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Megavitamin Arthritis Treatment, Part 2

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160-203 minutes

Joint Dysfunction, Part 2
Home

CHAPTER 2

To read Chapter 3, click this link: http://www.doctoryourself.com/kaufman8.html

To return to Chapter 1: http://www.doctoryourself.com/kaufman6.html

THE COMMON FORM OF JOINT DYSFUNCTION
by William Kaufman, M.D., Ph.D. (1949)

Edited by Andrew W. Saul

(Dr. Kaufman now discusses physical and psychological stresses, allergy, posture, obesity and other factors that may interact or interfere with niacinamide megavitamin therapy for arthritis. This chapter's three original photographs are not provided here, but may be seen in the original text, available through this website. For ordering information, you may either click here *or scroll to the very bottom of this webpage.)*

References cited in this chapter are posted at http://www.doctoryourself.com/kaufman11.html

Four Complicating Syndromes Frequently Coexisting with Joint Dysfunction

It might appear to the reader that the niacinamide treatment of a patient with joint dysfunction is a more or less mechanical and uninteresting procedure. However, in practice, the treatment of a patient with joint dysfunction is never a mechanical or dull routine, since therapy of joint dysfunction and commonly occurring complicating syndromes must always be adapted to the special needs of the individual patient. For the most part, the treatment of a patient with joint dysfunction is a constantly interesting and instructive discipline both for the patient and physician.

Commonly occurring complicating syndromes coexisting with joint dysfunction must often be corrected if the patient is to be able and willing to take niacinamide therapy as prescribed, and if he is to feel well ultimately. Even though joint dysfunction improves to the level of 96-100 (no joint dysfunction) in response to adequate niacinamide therapy, the patient may have continuing articular and non-articular symptoms, of one or more of these complicating syndromes, and he may erroneously conclude that the niacinamide treatment of his joint dysfunction has failed. On the other hand, whether or not a patient is taking niacinamide treatment, when these complicating syndromes are corrected, he may have an improved sense of well-being and freedom from articular and non-articular symptoms, but it does not follow necessarily that his joint dysfunction is improving, since serial re-measurements of his Joint Range Index may indicate that his joint dysfunction may be unimproved or worsened.

In the treatment of a patient with joint dysfunction who has one or more of four complicating syndromes frequently coexisting with joint dysfunction, the physician must correctly identify the basis of the patient's articular and non-articular symptoms, and must institute concurrently the appropriate specific therapy required for the successful

management of joint dysfunction and any of these four syndromes which the patient may have:

(a) the delayed post-traumatic articular syndrome (see page 79);
(b) the chronic allergic syndromes (page 96);
(c) the sodium retention syndrome (page 114);
(d) the syndrome of psychogenically induced, sustained hypertonia of somatic muscle (page 115).

The articular symptoms of any one or any combination of these four syndromes may be present in a patient without joint dysfunction, or may be absent in a patient with joint dysfunction (with or without clinically obvious arthritis); and may occur in a patient with joint dysfunction before niacinamide therapy is instituted, during the course of adequate niacinamide therapy, when adequate niacinamide therapy is replaced by inadequate niacinamide therapy or upon premature cessation of niacinamide therapy. The articular and non-articular symptoms of bodily discomfort of these four syndromes may vary in intensity, duration and extensiveness. With each of these syndromes a patient may have a steady state of discomfort which persists until the syndrome is successfully treated.

The four complicating syndromes frequently coexisting with joint dysfunction will be described as if each were an independent clinical entity. Often, the successful management of any one of these syndromes will not materially influence the clinical course of the other untreated coexisting syndromes. At times, however, these complicating syndromes may be interrelated, in the sense that when one of the untreated complicating syndromes becomes more severe, the other coexisting untreated complicating syndromes also become more severe; and in the sense that the successful treatment of one of the complicating syndromes may simultaneously ameliorate or lessen the intensity of symptoms of the other untreated coexisting complicating syndromes. Clinically, it may be very easy, or it may be extremely difficult, to ascertain the etiologic basis of the patient's articular and non-articular symptoms. The most helpful clues to the etiology of the patient's symptoms are obtained from careful clinical study, including a detailed history of the onset and development of symptoms, re-examination of the patient, an analysis of the food-symptom diary kept by the patient (see page 103), and an evaluation of the patient's response to a trial of therapy directed toward the amelioration of the symptoms of a given complicating syndrome.

Although from the physician's point of view, the patient's symptoms are subjective phenomena, to the patient his symptoms are real and have objective existence. By giving verbal expression to his symptoms, the patient is exteriorizing the fact that he does not feel well, and implying that if his symptoms could be made to disappear, he would feel well.

The physician must give careful consideration to the possible meaning of all the patient's symptoms, whether or not they seem to be trivial, atypical or bizarre at the time of their recital. The physician should regard the patient's symptoms as direct or indirect clues to the nature of the patient's ill health, even if the clinical meaning of these symptoms continues to be inapparent. Once the etiology of the patient's symptoms is recognized by the physician, it often becomes possible to institute appropriate treatment which, in time, ameliorates these symptoms. While the etiologic basis of some symptoms may be readily perceived by the physician, the clinical significance of other symptoms may remain obscure for a long time or may never be ascertained. Even some symptoms which at first hearing appear fantastic to the physician may prove, in time, to have a definite clinical basis which can be identified. When the nature of the patient's disorder becomes manifest, it is often found that most patients with puzzling symptoms were entirely accurate and honest in their reporting of symptoms. It is the rare patient who deliberately distorts facts and invents complaints and illnesses, and even such a patient by so doing gives valuable clues to the nature of his illness.

In evaluating the clinical significance of articular and non-articular symptoms, it is necessary to remember that the patient's prevailing emotional state influences the nature of his complaints (29) (182) (242). A patient who is mildly depressed may

complain at great length about his various symptoms, and may express considerable doubt that he will ever get well. On the other hand, a patient who is euphoric will complain little or not at all of articular and non-articular symptoms, and his general attitude toward all of his life situations will be optimistic. A patient who has feelings of anxiety, guilt, hostility or frustration may find substitute satisfaction in complaining bitterly about articular and non-articular symptoms. A patient whose attention is fixed on his symptoms will have many complaints excepting when his attention is distracted by more interesting matters. A patient with a rigid conception of his own perfection seldom will complain of symptoms. Occasionally, a patient unconsciously attempts to gain the approval of the physician by exaggerating his favorable response to therapy. On the other hand, a patient may use his complaints about poor health to "punish" the physician (authoritarian figure) by insisting with evident satisfaction that his health has been unimproved or worsened by treatment, when it is obvious from physical examination and from the remainder of the patient's story that he has in fact improved physically. At times, when a patient has secondary gains from his illness, he seems impelled to complain about his symptoms, and even when he has improved as a result of treatment and has fewer symptoms, he continues to complain more and more about less and less.

DELAYED POST-TRAUMATIC ARTICULAR SYNDROME
(In this section, there is excluded from discussion such severe accidental injuries as lacerations of the supporting structures of joints, bone fractures involving joint structures, torn articular cartilages causing internal derangement of joints, and penetrating joint wounds.)

Without knowledge of the clinical patterns of the delayed post-traumatic articular syndrome, the cause of many articular symptoms and signs often remains obscure. With knowledge of such patterns, and with knowledge of the patient's physical activities, occupation, and emotional tensions, the physician can often identify the basis for the patient's troublesome articular symptoms and signs, and can advise the patient how to modify his way of living so that in the future he will be less likely to experience such symptoms and signs.

The delayed post-traumatic articular syndrome is the consequence of certain types of mechanical joint injury:

(a) articular trauma which is likely to occur in the course of more or less ordinary physical activity;

(b) alterations in the alignment of joints due to certain acquired habits of posture, or indirectly due to niacinamide-induced improvement in joint mobility;

(c) psychogenically induced, sustained hypertonia of somatic muscle.

In general, the severity of the patient's delayed post-traumatic articular symptoms seems to depend on the following factors: the clinical grade of severity of his joint dysfunction; the severity, repetitiveness and duration of the inciting mechanical joint injury; the patient's prevailing moods; and his attitudes toward his symptoms and life situations.

Mechanical joint injury may be well tolerated by persons with the milder grades of joint dysfunction, who will have either no clinically discernible articular sequelae or will develop relatively mild symptoms and signs of the delayed post-traumatic articular syndrome for relatively short periods of time; but mechanical joint injury usually is poorly tolerated by persons with the more severe clinical grades of joint dysfunction, who tend to develop severe symptoms and signs of the delayed post-traumatic articular syndrome which last for relatively long periods of time (97). In general, immediate and delayed post-traumatic articular symptoms and signs tend to be more severe in untreated persons with joint dysfunction than in persons with joint dysfunction who are receiving adequate amounts of niacinamide. A patient with joint dysfunction who is receiving inadequate niacinamide therapy is more likely to suffer from the delayed post-traumatic articular syndrome than if he were receiving adequate niacinamide therapy. If his

niacinamide intake is increased from inadequate to adequate levels, even though mechanical joint injury continues at the same level as previously, the niacinamide-induced reparative process will often preponderate over the trauma-induced deteriorative process, and the delayed articular post-traumatic syndrome will be ameliorated.

The immediate effects of a single episode of extremely severe joint injury are well understood because of the close temporal relationship between the articular injury and the ensuing articular symptoms of discomfort, pain and disability, which may be associated with one or more of the following physical signs in the mechanically injured joint region: tenderness to palpation, swelling, heat, redness, congestion of the superficial circumarticular veins, spasm of somatic muscles operating the injured joint, and painful or painless limitation of active and passive articular movement. The delayed effects of such severe mechanical joint injury may include a continuance of articular discomfort, pain and disability lasting for months or years, and clinically well-defined arthritic changes in the injured joints (33) (121) (19) (131).

The immediate effects of a single episode of a less severe grade of mechanical joint injury are also well understood, but the delayed effects of such an injury to the joints have not been given the clinical attention they deserve. Because there is often an asymptomatic period of two to four days between the subsidence of the immediate post-traumatic articular symptoms and the appearance of the delayed post-traumatic articular syndrome, the physician and patient may be unable to perceive the causal relationship between the inciting mechanical joint injury and the delayed post-traumatic articular symptoms. When the delayed post-traumatic symptoms of joint discomfort, pain and disability occur three or four days after the inciting joint injury, there may be one or more of the following objective findings in the injured articular regions: tenderness to palpation, swelling, heat, redness, congestion of the superficial circumarticular veins, spasm of the somatic muscles operating the injured joint, and painful or painless limitation of active and passive articular movement. These delayed post-traumatic articular symptoms and signs may be more severe and more persistent than those occurring immediately after joint injury, and gradually decrease in severity, usually disappearing by the tenth to fourteenth day following the inciting injury to the joint. Occasionally, the delayed post-traumatic articular syndrome may persist for a month or more after a single joint injury, particularly when the patient's joint dysfunction is extremely severe, or when the inciting trauma is unusually great. At times, there may be no articular symptoms and signs immediately following mechanical joint injury, or such immediate articular symptoms as may appear immediately after the injury may seem so insignificant to the patient that he disregards them. Sometimes, the only sign of the delayed post-traumatic articular syndrome may be increased painless limitation in the ranges of movement of the injured joint. Even relatively slight injury, when sufficiently repetitive, may lead, in time, to a steady state of articular discomfort, pain and disability, and to the appearance of clinically obvious arthritic deformities in the injured joint region (96).

Sometimes, the cause of delayed post-traumatic articular symptoms may be identified only with difficulty after a prolonged period of clinical study. When a patient with joint dysfunction suddenly experiences a single isolated episode of joint pain and disability, or gradually develops a persistent state of articular discomfort (with or without periodic exacerbations) or merely an asymptomatic lowering of the ranges of joint motion, careful clinical study may disclose the fact that in the performance of a particular physical act either once or repetitively, the patient inadvertently or unknowingly injured the affected joints, or may disclose the fact that the patient has developed psychogenically induced, sustained hypertonia of somatic muscle of sufficient severity to injure his joints. Joints used statically or dynamically in the performance of everyday activities may incur mechanical trauma sufficiently severe to cause a single episode of articular discomfort, pain and disability, or may incur mechanical trauma sufficiently severe and repetitive to cause a steady state of articular discomfort, pain and disability.

When a patient has joint dysfunction of a high clinical grade of severity, his articular structures are particularly vulnerable to lesser grades of joint trauma, which may give

rise to the more severe and persistent symptoms and signs of the delayed post-traumatic articular syndrome. When a patient is recovering satisfactorily from joint dysfunction in response to continuously adequate niacinamide therapy, and a specific joint is subjected to a single episode of moderate injury, usually there is temporarily a delayed post-traumatic decrease in the range of movement of this joint -with or without accompanying symptoms of the delayed post-traumatic articular syndrome - although his uninjured joints continue to improve at a satisfactory rate. If the articular injury is more or less continuous, the range of movement of the injured joint decreases, and, in time, tends to stabilize for as long as the niacinamide-induced reparative process balances the trauma-induced deteriorative process in the injured joint. At this time, an increase in the patient's niacinamide intake does not materially improve the range of movement of the continuously injured joint, except in some instances where previous levels of niacinamide treatment have been inadequate. However, a decrease in niacinamide intake causes the range of movement of the continuously injured joint to decrease at a more rapid rate than if adequate amounts of niacinamide were taken continuously.

The Joint Range Index may or may not be significantly depressed by the post-traumatically decreased range of movement of a single joint. It is, therefore, necessary to analyze the component joint ranges which are measured for the computation of the Joint Range Index in order to observe which joints show post-traumatically decreased ranges of joint movement and which joints simultaneously have made satisfactory improvement in the ranges of joint movement for the period of observation during which the patient was ingesting continuously adequate amounts of niacinamide.

It is often possible to identify the type of behavior which caused mechanical injury of certain joints from an analysis of the distribution of joints with decreased ranges of movement and those with increased ranges of movement, and from knowledge of the patient and his physical activities and hobbies at various seasons of the year, and of his emotional tensions. For example, when the fingers of the right hand, right wrist and right shoulder
show decreased movement, and the patient has recently returned from a train trip, one can establish that the most likely cause of the decreased ranges of movement was the carrying of a suitcase. When mechanical articular injury is sufficiently generalized, there is a delayed post-traumatic decrease in the ranges of movement of the injured joints and in the Joint Range Index even though the patient with joint dysfunction is ingesting continuously adequate amounts of niacinamide; however, with cessation of joint injury there is usually a satisfactory rise in the Joint Range Index in response to adequate niacinamide therapy.

Certain physical activities have been identified as causes of the delayed post-traumatic articular syndrome in some patients at various times during this study, and include: sawing, planing, hammering, house-painting, weeding, spading, hoeing, spraying, hedge-clipping, lawn-mowing, bowling, sailing, rowing, paddling a canoe, fly-fishing, driving a car, knitting, crocheting, tatting, wringing of clothes, house-cleaning, cleaning, scrubbing floors, waxing floors. In some persons the repetitive performance of a physically awkward act may cause joint injury; e.g., the frequent daily use of a desk telephone with a short cord, which requires the user to twist his body into an awkward position each time he uses the telephone. In some patients, holding the joints in a fixed position and carrying moderate weights for relatively short or long periods of time may give rise to a delayed post-traumatic cycle of joint discomfort and disability; e.g., maintaining one knee and ankle fixed in an awkward position by sitting on the medial aspect of the ipsilateral heel, or sitting in a chair with the dorsum of the ipsilateral foot twisted behind one leg of the chair; hanging onto an overhead strap in a subway or bus; holding a knitting bag, handbag, shopping bag, brief-case, suitcase, or even holding a strong dog in leash. Similarly, certain jerky movements requiring the sudden exertion of extra muscular force will also give rise to a post-traumatic cycle of joint discomfort and disability; e.g., opening a window or drawer that "sticks," or loosening a stubborn jar cover with a strong steady twisting movement, or opening and closing a "tight" water faucet. Joint trauma may occur during the night when the patient maintains awkward sleeping postures for relatively long periods of time, particularly if he simultaneously has

during sleep psychogenically induced, sustained hypertonia of somatic muscle. Certain recently acquired or old methods of walking which the patient habitually uses will cause injury to the knee and hip joints and will cause a steady state of symptoms of articular discomfort, pain and disability, and signs of impaired mobility of hip and knee joints.

