MUSCULOSKELETAL DISORDERS

Donald Oken

American Handbook of Psychiatry

Musculoskeletal Disorders

Donald Oken

e-Book 2015 International Psychotherapy Institute

From *American Handbook of Psychiatry: Volume 4* edited by Silvano Arieti, Morton F. Reiser

Table of Contents

Musculoskeletal Disorders

Introduction

This chapter will discuss five clinical disorders: psychogenic rheumatism, occupational cramp, rheumatoid arthritis, parkinsonism, and gout. It is immediately apparent, therefore, that its title is a misnomer in one important sense. The basic pathophysiological processes involved in at least three of these disorders almost certainly lie outside the musculoskeletal system. A more accurate title might have referred to disorders with predominant musculoskeletal manifestations. The logic of their selection based on common symptomatic features is reinforced by a significant resemblance among the psychological factors that have been found associated with each.

The fact that the psychological findings reported for these disorders overlap poses questions about their precision, if not their validity. The similarities, however, may be quite real. If so, this raises a suspicion that the findings are similar for the same reason that the disorders are, i.e., their shared manifestations. The presence of any disease that causes severe limitation of motility, as well as pain and related symptoms in the muscles and joints, will produce psychological reactions to these symptoms. Perhaps the greatest problem in obtaining and interpreting data on patients with

musculoskeletal disorders lies in separating these consequences of disease from factors which are antecedent. Some of the data does bear on factors antedating onset and these are of particular interest. But the entire subject area contains a wealth of findings that merit interest, and illustrate both the successes and difficulties of the psychosomatic approach.

Prior to the consideration of the disorders themselves is a section which reviews the available data in the area of basic psychophysiology most relevant to the musculoskeletal system, i.e., muscle tension. The rationale for this choice can be questioned for the very reason and to the same degree that the chapter may be mislabeled. Nevertheless, muscle tension does seem to have direct relevance to two of the disorders, and indirect relevance to at least two others. It also represents an important area of psychophysiology in its own right. Other relevant areas of psychophysiology will not be neglected, but will be considered in direct relation to the disorders in which they may be implicated.

Basic Psychophysiology

It hardly needs to be pointed out that posture, gait, facial expression, and gestures represent basic characteristics of the personality. Individuals have their own relatively unique styles of motor expression which are readily recognizable both to themselves and those who know them. The style of a

person's motor activity also provides revealing manifestations of his psychological attributes. Many studies (reviewed by Plutchik) are unanimous in showing that motor expression reliably communicates characteristic attitudes.

Freud pioneered our understanding of the significance of "meaningless" motor acts as manifestations of specific unconscious motivational forces. Psychoanalysts who followed have provided further insights about the meanings behind various movements and postures. The individualized nature of these activities makes an ideographic clinical approach such as psychoanalysis the most appropriate method for their psychological study. No comparable method existed on the motor side for a long time. Now an ingenious beginning toward a science of "Kinesics" has been launched by Birdwhistell.

Expressive behavior has obvious relevance to both normal personality and psychopathology, the domain of psychiatry. The psychosomatic approach requires a focus on psychophysiological mechanisms, rather than motor behavior. Within this framework, our interest is in muscle tension.

Muscle Tension

The term "tension," with its inherent dual meaning of a psychological as well as muscular phenomenon, implies the existence of a psychosomatic

relationship. While this may be revealing in a very general sense, a scientific understanding of that relationship requires specific, precise definition of both its psychological and somatic components. The vagueness of the term tension to represent a psychological phenomenon is obvious. It may be less evident that there has been considerable variation in its use on the somatic side as well. The common feature which appears to underlie these various usages is skeletal muscle contraction; and it seems most sensible to define muscle tension simply in that way. In so doing we bypass issues of central neurophysiological states, and ignore the highly unlikely possibility that differing types of contraction exist.

This still leaves the problem of operational definition. The methods used to measure muscle tension have varied widely. As early as 1942, R. C. Davis catalogued six different modes of measurement, each including multiple differing techniques. Davis' careful analysis led him to conclude that the best method is the electronically integrated electromyogram (EMG) recorded from electrodes on the skin. There is now general agreement with his viewpoint that the surface EMG represents the most definite, sensitive, reliable, and practically useful method of measuring muscle tension. A manual providing a detailed description of the technique is available. Care must be exerted to choose electronic components with appropriate characteristics. With such equipment, the EMG is a direct, linear correlate of isometric skeletal muscle contraction over most of its range. It appears also to correlate with subjective

feelings of muscle tension.

Nevertheless other methods continue to be used. Among these are a resonance technique which records combined motion and tension; eye-blink rate; the speed and accuracy of fine psychomotor activity; the after contraction phenomenon; several techniques for quantifying movement, including a "motility bed"; and many others. All may have value. But they are measuring different phenomena than the muscle action potentials which the EMG detects. They *cannot* be considered equivalent either to the EMG or to one another.

Another problem lies in the choice of the muscle groups to be measured. Factor analyses applied to data from several independent studies show convincingly that a *general factor* of muscle tension does exist. This indicates that there is a tendency for tension to occur to a similar degree in muscles throughout the body. However, this tendency is limited. The general factor neither includes all muscles nor does it represent close to all the variance of those it does include. Additional factor clusters also emerge from the analyses that represent more localized patterning of tension. Moreover, it has been demonstrated that *response specificity* occurs for the muscles as well as autonomic variables. This phenomenon, originally identified by Lacey, represents the tendency for individuals to react physiologically to differing stimuli in a preferential fashion, with maximum response in a particular,

differing response system. The determinants, or even correlates, of the "choice" of a specific site of muscular responsivity remain unknown. *Stimulus specificity,* the patterning of responses due to the specific effects of a given stimulus, also seems to occur. The study by Voas also indicated that muscle tension measured in seven separate sites varied not only for different individuals but within the same individual during different conditions. In any single research, therefore, confidence can be placed on the findings *only* as they apply to those muscles specifically measured. Very few studies have included EMG's from multiple sites. Yet the results from one or two sites are frequently interpreted as if they represented a valid index of generalized tension. One of the favored sites is the frontalis. Here the dangers of erroneous extrapolation are especially great, for this muscle seems to be one of those least related to the general tension factor. In a few instances there are several studies focused on a given research question which together include sufficient sites to permit a reasonable capacity to infer that generalized tension has been demonstrated. There are also, of course, questions for which measurement of tension in only a single site is relevant. If, for example, one wishes to know if a symptom arises from muscle tension, then the tension at its locus represents the only relevant measure.

Additional methodological problems arise about the selection of variables for analysis. Is the variable of choice the resting EMG level, or that reached after stimulation? Is it the mean response or the peak? Is duration of

response more important than its magnitude, or, returning to the matter of the locus of response, is it better to know when tension involves a greater number of muscle areas or the body as a whole? There are no a priori answers. All these indices have been used. Any of them may be valid (i.e., relevant) to a *given* problem. At times, the use of several adds up to a broader understanding. But the lesson is clear; each study must be examined carefully to learn precisely what was measured and how, and the results interpreted in those terms.

Muscle Tension and Personality

Systematic attempts to understand the relation of specific personality traits to muscle tension began to develop around the turn of the century. One of the first was provided by William James, who divided people into hyper- and hypoactive types; the former being excitable, hypermotile, and tense, and the latter phlegmatic, calm and relaxed. Many other observers have suggested a similar dichotomy, often emphasizing that hyperactivity occurred at times of emotional stimulation. These and related studies have been the subject of an extensive review by Iris Balshan Goldstein.

It seems likely that emotional "excitability" corresponds closely to what we would now term "trait anxiety." To be distinguished from the state of anxiety, this term refers to the characterological tendency to respond with

anxiety to many situations, including those of low threat, i.e., a chronic proneness to anxiety. The threshold may be so low that anxiety is experienced even at seeming rest. Using this better defined concept, Balshan Goldstein found that normal women, scoring high on trait anxiety scales, had higher levels of tension (in sixteen muscles) during noise stimulation than did those with low scores. This greater responsiveness was negatively related to scores for *restraint,* and positively to scores for *general activity* in the Guilford-Zimmerman Temperament Survey, but not to any of its other trait variables. In a later very similar study, this time using a patient group, Balshan Goldstein found the same relationship between EMG-measured tension and trait anxiety. Rossi also demonstrated a relation between these two variables. Trait anxiety has also been related to eye-blink rate, a measure that has been proposed as an index of overall tension.

In both of Balshan Goldstein's studies, tension levels at rest were very low for all groups. Malmo also believes that stressful stimulation usually is required to bring out the higher tension levels of more anxious people. Others have suggested that anxious individuals will have significantly greater tension even at rest. The explanation for the discrepancy may lie in the degree of success in creating sufficiently non-stimulating conditions. The anxiety-inducing effects of exposure to the psychosomatic laboratory itself makes it difficult to achieve truly basal conditions.

Trait anxiety would be expected to be associated with evidences of maladjustment. This too has been shown to relate to muscle tension. Duffy found that children with lower ratings of adjustment in nursery school manifested higher pressure in squeezing a hand dynamometer. Adults with increased EMG tension (in the arm, shoulder and back) have been noted to react to minor environmental changes with anger and irritation. Similarly, Wenger found that boys judged to be most tense physically tended to be emotional, irritable, aggressive, and unstable, and that ratings of tension in children correlated with emotionality, carelessness, distractibility, and impulsiveness. There is evidence of the same relationship in laboratory settings as well.

Lacey has reported data suggesting that impulsivity and hyperkinetic traits are associated with tendencies to exhibit bursts of sympathetic-like activity, or continuous oscillatory changes, in *autonomic* variables. Although he did not measure muscle tension, its correlation with these behaviors has just been indicated. Lacey's interesting idea remains conjectural, however, since the only attempt at replication has proved negative.

The interrelationships among autonomic variables, muscle tension, and personality represents a barely touched-upon subject that may have considerable significance. One interesting possibility is opened by the study of Kempe. This revealed contrasting traits between individuals whose stress

response was primarily autonomic and those who tended to develop generalized muscle tension. Those with tension responses tended to be aloof, to deny emotion, and to intellectualize easily. The autonomic responders were emotional, insecure, and prone to worry. Although stress does have some tendency to result in a generalized "activation" of all physiological responses (as will be considered shortly), there is also evidence that the autonomic and muscle systems may have a reciprocal relationship under some circumstances. Malmo and Shagass noted this reciprocity between heart rate, and both motor responses and EMG levels in the arm in a group of psychiatric patients exposed to a painful stimulus.

In this latter study, some of the motor responses were "defensive," i.e., they served to terminate the painful stimulus. This raises the issue of the role of psychological defense in producing muscle tension. The traits reported by Kempe to characterize those who respond to stress with generalized muscle tension suggest a personality in which defenses against affects are predominant. This is an issue of great importance. The control of affect, particularly anger, has been postulated as a major pathogenic mechanism in several of the musculoskeletal disorders to be discussed in subsequent sections.

This relationship has been suggested by many psychoanalytic observations. Abraham reported in 1913 that "during analysis patients show

inhibitions of those bodily movements which derive from a *repressed* [my emphasis] erotic pleasure in movement." Ferenczi noted the frequent reciprocity between thought and action, and observed that many patients exhibit stiffness of the limbs during a state of resistance, which disappears following its resolution. The tendency for the affect-controlled compulsive character to manifest generalized muscular rigidity was explored in detail by Reich, who clarified its ties to the rigidity of defense in the important term "character armor." He pointed out how physical relaxation can result in a loosening of defenses and freeing of affective expression. Similarly, Barlow reported that following relaxation "one may see uncontrollable reactions, such as laughter and weeping." Fenichel comments that "pathogenic defense always means the blocking of certain movements." He reported that a patient "who can no longer avoid seeing an interpretation is correct, but nevertheless tries to, frequently shows cramping of his entire muscular system or of certain parts of it." The most detailed psychoanalytic studies were carried out by Felix Deutsch who reported on the presence of individually characteristic infantile postures and movements associated with the regressive nature of the psychoanalytic situation. A portion of what he observed were defensive movements, e.g., rigidity related to the *suppression* of thoughts, a point also emphasized by Ascher. In sum, these observations focus on the role of muscle tension in relation to defense, and often specifically in relation to control of affective expression. Plutchik summarizes the implication of these and other

studies in much the same way. He comments also on the ubiquity of reference to this connection in everyday language (being rigid, keeping one's chin up, etc.). There is, in addition, the familiar observation that being self-protectively "on guard" in the face of danger is associated with muscular tenseness. Also consistent with the reciprocity noted by Kempe are Kepecs' psychoanalytic observations of a reciprocity between muscle and secretory activity, including weeping and exudation into the skin. He suggests that when the muscles are about to be activated during rage but this undergoes inhibition, the result may be actual weeping or weeping lesions of the skin.

An attempt was made by the Michael Reese group to obtain experimental verification of the relationship between muscle tension and "control." In the initial study a series of hospitalized depressed patients were selected for study because of the motoric disturbances found in this type of disorder. These patients were exposed to a stressful situation, and one designed to stimulate efforts at self-control, while EMG recordings were obtained from seven muscles. The temporary activation of efforts at self-control did not elevate muscle tension. However, there were a number of significant correlations between tension in four muscles (frontalis, trapezius, quadriceps, and particularly the biceps) and several personality trait measures that seem consistent with the postulated relationship. Tension in these muscles was greater in the patients who were characterologically less emotional, less anxious, more prone to use fantasy than action, and who had a

more rigid definition of their body boundaries. An attempt to replicate these findings on a different study population, a mixed group of psychiatric outpatients, failed. A careful analysis of the discrepancies suggests that their major source lay in the differences between the psychological characteristics of the patients in the two studies. Whatever the explanation, one can only conclude that the clinical observation that defensive character armoring is reflected in elevated levels of muscle tension remains unverified in the laboratory.

The basis for selection of a measure of body boundary definiteness stemmed from the work of Fisher and Cleveland. These researchers have conducted a number of studies of body image focused on the degree to which an individual conceives of his body boundary as representing a distinct barrier between internal and external interchanges. They developed the hypothesis that a high "barrier" measure is associated with exterior psychosomatic disorders, including *arthritis,* and with greater reactivity of the physiological systems nearer the surface of the body. In several studies, it was found that subjects with a high barrier score developed greater levels of muscle tension in response to stressful stimulation than did those with a low barrier score. Details of their work and that of others on this subject are available in two books.

Stress, Arousal, Emotion, and Defense

There is solid evidence that the psychological stress response is accompanied by an increase in muscle tension in most people. In Duffy's studies, the greatest rises in tension occurred with emotional stress. It has been repeatedly demonstrated that when elevations of muscle tension occur during interviews, specifically stressful material is being discussed. In one interesting study, jaw muscle tension was high following criticism by the therapist, but fell after praise, in *both* the patient and therapist.

Not only does tension increase under stress, it spreads to involve larger areas of the body. There is a tendency for an individual to lose his ability to discretely utilize specific muscles required for appropriate action, and for tension to spread progressively from that site. Luria's classical studies demonstrated that affectively meaningful stimuli produced a disorganization of purposeful responses in the hand, which became impulsive and tremulous; there was "overflow" of tension to the muscles of the nonactive hand. Factor analyses of the data collected in the Michael Reese studies mentioned above also revealed that muscle responses became more diffuse as stress intensified.

There is a school of thought which asserts that there are no real physiological differences among the various states of affect associated with stress, nor indeed among these and other states of heightened motivation and mental effort. All such states are seen as representing points of elevation

along a single continuum. Their qualitative differences are ascribed to added cognitive elements and components of "directionality" (approach avoidance). The central underlying phenomenon has been termed "arousal" or "activation," the former emphasizing its behavioral properties, the latter its neurophysiological ones. A detailed consideration of this theory and the evidence upon which it is based is available, and does not concern us here.

What we do need to note is that the theory postulates that the level of muscle tension (along with manifestations of sympathetic nervous system activity) parallels the level of activation. Consistent with this are the findings of increased tension associated with learning, motivation, difficulty of mental work, reaction time, attention, thinking, etc. (References to this work can be found in Balshan Goldstein's review.) Certainly emotional stresses of essentially every kind have been demonstrated to be associated with increased muscle tension. One interesting exception is the occurrence of a precipitous drop in tension, to the point of physical collapse, associated with sudden surges of intense emotion, the phenomenon known as *tonusverlust.*

Levels of activation may be applied not only to classify stimuli (stronger stimuli being more activating) but to characterize individuals as well. There is a tendency for people to retain their respective ranks in their levels of muscle tension across a variety of stimulus situations. Individuals at the poles of such a ranking would presumably be the same ones identified by the hyper- vs.

hypoactive personality typology already described.

While activation theory may have value in understanding states of affective arousal in general, there are sound reasons for looking beyond it to more specific issues. As Lacey has so well clarified for autonomic responses, specific "fractionation" of the general response occurs, with variations ascribable to the stimulus, to the individual, and probably to the quality of the internal response state evoked. It has been indicated already that the tendency for muscles to react in a generalized fashion is not a strong one, and that there is distinct evidence that more discrete response patterns occur. It is now a commonplace laboratory observation that rises in muscle tension during arousal often can be demonstrated *if* the "correct" muscle is found, but this site will vary among individuals.

Whatever activation theory does explain, it necessarily begs questions relevant to the occurrence of specific sites of tension in specific people under specific circumstances, compelling us to look further.

