Rheumatoid Arthritis With Special Emphasis On The Psychosomatic Implications

Jeanne Huntley
Carroll College

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RHEUMATOID ARTHRITIS WITH SPECIAL EMPHASIS ON THE PSYCHOSOMATIC IMPLICATIONS

Submitted in Partial Fulfillment of the Requirements for Graduation with Honors to the Department of Psychology at Carroll College, Helena, Montana

Jeanne Huntley
March 31, 1980
This thesis for honors recognition has been approved for the Department of Psychology.

[Signatures]

March 31, 1980
Date
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INTRODUCTION

The name rheumatoid arthritis (RA) was introduced by Dr. Sir Alfred Baring Garrod in the nineteenth century when he was able to distinguish between it and gout by an elevation of uric acid in the latter.\(^1\)

It is possible that many historic figures had RA including Constantine IX, Christopher Columbus, Mary, Queen of Scots, and President James Madison.\(^2\) Additionally, it has been suggested that Hippocrates alluded to the disorder in many of his writings.\(^3\)

Although schools of thought differ as to the existence of RA in earlier times, evidence of articular erosive disease in Indian skeletal remains collected by the Museum of Anthropology in Mexico City may settle this point.\(^4\) Also, further X-ray evaluations of bones from ancient Egyptian pharaohs may offer additional insight into this question.

CLASSIFICATION

RA is just one of over 100 different forms of arthritis. Literally, arthritis means "inflammation of the joint" (from the Greek "arthron", meaning "joint", and "itis", meaning inflammation) and includes five major groups of disorders. These categories include:

1. The infectious cases caused by a specific microorganism;

2. cases that are possibly infectious but of unproved
etiology;
3. cases representing degenerative forms of joint disease;
4. cases in which the arthritis results from direct trauma to the joint; and
5. cases of metabolic arthritis (e.g., gout).

For unity and clarification of the many types of arthritis, the American Rheumatism Association in 1963 recommended the following table for general use.

Nomenclature and Classification of Arthritis and Rheumatism

I. Polyarthritis of unknown etiology
   A. Rheumatoid arthritis
   B. Juvenile rheumatoid arthritis (including Still's disease)
   C. Ankylosing spondylitis
   D. Psoriatic arthritis
   E. Reiter's syndrome
   F. Others

II. "Connective tissue" disorders (acquired)
   A. Systemic lupus erythematosus
   B. Progressive systemic sclerosis (scleroderma)
   C. Polymyositis and dermatomyositis
   D. Necrotizing arteritis and other forms of vasculitis
      1. Polyarteritis nodosa
      2. Hypersensitivity angiitis
3. Wegener's granulomatosis
4. Takayasu's (pulseless) disease
5. Cogan's syndrome
6. Giant cell arteritis (including polymyalgia rheumatica)

E. Amyloidosis
F. Others

III. Rheumatic fever

IV. Degenerative joint disease (osteoarthritis, osteoarthrosis)
   A. Primary
   B. Secondary

V. Nonarticular rheumatism
   A. Fibrositis
   B. Intervertebral disk and low back syndromes
   C. Myositis and myalgia
   D. Tendinitis and peritendinitis (bursitis)
   E. Tenosynovitis
   F. Fasciitis
   G. Carpal tunnel syndrome
   H. Others

(see also Shoulder-hand syndrome, VIII, C)

VI. Diseases with which arthritis is frequently associated
   A. Sarcoidosis
   B. Relapsing polychondritis
   C. Schönlein-Henoch purpura
   D. Ulcerative colitis
E. Regional enteritis
F. Whipple's disease
G. Sjogren's syndrome
H. Familial Mediterranean fever
I. Others
    (see also Psoriatic arthritis, I, D)

VII. Associated with known infectious agents

A. Bacterial
   1. Gonococcus
   2. Meningococcus
   3. Pneumococcus
   4. Streptococcus
   5. Staphylococcus
   6. Salmonella
   7. Brucella
   8. Streptobacillus moniliformis (Haverhill fever)
   9. Mycobacterium tuberculosis
  10. Treponema pallidum (syphilis)
  11. Treponema pertenue (yaws)
  12. Others
      (see also Rheumatic fever, III)