Joint trauma may occur also when a patient with joint dysfunction (with or without clinicallv obvious arthritic deformities) has mental tensions which are exteriorized through psychogenically induced, sustained hypertonia of somatic muscle. Although such a patient may erroneously believe that he is completely relaxed, the coapting pressures exerted continuously against articular surfaces, and the accompanying tensions on periarticular structures often cause continuous joint trauma for as long as this sustained somatic muscle hypertonia persists. When psychogenically induced, sustained hypertonia of somatic muscle is present and the patient uses his joints in everyday activities, there is joint trauma in excess of what would have occurred in the performance of these activities in the absence of sustained hypertonia of somatic muscle. Psychogenically induced, sustained hypertonia of somatic muscle in persons with the more severe grades of joint dysfunction may cause articular swelling, redness, increased congestion of the superficial circumarticular veins, increased heat, spasm of the somatic muscles operating the joints, stiffness, and limitation in the ranges of active and passive joint movement. In time, repetitive joint trauma from this source will favor the appearance of clinically obvious arthritic deformities. Ordinarily, the patient is unaware of his mental tensions and his psychogenically induced, sustained hypertonia of somatic muscle, although he is very aware of his symptoms due to the delayed post-traumatic articular syndrome.

Many persons with joint dysfunction (with or without clinical or radiographic evidence of arthritic changes in joints) may be unaware of any articular discomfort or disorder until joint trauma gives rise to the delayed post-traumatic articular syndrome. The anxiety and mental tension developed by such patients as a result of this articular discomfort, pain and disability (particularly when a steady state of articular discomfort is reached) often create secondary psychogenically induced, sustained hypertonia of somatic muscle which is sufficiently severe to perpetuate joint injury and its sequelae.

TREATMENT OF THE DELAYED POST-TRAUMATIC ARTICULAR SYNDROME

Treatment of the delayed post-traumatic articular syndrome should be directed toward preventing the joint traumata which produce this syndrome and toward giving the patient relief from whatever delayed post-traumatic articular symptoms he may have. Since joint injury may be caused by ordinary or unusual, essential or non-essential daily activities, it is not always possible to prevent articular trauma, even when the physical act producing joint injury is known. However, once the causation of mechanical joint injury is recognized, the patient should be advised how to keep joint injuries to a minimum in the performance of his essential everyday physical activities. A patient who understands the temporal and causal relationship between the mechanical joint injuries of everyday activities and the symptoms of the delayed post-traumatic articular syndrome is likely to modify his activities so that mechanical injury to his joints will be minimal and, when possible, to avoid those unessential physical activities which may actuate the delayed post-traumatic articular syndrome.

Many patients erroneously believe that "exercise loosens the joints." It is often necessary to demonstrate to a patient that after exercise his Joint Range Index and the ranges of movement of his exercised joints are depressed, sometimes for days or weeks. In patients who have the lesser clinical grades of joint dysfunction, such delayed post-traumatic depression of joint ranges may not be sufficiently severe or prolonged to warrant the interdiction by the physician of all unessential physical exercise. However, in patients who have the more severe clinical grades of joint dysfunction, such delayed post-traumatic depression of the joint ranges may be sufficiently marked and prolonged to impede satisfactory joint recovery in response to niacinamide therapy. For each patient, where possible, physical exercise should be adjusted so that the resultant joint injury will not materially impede satisfactory niacinamide-induced recovery from joint dysfunction.

Although physically strenuous exercise may give some patients with the more severe grades of joint dysfunction temporary benefit through transient release of psychogenically induced, sustained hypertonia of somatic muscle, the joints are not benefited by such exercise. It may be desirable to permit a patient with unresolved mental tensions to continue to enjoy his strenuous physical exercise, since the advantages of obtaining transitory relief from sustained hypertonia of somatic muscle may outweigh the disadvantages of actuating the post-traumatic articular syndrome. However, in time, with satisfactory psychotherapeutic resolution of his emotional tensions, the patient usually is relieved of his psychogenically induced, sustained hypertonia of somatic muscle, and consequently does not have the urgent need for seeking emotional release through excessive physical activity.

The more niacinamide-induced recovery a patient has had from his initial clinical grade of joint dysfunction, the better he will be able to tolerate the articular trauma of his everyday activities. The substitution of inadequate for adequate niacinamide therapy, or the premature cessation of adequate niacinamide therapy, tends to make the delayed post-traumatic syndrome more severe. Continuously adequate niacinamide therapy helps to minimize the symptoms and signs of the delayed post-traumatic articular syndrome but does not prevent their occurrence.

The use of plain or enteric coated aspirin (0.3 to 0.6 g per dose) or enteric coated sodium salicylate (0.6 g per dose) distributed as needed during the day - in a person having no intolerance for these drugs - often gives the patient relief from his localized or generalized post-traumatic articular symptoms. Rarely, for the relief of articular pain, it is necessary to give additionally codeine (0.030 to 0.060 g per dose) or demerol (0.100 to 0.150 g per dose), as required. Procaine hydrochloride infiltration of an injured joint region has not been used (33) (211), nor were intravenous procaine hydrochloride injections used (63).

Relative rest of the injured joints tends to hasten recovery from the delayed post-traumatic articular syndrome, provided that there is daily movement of the joint, without weight-bearing, through the fullest possible ranges of active and passive movement. When the delayed posttraumatic articular syndrome occurs in a given joint region, it is often helpful to apply massive hot, wet, Epsom salt dressings (for 30 minutes 3 or 4 times daily) to a large region, including and surrounding the injured joint. Moist heat seems to be more efficacious than dry heat, although it is often more convenient to use dry heat (heating pad, or heat from an electric incandescent bulb). With the use of moist or dry heat special care must be taken not to burn the patient. Certain types of massage administered to injured articular regions may be helpful in giving some patients subjective relief from localized post-traumatic articular symptoms. A patient who injures his joints and develops generalized delayed post-traumatic articular symptoms may have temporary relief from these symptoms by soaking in a tepid bath for 20 or 30 minutes. In selected instances, a suitable type of body massage following the bath may give additional benefit.

MISCELLANEOUS TYPES OF MECHANICAL JOINT INJURY AND THEIR TREATMENT

Posture. Certain types of posture in sitting, standing; walking and working cause mechanical joint injury, regardless of the patient's clinical grade of joint dysfunction, whether or not he is receiving adequate niacinamide treatment. Often there is a correlation between the patient's posture and his symptoms of bodily fatigue and joint discomfort, pain and disability, and therefore the physician must constantly analyze the patient's static and dynamic postures and make appropriate suggestions for the correction of faulty posture. A few commonly occurring types of static and dynamic postural abnormalities are described below, together with suggestions for their treatment. No general discussion of posture is included, since a number of excellent descriptions of what constitutes good posture are available in the literature (33) (84) (73).

It was observed that many patients who were making satisfactory recovery from severe or extremely severe joint dysfunction in response to adequate niacinamide therapy

(even those who had reached the level of slight joint dysfunction or no joint dysfunction) had continuance or worsening of symptoms referable to hip and knee joints and to the muscles of their lower extremities, and that objectively, recovery of movement in hip and knee joints lagged behind recovery of movement in other moveable joints. When it was recognized that these patients were continuing to use habitually the abnormal posture described below, even though therapeutically increased ranges of joint movement permitted more efficient walking posture, appropriate suggestions were made for the correction of improper postures. When the patient taking adequate niacinamide therapy adopted these suggested changes in walking posture, he experienced some immediate relief from his symptoms and, in time, when the recommended posture became habitual, he usually became entirely free from symptoms referable to his hip and knee joints and to his lower extremity muscles, and the rate of recovery in the ranges of hip and knee joint movement was accelerated. Now that patients are routinely advised, as described below, to modify improper walking posture at the outset of niacinamide therapy, the continuance or accentuation of this pattern of articular and muscular symptoms of the lower extremities is seldom seen, and recovery of movement in hip and knee joints parallels that of other joints in response to adequate niacinamide therapy.

This commonly occurring postural abnormality of standing and walking results chiefly from sustained hypertonia of the quadriceps muscles, associated with various degrees of cocontraction (sustained hypertonia) of the flexor and adductor muscles of the thighs. At first this postural abnormality may occur only as an unconsciously adopted accompaniment of unresolved emotional problems, which initiate psychogenically induced, sustained hypertonia of somatic muscle. In time, such postures and the sustained hypertonia of somatic muscle may become habitual, whether or not the patient continues to have unresolved emotional problems. In the standing position, the patient's muscles contract more forcefully than necessary to maintain his stance efficiently. In addition, the patient usually has increased pelvic tilt and increased lumbar lordosis, and holds his head in a forward position which accentuates the thoracicocervical curve. Any dorsal kyphosis the patient may have seems to become more prominent as a result of this abnormal posture. Often in this posture the patient's abdominal muscles become so lax that his abdomen becomes pendulous (6). In walking, the person with sustained muscular hypertonia tends to maintain the poor standing posture described above. In forward progression, he tends to inhibit the natural swinging movement of the arms. With each consecutive step, the ipsilateral trunk-thigh muscles elevate the thigh sufficiently to permit pendulum-like swinging of the entire ipsilateral lower extremity as a more or less rigid unit, with little or no associated knee movement. Upon simultaneous palpation of the anterior and posterior thigh muscles of the patient as he walks, it is possible for the physician to detect a high degree of cocontraction of antagonists and protagonists of the hip and knee joint movement without palpable relaxation of these thigh muscles during walking. It is tiring for the patient to stand and walk in the manner described above. He also experiences a sense of resistance to walking which he describes as dragginess, heaviness, weakness, unsteadiness and stiffness of the lower extremities. He may have pain, discomfort and stiffness in the muscles of his thighs, back and neck; there are often associated symptoms of discomfort, pain and disability in the hip and knee joints. In addition, the patient may have pain and discomfort in the joints of his lumbosacral region, in his upper thoracic spine, and in the cervical spine. He may have noticed that over a period of time he has become "round-shouldered," that it is hard for him to straighten up, and that his "stomach" has become more prominent. When such a posture is habitual for many years, the patient with joint dysfunction suffers from the steady state of the post-traumatic articular syndrome, and is likely to develop arthritic changes in the various joint regions subjected to excessive mechanical trauma, resulting in part from improper alignment of joints, and in part from continuously sustained hypertonia of somatic muscle.

Such a patient is shown how to modify 'his gait so that he consciously lifts his feet, raising and flexing each knee alternately with each successive step, instead of walking stiff-kneed. He may notice at once that walking in this way is relatively effortless and comfortable as compared with his usual gait, which caused his lower extremities to feel

draggy, heavy, weak, unsteady and stiff, and his thigh muscles to feel painful and uncomfortable. With this correction in gait, simultaneous palpation of anterior and posterior thigh muscles will indicate that there is alternately well-coordinated contraction and relaxation of the opposing thigh muscles. When, in addition, the patient learns to hold himself as tall as possible in standing, walking and sitting, he may lose his pelvic tilt, lumbar lordosis and anterior neck flexion. The patient must practice the therapeutically suggested alterations in posture so that ultimately he habitually uses those static and dynamic postures which cause the least injury to his joints, and as a result he will no longer be troubled with symptoms from this type of improper p05ture. When a patient has marked limitation in ranges of movement of hip and knee joints before niacinamide therapy is instituted, he is unable to correct his gait in the manner suggested. When niacinamide-induced recovery permits sufficient increase in hip and knee movement, this correction of gait is possible. Occasionally, irreversible arthritic joint changes are present which make this improvement in posture mechanically impossible.

Sacro-iliac Joint Strains. A patient with a history of recurrent sacro-iliac strains is given certain suggestions concerning posture which are often helpful in preventing recurrences of such strains: he should avoid twisting his trunk in the performance of any physical act while standing with his trunk bent at an angle of 35 to 55 degrees with his thighs, since this maneuver is frequently the cause of sacro-iliac strain. He should not "cross his knees" when sitting. He should not stand asymmetrically with most of his body weight resting on one foot. He should sleep on a non-sagging bed.

High Heels. Women who wear high-heeled shoes are likely to have postural back strains caused by compensatory lumbar lordosis, pelvic tilt, flexion of the neck and slight bending of the knees - all of which are necessary to maintain balance in the erect posture when high heels are worn. Some women wearing high-heeled shoes may have a steady state of back fatigue, discomfort and pain from such postural strains, while others may have these symptoms only when they are on their feet a great deal, or when they carry unaccustomed weights. Symptoms from postural strain are accentuated by the alternate wearing of high-heeled and low-heeled shoes. Women are advised to wear slippers and shoes having heels of uniform height, preferably low or medium heels.

Lifts. Often patients who were obliged to wear lifts continuously on their shoes to alleviate hip and knee discomfort prior to adequate niacinamide therapy found during niacinamide-induced recovery of joint mobility that discomfort of hip and knee joints increased in severity. However, when the lifts were removed, this discomfort disappeared.

Obesity. The excess weight of the moderately overweight patient increases mechanical injury of the weight-bearing joints (hips, knees, ankles, small joints of the feet). The excess weight of the markedly overweight patient causes more severe mechanical injury of these joints and, in addition, during standing and walking the patient has postural strain from balancing his heavy, often pendulous abdomen, and develops associated articular symptoms of fatigue, discomfort and pain in various portions of his back. Adequate weight reduction is part of the treatment of such patients with joint dysfunction, and a prerequisite for this is often the successful resolution of the patient's emotional problems (23) (138).

Painful Feet. A patient with painful feet may adopt awkward bodily postures which subject many joints of the body to excessive mechanical injury.

It is not uncommon to find that considerable foot pain is caused by the wearing of shoes which have unevenly worn heels or projecting irregularities of the insoles. A patient who habitually dorsifiexes his toes while wearing shoes, often develops considerable discomfort of the feet and legs. When such a patient is made aware that he habitually dorsiflexes his toes, he can eventually break himself of this habit, and he will be free from discomfort from this source.

During the course of adequate niacinamide therapy, a patient with joint tilt, dysfunction may develop considerable pain and discomfort in the ball of the foot and in one or more of the four small toes of the feet even though he has continued to wear

footgear (shoes, slippers, socks or stockings) which was comfortable previously. When one foot is significantly longer than the other, the foot pain experienced during niacinamide therapy may be more severe in the longer foot, or present only in the longer foot.

As part of the progressive retrograde changes of untreated joint dysfunction, over a period of years many patients develop in the four small toes mild, moderate or marked deformities, consisting of partial flexion of the interphalangeal joints, and partial extension of the corresponding metatarsophalangeal joints; thus, one or more of the four small toes of each foot are "curled" to various degrees. Where there is a significant disparity in the length of the two feet, the "curling" of the toes of the longer foot is the more pronounced than that of the shorter foot. Such "curled" toe deformities are much more common in women than in men, presumably because the higher heels and narrower toe caps of women's shoes are additional factors which mechanically favor the formation of "curled" toes. With niacinamide-induced articular improvement, there is a gradual "uncurling" of the deformed toes, with virtual lengthening of the feet which is particularly prominent on weight-bearing. Consequently, footgear of a size entirely comfortable prior to niacinamide-induced joint reconstitution becomes painfully short, with resultant injury to the feet. When such an injury has taken place, the patient often complains of pain, burning, throbbing and swelling in the ball of the foot. These symptoms usually are most severe on the plantar surfaces of the second, third and fourth metatarsophalangeal joints. Examination reveals redness, swelling, heat and exquisite tenderness to digital pressure on the ball of the foot. There may be swelling, pain and redness of the interphalangeal joints of the four small toes. The skin of the dorsolateral surfaces of the fourth and fifth toes near the interphalangeal joints may be irritated, swollen, painful and reddened from rubbing against the lining of the shoes, and at times there may be, in addition, secondary infection. Callusing of the skin of the ball of the foot, and corns in the rubbed areas on the toes are commonly found.