Anxiety plays a special role in the psychological stress response, being both an indicator of its presence and a precursor of further response. Hence it is not surprising that anxiety is the affect most clearly implicated in stress-associated rises in muscle tension. Motor hyperactivity and "nervous" mannerisms are widely accepted as evidence of everyday anxiety. Tremor,

purposeless movements, restlessness, and other motor symptoms are regular features of clinical states of anxiety. Cameron's systematic analysis of symptoms occurring in patients with anxiety revealed that the "skeletal muscle pattern" represented the largest subgroup. Balshan Goldstein concluded from her extensive review that muscle tension tends to be high, "particularly in disorders in which anxiety is the major concomitant." In studies of the stress responses of psychiatric patients belonging to several diagnostic categories, Malmo and Shagass found that the degree of anxiety paralleled the extent of the rise in muscle tension, regardless of the diagnosis (with some exceptions among chronic schizophrenics). Two separate studies of hypnotically induced emotions demonstrated that rises in muscle tension were greater following an anxiety or fear suggestion than one for depression. A detailed laboratory study of muscle tension in psychiatric outpatients by the Michael Reese group, involving multiple psychological variables, revealed that the increases in muscle tension occurring with stress were related most closely to ratings of anxiety. Both Jacobson and Wolpe consider muscle tension as an inherent component of the state of anxiety, so that their anxiety treatment techniques are based on tension reduction.

Anxiety also causes a prolongation of the muscle-tension response to stressful stimulation. The exaggerated startle reaction so typical of anxiety seems to be due to this rather than an increased magnitude of response. Anxious patients manifested a continuing rise in tension following a sudden

noise stimulus at a time when the responses in normals were falling and had almost disappeared.

Anger also is relevant. The subjective sense of increased tension associated with rising anger is a familiar experience. A specific attempt to differentiate the physiological patterns specifically characteristic of fear vs. anger revealed that both were associated with elevated frontalis tension, with the levels during anger exceeding those for fear.

The state of frustration may not be identical to that of anger, but it is certainly one closely akin. In his thoughtful review, Plutchik marshalled a substantial body of evidence linking frustration and conflict to muscle tension. He cites a variety of studies which confirm the observable link between frustration, anger, and hypermotility, and of tension arising in situations where internal conflicts block action. From these and other data he concludes that chronic muscular tensions represent "a continuous state of readiness, indicating present day conflict or frustrations" that "reflect attitudes which are usually verbally unexpressed." Freeman also felt frustration and conflict were central to the development of muscle tension.

If we take a second look at the studies of stress, maladjustment and trait anxiety, it becomes apparent that irritation, irritability, impatience, and similar states are frequently part of the reported affective reactions. Given the

complexity of human beings, states in which a single "pure" affect develop are rare. Experience in the psychosomatic laboratory confirms this; even with the specific attempt to stimulate a given affect, mixed states occur. Certainly the physiological features of anxiety, anger, and other affects overlap, requiring great sophistication in research methodology to delineate their minor, quantitative differences. It may be that each affect is associated with a somewhat specific *patterning* of tension, i.e., with prediliction for certain muscle groups. Bull has suggested that feeling states arise secondarily as the perception of the patterned muscular responses which, she feels, represent the primary emotional state, a derivative of the James-Lange theory. The term she uses for emotional states is "attitude," one that has both affective and positional connotations.

But in every situation of stress and affect arousal it is equally certain that the reaction also will include elements of ego defense. It is impossible to conceive of a state of stress in human beings unaccompanied by the mobilization of *defenses and coping mechanisms.* The states of frustration and conflict, discussed above, also include a component of defense. Even simpler demands for performance, problem solving, attention, etc., necessarily call forth adaptive efforts. Thus we are brought back again to the possible role of *control* and defense mechanisms in the production of muscle tension, especially when it is sustained. It is possible that it is these processes, rather than the affect, that is the crucial factor in stress-induced tension.

The two studies which attempted specifically to identify the role of *control* were, as indicated, negative. The focus of these was on relatively conscious and immediate exertions of control efforts, i.e., self-control. Perhaps a better definition of control related to more automatic processes, or a focus on other defense and coping mechanisms, especially those linked to character, might have better payoff. One report, which touches on this subject, linked the responsiveness of frontalis muscle tension to ego strength, as measured by the MMPI (Minnesota Multiphasic Personality Inventory). Given the known inverse relationship of ego strength to such factors as trait anxiety, which correlate with muscle tension, this seems puzzling. The explanation may lie in the greater defensive response of the high ego-strength subjects. Unfortunately the design of the study included no observations of this dimension.

Any stressful situation will inevitably tend to cause rises in all these factors concomitantly, i.e., a mixture of various affects, defenses, and coping maneuvers. A researcher will therefore find the *explanatory* variables he happens to look for. The present state of our knowledge suggests that all these factors may bear a relationship to muscle tension. It seems sensible to view tension as representing a final common pathway of peripheral response which is related to a variety of central psychological states. Within this very general situation, it may be that differential patterning of muscle response occurs in relation to specific states. To determine if this is so requires more

sophisticated research which can examine a variety of psychological factors simultaneously, each in relation to tension measures taken from many muscles. Until then, we are left with many interesting conjectures but only a few dependable conclusions.

Psychiatric Disorders

There is one direction in which many findings do converge. Every one of the aforementioned factors suggests that elevated muscle tension should be found in psychiatric patients. Who, if not psychiatric patients, are prone to manifest anxiety, conflict, stress sensitivity, etc.? The verification of this is ample. The overall picture shows that patients with essentially every disorder exhibit a heightened muscle tension under many conditions. In fact, several of the studies already discussed have used patient groups precisely for this reason. Therein lies the problem. In such instances it is difficult to discern if the findings bear a relationship to the disorder per se. The burden of proof must rest in the other direction, given the widespread presence of elevated tension across diagnostic groups.

Several of Malmo's studies have specifically suggested that anxiety is the primary significant factor. Also consistent is Martin's finding that "dysthymic" (anxious, depressed, and/or obsessional) neurotics displayed significantly more forearm and frontalis-tension at rest than did hysterics. A similar

comparison by Balshan Goldstein revealed like differences which, however, were small and not statistically significant. There seems little point, therefore, in discussing the many studies in which mixed, nonspecified or anxious patient groups have been studied.

Several studies focusing on *schizophrenia* have pointed to elevated levels of tension in these patients, at least under "resting" conditions. Whatmore and Ellis found resting EMG tension recorded from four areas (forehead, jaw, forearm, and leg) to be higher in a group of twenty-one schizophrenics than in ten normal controls. The nature of the patient group was not specified beyond statements that they had "clear-cut" diagnoses, and were "without signs of deterioration." Malmo and his coworkers compared neck and forearm EMG levels in seventeen *chronic* schizophrenics, with groups of "acute psychotics," neurotics, and normal controls. All patient groups had high levels of tension at rest, whereas the normals did not. Stressful stimulation produced an increase in tension for all groups, but the rise was significantly lower for the chronic schizophrenics than the other patient groups, with the normals falling between. The schizophrenics were especially less responsive if the stimulus was brief. Very similar results to those of Malmo were obtained by Williams using a resonance (non-EMG) technique.

In a previous study from the same laboratory, "early" schizophrenics

were found to be similar to *very* anxious neurotics. Both showed high levels of neck tension in response to painful stimulation. The schizophrenics, however, manifested poor discrimination among the various levels of stimulus intensity. Martin also reported that early schizophrenics were significantly more tense (frontalis and forearm EMG) than normals. But the difference was present at rest and disappeared following stimulation.

In a complex and methodologically sophisticated study, Balshan Goldstein compared muscle tension in psychotics, neurotics, patients with character disorders, and normal controls. All groups included contained fifteen subjects, and both sexes. The psychotics were *not chronic* and included ten schizophrenics. The EMG records were obtained from seven muscles at rest and in response to a noise stimulus. The psychotics had generally higher tension levels at rest, and distinctly greater responses to the noise. The differences were significant for the sternocleidomastoid, frontalis, biceps, and forearm extensor muscles, especially the forearm. Both the normals and patients with character disorders had low levels and responses, while the neurotics fell between. This study incorporated a unique feature in that the neurotic and psychotic groups were equated for their levels of anxiety on the Taylor MAS scale (manifest anxiety scale). Thus their differences in tension seem reliably related to the diagnostic difference itself. This is the only study about which such a statement can be made with confidence.

Whatmore and Ellis also conducted studies of *depression.* In their initial project, six severely retarded patients, as well as thirteen patients with agitated or mixed pictures, were compared with matched controls. All were female. Resting EMG recordings (forehead, jaw, forearm, and leg) revealed high tension in all areas, with the greatest differences in the jaw muscles, and the least in the frontalis. The highest levels occurred in the *retarded* depressives. On the basis of these data Whatmore and Ellis formulated a theory that persistent muscular hypertension at rest, which they termed "hyperponesis," reflected a central neurophysiological state that was an inherent aspect of the depressive disorder. This formulation, however, is not only vague but loses some credibility from their own previous similar findings in schizophrenics, of which they make no mention. Nevertheless their data, particularly those in retarded patients, are of great interest.

In their second project, five previously studied severely retarded depressed patients were followed with the same measures longitudinally through remissions and relapses. Increased tension was again demonstrated during periods of depression. Although the levels decreased temporarily during successful treatment, they soon returned to the previous high levels which persisted "indefinitely." In one patient, a relapse was just preceded by an increase above the already elevated level. Whatmore and Ellis interpret these data as further support for their concept of "hyperponesis."

Martin and Rees found increased forearm tension in female depressives during a reaction-time test, compared to controls. While patients' mean reaction times were slower, some of their responses were as fast as the normals, suggesting that the difference may have been due to reduced motivation.

The degree of elevation in forearm tension was found to correlate with the severity of depression in a group of patients (of both sexes) studied by Noble and Lader. Tension correlated also with the level of anxiety. In contrast to Whatmore and Ellis' findings, the elevated resting level dropped significantly following electroconvulsive therapy (ECT). Mental arithmetic produced an increase in tension to about the same levels both before and after ECT, the extent of the change being greater afterwards because of the lower resting level. A positive relationship between the intensity of depression and the level of resting tension in the forearm, and to some extent the frontalis, was found also in another study of depressed patients, by Rimon et al. This was more certain for males than females. Jaw tension however, which Whatmore and Ellis reported as showing the highest levels, had a negative relationship with the severity of depression in both sexes. The posttreatment data are confusing. Patients who made a good recovery showed an increase in tension, while those who had a poor recovery had a decrease.

Data on depressives was obtained also in the aforementioned study by Balshan Goldstein. Tension levels were recorded from seven muscles in *nonpsycliotic* depressed patients and matched groups of nondepressed neurotics and controls. Each contained thirteen women and eight men. Following a stimulus, the depressed patients had significantly higher levels of tension in the trapezius and frontalis; in the forearm the difference fell just short of significance. Similar, but insignificant, differences were present at rest. No differences in jaw tension were found, a result in accord with the findings of Rimon but directly contrary to those of Whatmore and Ellis. It seems likely that Goldstein's subjects were less severely depressed than those in either of these two studies, which had included psychotic patients. Whatmore and Ellis' patients also were older, a factor which Rimon found to be associated with higher levels of tension.

The problem of relating specific *sites* of tension to specific disorders is illustrated by the data on the forearm extensors. Elevated tension in these muscles is the most reliable finding in depressives, having been reported in all the studies discussed above. However, it has also been reported to occur in schizophrenics in three separate studies.

A recent study has demonstrated a decrease in the postural reflex activity of the shoulder (supraspinatus) in depressed patients. This correlated, in general, with the level of depression. These findings fit with the

commonly observable slumped posture of depressives. An essential point is that this interesting technique is entirely different from that used in the other studies considered above.

It might be mentioned also that psychotics of all types are said to differ from neurotics in having significant disturbances of *fine* psychomotor activity. Finally, it should be noted that characteristic microscopic muscle lesions and an elevation in circulating levels of certain muscle enzymes have been reported to occur in patients with a variety of *acute* psychoses.

Conclusions

It seems possible to come to a few general conclusions despite the differing and somewhat confusing nature of this array of data:

1. There is a tendency for individuals to be characterized by different levels of muscle tension and to maintain this relative level in various situations. Perhaps the best personality correlate of higher levels of tension is the tendency to experience anxiety and to manifest other forms of emotional hyperreactivity.

2. Individuals have a proclivity to develop tension, when stimulated, primarily in specific sites characteristic for themselves. Under conditions of increased arousal, tension tends to become progressively more generalized,

and the differences among individuals thus tend to disappear.

3. Under stressful conditions, particularly those which induce anxiety, muscle tension rises. At high anxiety levels this increase in response is likely to be accompanied by its prolongation in time, and by its spread, leading to disruption of motor coordination. The relationship of stress-induced tension with heightened anxiety is clearest, but it may be related also to anger and other affects, to the mobilization of certain defenses, to the state of conflict or frustration, or to all of these.

4. Patients with psychiatric disorders of every type are likely to exhibit high levels of muscle tension, especially after stressful stimulation. It is uncertain if this relates to the disorders themselves or is merely a reflection of anxiety and the other factors just mentioned. Increased tension may be especially characteristic of two disorders, schizophrenia and the depressions, above and beyond the presence of anxiety. For the depressions, where the evidence seems particularly strong, the level of tension seems to parallel the severity of the disorder, even in the presence of overt psychomotor retardation.

5. No convincing evidence yet exists to relate specific patterns or sites of muscle tension to specific affect states or to specific psychiatric diagnostic entities, despite several interesting suggestions. This state of knowledge may

be partly a product of the fact that few studies have obtained tension measures from more than a limited number of sites.

6. Clarification of many of these problems seems amenable to research embodying now existing technical and methodological knowledge.

These conclusions reflect the substantial body of research on the basic psychophysiology of muscle tension. None of them bears directly on the role of muscle tension in the musculoskeletal disorders. They do, however, have some relevance to the psychological characteristics which have been suggested as playing a significant role in these disorders, as will be seen. There are also a few studies in which tension measures have been made directly in patients with given disorders. These will be considered, as appropriate, within the following sections which deal with each disorder.

Psychogenic Rheumatism

While the term “rheumatism” lacks precise meaning, that very quality may have a particular aptness in grouping together a grab bag of disabilities which have common underlying features. The complaints may include a variety of aches, pains, weakness, stiffness, and other uncomfortable sensations in the muscles or joints, as well as subjective swelling, tenderness, and limitation of motion. These may involve a single area, several, or occur “all over” the body, and they may be migratory. The diagnosis of psychogenic

rheumatism is applied to such disorders in which the symptoms and disability occur in the absence of established "organic" disease or are significantly out of proportion to its extent. Clinically, the complaints often have a vague or odd quality, may not conform to expected anatomic distributions, and may be unrelieved by analgesics or physical therapies. Abnormal, sometimes bizarre, posture and movements may develop. However, the symptoms can closely mimic known *organic* diseases.

The overall incidence of minor isolated rheumatic symptoms must be 100 percent. (Who has not had unexplainable aches and pains?) But there are no clear figures for the prevalence of disability significant enough to require diagnostic labeling. It is established that this disorder occurs frequently enough to comprise a major portion of the patients with diagnosed rheumatic disease. In several large series, reviewed by Boland, it consistently ranked among the top three causes of rheumatic disease, along with rheumatoid and osteoarthritis. The incidence seems to be particularly high in military personnel, where figures as high as 34 and 42 percent of the admissions to special rheumatic centers have been reported. In this setting, its onset is especially common just prior to overseas or combat duty.

Some further idea of the vast extent of rheumatic disease and its disability, particularly in the military, is provided by data on veterans' pensions. In 1931 (an era when the number of veterans was far fewer than it

is today and their care was far less complete) the Federal government was providing pensions to more than 35,000 disabled veterans in this category, at an annual cost exceeding 10 million dollars!

Classification and Mechanisms

Boland, who provides one of the best general reviews of the subject, refers to the disorder also as "psychoneurotic musculoskeletal complaints." He does so to emphasize the very high incidence of coexisting neurotic symptoms and predisposing "neurotic traits" he finds to be characteristic. Ehrlich also describes it as a form of psychoneurosis. The studies on which this impression is based, however, lacked adequate controls, and the ubiquity of psychiatric symptoms when these are skillfully sought after makes one cautious about this viewpoint. It seems likely that many cases of lesser severity which do not require referral to a rheumatologist or psychiatrist will fail to demonstrate overt psychopathology.

Boland classifies psychogenic rheumatism into three subtypes: (1) pure; (2) superimposed (functional overlay); and (3) residual (functional prolongation).22 The last most often follows trauma, while the superimposed type is usually associated with more minor rheumatic diseases, rather than with a serious articular disease such as rheumatoid arthritis. *Fibrositis* is traditionally included as one minor condition particularly prone to

psychogenic overlay. But there is good reason to question whether this entity exists at all. Its diagnosis is based on subjective complaints. Though nodules are sometimes palpable, Halliday has pointed out that these can be found (as?) often, if sought for, in *a*symptomatic individuals. In one study cited by Boland, 70 percent of cases labeled as fibrositis showed "significant psychiatric disorders." Although acknowledging the difficulties in diagnosis, Hench and Boland suggest that, with care, the two can be differentiated clinically. They characterize fibrositis as having more localized and definite symptoms which are highly responsive to external temperature and humidity (rather than to mood, distraction, or emotional stress), as having a better response to the usual therapies, as associated with less disability, and as occurring in less neurotic patients who are more calm and cooperative during examination, and who may evidence a more "objective" attitude about their illness.

Halliday, the earliest serious student of the problem, classified fibrositis as one of three types of psychogenic rheumatism. The others were hysterical pains, seen most often in people in hazardous occupations, and symptoms arising as manifestations of a psychoneurotic anxiety state or psychotic depression. Paul, who studied back pain, divided that condition into four categories. These included: (1) pain due to muscle tension "of conversion origin;" (2) pain of conversion origin without increased muscle tension; (3) pain due to muscle tension of "anxiety-tension origin;" and (4) any of these

with associated back disease or injury.

