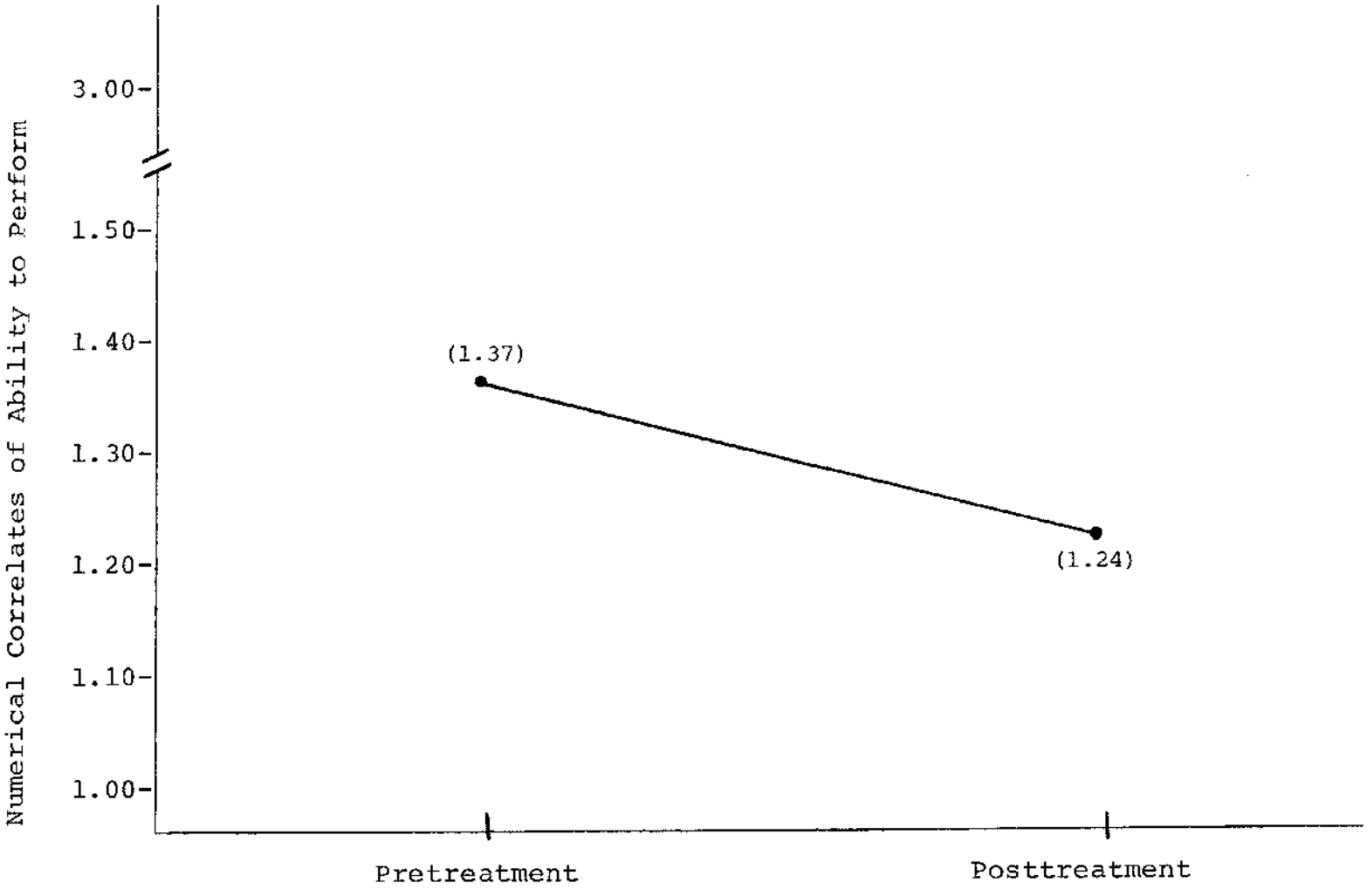

Figure 9. Performance on activities of daily living as measured on the functional evaluation for rheumatoid arthritis for combined groups. (1 = Always Can; 2 = Sometimes Can; 3 = Never Can)

Table 19

Analysis of Variance on Reported Changes in Functional Performance

Source	df	MS	F
Between Subjects	21		
Increase vs. Decrease (B)	1	26.27	1.37
Error B	20	19.12	
Within Subjects	22		
Changes for Combined Groups (A)	1	17.82	9.29**
A X B	1	.82	< 1
Error W	20	1.92	

*$p < .05$.

**$p < .01$.

Table 20 presents the analysis of the number of diseased joints recorded pre- and posttreatment. No significant changes are evident.

A second and broader-based physical therapist's assessment of each patient, pre- and posttreatment, indicates an absence of discernable improvement. A t test for differences between groups yields nonsignificant results (t [22] = 1.4).

Table 20

Analysis of Variance on Observed Changes in the Number of Impaired Joints

Source	df	MS	F
Between Subjects	23		
Increase vs. Decrease (B)	1	1416.36	3.12
Error B	22	453.43	
Within Subjects	24		
Changes for Combined Groups (A)	1	21.04	3.11
A X B	1	.04	< 1
Error W	22	6.75	

Table 21 summarizes the data analysis generated from the subjects' responding to the Profile of Moods State test administered pre-, mid-, and posttreatment. Of the six dimensions assessed, significant changes are seen only in the area of subjective tension.

Table 21

Analysis of Variance on Changes in Psychological Configuration as Measured by the Profile of Moods State Test

Source	df	MS	F
Tension			

Table 21--Continued

Source	df	MS	F
Between Subjects	23		
Increase vs. Decrease (B)	1	70.17	1.13
Error B	22	62.30	
Within Subjects	48		
Changes for Combined Groups (A)	2	32.07	6.43**
A X B	2	57.89	1.16
Error W	44	49.81	
Depression			
Between Subjects	23		
Increase vs. Decrease (B)	1	20.64	< 1
Error B	22	71.26	
Within Subjects	48		
Changes for Combined Groups (A)	2	32.90	< 1
A X B	2	117.15	2.54
Error W	44	46.06	
Anxiety			
Between Subjects	23		
Increase vs. Decrease (B)	1	69.35	< 1
Error B	22	95.86	

Table 21--Continued

Source	df	MS	F
Within Subjects	48		
Changes for Combined Groups (A)	2	224.45	3.05
A X B	2	117.95	1.60
Error W	44	73.54	
Vitality			
Between Subjects	23		
Increase vs. Decrease (B)	1	231.54	1.12
Error B	22	205.95	
Within Subjects	48		
Changes for Combined Groups (A)	2	1.61	< 1
A X B	2	102.39	1.32
Error W	44	77.40	
Fatigue			
Between Subjects	23		
Increase vs. Decrease (B)	1	58.33	< 1
Error B	22	204.01	
Within Subjects	48		
Changes for Combined Groups (A)	2	239.47	2.33

Table 21--Continued

Source	df	MS	F
A X B	2	223.80	2.17
Error W	44	102.86	
C Scale			
Between Subjects	23		
Increase vs. Decrease (B)	1	1.26	< 1
Error B	22	58.43	
Within Subjects	48		
Changes for Combined Groups (A)	2	68.06	1.96
A X B	2	41.78	1.21
Error W	44	34.64	

*$p < .05$.