The patient is advised to stay off his feet for several days, to immerse his lower extremities in hot Epsom salt solution up to the mid-calf region for 30 minutes three or four times a day, and to obtain footgear correctly fitted to his "new" foot size, measured to his foot size when he is in a standing, weight-bearing. position. At any time the wearing of footgear that is too small will cause a recurrence of this type of foot discomfort.

SOME EXAMPLES OF MECHANICAL JOINT INJURY

CASE U, No.178, female, age 43, housewife, married.

This patient, who had slight dysfunction (Joint Range Index 88.1) without arthritis, complained when first seen that she had had daily, for a number of years, pain, swelling and stiffness in the joints of her hands, more marked at all times in the right hand than in the left hand. She was unable to attribute her discomfort and disability to any specific act which might have injured her joints. Her articular symptoms were always much worse on Wednesdays and Thursdays, and by the following Monday were noticeably better, although she was never completely free from joint discomfort.

Upon questioning, it was found that for many years she ironed every Monday for about five hours continuously. When she was asked to demonstrate her method of ironing, it was observed that she exerted strong and persistent pressure in gripping the handle of the iron tightly with the fingers of her right hand, and exerted a strong downward pressure with her right wrist as she moved the iron back and forth. The left hand grasped the edge of the garment tightly between thumb and forefinger as she stretched the cloth in the course of her ironing. She stated that three or four days after ironing, her chief discomfort in the right hand was in the wrist and in all of the joints of the thumb and fingers. In her left hand, pain was limited to the wrist and the joints of the thumb and forefinger.

Since there seemed to be a causal relationship between the method of ironing and the patient's joint symptoms, she was advised to distribute her ironing through the week so that she would do no more than one hour of ironing on any one day. She was also

instructed to use no more than the minimal muscular force necessary to perform her ironing.

After a month of such a program, she was free from articular pain, swelling and stiffness for the first time in many years. For three years she has had no difficulty referable to the joints of her hands and wrists, even though she continues to do the same amount of housework and ironing.

This patient had a post-traumatic pattern of persistent articular pain and disability resulting from repetitive episodes of mild joint trauma occurring every 7 days, with cyclic exacerbations of articular difficulties for 3 or 4 days after joint trauma was sustained.

CASE V, No.452, female, age 61, invalid, married.

When first seen, this patient had extremely severe joint dysfunction (Joint Range Index 52.2) and severe rheumatoid arthritis, as well as a post-traumatic pattern of immediate and delayed articular pain, discomfort and disability resulting from a single episode of mild joint trauma.

She had performed what was for her the unusually difficult task of addressing 20 envelopes for Christmas cards, holding the pen in her right hand. Ordinarily, her husband would have performed this service for her, but he was away on a business trip, and she did not wish to ask anyone else to relieve her of this obligation. When she completed her writing, she experienced uncomfortable cramps, fatigue and unusual stiffness in her right hand, which lasted for about 30 minutes. She was free from further unusual discomfort in her right hand until four days later, when she suddenly experienced severe, persistent articular pain and increased stiffness in the joints of her right thumb, first and second fingers, and, to a slightly lesser extent, in the joints of the fourth and fifth fingers. Her pain, articular swelling and stiffness persisted at a severe level for four days, with gradual subsidence of the delayed post-traumatic articular syndrome over a period of one month, which corresponded to her first month of niacinamide therapy. At the time of her second office visit, there was no evidence of the delayed post-traumatic articular syndrome.

CASE W. This 65-year-old woman accidentally cut the digitorum profundus tendon of her right forefinger 16 years before the photographs of Figure 35 were taken. At the time of the initial examination her Joint Range Index was 71.5, indicating moderate joint dysfunction.

Since the accident, the right forefinger could be flexed to a limited extent, and was moved during the course of her daily work, but not to a sufficient degree to be useful in the performance of household tasks. Thus, the right forefinger was not exposed to the more severe mechanical joint in-juries of housework and psychogenically induced, sustained hypertonia of somatic muscle. This patient was extremely right-handed, and grasped her various household implements with great force, probably because she did not have full use of her right forefinger.

There was no clinical evidence of impairment of innervation or circulation to the right forefinger. Sensations of heat, cold, pain, light touch, vibration, motion and position were normal in all the digits of the right hand. All the digits of the right hand were equally warm, and of the same color (210).

Because the interphalangeal joints of the right forefinger had been subjected to little mechanical injury, they had no articular deformities. However, the joints of other digits of the right hand were markedly deformed, presumably because of repetitive mechanical joint injury incurred by the tight grasping of household utensils, and by psychogenically induced, sustained hypertonia of somatic muscle. There was marked limitation of movement of the interphalangeal joints of the deformed digits, but not of the interphalangeal joints of the right forefinger.

CHRONIC ALLERGIC SYNDRONIES
Certain food-induced articular and non-articular allergic symptoms which are described

below may obscure partially or completely a patient's subjective appreciation of improvement in response to adequate niacinamide therapy, even though objectively satisfactory improvement in joint function is demonstrated by continuously rising values of the Joint Range Index on serial re-measurements of joint ranges. While these allergic reactions usually do not include any significant degree of limitation in ranges of joint movement, they may be responsible for considerable articular pain and discomfort, in addition to other symptoms of bodily discomfort. It is, therefore, of considerable importance in the medical management of a patient with joint dysfunction to distinguish between the symptoms of aniacinamidosis, which are ameliorated in time by adequate niacinamide therapy, and allergic syndromes which are ameliorated in time only by elimination of the offending allergen, or by hyposensitization to the offending allergen.

Although many diverse clinical manifestations of food allergy may occur in persons with joint dysfunction, three syndromes occur frequently in response to the ingestion of an offending food or foods: (a) Allergic Pain Syndrome (223) (167) (221), (b) Allergic Fatigue Syndrome (223) (167) (152) (153), (c) Allergic Mental Syndrome (223) (167) (151) (152) (153) (220) (40) (166) (213) (31) (165). These syndromes are described below. (Rarely, the allergic pain syndrome occurred when there was an active dental or tonsillar focus of infection, and was alleviated when the source of infection was eradicated. Only three examples of such benefit were observed in this series of 455 cases.)

These syndromes may occur separately in various degrees of severity and chronicity, or in any combination, and may be associated with a number of allergic symptoms not specifically included in the description of these syndromes. Clinical manifestations of these allergic syndromes may appear almost immediately after the ingestion of an offending food material and may continue for a few hours or a few days; or they may appear after a latent period of 12-76 hours following the ingestion of the allergen, and continue for as long as two weeks, gradually decreasing in severity during this interval. The daily ingestion of an offending food or food material produces a more or less steady state of allergic symptoms, with some exacerbation of these symptoms soon after the ingestion of this food.

Clinical proof that a suspected food is responsible for a patient's allergic symptoms is obtained (a) when such symptoms disappear when the offending food material is completely excluded from his diet for a sufficient period of time (2-3 weeks), and (b) when there is a recurrence of the initial pattern of allergic symptoms upon ingestion of the offending food material soon after he has become symptom-free as a result of abstinence from the allergenic food for a sufficient period of time; i.e., before abstinence from the food has been sufficiently prolonged for hyposensitization to have occurred.

(In addition to these three syndromes of allergic food reaction, offending foods have caused in patients with joint dysfunction the following types of allergic symptoms, which could be produced by the ingestion of the offending food, and could be eliminated by complete avoidance of the offending food:

Skin: Hives, angioneurotic edema, chronic pruritus, chronic skin lesions
Mucous membranes: Angioneurotic edema, canker sores.
Eyes: Chronic conjunctivitis.
Head: Cephalgia, including migraine.
Respiratory tract: Sneezing, postnasal drip, vasomotor rhinitis, recurrent sore throats, recurrent colds, sinusitis, asthma.
Gastro-intestinal: Nausea, vomiting, abdominal pain and cramps, heartburn, water brash, diarrhea, bilious attacks.)

While the ingestion of any food material can produce allergic symptoms in allergic persons, certain foods (chocolate, citrus fruits, tomato, pineapple, whole wheat, corn, milk, eggs and nuts) seem to be the most frequent offenders in the production of the allergic syndromes described below.

An oral threshold dose of an offending food is defined as the smallest quantity of that food which, when ingested not oftener than once every two weeks, will produce allergic

symptoms in a person sensitive to this food. An oral sub-threshold dose of an offending food is defined as that amount ingested not oftener than once every two weeks which will produce no clinically discernible allergic reactions in a person sensitive to this food. However, if sub-threshold doses of a single offending food are eaten daily by a person who is sensitive to this allergenic food in threshold doses, in a few days or weeks there may be precipitated a clinically obvious allergic reaction, which probably represents the summation of clinically inapparent allergic reactions which have reached an intensity exceeding the threshold for the production of allergic symptoms.

If sub-threshold amounts of several offending foods are eaten on the same day, an allergic reaction to these may occur, even though such foods when eaten separately on different days do not give rise to a clinically apparent allergic reaction. It has been noted that single sub-threshold doses of different offending foods ingested on consecutive days may precipitate a clinically obvious allergic reaction.

In many persons with severe food allergies, the amount of the offending food which precipitates clinically significant allergic reactions is so small that every trace of this food must be eliminated from the patient's diet if he is to have relief from his allergic symptoms.

When an offending food is eliminated from the diet for a sufficiently long period of time, the tolerance gained with clinical hyposensitization may be excellent and apparently unlimited; or it may be moderate and easily broken down, either by too frequent ingestion of the food in small or moderate amounts, or by the single ingestion of an excessive quantity of this food; or, the tolerance may be so slight that it may be easily broken down by the single ingestion of a very small amount of the offending food material.

Whether or not the patient has a personal or family history of allergy, at any time he may become sensitized to any food and have any pattern of food-induced allergic symptoms, which may vary in severity, chronicity and extensiveness from time to time (158) (159) (160).

Transient sensitization to certain foods has been observed in many patients with upper respiratory infections ("colds") who have a continuance of their acute coryza, malaise and lymphadenopathy as a result of a practice widely used in the treatment of "colds," particularly, during the early days of the "cold," namely, the ingestion daily of a quart or more of such liquids as citrus fruit juices, pineapple juice, tomato juice, milk and chocolate milk. When a person with a limited tolerance for these food materials takes these liquids in larger quantities than usual for him, his oral threshold dose is exceeded, and an allergic tissue reaction is produced which resembles that of "infectious colds." Often, this food-induced allergic reaction prolongs "cold-like" symptoms for several weeks. However, when the patient eats his usual diet and takes 8 to 10 glasses of water daily instead of large quantities of the above fluids, this food-induced allergic reaction is avoided and the patient recovers much more rapidly from his "cold."

Cyclic food resensitization is likely to occur when certain foods in season are eaten daily in ordinary or excessive amounts; e.g., tomatoes, citrus fruits, pineapple, strawberries, peaches, melon, corn; and hyposensitization is likely to occur when these foods are not in season, and the patient excludes them from his diet, or limits the amounts ingested. To avoid cyclic resensitization, an allergic patient is advised to vary his diet as much as possible throughout the year, and not to have too frequent or excessive ingestion of any one food (158) (159) (160).

Sometimes a patient with pollinosis will observe during his hay fever season that his reactions to known allergenic foods tend to be more severe, and that certain foods, which he could ingest with impunity at other seasons, give rise to allergic food reactions. Conversely, the ingestion of certain foods during his hay fever season may worsen his symptoms of pollinosis.

Extremes of environmental temperature occasionally increase the severity of the patient's reaction to the ingestion of an allergenic food.

A given food may cause allergic symptoms only when the patient is emotionally disturbed; or, a person who reacts to the ingestion of an allergenic food may react more violently if this food is ingested at a time when he is emotionally disturbed. Many patients suffering from severe allergic symptoms have considerable secondary anxiety concerning their allergic ailment, and often associated psychosomatic symptoms are so severe that they dominate the clinical picture, and the patient is considered to be psychoneurotic.

Excessive ingestion or excessive retention of dietary sodium tends to make the allergic reaction to allergenic foods more severe (99).

In women, a cyclic variation in the allergic pattern has been noted, so that clinical evidence of food allergy may occur only during the two weeks before, but not during the two weeks after, the menstrual period; or, food-induced allergic symptoms may be present throughout the month, but accentuated during the two weeks before the period (223) (167).

Allergic Pain Syndrome. In certain allergic persons, the ingestion of a threshold amount of an offending food material causes primarily mild, moderate or severe generalized pain in somatic muscle, tendon, periosteum, and periarticular and articular structures. A patient experiencing the allergic pain syndrome avoids all unnecessary physical exertion, since ordinary physical activity causes him pain and discomfort. Physical examination may disclose tenderness to palpation of somatic muscle, tendon, periosteum and periarticular structures. When the blood pressure cuff is inflated during the measurement of blood pressure, the patient may spontaneously complain of severe pain in the muscles of his arm. Somatic muscle is hypotonic and feels flaccid. Active and passive movement of joints may cause articular pain. The pain of this syndrome is usually not alleviated by the ingestion of aspirin, and if the patient is allergic to aspirin, the ingestion of this drug may even be responsible for the initiation and continuance of his allergic pain syndrome. Body massage usually worsens his pain and discomfort. In persons having the allergic pain syndrome, relatively slight mechanical joint injury will evoke severe and prolonged symptoms and signs of the delayed post-traumatic articular syndrome.

Allergic Fatigue Syndrome. In certain allergic persons, the ingestion of a threshold amount of an offending food material causes primarily extreme muscular fatigue, which is often associated with cervical lymphadenopathy (rarely, generalized lymphadenopathy), lymphocytosis and hypothermia (although occasionally there is a moderate elevation in temperature). Physical activity intensifies this allergic fatigue, but prolonged rest does not relieve the patient's symptoms of fatigue.

Allergic Mental Syndrome. In certain allergic persons, the ingestion of a threshold amount of an offending food material causes primarily mental symptoms, including mental fatigue, depression and confusion. The person may complain of disagreeable "mental fogginess or haziness," "a feeling of partial anesthesia," or a "feeling of being drugged." Thought processes are slowed. The patient may have unwarranted irritability, unreasonableness, temper tantrums, loss of memory, inability to concentrate, restlessness, sleepiness (although occasionally insomnia is noted). The patient’s mental inertia may be so severe that he finds it difficult to make decisions about even uncomplicated matters. He vacillates, procrastinates, and has trouble in carrying out even the simplest plans that he has made. He may require long naps during the day and may sleep long hours at night without relief from such mental fatigue. He knows that "something is wrong" with him, and he can describe his pattern of mental symptoms, although usually he is reluctant to do so because such symptoms have been made light of by his family and friends. A patient may refuse to discuss his pattern of allergic mental symptoms with the physician at the time of the initial visit, fearing that such symptoms are indicative of mental disease (insanity). He often complains that "life is not worth living” feeling this way.

The allergic patient with this mental syndrome may be secondarily disturbed because his family and physicians consider him to be a chronic grumbler and complainer. He feels emotionally insecure because he has been unable to obtain therapeutic relief from

his allergic symptoms. Often such a person, with the tentative diagnosis of "psychasthenia," "neurasthenia," "nervous exhaustion," "psychoneurosis," or "psychosomatic fatigue," is referred to a psychiatrist, who, after studying the patient, believes that the patient's problems are psychosomatic in origin, not realizing that food allergy has created a somatopsychic disorder, which can be corrected by the removal of the offending food material from the patient's diet, but not by psychotherapy.