Each of these classifications has some heuristic value, but a fully satisfactory taxonomy remains to be developed. One problem centers around symptoms arising from conversion (hysterical) mechanisms. Because they represent psychogenic rather than psychophysiological disturbances, conventionally these are excluded from the "psychosomatic" category. This may be entirely appropriate for such curious and blatant examples of hysteria as camptocormia, or the "stiff-man" syndrome. But it is likely that more minor conversion mechanisms are involved in some of the ordinary disorders diagnosed as psychogenic rheumatism. This may occur as a secondary elaboration of symptoms which initially developed on a psychophysiological basis. Faulty postural compensations also can add a significant element to symptoms which arose originally on a psychological basis.

Nevertheless in most instances the major pathogenic mechanism seems to be a psychophysiological one, i.e., elevated muscle tension. Of the several major clinical disorders considered in this chapter in which increased muscle tension has been hypothesized to be the significant mechanism, the evidence seems by far most dependable for psychogenic rheumatism. Clear signs of localized muscle tension are often readily apparent in the physical examination of patients with the symptoms of psychogenic rheumatism. This clinical finding has been confirmed by EMG measures. Particularly relevant is

the phenomenon termed "symptom specificity," elucidated by the excellent research of Malmo and Shagass. Symptom specificity refers to the fact that the stress response of some individuals is characterized by a distinct predilection for hyperactivity in a given muscle (or autonomic variable) leading to symptoms directly referable to that increased activity. Thus, patients with a history of head and neck pains responded to psychological stress with greater rises in neck-muscle tension than did controls or patients with cardiovascular symptoms, while the latter patients had the greatest cardiovascular changes. In longitudinal studies, several headache patients manifested periods of increased head and neck tension occurring during stress, and it was at such times that episodes of their typical pain occurred.

Rinehart, disciple of Jacobson's "progressive relaxation," also identifies elevated muscle tension as the primary issue in psychogenic rheumatism. His entirely anecdotal report loses all credibility, however, by lumping together *all* rheumatic disease (including rheumatoid and osteoarthritis) as having the identical pathophysiology and pathogenesis, as does the entire school of "progressive relaxation" by its indiscriminate implication of muscular hypertension as being the central defect in a large array of disease processes.

Psychogenesis

There are few disorders in which patients' use of symbolic *organ*

language is as conspicuous as in psychogenic rheumatism. One is struck, as were Halliday, Weiss, and Paul, by these patients' references to themselves, their reactions, and their world in terms replete with musculoskeletal connotations. They are people who "wouldn't stoop" to certain behavior, even when they meet "stiff' situations, as might a "spineless" person; they manage somehow to "limp along" and "not buckle," even when things are a "pain in the neck," etc.

Two themes are especially prominent in their expressions: *anger* and *its control*. These same issues were identified by Weiss as the characteristic feature of forty patients with varied rheumatic symptoms. His interviews revealed "chronic resentment" and "smoldering discontent" to be their "special emotional problem." They were "burned up" and "aching to" express their hostility, but were unable to do so because of repressing forces. Weiss went on to comment that muscles "serve as the means for defense and attack in the struggle for existence," and that chronic muscle tension arises when the expression of aggression is inhibited.

The very careful study of seventy-five diverse backache patients led Holmes and Wolff to quite similar conclusions, despite their different (nonpsychoanalytic) frame of reference. They were able to discern a common "basic personality" characterized by an "action orientation" going back to childhood, and by a "basic insecurity" which had led to a wary, tentative, "on-

guard" approach to people and life situations. These patients had many obsessive-compulsive character traits and a strong need to "keep the peace." Episodes of back pain occurred in life situations in which they felt disapproved, unreceiving of deserved praise, or criticized despite their efforts, and thus felt "anger and resentment" over being taken advantage of, but could not take action without increasing their insecurity. They dealt with this conflict by being even more "on guard." Support for this formulation came from EMG measures taken during interviews. Striking increases in muscle tension occurred during the discussion of stressful events of the specific type noted, which were especially prone to occur when the current life situation was of the same nature.

Something very like the "on-guard" attitude seems to characterize the response of these patients during medical examinations. Hench and Boland describe the attitude of "touch me not" as being sufficiently typical to constitute a clue to diagnosis. The obsessive-compulsive traits also were noted by both Halliday and by Rinehart. This makes sense, given the psychodynamic relationships of such defenses to the control of anger, and their common "anal" genetic origin—a shorthand designation for a developmental period in which the main issue is the mastery and control of *muscular* activity.

However, the clinical descriptions provided in these and several other

reports include some patients who have manifest anxiety. This was the major quality communicated in Boland's descriptions, as already noted. The question therefore exists whether it is the anger, its control, or anxiety over that or other conflicts, which is the relevant factor. Basic research, as I have noted in an earlier section, leaves this matter open. Perhaps the wisest present position that can be taken is that one must determine individually for each patient the psychological factors that are involved in *his* increased muscle tension.

Chapman makes the interesting observation that these disorders occur mainly in muscles that have important uses in animals, but are of lesser importance in man. Just why a given site is involved in a given person is not always clear. What psychological factors contribute to the "symptom specificity"? Sometimes the specific symbolic meanings of the part seem to be involved. These may become apparent in the organ language used to describe the symptoms. One interesting lead is provided by experimental findings which suggest that hostility is related to tension in the arm, and that, in women, sexual problems are related to leg tension. Further work of this type is needed.

Treatment

As always, good treatment rests on accurate diagnosis. Little can be

added to Halliday's advice that this requires establishing *what kind* of a person this is, why did he take ill *when* he did, and in the *manner* that he did. In many mild cases, the kind of doctor-patient relationship inherent in this approach itself leads to a reduction in the intensity of conflicts, allowing for recovery. At an early stage the usual simpler techniques of symptomatic relief, reassurance, support, ventilation, and environmental manipulation usually suffice. Muscle relaxants may be very useful for the anxious patient, especially if these also have "tranquilizer" effects. These agents require more cautious use in rigidly controlled patients, who suffer from generalized muscle tension. "Relaxation" may have a paradoxical effect by threatening such patients with an inability to maintain their defenses.

Major and prolonged illnesses represent a far more difficult problem. There is great value in early intervention before symptoms have become fixed and elements of secondary gain are superimposed. As Hench and Boland understate it: "Our pleasure at being able to reassure soldiers . . . that they do not have arthritis . . . and need not fear the presence of a crippling disease is tempered by the difficulty in helping them develop insight and to accept the diagnosis." Physical and pharmacological therapies specifically associated with "organic" forms of arthritis should be avoided, since the patient uses these to reinforce his denial of the psychogenic nature of his illness. The physician's firmness in this regard does not require being argumentative and must be combined with an accepting open attitude to the patient. The

treatment of persistent symptoms in its further details merges into the entire body of techniques of psychotherapy.

Special therapeutic techniques designed specifically to reduce muscle tension promise usefulness in those many instances where muscular hypertension can be implicated. These include “autogenic training,” “progressive relaxation,” hypnosis, and the “reciprocal inhibition,” associated with behavior therapies. All of these techniques have been reported to produce significant immediate relaxation in selected individual instances during training sessions. This has been confirmed by EMG measures. But it is yet far from clear how much generalization occurs to situations outside sessions, whether reports of outside subjective improvement are also associated with demonstrable reductions in EMG levels, or how widely applicable such methods are to different patients. The body of literature on these techniques is very large, but almost none of the “research” reports can be said to embody even the most minimal principles of scientific methodology, though beginnings are being made.

Occupational Cramp

The occupational cramps include a large number of functional disorders characterized by the impairment of a specific learned occupational motor skill. The dysfunction typically is associated with muscle spasm, and pain or

severe discomfort of the involved part, although the specific clinical features vary. An excellent brief review of the subject is available.

The varieties of cramp are apparently as numerous as there are occupational motor acts. Some thirty-four varieties affecting the upper extremity alone have been delineated, ranging from telegraphist's, to cigar maker's, to violinist's cramp! The various forms seem to have much in common. The incidence of each seems merely to reflect the prevalence of the skill. For this reason writing is by far most often affected.

Writers cramp is well known and certainly the best studied of these disorders. Crisp and Moldofsky have reviewed the subject extensively. It exists essentially in all cultures. Its incidence is reported as being 0.1 percent of "neuropsychiatric cases," though the meaning of this figure is obscure, given the impossibility of defining the base. Mild cases are probably quite numerous. As with the other occupational cramps, its psychogenic nature is evident from the fact that a specific skill is involved, while unrelated activities involving the same muscles and movements are spared or minimally affected. The disability tends to increase with stress, and concomitant neurotic symptoms are usually found.

Many psychologists would formulate the origins of the disorder as a "faulty learning experience." This viewpoint has become more attractive

because of the resurgent interest in the application of learning models to behavioral disorders. But the idea is not new, and corresponds closely to that held by Pierre Janet. Techniques of "reeducation" have, indeed, proved somewhat efficacious. This is more likely to be the case when they are combined with other techniques of effective psychotherapy. Partly, such reeducation may be required because of the superimposition of secondary maladaptive compensatory positions and movements, which do represent a form of erroneous learning. Frustration and anxiety over anticipated failure play a role in this development. In any event, the faulty-learning-habit explanation begs the questions of *why* the learning was deviant, and why the disorder developed *when* it did.

Psychiatrists have tended generally to classify writer's cramp as a psychoneurotic symptom, though most formulations have been vague. Most writers have tended to view it as an hysterical symptom, though noting that it might occur in conjunction with an obsessional state. Cameron viewed it as an hysterical symptom related to ambivalence over the writing activity, and Glover felt it represented an hysterical conversion mechanism.

Crisp and Moldofsky present compelling arguments for placing it instead in the psychosomatic (psychophysiological) category. Careful study revealed that their patients lacked the classical hysterical features, i.e., they were neither bland nor manipulative. In fact, obsessive-compulsive traits

were usual. Secondary gain played a minimal role; the actual inability to write was rare. Instead, there was a "continuing unresolved ambivalence" over writing, with expressed resentment over having to do so. Symptomatic exacerbations corresponded closely with periods of intensified resentment. Thus, the disorder seems to represent a concomitant of affective disturbance, rather than a neurotic defense against it. Finally, a physiological disturbance of the motor system does seem to be present and to extend beyond the symptom. Von Reis has provided EMG evidence of widespread muscle tension throughout the arm of the involved hand, occurring even at rest.

Crisp and Moldofsky's experience also underlines the value of an overall treatment program which combines psychotherapy with both relaxation exercises and reeducation. They emphasize that the *transference relationship* plays a key role in the effectiveness of treatment, sufficiently so that it can be used as a predictor of therapeutic success.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is an inflammatory disorder of connective tissue, with polyarthritis as its most characteristic feature. The arthritis has a predilection for the more peripheral and smaller joints, and is typically symmetrical. The pathological process in the joints is one of acute proliferative inflammation which attacks the synovial membrane. This results

in the formation of a granulomatous pannus, with damage and destruction of the underlying cartilage. Adhesions and scar formation tend to occur, eventuating in disability and deformity which may be crippling. Any of the connective tissues throughout the body may be involved in a similar inflammatory process. Constitutional symptoms are common. The typical course of the disease is dominated by unexplained exacerbations and remissions, which may be total. Attacks may vary greatly in the severity of the arthritis and other symptoms, and there are marked differences in the long-term course of those afflicted.

The disease is worldwide, though it may be less common in the tropics. It occurs at all ages, the most common onset being the mid-30s. Females are affected about two and one half times more frequently than men, with onset in middle age not uncommon. It is generally said to occur in approximately 3 percent of the population. Precise figures, however, are uncertain because of its episodic and varying course, even with the arduous development of standard diagnostic criteria. It has been suggested that RA may be a much more common and benign disorder overall than is generally believed.

Rheumatoid arthritis is classified as one of the *collagen* or connective tissue diseases, grouped together because of their common histopathological features, symptoms, therapeutic response to steroids, and other similarities. The etiology of all these disorders is unknown. There are strong indications

that alterations in the immune response are involved. Even if this is confirmed, the causative factors that initiate and underlie this immunological disturbance require clarification.

Genetic factors also have been implicated, since RA has a high familial incidence; but such a finding can arise from environmental as well as genetic factors, and definitive data are lacking. Genetic explanations are inadequate to explain certain peculiarities of its familial incidence, for example its increased frequency in spouses.

Psychosomatic Correlations; the "RA Personality"

The notion that RA might be a psychosomatic disorder is an ancient one. Paulus Aegineta, who lived in the sixth or seventh century, ascribed attacks to "sorrow, care, watchfulness and other passions of the mind." In modem times, well over 100 papers and books have appeared which have linked psychological and social factors to the disease. The bulk of this work has been examined in several thoughtful and comprehensive reviews (see references 133, 179, 187, 236, and 267). The book by Prick and Van de Loo, published in 1964, contains summaries of most of the then available studies, including many by European workers not included in other sources. Further information is provided by the *Annual Rheumatism Review*, published since 1935.

The vast preponderance of reports which deal directly with the role of psychological factors in RA are in agreement that stress is associated with attacks, if not the origin of the disease. Much of this material, unfortunately, is anecdotal and impressionistic. It is little different from what has been reported for innumerable other disease states. Seemingly every type of stressful situation has been implicated. On the other hand, the most extensive clinical epidemiological study of arthritis thus far carried out, reported a failure to find evidence of stressful situations associated with the onset of RA. It is difficult to place much reliance on this report either, however, because of the grossly superficial methods used to collect the psychological data.

Some studies have attempted to go beyond this level of generality to identify personality features that might characterize the RA patient. This work has provided descriptions of these patients as shy, leading quiet lives, and feeling inadequate and inferior; as self-sacrificing and needing to serve others; as conscientious, dutiful and compulsive; as having a strict, rigid, moralistic conscience; and as manifesting a tendency to depression. This offers too much diversity to indicate that there is any one simple overt "RA personality type."

Still other studies have probed deeper to attempt to identify underlying conflicts and defenses. These suggest that there may well be specific psychological attributes which characterize RA patients at this deeper level. A

preponderance of this work suggests in a variety of ways that the central issue for these patients relates *to the control or containment of anger* (see references 20, 39, 47, 123, 125, 150, and 222) Thus, RA patients are felt to have a great deal of unconscious or unexpressed anger (see references 20, 125, 149, 150, and 172); but they sharply restrict their overt expressions of hostility (and other emotions) (see references 20, 39, 47, 104, 125, 150, and 172); and avoid situations likely to result in disagreement; because anger, they report, "is likely to make their joints worse." Swaim links the recovery from RA to the patient's capacity to develop a satisfactory overall philosophy of life leading to a state of spiritual harmony. It would be easy to dismiss this as unscientific and irrelevant, were it not for the fact that this author is a renowned rheumatologist, and that his approach involves persuading his patients to "give their *resentments* to God." (My emphasis.)

RA patients also have been reported to engage in a great deal of physical activity in the form of sports and hard work. The preference for these activities is reported to go back to childhood, and has been interpreted as representing a preferred method for safely discharging aggression (see references 15, 25, 38, 47, 125, 150, and 172). Thus it has been suggested that RA may be precipitated when this channel for discharge becomes blocked. Arthritics of both sexes have been reported also to suffer from disturbances in sexual identification (see references 15, 25, 125, 147, 172, and 253). In the females, this has been described as having the features of a classical

masculine protest reaction, which seems linked to their involvement in active physical sports as girls.

Perhaps fears of disagreements and the need to maintain tight emotional control explain their reported difficulty in establishing close relationships, and also their shy, quiet attributes already mentioned. Most of the other overt personality traits noted (being compulsive, self-sacrificing and depressed, having a strict conscience, etc.) are equally consistent with conflicts over hostility and aggression, as these can be understood within a psychoanalytic framework. The seemingly diverse overt personality traits, therefore, exhibit psychodynamic coherence.

Another reported finding is a frequent history of separation or loss of a parent figure during childhood (see references 15, 20, 25, 36, 146, 150, 222, and 243). It has been suggested that grief and separation are important antecedent stresses in relation to the development of an attack. In two studies, however, the control groups showed an equal or higher incidence of early parental loss.

All these trends were noted by King in his 1955 review. Justifiably, he was critical of methodological deficiencies in all the studies then available. Most were impressionistic and lacked control for bias; almost none used comparison groups; diagnosis was loose; and all were retrospective and thus

potentially interpretable in terms of the consequences of RA, etc. Yet, as King indicated, there is an impressive consistency to this work, especially in the prominence of problems related to aggression.

The attempt to develop a comprehensive, coherent schema, that utilized psychoanalytic insights, in order to explain the genesis of RA and other psychosomatic diseases reached its acme in the work of Franz Alexander and his co-workers. For over thirty years this group carried out a series of intensive studies of patients with several psychosomatic disorders to elicit a "specific dynamic configuration" characterizing each. It was their view that such a configuration, in combination with certain equally specific but unknown somatic factors, led to susceptibility to the disease. The disorder would then appear in situations whose nature was specifically such as to severely aggravate the conflict nuclear to the configuration.

The formulation developed for RA indicated that these patients: (1) had overprotective, restrictive parents who stimulated rebellious feelings together with heightened anxiety over the expression of these; (2) the rebellion was discharged in childhood via sports and physical activity, associated in women with a masculine protest reaction; (3) progressively in later life hostility was expressed through masochistic self-sacrifice which served to control the environment while denying hostility (benevolent tyranny); (4) an interruption in the availability or success of this pattern led

to rising anger and increased conflict over its expression; (5) this led to simultaneous increased tension in both sets of opposing (because of the conflict) muscles; and (6) this led somehow to arthritis.