B. Rickettsial

C. Viral
   1. Rubella
   2. Mumps
   3. Viral hepatitis
   4. Others
D. Fungal
E. Parasitic

VIII. Traumatic and/or neurogenic disorders
A. Traumatic arthritis (the result of direct trauma)
B. Neuropathic arthropathy (Charcot joints)
   1. Syphilis (tabes dorsalis)
   2. Diabetes mellitus (diabetic neuropathy)
   3. Syringomyelia
   4. Myelomeningocele
   5. Congenital insensitivity to pain (including familial dysautonomia)
   6. Others
C. Shoulder-hand syndrome
D. Mechanical derangement of joints
E. Others
   (see also Degenerative joint disease, IV; Carpal tunnel syndrome V, G)

IX. Associated with known or strongly suspected biochemical or endocrine abnormalities
A. Gout
B. Chondrocalcinosis articularis ("pseudogout")
C. Alkaptonuria (ochronosis)
D. Hemophilia
E. Sickle cell disease and other hemoglobinopathies
F. Agammaglobulinemia (hypogammaglobulinemia)
G. Gaucher's disease
H. Hyperparathyroidism
I. Acromegaly
J. Thyroid acropachy
K. Hypothyroidism
L. Scurvy (hypovitaminosis C)
M. Hyperlipoproteinemia type II (xanthoma tuberosum and tendinosum)
N. Fabry's disease (angiokeratoma corporis diffusum or glycolipid lipidosis)
O. Hemochromatosis
P. Others
   (see also inherited and congenital disorders, XII)

X. Neoplasms
   A. Synovioma
   B. Primary juxta-articular bone tumors
   C. Metastatic malignant tumors
   D. Leukemia
   E. Multiple myeloma
   F. Benign tumors of articular tissue
   G. Others
      (see also Hypertrophic osteoarthrophy, XIII, I)

XI. Allergy and drug reactions
   A. Arthritis due to specific allergens (e.g., serum sickness)
   B. Arthritis due to drugs
   C. Others
      (see also Systemic lupus erythematosus, II, A, for Drug-induced lupus-like syndromes, e.g., hydralazine and procaineamide syndromes; Hyper-
sensitivity angiitis, II, D, 2)

XII. Inherited and congenital disorders
A. Marfan syndrome
B. Homocystinuria
C. Ehlers-Danlos syndrome
D. Osteogenesis imperfecta
E. Pseudoxanthoma elasticum
F. Cutis laxa
G. Mucopolysaccharidoses (including Hurler's syndrome)
H. Arthrogryposis multiplex congenita
I. Hypermobility syndromes
J. Myositis (or fibrodysplasia) ossificans progressiva
K. Tumoral calcinosis
L. Werner's syndrome
M. Congenital dysplasia of the hip
N. Others
   (see also Arthropathy associated with known biochemical or endocrine abnormalities, IX)

XIII. Miscellaneous disorders
A. Pigmented villonodular synovitis and tenosynovitis
B. Behcet's syndrome
C. Erythema nodosum
D. Relapsing panniculitis (Weber-Christian disease)
E. Avascular necrosis of bone
F. Juvenile osteochondritis
G. Osteochondritis dissecans
H. Erythema multiforme (Stevens-Johnson syndrome)
I. Hypertrophic osteoarthropathy
J. Multicentric reticulohistiocytosis
K. Disseminated lipogranulomatosis (Farber's disease)
L. Familial lipochrome pigmentary arthritis
M. Tietze's syndrome
N. Thrombotic thrombocytopenic purpura
O. Others

This table illustrates that RA is distinctly different from similar forms of arthritis.