**$p < .01$.

Figure 10 graphically illustrates this linear decline for the combined groups.

A second psychological measure involves the subjects' self-reports of the percentage of time during which they feel their mood is affected by their disease. Table 22 is a summary of the analysis of these data. A significant decline in the affected-mood time is seen for the combined groups.

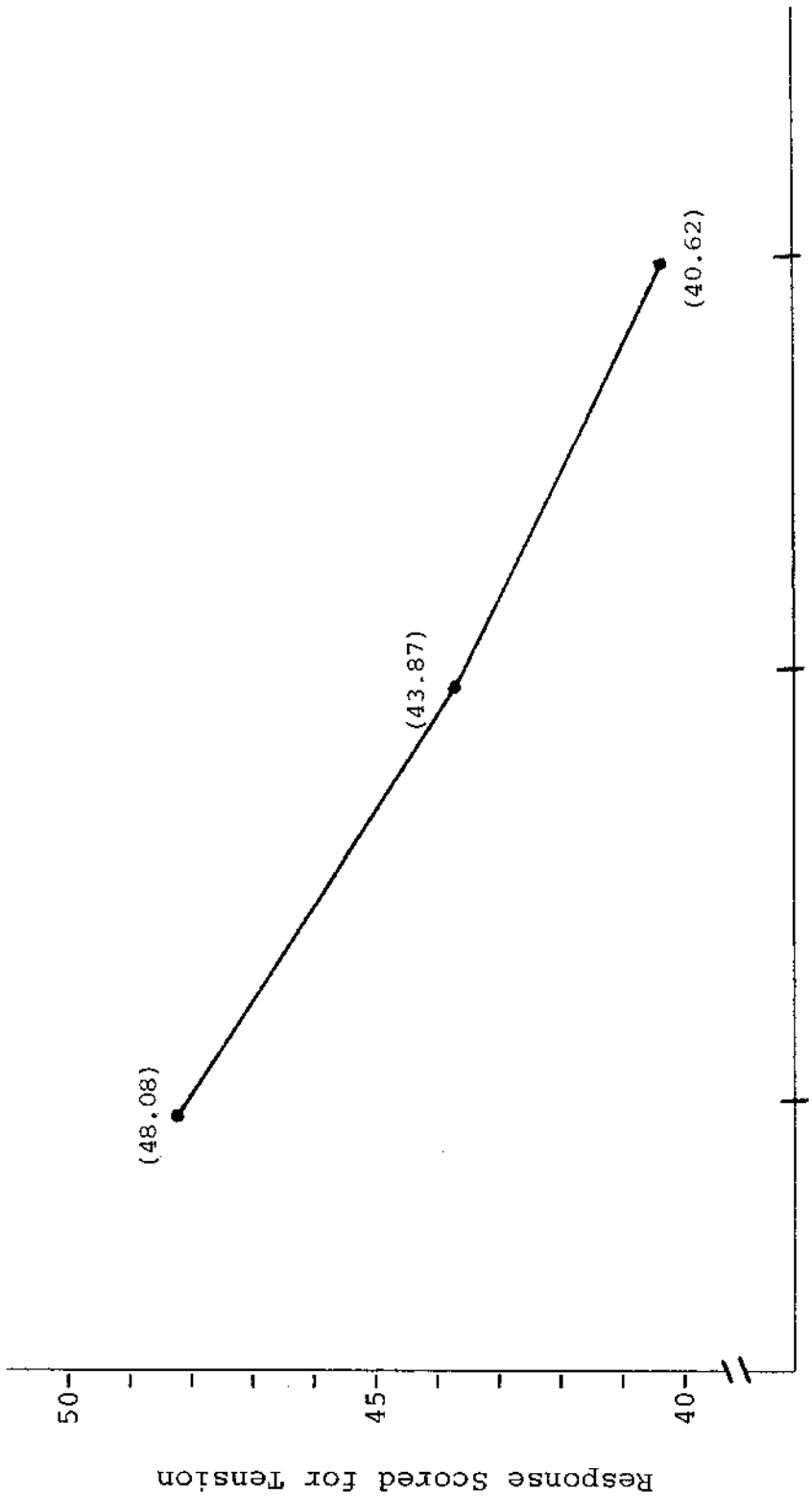

Figure 10. Level of tension experienced, as measured by the Profile of Moods Scale test, for combined groups.

Table 22

Analysis of Variance on Reported Changes in Degree of Disease-Related Mood Affect

Source	df	MS	F
Between Subjects	17		
Increase vs. Decrease (B)	1	2660.02	< 1
Error B	16	3025.71	
Within Subjects	36		
Changes for Combined Groups (A)	2	501.41	3.78*
A X B	2	5.85	< 1
Error W	32	132.59	

*$p < .05$.

**$p < .01$.

Figure 11 is a graphic illustration of the pattern. The majority of the improvement appears to be between mid- and posttreatment measures.

Discussion

The results of this research are interpreted as partially answering the question of whether relaxation training and biofeedback training of peripheral skin temperature can positively influence the functional, physical, and psychological aspects of rheumatoid arthritis. Contrary to the belief that both the relaxation and skin temperature control response would be learned and yield positive effects, only the

Figure 11. Percentage of time in which subject's mood was disease-affected for combined groups.

relaxation response appears to be correlated with improvement of the disease on certain of the considered dimensions.

Although statistically significant changes in skin temperature are shown for both the increase and decrease temperature groups, the changes are generally in the warmer direction. Further, the majority of the change is associated with relaxation training, and only statistically nonsignificant changes are recorded subsequent to the addition of the biofeedback training. A suggestive trend is seen in the performance of the decrease group in the last four training sessions. The data for the period shows that rheumatoids may, in fact, be able to consciously lower peripheral skin temperature. Electromyographic data for the same period are interpreted as confirming that these subjects were concomitantly relaxed, relative to baseline levels, and were in fact able to further decrease muscular tension during the biofeedback training. Admittedly, the performance of 12 subjects across only four sessions is scant evidence, and the overall data do support the general notion that an increase in peripheral skin temperature typically occurs in a relaxed state. However, from examining the data, it appears possible that with the application of biofeedback training this peripheral skin temperature pattern might be reversed for the rheumatoid population. The occurrence of this pattern is apparently neither widespread nor dramatic enough to differentiate the two treatment groups, as there are no reliable between-group

differences. Whether such a reversal would be desirable has yet to be answered.