Methodological considerations momentarily aside, the formulation has an elegant quality in its internal consistency and its integration of diverse observational data. Its correctness, moreover, need not be limited by its etiological implications. There is no reason why this formulation cannot be correct at the *descriptive* level, even if it proves inadequate at the *explanatory* level. The two are separable. The former requires merely establishing that the postulated psychodynamic features are characteristic of RA patients. Validation of the latter requires additional data demonstrating that these characteristics existed prior to the onset of the disease, and represented necessary and/or sufficient factors for its occurrence. Whether this formulation will be acceptable even in this limited descriptive sense depends ultimately on one's willingness to accept conceptualizations of human behavior in psychoanalytic terms. One can only note that for clinicians with this orientation who have actual experience working with arthritics, the formulation does seem remarkably applicable to many RA patients.

The study on which it is based does, of course, exhibit grave methodological faults. The patients were interviewed with full knowledge of their diagnosis and of the personality traits reported by previous

investigators, and no control groups were used. Some reassurance is provided by a later systematic validation study conducted by the Alexander group. Using eighty-three patients representing seven major psychosomatic disorders for which formulations had been developed, "blind" diagnoses were made on case records from which medical diagnostic clues were deleted. Psychoanalytic judges were able to make the correct diagnosis significantly more often than at chance expectancy, and also significantly better than could a group of internists judging the same data. The degree of success in identifying the RA patients was particularly high.

Additional confirmation comes from studies carried out by Cleveland and Fisher utilizing psychological test data. They compared RA patients with a matched control group of patients suffering from back pain, utilizing a battery of projective tests, such as Rorschach, TAT (thematic apperception test), and DAP (draw-a-person), to elicit unconscious fantasies. The fantasies of the RA patient "were so unique that three psychologists were able to differentiate with only one error" the RA patients from the controls. During interviews the RA patient was noted to be an "overtly calm" person who "rarely expresses or feels anger." But the test data revealed that "covertly he seems to be containing a large amount of hostile feelings." The RA patients displayed evidence of a relatively unique body image, characterized "as a kind of hollow container filled with uncontrolled fluid material and surrounded by a hard, unpenetrable surface." This external "barrier" quality was

conceptualized as playing a major role in the defense against hostile expression.

Cleveland and Fisher also compared RA with ulcer patients. Both groups evidenced strong hostility. But the RA patients were distinctive in their use of physical activity as a technique for handling this, and they more frequently gave a history of greater participation in rugged physical activity during early life. Subsequent studies utilizing similar test data have confirmed the findings with regard both to body image and contained hostility. The general nature of these findings is also remarkably consistent with the early, less systematic Rorschach studies of Booth.

Further information has been provided by the series of careful psychosocial studies carried out by Cobb, King, and their associates, using sophisticated survey research methods. These data confirmed previous observations that RA is more prevalent in the lower classes. (As one observer has put it, "RA seems to be a disease of losers ... all evidences of the disease were commoner in those with low incomes or little education.") Discrepancy between income and education, which King and Cobb point to as an indicator of social-status stress, was especially associated with RA. Other indices of social stress also were more common.

Certain factors appeared prevalent specifically in *women* with RA. They

had come from parental homes with high social-status stress; they reported mother's authority and discipline as more arbitrary and controlling; their recalled reaction to this was high covert hostility but very low overt resistance and aggressiveness; and they evidenced strong identification with mother despite her negative image. As adults, the conflicts over the control of anger remained in evidence, and they manifested evidence of poorer mental health functioning. Their own marriages too were likely to be with husbands of incongruent social status, and to be characterized by much hostility. Men married to these women are more likely to have peptic ulcers. Taken all together, these data on women are in remarkable agreement with the psychoanalytically derived formulation of the Alexander group. The information regarding the families of origin is of special interest in that it represents reasonably objective evidence of stress *antecedent* to the disease onset.

Cobb and his associates also report that RA patients had a higher incidence of divorce but put up with an unsatisfactory marriage longer than those free of the disease. They interpret this as evidence of the suppression of hostility. A Swedish study failed to confirm the higher divorce rate for RA patients. The implication that this represents refutation, however, is incorrect, pointing up an important methodological clarification about this type of research. Divorce and similar social indices represent culture-bound phenomena with multiple determinants. Within any one society, divorce can

be interpreted as an evidence of interpersonal disturbance. The fact that it does not occur differentially in another society may merely mean that other cultural determinants there make it sufficiently accessible or inaccessible to its members, so that psychological factors become irrelevant. In such a culture interpersonal difficulties, when present, will be evidenced in alternative aspects of social behavior. The data of Cobb et al. deserve the test of replication within the United States; but this would represent their only valid test.

It seems appropriate at this point to mention the reported relationship of RA to schizophrenia. Two studies have suggested that the disorders rarely occur simultaneously. In one study, not a single case of arthritis was found among 2200 patients at a mental hospital. In another, only twenty arthritics were found among more than 15,000 hospitalized psychotics. Both studies exhibit serious methodological deficiencies. Yet, the magnitude of these findings is impressive. If they are correct, it is difficult to interpret their meaning. Some observers have suggested that RA patients are, underneath, seriously disturbed psychologically, and that RA may thus constitute an organismic defense against psychosis.

Objective Tests

The aforementioned studies by Cobb, King, et al. and by Cleveland and

Fisher represented a major advance in methodological sophistication. Nevertheless, these too had shortcomings that have been pointed out by Scotch and Geiger. In an effort to introduce greater precision, a number of investigators have carried out a series of studies using better defined samples and better control groups, and relying on objective psychological tests, usually the MMPI.

The choice of such tests deserves comment. They can be easily and directly scored, have well-established baseline data on large normal and other samples, and provide concrete quantifiable data. They can provide measures of static traits or trait clusters, including nomothetic expressions of underlying motivational forces (e.g., hostility, dependency) one at a time. These data are important, valid and reliable. But they are simply not relevant to every problem. These tests do not provide information about dynamic relationships among motivational forces and defenses, nor their ideographic expressions. They cannot provide any direct information about the presence of a complex psychodynamic configuration of the type proposed by Alexander. Some reviewers have reacted to "projective" psychological tests as if that adjective represented the converse of objective, i.e., as if data derived from such instruments lacks validity. Projective tests are, of course, less precise and quantifiable. But, in fact, the concern of such critics with the validity of projective methods seems to reflect their skepticism about the acceptability of psychodynamic conceptualizations per se. Both types of tests

are valid. Each has its disadvantages as well as advantages, and the appropriateness of each varies with the purposes for which they are used.

A number of studies have utilized the MMPI. An almost universal finding is elevation of the "neurotic triad" of the hypochondriasis, hysteria, and depression scales (see references 29, 81, 189, 197, 209, 262, and 268). It seems clear that the RA patient describes himself as characterized by neurotic trends. On the other hand, the scale elevations are less than those found in neurotic patients, and the picture is inconsistent with the view that RA patients are seriously disturbed and near psychotic. The fact that these findings are indistinguishable from those in neurotic patients has been interpreted as running counter to the expectation that a specific RA personality pattern exists. A question also can be raised as to whether these abnormal MMPI findings merely reflect the presence of the symptoms and disability which RA produces. To get more information on this, several investigators have carried out item analyses to determine if the scale elevations result entirely from responses which reflect these manifestations of the disease. The results have been conflicting.

Bourestom and Howard described similarities in the MMPI-scale elevations among patients with RA, multiple sclerosis, and spinal-cord injuries. They also found overall differences which "support the hypothesis of some specific personality correlates associated with the three disabilities." In

addition, sex-linked differences were found within each group, and the male arthritics seemed somewhat different from all other groups. Nalven analyzed MMPI responses in terms of hostility, and also obtained scores on three special hostility scales. The data failed to provide evidence of increased hostility in RA patients but did suggest that they had the problem of overcontrol of hostility. Geist used projective tests and a questionnaire battery as well as the MMPI. His MMPI data indicated neurotic trends similar to the earlier studies. The other instruments revealed signs of inhibited chronic aggression in the RA patients. They also indicated the presence of obsessive compulsive defenses, participation and interest in sports prior to disease onset, and a suggestion that the families of origin were characterized by matriarchal discipline. Unfortunately his sample was relatively small (twenty-two), and his questionnaire lacked external validation.

Robinson and his co-workers used Catells' *16 Personality Factor Inventory* (16PF) to study patients with recent ("new") as well as long-standing ("old") RA, and similar matched groups of tuberculosis, diabetes, and hypertension patients. The new RA patients differed little from the other new disease groups, but did show deviations from the test norms indicating neurotic trends similar to that revealed by the MMPI. In contrast to the other illness groups, the new and old RA patients were significantly similar to one another, which these authors interpreted as evidence supporting the existence of an RA personality type. As a whole, the RA patients manifested

emotional instability, introversion, guilt, and depression proneness, exaggerated dependency needs, and trends toward compulsivity and tension. This combination, they concluded, reflects "a person who restricts his emotional expression, including expression of aggression."

The 16PF was used also by Moldofsky and Rothman in a complex study of symptoms, treatment and personality in a group of RA patients. The patients as a group had test scores which, compared to norms, revealed low ego strength, emotional instability, dependency, and conformity, i.e., again, similar findings to the MMPI studies. The investigators concluded from this that there was no specific RA personality. The personality traits did not show any relationship to disease activity, but patients on steroids manifested more severe emotional symptoms. Wolff comments that "the results obtained in these two 16PF studies differed considerably, strongly suggesting that large variations in personality patterns exist in RA." Here again, the nature of the data does not seem to be considered sufficiently.

The most creatively designed studies utilizing the MMPI were carried out by Moos and Solomon. They scored the MMPI on a variety of derived scales developed to reveal underlying personality traits, as well as on the conventional scales, and also utilized ratings and content analyses of interviews. Women with RA were compared with their healthy sisters, and with other female relatives. The patients displayed more compliance-

subservience, depression, conservatism-security, and sensitivity to anger than did their sisters. They also manifested clear and striking differences from their sisters in their self-descriptions, in the extent of their masochism, self-sacrifice, and denial of hostility, in their perceptions of the amount of rejection they perceived from their mothers and of the strictness of their fathers. Compared with female relatives in general, the patients scored higher on scales reflecting physical symptoms, depression and apathy, psychological rigidity, and neurotic symptoms indicative of anxiety, masochism, self-alienation and over-compliance. No clear differences in physical interests or activity could be elicited. The RA patients also displayed some similarities to patients with other psychosomatic conditions. As the authors note, this finding cannot be interpreted merely as lack of specificity, for it was not general. There were similarities to both ulcer and hypertension patients, but in entirely *different* ways. The point is that the dynamic formulations for different disorders do overlap, not merely because they lack precision but also because each is a constellation of transacting traits which include some common elements.

This work provides meaningful information about personality in RA that goes well beyond the earlier, less sophisticated MMPI studies. At the same time, it is entirely consistent with that work in revealing the general pattern of nonspecific neurotic symptoms characteristic of the RA patient. As did some earlier workers, Moos and Solomon carried out a careful item

analysis to see if this was explainable on the basis of RA symptoms alone, which substantiated the impression that it was not. (They also cite an unpublished study by D. Cohen that provided a similar result.) Moos and Solomon take pains to point out, however, that this tendency of the RA patient to display neurotic symptoms could be a nonspecific consequence of the presence of a painful, disabling disease. They reaffirm the point, made by every serious student of the problem, that the interpretation of reported distinctive personality findings requires longitudinal studies to determine if these existed antecedent to the onset, and there are absolutely no such data as of 1972.

A later study carried out by Meyerowitz, Jacox, and Hess is of special interest because of its methodological approach. Detailed psychological test and clinical studies were carried out on eight sets of female monozygotic twins, discordant for RA. No consistent differences in personality could be found. There was nothing distinctive about the patients' conflicts or handling of hostility. *Both* groups showed a conspicuous preference for physical activity, and a need to serve and take responsibility for others. The single consistent difference pertained to the involvement of the ultimately affected twin, prior to disease onset, in events experienced as "demanding and restricting." To these, the patients responded "with their characteristic heightened activity, but increasing stress was experienced to the point of being unable to cope."

In a subsequent review, Meyerowitz points to the importance of distinguishing such contributions to disease onset from those purporting to explain etiology, as well as from those which merely delineate the effects of psychological factors upon the course of the disease. Nevertheless, this finding is strongly reminiscent of the etiological specificity hypothesis proposed for RA by Graham and Grace. These latter investigators proposed that psychosomatic disorders are characterized by the presence of a distinctive "attitude" prior to the disease onset. The latter was defined as a feeling state combined with a disposition towards some action. A concomitant specific physiological state is proposed, which is implicated in the pathogenesis of the given disorder. For RA, the attitude is defined as follows: "Felt tied down and wanted to get free (felt restrained, restricted, confined, and wanted to be able to move around)." The problems of evaluating the retrospective report of this particular attitude provided by a patient currently affected by the characteristic effects of RA are too obvious to detail.

Subgroups

An apposite criticism of many of the reported studies is their failure to provide a detailed description of the particular patients studied. The specific characteristics of a given sample can affect the data in significant ways. Findings may derive from characteristics of the sample entirely extraneous to the presence of RA. As well as being misleading, these may obscure the

manifestations of factors that are specific to the disorder. There is also the possibility that seeming discrepancies among studies may reflect the existence of variants of the disorder (subgroups), each of which has differing psychological characteristics. The most obvious example of this relates to the sex of the patients studied. Most studies have been restricted to a single sex, or have failed to consider the male and female patients separately. Yet in two instances already mentioned, where both sexes were included and their data were compared, differences were found. One would hardly expect otherwise. If psychological factors are involved in RA, their role is complex and related to deeper layers of the personality where sex-linked motivations and characteristics exist. At the least, similar dynamic forces will manifest themselves differently within the total psychological economies of men and women. It is possible also that the relevant factors themselves differ for the two sexes.

The importance of elucidating characteristics to identify still other possible subgroups is well illustrated by the careful study of Rimon. This Finnish researcher conducted detailed physical and psychiatric examinations of 100 female clinic patients with RA, and compared them to the same number with other "somatic diseases." He identified fifty-five of the RA patients in whom there was a correlation between emotional conflict situations and the disease onset, and thirty-three in whom there was no such correlation. (The balance represented an inbetween situation.) There were

significant differences between the two. In the group where the disease onset followed a significant conflict, recurrences also tended to follow similar conflict situations, and the course of the disease was one of sudden onset with distinct and often acute symptoms and a much more rapid progression. The patients had few affected relatives. In the group without conflict correlation, the onset was slow and the progression of symptoms delayed, and these patients had a relatively high family incidence of RA. This latter group had an evident incapability of expressing hostile feelings. (For the whole group, "problems in aggression dynamics were of minor incidence" at the "rational" level, again suggesting the irrelevance of data at this level.) A detailed look at ten patients, whose disease had a malignant progression, revealed that half had an exceptionally heavy genetic predisposition, while the other half manifested a great "psychic vulnerability." The latter was indicated by evidence of ego disorganization with overt depression. This research, therefore, suggests that two separate groups of people have a predilection to develop RA. In one, heredity plays the major role, whereas for the other psychological stress is the significant factor; the disease progresses differently for each.

Additional confirmation will be required before we can accept Rimon's findings as generally valid. There are data available from an earlier study by King and Cobb which furnish indirect support for some of Rimon's findings. They compared thirty-two severe RA patients, 25 percent of them

hospitalized, with a group of normal controls, and also with data obtained previously on a group of normals and twenty-one patients diagnosed as having mild RA. The severe cases showed poorer maternal identification and felt a lack of a positive relationship with their mothers, perceiving her as giving insufficient attention and affection. They viewed both parents as having been strict and uncompromising. In contrast, the mild cases were like the controls. If we assume that these psychosocial variables parallel psychological stress proneness, then their more severe disease seems to match the more acute and rapid disease progression in Rimon's comparable group.

Also somewhat confirmatory is a study by Moos and Solomon which found that patients with greater functional incapacity from their disease had poorer ego strength and evidences of a variety of abnormal personality traits, as measured by the MMPI.192 These researchers took pains to match the two groups on many variables, but their efforts failed on one. The more incapacitated group had a shorter duration of illness, and since the groups were matched for the stage of disease, this means the rate of progression had been more rapid in that group. The greater psychopathology which they manifested is thus consistent with Rimon's data. Another study by the same investigators indirectly points in a different direction. In asymptomatic relatives of RA patients, those who lacked the serum rheumatic factor had greater evidence of emotional disturbance on the MMPI. This led the

investigators to conclude that only those individuals who have the hereditary predisposition *and* experience emotional distress are likely to develop RA.

Regardless of the plausibility of that suggestion, the important issue is the need for further studies like that of Rimon which attempt to delineate subgroups. A further illustration of this approach, as well as the use of an anteriospective design, is provided by the work of Moldofsky and Chester who observed two contrasting pain patterns in a group of sixteen RA patients. In patients manifesting the "synchronous" pattern, changes in arthritic symptoms occurred concomitantly with, or just after, mood changes, primarily those of anxiety or hostility. Patients with the "paradoxical" pattern were characterized by an inverse relationship between joint symptoms and feelings of hopelessness. No clinical or socioeconomic variables differentiated the two groups. However, the paradoxical group demonstrated a less favorable outcome when followed longitudinally.

Studies which focus on treatment outcome can contribute to the same end. Factors which are discovered to predict differential responsiveness to differing therapies may serve as clues to subgroups basic to the disorder itself. McGlaughlin et al. found personality differences between patients who responded well to ACTH (adrenocorticotropic hormone) and those who do not. Those who had good dream recall, indicating a higher level of ego function, responded better. Apropos of the point made earlier, there also

were differences between the findings for the male and female patients. The characteristic conflict in both was between hostility and dependency; but the males dealt with this by compulsive defenses and withdrawal, whereas the females relied more on physical activity and on the control of others through self-sacrifice.