Although the purpose of this paper is to focus on RA, it seems important to briefly investigate other types of arthritis that the general public frequently confuses with RA. The précis on the following two pages is an attempt at this process.
<table>
<thead>
<tr>
<th>Type</th>
<th>Characteristics</th>
<th>Usual Age of Onset</th>
<th>Sex Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatoid arthritis (RA)</td>
<td>Usually starts in hands or feet. Joints involved symmetrically. Many joints affected. Destructive, crippling.</td>
<td>35 to 45.</td>
<td>Women affected two to three times more than men.</td>
</tr>
<tr>
<td>Juvenile RA (Still's disease)</td>
<td>Knees, elbows often affected. Generalized illness, usually with fever at onset.</td>
<td>Infancy to late teens.</td>
<td>Sex preference not notable.</td>
</tr>
<tr>
<td>Osteoarthritis (degenerative joint disease)</td>
<td>Affects weight-bearing joints most. Wear-and-tear damage to joints.</td>
<td>Middle years or earlier.</td>
<td>More common in men before 45, in women after 45.</td>
</tr>
<tr>
<td>Gout (gouty arthritis)</td>
<td>&quot;Chemical&quot; arthritis with high blood uric acid level and deposits of urate crystals in or near affected joints. Exceedingly painful acute attacks in single joints.</td>
<td>Middle years or earlier.</td>
<td>Men more than premenopausal women; after menopause men, women equally.</td>
</tr>
<tr>
<td>Pseudogout</td>
<td>Another &quot;chemical&quot; arthritis due to calcium-pyrophosphate deposits in cartilage of joints. Hands and feet often affected. Can mimic gout.</td>
<td>Middle years or earlier.</td>
<td>Men and women equally.</td>
</tr>
<tr>
<td>Traumatic arthritis</td>
<td>Results of acute injury to joint. May have bleeding into joint or effusion of joint fluid.</td>
<td>Any age.</td>
<td>Men and women equally.</td>
</tr>
<tr>
<td>Septic arthritis</td>
<td>Due to bacterial infection in a joint. Bacteria may enter from outside (skin break) or invade via the bloodstream. Common bacteria: staph, TB, typhoid, gonorrhea.</td>
<td>Any age.</td>
<td>Men and women equally.</td>
</tr>
<tr>
<td>Reiter's syndrome</td>
<td>An arthritis, usually on one side, associated with inflammation of the eye and the urinary tract.</td>
<td>Young adulthood.</td>
<td>Mostly men; rarely seen in women.</td>
</tr>
<tr>
<td>Ankylosing spondylitis (Marie-Strumpell disease)</td>
<td>A progressive arthritis involving inflammation and then &quot;freezing&quot; of the lower spine and sacroiliac joints. Upper spine may later be involved.</td>
<td>10 to 30 Uncommon to start later.</td>
<td>90 percent of patients are men.</td>
</tr>
<tr>
<td>Neurogenic joint disease (Charcot's joints)</td>
<td>A destructive arthritis, usually of the hips, knees, or feet, caused by nervous system illness. Often occurs in advanced spinal cord syphilis, but may arise in diabetes, spinal cord defects or any other conditions.</td>
<td>Any age.</td>
<td>Men and women equally.</td>
</tr>
<tr>
<td>Arthritis in other illnesses</td>
<td>Can occur in simple illnesses like flu (joint aching) or serious illnesses like acute rheumatic fever, lupus erythematosus and others.</td>
<td>Depends on the general illness.</td>
<td>Depends on the general illness.</td>
</tr>
<tr>
<td>Treatment</td>
<td>Remarks</td>
<td>Prognosis</td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------------------------------------</td>
<td>----------------------------------------</td>
<td>-----------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Anti-inflammatory (AI) drugs; special drugs; physical measures; joint-replacement surgery when indicated.</td>
<td>Not hereditary. Long, chronic course.</td>
<td>No cure, but can be controlled with early, vigorous treatment.</td>
<td></td>
</tr>
<tr>
<td>AI drugs; special drugs; physical measures.</td>
<td>Often acute, feverish onset.</td>
<td>For youngsters, 75 to 80 percent complete remission. Rate poorer for late teenage onset.</td>
<td></td>
</tr>
<tr>
<td>AI drugs; cortisone in individual joints; physical measures; weight reduction; joint-replacement surgery when indicated.</td>
<td>Cause unknown. Heavy physical labor predisposes. Joint stiffening, nighttime aching common. Chronic progressive course.</td>
<td>No cure, but control is moderately good with diligent treatment.</td>
<td></td>
</tr>
<tr>
<td>Reduction of blood uric acid levels, then continuing medical maintenance; special drug treatment for acute attacks.</td>
<td>Underlying cause is an inborn metabolic defect. remission on proper medication; Not believed hereditary. continuing therapy to maintain the remission.</td>
<td>Excellent chance for complete remission. Rate poorer for late teenage onset.</td>
<td></td>
</tr>
<tr>
<td>AI drugs; cortisone injected into individual joints.</td>
<td>Occurs intermittently.</td>
<td>Excellent for control, with no crippling effect, though it does not generally clear up completely.</td>
<td></td>
</tr>
<tr>
<td>Physical measures (ice pack at first, then moist heat); rest joint; check to rule out fracture; aspirin for pain; drainage of joint.</td>
<td>As much an injury as an arthritis. Needs time to heal.</td>
<td>Excellent outlook for complete restoration to normal.</td>
<td></td>
</tr>
<tr>
<td>Identify bacteria; then specific antibiotic treatment.</td>
<td>Untreated gonorrhea is most common cause. Bacteria can destroy joint. Knees most commonly affected.</td>
<td>Excellent chance for cure under proper treatment.</td>
<td></td>
</tr>
<tr>
<td>Same as for rheumatoid arthritis. Antibiotics often used initially.</td>
<td>May be caused by a virus or bacteria-like organism.</td>
<td>May subside permanently but often recurs off and on indefinitely.</td>
<td></td>
</tr>
<tr>
<td>AI drugs; physical measures including regular back exercises; mild pain medication as needed.</td>
<td>Possibly due to hereditary factors. Can impair breathing when upper spine involved.</td>
<td>Tends to smoulder and progress for 10 to 20 years, then may quiet down, but joint damage does not improve.</td>
<td></td>
</tr>
<tr>
<td>Bracing or fixation of badly damaged joints; treatment of underlying neurological condition when possible.</td>
<td>Proof that we need unimpaired nervous system and reflexes for joints to work properly.</td>
<td>Poor unless condition can be discovered early and underlying neurological condition treated.</td>
<td></td>
</tr>
<tr>
<td>Symptomatic while the general illness is being treated.</td>
<td>Joint pain or inflammation can occur as part of many illnesses ranging from virus to cancer.</td>
<td>May clear up completely and permanently when underlying illness is successfully treated.</td>
<td></td>
</tr>
</tbody>
</table>
To define RA even more specifically, the American Rheumatism Association (ARA) proposed eleven symptoms, signs and laboratory examination criteria that are especially relevant for a positive RA diagnosis. Five or more definite criteria indicate that a person has a 70% chance of having RA.