A few patients with joint dysfunction have, in addition to the allergic mental syndrome, a primary neuropsychiatric disturbance. In such in-stances, treatment must include adequate niacinamide therapy, exclusion of the offending food material from the diet and expert psychotherapy.

TREATMENT OF CIIRONIC ALLERGIC FOOD SYNDROMES

Skin testing was rarely used in attempting to identify allergenic foods, since false-negative scratch or intracutaneous skin reactions may be obtained for a given food or group of foods, the ingestion of which causes the patient to experience clinically important allergic reactions, and falsepositive skin reactions may be obtained for food materials, the ingestion of which is clinically well tolerated by the patient (167) (152) (153) (151) (158) (159) (160) (81) (150).

Elimination diets, especially the diets of Rowe (167) (171), were used and modified empirically as necessary in the attempt to rid the patient of his food-induced allergic symptoms. At times, it may be extremely difficult to select a basic elimination diet which will accomplish this. When symptoms due to food allergy are not abated in 7 to 14 days, the patient is probably allergic to one or more foods in the elimination diet. While an elimination diet containing few foods sometimes gives relief from allergic symptoms, the too-frequent ingestion of the small number of foods in such a diet favors sensitization of the patient to any of the allowed foods. When new foods are added to the patient's basic elimination diet after he has been free from his chronic allergic food symptoms for two weeks, the patient should keep an accurate food-symptom diary which permits the physician to assess the patient's clinical reactions to the ingestion of the newly added food materials. If any added food seems to be giving rise to allergic symptoms, its use is interdicted. The patient's elimination diet is liberalized as rapidly as possible by the addition of those foods which by trial he is able to ingest repeatedly without experiencing allergic symptoms. It is possible at any time for an allergic patient to become sensitized to foods that formerly he tolerated well, and when symptoms suggestive of allergic reaction to the ingestion of foods recur, it is necessary to resume the search for offending food materials.

The polypropeptan method (223) of specific desensitization to twelve basic foods was given a limited trial, and good results were obtained in some patients. The food-symptom diary was useful in observing the clinical effects of specific desensitization, and the patient's reaction to the subsequent addition of new foods not included in the basic list of twelve foods. When the ingestion of new foods caused allergic symptoms, and specific propeptans were available for treatment, the patient was desensitized to these foods; when specific propeptans were not available, the use of these foods was interdicted.

The method of individual food-testing advocated by Rinkel and others (159) (155) was not employed.

At times the antihistaminics were employed as palliative measures in the treatment of hay fever symptoms and of certain pruritic skin conditions (hives, contact dermatitis). The chronic allergic food syndromes described in this volume did not seem to respond to treatment with antihistaminic drugs.

While it is tedious and time-consuming for the patient to keep an accurate food-symptom diary, and for the physician to analyze such a diary, this method of clinical investigation has been most helpful in the identification of specific foods causing allergic symptoms, and in the evaluation of the patient's response to elimination of allergenic foods from his diet. Some patients were unwilling or unable to cooperate in keeping a

food-symptom diary and in restricting their diet as suggested. About 70% of persons who had symptoms suggestive of chronic allergic food syndromes were willing and able to cooperate in this exacting program.

The diary is kept in a standard stenographer's notebook, with a central dividing line on each page. Each notebook page contains the record of one day only. The diet (including all snacks, condiments and food-tasting) and the time of ingestion of each meal are noted in sequence in the left-hand column of each page. The patient is instructed to be specific in his description of the types of food eaten and, wherever possible, to list the ingredients of such mixtures as soups and salads. In the right-hand column, the patient lists his complaints, including the time of onset, degree of severity and duration of symptoms. A diary which is carelessly kept or has days omitted is not reviewed. It is not considered desirable in most instances to study food-symptom diaries which are kept for less than one month.

Certain additional information included in the diary is of value in the analysis of the patient's allergic and non-allergic symptoms. The patient records his emotional upsets, since these may cause reactions to allergenic foods to seem more severe, or may be accompanied or followed by psychosomatic symptoms. Any unusual physical activity is recorded, since this often actuates a delayed post-traumatic articular syndrome which might otherwise be confused with certain types of chronic allergic food reactions. Women record the days of menstrual flow, so that any pre-menstrual accentuation of allergic or sodium retention symptoms can be detected.

Such a diary gives more objective and accurate information concerning the patient's pattern of symptoms than his verbal impressions of how he has felt for a given period of time. Through the use of the food-symptom diary, it is possible for the physician to analyze accurately the patient's clinical reactions to:

(a) the ingestion of threshold amounts of allergenic foods;

(b) the elimination of allergenic foods from the patient's diet

(c) the re-introduction of allergenic foods after a period of abstinence, to test the degree of clinical hyposensitization to the offending food material, and to detect promptly any clinical evidences of resensitization to such foods; and

(d) the daily ingestion of sub-threshold amounts of allergenic foods for a sufficient period of time to produce summation effects.

In addition to data relative to allergic syndromes, objective analysis of the food-symptom diary yields other clinical information about the patient, and may be helpful in differentiating the symptoms of chronic allergic syndromes, delayed post-traumatic syndrome, sodium retention syndrome and psychosomatic syndromes. A carefully kept food-symptom diary indicates the regularity or irregularity of the patient's living and eating habits; the variety or monotony of his diet; his caloric intake; the relative amounts of protein, carbohydrate and fat in his diet; the quality and quantity of dietary protein; his vitamin and mineral intake; and his caffeine and alcohol intake.

The identification of offending foods is relatively simple when the diary shows days when the patient is entirely free from allergic symptoms, and is relatively difficult when the diary indicates that the patient is never free from allergic symptoms. When an offending food is eaten less often than once a week, the allergic symptoms following the ingestion of this food usually appear after a latent period of half an hour to 72 hours (usually 12-24 hours) and usually last from 4 hours to 4 days. When the patient's diary reveals that he is never without allergic symptoms, the only clues to the identity of the offending food material are obtained by noting variations in the intensity of symptoms. Slight intensification of symptoms usually follows soon after the ingestion of an offending food, and slight diminution in the intensity of allergic symptoms is noted when such offending food material is absent from the diet for a day or more. It is in such instances that the use of elimination diets or polypropeptan therapy is most helpful in alleviating the steady state of allergic symptoms. Once the patient has become

symptom-free, the effects of the addition of new foods can be ascertained from a study of the food-symptom diary.

When analysis of the food-symptom diary suggests that the patient is having food-induced allergic symptoms, recommendations are made that the suspected allergenic food material be completely eliminated in all forms from the patient's diet. The patient continues to keep his food-symptom diary so that the effects of restriction of suspected allergenic food materials can be observed. Specific dietary advice is always given to the patient so that, after exclusion of suspected foods, his diet is adequate in protein, calories and minerals. When milk and milk products are excluded from his diet for any prolonged period of time, suitable calcium preparations are administered to offset the resulting dietary loss of calcium. Patients who habitually use large amounts of salt in the diet or who seem to have symptoms resulting from excessive sodium retention are asked to limit their salt intake.

When a patient is allergic to many foods, usually only a few of the allergenic foods which cause his symptoms can be identified upon analysis of the first month's diary. Even when a few of the offending foods are eliminated from his diet, the subsequent food-symptom diary often shows one or more of the following alterations in the pattern of allergic symptoms:

(a) a lessening in intensity of symptoms, (b) longer intervals of freedom from such symptoms, (c) the elimination of certain allergic symptoms but not of others; e.g., the allergic pain syndrome may be eliminated, but allergic pruritus may persist. Such changes in the patient's allergic symptomatology may be noted usually within two or three weeks after the exclusion of the chief offending foods from his diet, although occasionally benefits may be noted from the exclusion of allergenic foods as early as the third or fourth day. Eventually, when all of the offending foods are eliminated from the allergic patient's diet, he becomes symptom-free, and the food-symptom diary may be discontinued. Should allergic symptoms recur, he is asked to resume keeping the food-symptom diary.

If the offending food is re-introduced into the patient's diet after several months of abstinence, his reaction to the ingestion of this food may be of the original intensity, or less severe, or absent, depending upon the degree of hyposensitization to the offending food which occurred in the time during which this food was excluded from the patient's diet. In most persons, in order to achieve complete clinical hyposensitization to the offending food, it may be necessary to exclude this food for a year or longer. Rarely, even with prolonged exclusion from the diet, there is no demonstrable clinical hyposensitization to an offending food (150) (167) (219).

When it has been demonstrated that a patient has become clinically hyposensitized to a single ingestion of a food which formerly caused allergenic symptoms, he is instructed to have this food infrequently, in amounts limited to average servings, in order not to become resensitized to this food (160) (199). He is advised not to have this food more than once a month for six months; for the next six months, not to have the food more often than once every two weeks; and thereafter, not to have the food more often than every fifth day. If at any time there is a recurrence of symptoms due to ingestion of this food, it must be excluded from the diet to permit clinical hyposensitization to take place again.

Sometimes, a patient wants to find out for himself whether he is really allergic to a given food, or whether it is his "imagination" or an idea of the doctor's. When he ingests the allergenic food before hyposensitization has occurred, he has his usual allergic symptoms. However, he will not have allergic symptoms in response to the ingestion of this food if he has become hyposensitized.

Although suggestion may play a part in the production of the allergic patient's symptoms and in his relief from symptoms following the exclusion of allergenic foods from his diet, the reaction of truly allergic persons, who are markedly sensitive to a given food, is predominantly due to bodily changes produced by the ingestion of allergenic food materials. This becomes strikingly clear when such a patient inadvertently and

unknowingly ingests an allergenic food material, and subsequently develops his typical pattern of allergic symptoms (233) (22) (61) (93) (151) (152) (153) (184).

Although a patient may complain that a diet which eliminates certain offending foods is monotonous, analysis of his diet when his choice of foods is unrestricted is likely to show little or no variety in his daily menus. An emotionally well-adjusted person is usually able to tolerate dietary restrictions without developing anxiety, even though essential or favorite foods may be interdicted. Sometimes a person who has developed excessive guilt in response to certain life situations seems to welcome food restriction, since symbolically this constitutes punishment for his real or fancied sins.

Certain patients are unable to cooperate adequately in the investigation and treatment of their allergic food syndromes because of their emotional disorders; e.g., a patient may develop considerable anxiety in response to dietary restriction of important or unimportant foods, perhaps because deprivation of food symbolizes a threat to his security and re-activates old, unresolved conflicts, and he will drop treatment; or, a patient who has secondary gains from the continuance of his allergic symptoms may protest that he will "do anything to feel well," but he impedes in every possible way the efforts of the physician to work out a solution to his allergic problems. Treatment of the chronic allergic food syndromes of such patients will not be successful until the basic emotional disorder has been corrected by psychotherapy (218).

SOME EXAMPLES OF FOOD-INDUCED ALLERGIC REACTIONS

CASE AA. This case history demonstrates the clinical effects of the ingestion at different times of various amounts of an offending food, to which the patient was moderately allergic, and subsequent hyposensitization resulting from abstinence from this food for a sufficiently long period of time.

A 52-year-old widow complained of severe headaches recurring cyclically every Sunday for many years, and occasional milder headaches at other times. These headaches had not been modified by her uneventful "change of life," which occurred when she was 46 years old. Every Sunday morning she awoke with a violent, throbbing bitemporal cephalgia (headache) associated with photophobia, nausea and vomiting. The severe headache persisted for 24 hours, and was succeeded by dull head discomfort persisting for as long as 48 hours. No previous medical measures had prevented the cyclic recurrence of her headaches or had given her appreciable relief from the pain and associated discomfort of this type of cephalgia.

She was asked to keep a food-symptom diary for one month. This record revealed that every Saturday night she ate as many as fifteen pieces of chocolate candy during the course of a weekly bridge game, although at other times during this month she did not eat chocolate. On the basis of her food-symptom diary, it was suspected that chocolate was the chief offending food material. Upon advice, she eliminated chocolate completely from her diet for two weeks, during which interval, for the first time in 10 years, she was free from her usual Sunday headaches and associated symptoms. As a confirmatory test, she re-introduced chocolate into her diet, and reported that approximately 12 hours after the ingestion of 10 pieces of chocolate candy she experienced her usual severe cephalgia with associated nausea and vomiting. Thus, it was confirmed clinically that chocolate was the offending food:

(a) by the absence of her headaches upon the exclusion of chocolate from her diet, and

(b) by the reappearance of her usual severe cephalgia upon re-introduction of a given amount of chocolate into her diet before clinical hyposensitization had taken place.

The patient cooperated over a period of many months in a sequential clinical investigation:

1) to determine the smallest oral dose of chocolate which would cause her usual headache; i.e., the amount of chocolate that was the threshold oral dose for the production of her usual allergic symptoms:

When she had abstained from chocolate for two weeks and was free from her usual headaches during this interval, the smallest amount of chocolate ingested at one time which produced her usual headaches after a latent period of approximately 12 hours, was four squares of a popular chocolate bar.

2) to determine that oral dose of chocolate ingested once every two weeks which was sub-threshold for the production of her allergic symptoms; i.e., the amount of chocolate ingested once every two weeks that was insufficient to cause her headaches:

When she had abstained from chocolate for two weeks and was free from her usual headaches during this interval, she was able to eat three squares of chocolate once every two weeks for three successive two-week periods without precipitating her usual headache.

3) to demonstrate that in time the ingestion of sub-threshold doses of chocolate under certain conditions caused the appearance of her allergic symptoms.

When she had abstained from chocolate for two weeks and was free from her usual headaches during this interval, the daily ingestion of one square of chocolate for four successive days caused no clinical symptoms until the fifth day, when she awakened with her usual severe headache.

For the next two-week period, she abstained from chocolate and was free from her usual headaches during this interval. Then she ate three squares of chocolate on one day, without experiencing any clinically apparent allergic reaction. When she repeated the same oral dose of chocolate four days later, she developed her usual severe headache on the following day.

In this instance, it would appear that eventually the threshold necessary for the production of her allergic symptoms was exceeded as a result of the summation of clinically inapparent allergic reactions following the repetitive ingestion of sub-threshold amounts of the offending food.

When chocolate was excluded in all forms from this patient's diet for one year, during this time she was completely free from headaches, and became sufficiently hyposensitized so that she could eat four or five pieces of chocolate candy daily for weeks at a time without experiencing headaches or other allergic symptoms.

CASE BB. This case history demonstrates the occurrence of the Allergic Fatigue Syndrome and the Allergic Pain Syndrome following the ingestion of an offending food to which the patient was severely allergic, and subsequent hyposensitization resulting from abstinence from this food for a sufficiently long period of time.

This 36-year-old physician experienced for more than 8 years severe and persistent generalized periosteal pain, excessive and persistent fatigue that was not relieved by rest, and recurrent afebrile attacks of swollen lymph nodes. These symptoms at irregular intervals reached such intensity that periodically he had to take to bed for a week or more, until there was some remission in his symptoms.

On two occasions 9 and 10 years ago, he experienced two distinct severe episodes of classic acute infectious mononucleosis. Although his heterophile antibody test was strongly positive during the two acute attacks of infectious mononucleosis, it was negative subsequently. However, for at least 6 years, stained smears of his peripheral blood revealed the presence of atypical lymphocytes resembling those usually found in infectious mononucleosis (154).

He was studied over a period of many years by internists, allergists, hematologists and psychiatrists. Chronic afebrile brucellosis was excluded through a battery of tests (74). Skin testing indicated that he was allergic to a variety of inhalants and foods. Over a period of several years he was desensitized to various pollens and house dust with relief of his seasonal hay fever, but with no relief of his presenting symptoms of pain, fatigue and recurrent swollen lymph glands. The elimination from his diet for several

years of a number of foods to which he showed strongly positive skin reactions (milk, eggs, wheat, nuts, lettuce, legumes) did not appreciably alter his chronic illness. It was, therefore, believed that his food allergies did not contribute to his chronic symptomatology.