A study carried out by Wolff is also of interest in its predictive design, as well as its use of a measure of "pain sensitivity range" he developed. Lower preoperative pain sensitivity served to predict a more favorable outcome to surgical rehabilitative procedures. It seems likely, however, that this variable is more relevant to rehabilitation potential in general than to RA or any subtypes which it may include.

Mechanisms

Any theory which postulates that psychological factors play an etiological role in RA must account for a psychophysiological mechanism through which these factors produce its characteristic joint lesions. The psychoanalytically oriented investigators of RA typically have suggested that conflict over the expression of hostility causes increased muscle tension, and that this leads somehow to the joint pathology. Studies were conducted by two of these research groups dealing with the first part of this proposed chain of events, i.e., the psychophysiology of muscle tension in RA patients.

Morrison, Short, Ludwig, et al. studied thirty-four RA patients, using both surface and needle EMG.193 Half of the RA patients evidenced inconstant spontaneous spiking in some muscles adjacent to the affected joints, when they were apparently relaxed. The normals showed no such activity.

Gottschalk and his co-workers, associates of Alexander, carried out two studies. In the first, multiple handwriting variables were measured in RA patients, in a mixed group of fifteen psychiatric patients that contained nine hypertensives, and in fifteen normal subjects. *Both* patient groups exhibited greater variability on several measures than did the normal subjects. These data were interpreted as evidence of a disturbance in the synergistic use of muscles related to neurotic conflict or pent-up aggression. In their second study, EMG records were obtained on groups of equal size (six) of RA patients, RA patients in psychoanalysis, hypertensives, and normal subjects. Initially, measures were taken from the forearm muscles at rest, and during actual and imagined movements. The RA patients in analysis and the controls showed generally lower tension than the medically treated RA patients and the hypertensives. It was concluded that both RA and hypertension are associated with increased muscle tension and that this may be reduced by psychoanalysis. The second part of this study recorded arm and leg tension in the patients in analysis before and after emotional stress. There were marked reactions "partly predictable" from knowledge of the analysis. "In general," when defenses allowed expression of hostile impulses the tension decreased,

and when there was inadequate means of coping with these impulses, the tension rose.

Surprisingly, there seem to be but two subsequent studies on this issue. Southworth obtained surface EMG recordings from the trapezius and frontalis in patients with RA and those with peptic ulcer before and after a stressful word association test. The only difference was in a more prolonged elevation subsequent to the stress in the RA group, confined to the trapezius. (Moos and Engel interpreted this as a "lack of adaptation," i.e., perseveration, in the muscle responses of arthritics, and suggest that findings might have been enhanced had "symptomatic" muscles been selected for study.) The word association data failed to indicate conflicts over aggression in either group; conflicts over dependency were equally present in both. However, a computed overall "emotional disturbance score" correlated negatively with trapezius tension, which Southworth interprets as evidence that discharge via emotional and muscular tension are inversely related.

Moos and Engel carried out a study of their own. They attempted to determine if hypertensives and RA patients would demonstrate "response specificity" (see p. 728). A complex conditioning technique was used to study twelve female patients in each group. No conditioning occurred. The RA patients showed generally higher tension in their symptomatic muscles, and greater reactivity in both symptomatic and asymptomatic muscles, as

measured by EMG. They also demonstrated a failure to "adapt" (i.e., a persisting response) in their muscle responses, although their blood pressure responses did adapt; hypertensives displayed the reverse.

Even if we accept these findings, major problems arise with regard to their interpretation. First, the presence of acute arthritis or any painful process is associated with splinting of the involved part by increased muscle tension. Hence increased tension, specifically around a symptomatic joint, may be a consequence rather than a cause of arthritis. The only finding that even partly confronts this issue is a mention by Morrison et al. of a single instance in which occasional spikes recorded from a resting muscle preceded by one week the appearance of arthritis in the adjacent joint. Second, and more important, among the various extra-arthritic manifestations of RA is a *myositis* associated with round-cell infiltration, indistinguishable from that found in the other collagen diseases. There is at least as much reason to implicate this as the basis for increased muscle tension as a psychophysiological mechanism.

To clarify the relationship of conflicts over the expression of anger to muscle tension in RA will require studies made during periods of total remission or, better still, prior to the initial onset of the disease. (Data from normal subjects who do not develop RA may not be adequate, since RA patients could differ in the presence of just such a relationship.) A

longitudinal approach allowing for observations antecedent to onset will also answer the question as to whether the psychological findings arise as the result of the disorder or precede it. Every thoughtful student of the field has emphasized the need for such longitudinal studies, while recognizing the immense practical problems which have precluded their accomplishment thus far.

The hypothesis that muscle tension represented the pathogenic link between psychological factors and arthritis seemed plausible at the time it was proposed. Since then, our knowledge of RA has vastly increased. If RA is a systemic disease of collagen tissue, as we now believe, that hypothesis no longer seems adequate. An acceptable psychosomatic theory must account for the specific pathogenic process central to a disease, as Engel has pointed out so beautifully. This viewpoint in no way affects the validity of any of the above mentioned psychological findings per se, which either may play no etiological role, or may do so via another pathogenic mechanism.

One such alternative mechanism has, in fact, been proposed. There is accumulating evidence strongly suggesting that all of the collagen disorders, RA among them, represent “autoimmune” diseases. Although the initiating mechanism remains obscure, a hypersensitivity mechanism seems to be involved in their pathogenesis. Consistent with this, the serum of RA patients typically (though not invariably) contains a macroglobulin “rheumatoid

factor." This has been identified as a complex of an unknown 19S macroglobulin with normal gamma globulin, suggesting that it represents an immune reaction to the patient's own serum protein. Solomon and Moos have suggested that RA may arise via stress-induced alterations of immunological mechanisms. They have marshalled data from a large variety of studies which suggest that emotional factors can alter immune mechanisms. Among these was Fessel's work demonstrating elevated 19S protein in stressed prisoners, and their own demonstration of elevated immunoglobulins in psychiatric patients. Hendrie et al. also found elevated gamma globulin levels in female psychiatric patients, but not in males, and confirmed Solomon's finding that this bore no specific relation to schizophrenia. The relevance of this body of research to RA is entirely indirect. Thus Solomon and Moos label their hypothesis a "speculative theoretical interpretation." However it clearly merits further pursuit, using both RA patients and experimental animals.

This new approach, as well as the whole thread of the discussion in this section, tells us a good deal about the present state of the field. The evolution of psychosomatic research on RA has reached the stage of progress which provides a clear guideline for its future direction. The major focus of future research must be on the painstaking clarification of mechanisms through which psychological and somatic processes interface. These must be chosen in terms of their relevance to the best available knowledge of the pathogenic processes centrally involved in the disorder.

Parkinsonism

The parkinsonian syndrome is a progressive neurological disorder manifested by muscular tremor, weakness, and rigidity, often associated with autonomic symptoms, that arises from abnormalities of the basal ganglia. In some instances, lesions result from an attack of encephalitis, and, less commonly, trauma, toxins, neurosyphilis, and possibly arteriosclerosis have been implicated. But there remain a large group of patients, most likely a majority, in whom no such specific cause is found, and where the disorder is ascribed to *ideopathic* degeneration of the basal ganglia. Under these circumstances it has been suggested that the disorder may belong to the *psychosomatic* category. The basis for this viewpoint came from a number of observations that: (1) the onset followed emotional stress; (2) that those afflicted had a characteristic personality configuration; and (3) that psychiatric symptomatology is frequently exhibited.

An early impetus for this viewpoint came from Jeliffe, who described the "obvious resemblances" between parkinsonians and catatonics. He indicated that, in both, the motility disturbances served to bind unconscious hostility; and likened the parkinsonian posture to that of a boxer.

Jackson and his co-workers, although taking a more traditional *organic* stance regarding etiology, observed that "the direct exciting causes are as a rule psychogenic." They called attention to the great frequency and variety of

psychiatric symptoms found in the disorder, noting that often these *preceded* the neurological signs. Depression was by far the commonest finding in their large series of patients, but many other symptoms were noted, including delusions, hallucinations, and agitation. It seems likely that some of the striking nature of these findings resulted from sampling bias, i.e., the authors were located at a state hospital. A more recent and conservative study carried out by Schwab, Fabing, and Prichard also emphasized the number and variety of psychiatric symptoms to be found in parkinsonians. These neurologists grouped the psychiatric disturbances into four categories: (1) unrelated, e.g., antedating the disorder; (2) reactive; (3) secondary to medication; and (4) paroxysmal, often associated with oculogyric crises and attributed to the CNS (central nervous system) pathology. The latter included episodes of anxiety, depression, compulsive acts, agitation, paranoia, and other symptom clusters.

The Parkinsonian Personality

The first major systematic studies of psychogenesis were carried out by Booth. Several series of patients, both here and in Germany, were studied by clinical interviews as well as the Rorschach. For the first time, control groups were utilized. From these data Booth concluded definitely that a "specific personality type" was to be found. He described this as characterized by an "urge to action" through motor activity and industriousness, and by a "striving for independence, authority and success within a rigid, usually

moralistic, behavior pattern." He adds that "the balance between success, aggression, and morals appears to be unusually delicate." Of special interest to us, Booth's controls included a group of patients with rheumatoid arthritis. He was struck by their many similarities to parkinsonians. Both disorders, he concluded, are dominated by an urge for independent action in which "obstacles are likely to provoke aggressiveness," and both are subject to conflicts between these feelings and a strict rigid conscience. The difference lay in their emotional relationships. Whereas the arthritics had a "*defensive* attitude against emotional involvement and outside influences," the parkinsonians were "*activated* by emotional experience coming from the environment" (my emphases). Booth's work represents one of the rare instances in which subtle psychological differences among given "psychosomatic" diseases have been dissected from their similarities. (These studies also included hypertensives, and delineated a different personality constellation for that disorder which will not be considered here). Unfortunately, methodological flaws open all of Booth's work to question, i.e., the patients were interviewed and tested by him alone, and his interpretations were made with full foreknowledge of the nature of the diagnoses.

Three clinical studies provided additional data that coincide with Booth's findings. Unfortunately, all are mere anecdotal reports devoid of controls. Mitscherlich reported on the presence of chronic emotional tension

in relation to aggression, leading to "readiness to motor activity without the possibility to realize it," resulting in a personality presenting as "coolness." Shaskan et al. regarded the needs for conformity and "virtuousness" to be so great as to produce an "unusually satisfactory" adjustment to the disease (a finding that seems at considerable variance from that of most observers). Sands considered these needs as sufficiently striking to characterize parkinsonians as having a "masked personality." The last two investigators emphasized that intense anxiety, anger, and conflict lay beneath the superficial calm and conformity.

Machover, who like Booth utilized Rorschach data, failed to confirm these observations. He found *no* evidence of any consistent personality picture. What little homogeneity he did elicit was limited to signs of cognitive interference, dependence, affective instability, inertia, and passivity—data at considerable variance with the industriousness and striving for independence reported by Booth. These were related to the duration of the disease and were explained as the consequences of *living with* a disabling disease that severely constrained activity. Unfortunately, he failed to use controls.

Stronger support for these negative findings have come from the program of well-designed studies of Riklan, Differ, and their associates. Their findings, together with a comprehensive review of the background of the problem, have been collected in an excellent monograph. Extensive data were

derived from systematic interviews with both patients and family members; from detailed clinical observations and examinations; and from a battery of psychological tests, both objective and projective, which tapped cognitive as well as personality attributes; and from controls. These data led to the conclusion that parkinsonians do *not* reveal distinctive behavioral features. Neither a "parkinsonian personality," nor even a typical reaction to the disease (such as reported by Machover) was found. Impairment in perceptual-cognitive functions was sometimes present but this covaried with the *severity* of the neurological impairment, not its duration. The investigators tend to view it as due to the disease process itself, not its symptoms.

Support for the findings in the cognitive area comes from a study by Talland. On the basis of a specially selected group of cognitive tests "No definite signs of impairment could be established." Patients off medication generally did better on all tests, suggesting that some of the defects observed clinically might be due to drug effects. Talland was by no means unaware of the existence of patients with severe intellectual impairment. But he differentiates these few from parkinsonians generally, and suggests that they may represent an etiologically distinct subgroup in which the pathological process directly disrupts brain functions. Riklan and his co-workers extended this line of thinking even further. The diversity of their findings led them to conclude that "parkinsonism refers to a number of complex and composite neurological syndromes," and, "it would be theoretically inconsistent and

practically useless to propose that parkinsonian patients define an entity generalizing its own behavioral characteristics." Here precisely lies the nub of a problem in accepting their negative findings as the final word on the question of a parkinsonian personality. Each subtype of the disorder has special features which act to obscure any held in common. If a specific personality configuration should exist, its presence can only be determined by eliminating these extraneous sources of variance. This requires the selection of uniform subgroups for study. The first task is to exclude patients whose disease arises from known exogenous factors, as well as all those in whom there is any evidence of intellectual or other diffuse neurological impairment. Demographic variables should then be used to create still more homogeneous subgroups. Even then, problems remain. One is left with patients whose impairment varies in extent and locus, and whose psychological reactions to these differ. Perhaps the question will only be answered when longitudinal prospective studies can be done. In any event, the present weight of the evidence favors caution about the existence of a parkinson-specific personality.

In contrast, the influence of psychological factors on parkinsonian symptoms, is an established fact. Symptoms frequently are increased in the presence of strong emotion, stress, and fatigue. This tells us nothing about etiology, since all diseases necessarily are responsive to psychological influence. A disorder of the CNS itself would seem especially likely to exhibit a

sensitivity to emotional arousal. The CNS is characterized by rich interconnections among its parts, and the hypothalamic and limbic structures which subserve emotion have known effects upon the musculature. Clinically this is apparent in the familiar extrapyramidal side effects of the phenothiazines. Nor can we make any etiological inferences from the symptomatic improvement that can result from psychotherapy.

However it is difficult indeed to explain away the careful report by Grinker and Spiegel of the development of the full-blown parkinsonian syndrome in cases of *combat exhaustion*, These patients were indistinguishable from typical "organic" parkinsonians, except that the entire picture rapidly disappeared with psychiatric treatment. A report also has appeared in which parkinsonian symptoms developed during the course of a schizophrenic illness, and disappeared following a lobotomy concomitant with symptomatic recovery from the psychosis. Psychodynamic study of this patient suggested that "the parkinson syndrome may have developed as a defense against the patient's violent hostility" (reminiscent of the aforementioned formulations of Jeliffe and Booth) as well as serving regressive needs.

Somatopsychic Correlations

Cooper's development of a stereotactic neurosurgical approach to

parkinsonism which involves the production of lesions in the thalamus and corpus pallidum, provides a new avenue for increasing our understanding of the functions of these parts of the brain. This work has been the subject of an excellent review by Crown, (which also considers some of the material covered above). In the *cognitive* sphere the findings are reasonably definite. There are transient losses postoperatively, which seem more related to verbal skills with left-sided lesions, and to performance and visual-motor skills with those on the right side. More important, no permanent cognitive defects can be ascertained.

A detailed clinico-pathological correlational study is available which clarifies some of the subtle language and speech effects of thalamic surgery.

Some interesting findings emerge with regard to *personality* effects following surgery, though these are far from clear. Riklan and Levita mention only two changes which seemed to be persistent. These were a defect in body image and a reduction in perceptual integration and nonspecific drive or energy. The latter, according to these workers, may reflect reduced kinesthetic feedback to the reticular activating system, a kind of relative sensory deprivation. This intriguing hypothesis receives some support from another of their studies. In a group of parkinsonians, a negative correlation was demonstrated between "activation level" (as measured by skin resistance) and the degree of voluntary motor impairment.

The reports of *mood* changes after surgery are somewhat conflicting. McFie comments on the "improved emotional reactions *following the alleviation of symptoms*" (my emphasis), and on the frequency of overt euphoria which he linked to right-sided surgery. He likens the reaction to that found with leukotomy. Hays also found euphoria to be the commonest affective reaction. However he attributed this to the specific CNS effects of the surgery, and found that it was *not* related to the degree of improvement. In contrast, Asso found that most affective changes were those of anxiety or depression, and that euphoria was rare. Unfortunately, there is no direct way to compare these studies. Uniform rating scales were not used, nor is it clear if there were equivalent preoperative levels of depression or expectancies of benefit from the surgery in the three groups.

Obviously, we have still much to learn about the role of these brain structures in personality and mood.

Gout

Gout is a disorder of uric acid metabolism in which symptoms arise from the formation of urate deposits in various body areas. The usual site for deposition is one or more of the joints, producing an acute, often exquisitely painful arthritis. Chronic joint deformities occasionally eventuate. The other major sites are the subcutaneous tissues, giving rise to the development of

nodular *tophi*, and the kidneys, resulting in nephritis and stone formation. The metabolic dyscrasia is ordinarily manifested by an elevated concentration of uric acid in the blood and other body fluids, but may only be evident in other subtler but measurable abnormalities (e.g., an increased size of the uric acid pool). Most research has identified the major metabolic defect as being an overproduction of uric acid, though its decreased conversion or excretion, or both, may also play a role.

Dietary overindulgence (increased purine intake), alcohol, exercise, and certain drugs can increase uric acid levels transiently to precipitate an acute attack. (The classically ascribed causative role of venery, however, seems dubious!) Trauma, infection, surgery, and other physical stressors also may act as precipitants. So can acute emotional stress. Sydenham, himself a victim, as quoted by Talbott, advised gout sufferers to "keep the mind quiet." However, it is now well substantiated that these factors have relevance only to the precipitation of acute attacks, and only to a small minority of these, whereas the pertinent issue for the disorder is a metabolic fault, in which such factors play no important role.