Rheumatoid Arthritis Diagnostic Criteria (ARA 1958)

1. Morning stiffness
2. Pain on motion or tenderness in at least one joint
3. Swelling (soft tissue thickening or fluid, not bony overgrowth alone)
4. Swelling of at least one other joint
5. Symmetrical joint swelling with simultaneous involvement of the same joint on both sides of the body. Terminal phalangeal joint involvement will not satisfy the criterion.
6. Subcutaneous nodules over bony prominences on extensor surfaces
7. Roentgenographic changes typical of RA (which must include at least bony decalcification localized to or greatest around the involved joints and not just degenerative changes
8. Positive agglutination (anti-gammaglobulin) test
9. Poor mucin precipitate from synovial fluid (with shreds of cloudy solution)
10. Characteristic histologic changes in synovial membrane
11. Characteristic histologic changes in nodules
Morning Stiffness

Since Lansbury first drew attention in 1956 to the possibility of quantifying morning stiffness, there has been a growing awareness of its value as an index of rheumatoid activity. It appears in 97% of untreated RA patients and lasts for approximately 3½ hours after rising. As patients go into remission, the duration of morning stiffness steadily declines.

Pain on Motion or Tenderness in at Least One Joint

Pain, being a subjective phenomena, is hard to quantify. However, several authors have reported on a count of aspirin or other analgesic tablets needed per day, as a measure of pain. In studies done by Lansbury data indicated that as the patient improved, the aspirin intake was lowered, and vice versa.