The learning of the relaxation response is confirmed by examining the electromyographic data collected at baseline and subsequent to relaxation and biofeedback training. The issue of whether the relaxation methodology is efficacious is clear from the differences between baseline and post-training relaxation measures.

The impact of the unsuccessful biofeedback training is apparent since the biofeedback training is the only factor differentiating the two treatment groups. With this factor being essentially removed, between-group differences would not be expected and, in fact, are not substantiated.

Although there are no differential shifts on measures of discomfort and incapacitation, positive changes do occur generally, and are encouraging. Apparently muscular relaxation is the only active treatment component and its application may well have positive implications regarding the way in which a rheumatoid experiences her disease. Reportedly, the subjective level of pain, percentage of time spent hurting, and percentage of the body hurting all indicate reduction within and/or across treatment session with some relief being perceived by the subjects. This relief is noteworthy because of the consistent achievement without medication.

The absence of shifts, differential or otherwise, on the two locus of control measures is believed to be partially

related to the subjects' failure to learn the biofeedback response. It is possible that some subjects may have perceived themselves as failing, a perception which may well have undermined any increasing sense of mastery or internal locus of control. It should also be noted that many of the subjects had suffered for years and had been exposed to numerous treatments and "cures," so were consequently not easily swayed in their perceptions or expectations. Some success experienced in the relaxation training segment possibly averted complete frustration or perceived helplessness, perhaps offsetting any tendencies to move in the external direction.

The failure to determine any reliable changes in disease-related imagery is believed to be due to a lack of sophistication for both the subject and the experimenter. A review of the Image A protocols and the patients' drawings reveals a rather impoverished ideational system specific to the disease. Concomitantly, it is believed that the evaluation mechanisms lacked in the control and specificity necessary for quantitative analysis. Perhaps patient education would be helpful, with an aim toward achieving a level of understanding concerning both disease and treatment.

Of the six measures employed as physical criteria behavior, four are measures of active, and two are indices of more passive endeavors. Positive results are confined to the passive measures, with improvement seen in the increased number

of hours slept and decrease in the number of times a subject awoke during the night. It appears that although some changes in the life patterns occur, the subjects either are not motivated to initiate more actively oriented changes or they do not perceive themselves as improved enough to support the endeavor. It is speculated that the subjects are not motivated to attempt new patterns because increased activity levels have historically antedated exacerbations of pain and discomfort. Based on such history, this restraint may be well advised.

The subjects' tendency to initiate new physical, work, or leisure-related activities may be lacking, but they report improved performance regarding day-to-day tasks on the functional evaluation for rheumatoid arthritis. It should be noted that the data patterns for pain and discomfort are continually in the positive direction showing that increases in activity do not necessarily lead to exacerbations. Many of these tasks, however, are quite simple and the improvement might be a function of the reductions in muscular tension leading to greater flexibility of the joints. This flexibility would allow better performance without necessarily requiring greater muscular output.

The physical therapists' general conclusion (that the subjects did not significantly differ as a function of treatment received) is considered valid. Such a finding does not, however, undermine the general conclusion that the rheumatoids

felt better. Results indicate that while joint parameters are unchanged, the experience of pain in those joints is, and functional performance did improve. This finding leads to the suggestion that relaxation therapy be considered as an adjunct to medical or physical treatment, and not necessarily be employed singularly.

Consistent with all other data, no differential shifts emerge among psychological measures. Concomitant with decrements in muscular tension, however, is the general decline in psychological tension measured on the mood scale. This, coupled with the decrease in time in which the subjects' mood are disease-affected, adds to the list of indices supporting the conclusion that gains are evident regarding the patients' personal experience with rheumatoid arthritis.

While it may be that the progress reflected in the reported data is associated with relaxation training, it is possible that other factors contribute to the variance. It is possible that expectancy sets or the demand characteristics of the situation are causitive. Future research should consider the inclusion of control and/or attention/control groups.

Further consideration of biofeedback training is encouraged, as previous researchers have demonstrated that skin temperature control (at least in the warming direction) is a learnable response (Blanchard & Haynes, 1975; Jacobson et al., 1973). Further experimentation with the rheumatoid population

is necessary to determine if the manipulation, in fact, is helpful. Biofeedback training of the electromyographic control response should also be considered as a possible facilitation to learning the relaxation response.

In conclusion, it seems that relaxation training should be given serious consideration as an adjunct to other modes of treatment for the rheumatoid arthritic. It should also be noted that the relaxation training in the present study is basically automated; the training could be negotiated easily by technicians or other support personnel, or adapted to and implemented in the patient's home.

Appendix A

Letter of Explanation

Title of Study: A Comparison of the Psychological Effects of Two Adjunctive Treatments for the Rheumatoid Arthritic

Investigator's Name: Phillip C. McGraw, M. A.

Lay Statement of Insure Informed Consent:

You have been diagnosed as having Rheumatoid Arthritis. Very often patients with this diagnosis have problems in adjusting to the disease, the pain and stiffness, and the medication. This can interfere with your outlook on life. Sometimes, however, patients are able to live quite well with their disability. Regardless of how arthritis has affected you, we would like to ask your help in investigating these emotional aspects and in studying the effect of physical therapy on adjusting the problem arthritics may encounter.

Physical therapy has been used for a long time with arthritic patients, and includes such things as exercises, heat or cold packs, paraffin baths, and instruction as to improving your activities of daily living. There are no obvious risks to your health, and many patients find it helps them move and feel better.

You will be asked to participate in the study twice a week for 45 minutes each time, for about six weeks. Your progress will be checked by extensive testing by the physical

the psychologist and the physician and your records will be kept confidential. You will be asked to come in for follow-up examinations three, six, and nine months after your treatment.

Appendix B

Consent Form

Your participation in this study will help us better understand the emotional difficulties of having rheumatoid arthritis and how it can change your life, as well as to learn techniques which are most effective in dealing with these problems. With this information we can then provide more comprehensive treatment for other patients.

You are under no responsibility to continue in the treatment study should you wish to withdraw your consent, nor would failure to sign the consent form influence the care you will receive in this hospital.

Any questions you have will be fully answered.

Consent:

Having read the information statement and had the opportunity to ask questions, I hereby willingly consent to be tested.