The psychiatrists doubted that his symptoms were the expression of serious maladjustments to life situations or hypochrondriasis, although they noted moderate anxiety about his health. The consensus of his attending physicians was that he had a chronic recurrent type of infectious mononucleosis. He was advised to "take it easy, to adopt a philosophical attitude" toward his illness, and take it in his "stride."

During the meat shortage in 1944, for a period of several months this patient had a "spontaneous" remission from his illness. He was astonished and delighted to feel well for the first time in more than 8 years. However, he did not realize at the time that this period of freedom from symptoms corresponded exactly to the interval during which he ate no beef. (Previous skin-testing to beef had given a negative reaction both by the scratch and intracutaneous methods.) After several months of a beef-free diet, he ate his first beef-containing meal, and immediately there was a recurrence of his Allergic Pain Syndrome and Allergic Fatigue Syndrome and lymphadenopathy. Following the ingestion of this beef-containing meal, there was for 10 days no appreciable decrease in the intensity of his allergic symptoms.

This patient was advised to abstain from beef except for a test meal of beef to be taken once every 6 months. After 6 months of abstinence from beef, he was symptom4ree until he ingested a beef-containing test meal, which again caused a recurrence of his allergic symptoms, this time lasting only 3 days.

For another six-month period of abstinence from beef, he was symptom-free, until he ingested his next beef-containing test meal, which was followed by an allergic reaction lasting only 36 hours. After the next beef-free and symptom-free six months, the ingestion of a beef-containing test meal caused a recurrence of his allergic symptoms lasting only 8 hours. Finally, after 6 months more of abstinence from beef and freedom from allergic symptoms, the next ingestion of a test meal of beef caused no subjectively discernible allergic reaction.

During the 2 ½ years in which he abstained from beef (save for single test meals of beef every 6 months) there was a progressive clinical hyposensitization to beef, as indicated by a gradual decrease in the intensity and severity of the beef-induced allergic reactions. Future study will indicate whether or not clinical hyposensitization is sufficiently complete so that beef may again be introduced into his diet in moderate amounts at weekly or bi-weekly intervals without causing re-sensitization to beef and a recurrence of this patient's Allergic Pain Syndrome, Allergic Fatigue Syndrome and lymphadenopathy.

CASE CC. This case history demonstrates the occurrence of the Allergic Mental Syndrome in response to the ingestion of an offending food, and the absence of this syndrome upon exclusion of the offending food from the diet.

This 19-year-old college girl had an excellent academic record for her freshman and sophomore years, being in the first 10 per cent of her class scholastically. Early in the first semester of her junior year, she noticed for the first time that she was foggy mentally and forgetful, and that it was difficult for her to concentrate on her studies, as a result of which she fell behind in her required class work. After a few weeks, her mental fatigue became so severe that she frequently overslept in the morning, missing classes. There was no lessening in the intensity of her mental fatigue as a result of extra sleep, and she often felt "drugged" on awakening. Frequently she was so sleepy in the afternoon that she had to take long naps.

In an attempt to overcome her deficiencies in her school work, she gave up all social activities, no longer having "dates" or going to the movies; instead, she devoted all her time to study. She became emotionally upset when she realized that course material that should have been easy for her to master, was not; and that she could not recall

things which she had learned and understood well at an earlier time. She was failing in her school work, and had received notice from the Dean's office that she was taking excessive class cuts.

Before Thanksgiving she was advised by the Dean to consult a psychiatrist in order to determine the cause of her recent scholastic inadequacies and the cause of the recent changes in her personality which had been noted by instructors.

When she was first seen, she volunteered that she was afraid she was losing her mind. Her history revealed that during the last semester of her sophomore year she took a course in nutrition and decided to improve her protein intake by adding eggs to her diet, in spite of the fact that previously she had disliked eggs and always avoided eating them. Accordingly, during the first semester of her junior year, she forced herself to eat one or two eggs daily.

Upon questioning, it became apparent that her troubles in college began soon after the introduction of eggs into her diet. In view of this, she was asked to stop the ingestion of eggs and all egg-containing foods. She was also asked to keep a food-symptom diary (which proved to be unnecessary, since within 2 weeks after the exclusion of eggs from her diet, she again felt mentally alert, was free from the disagreeable fogginess and sleepiness, and was able to do her school work easily and efficiently). In spite of her poor scholastic showing during the beginning of the first semester, she was able to complete the first semester of her junior year with a B-plus average.

This patient has not been significantly desensitized to eggs or egg-containing foods after two years of abstinence from these foods.

CASE DD. This case history demonstrates the occurrence of the Allergic Pain Syndrome in response to the ingestion of a number of offending foods to which the patient was severely allergic, and the absence of this syndrome upon exclusion of the offending foods from the diet.

Since the age of 10, this 35-year-old housewife had not been free at any time from generalized subcutaneous edema and severe bone, joint and muscle pain. These symptoms were cyclically increased in intensity during the 10-day interval preceding her periods, and also seemed to be aggravated by weather changes. She did not complain of physical or mental fatigue, and had no mental symptoms save for premenstrnal tension. Her basal metabolic rate was within the normal range. She had been studied and treated by many physicians over a period of many years without obtaining even slight relief from her symptoms. Most of the physicians thought she had some form of chronic "rheumatism," and in addition that she suffered from hypochondriasis.

Physical examination disclosed that she had moderate joint dysfunction, which responded satisfactorily to the. subsequent institution of therapy with adequate amounts of niacinamide, but her syndrome of subcutaneous edema and pain was not materially affected by this treatment.

The premenstrual accentuation of her subcutaneous swellings and pain and premenstrual tension were controlled by a moderately low-sodium diet throughout the month, supplemented by 1 g of enteric coated ammonium chloride three times a day after meals and at bedtime, which was administered daily for the two weeks preceding her periods. Because her symptoms of pain persisted, even though her Joint Range Index increased satisfactorily over a period of time in response to niacinamide therapy, and even though the premenstrual accentuation of her symptoms was prevented by a low-sodium diet, an allergic cause for her symptoms was sought and found.

Analysis of her food-symptom diary over a period of several months indicated that she was severely allergic to wheat, eggs, pork and nuts. When these foods were excluded from her diet for 3 weeks, she became completely free from subcutaneous edema and bodily pain for the first time in her life that she could remember.

Adequate niacinamide therapy caused satisfactory improvement in the Joint Range Index, but did not alleviate the Allergic Pain Syndrome. Salt restriction alleviated only the symptoms due to the salt-retention syndrome. Exclusion of allergenic foods from the diet relieved the Allergic Pain Syndrome. Thus, in order for this patient to feel well, it was necessary to institute the proper medical treatment for three separate clinical entities: joint dysfunction, sodium retention syndrome and food-induced Allergic Pain Syndrome.

CASE EE. This case history demonstrates the occurrence of the Allergic Fatigue Syndrome in response to the ingestion of a number of offending foods to which the patient was severely allergic, and the absence of this syndrome upon exclusion of the offending foods from the diet.

This 20-year-old male, a college senior, complained for 3 years of persistent physical weakness and exhaustion. He also had at times swollen neck glands. Although he was so tired at times that he was unable to attend classes, he had no difficulty in mastering his course work, and his scholastic record was excellent.

Over a period of several years, studies in the college Health Service gave no positive evidence for active tuberculosis, brucellosis or infectious mononucleosis as a cause of his fatigue. Physical examination showed that he had a slight grade of joint dysfunction, and that his muscles were hypotonic and weak. His joint dysfunction responded to adequate treatment with niacinamide, but his physical fatigue and weakness and his muscle hypotonia did not improve.

A study of his food-symptom diary showed a daily ingestion of chocolate and tomato-containing foods. Clinical proof was obtained that chocolate and tomato were the chief offending substances. Within 10 days after eliminating these foods from his diet, he had complete relief from his physical fatigue and muscle hypotonia. Subsequently, there were no recurrences of his symptoms, save when occasionally he "forgot" and ate the offending foods.

SODIUM RETENTION SYNDROME

Certain patients have complaints of bodily discomfort which disappear when the sodium content of the body is decreased by appropriate therapy, and recur whenever excessive sodium retention recurs. It is believed, therefore, that these symptoms are the result of excessive retention of sodium in the body, whatever the cause of this may be (216) (144).

It is well known that excessive sodium retention (as well as chloride and water retention) occurs cyclically in women who have premenstrual tension, and that excessive ingestion of sodium in the diet or in medicaments accentuates premenstrual tension. Any or all of the following symptoms and signs may occur in a mild or severe form starting 10-14 days before the onset of the period, and usually subside during the period or immediately thereafter: gradual enlargement, swelling and bloating of the body so that garments become uncomfortably tight; a gain of 2-5 pounds in weight, which is lost soon after the period begins; myalgia, arthralgia, backache, cephalgia (including migraine), nausea, sensations of intestinal bloating, pelvic discomfort, labial itching; emotional instability (including nervousness, touchiness, crying spells, irritability, quarrelsomeness, dopiness and depression); excessive fatigue, inability to concentrate, impaired memory, clumsiness, insomnia, hyperkinesis, erratic behavior, increased sexual desire. There may be noticeable edema of the face and various portions of the body. The breasts are usually enlarged and tender to palpation, and somatic muscle and periosteum are likely to be tender to digital palpation.

In most other clinical forms of sodium retention the symptoms and signs are less severe and less extensive than those found in severe premenstrual tension, and consist chiefly of arthralgia, nervousness, insomnia, dizziness and increased blood pressure (65) (142) (143) (190) (222) (128). Sodium retention of a degree sufficient to cause symptoms may occur in persons who are ingesting excessive amounts of sodium. However, some persons have a significant degree of sodium retention with even a moderate intake of

sodium. Reduction of excessive amounts of sodium in the body by appropriate therapy gives complete relief from the above symptoms, and in some persons there may be a decrease in blood pressure as a result of salt restriction.

With excessive sodium retention, there may be an accentuation of symptoms due to coexisting complicating syndromes (the delayed post-traumatic articular syndrome, chronic allergic food syndromes, and psychosomatic syndromes). When the sodium content of the body is decreased by therapy, there is a decrease in the severity of the symptoms due to sodium retention, and usually a decrease in the intensity of symptoms of the coexisting complicating syndromes.

The symptoms and signs of excessive sodium retention (64) (88) (139) are usually controlled when the patient limits the amount of salt in his diet, drinks 8 to 10 glasses of water daily and, when indicated, takes enteric coated ammonium chloride tablets (1 to 3 g, t.i.d. p.c. and h.s.)

Although severe sodium chloride restriction has been known to cause symptoms which are usually alleviated by the administration of salt (175) (28), no patient in this series who was treated for the sodium retention syndrome developed such symptoms.

SYNDROME OF PSYCHOGENICALLY INDUCED, SUSTAINED HYPERTONIA OF SOMATIC MUSCLE

GENERAL CONSIDERERATION OF PSYCHOSOMATIC SYMPTOMS IN JOINT DYSFUNCTION

Either before treatment for joint dysfunction was undertaken or subsequently, most patients included in this study suffered at one time or another from a variety of transient or persistent, mild, moderate or severe symptoms of bodily discomfort, which were interpreted as being collateral to primary mental tension (43) (235) (203) (49). Psychosomatic symptoms occurring during the course of treatment of joint dysfunction often caused the patient much subjective discomfort, and obscured his appreciation of improvement in joint dysfunction in response to adequate niacinamide therapy, even though satisfactory improvement in joint dysfunction was demonstrated objectively by continuously rising values of the Joint Range Index as determined serially during the course of adequate niacinamide therapy.

In most instances, the existence of psychosomatic illness (where symptoms of bodily discomfort are caused, intensified or perpetuated by mental influence) can be validated when the symptoms of bodily discomfort are consonant with the emotional problems of the patient (235) (50) (110) and disappear upon satisfactory discharge of the inciting mental tension; and when, upon careful clinical investigation, no evidence can be found for co-existing somatic disease which could produce such symptoms (237). In some instances, symptoms of bodily discomfort initiated by mental tensions may persist as habit patterns even when the inciting mental tensions are adequately discharged. In the treatment of a patient with such symptom-producing non-purposive habit patterns, the patient must be re-educated before these mechanisms of habitual behavior can be extinguished (100) (122).

It is not uncommon to find in a person with psychosomatic symptoms the coexistence of clinically significant asymptomatic or symptom-producing somatic disease. Symptoms of primarily somatic disease may or may not be similar to symptoms of a coexisting psychosomatic illness. When the symptoms due to concurrent somatic disease and psychosomatic illness are similar or identical, the relative relief obtained from removal of the psychosomatic component by psychotherapy may be such that the patient temporarily feels greatly benefited. If the patient and physician are satisfied with such a therapeutic result, serious somatic disease may be overlooked until it produces such symptoms and signs that its presence cannot be ignored, and by this time, the somatic disease process may not be amenable to any form of therapy.

While patients differ in the degree of susceptibility to externalization of their mental tensions through psychogenic symptoms of bodily discomfort, any patient may develop

psychosomatic symptoms either transiently or persistently if the mental stresses to which he is exposed are for him sufficiently severe, sufficiently prolonged, or sufficiently repetitive (111) (110) (2) (3).

Once psychosomatic symptoms occur, patients differ in their ability to become free from such symptoms, either spontaneously or through directed therapy. It is well known that symptom-producing alterations in visceral function and in somatic muscle tone are the usual accompaniments of many emotional states, such as anxiety, fear, panic, resentment, hostility and rage (27) (241) (133) (55). In acute and subacute emotional states, symptoms of bodily discomfort produced by psychogenic alterations in visceral function often preponderate, while in chronic emotional states, symptoms of bodily discomfort produced by psychogenically sustained hypertonia of somatic muscle preponderate. In acute, subacute and chronic emotional states, the patient's collateral emotional response to the unpleasant sensory concomitants of psychogenically altered bodily function produces a heightening of his total emotional tensions and an increase in the severity, extensiveness and duration of his psychosomatic symptoms (29) (182). When these psychogenic symptoms of bodily discomfort become severe enough, they often serve to deflect the patient's attention from his primary mental tension and anxiety to his collateral somatic dysfunction. Thus, temporarily he may feel relieved, and may not be disturbed consciously by his primary mental tension, although in time he may develop secondarily considerable mental tension and anxiety concerning his continuing psychosomatic symptoms and their possible meaning to his health and his future security. Before instituting treatment of the patient with psychosomatic symptoms, the physician should try to evaluate the part played by the psychosomatic symptoms in the maintenance of the patient's biodynamic homeostasis, and the emotional resources which the patient could muster to deal with his basic emotional problems if his psychosomatic symptoms were prematurely removed by ill-advised psychotherapy.

A patient who is usually well-adjusted may have psychosomatic symptoms only when he is suffering from an acute or subacute tensional situation, but a severely psychoneurotic patient may never be entirely free from reciprocally interacting psychogenic symptoms of bodily discomfort and mental tensions. In some patients the severity, extensiveness and subjective awareness of psychosomatic symptoms may seem directly proportional to the severity of the existing mental stress, while in others no such direct relationship obtains. In some patients, exposure to any degree of mental stress always seems to call forth the same fixed pattern of psychosomatic symptoms and mental tensions. Psychosomatic symptoms may be absent, or present at low levels of intensity, extensiveness and psychic awareness when the patient's chronic psychoneurosis is compensated, and are usually present at high levels of intensity, extensiveness and psychic awareness when the psychoneurosis becomes decompensated. Usually, the more aware the patient is of his psychosomatic symptoms, the less aware he is of his primary mental tensions; indeed, if he is aware of any mental tensions at all, he usually attributes these to his intense concern about his presenting psychosomatic symptoms and their meaning to his health and future security.