Swelling

Researchers indicated that RA may be initiated by effusions from the synovial sac causing an inflammation of the synovial lining. Initial swelling due to synovitis (inflammation of the synovial lining) is usually most apparent over the extensor surfaces, "where the articular capsule is more distensible." Additionally, swelling is usually spindle-shaped when first noticed in the extremities. Symmetrical Joint Swelling

Any synovial joint of the body may be involved, but especially the knees and small joints of the hands, wrist, and feet. This process may begin with an asymmetric distribution, but gradually becomes symmetric.
Subcutaneous Nodules

Approximately 20 to 25% of all patients with RA manifest such nodules eventually. Topically, these rheumatoid nodules may be mistaken for similar nodules caused by gout, sebaceous cysts, basal cell carcinoma, and ganglions of the hand. However, it is histologically distinct and thus easily diagnosed with laboratory analysis. These nodules are most common over the olecranon process of the ulna, less common over the finger joints and least common over the joints of the foot.

Roentgenographic Changes Typical of RA

Typical X-rays of a person with RA indicate soft tissue swelling, narrowing of the interosseous space, and reduced bone mass.

Narrowing of the interosseous space reflects cartilage loss. This process is uniform and suggests an enzymatic degradation of cartilage. In some cases, a widening of the joint may be seen, reflecting "capsular distention by hypertrophied synovium and excessive fluid."

The reduction of bone mass associated with RA is brought about by the resorption of cancellous bone which produces demineralization. Articular erosions are a form of bone resorption along the surface of subchondral compact bone. They are the most distinctive radiologic manifestation of RA and their radiographic appearance provides an important parameter for assessment of activity of disease and response to therapy.

Erosions generally begin at joint areas most closely connected with the synovial fluid. As the erosive process progresses, greater destruction and resorption of the bone are seen,
which produces a whittled, or alternatively, cavitated appearance. The later are termed "pocketed erosions".\textsuperscript{18}

**Positive Agglutination Test**

The term rheumatoid factor evolved from the observations of Waaler, who noted that serum from a high proportion of patients with rheumatoid arthritis agglutinated sheep erythrocytes sensitized with rabbit antibodies.\textsuperscript{19} Seropositivity of patients with RA is usually defined by the latex fixation test or red cell agglutination test. These assays reflect the presence of IgM-rheumatoid factors. The presence of rheumatoid factors is not unique to RA, and a positive test for these antibodies should never be used as the sole criterion for the diagnosis of RA.\textsuperscript{20} Additional evidence suggests that only 70\% of persons afflicted with RA show seropositivity (positive rheumatoid factor).\textsuperscript{21}

**Poor Mucin Precipitate from Synovial Fluid**

Normal synovial fluid does not clot as it lacks fibrinogen as well as prothrombin, factors V and VII, tissue thromboplastin, and antithrombin. But most pathologic fluids do clot, and the rapidity of clotting and the size of the clot is roughly proportional to the severity of inflammation present.\textsuperscript{22}

Clots are categorized by a "mucin clot" test in which supernatant from a centrifuged specimen is transferred to a clean glass tube. A few drops of glacial acetic acid are placed on the surface of the fluid. The heavier acid settles to the bottom of the tube leaving a dense white precipitate of protein hyaluronate.\textsuperscript{23}
The mechanism for the failure of most fluids from inflamed joints to form good mucin clots is not well understood.24

Characteristic Histologic Changes in Synovial Membrane

As time passes the synovial membrane of the RA becomes characteristically hypertrophied, edematous, and inflamed. The lining cells become multilayered, occasionally reaching a depth of 10 to 20 cells with multinucleated giant cells interspersed among them. Additionally, lymphocytes and plasma cells infiltrate the synovium.25

CLINICAL FEATURES

In the majority of adults, RA begins "insidiously", with gradual appearance of stiffness (a subjective sensation of lack of free movement, most notable after a period of rest), pain and signs of joint inflammation involving a few joints. This is commonly accompanied by or preceded by fatigue, fever, loss of appetite, diffuse myalgia (pain in one or more muscles), and malaise. The hands and feet are commonly affected initially, but any synovial joint may be involved at any time.25

Course of Disease

The natural history of RA is extremely variable, with some patients (10-15%) having very mild disease of brief duration and some (10%) at the other extreme experiencing progressive disease involving many joints. Most persons with RA experience gradual increases and decreases in the extent and severity of joint inflammation over many years.27