The disease was known as early as the fifth century B.C., when it was described by Heiron, a resident of ancient Syracuse. Its hereditary nature has been recognized nearly as long. Modern studies, while confirming the genetic factor, have amplified our understanding of it in important ways. It is likely

that multiple genes are involved, and that their effects are simply additive rather than interactive. The heritability factor has been quantified as ranging from approximately one fifth to one third, and being distinctly less important for males than females. Twin studies together with those of family incidence suggest strongly that, for males, environmental influences may outweigh the genetic as determinants of uric acid levels.

The relevance of this sex difference becomes apparent in the fact that more than 95 percent of gout occurs in men. Mean uric acid levels in males are approximately 5 mg. percent, a value about 1 mg. percent higher than that for women beginning from puberty. The difference converges somewhat in late life because of a rise in the level for women at menopause. The precipitation of uric acid in the tissues is, of course, a function of its concentration. As levels progressively exceed its solubility (ca. 6.4 mg. percent) there is a parallel increase in the probability that crystallization and deposition will occur.

The observation that the disease is more common among the affluent, eminent, and successful also goes back to antiquity. With the clarification of the role of uric acid, it has become possible to examine this relationship more closely. A substantial number of modern studies have provided both confirmation of its general validity, and a clarification that the crucial issue is not socioeconomic status per se, but the psychological characteristics of drive

achievement and leadership. The latter, of course, build the path to success and prominence. Mueller and his associates have provided an extensive review of this work.

The relationship with social status has been demonstrated in a variety of groups. Uric acid levels were found to be higher in executives than both craftsmen and normal controls, and higher in medical than high school students. Within a single plant, Oak Ridge, the highest mean values were found in the Ph.D. scientists and the lowest in craftsmen, with the supervisors and inspectors falling in between. Dodge and Mikkelson, as cited by Mueller, found the age corrected urate levels of professionals and executives to be higher than those of workers in unskilled jobs and farmers. State white-collar employees had a higher mean level than the general population.

By comparing subsamples within some of these and similar groups, it has become possible to delineate the role of *achievement-related behavior* from overall socioeconomic characteristics, since each group is more-or-less homogeneous with regard to the latter. Thus, in a Scottish study, the top executives had urate levels exceeding those found in those of lower rank. Executives enrolled in a summer Executive Development Program, and thus presumably of greater ambition, had uric acid levels exceeding those of an unselected group of executives. Among the state employees, those with the greatest number of job changes (considered to be an index of upward

mobility) had the higher levels. In a study of a group of men anticipating job termination, those with high uric acid levels were more likely to resign early to find another job.

Even closer to the point, within a sample of university professors, interview ratings of achievement motivation were found to correlate at a level of r = 66 with serum uric acid values. Similarly, Jenkins et al. found significant correlations, within a large group of supermarket employees, between serum urate levels and test items related to drive, competitiveness and challenging life circumstances, as cited by Mueller from a personal communication. Further confirmation is provided by a pilot study that utilized a measure of the motivational trait free from overt achievement behavior. Patients with gout and hyperuricemia had measurably higher levels of *need achievement* than did a control group of social work students.

The relationship holds also in regard to educational variables. The uric acid levels of high school and college students were positively related to the extent of their extra curricular activities, including those of a social and nonathletic type, and with test measures of achievement motivation. There does not appear to be any simple relationship of uric acid to grades. However, high school students with poor grades turned out to be more likely to go to college if they had high uric acid levels than if they did not, and within this group, uric acid levels correlated with the length of time they remained in

college. Approached from the other side, students attending or planning to attend college had higher uric acid values than those without such plans, above and beyond any association with grades.

A few reports have appeared that fail to demonstrate relationships between uric acid and social class. But, as of 1971, the weight of the evidence is so preponderant, both in numbers of studies and their meaningful consistencies, that it is difficult to be skeptical. It should be emphasized, however, that, with a single exception, all the findings involve studies done exclusively on *men*. Given the sex incidence of hyperuricemia and gout, this is not surprising. Moreover, the apparently greater role of genetic factors in women may dilute the effect of other variables, including those related to achievement. The one study done on women did show a suggestive relationship. In nursing students, a positive association was found between uric acid and extracurricular activities. It will be important to learn if this relationship does indeed hold true also for women.

In one of the aforementioned studies, uric acid measures were taken also on a group of women, the subjects' spouses. Because achievement indices were recorded only for their husbands, this study provides no contribution to the question of the relationship of this variable in women. These data are helpful in another way. The uric acid levels of the executives' and professionals' wives did not exceed that of the wives of the less skilled

workers. Thus, there is further support for believing that it is not their living styles (dietary and drinking habits etc.), nor other aspects of social class per se which are involved in the elevated uric acid levels found in their husbands. It might be added that various details of design in several of the other studies mentioned above lead to further confidence that such exogenous factors are not responsible for the findings.

There is one additional facet of this whole body of research that is of unique significance. Because the subjects had hyperuricemia but not gout, the psychological findings cannot be ascribed to any secondary effects of suffering from that disease. In this sense, this is prospective research, free from the potential error inherent in the retrospective method.

Assuming that the relationship between uric acid and achievement behavior and/or motivation does exist, how are we to understand it? The possibility arises that uric acid overproduction is a concomitant of chronic stress arising from the drive to success and the effort attendant upon its achievement. There are two studies which provide direct evidence that short-term psychological stress is associated with a rise in uric acid. In a group of Navy frogmen during training, Bahe and his co-workers found uric acid elevations just prior to the start of training (the familiar pre-experimental anticipation effect that occurs in many stress variables.) Bises in uric acid also occurred during periods when the trainees approached demanding tasks with

an “optimistic” attitude, while drops were noted during a period when they felt “overburdened” and less assured of success. Similar findings occurred in a study of stably employed men experiencing job loss because of a plant closing. Anticipation of job loss was associated with elevations of uric acid which dropped following new employment. The duration of the rise tended to parallel the length of time it had taken to find the new job. Of special interest, those men who resigned prior to termination to obtain a new job had *stable* higher uric acid levels. This latter behavior not only implies greater achievement drive, it also suggests a greater degree of optimism. Furthermore, for a small subgroup in which psychological measures could be made reliably, a combined rating of sadness, low self-esteem, and anxiety correlated negatively with uric acid levels.

Thus, these studies contribute to the understanding of the relationship of uric acid to transient emotional states, as well as to the larger body of work on its relationship to enduring personality traits. They also provide an intriguing lead for better *delineating achievement* drive in terms of the attitude associated with it. Additional confirmation of this lead can be found in closer scrutiny of some of the previously mentioned studies of the achievement trait. More frequent job change seems interpretable in terms of optimism and a sense of active mastery, as well as of upward mobility. So does the willingness to attempt college. Moreover the uric acid levels in college professors, which were positively correlated with ratings of

achievement motivation, were concomitantly negatively correlated with reported feelings of being overburdened and worried about their jobs. Also consistent with this point is the finding that high school students with lower uric acid levels had more unrealistic vocational expectations and aspirations than those with high levels (the degree to which such goals are unrealistic being a concomitant of achievement avoidance) .

It would be difficult to overestimate the importance of this type of clarification. Such traits as *drive, achievement*, and *leadership* represent global qualities which may subsume or even obscure more narrowly and precisely defined personality attributes. It is essential to separate the latter from the grosser traits within which they are imbedded. (An excellent example of such an endeavor is to be found in Jenkin's delineation of the separate traits included within the *coronary-prone personality*.) The justification for this viewpoint comes not only from considerations of logic but from its demonstrated payoff. Thus, in several of the studies reported, cholesterol levels were measured also and showed a very different relationship with the psychological variables than did uric acid. In general the relation of cholesterol to the personality continuum of the optimism sense of mastery vs. less assured overburdened was just opposite to that for uric acid. Since coronary disease is accompanied by elevation in the mean levels both of cholesterol and uric acid, the clarification of the differential personality correlates of the two substances is of considerable interest.

The correlation between personality traits and biochemical or physiological variables does not, of course, indicate a causal relationship. Either or both may be mere derivative products of other, more central factors. Even if the relationship were causal, the psychological variable need not be the primary factor. As a matter of fact, Orowan has offered the interesting converse hypothesis that uric acid acts as an endogenous cortical stimulant. Consistent with this is the positive relationship between uric acid and IQ levels. However, this correlation, albeit statistically significant, is very low ($r \cong 0.1$), and, in any event, this hardly constitutes validation of the hypothesis.

Clearly there remains much work to be done. In this instance such a statement is no mere cliche. Rather it reflects an existing stage of accomplishment, important not merely in itself but in the clear directions it provides for further research. The elucidation of the relationship of uric acid and personality represents one of the brighter areas of psychosomatic research.

Bibliography

Abraham, K. (1913) "A Constitutional Basis of Locomotor Anxiety," in *Selected Papers on Psychoanalysis,* pp. 235-243. London: Hogarth, 1949.

Acheson, R. M. and W. O'Brien. "Some Factors Associated with Serum Uric Acid in the New Haven Survey of Joint Disease," in P. H. Bennett and P. Wood, eds., *Population Studies of the Rheumatic Diseases,* pp. 365-370. Amsterdam: Excerpta Medica Foundation, 1968.

Alexander, F. *Psychosomatic Medicine. New York: Norton, 1950.*

Alexander, F., T. French, and G. H. Pollock. *Psychosomatic Specificity.* Chicago: University of Chicago Press, 1968.

Allport, G. W. and P. Vernon. *Studies in Expressive Movement.* New York: Macmillan, 1933.

Amkraut, A. A., F. Solomon, and C. Kraemer. "Stress, Early Experience and Adjuvant-Induced Arthritis in the Rat," *Psychosom. Med.,* 33 (1971), 203-214.

Anumonye, A., J. Dobson, S. Oppenheim et al. "Plasma Uric Acid Concentrations among Edinburgh Business Executives," *JAMA,* 208 (1969), 1141-1148.

Ascher, E. "Motor Attitudes and Psychotherapy," *Psychosom. Med.,* 11 (1949), 228-234.

Asso, D., S. Crown, J. A. Russell et al. "Psychological Aspects of the Stereotactic Treatment of Parkinsonism," *Br. J. Psychiatry,* 115 (1969), 541-553.

Avni, J. and J. Chaco. "Objective Measurements of Postural Changes in Depression." Paper presented at the Annual Meeting of the American Psychosomatic Society, Boston, April 1972. Unpublished.

Ax, A. F. "The Physiological Differentiation between Fear and Anger in Humans," *Psychosom. Med*., 15 (1953), 433-442.

Ax, A. F., J. Bamford et al. "Autonomic Response Patterning of Chronic Schizophrenics," *Psychosom. Med.,* 31 (1969), 353-364.

Balshan, I. D. "Muscle Tension and Personality in Women," *Arch. Gen. Psychiatry,* 7 (1962), 436-448.

Barlow, W. "Anxiety and Muscle Tension," in D. O'Neill, ed., *Modern Trends in Psychosomatic Medicine.* New York: Hoeber, 1955.

Bennett, L. A. "The Personality of the Rheumatoid Arthritis Patient." Master's thesis, McGill University, Montreal, 1952. Unpublished.

Bennett, P. and P. H. N. Wood, eds., *Population Studies of the Rheumatic Diseases,* International Congress Series no. 148. Amsterdam: Excerpta Medica Foundation, 1968.

Birdwhistell, R. L. *Introduction to Kinesics.* Louisville: University of Louisville Press, 1952.

---- . Kinesics and Context. Philadelphia: University of Pennsylvania Press, 1970.

Bland, J. H., ed. "Symposium on Rheumatoid Arthritis," *Med. Clin. North Am.,* 52 (1968), 477-769.

Blom, G. E. and G. Nicholls. "Emotional Factors in Children with Rheumatoid Arthritis," *Am. J. Orthopsychiatry,* 24 (1954), 588-600.

Boland, E. W. "Psychogenic Rheumatism: The Musculoskeletal Expression of Psychoneurosis," *Calif. Med.,* 68 (1948), 273-279.

----. "Psychogenic Factors in Rheumatic Disease," in J. L. Hollander, ed., Arthritis and Allied Conditions, 6th ed. Philadelphia: Lea & Febiger, 1960.

Boland, E. W. and W. P. Corr. "Psychogenic Rheumatism," *JAMA,* 123 (1943), 805-809.

Boman, K. "Effect of Emotional Stress on Spasticity and Rigidity," *J. Psychosom. Res.,* 15 (1971), 107-112.

Booth, G. "Personality and Chronic Arthritis," *J. Nerv. Ment. Dis.,* 85 (1937), 637-662.

----. "Objective Techniques in Personality Testing," *Arch. Neurol. Psychiatry,* 42 (1939), 514-530.

----. "Organ Function and Form Perception," *Psychosom. Med*., 8 (1946), 367-385.

----. "Psychodynamics in Parkinsonism," *Psychosom. Med*., 10 (1948), 1-14.

Bourestom, N. C. and M. T. Howard. Personality Characteristics of Three Disability Groups," *Arch. Phys. Med. Rehahil.,* 46 (1965). 626-632.

Brooks, G. W. and E. Mueller. "Serum Urate Concentrations among University Professors," *JAMA,* 195 (1966), 415-418.

Bull, N. *The Attitude Theory of Emotion.* New York: Coolidge Foundation, 1951.

Cameron, D. E. "Observations on the Patterns of Anxiety," *Am. J. Psychiatry,* 101 (1944), 36-41.

Cameron, N. *The Psychology of Behavior Disorders. Boston: Houghton Mifflin, 1947.*

Chapman, A. H. *Management of Emotional Disorders: A Manual for Physicians.* Philadelphia: Lippincott, 1962.

Christian, C., ed. "Nineteenth Rheumatism Review," *Arthritis Rheum.,* 13 (1970), 474-711.

Cleveland, S. and S. Fisher. "Behavior and Unconscious Fantasies of Patients with Rheumatoid Arthritis," *Psychosom. Med.,* 16 (1954), 327-333.

----. "A Comparison of Psychological Characteristics and Physiological Reactivity in Ulcer and Rheumatoid Arthritis Groups: I. Psychological Measures," Psychosom. Med., 22 (1960), 283-289.

Cleveland, S., E. E. Reitman, and E. J. Brewer, Jr. "Psychological Factors in Juvenile Rheumatoid Arthritis," *Arthritis Rheum.,* 8 (1965), 1152-1158.

Cobb, Sidney. "Contained Hostility in Rheumatoid Arthritis," *Arthritis Rheum.,* 2 (1959), 419-425.

----. "Hyperuricemia in Executives," in J. H. Kellgren, ed., The Epidemiology of

Chronic Rheumatism, pp. 182-196. Philadelphia: Davis, 1963.

----. *The Frequency of Rheumatic Diseases.* Cambridge, Mass.: Harvard University Press, 1969.

Cobb, Sidney and T. Lincoln. "On the Frequency of Individuals Who Suffer Occasional Attacks of Rheumatoid Arthritis," *Arthritis Rheum.,* 3 (1960), 48.

Cobb, Sidney, M. Miller, and M. Wieland. "On the Relationship between Divorce and Rheumatoid Arthritis," *Arthritis Rheum.,* 2 (1959), 414-418.

Cobb, Sidney, W. J. Schull, E. Harburg et al. "The Intrafamilial Transmission of Rheumatoid

Arthritis," *J. Chronic Dis.,* 22 (1968), 195-296.

Cobb, Stanley, W. Bauer, and I. Whiting. "Environmental Factors in Rheumatoid Arthritis," *JAMA,* 113 (1939), 668-670.

Cooper, I. S. *The Neurosurgical Alleviation of Parkinsonism.* Springfield, Ill.: Charles C. Thomas, 1956.

Cormier, B. M. and E. Wittkower. "Psychological Aspects of Rheumatoid Arthritis," *Can. Med. Assoc. J.,* 77 (1957), 533-541.

Crisp, A. H. and H. Moldofsky. "A Psychosomatic Study of Writer's Cramp," *Br. J. Psychiatry,* 111 (1965), 841-858.

Crown, S. "Psychosomatic Aspects of Parkinsonism," *J. Psychosom. Res.,* 15 (1971), 451-459.

Damaser, E. C., R. Shor, and M. Orne. "Physiological Effects during Hypnotically Requested Emotions," *Psychosom. Med.,* 25 (1963), 334-343.

Davidowitz, J., A. N. Brown-MAYERS, R. Kohl et al. "An Electromyographic Study of Muscular Tension," *J. Psychol.,* 40 (1955), 85-94.

Davis, F. H. and R. B. Malmo. "Electromyographic Recording during Interview," *Am. J. Psychiatry,* 107 (1951), 908-915.

Davis, J. F. *Manual of Surface Electromyography. Montreal: Allan Memorial Institute of Psychiatry, 1952.*

Davis, J. F., R. B. Malmo, and C. Shagass. "Electromyographic Reaction to Strong Auditory Stimulation in Psychiatric Patients," *Can. J. Psychol.,* 8 (1954), 177-186.

Davis, R. C. "Methods of Measuring Muscular Tension," *Psychol. Bull.,* 39 (1942), 329-346.

Decker, J. L. "The Epidemiology of Hyperuricemia: A Summary," in G. Katona and J. R. Gill, eds., *Panamerican Rheumatology,* pp. 74-75. Amsterdam: Excerpta Medica Foundation, 1969.

Deutsch, F. "Analysis of Postural Behavior," *Psychoanal. Q.,* 16 (1947), 195-213.

Duffy, E. "The Measurement of Muscular Tension as a Technique for the Study of Emotional Tendencies," *Am. J. Psychol.,* 44 (1932), 146-162.

----. "The Relation between Muscular Tension and Quality of Performance," *Am. J. Psychol*., 44 (1932), 535-546.

----. "Level of Muscular Tension as an Aspect of Personality," *J. Gen. Psychol*., 35 (1946), 161-171.