A patient with joint dysfunction (with or without obvious arthritic deformities) who also has a compensated psychoneurosis will tolerate a more or less steady state of reciprocal emotional and psychosomatic discomfort which he considers to be normal for him. The continuance of his troublesome symptoms and the secondary gains he derives from his chronic compensated psychoneurosis may in time afford him a considerable degree of emotional security and satisfaction. It is only when his psychoneurosis becomes decompensated that such a patient will develop intolerable anxiety and intolerable psychosomatic symptoms, and when his psychoneurosis again compensates either spontaneously or through psychotherapy, he will revert to his original steady state of tolerable emotional and psychosomatic discomfort, and he may feel that he has been cured and is normal again. Frequently the treatment of psychogenic syndromes of bodily discomfort is rendered difficult by the unwillingness of the patient with such syndromes to cooperate in an investigation of the mental and emotional factors which are etiologically related to his presenting psychosomatic illness.

While it is not the purpose of this volume to describe all types of psychosomatic symptoms observed in the group of patients treated for joint dysfunction, in this section consideration will be given to articular and nonarticular psychosomatic symptoms arising directly or indirectly from psychogenically induced, sustained somatic muscle hypertonia, and appropriate suggestions for the management of this syndrome will be offered.

In planning appropriate treatment for psychogenically induced, sustained hypertonia of somatic muscle, the physician should try to understand the basis of the patient's psychosomatic symptoms m terms of the interactions of endogenous and exogenous operational factors which made the patient the person that he is, predisposing him to his illness, initiating his psychosomatic disorder, and causing his illness to persist (168).

PSYCHOBIOLOCIC STUDY OF THE PATIENT WITH JOINT DYSFUNCTION

In the clinical analysis of the patient's health problems (236) (66) (235) (239) (100) (117) (157), coexisting psychic, somatic psychosomatic and somatopsychic phenomena were regarded as dynamic, interrelated and integrated manifestations of the functioning human psychobiologic unit. However, certain techniques were primarily employed in the study and treatment of psychic aspects of disease, and other techniques were primarily employed in the study and treatment of somatic aspects of disorders. Combinations of these techniques were used to identify, study and treat (a) psychosomatic disorders, in which symptoms of bodily discomfort are collateral to primary mental disorders, and usually disappear when aberrant mental functioning is corrected, and (b) somatopsychic disorders, in which mental disorders are collateral to primary somatic disorders, and usually disappear when aberrant somatic physiology is corrected.

The clinical study of each patient was performed unhurriedly in order to give the patient adequate time to express his complaints fully, and to give the examiner sufficient time to collect the necessary clinical data, and, upon reflection, to make the necessary clinical correlations, and to evolve a reasonable plan of treatment for the patient. Throughout the clinical study, without appearing to do so, the physician continuously observed and evaluated the verbal and somatic reactions which exteriorized some of the patient's emotional responses during the elicitation of the history, during various procedures of the physical examination, and during the summary of the patient's health problems and the recommended therapy.

As an approach to the understanding of the patient's emotional and psychological problems, during the course of the initial interview, information was obtained, sometimes by indirection, concerning many matters which were independent of joint dysfunction but were often responsible for the evolution and persistence of the patient's presenting attitudes, moods, sentiments, conflicts and psychosomatic symptoms. Such information included data concerning the patient's childhood, family problems, home life, educational background, social background, religious background, emotional background, worries and plans concerning the future, his work experience, adjustments to various life situations (including his illness), the patient's interpretation of the cause and significance of his symptoms and illness, the persons he has known or heard of who have similar symptoms, and any apparent temporal relationship between the occurrence of emotionally upsetting events and the onset of his symptoms. The patient was encouraged to summarize what he considered to be his "good and bad points," and the 'best and worst periods" of his life. An attempt was made to assess his attitude toward his failures and successes, toward his mental and physical handicaps, toward his "sacrifices" for the benefit of other members of his family, and toward ailing members of his family. It was often helpful to know the patient's schedule of activities during an average day and week, and during weekends, holidays and vacations, since he may have symptoms only at certain times: at home, at work, in church, on a vacation, on weekends or holidays, when meeting or visiting certain individuals.

It is important to keep in mind that persons with the more severe grades of joint dysfunction who have psychogenic articular and non-articular symptoms often have repressed resentment, hostility, rage and aggressive-ness which are chiefly exteriorized by localized or generalized sustained hypertonia of somatic muscle (89). In the older

age groups particularly, psychogenically induced, sustained hypertonia of somatic muscle is caused by fear of economic insecurity, of losing dominance in a family or business group, of having a "serious" illness (e.g., cancer, strokes, loss of mental faculties), and fear of dying (170).

It was the writer's aim to gather the raw material of the history by haviug the patient tell his story in a natural way, with only such comment or questions from the physician as were needed to indicate that the physician was sympathetically interested in the patient's problems, and to explore those portions of the medical history about which the physician wished to obtain more information. During the elicitation of the history, and subsequently, the greatest care was taken not to suggest to the patient the existence of clinical problems either by interrogation, comment or implication.

It was found that one of the most fruitful sources of information about the patient's emotional makeup was his behavior in the doctor's office. Often, his emotional reactions to the discussion of events, circumstances or persons were exteriorized by his mannerisms, by changes (or lack of appropriate changes) in his facial expression, by alterations in his voice, posture, color, neck artery pulsations, respirations; by aerophagia, by sweating, by crying, by his asides and by his gait. The patient's attitude toward the physician and his assistant sometimes gave valuable clues to certain of the patient's emotional problems. At times, clues to an emotionally charged situation were obtained during the interview; e.g., when the patient in the rnidst of a sentence "forgot" what he was going to say and couldn't "recall" it; when a patient abruptly terminated discussion of a given subject and was unwilling to resume such a discussion; when a patient made a spontaneous and revealing statement, followed by a prolonged discourse intended to correct any "false impression" the examiner may have received from the patient's initial statement; when a patient asked question after question about non-personal or personal medical matters or digressed, talking at great length about emotionally neutral subjects in order to avoid an emotionally painful topic of discussion; when a patient exaggerated or minimized the importance of certain matters in his life history.

At times, a knowledge of patterns of symptom-language and a knowledge of fundamental dynamic patterns of certain psychosomatic disorders were helpful in interpreting the meaning of the patient's psychosomatic complaints and in facilitating the analysis of his central emotional problem (235).

The interpretation of symbolic body language is a valuable, but not infallible, guide for the physician in the identification of a patient's emotional reaction to some problem which he is facing. For example, psychogenically induced, sustained hypertonia of epicranial muscles which results in headache may indicate that the patient is faced with a situation for which there seems to be no satisfactory solution; increased jaw muscle tension may be the sign of determination to perform some difficult or unpleasant task; painful sustained muscle tension of the tongue and throat muscles may indicate that the person has something he wants to say, but can't; pain in the neck may symbolize the patient's preparedness for defensive or aggressive action; pain in the left pectoral muscle may be present when the person has sustained a loss; pressure in the anterior portion of the chest may indicate that the person is sad, grief-stricken or guilty; vaginismus may indicate a defensive reaction against having sexual relations; pain in the right upper extremity may indicate a repressed desire to strike someone; while unilateral thigh and leg pain may indicate that the person wishes to kick someone.

When one elicits data of a personal nature, allowance must be made (even in a non-psychotic patient) for differences between reality and the consciously or unconsciously revised account which the patient gives the physician. Properly interpreted, such conscious or unconscious revision may be more indicative of the patient's emotional problems, prevailing moods and goal-direction than any "true" statement.

After the history was elicited and rapport established, the patient was routinely asked if he wished to talk about any additional matters. It was found that frequently a patient took advantage of this opportunity to reveal those personal, and often most troublesome problems, which he had refrained from mentioning earlier in the interview.

Next, the physical examination and routine laboratory studies were performed. During the physical examination, no comment was made by the physician which might cause the patient anxiety. At no time during the physical examination was the patient led to believe that the examiner was unduly interested in any one part of the examination, or was giving unusual attention to any one part of the patient's body. The patient's apparent reaction to the physical examination was noted.

When the clinical study of the patient was completed, the findings were related factually in terms which the patient could understand. Care was taken not to suggest to the patient physical, emotional or mental disorders which he did not have. He was told how his health compared with what is judged by present-day standards to be "average good health" for his age and sex. He was given an opportunity to ask questions, and when necessary those points which were not clear to him were amplified and rephrased. When correctable disorders were found, if these seemed to be of sufficient clinical importance, appropriate therapy was prescribed. When remediless disorders existed, the examiner always tried to apprise the patient of such findings in a manner which would give rise to the least amount of anxiety and, whenever possible, palliative measures were employed to make the patient more comfortable, to retard the progress of his disorder, or to prevent complications of disease (41). A patient with a remediless disorder was often comforted by the thought that even though there was not an efficacious treatment for his disease, medical progress was such that in time new discoveries might offer him or other sufferers a remedy for the correction of his disorder.

The objectives of the recommended program of medical therapy were outlined for the patient, and the expected response to such therapy was described. If special laboratory studies or additional clinical studies were recommended, the reasons for desiring them were explained to the patient.

Clinical study of the patient continued when he returned for necessary re-examinations, and the physician's initial impressions and conclusions concerning the patient and his health problems were modified as necessary upon further reflection, or when new clinical data became available. The clinical management of joint dysfunction was carried on as previously described, and concurrently other health problems which the patient presented were given appropriate treatment.

PSYCHOGENICALLY INDUCED, SUSTAINED HYPERTONIA OF SOMATIC MUSCLE

Although symptom-producing, localized or generalized hypertonia of somatic muscle may be caused by any etiologic agent which maintains the central excitatory state of the motoneurones innervating the somatic muscle region at their discharge level (36) (194), it is most commonly caused by psychogenically induced, sustained hypertonia of somatic muscle. Psychogenically induced, sustained hypertonia of somatic muscle gives rise to protean clinical manifestations, which may occur in persons with or without joint dysfunction, and with or without obvious arthritis. The localized or generalized patterns of symptoms resulting from such sustained hypertonia of somatic muscle at times may simulate well-known somatic disease patterns, although at other times they may not be typical of any known somatic disease. The patient who experiences articular and non-articular symptoms as a result of psychogenically induced, sustained hypertonia of somatic muscle often seeks medical advice because he mistakenly believes that he is seriously ifi with a somatic disease, and often develops considerable anxiety about the possible meaning of his illness (60) (232) (224) (75).

The syndrome of psychogenically induced, sustained hypertonia of somatic muscle may or may not occur in association with other psychogenic syndromes.

Localized or generalized psychogenically sustained postural hypertonia of somatic musculature often symbolizes the preparedness of the human psychobiologic unit for aggressive or defensive action against extrinsic or intrinsic noxious factors, with sulficiently strong concurrent central inhibition to prevent such completion of the required goal-directed action as would permit, at least temporarily, a satisfying discharge of both the prevailing somatic and psychic tensions (215) (241) (122) (133) (55). In this sense, psychogenically sustained hypertonia of somatic musculature is

operationally a compromise adjustment of the human psychobiologic unit, and represents part of the dynamic pattern of somatization of unresolved conflicts, repressions, resentments, and indecisiveness. Psychogenically sustained somatic muscle hypertonia may in time become habitual as the adaptive response to even the most trivial threat to the patients emotional security, and may include substitutive behavior of various types, replacing inhibited goal-directed action. Even when the initiating psychic tensions responsible for the creation of the sustained somatic muscle hypertonia have been adequately discharged, the retention of hypertonic muscular habit patterns may result in a steady state of bodily discomfort which the patient in time comes to regard as being normal for him. Such a patient will complain only of exacerbations of his bodily discomfort when an old emotional or psychic tension is revived, or when he is exposed to a new emotional or psychic stress (25).

Psychogenically induced, sustained hypertonia of somatic muscle, when sufficiently intense, prolonged or repetitive, may cause the following symptoms and physical signs: fatigue, stiffness, aching, soreness, pain, paresthesias, limitation of joint movement, joint traumatization (including the evolution of the delayed post-traumatic articular syndrome), and, rarely, muscle spasm and muscle tremor. The symptoms and signs of psychogenically induced, sustained hypertonia of somatic muscle may vary from time to time in extensiveness, intensity, duration, repetitiveness, and are likely to be more severe when the environmental temperature is low than when it is high. In general, the more severe the patient's psychic tensions are, the more likely he is to experience irradiation of extensiveness, increased severity and increased awareness of symptoms of bodily discomfort and physical signs resulting directly or indirectly from psychogenically induced, sustained hypertonia of somatic muscle. Once symptom-producing, sustained hypertonia of somatic muscle has been initiated by psychogenic influence, it may be maintained through psychogenic influence and/or through self-exciting lower neuronal reflex arcs actuated by stimulation of afferent end organs within the substance of the contracting muscle itself and within the tendinous origins and insertions of the contracting muscle (137) (55) (242).

The validity of the concept that sustained hypertonia of somatic muscle may cause symptoms of discomfort has been established by others who used special techniques of study, including electromyography and procaine injections into the symptom-producing contracting muscles (242) (243) (217). In patients studied by the writer, the presence of sustained hypertonia of somatic muscle was inferred from characteristic symptoms and signs which were present when a sufficient degree of muscular hypertonia was present, and which were absent when this degree of sustained hypertonia disappeared.

COMMONLY OCCURRING SYMPTOMS OF REGIONALLY SUSTAINED HYPERTONIA OF SOMATIC MUSCLE

Skull Muscles (242). A patient with psychogenically induced, sustained hypertonia of the epicranial muscles may complain of sensations of pulling, heaviness, soreness, tightness, "tight band around the head," scalp discomfort on combing the hair, small painful areas of the scalp, crawling sensations, or headache. These symptoms may occur unilaterally or bilaterally, and may be limited to the occipital, temporal, parietal or frontal regions, or to any combination of these regions.

A patient with psychogenically induced, sustained hypertonia of jaw muscles may complain of aching or pain in the teeth, gums, temporomandibular joints, jaw muscles, and may be aware of clicking sounds or full sensations in his ears. He may find it difficult to open his jaws widely. A person with complete upper and lower dentures may complain "even my false teeth hurt," and a person with partial dentures often complains that he is unable to get a comfortable denture. Patients with sustained hypertonia of jaw muscles often have extensive clinical and x-ray studies of teeth, sinuses and temporomandibular joints, which are usually negative.

A patient with psychogenically induced, sustained hypertonia of facial muscles may complain that his face feels swollen, mask-like, frozen, stiff, tender or tight. He may have burning, crawling, or tingling sensations in the face. A patient who frowns constantly may complain of feeling pressure, discomfort or fullness in the region of the

bridge of the nose and glabella. Symptoms referable to sustained contraction of the orbicularis oris and orbicularis oculi muscles are described below, under Sphincter Muscles.

A patient with psychogenically induced, sustained hypertonia of tongue muscles may complain that his tongue feels sore, "stiff as a board," tired, or "1ike a piece of raw meat." Limited to the anterior and lateral aspects of the tongue there may be lingual pain, burning, tingling, abrasions (with or without secondary infection).

Neck Muscles (242). A patient with psychogenically induced, sustained hypertonia of neck muscles may have in the back of his neck, unilaterally or bilaterally, aching, pain, tightness, drawing sensations, pulling or burning sensations, with or without radiation of pain or discomfort upward toward the base of the skull and downward toward the upper thoracic spine. He may have stiffness and limitation of neck movement. With severe posterior neck pain there may be reflexly sustained, symptom-producing contraction of epicranial and facial muscles. Rarely, sustained hypertonia of anterior and posteri9r cervical muscles may cause symptoms suggestive of cervical radiculitis or of the scalenus anticus syndrome.

Sustained hypertonia of pharyngeal muscles may cause throat symptoms of tightness, soreness or dysphagia. Sometimes the patient will complain of pain limited to the region overlying the most lateral portions of the hyoid bone. Sustained hypertonia of intrinsic laryngeal muscles may cause the patient to complain of vocal fatigability, hoarseness, or poor control of voice (21). A patient with psychogenically induced, sustained hypertonia of pharyngeal, external laryngeal and anterior neck muscles may complain of a 'clump in the throat that can't be swallowed," and he may try to relieve his throat symptoms by repetitive swallowing, by clearing his throat or by brief, non-productive coughing.