Physical Findings

Patients with RA may have fever related to their disease.
Fever occurs in 2% of ambulatory patients in the morning. Recent surveys suggested that 20% of hospitalized RA patients, whose hospitalization may indicate a more severe disease, have average elevated temperatures of 37.5°C. Further evidence reveals that the occurrence of fever in RA patients is correlated with active disease. 28

Tendon and tendon sheaths show signs of inflammation in the RA patient. Tendon involvement is very common about the hands, wrists, feet and ankles. 29

A person with RA usually experiences joint deformities. These include contractures, which are permanent losses of motion of a joint, ankylosis, which is complete loss of motion of a joint, subluxation which is partial displacement of a bony part of a joint, and dislocation and angular deformities. 30

No deformities are unique to RA. Several deformities of the hand are common, however, in longstanding disease, including ulnar drift of the fingers (angular deformity at metacarpophalangeal (MCP) joints with angulation of fingers to the ulnar side of the hand); boutonniere deformity (combination of proximal interphalangeal joint (PIP contracture preventing full extension and hyperextension of distal interphalangeal (DIP) joint); swan-neck deformity (fixed hyperextension of PIP joint with DIP joint in position of flexion). 31

The common deformities just described are not seen early in the course of RA. They are the result of damage to tissues caused by the inflammatory process, and therefore appear only after disease has been present for some time. 32
Muscle weakness in patients with RA is common. It may be due to pain causing inhibition of muscle contraction or to prolonged impaired use of the muscle. In some patients with RA rather severe weakness of muscle occurs along with atrophy, which is poorly understood. In nearly 100% of RA patients with active joint inflammation there is more measurable decrease of grip strength, the variations of which correlate with changes in severity of joint inflammation.\(^{33}\)

**Laboratory Findings\(^{34}\)**

- **Blood:** Approximately half of the patients with RA have mild to modest anemia. The white blood count (WBC), however, is usually lowered.
- **Urinalysis:** No abnormalities are associated with RA.
- **Erythrocyte sedimentation rates (ESR):** The ESR is elevated above normal in almost all RA patients with active joint inflammation, and the degree of elevation correlates well with the severity of disease. It is, however, a non-specific test, having no value in the diagnosis of RA.

The rate of sedimentation of erythrocytes is related to the degree of roucoux formation. This is enhanced by increases in the concentration of large asymmetric molecules in plasma, especially fibrinogen and alpha-2 globulin. Increases in fibrinogen and alpha-2 globulin occur nonspecifically in the presence of inflammation by diverse causes. In RA, gamma globulin levels are often increased as well.

**Synovial Fluid Findings\(^{35}\)**

WBC's are increased by 40-60% in the synovial fluid.
EXTRA-ARTICULAR FEATURES OF RHEUMATOID DISEASE

Persons who have had well-established RA for several years are likely to develop additional complications.

One such complication is Sjogren's syndrome, which is an inflammatory process involving lacrimal and/or salivary glands. Impaired secretory function is the consequence of this syndrome.36

Another complication that may evolve out of long-term RA is Felty's syndrome, which is characterized by enlargement of the spleen, leukopenia and pigmented spots on the skin of the lower extremities.37

Pericarditis and pleuropulmonary disease are found 40% of the time at autopsies of RA persons. However, these afflictions are noted less clinically.38

Finally, necrotizing vasculitis, the most serious "manifestation extra-articular disease", often involves skin, muscles, peripheral nerves, and at times, the vascular system. It causes ulceration or gangrene of skin, peripheral neuropathy and severe weakness. A less serious form of vasculitis occurs somewhat more frequently and involves the skin of the distal legs. This type of vasculitis causes chronic leg ulcers.39

TREATMENT

Drugs

Aspirin is the mainstay of therapy for mild to moderately severe RA. Patients usually are prescribed approximately 3.6 gm per day, however, 4.8 grams can be tolerated in some instances.40 The principle side effects of aspirin involve
the gastrointestinal tract. Peptic ulcers may occur, and slight fecal blood loss can be expected in the majority of patients treated with aspirin.\textsuperscript{41}

Periods of rest during the day are helpful. Complete inactivity should be avoided, however, unless severe disease exists with markedly painful swollen joints. Lightweight splints or shells may be used to assist in immobilizing the wrist and knee joints, relieve muscle spasms, and insure correct position, especially at night. Complete immobilization of a joint with plaster splints for periods of more than four weeks may lead to increasing joint stiffness and muscle atrophy which favor the development of contractures and ankylosis.\textsuperscript{42}