----. *Activation and Behavior.* New *York: Wiley, 1962.*

Dunbar, H. F. *Emotions and Bodily Changes.* New York: Columbia University Press, 1954.

Dunn, j., W. G. Brooks, J. Mausner et al. "Social Class Gradient of Serum Uric Acid Levels in Males," *JAMA,* 185 (1963), 431-436.

Ehrlich, G. E. "Psychosomatic Aspects of Musculoskeletal Disorders," *Postgrad. Med.,* 38 (1965), 614-619.

Eleftheriou, B. and J. P. Scott. *The Physiology of Aggression and Defeat.* New York: Plenum, 1971.

Engel, G. L. "Studies of Ulcerative Colitis: I Clinical Data Bearing on the Nature of the Somatic Process," *Psychosom. Med.,* 16 (1954), 496-501.

----. "Studies of Ulcerative Colitis: II. The Nature of the Somatic Processes and the Adequacy of Psychosomatic Hypotheses," Am. J. Med., 16 (1954), 416-433.

----. "Studies of Ulcerative Colitis: III. The Nature of the Psychologic Processes," *Am. J. Med*., 19 (1955), 231-256.

Fenichel, O. *The Psychoanalytic Theory of Neurosis.* New York: Norton, 1945.

Ferenczi, S. (1919) "Thinking and Muscle Innervation," in J. Rickman, ed. *Further Contributions to the Theory and Technique of Psychoanalysis,* pp. 230-232. New York: Basic Books, 1952.

Fessel, W. J. "Mental Stress, Blood Proteins and the Hypothalamus," *Arch. Gen. Psychiatry,* 7 (1962), 427-435.

Fischman, D. A., H. Meltzer, and R. Poppei. "Disruption of Myofibrils in the Skeletal Muscle of Psychotic Patients," *Arch. Gen. Psychiatry,* 23 (1970), 503-513.

Fisher, S. *Body Experience in Fantasy and Behavior.* New York: Appleton-Century-Crofts, 1970.

Fisher, S. and S. E. Cleveland. "A Comparison of Psychological Characteristics and Physiological Reactivity in Ulcer and Rheumatoid Arthritis Groups: II. Differences in Physiological Reactivity," *Psychosom. Med.,* 22 (1960), 290-293.

----. *Body Image and Personality,* 2nd ed. New York: Dover, 1968.

Freeman, G. L. "Postural Tensions and the Conflict Situation," *Psychol. Rev.,* 46 (1939). 226-240.

French, J. G., H. J. Dodge, M. O. Kjelsberg et al. "A Study of Familial Aggregation of Serum Uric Acid Levels in the Population of Tecumseh, Michigan, 1959-60," *Am. J. Epidemiol.,* 86 (1967), 214-224.

French, T. and L. Shapiro. "The Use of Dream Analysis in Psychosomatic Research," *Psychosom. Med.,* 11 (1949), 110-112.

Freud, S. *The Problem of Anxiety.* New York: Norton, 1936.

----. (1901) *The Psychopathology of Everyday Life, in J. Strachey, ed., Standard Edition,* Vol. 6. London: Hogarth, 1953.

Geist, H. *The Psychological Aspects of Rheumatoid Arthritis.* Springfield, Ill.: Charles C. Thomas, 1966.

Gellhorn, E. *Physiological Foundations of Neurology and Psychiatry.* Minneapolis: University of Minnesota Press, 1953.

Gertler, M. M., S. M. Garn, and S. A. Levine. "Serum Uric Acid in Relation to Age and Physique in Health and in Coronary Heart Disease," *Ann. Intern. Med.,* 34 (1951), 1421-1431.

Glover, E. *Psycho-analysis.* London: Staples Press, 1949.

Gold, S. "Psycho-Genesis in the 'Stiff-Man Syndrome'," *Guys Hosp. Rep.,* 114 (1965), 279-285.

Goldstein Balshan, I. "Role of Muscle Tension in Personality Theory," *Psychol. Bull.,* 61 (1964), 413-425.

----. "Physiological Responses in Anxious Women Patients," Arch. Gen. Psychiatry, 10 (1964), 382-388.

----. "The Relationship of Muscle Tension and Autonomic Activity to Psychiatric Disorders," *Psychosom. Med.,* 27 (1965), 39-52.

Goldstein Balshan, I., R. R. Grinker, Sr., H. A. Heath et al. "Study in Psychophysiology of Muscle Tension: I. Response Specificity," *Arch. Gen. Psychiatry,* 11 (1964), 322-330.

Gordon, R. E., R. Lindeman, and K. Gordon. "Some Psychological and Biochemical Correlates of College Achievement," *J. Am. College Health Assoc.,* 15 (1967), 326-331.

Gottschalk, L., M. Serota, and K. Roman. "Handwriting in Rheumatoid Arthritics," *Psychosom. Med.,* 11 (1949), 354-360.

Gottschalk, L., H. Serota, and L. Shapiro. "Psychologic Conflict and Neuromuscular Tension," *Psychosom. Med.,* 12 (1950), 315-319.

Grace, W. J. and D. Graham. "Relationship of Specific Attitudes and Emotions to Certain Bodily Diseases," *Psychosom. Med.,* 14 (1952), 243-251.

Graham, D. T., R. M. Lundy, L. S. Benjamin et al. "Specific Attitudes in Initial Interviews with Patients Having Different 'Psychosomatic' Diseases," *Psychosom. Med.,* 24 (1962), 257-266.

Gregg, D. "The Paucity of Arthritis among Psychotic Cases," *Am. J. Psychiatry,* 95 (1939). 853-856.

Grinker, R. R., Sr., S. J. Korchin, Basowitz et al. "A Theoretical and Experimental Approach to Problems of Anxiety," *Arch Neurol. Psychiatry,* 76 (1956), 420-431.

Grinker, R. R., Sr. and J. Spiegel. *War Neurosis in North Africa: The Tunisian Campaign.* New York: Josiah Macy Foundation, 1943.

Grokoest, A. W., I. Snyder, and R. Schlaeger. *Juvenile Rheumatoid Arthritis.* Boston: Little, Brown, 1962.

Grossman, W. I. and H. Weiner. "Some Factors Affecting the Reliability of Surface Electromyography," *Psychosom. Med.,* 28 (1966), 78-83.

Guggenheim, P. and L. Cohen. "A Case of Schizophrenia in which Manifestations of Parkinsonism Appeared during the Course of the Psychosis and Disappeared after Lobotomy," *Psychosom. Med.,* 20 (1958), 151-160.

Gutman, A. B., ed. "Proceedings of Conference on Gout and Purine Metabolism," *Arthritis Rheum.,* 8 (1965), 589-910.

Halliday, J. L. "Psychological Factors in Rheumatism," *Br. Med. J.,* 1 (1937), 213-217; 264-269.

----. "The Concept of Psychogenic Rheumatism," *Ann. Intern. Med.,* 15 (1941), 666-677.

----. "The Psychological Approach to Rheumatism," *Proc. R. Soc. Med.,* 35 (1941), 455-457.

----. "Concept of a Psychosomatic Affection," *Lancet,* 2 (1943), 692-696.

Hays, P., et al. "Psychological Changes following Surgical Treatment of Parkinsonism," *Am. J. Psychiatry,* 123 (1966), 657¯663.

Heath, H. A., D. Oken, and W. G. Shipman. "Muscle Tension and Personality," *Arch. Gen. Psychiatry,* 16 (1967), 720-726.

Heath, H. A., D. Oken, and W. G. Shipman et al. "Three Factor Analyses of Electromyographic Data Under Varying Conditions," *Multivariate Behav. Res.,* 2 (1967), 263-280.

Hellgren, L. "Marital Status in Rheumatoid Arthritis," *Acta Rheumatol. Scand.,* 15 (1969), 271-276.

Hench, P. S. and E. W. Boland. "The Management of Chronic Arthritis and Other Rheumatic

Diseases among Soldiers of the United States Army," *Ann. Intern. Med.,* 24 (1946), 808-825.

Hendrie, H. C., F. Paraskevas, and J. D. Adamson. "Stress, Immunoglobulin Levels and Early Polyarthritis," *J. Psychosom. Res.,* 15 (1971), 337-342.

----. "Gamma Globulin Levels in Psychiatric Patients," *Can. Psychiatr. Assoc. J.,* 17 (1972), 93-97.

Hollander, J. L., ed. *Arthritis and Allied Conditions: A Textbook of Rheumatology,* 6th ed. Philadelphia: Lea & Febiger, 1960.

Holmes, T. H. and H. Wolff. "Life Situations, Emotions, and Backache," *Psychosom. Med.,* 14 (1952), 18-33.

Inman, V. T., H. J. Ralston, J. B. de C. M. Saunders et al. "Relation of Human Electromyogram to Muscular Tension," *Electroencephalogr. Clin. Neurophysiol.,* 4 (1952), 187-194.

Jackson, J. A., G. B. M. Free, and H. V. Pike. "The Psychic Manifestations in Paralysis Agitans," *Arch. Neurol. Psychiatry,* 10 (1923), 680-684.

Jacobson, E. *Progressive Relaxation.* Chicago: University of Chicago Press, 1938.

Jacobson, E., ed. *Tension in Medicine.* Springfield, Ill.: Charles C. Thomas, 1967.

James, W. *The Principles of Psychology.* New York: Holt, 1890.

Jeffrey, M. R. and J. Ball. *The Epidemiology of Chronic Rheumatism.* Philadelphia: Davis, 1963.

Jelliffe, S. E. "The Mental Pictures in Schizophrenia and in Epidemic Encephalitis," Am. *J. Psychiatry,* 6 (1926), 413-465.

----. "The Parkinsonian Body Posture," *Psychoanal. Rev.,* 27 (1940), 467.

Jelliffe, S. E. and W. A. White. *Diseases of the Nervous System.* Philadelphia: Lea & Febiger, 1935.

Jenkins, C. D., S. J. Zyzanski, and R. H. Rosenman. "Progress toward Validation of a Computer-

Scored Test for the Type A Coronary-Prone Behavior Pattern," *Psychosom. Med.,* 33 (1971), 193-202.

Johnson, A. M., L. Shapiro, and F. Alexander. "Preliminary Report on a Psychosomatic Study of Rheumatoid Arthritis," *Psychosom. Med.,* 9 (1947), 295-300.

Kasl, S. V., G. W. Brooks, and Sidney Cobb. "Serum Urate Concentrations in Male High School Students, a Predictor of College Attendance," *JAMA,* 198 (1966), 713-716.

Kasl, S. V., G. W. Brooks, and W. Rodgers. "Serum Uric Acid and Cholesterol in Achievement Behavior and Motivation: I. The Relationship to Ability, Grades, Test Performance, and Motivation," *JAMA,* 213 (1968), 1158-1164.

----. "Serum Uric Acid and Cholesterol in Achievement Behavior and Motivation: I. The Relationship to College Attendance, Extracurricular and Social Activities, and Vocational Aspirations," *JAMA*, 213 (1970), 1291-1299.

Kasl, S. V., Sidney Cobb, and G. W. Brooks. "Changes in Serum Uric Acid and Serum Cholesterol in Men Undergoing Job Loss*," JAMA,* 206 (1968), 1500-1507.

Kempe, J. E. "An Experimental Investigation of the Relationship Between Certain Personality Characteristics and Physiological Responses to Stress in a Normal Population." Ph.D. dissertation, Michigan State University, 1956. Unpublished.

Kepecs, J. G. "Some Patterns of Somatic Displacement," *Psychosom. Med.,* 15 (1953), 425-432.

King, H. E. "Psychomotility: A Dimension of Behavior Disorder," *Proc. Am. Psychopathol. Assoc.,* 58 (1969), 99-128.

King, S. H. "Psychosocial Factors Associated With Rheumatoid Arthritis," *J. Chronic Dis.,* 2 (1955), 287-302.

King, S. H. and Sidney Cobb. "Psychosocial Factors in the Epidemiology of Rheumatoid Arthritis," *J. Chronic Dis.,* 7 (1958), 466-475.

----. "Psychosocial Studies of Rheumatoid Arthritis: Parental Factors Compared in Cases and

Controls," *Arthritis Rheum.,* 2 (1959). 322-331.

Lacey, J. I. "Psychophysiological Approaches to the Evaluation of Psychotherapeutic Process and Outcome," in E. A. Rubinstein and M. B. Parloff, eds., *Research in Psychotherapy,* pp. 160-208. Washington, D.C.: American Psychological Association, 1959.

----. "Somatic Response Patterning and Stress: Some Revisions of Activation Theory," in M. H. Appley and R. Trumbull, eds., Psychological Stress, pp. 14-37. New York: Appleton-Century-Crofts, 1967.

Lacey, J. I., D. E. Bateman, and R. Vanlehn. "Autonomic Response Specificity," *Psychosom. Med.,* 15 (1953), 8-21.

Lacey, J. I. and B. Lacey. "The Relationship of Resting Autonomic Activity to Motor Impulsivity," *Res. Publ. Assoc. Res. Nerv. Ment. Dis.*, 36 (1956), 144-209.

Lader, M. H. and A. M. Matthews. "Electromyographic Studies of Tension," *J. Psychosom. Res.,* 15 (1971), 479-486.

Lanese, R. R., G. Gresham, and M. D. Keller. "Behavioral and Physiological Characteristics in Hyperuricemia," *JAMA,* 207 (1969), 1878-1882.

Lewis-Fanning, E. "Report on an Enquiry into the Aetiological Factors Associated with Rheumatoid Arthritis," *Ann. Rheum. Dis.*, 9 (1950), Suppl.

Lindsley, D. B. "Emotion," in S. S. Stevens, ed., *Handbook of Experimental Psychology,* pp. 473-516. New York: Wiley, 1951.

Lippold, O. C. J. "The Relation between Integrated Action Potentials in a Human Muscle and Its Isometric Tension," *J. Physiol.,* 117 (1952), 492-499.

Lovaas, O. I. "The Relationship of Induced Muscular Tension, Tension Level, and Manifest Anxiety in Learning," *J. Exp. Psychol.,* 59 (1960), 145-152.

Lowman, E. W., ed. *Arthritis: General Principles, Physical Medicine, Rehabilitation.* Boston: Little, Brown, 1959.

Lowman, E. W., S. Miller, P. Lee et al. “Psycho-Social Factors in Rehabilitation of the Chronic Rheumatoid Arthritic,” *Ann. Rheum. Dis.,* 13 (1954), 312-316.

Ludwig, A. O. “Emotional Factors in Rheumatoid Arthritis,” *Physiother. Rev.,* 29 (1949), 339-342.

----. “Psychogenic Factors in Rheumatoid Arthritis,” *Bull. Rheum. Dis.,* 2 (1952), 33-34.

----. “Rheumatoid Arthritis,” in E. D. Wittkower and R. A. Cleghorn, eds., *Recent Developments in Psychosomatic Medicine*, pp. 232-244. Philadelphia: Lippincott, 1954.

Lundervold, A. “An Electromyographic Investigation of Tense and Relaxed Subjects,” *J. Nerv. Ment. Dis.,* 115 (1952), 512-525.

Luria, A. *The Nature of Human Conflicts.* New York: Liveright, 1932.

Luthe, W., ed. *Autogenic Training: Psychosomatic Correlations.* New York: Grune & Stratton, 1964.

Machover, S. “Rorschach Study on the Nature and Origin of Common Factors in the Personalities of Parkinsonians,” *Psychosom. Med.,* 19 (1957), 332-338.

MacLean, P. D. “The Limbic (‘Visceral Brain’) in Relation to Central Gray and Reticulum of the Brain Stem,” *Psychosom. Med.,* 17 (1955), 355-366.

Malmo, R. B. “Symptom Mechanisms in Psychiatric Patients,” *Trans. N.Y. Acad. Sci.,* 18 (1956), 545-549.

----. “Activation: A Neuropsychological Dimension,” *Psychol. Rev*., 66 (1959), 367-386.

Malmo, R. B., T. J. Boag, and A. A. Smith. “Physiological Study of Personal Interaction,” *Psychosom. Med.,* 19 (1957), 105-119.

Malmo, R. B. and C. Shagass. “Physiologic Studies of Reaction to Stress in Anxiety and Early Schizophrenia,” *Psychosom. Med.,* 11 (1949), 9-24.

----. “Physiologic Study of Symptom Mechanisms in Psychiatric Patients under Stress,” *Psychosom. Med*., 11 (1949), 25-29.

----. “Studies of Blood Pressure in Psychiatric Patients under Stress,” *Psychosom. Med*., 14 (1952), 82-93.

Malmo, R. B., C. Shagass, and F. H. Davis. “Symptom Specificity and Bodily Reactions during Psychiatric Interview,” *Psychosom. Med.,* 12 (1950), 363-376.

Malmo, R. B., C. Shagass, and J. F. Davis. “A Method for the Investigation of Somatic Response Mechanisms in Psychoneurosis,” *Science,* 112 (1950), 325-328.

----. “Electromyographic Studies of Muscular Tension in Psychiatric Patients under Stress,” *J. Clin. Exp. Psychopathol*., 12 (1951), 45-66.

Malmo, R. B., C. Shagass, and A. Smith. “Responsiveness in Chronic Schizophrenia,” *J. Pers.,* 19 (1951), 359-375.

Malmo, R. B., A. A. Smith, and W. A. Kohlmeyer. “Motor Manifestation of Conflict in Interview: A Case Study,” *J. Abnorm. Soc. Psychol.,* 52 (1956), 268-271.

Marschall, W. “The Psychopathology and Treatment of the Parkinsonian Syndrome and Other Post-Encephalitic Sequelae,” *J. Nerv. Ment. Dis.,* 84 (1936), 27-45.