Date _____ Signed _____
(Patient - if 18 or older)

Time _____ Witness _____

Appendix C

Social History Interview Questionnaire

I. Name_____ Age____ Date_____

II. Date of First Symptoms_____ Education_____

Sudden Onset _____ Religion_____

Insidious Onset _____ Occupation_____

Comments (ask patient details):

III. Birth Order

___1. first born
___2. second born
___3. third born
___4. fourth born
___5. other middle siblings
___6. last born (of how many___)

IV. What makes you angry?

V. What do you do when you get angry (you may answer never, sometimes, always)?

Pre	Now	
25		
___	___	1. withdraw, pout, get quiet
___	___	2. yell or curse
___	___	3. fight
___	___	4. throw things
___	___	5. work very hard at something to help get over it
___	___	6. punish yourself, dislike yourself
___	___	7. cry

VI. Physical activities when young _____

_____

_____

Initial	3 weeks	Posttreatment	Follow-up	Follow-up		
					I.	How many hours a day do you experience pain?
___	___	___	___	___		1. I do not have pain
___	___	___	___	___		2. 1-8 hours per day
___	___	___	___	___		3. 9-16 hours per day
___	___	___	___	___		4. 17-23 hours per day
___	___	___	___	___		5. Constantly
___	___	___	___	___	II.	How many hours sleep do you get each night?
___	___	___	___	___	III.	How many times do you wake up?
					IV.	Check changes in work (including housework) activities since diagnosis.
___	___	___	___	___		1. do more
___	___	___	___	___		2. no change
___	___	___	___	___		3. considerable change
___	___	___	___	___		4. drastic change, I cannot do what I did before
					V.	Changes in leisure activities
___	___	___	___	___		1. Some activities I engaged in more (Specify) _____ _____
___	___	___	___	___		2. No change. I participate in the same activities as before.
___	___	___	___	___		3. Some activities (but not all) I engage in less frequently.
___	___	___	___	___		4. I have had to curtail or decrease all leisure activities
					VI.	Pain/discomfort scales (0-100)
___	___	___	___	___		1. pain severity
___	___	___	___	___		2. physical activity
___	___	___	___	___		3. percent of time pain felt

Initial	3 weeks	Posttreatment	Follow-up	Follow-up	
___	___	___	___	___	4. effect on mood
___	___	___	___	___	5. percent of body hurting

Appendix D

Levinson Locus of Control Scale*

1. Whether or not I get to be a leader depends mostly on my ability.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

2. To a great extent, my life is controlled by accidental happenings.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

3. I feel like what happens in may life is mostly determined by powerful people.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

4. Whether or not I get into a car accident depends mostly on how good a driver I am.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

5. When I make plans, I am almost certain to make them work.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree

*Revision of form used here to show sample of items and responses.

___ Slightly agree
___ Agree somewhat
___ Strongly agree

6. Often there is no chance of protecting my personal interest from bad luck happenings.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

7. When I get what I want, it's usually because I'm lucky.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

8. Although I might have good ability, I will not be given leadership responsibility without appealing to those in positions of power.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

9. How many friends I have depends on how nice a person I am.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

10. My life is chiefly controlled by powerful others.

___ Strongly disagree
___ Disagree somewhat
___ Slightly disagree
___ Slightly agree
___ Agree somewhat
___ Strongly agree

Appendix E

Health Locus of Control[a]

1. If I take care of myself, I can avoid illness.
2. Whenever I get sick it is because of something I've done or not done.
3. Good health is largely a matter of good fortune.
4. No matter what I do, if I am going to get sick, I will get sick.
5. Most people do not realize the extent to which their illnesses are controlled by accidental happenings.
6. I can only do what my doctor tells me to do.
7. There are so many strange diseases around that you can never know how or when you might pick one up.
8. When I feel ill, I know it is because I have not been getting the proper exercise or eating right.
9. People who never get sick are just plain lucky.
10. People's ill health results from their own carelessness.
11. I am directly responsible for my health.

[a]Wallston, Wallston, Kaplan, & Maides, 1976

Appendix F

Profile of Moods State*

Name_____ Date_____

Below is a list of words that describe feelings people have. Please read each one carefully. Then fill in one space under the answer to the right which best describes how you have been feeling during the past week including today.

		Not at all	A little	Moderately	Quite a bit	Extremely
1.	Friendly	____	____	____	____	____
2.	Tense	____	____	____	____	____
3.	Angry	____	____	____	____	____
4.	Worn out	____	____	____	____	____
5.	Unhappy	____	____	____	____	____
6.	Clear-headed	____	____	____	____	____
7.	Lively	____	____	____	____	____
8.	Confused	____	____	____	____	____
9.	Sorry for things done	____	____	____	____	____
10.	Shaky	____	____	____	____	____
11.	Listless	____	____	____	____	____
12.	Peeved	____	____	____	____	____
13.	Considerate	____	____	____	____	____
14.	Sad	____	____	____	____	____

*Revision of form used here to show sample of items.

Appendix G

Image A Questions

1. If you had x-ray vision and could see your most diseased joint; what would it look like? (Elicit as complete a description as possible).

2. How strong is that joint? (How many lbs. can it lift, etc.).

3. What would the joint feel like if you could touch it? (hard, soft, rough, porous, smooth, etc.) (let patient supply several adjectives)

4. Describe what your white blood cells around your damaged joint look like.

5. Describe the movement of your white blood cells. How fast, etc.

6. Describe the fluid around your abnormal joints. What consistency? What does it do?

7. How do healthy vs. unhealthy joints look different? Act different? Feel different?

8. Describe your treatment? What does it do?

9. Do you think it works to cure arthritis? (How?)

10. Do you think it relieves pain? How?

11. Do you think it reduces swelling? How?

12. Describe any healing you think is taking place.

13. What do you think your chances (percentage) are of returning to health? (Ask patient to be extremely honest about this)

14. (Score on symbolism)

15. (Score on overall strength/weakness of ability to do image task)

16. (Score on clinical impression of disease process)

Appendix H

Physical Therapy Evaluation
(Sample Items)

Name_____ Date_____

Age_____ Sex_____ Occupation_____

Diagnosis _____ How Long_____

Any Pain _____ Is is Constant _____

When is it Worse _____

Rate Pain on a Scale of 1 to 10 _____

1 = minor, offasional, hardly noticeable pain
10 = major, constant, debilitating pain

50' walking time _____

	ROM		STRENGTH	
SHOULDER	L	R	L	R
Flexion (160)				
Extension (40)				
Abduction (160)				
Abduction				
Internal rotation (90)				
External rotation (90)				
ELBOW				
Flexion (140)				
Extension				
FOREARM				
Pronation (90)				
Supination (90)				
WRIST				
Flexion (60)				
Extension (65)				
Ulnar deviation (45)				
Radial deviation (25)				
ANKLES				
Dorsiflexion				
Plantarflexion				
Inversion				
Eversion				

Appendix I

Functional Evaluation for Rheumatoid Arthritis (Sample Items)