The majority of patients will respond with improvement to these conservative measures carried out over the course of at least two months. However, if these conditions continue to exist, chloroquine or hydroxychloroquine may be prescribed. The maximum amount of chloroquine prescribed is usually 250 mg and the maximum amount of hydroxychloroquine prescribed is 200 mg.\textsuperscript{43}

The principle side effects of the two drugs are nausea, skin eruptions, and visual disturbances. Visual disturbances are caused by the accumulation of either one of the drugs in the cornea, which causes dimness of vision or a sensation of halos.\textsuperscript{44}

Another treatment used for persons with RA is the injection of gold salts. Most authorities consider gold to have a definite place in the course of treatment, and con-
trolled studies provide some support for this view. The advocates of chrysotherapy generally recommend that gold be started only after a preliminary period of observation which indicates that the disease activity is inadequately controlled by more conservative measures. The decision to use chrysotherapy commits the patient to a long-term course of treatment. In this treatment, the patient receives a test intramuscular injection containing 10 mg of gold. If there are no outward or local reactions, this is followed in a week by 25 mg, and thereafter weekly injections containing 50 mg of gold. Improvement is seldom noted until approximately 200 to 400 mg of gold have been given. If no change occurs after the administration of a full gram of gold, the injections are stopped. If, however, there is significant improvement the patient continues to receive "maintenance" injection containing 50 mg of gold at approximately monthly intervals. The manner in which gold produces a therapeutic effect is not known; possibly it is related to inhibition of lysosomal enzymes.\textsuperscript{45} The principle side effects of gold treatment are skin rashes, stomatitis, and ulcers. Additionally, marrow depression and renal damage have been noted.\textsuperscript{46}

Severe forms of RA that do not respond to the above-mentioned treatment may respond positively to corticosteroids. The most common drugs used are prednisone and cortisone and the maximum dosage is 10 mg/day. When injected into joints in small doses, steroids can markedly reduce pain and inflammation within hours. However, if taken orally for long periods of time, the side effects can be devastating.\textsuperscript{47}
principle side effects of oral treatment are weight gain, moon faces of Cushing's syndrome, acne, ecchumoses, hirsutism, diabetes, peptic ulcers, osteoporosis, cataracts, mental disturbances, and activation of tuberculosis and high blood pressure.\(^{48}\)

Phenylbutazone and indomethacin may temporarily suppress joint inflammation in RA, but these drugs are not generally recommended for long-term uses because of the frequency and severity of side effects and the lack of sustained benefits. The principle side effects of this treatment includes rash, peptic ulcers, edema, and blood dyscrasias. Additionally, headaches and light-headedness are common problems to patients on indomethacin.\(^{49}\)

Other drugs prescribed for RA include antimalaria drugs, penicillamine, and immunosuppressive drugs.

The principle side effect of an antimalaria drug such as chloroquine is retinal damage.\(^{50}\)

The principle side effects of penicillamine are kidney dysfunction, gastrointestinal distress, and aplastic anemia.\(^{51}\)

The principle side effects of immunosuppressive drugs such as chlorambucil, methotrexate and cyclophosphamide are marrow suppression, susceptibility to infection, liver damage, cystitis and carcinoma of the urinary bladder, and mucous membrane lesions.\(^{52}\)

Joint surgery procedures are progressing rapidly, but debate continues about the best techniques and timing for surgery, rebuilding or replacing joints with artificial ones, and even the appropriateness of surgery for any single patient.\(^{53}\)
Although surgery may not relieve pain, it may save another joint on which the patient has put undue stress to compensate for lost function in the arthritic joint.  

Typical of recent progress is a new technique, which is less drastic than total hip joint replacement. This technique was performed on approximately 100,000 persons in the U. S. last year, and consisted of replacing the head of the femur with a metal cap. In this instance, the socket of the pelvic bone is lined with polyethylene and the metal-to-polyethylene joint surface permits relatively stress-free movement.