Chest Muscles. A patient with or without coronary artery disease who has psychogenically induced, sustained hypertonia of chest muscles may complain of heaviness, pressure, or pain in his anterior chest. He may complain of pain or pressure limited to the left pectoral muscles, and report that these muscles feel bruised, although he can recall no antecedent external injury to this region. He may have pain along the insertion of diaphragmatic muscle into the thoracic wall, usually on the left side (240). Respiratory dysrhythmias (including the hyperventilation syndrome) (46) (209) may be associated with such symptoms of chest discomfort.

Abdominal Muscles. Symptom-producing, psychogenically induced, sustained hypertonia of abdominal muscles occurs rarely, and causes soreness, tenderness or a "bruised feeling" in the contracting muscles.

Thoracic and Lumbar Vertebral Muscles. A patient with psychogenically induced, sustained hypertonia of muscles of the thoracic and lumbar spine may complain of stiffness, pain, limitation of movement of the spine and, rarely, radicular pain (180).

Extremity Muscles. A patient with psychogenically induced, sustained hypertonia of somatic extremity muscles may have discomfort which seems to originate in joints, periarticular structures, tendons, muscles, or any combination of these structures. There may be limitation of articular movement, stiffness, awkwardness in the use of extremities, joint swelling and pain, as well as muscular fatigue, aching, soreness and pain. The patient may complain of dysequilibrium in waliiing when sustained hypertonia of somatic muscle is preponderanily localized to the right or left side of the body. When walking, he may complain of hip and knee pain, and of dragginess, heaviness and stiffness of his lower extremities which results from sustained hypertonia of opposing co-contracting thigh muscles. Tight crossing of thighs in the sitting position may produce immediate or delayed symptoms of discomfort in the thigh muscles at the site of muscle compression. A patient in crossing his knees may exert prolonged pressure on the common peroneal nerve of the overlying leg, producing paresthesias in the sensory distribution of the nerve, and the feeling that the ipsilateral foot is heavy; rarely, complete external peroneal nerve palsy may develop from this source. Some patients

under emotional tension habitually and unconsciously dorsifiex the toes while wearing their ordinary footgear and develop discomfort in the foot and anterior region of the leg.

Sphincter Muscles. Psychogenically induced, sustained hypertonia of somatic sphincter muscles may cause a variety of symptoms, depending on the sphincter region involved; e.g., sustained hypertonia of the orbicularis oculi muscles may give rise to feelings of eyestrain, fatigue, discomfort, paresthesias of the lids, excessive blinking, and cephalgia. Sustained hypertonia of the orbicularis oris muscle (with or without sustained hypertonia of the masseter muscles) may cause labial sensations of tightness, swelling, pain, and paresthesias (particularly, tingling). Tight apposition of the inner surfaces of the lips against the labial surfaces of the teeth may cause the patient to complain of mucous membrane abrasions, with or without secondary infection. Sustained hypertonia of the urinary sphincter muscles may result in difficulty in starting the urinary stream. Sustained hypertonia of the vaginal sphincter muscles, with or without associated sustained hypertonia of other perineal muscles, may give rise to symptoms of vaginismus, vaginal paresthesias, dyspareunia. Sustained hypertonia of the somatic muscle of the anal sphincter (136), with or without associated sustained hypertonia of other perineal muscles, may cause constipation, sensations of incomplete evacuation of stool, rectal pain, and may be responsible for hemorrhoidal symptoms in certain individuals. Coccygeal pain and discomfort may arise from sustained hypertonia of the levator ani and other perineal muscles attaching to the coccyx.

COMMONLY OCCURRING PHYSICAL SIGNS OF PSYCHOGENICALLY INDUCED, SUSTAINED HYPERTONIA OF SOMATIC MUSCLE

Psychogenically induced, sustained hypertonia of somatic muscle is not regularly associated with small or large pupils, tachycardia or bradycardia, excessive sweating, dryness of the mouth or ptyalorrhea, muscle tremors, muscle spasm, nausea, diarrhea, polyuria, or elevation of blood pressure, although a patient with psychogenically induced, sustained hypertonia of somatic muscle who develops acute anxiety may have these somatic and visceral symptoms and sigus of vegetative nervous system imbalance (55) (133) (26) (241).

In general, with symptom-producing, psychogenically induced, sustained hypertonia of somatic muscle, the patient's sitting or standing posture, gait, body movements and mannerisms may appear tense, jerky, and not smoothly integrated. He may hold one shoulder higher than the other; he may keep one arm close to his body when sitting or walking, or he may hold both arms stiffly by his sides when walking. Occasionally he may walk with a stoop; more often, he walks stiff-kneed, with increased lumbar lordosis, increased dorsal kyphosis, with his neck in partial flexion and his head thrust forward (see page 88). Periodically, he may extend, flex and rotate his neck, and rub the back of his neck as if to get relief from neck discomfort. A patient may change his sitting position suddenly, or move his extremities suddenly from one position to another. Another type of patient may cross his thighs tightly when sitting and may sit rigidly, in a slouched position, with marked flexion of his spine in the thoracic and lumbar regions, without moving for long periods of time. One or both fists may be clenched for long periods of time. Tight grasping of objects (pens, pencils, knitting needles, papers, books) in the hands may be noted in persons with sustained hypertonia of somatic muscle.

The patient's facial expression often tends to be fixed, tense and relatively immobile. Smooth, well-integrated and spontaneous transitions from one facial expression to another do not readily occur, and transient grimacing or tic-like movements may precede changes in facial expression. Each time certain emotionally charged subjects are mentioned, the patient may present a stereotyped facial expression, or may blink frequently. A steady state of jaw muscle tension may be noted in some patients; in others, variations in jaw muscle tension may be noted when the mouth is closed and the patient alternately tenses and relaxes his jaw muscles. The patient may swall9w frequently, and at times rhythmically, once every 30 to 120 seconds, and have the usual consequences of excessive aerophagia (96).

In persons with sustained hypertoma of the orbicularis oris muscles, one may see abrasions on the dental surfaces of the lips, with or without secondary infection. There

may be excessive attrition of the teeth; there may be indentations along the lateral margins of the tongue due to prolonged pressure of the tongue against the lingual surfaces of the teeth. In many patients, a leukoplakic line can be seen, usually bilaterally, on the buccal surfaces of the cheeks along the line of closure of the teeth. This may result either from habitual mouth suction, which draws a portion of the buccal inucous membrane within the region of closure of the opposing teeth, or from sustained contraction of the buccinator muscles which push a portion of this membrane within the region of closure of the opposing teeth. Dentures make relatively deep impressions in the supporting mucous membrane as a result of the patient's sustained jaw muscle contraction.

The patient may sigh frequently, and show other types of respiratory dysrhythmia. His voice may be poorly modulated. The rhythm of his speech may be jerky, there may be elision of syllables, and he may stutter (126). He may noisily clear his throat repetitively, or have a recurrent, brief, non-productive cough.

Palpation of somatic muscle in the apparently relaxed patient who has psychogenically sustained hypertonia may or may not reveal increased somatic muscle firmness. In some patients, the very act of palpating somatic muscle causes muscular hypertonicity to disappear for a short time after completion of the palpatory maneuver. In others, palpation increases the imtial degree of hypertonia. Palpation of hypertonic somatic muscle may reveal small, exceedingly tender islands of tissue - trigger spots (217) - and when these are palpated firmly, there is irradiation of pain to the substance of the entire muscle, reflexly to distant muscles, and to the nearest joint. This may be easily demonstrated in persons with habitual hyper tonia of the left pectoral muscles, in whom radiation of discomfort may be to the left shoulder and arm, and to the left side of the neck. Transient psychogenically induced, sustained hypertonia of abdominal muscles, which is usually present only in the recumbent position, may be sufficiently marked to render abdominal examination difficult. Resistance to alternate passive flexion and extension of extremity muscles may be the only physical sign of increased muscle tonus in the apparently relaxed patient who shows no evidence of organic central nervous system disease. The tendon reflexes are usually increased in amplitude, with a short latent period. Rebound contraction is often prominent. Sustained cocontraction in opposing muscles may be of sufficient degree to prevent visible reflex response to tendon tapping, but not palpable reflex contraction. In patients with sustained muscular hypertonia, ankle and patellar clonus, Hoffman and Babinski Sigus were not elicited. Occasionally, swaying in the Romberg position was noted when a patient had sustained hypertonia of somatic muscle, which was relatively greater in one half of the body than the other. Such persons may sway when walking, or may veer n6ticeably to one side.

It may be difficult to examine the palpebral conjunctivae because of marked hypertonia of the orbicularis oculi muscle. Because of sustained hypertonia of the orbicularis oris muscle (and buccinator muscle), lips may be hard to retract with a tongue blade in attempting to expose teeth and gingiva for inspection. In women patients, the introitus may be markedly narrowed by spasm of the vaginal sphincter muscles, and if digital examination is attempted, the patient will have severe pain and discomfort from this maneuver, and pelvic examination will be unsatisfactory. A simple, effective method of causing relaxation of the vaginal sphincter muscles is to ask the patient to relax her perianal muscles, which simultaneously causes vaginal sphincter muscles to relax. Rectal examination may be rendered difficult because of sustained hypertonia of muscles of the anal sphincter and pelvic floor. Spasm of these muscles may cause rectal examination to be a painful procedure for the patient, even though the gloved examining finger is adequately lubricated. Spasm of sphincter muscles may cause hemorrhoidal veins to become prominent.

TREATMENT OF PSYCHOGENICALLY INDUCED, SUSTAINED HYPERTONIA OF SOMATIC MUSCLE

Psychogenic symptoms were exceedingly prevalent in the group of patients studied, and for this reason the writer found it necessary to obtain a working knowledge of psychiatric principles and treatment, through study of available literature (122) (50)

(100) (111) (66) (239) (181) (234) (235) (5) (156) (53) (18) (130) (133) (169) (170) (207) (124) (125) (4) (9) (13) (94) (237), and through careful evaluation of the effects of psychotherapy on patients whose emotional disorders were exteriorized through psychosomatic symptoms. Various methods of treatment of psychogenic syndromes were tested and adapted to the special needs of each patient so that he could be helped to become accustomed to dealing more directly, more realistically and more effectively with his problems of everyday living. The ultimate objectives of reconstructive psychotherapy of patients with emotional disorders and psychosomatic symptoms were to enable the patient to have, in time, freedom from his psychosomatic symptoms, the ability to solve his problems of living with greater efficiency and with more emotional maturity, and a sustained feeling of emotional and physical well-being. Sometimes, these therapeutic objectives could be attained only when the patient was referred to a competent psychiatrist for study and treatment. Sometimes, these therapeutic objectives were unattainable whether an internist or psychiatrist managed the patient's emotional disorder (83). Usually, the internist could successfully manage the patient's psychosomatic problems and attain the desired objectives (235) (156). Some patients were helped to resolve their presenting problems and to have relief of psychosomatic symptoms in a relatively short period of time; in other persons, the same degree of therapeutic success was achieved over a period of several years; and in other individuals, no sustained psychotherapeutic progress was maintained, and any benefit the patient had was chiefly from supportive psychotherapy.

It is usually possible to manage most of a patient's emotional problems which are exteriorized through psychosomatic symptoms, as an integral part of the general medical treatment of his joint dysfunction. A patient is most likely to have complete and lasting relief from a psychosomatic syndrome when the emotional tensions which initiated his psychogenic symptoms and signs are corrected by successful reconstructive psycho-therapy. Usually partial or temporary relief from psychosomatic syndromes may occur when the emotional tensions of the patient are lessened by helpful changes in his external environment or by supportive psychotherapy. In treating a patient with psychogenically induced, sustained hypertonia of somatic muscle, it is sometimes possible to alleviate collateral psychosomatic symptoms without improving the patient's primary emotional disorder (83) (see Case II, page 140), and when the patient feels more comfortable physically, he will have relief from his secondary anxiety concerning the meaning of his psychosomatic symptoms. Under such circumstances, his primary emotional disorder is likely to be more accessible to study and therapy.

Some psychogenic syndromes are collateral to aberrant physiology; e.g., athiaminosis (148), aniacinamidosis (206) (214) (127) (32), menopausal syndrome (121) (33), starvation (80), adrenal gland hypofunction (47). In most instances of this sort, when somatic dysfunction is corrected, the patient will have prompt recovery from collateral psychogenic syndromes; when such somatic dysfunction recurs, there will be a recurrence of the collateral psychogenic syndromes (208).

The patient with psychosomatic symptoms is usually unaware that there is a positive correlation between his emotional reactions to troublesome life situations and his symptoms of bodily discomfort, and he is likely to regard his known emotional problems and physical symptoms as representing two distinct and unrelated conditions. He usually believes that he has his emotional problems "under pretty good control" (169), and seeks medical advice for the relief of his symptoms of bodily discomfort. The patient's failure to perceive the reciprocal relationship between his emotional and psychosomatic problems, and his preoccupation with psychosomatic symptoms seem to represent a type of unconscious adaptive adjustment which he has made to his emotional disorder so that he can maintain emotional homeostasis. Much supportive and preparatory psychotherapy may be required before a patient will be ready to accept reconstructive psychotherapy directed toward the solution of his basic emotional problems. When he attains sufficient insight into the nature of his presenting emotional and psychosomatic problems, he may be enabled to resolve these problems, and when he acquires habitually more mature methods of dealing with his problems of everyday living, he will have no further need for "protective" psychosomatic symptoms, which, consequently, often disappear.

Data concerning the patient's emotional life must be collected and evaluated as objectively and dispassionately as any other clinical data. In analyzing a patient's emotional attitudes and behavior, the internist must learn to recognize positive and negative transference phenomena and to understand their meaning (50) (4) (239). It is relatively easy for a physician to tolerate positive transference reactions as being "natural and proper," and to use them to good therapeutic advantage. However, it is relatively difficult for a physician to tolerate negative transference reactions with equanimity, unless he is prepared to seek the dynamic basis for such emotional behavior, and to manage such negative transference reactions so that they will not unduly hinder attainment of the desired therapeutic goals. Some-times, negative transference reactions are so intense that all attempts at therapy will fail. The physician must also become aware of his own positive and negative counter-transference reactions to a patient, and must learn to modify these as required by the best interests of the patient.

From information elicited during the course of clinical study, the physician can often detect certain basic, repetitive, and emotionally immature patterns in a patient's reactions to various life situations. A patient with emotional disorders and psychosomatic symptoms often has certain prevailing attitudes and derivative defense reactions which cause him to exhibit, without being aware of their existence, certain patterns of emotional behavior which are detrimental to him in his interpersonal relationships. When mental tensions generated directly or indirectly by such pervasive attitudes and their derivative defense reactions become sufficiently severe, the patient may complain of symptoms resulting from psychogenically induced, sustained hypertonia of somatic muscle or other psychogenic syndromes. The patient's awareness of psychosomatic symptoms supersedes his awareness of his primary emotional tensions and produces collateral anxiety concerning the meaning of his psychosomatic symptoms of bodily discomfort.

Before attempting to treat a person with psychosomatic symptoms, the physician should try to understand the background for the development and retention of the patient's emotionally immature attitudes. The physician should appreciate what difficulties the patient is likely to experience in trying to improve these attitudes and gain emotional maturity. Sometimes, by suffering from symptoms or an illness (psychosomatic), the patient may be making the best adjustment he can to a difficult life situation, and when this is so, the physician must realize that the patient may be better off with his symptoms and illness than without them, and in such instances psychotherapy should be chiefly supportive.