Name _____ Date _____

Dominant Hand _____

Score Activities on Scale of 1 to 3

1 = Always can
2 = Sometimes can
3 = Never can

ACTIVITIES	SCORE	COMMENTS
PERSONAL HYGIENE		
1. Wash hands, face		
2. Brush teeth		
3. Shave or make up		
DRESSING		
1. Take clothes from closet		
2. Put on, remove button blouse		
socks or hose		
slacks or shorts		
3. Wind watch		
EATING		
1. Pass food at table		
2. Use salt shaker		
3. Cut with knife		
HOUSEHOLD		
1. Pick up object from table		
2. Wash, dry dishes (heavy pans)		
3. Empty trash		
4. Dust, wax furniture		
5. Hang up wash		
6. Carry grocery bag		
LOCOMOTION		
1. Ambulate unassisted		
2. Ambulate with crutches, cane		
3. Propell wheelchair		
COMMUNICATION		
1. Write name		
2. Use eraser		
3. Dial phone		
APPARATUS		
1. Put on, remove adaptive apparatus		

Concomitant Diseases:	Limits Activity None	Some	Greatly
1) _____	____	____	____
2) _____	____	____	____
3) _____	____	____	____

Onset of disease_____ Duration of disease_____

Patient's subjective assessment of disease:

crippled ___ somewhat disabled ___

almost crippled ___ few problems ___

disabled ___ no problems ___

Nonsteroidal antiinflammatory agents	____	____
Gold	____	____
Penicillamine	____	____
Steroids	____	____
Cytoxan/Immuran	____	____
Other - _____	____	____

Psychoactive Drugs:	Ever	Past 3 Mo.	Present
Benzodiazepams	____	____	____
Tricyclic antidepressants	____	____	____
Barbiturates	____	____	____
Phenothiazines	____	____	____
Steroids	____	____	____
Other - _____	____	____	____

Patient's present meds.:

1) _____

2) _____

3) _____

4) _____

5) _____

References

Achterberg, J. Perceptual program in relaxation and guided imagery. Dallas: Medisette, Inc., 1978. (a)

Achterberg, J. Personal communication, February, 1978. (b)

Achterberg, J., & Lawlis, G. F. Imagery of cancer (Image A): An evaluation tool. Champaign, Ill.: Institute for Personality and Ability Testing, 1978.

Alexander, A. B., Miklich, D. R., & Hershkoff, H. The immediate effects of systematic relaxation training on peak expiratory flow rates in asthmatic children. Psychosomatic Medicine, 1972, 34(5), 388-391.

Alexander, B. A. Chronic asthma. In R. B. Williams, Jr., & N. D. Gentry (Eds.), Behavioral approaches to medical treatment. Cambridge: Ballinger, 1977.

Alexander, F. Psychosomatic medicine: Its principles and application. New York: Norton, 1977.

Alexander, F., French, T. M., & Pollock, G. H. Psychosomatic specificity: Experimental study and results. Chicago: University of Chicago Press, 1968.

Alexander, W. R. M., Stewart, S. M., & Duthie, J. J. R. Etiological factors in rheumatoid arthritis. In J. J. R. Duthie & W. R. M. Alexander (Eds.), Rheumatic diseases. Edinburgh: Edinburgh University Press, 1968. (Pfizer Medical Monographs No. 3)

American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-II), 2nd Ed. The

American Psychiatric Association, Washington, D. C., 1961.

Barchiton, J. Analysis of a woman with incipient rheumatoid arthritis. International Journal of Psychoanalysis, 1963, 44, 88-91.

Basset, J. E., Blanchard, E. B., & Estes, L. D. Effects of instructional-expectancy sets on relaxation training with prisoners. Journal of Community Psychology, 1977, 5(2), 166-170.

Bennett, P. H., & Burch, T.A. New York symposium on population studies in the rheumatoid diseases: New diagnostic criteria. Bulletin on Rheumatoid Disease, 1967, 14, 301-302.

Bennett, T., Hoskins, D. J., Hampton, J. R. Cardiovascular reflex response to apnolic face immersion and mental stress in diabetic subjects. Cardiovascular Research, 1976, 10(2), 192-199.

Benson, H. The relaxation response. New York: William Morrow & Co., 1975.

Bernstein, D. A., & Borkovec, T. C. Progressive relaxation training: A manual for the helping profession. Champaign, Ill.: Research Press, 1973.

Blanchard, E. B., & Haynes, M. R. Biofeedback treatment of a case of Raynaud's disease. Journal of Behavior Therapy and Experimental Psychiatry, 1975, 6, 230-234.

Blom, G. E., & Nichols, G. Emotional factors in children with rheumatoid arthritis. American Journal of Orthopsychiatry, 1953, 24, 101-104.

Booth, G. C. The psychological approach in therapy of rheumatoid arthritis. Rheumatism, 1939, 27, 38-44.

Bourestom, N. C., & Howard, M. T. Personality characteristics of three disability groups. Archives of Physical Medicine, 1965, 36, 626-629.

Budzynski, T. H., Stoyva, J. M., Adler, C. S., & Mullaney, D. J. EMG biofeedback and tension headache: A controlled outcome study. Psychosomatic Medicine, 1973, 35, 484-496.

Cannon, W. B. Bodily changes in pain, hunger, fear, and rage. (2nd Ed.). New York: Appleton-Century-Crofts, 1929.

Carpenter, J. O., & David, L. J. Medical recommendations: Followed or ignored? Factors influencing compliance in arthritis. Archives of Physical Medicine and Rehabilitation, 57(5), 1976, 241-246.

Cleveland, S. E., & Fisher, S. A comparison of psychological characteristics and physiological reactivity in ulcer and rheumatoid arthritis groups I. Psychosomatic Medicine, 1960, 22, 283-288.

Cobb, S. Contained hostility in rheumatoid arthritis. Arthritis Rheumatism, 1959, 2, 419-423.

Cobb, S. The epidemiology of rheumatoid arthritis. Arthritis Rheumatism, 1965, 8(11), 76-78.

Cobb, S., & Hall, W. A. A newly identified cluster of diseases--rheumatoid arthritis, peptic ulcer, and tuberculosis. Journal of the American Medical Association, 1965, 193, 1077-1079.

Cobb, S., Schull, W. J., Harburg, E., & Kasl, S. The intrafamilical transmission of rheumatoid arthritis: Summary of findings. Journal of Chronic Disease, 1969, 22, 193-195.

Coleman, J. C. Abnormal psychology and modern life (4rd ed.). Chicago: Scotts Foresman, 1968.

Cormier, B. M., Wittkower, E. D., Marcotte, V., & Forget, F. Psychological aspects of rheumatoid arthritis. Canadian Medical Association Journal, 1957, 77, 533-545.

Craighead, W. E. The role of muscular relaxation in systematic desensitization. In R. Rubin (Ed.), Advances in behavior therapy (Vol. 5). New York: Academic Press, 1973.