Martin, I. “Levels of Muscle Activity in Psychiatric Patients,” *Acta Psychol.* (*Amst.),* 12 (1956), 326-341.

Martin, I. and H. J. Grosz. “Hypnotically Induced Emotions,” *Arch. Gen. Psychiatry,* 11 (1964). 203-213.

Martin, I. and L. Rees. “Reaction Times and Somatic Reactivity in Depressed Patients,” *J. Psychosom. Res.,* 9 (1966), 375-382.

McFie, J. “Psychological Effects of Stereotaxic Operations for the Relief of Parkinsonian Symptoms,” *J. Ment. Sci.,* 106 (1960), 1512-1517.

McLaughlin, J. T., R. N. Zabarenko, P. B. Diana et al. “Emotional Reactions of Rheumatoid Arthritics to ACTH,” *Psychosom. Med.,* 15 (1953), 187-199.

Meltzer, H. Y. and W. Engel. “Histo-chemical Abnormalities of Skeletal Muscle in Acutely Psychotic Patients,” *Arch. Gen. Psychiatry,* 23 (1970), 492-502.

Meltzer, H. Y. and R. Moline. “Muscle Abnormalities in Acute Psychoses,” *Arch. Gen. Psychiatry,* 23 (1970), 481-491.

Mersy, D. J., T. Auyong, and R. Eelkema. “Relationship of Serum Urate Levels to Occupation and Mental Retardation,” *Psychosomatics,* 9 (1968), 199-201.

Meyer, D. R. “On the Interaction of Simultaneous Responses,” *Psychol. Bull.,* 50 (1953), 204-220.

Meyer, D. R., H. P. Bahrick, and P. M. Fitts. “Incentive, Anxiety, and the Human Blink Rate,” *J. Exp. Psychol.,* 45 (1953), 183-187.

Meyerowitz, S. “Editorial: Psychosocial Factors in the Etiology of Somatic Disease,” *Ann. Intern. Med.,* 72 (1970), 753-754.

----. “The Continuing Investigation of Psychosocial Variables in Rheumatoid Arthritis,” in A. Hill, ed., *Modern Trends in Rheumatology*, pp. 92-105. New York: Appleton-Century-Crofts, 1971.

Meyerowitz, S., R. Jacox, and D. W. Hess. “Monozygotic Twins Discordant for Rheumatoid Arthritis: A Genetic, Clinical and Psychological Study of 8 Sets,” *Arthritis Rheum.,* 11 (1968), 1-21.

Mikkelsen, W. M., H. Dodge, and H. Valkenburg. “The Distribution of Serum Uric Acid Values in a Population Unselected as to Gout or Hyperuricemia,” *Am. J. Med.,* 39 (1965), 242-251.

Mitscherlich, M. “The Psychic State of Patients Suffering from Parkinsonism,” in A. Jores and H. Freyberger, eds., *Advances in Psychosomatic Medicine,* pp. 317-324. New York: Brunner, 1961.

Moldofsky, H. “Occupational Cramp,” *J. Psychosom. Res.,* 15 (1971), 439-444.

Moldofsky, H. and W. J. Chester. “Pain and Mood Patterns in Patients with Rheumatoid Arthritis,”

Psychosom. Med., 32 (1970), 309-318.

Moldofsky, H. and A. I. Rothman. "Personality, Disease Parameters and Medication in Rheumatoid Arthritis," *Arthritis Rheum.,* 13 (1970), 338-339.

Montoye, H. J., J. A. Faulkner, H. J. Dodge et al. "Serum Uric Acid Concentration among Business Executives," *Ann. Intern. Med.,* 66 (1967), 838-850.

Moos, R. H. "Personality Factors Associated With Rheumatoid Arthritis: A Review," *J. Chronic Dis.*, 17 (1964), 41-55.

Moos, R. H. and B. T. Engel. "Psychophysiological Reactions in Hypertensive and Arthritic Patients," *J. Psychosom. Res.,* 6 (1962), 227-241.

Moos, R. H., and G. Solomon. "Minnesota Multiphasic Personality Inventory Response Patterns in Patients with Rheumatoid Arthritis," *J. Psychosom. Res.,* 8 (1964), 17 28.

----. "Psychologic Comparisons between Women with Rheumatoid Arthritis and Their Non-arthritic Sisters: I. Personality Test and Interview Rating Data," *Psychosom. Med*., 27 (1965), 135-149.

----. "Psychologic Comparisons Between Women with Rheumatoid Arthritis and Their Non-arthritic Sisters: II. Content Analysis of Interviews," *Psychosom. Med*., 27 (1965), 150-164.

----. "Personality Correlates of the Degree of Functional Incapacity of Patients with Physical Disease," *J. Chronic Dis*., 18 (1965), 1019-1038.

Morrison, L. R., C. L. Short, A. O. Ludwig et al. "The Neuromuscular System in Rheumatoid Arthritis," *Am. J. Med. Sci.,* 214 (1947), 2-49.

Mueller, A. D. and A. M. Lefkovits. "Personality Structure and Dynamics of Patients with Rheumatoid Arthritis," *J. Clin. Psychol.,* 12 (1956), 143-147.

Mueller, A. D., A. M. Lefkovits, J. E. Bryant et al. "Some Psycho-social Factors in Patients with Rheumatoid Arthritis," *Arthritis Rheum.,* 4 (1961), 275-282.

Mueller, E. F., S. V. Kasl, G. W. Brooks et al. “Psychosocial Correlates of Serum Urate Levels,” *Psychol. Bull.,* 73 (1970), 238-257.

Nalven, F. B. and J. O’Brien. “Personality Patterns of Rheumatoid Arthritic Patients,” *Arthritis Rheum.,* 7 (1964), 18-28.

Nidevar, J. E. A *Factor Analytic Study of General Muscle Tension.* Ph.D. dissertation, University of California at Los Angeles, 1959. Unpublished.

Nissen, H. A. “Chronic Arthritis and Its Treatment,” *N. Engl. J. Med.,* 210 (1934), 1109-1115.

Nissen, H. A. and K. A. Spencer. “The Psychogenic Problem (Endocrinal and Metabolic) in Chronic Arthritis,” *N. Engl. J. Med*., 214 (1936), 576-581.

Noble, P. J. and M. H. Lader. “An Electromyographic Study of Depressed Patients,” *J. Psychosom. Res.,* 15 (1971), 233-239.

Oken, D. “An Experimental Study of Suppressed Anger and Blood Pressure,” *Arch. Gen. Psychiatry,* 2 (1960), 441-456.

----. “The Role of Defense in Psychological Stress,” in R. Roessler and N. S. Greenfield, eds., *Physiological Correlates of Psychological Disorder*, pp. 193-210. Madison: The University of Wisconsin Press, 1962.

Orowan, E. “Origins of Man,” *Nature,* 175 (1955), 683-684.

Paul, L. “Psychosomatic Aspects of Low Back Pain,” *Psychosom. Med.,* 12 (1950), 116-124.

----. “Mephenesin in Anxiety-tension States,” *Psychosom. Med*., 14 (1952), 378-382.

Ploski, H., E. Levita, and M. Riklan. “Impairment of Voluntary Movement in Parkinson’s Disease in Relation to Activation Level, Autonomic Malfunction, and Personality Rigidity,” *Psychosom. Med.,* 28 (1966), 70-77.

Plutchik, R. “The Role of Muscular Tension in Maladjustment,” *J. Gen. Psychol.,* 50 (1954), 45-62.

Polley, H., W. M. Swenson, and R. M. Steinhilber. "Personality Characteristics of Patients with Rheumatoid Arthritis," *Psychosomatics,* 11 (1970), 45-49.

Popert, A. J. and J. Hewitt. "Gout and Hyperuricaemia in Rural and Urban Populations," *Ann. Rheum. Dis.,* 21 (1962), 154-163.

Price, J. P., M. H. Clare, and F. H. Ewerhardt. "Studies in Low Backache with Persistent Muscle Spasm," *Arch. Phys. Med.,* 29 (1948), 703-709.

Prick, J. J. G., and K. Van de Loo. *The Psychosomatic Approach to Primary Chronic Rheumatoid Arthritis.* Philadelphia: Davis, 1964.

Rahe, R. H. and R. Arthur. "Stressful Underwater Demolition Training. Serum Urate and Cholesterol Variability," *JAMA,* 202 (1967), 1052-1054.

Rahe, R. H. et al. "Serum Uric Acid and Cholesterol Variability," *JAMA,* 206 (1968), 2875-2880.

Reich, W. *Character-Analysis,* 2nd ed. New York: Orgone Institute Press, 1949.

Reis, G. von. "Electromyographical Studies in Writer's Cramp," *Acta. Med. Scand.,* 149 (1954), 253-260.

Riklan, M. and E. Levita. *Subcortical Correlates of Human Behavior.* Baltimore: Williams & Wilkins, 1969.

Rimón, R. "A Psychosomatic Approach to Rheumatoid Arthritis: A Clinical Study of 100 Female Patients," *Acta Rheumatol. Scand., (Suppl).* 13 (1969), 1-154.

Rimón, R., A. Stenback, and E. Huhmar. "Electromyographic Findings in Depressive Patients," *J. Psychosom. Res.,* 10 (1966), 159-170.

Rinehart, R. E. "Physiologic Approach to Rheumatic Disease," in E. Jacobson, ed., *Tension in Medicine,* pp. 55-70. Springfield, Ill.: Charles C. Thomas, 1967.

Robb, J. H. and B. S. Rose. "Rheumatoid Arthritis and Maternal Deprivation: A Case Study in the Use of a Social Survey," *Br. J. Med. Psychol.,* 38 (1965), 147-159.

Robinson, C. E. G. “Emotional Factors and Rheumatoid Arthritis,” *Can. Med. Assoc. J*., 77 (1957). 344-345.

Robinson, H., R. Kirk, and R. Grye. “A Psychological Study of Rheumatoid Arthritis and Selected Controls,” *J. Chronic Dis.,* 23 (1971). 791-801.

Roessler, R., A. Alexander, and N. Greenfield. “Ego Strength and Physiological Responsivity: I,” *Arch. Gen. Psychiatry,* 8 (1963), 142-154.

Rossi, A. M. “An Evaluation of the Manifest Anxiety Scale by the Use of Electromyography,” *J. Exp. Psychol*., 58 (1959), 64-69.

Ruesch, J. and J. Finesinger. “The Relation between Electromyographic Measurements and Subjective Reports of Muscular Relaxation,” *Psychosom. Med.,* 5 (1943), 132-138.

Sabshin, M., et al. “Significance of Pre-experimental Studies in the Psychosomatic Laboratory,” *Arch. Neurol. Psychiatry,* 78 (1957). 207-219.

Sainsbury, P. “Muscle Responses: Muscle Tension and Expressive Movement,” *J. Psychosom. Res.,* 8 (1964), 179-185.

Sainsbury, P. and W. R. Costain. “The Measurement of Psychomotor Activity: Some Clinical Applications,” *J. Psychosom. Res.,* 15 (1971), 487-494.

Samra, K., M. Riklan, E. Levita et al. “Language and Speech Correlates of Anatomically Verified Lesions in Thalamic Surgery for Parkinsonism,” *J. Speech Hear. Res.,* 12 (1969), 510-540.

Sandler, S. A. “Camptocormia, or the Functional Rent Rack,” *Psychosom. Med.,* 9 (1947), 197-204.

Sands, I. “The Type of Personality Susceptible to Parkinson Disease,” *J. Mt. Sinai Hosp. (N.Y.),* 9 (1942), 792-794.

Sapirstein, M. R. “The Effect of Anxiety on Human After-Discharges,” *Psychosom. Med.,* 10 (1948), 145-155-

Sarwer-Foner, G. J. "Psychoanalytic Theories of Activity-Passivity Conflicts and the Continuum of Ego Defenses," *Arch. Neurol. Psychiatry,* 78 (1957), 413-418.

Schwab, R. S., H. Fabing, and J. Prichard. "Psychiatric Symptoms and Syndromes in Parkinson's Disease," *Am. J. Psychiatry,* 107 (1950), 901-907.

Scotch, N. A. and H. Geigeh. "The Epidemiology of Rheumatoid Arthritis," *J. Chronic Dis.,* 15 (1962), 1037-1067.

Seashore, R. H. "Work and Motor Performance," in S. Stevens, ed., *Handbook of Experimental Psychology,* pp. 1341-1362. New York: Wiley, 1951.

Shagass, C. and R. B. Malmo. "Psychodynamic Themes and Localized Muscular Tension during Psychotherapy," *Psychosom. Med.,* 16 (1954), 295-314.

Shaskan, D., H. Yarnell, and K. Alper "Physical, Psychiatric and Psychometric Studies of Post-Encephalitic Parkinsonism," *J. Nerv. Ment. Dis.,* 96 (1942), 652-662.

Shipman, W. G., H. Heath, and D. Oken. "Response Specificity Among Muscular and Autonomic Variables," *Arch. Gen. Psychiatry,* 23 (1970), 369-377.

Shipman, W. G., D. Oken, I. Balshan Goldstein et al. "Study in Psychophysiology of Muscle Tension: II. Personality Factors," *Arch. Gen. Psychiatry,* 11 (1964), 330-345.

Shipman, W. G., D. Oken, and H. Heath. "Muscle Tension and Effort at Self-Control during Anxiety," *Arch. Gen. Psychiatry,* 23 (1970), 359-368.

Shochet, B. R., E. T. Lisansky, A. F. Schubert et al. "A Medical-Psychiatric Study of Patients with Rheumatoid Arthritis," *Psychosomatics,* 10 (1969), 271-279.

Solomon, G. F. and R. Moos. "Emotions, Immunity and Disease," *Arch. Gen. Psychiatry, 11* (1964), 657-674.

----. "The Relationship of Personality to the Presence of Rheumatoid Factor in Asymptomatic Relatives of Patients with Rheumatoid Arthritis," *Psychosom. Med*., 27 (1965), 350-360.

Southworth, J. A. "Muscular Tension as a Response to Psychological Stress in Rheumatoid Arthritis and Peptic Ulcer," *Genet. Psychol. Monogr.,* 57 (1958), 337-392.

Spielberger, C. D. "Theory and Research on Anxiety," in C. D. Spielberger, ed., *Anxiety and Behavior,* pp. 3-20. New York: Academic, 1966.

Stetten, D. and J. Hearon. "Intellectual Level Measured by Army Classification Battery and Serum Uric Acid Concentration," *Science,* 129 (1959), 1737.

Stonehill, E. and A. H. Crisp. "Problems in the Measurement of Sleep with Particular Reference to the Development of a Motility Bed," *J. Psychosom. Res.,* 15 (1970). 495-499.

Swaim, L. J. *Arthritis Medicine and the Spiritual Laws.* Philadelphia: Chilton, 1962.

Talbott, J. H. *Gout,* 2nd ed. New York: Grune & Stratton, 1964.

Talland, G. A. "Cognitive Function in Parkinson's Disease," *J. Nerv. Ment. Dis.,* 135 (1962), 196-205.

Thomas, G. W. "Psychic Factors in Rheumatoid Arthritis," *Am. J. Psychiatry,* 93 (1936), 693-710.

Tyndel, M. "The Other Side of A One-Sided Approach," *Am. J. Psychiatry,* 127 (1971), 1101.

Voas, R. B. "Generalization and Consistency of Muscle Tension Levels." Ph.D. dissertation, University of California at Los Angeles, 1952. Unpublished.

Weiss, E. "Psychogenic Rheumatism," *Ann. Intern. Med.,* 26 (1947), 890-900.

Wenger, M. A. "Some Relationships between Muscular Processes and Personality and Their Factorial Analysis," *Child Dev.,* 9 (1938), 261-276.

----. "An Attempt to Appraise Individual Differences in Level of Muscular Tension," J. Exp. Psychol., 32 (1943), 213-225.

Whatmore, G. B. and R. M. Ellis, Jr. "Some Motor Aspects of Schizophrenia: An EMG Study," *Am. J. Psychiatry,* 114 (1958), 882-889.

----. “Some Neurophysiologic Aspects of Depressed States: An Electromyographic Study,” *Arch. Gen. Psychiatry*, 1 (1959), 70-80.

----. “Further Neurophysiologic Aspects of Depressed States,” *Arch. Gen. Psychiatry*, 6 (1962), 243-253.

Wiener, D. N. “Personality Characteristics of Selected Disability Groups,” *Genet. Psychol. Monogr.,* 45 (1952), 175-255.

Williams, J. G. L. “Use of a Resonance Technique to Measure Muscle Activity in Neurotic and Schizophrenic Patients,” *Psychosom. Med.,* 26 (1964), 20-28.

Williams, R. and A. Krasnoff. “Body Image and Physiological Patterns in Patients with Peptic Ulcer and Rheumatoid Arthritis,” *Psychosom. Med.,* 26 (1964), 701-709.

Williams, T. A., J. Schachter, and R. Rowe. “Spontaneous Autonomic Activity, Anxiety, and ‘Hyperkinetic Impulsivity’,” *Psychosom. Med.,* 27 (1965), 9-18.

Wolff, B. B. “Experimental Pain Responses as Predictors of Outcome of Surgical Rehabilitation in Rheumatoid Arthritis,” (Abstract), *Arthritis Rheum.,* 11 (1968), 519-520.

----. “Current Psychosocial Concepts in Rheumatoid Arthritis,” Bull. Rheum. Dis., 22 (1972), 656-660.

Wolff, B. B. and R. Farr. “Personality Characteristics in Rheumatoid Arthritis,” (Abstract), *Arthritis Rheum.,* 7 (1964), 354.

Wolpe, J. *Psychotherapy by Reciprocal Inhibition.* Stanford: Stanford University Press, 1958.