Psychotherapy of the patient starts when the patient and physician first meet, and continues throughout the course of treatment of joint dysfunction and associated non-psychogenic syndromes. Often, a patient derives positive psychotherapeutic benefits when his physical and emotional problems are delimited and a broad program of therapy is outlined for him by the physician. Occasionally, during the first visit a patient is able to gain insight into the dynamics of his most pressing emotional problems, and has considerable relief from some of his most pressing anxieties without any directed psychotherapy on the part of the physician other than the sympathetic elicitation of the patient's history, the performance of a careful physical examination, and the subsequent detailed discussion by the physician of the patient's clinical problems and the proposed form of therapy. Sometimes, during the initial clinical study a patient will have almost immediate, and often lasting, relief from psychosomatic symptoms, without insight into the dynamics of his emotional problems, from the reassurance that he has no serious physical disease and that his symptoms of bodily discomfort are of emotional origin. Other patients seem to be uninterested, or disturbed by the objective analysis of their state of health, and either refuse to accept therapy as recommended, or prematurely discontinue therapy.

The technique of treatment of psychosomatic symptoms as described below is adapted to the specific needs of the individual patient with psychogenically induced, sustained hypertonia of somatic muscle.

During the course of the initial clinical study, the physician introduces the patient to the concept of psychosomatic illness by citing commonplace examples of psychosomatic reactions, selected so that they have no direct or immediate application to the patient's emotional disorder and so that they illustrate patterns of psychosomatic reactions which differ from those experienced by the patient. Usually, the patient accepts the validity of the general concept that emotional experiences may be accompanied by symptoms and signs of bodily dysfunction, and that these symptoms and signs are an involuntary consequence and accompaniment of underlying attitudes. Gradually, the physician modifies the discussion so that concepts of psychosomatic illness and treatment are described as they relate more directly to the patient's health problems. In order to give the patient a new point of departure in thinking of his psychosomatic illness as the exteriorization of his emotional tensions, clinical data obtained during the initial study of the patient (including his own description of his reactions to various life situations) are used to illustrate how his emotional behavior has been conditioned by certain pervasive attitudes and the derivative defenses of such attitudes. When this psychosomatic relationship is revealed to the patient in an objective manner by the physician, often the patient can perceive at once that the explanation of his illness is consistent with his subjective experiences and with reality.

The patient is told that his symptoms and signs resulting from psychogenically induced, sustained hypertonia of somatic muscle are not imaginary, and usually are not indicative of serious somatic disease. His psychosomatic symptoms and signs have physiological, anatomical and psychological bases, which are explained to him in simple terms. When the patient is seen in the physician's office to exhibit regionally sustained hypertonia of somatic muscle, this fact is called to his attention, not to embarrass him, but to make him aware that his sustained muscular hypertonia is objectively demonstrable. He is told that his symptoms and signs of psychogenically induced, sustained muscular hypertonia have deflected his attention from his troublesome initiating emotional tensions to his bodily dysfunction, with the result that he has new anxieties concerning the possible meaning of his psychosomatic symptoms and signs.

It is often useful in the exposition of the symptomatology of psychogenically induced, sustained hypertonia of somatic muscle to illustrate to the patient that considerable muscular and periarticular discomfort and pain can be induced by voluntary sustained contraction of somatic muscle. This is done through the use of the procedure described below, or some variation thereof. The patient is asked to abduct his right upper extremity until it is at right angles to the sagittal plane of the body. The palmar surface of his hand faces the floor. Without changing this position of the upper limb, the patient is asked to flex his wrist maximally and to make a fist. He is then asked to elongate his right upper extremity as much as possible, through sustained muscular contraction. Usually, within a few seconds of such sustained muscular contraction, he will experience discomfort and pain in muscles, tendons and periarticular structures of the wrist region, in the muscles of the forearm, and to a lesser extent in the elbow and shoulder regions. He will often be astonished that his voluntary muscle contraction can produce such discomfort. During the period of voluntary sustained maximal contraction of upper extremity muscles, the sites of the patient's uncomfortable or painful sensations are tender to digital palpation, and feel tense. When the sustained muscular contraction is terminated by voluntary relaxation of the upper extremity muscles, the pain in the elbow region promptly lessens in intensity, although there may be some residual discomfort, for some time thereafter.

In addition, it can often be demonstrated to the patient that when he fixes his attention on a given anatomic region, he may have awareness of sensations which he would otherwise not perceive. The patient who has been seated for some time is asked whether or not he is conscious of any sensations from his buttocks pressing against the chair. Invariably, his initial answer is in the negative. He is then asked to concentrate his attention on any sensations which he may feel from his buttocks pressing against the chair. Most patients then report that they feel a sensation of increasing pressure on the buttocks, which reaches a maximum level of intensity which is often distinctly uncomfortable.

Typical examples are cited to illustrate that mental reactions can cause changes in bodily function, and, if these psychosomatic reactions are sufficiently persistent, changes in bodily structure. It is explained that persons with psychogenically induced, sustained hypertonia of somatic muscle are characterized by exteriorizing their emotional tensions through muscular contraction, which symbolically indicates readiness to perform certain defensive or offensive acts, together with sufficient concurrent inhibition to prevent execution of these acts. Persons with the syndrome of psychogenically induced, sustained hypertonia of somatic muscle usually harbor various degrees of repressed hostility, resentment, hatred or rage. When these feelings are directed against an individual whom the patient feels he should obey, respect, admire, love, or be grateful to, he develops guilt feelings which increase his tensions and make his symptoms more severe. The patient must understand that it is not unusual or "wrong" to have such mixed feelings. When the physician can verbalize some of the patient's destructive feelings, or when the patient can talk frankly and freely about his specific problems, without fear of censure by the physician, the patient may have relief from his emotional tensions.

Many patients are able to accept without resistance the idea that their somatic symptoms have a psychogenic basis. However, it is not unusual for a patient to exhibit some degree of resistance to the idea that his illness is a psychosomatic one, and the physician must be aware of the degree of such resistance, so that he can more effectively direct the subsequent course of treatment. When such resistance is mild or moderate, it does not interfere with successful treatment of the patient's psychosomatic disorder, since gradually the patient is able to find confirmation in his daily experiences of the psychosomatic nature of his disorder, and has relief from his psychosomatic symptoms when he is able to acquire and utilize habitually more mature attitudes in the solution of his problems of everyday living.

When a patient shows marked resistance to the suggestion that his ill-ness is a psychosomatic one, discussion of this subject is terminated for the time being by the physician with a reiteration of the nature of psychosomatic disorders in general. Subsequently, the treatment of such a patient is more or less limited to somatic aspects of disorders, and any improvement in the patient's emotional status is more or less fortuitous. In time, a number of patients who showed initially marked resistance to the concept of psychosomatic illness "discover" that they have certain symptoms or an increase in severity of symptoms only when they are exposed to situations which cause emotional tensions, and that their symptoms disappear or become less severe when they are not exposed to these types of situations. After a patient has made such a "discovery," he often becomes more amenable to treatment of his psychosomatic symptoms and underlying emotional disorders, provided that the physician is willing to let the patient take full credit for having made original and informative observations concerning the nature of his psychosomatic problems.

The most substantial relief from psychogenically induced, sustained hypertonia of somatic muscle is afforded by successful treatment of the patient's emotional disorder. However, certain ancillary procedures have been found helpful in giving the patient some relief from his symptoms of psychogenically induced, sustained hypertonia of somatic muscle. The patient may find that his symptoms are lessened when he takes a vacation; when he can interest himself in hobbies, sports, or in civic, social or church activities; or, when there is an external solution to his problems. Patients may be benefited when they are helped to schedule periods of work, exercise, rest and recreation, so that they can expend their physical and emotional energies without developing excessive physical or emotional fatigue. Sometimes, it is necessary to poit out to a patient the deleterious effects of indecision, worry and day-dreaming, and to assist him to establish better habits of thinking. Generalized muscular relaxation can be induced by tepid baths of 20-30 minutes' duration, or by the application of the methods of progressive relaxation (87). The local use of dry heat or the use of hot massive wet dressings applied for 30 minutes several times during the day are helpful in the relaxation of psychogenically induced, sustained hypertonia in certain muscular regions; e.g., posterior cervical region, pectoral muscles, shoulders. Some patients may have relief from symptoms described above only when they do strenuous physical work. In

acute tensional situations, the judicious use of medications (e.g., phenobarbital, belladonna, dexedrine, aspirin) for a limited period of time may be helpful.

SOME EXAMPLES OF PSYCHOGENICALLY SUSTAINED MUSCULAR HYPERTONIA AND ITS RESPONSE TO TREATMENT

CASE FF. Pain in the Neck. A 63-year-old manufacturer was recovering satisfactorily from joint dysfunction in response to adequate niacinamide therapy until he suddenly developed, in the back of his neck, severe pain which radiated into his upper thoracic spine. After this pain persisted for a week, he sought medical advice. He could recall no injury to his cervical spine. Physical examination revealed a decrease in the range of lateral rotation of the neck as compared to previous measurements, and tenderness and firmness of posterior neck muscles. When asked if anything was disturbing him, he stated that a few days before his neck pain started, one of his salesmen had promised his best customer delivery of goods at a date which could not be met and at a price that was far too low. The manufacturer was sure that he would lose his best customer, and was furious with his salesman, whom he planned to fire. "To top it all," he said, "the pain in my neck has been so bad I can't even think straight."

The probable relationship between his emotional tensions and the pain in his neck was explained to him, and in discussing his problems, it was suggested that he might meet with his customer and salesman in order to correct the error. This was done, and a satisfactory solution was evolved. The customer accepted the explanation; the salesman kept his job; and the manufacturer lost his pain in the neck. Three months later, the lateral ranges of neck movement had increased to the level of movement which he had before the joints of the cervical spine were injured by psychogenically induced, sustained hypertonia of neck muscles.

CASE CC. Painful Hands. A 50-year-old unmarried female office manager was recovering satisfactorily from joint dysfunction in response to adequate niacinamide therapy, until she developed painful swelling and stiffness of her fingers, which became progressively worse over a period of four months. She had not performed any unusual physical tasks which might have injured her finger joints. Measurement of the ranges of joint movement showed that all her joints had improved excepting her finger joints, which showed decreased ranges of movement. Her finger joints were markedly swollen, and felt somewhat warm to the touch, but were not red.

When asked if anything was upsetting her emotionally, she said she was afraid she might lose her job. Without consulting her, her superior had employed a young college graduate as assistant office manager, and the patient felt that this girl might replace her as office manager. She thought the new worker was cold, unfriendly and efficient, and was annoyed that the girl had already made several suggestions about improving office procedures. As she talked about the assistant office manager, her hands were clenched tightly. When asked what she would like to do to the girl, she said promptly, "I'd like to shake the living daylights out of her." This confession surprised her. When it was indicated that she looked upon the girl as a threat to her economic and emotional security, she realized that this was so. By clenching her hands tightly she was symbolically attacking her assistant, but actually she was injuring her hands by psychogenically induced, sustained hypertonia of forearm muscles. For temporary relief, she was asked to soak her hands in hot epsom salt solution for 30 minutes three or four times a day, and to try to avoid consciously clenching her hands. For more lasting relief, it would be necessary for her to overcome her intense feelings of hostility toward the girl, which came out of her own fear of insecurity. Probably, she was told, the assistant manager also felt insecure in her new position and wanted to please her manager.

A month later this patient reported that her hands gave her no discomfort, and were not swollen, although they still felt a little stiff. She had decided to take an interest in the assistant office manager and had had lunch with her several times. She thought that the assistant was a "swell person." The fact that she had learned that the girl planned to get married and leave the job in about a year undoubtedly had a great deal to do with her apparent improvement in her previously hostile attitude.

CASE HH. Generalized Sustained Muscular Hypertonia. A 38-year-old assistant factory supervisor suffered for years from constant fatigue and from discomfort and pain in his muscles and joints, which had not responded to medical treatment. Physical examination indicated that he had extremely severe joint dysfunction, and severe generalized sustained hypertonia of somatic muscle. He said that he had difficulty at work in getting along with his superiors, but not with his subordinates. He complained that younger men were promoted over his head, but solaced himself with the thought that this was because he had a mind of his own, and "stood up to" his superiors, who were "down on" him for this reason. When questioned about his childhood, he related that his father was frequently drunk, and was always strict with him, punishing him severely for the most trivial offenses. He was an obedient child, but secretly hated his father.

It was pointed out to the patient that the hostility he had originally directed against his father was unconsciously being directed against all persons in authority. The unconscious fear that his subordinates might feel hostile toward him caused him to be over-kind to them. When he showed marked resistance to the explanation that his psychosomatic symptoms were the result of his basic attitude of repressed hostility toward persons in authority, this subject was dropped and treatment for his joint dysfunction was instituted. He had strong resistance to niacinamide therapy, and did not take his medications as directed - which was interpreted as being another expression of resistance to a person in authority - in this case, the physician.

When he returned for a semi-annual recheck examination, he had become much more aware of his hostility toward his superiors, and was gradually succeeding in improving his attitudes toward them. He had his first promotion in years, which surprised him and encouraged him further. He had lost his articular and non-articular symptoms of generalized psychogenically induced, sustained hypertonia of somatic muscle, and had become completely free from his excessive fatigue and symptoms of severe bodily discomfort. His joint dysfunction had not improved because he felt so well that he thought it unnecessary to take medications of any sort.

CASE II. Excessive Aerophagia. A 44-year-old, unmarried traveling salesman complained of belching, passing much gas rectally, bloating, and a feeling that all the food he ate "turned to gas." His symptoms were most severe on weekends, when he was at home with his widowed mother. Not uncommonly during the weekend, he would wake at 2 A.M. with left upper abdominal pain radiating into his left lower anterior and anterolateral chest and lasting for several hours. He would try to get relief by belching and by taking antacids, but had little relief from these procedures. Enemas tended to make him feel faint and gave him severe abdominal cramps. He was sure that he had serious stomach or heart disease, even though serial electrocardiograms, gall-bladder and gastro-intestinal studies per-formed over a period of years were negative. He denied having emotional problems, would not discuss his relationship with his mother, and said that everything in his life was fine excepting for the gas.

In the office, he was observed to have rhythmic aerophagia at 20-second intervals, and to belch about every five minutes. His submental muscles were palpably tense, but he had no other evidence of psychogenically induced, sustained hypertonia of somatic muscle. His distended abdomen was tympanitic to percussion; borborygmi were heard; the large intestine could be outlined by palpation. Fluoroscopy showed a large gas bubble in his stomach, and relatively little movement of the left leaf of the diaphragm with respiration.

The patient was told that symptoms such as he had were usually the result of excessive aerophagia, emotionally induced. He showed such marked resistance to the idea that his symptoms could be initiated by emotional factors that subsequent discussion was confined to an explanation of the dynamics of the production of somatic symptoms by excessive aerophagia, and a description of the method of inhibiting his frequently recurring swallowing reflex. He was asked to hold between his teeth, for 20 minutes at a time four or five times daily, a cork which was sufficiently large to prevent him from swallowing. If he salivated during this procedure, he was not to swallow his saliva but to

let it drain out of his mouth, catching it in a towel; if he wanted to belch, he might do so, without removing the cork from his mouth.

For one month, he used this method of inhibiting his frequently recurring, rhythmic air-swallowing reflex, and reported that he no longer had trouble with "gas." In the office, at this time he was seen to swallow about once every 10 minutes, and did not belch once during a one-hour period of observation. The patient was pleased to have been "cured" of his trouble, and stated that his "gas" could not have been caused by emotional tensions which were unresolved, since he had the same emotional problems which he had had for years, which could not be relieved in a satisfactory way as long as a "certain person" was alive. He would not discuss this subject further.

Subsequently, he has been seen over a period of several years without any recurrence of his excessive aerophagia. When asked about the status of his emotional problems from time to time in this interval, he says "No change."

(End of Chapter 2. References cited in this chapter are posted at http://www.doctoryourself.com/kaufman11.html)

To go on to Chapter 3, click this link: http://www.doctoryourself.com/kaufman8.html

To go back to Chapter 1: http://www.doctoryourself.com/kaufman6.html