Creer, T. L., & Renne, C. M. Training social agents in the rehabilitation of chronically ill children. In M. E. Bernal (Ed.), Training in behavior modification. Belmont, Calif.: (in press).

Crown, S., Crown, J. M., & Fleming, A. Aspects of the psychology of rheumatoid disease. Rheumatological Rehabilitation, 1974, 13(4), 167-168.

Dement, W. C. Introduction to sleep and sleep disorders. Paper presented at the meeting of the Association for the

Advancement of Behavior Therapy, San Francisco, December, 1975.

DeWind, L. T., & Payne, J. H. Intestinal bypass surgery for morbid obesity, long-term results. Journal of the American Medical Association, 1976, 236(20), 2298-2301.

Duthie, J. J. R., Brown, P. E., Knox, J. D. E., & Thompson, M. Course and prognosis in rheumatoid arthritis. Annual of Rheumatoid Disease, 1975, 16, 411-417.

Engel, G. L. Psychophysiological gastrointestinal disorders: I. Peptic ulcer. In A. Freedman, H. Kaplan, & B. Sadock (Ed.), Comprehensive textbook of psychiatry, Vol. 2. Baltimore: Williams and Wilkins, 1975.

Engels, W. D., & Wittkower, D. Psychophysiological allergic and skin disorders. In A. Freedman, H. Kaplan, & B. Sadock (Ed.), Comprehensive textbook of psychiatry, Vol. 2. Baltimore: Williams and Wilkins, 1975.

Fowler, J. E., Budzynski, T. H., & Vandenbergh, R. L. Effects of an EMG biofeedback relaxation training program on diabetes: A case study. Biofeedback and Self Regulation, 1976, 1(1), 105-112.

Freedman, A. M., Kaplan, H. I., & Sadock, B. J. Comprehensive textbook of psychiatry, Vol. 2. Baltimore: Williams and Wilkins, 1975.

Freeling, N. W., & Shemberg, K. M. The aleviation of test-anxiety by systematic desensitization. Behavior Therapy and Research, 1970, 8, 293-296.

Freidman, A. P. Headaches. In A. Freedman, H. Kaplan, & B. Sadock, (Ed.), Comprehensive textbook of psychiatry, Vol. 2. Baltimore: Williams and Wilkins, 1975.

French, T. M., & Alexander, F. Psychogenic factors in bronchial asthma. Washington, D.C.: National Research Council, 1941.

Geist, H. The psychological aspects of rheumatoid arthritis. Springfield: Charles C. Thomas, 1966.

Geist, H. Can rheumatoid factors and anger equal arthritis? Medical World News, 1969, 10, 23.

Gentry, P. W. Psychological aspects of myocardial infarctions and coronary care. New York: Mosby, 1975.

Gentry, W. D., & Bernal, G. A. A. Chronic pain. In R. B. Williams, Jr., & W. D. Gentry (Eds.), Behavioral approaches to medical treatment. Baltimore: Ballinger, 1977.

Gershman, L. M., & Clouser, R. A. Treating insomnia with relaxation and desensitization in a group setting by an automated approach. Journal of Behavior Therapy and Experimental Psychiatry, 1974, 5, 31-36.

Gifford, S., & Gunderson, J. G. Cushing's disease as psychosomatic disorder. Perspectives in Biological Medicine, 1970, 13, 169-173.

Goldfried, M. R., & Davison, G. C. Clinical behavior therapy. New York: Holt, Rinehart and Winston, 1976.

Goldfried, M. R., & Trier, C. S. Effectiveness of relaxation as an active coping skill. Journal of Abnormal Psychology, 1974, 83, 348-355.

Gregg, D. The paucity of arthritis among psychotic patients. American Journal of Psychiatry, 1939, 95, 853-854.

Halliday, J. L. The concept of psychosomatic rheumatism. Annual of Internal Medicine, 1941, 15, 666-673.

Halliday, J. L. Psychological aspects of rheumatoid arthritis. Proc. R. Soc. Med., 1942, 35, 71-76.

Hartfall, S. J. Stress factors in the etiology of the rheumatoid disease. British Journal of Physical Medicine, 1955, 18, 16-21.

Heisel, J. S. Life changes as etiological factors in juvenile rheumatoid arthritis. Journal of Psychosomatic Research, 1972, 16, 411-417.

Hinkle, L. E., & Wolf, S. A. A summary of experimental evidence relating life stress to diabetes mellitus. Journal of Mt. Sinai Hospital, 1952, 19, 537-543.

Holmes, T. H., & Rahe, R. H. The social readjustment scale. Journal of Psychosomatic Research, 1967, 11(3), 213-217.

Hutchings, D. F., & Reinking, R. A. Tension headaches: What form of therapy is most effective? Biofeedback and Self-Regulation, 1976, 1(2), 183-190.

Jacobson, E. You must relax (3rd ed.). New York: McGraw-Hill, 1948.

Jacobson, A. M., Hackett, R. P., Surman, O. S., & Silverberg, E. L. Raynaud's phenomenon: Treatment with hypnotic and operant techniques. Journal of the American Medical Association, 1973, 225, 739-740.

Jivoff, L. Rehabilitation and rheumatoid arthritis. Bulletin on Rheumatic Disease, 1975-76, 26(2), 838-841.

Johansson, M., & Sullivan, L. Influence of treatment and change of climate in women and rheumatoid arthritis: A controlled prospective study of psychological, medical, and social effects. Scandanavian Journal of Rheumatology, 1975, 9 (supplement), 1-193.

Johnson, A., Shapiro, L., & Alexander, F. Preliminary report on a psychosomatic study of rheumatoid arthritis. Psychosomatic Medicine, 1947, 9, 295-302.

Katy, S., Vignos, P. J., & Moskowitz, R. W. Comprehensive outpatient care in rheumatoid arthritis. Journal of the American Medical Association, 1968, 206, 1249-1253.

Kellgren, J. H. Epidemiology of rheumatoid arthritis. Arthritis Rheumatoid, 1966, 9, 658-671.

Kellgren, J. H. Epidemiology of rheumatoid arthritis. In J. J. R. Duthie & W. R. M. Alexander (Eds.), Rheumatic Diseases. Edinburgh: Edinburgh University Press, 1968.

Kellgren, J. H., & Ball, J. Clinical significance of the rheumatoid serum factor. British Medical Journal, 1959, 1, 523-531.

Kilroy, A. W., Schaffner, W., Fleet, W. F., Jr., Lefkowitz, L. B., Jr., Karzon, D. T., & Fenichel, G. M. Two syndromes following rubella immunization. Journal of the American Medical Association, 1970, 214, 2287-2291.

King, S. H. Rheumatoid arthritis: An evaluation of the literature. Journal of Chronic Disease, 1955, 2, 287-298.

Knappe, P. H. The asthmatic and his environment. Journal of Nervous and Mental Disease, 1969, 149, 133-139.

Koran, L. M., & Hamburg, D. A. Psychophysiological endocrine disorders. In A. Freedman, H. Kaplan, & B. Sadock (Ed.), Comprehensive textbook of psychiatry, Vol. 2. Baltimore: Williams and Wilkins, 1975.

Lang, P. J., Melamed, B. G., & Hart, J. A psychophysiological analysis of fear modification using an automated desensitization procedure. Journal of Abnormal Psychology, 1970, 76, 220-234.

Lawrence, J. S., Valkenburg, H. A., Tuxford, A. F., & Collard, P. J. Rheumatoid factor in the United Kingdom: II. Associations with certain infections. Clinical Experimental Immunology, 1971, 9, 519-528.

Levinson, R. Reliability and validity of the I, P, & C-a multi-dimensional view of locus of control. Paper presented at the meeting of the American Psychological Association, Montreal, September, 1973.

Love, W. A., Montgomery, D. D., & Moeller, T. A. Working paper number one. In R. B. Williams & D. W. Gentry (Eds.), Behavioral approaches to medical treatment. Baltimore: Ballinger, 1977.

Ludwig, A. E. Psychogenic factors in rheumatoid arthritis. Bulletin of Rheumatoid Disease, 1954, 2, 33-37.

Ludwig, A. O. Rheumatoid arthritis. In A. M. Freedman & J. J. Kaplan (Eds.), Comprehensive textbook of psychiatry. Baltimore: Williams and Wilkins, 1962.

McGlynn, F. D. Experimental desensitization following three types of instructions. Behavior Research and Therapy, 1971, 9, 367-369.

Marks, V., & Rose, F. C. Hypoglycemia. Oxford: Blackwell, 1965.

Mason, J. W. Organization of psychoendocrine mechanism. Psychosomatic Medicine, 1968, 30, 508.

Medalie, J. E., & Goldbourt, U. Angina pectores among 10,000 men. II--Psychosocial and other risk factors as evidenced by a multivariate analysis of a five-year incidence study. American Journal of Medicine, 1976, 60(6), 910-921.

Meyerowitz, S. The continuing investigation of psychosocial variables in rheumatoid arthritis. Modern Trends in Rheumatology, 1971, 2, 92-105.

Meyerowitz, S., Jacox, R., & Hess, E. Monozygotic twins discordant for rheumatoid arthritis. Arthritis Rheumatism, 1968, 11(1), 111-114.

Michael, R. P., & Gibbons, J. L. Interrelationships between the endocrine system and neuropsychiatry. International Review of Neurobiology, 1963, 5, 253-254.

Mitchell, K. R., & White, R. G. Self-management of severe predormital insomnia. Journal of Behavior Therapy and Experimental Psychiatry, 1977, 8(1), 57-63.

Montgomery, D. D., Love, W. A., & Moeller, T.A. Working paper number two. In R. B. Williams, Jr., & D. W. Gentry (Eds.), Behavioral approaches to medical treatment. Baltimore: Ballinger, 1977.

Moos, R. H. Personality factors associated with rheumatoid arthritis: A review. Journal of Chronic Disease, 1964, 17, 41-59.

Moos, R. H., & Engel, B. T. Psychophysiological reactions in hypertensive and arthritic patients. Journal of Psychosomatic Research, 1962, 6, 227-231.

Moos, R. H., & Soloman, G. F. Minnesota Multiphasic Personality Inventory response patterns with rheumatoid arthritis. Journal of Psychosomatic Research, 1964, 8, 17-21.

Moos, R. H., & Soloman, G. F. Psychologic comparisons between women and their non-arthritic sisters. I: Personality test and interview rating data. Psychosomatic Medicine, 1965, 27(7), 135-150. (a)

Moos, R. H., & Soloman, G. F. Psychologic comparisons between women and their non-arthritic sisters. II: Content analysis of interviews. Psychosomatic Medicine, 1965, 27(7), 150-165. (b)

Morris, C. G. Psychology: An introduction. Englewood Cliffs, N. J.: Prentice-Hall, 1976.

Morrison, L., Short, C., Ludwig, A. O., & Schwab, R. The neuromuscular system in rheumatoid arthritis. Electromyographic and histologic observations. American Journal of Medical Science, 1974, 214, 33-37.

Mueller, A. D., & Lefkowitz, A. M. Personality structure and dynamics of patients with rheumatoid arthritis. Journal of Clinical Psychology, 1956, 12, 143-148.

Nalven, F. B., & O'Brien, J. F. On the use of the M.M.P.I. with rheumatoid arthritic patients. Arthritis Rheumatoid, 1968, 7, 18-29.

Nissen, H. A., & Spencer, K. A. The psychogenic problem (endocrine and metabolic) in chronic arthritis. New England Medical Journal, 1936, 214, 576-579.

Patel, C. H. Biofeedback-aided relaxation and meditation in the management of hypertension. Biofeedback and Self-Regulation, 1977, 2(1), 1-41.

Paul, G. L. Outcome of systematic desensitization. II: Controlled investigations of individual treatment technique variations, and current status. In C. M. Franks (Ed.), Behavior therapy: Appraisal and status. New York: McGraw-Hill, 1969.

Peck, C. L., & Kraft, G. H. Electromyographic biofeedback for pain related to muscle tension. A study of tension headache, back, and jaw pain. Archives of Surgery, 1977, 112(7), 889-895.

Pegg, S. M., Littler, T. R., & Littler, E. N. A trial of ice-therapy and exercise in chronic arthritis. Physiological Therapy, 1969, 55, 51-56.

Pelletier, K. Mind as healer, mind as slayer. New York: Delta Books, 1977.

Phillips, P. E., & Christian, C. L. Myxovirus antibody increases in human connective tissue disease. Science, 1970, 168, 982-984.

Pilkington, T. L. The coincidence of rheumatoid arthritis and schizophrenia. Journal of Nervous and Mental Disease, 1956, 124, 604-607.

Pipineli-Potamianou, A. Stress and anxiety in psychosomatic diseases: Research on cases of rheumatoid arthritis. Transnational Mental Health Research Newsletter, 1976, 18(2), 3-6, 13-14.

Profile of moods state. (Educational & Industrial Testing Service.)

Rakoff, A. E. Endocrine mechanisms in psychogenic amenorrhea. In R. P. Michael (Ed.), Endocrinology and human behavior. London: Oxford University Press, 1968.

Ratlif, R. G., & Stein, N. H. Treatment of neurodermotetes by behavior therapy: A case study. Behavior Therapy and Research, 1968, 6, 397-399.

Reading, C., & Mohr, R. D. Biofeedback control of migraine: A pilot study. British Journal of Social and Clinical Psychology, 1976, 15(4), 429-433.

Reeves, J. L. EMG-biofeedback reduction of tension headaches: A cognitive skills training approach. Biofeedback and Self-Regulation, 1976, 1(2), 217-225.

Rimm, D. C., & Masters, J. C. Behavior therapy: Techniques and empirical findings. New York: Academic Press, 1974.

Rimon, R. A psychosomatic approach to rheumatoid arthritis. Acta Rheumatical Scandinivica, 1969, 13, 1-11.

Robinson, C. E. Emotional factors and rheumatoid arthritis. Canadian Medical Association Journal, 1957, 77, 344-357.

Rodnan, G. P. Primer on the rheumatic diseases. Journal of the American Medical Association, 1973, 224, 663-669.

Ropes, M. W., Bennett, G. A., Cobb, S., Jacox, R., & Jesser, R. A. 1958 revision of diagnostic criteria for rheumatoid arthritis. Bulletin of Rheumatoid Disease, 1958, 9, 175-182.

Rosen, H., & Lidz, T. Emotional factors in the precipitation of recurrent diabetic acidosis. Psychosomatic Medicine, 1949, 11, 211-216.

Rothermilch, M. O., & Phillips, V. K. Rheumatoid arthritis in criminal and mentally ill populations. Arthritis Rheumatoid, 1963, 6, 81-86.

Schless, G. L., & von Laveren, S. R. Diabetic acidosis precipitated by stress. Diabetes, 1964, 13, 419-424.

Schochet, B., Lisansky, E., Schubart, A., Fiocco, V., Kurland, S., & Pope, D. M. A medical psychiatric study of patients with rheumatoid arthritis. Psychosomatics, 1969, 10, 3-8.

Schultz, A., & Luthe, W. Autogenic training (Vol. 1). New York: Grune and Stratton, 1969.

Schumacher, H. R., Jr. Synovial membrane and fluid morphologic alterations in early rheumatoid arthritis: Microvascular injury and virus-like particles. Annual of the New York Academy of Science, 1975, 256, 39-43.

Scotch, N. A., & Geiger, H. J. The epidemiology of rheumatoid arthritis: A review with special attention to social factors. Journal of Chronic Disease, 1962, 15, 1037-1042.

Selye, H. The physiology and pathology of exposure to stress. Montreal: Acta, 1950.

Shafii, M. Psychotherapeutic treatment for rheumatoid arthritis. Arch. Gen. Psychiatry, 1973, 29, 14-17.

Sharp, J. T. Mycoplasmas and arthritis. Arthritis Rheumatoid, 1971, 13, 263-266.

Short, C. L., Bauer, W., & Reynolds, W. E. Rheumatoid arthritis. Cambridge: Harvard University Press, 1957.

Silverman, A. J. Rheumatoid arthritis. In A. Freedman, H. Kaplan, & B. Sadock (Ed.), Comprehensive textbook of psychiatry, Vol. 2. Baltimore: Williams and Wilkins, 1975.

Solomon, G. F., & Moos, R. H. Emotion, immunity and disease: A speculative theoretical integration. Arch. Gen. Psychiatry, 1964, 11, 19-21.

Southworth, J. Muscular tension as a response to psychological stress in rheumatoid arthritis and peptic ulcer. Genetic Psychological Monographs, 1958, 57, 337-351.

Spergel, P. The rheumatoid arthritic personality--A psychodiagnostic myth. Unpublished study, 1972.

Stein, M., & Schiavi, R. Psychophysiological respiratory disorders. In A. Freedman, H. Kaplan, & B. Sadock (Eds.), Comprehensive textbook of psychiatry, Vol. 2. Baltimore: Williams and Wilkins, 1975.

Suinn, R. M. Type A behavior pattern. In R. B. Williams, Jr., & W. D. Gentry (Eds.), Behavioral approaches to medical treatment. Cambridge: Ballinger, 1977.

Suinn, R., & Richardson, F. Anxiety management training: A non-specific anxiety control. Behavior Therapy, 1971, 4, 498-503.

Szklo, M., Tonasciasio, J., & Gordis, L. Psychosocial factors and the risk of myocardial infarctions in white women. American Journal of Epidemiology, 1976, 103(3), 312-320.

Tasto, D. L., & Chesney, M. Muscle relaxation treatment for primary dysmenorrhea. Behavior Therapy, 1974, 5, 668-672.

Taylor, D. W. Treatment of excessive frequency of urination by desensitization. Journal of Behavior Therapy and Experimental Psychiatry, 1972, 3, 311-313.

Trueting, T. F. The role of emotional factors in the etiology and course of diabetes mellitus: A review of the recent literature. American Journal of Medical Science, 1962, 244, 93-102.

Trevatham, R. D., & Tatum, J. C. Rarity of concurrence of psychosis and rheumatoid arthritis in individual patients. Journal of Nervous and Mental Disease, 1954, 120, 85-88.

Wallston, B. S., Wallston, K. A., Kaplan, G. C., & Maides, S. A. Development and validation of the health locus of control scale. Journal of Consulting and Clinical Psychology, 1976, 44(4), 580-585.

Warren, S. L., Marmor, L., Liebes, D. M., & Hollins, R. L. An active agent from human rheumatoid arthritis which is transmissible in mice. Archives of Internal Medicine, 1969, 124, 629-633.

Weiner, H. M. Psychobiology and human disease. New York: Elsevier, 1977.

Weiss, J. M. Influence of psychological variables in stress-induced pathology. Ciba Foundation Symposium, 1972, 8, 253-257.

Wickramasekera, I. The application of verbal instructions and EMG feedback training to the management of tension headache: Preliminary observations. Headache, 1973, 13, 74-76.

Wickramasekera, I., Truong, X. T., Bush, M., & Orr, C. The management of rheumatoid arthritic pain: Preliminary observations. Biofeedback Behavior Therapy and Hypnosis, 1976, 47-55.

Williams, M. H. Recovery of mycoplasma from rheumatoid synovial fluid. In J. J. R. Duthie & W. R. M. Alexander (Eds.), Rheumatic diseases. Edinburgh: Edinburgh University Press, 1968.

Williams, R. B., & Gentry, W. D. Behavioral approaches to medical treatment. Cambridge: Ballinger, 1977.

Williams, R. C., Jr. Rheumatoid arthritis as a systemic disease (Vol. 4). Philadelphia: Saunders, 1974.

Wolff, H. G. Headache and other head pain (3rd Ed.). New York: Oxford Press, 1968.

Wyatt, H. J. Psychologic factors in arthritis. In S. Light (Ed.), Arthritis and physical medicine. Baltimore: Waverly Press, 1969, 176-190.

Zeisset, R. M. Desensitization and relaxation in the modification of psychiatric patients' interview behavior. Journal of Abnormal Psychology, 1968, 73, 18-24.