Physical Therapy (See Appendix 1)

Physical therapy (PT) is also utilized in the treatment of patients with RA. PT is intended to:

1. Maintain range of motion;
2. prevent disuse and atrophy of muscle;
3. minimize deformity;
4. provide proper rest;
5. minimize excessive articular trauma.

A PT's instructions include having the patient move all of his joints through a full range of motion once daily. This is most easily accomplished after the period of initial morning stiffness has subsided or shortly after or during a hot tub bath or shower. Instruction as to proper positioning of joints during rest or sleep is equally important in preventing contracture. The tendency to place pillows under a painful knee joint is a major contributing factor to the development of contractures and must be actively discouraged.
The patient is instructed to sleep in a position with knees and elbows fully extended and with the neck and wrists in a near neutral position.\textsuperscript{56}

Joint trauma can be minimized by the use of splints. Additionally, a soft cervical collar with Velcro straps to hold the neck in mid-position minimizes the pain when RA involves the cervical spine.\textsuperscript{57}

**POSSIBLE CAUSATIVE FACTORS**

Many etiologies have been postulated for RA, however, none have been completely substantiated by research data. It is still unclear whether RA is one disease with multiple causative factors or a "symptom complex" produced by a single causative factor.\textsuperscript{58}

**Infection**

It has been hypothesized that infectious pathogens may play a major role in RA. Current research indicates that chronic arthritis accompanies certain natural or experimentally induced bacterial infection in animals. For example, features similar to rheumatoid synovitis are seen in streptococcal infections of rabbits, in mycoplasmal and erysipelothrix infections in mammals and birds, and in mycobacterium infections in rats.\textsuperscript{59}

Even though the symptoms are similar, researchers have failed to isolate any of the above infectious agents from human material. However, Person and Sharp have initially found organisms in animals with RA induced by erysipelothrix. The organisms, though, disappeared when chronic inflammatory synovitis was established.\textsuperscript{60}
A possible viral causation of RA has been vigorously pursued. Direct viral identification, either through isolation or visualization by electron microscopy, has been unrewarding. Also, an increase in any antiviral antibody has not been detected in serum, synovial fluid or eluates from rheumatoid synovial tissues. However, the search goes on and researchers in this area suspect that RA may be caused by the incorporation of viral genome into the nucleic acid of host cells.  

Metabolic and Biochemical Abnormalities

A finding unique to RA is the low plasma concentration of the amino acid histidine. Additionally, explanted rheumatoid synovial cells show increased hyaluronic acid synthesis, glucose consumption, and lactate production. It has been suggested that the abnormalities are produced in response to a "connective tissue activating peptide" that is utilized in the enzymatic degradation of tissue.

Autoimmunity

It has been suggested that RA may be an autoimmune disorder with the combining elements of:

1. Genetic control possibly related to the activity of specific immune response genes;
2. immunologic control, which may be exerted by regulatory T-dependent lymphocytes;
3. possible viral influences;
4. sex hormone modulation of immune regulation.  

Genetic Factors

Although it was once thought that environmental factors
played a greater role in RA than genetic factors, it has recently been demonstrated that HLA-Dw4, a determinant in the D locus occurs three to four times more often in adults with RA than in non-rheumatoid controls.\textsuperscript{65}

Additionally, in the mouse, the immune response genes are located close to the HK locus on the 17 chromosome. Because of the similarities in the organization of the human HLA region, it is likely that the immune response genes will reside in an area adjacent to the HLA-D locus.\textsuperscript{66}

Although current data seems to indicate that RA may be caused by a genetic predisposition coupled with infectious agents, the search for the complete causative factor must also include a psychological investigation.

Development of Possible Psychological Causation

**Psychic Syndromes Expressed as Musculoskeletal Symptoms**

It is well-documented that musculoskeletal pain without other disease symptoms may be a mild form of disease or may arise because of psychological disturbances. Patients in the category of psychological disturbances usually complain of pain in joints and muscles. They experience soreness or tenderness of the involved parts. Stiffness is a frequent complaint and increased muscle tension may be found. Examination, however, shows no swelling or other objective signs of joint inflammation. Further study usually reveals that their psychological distress has manifested itself in the past in numerous somatic symptoms and is more widespread than that arising from the present problem alone.\textsuperscript{67}

The development of such symptoms has been accounted
for by two mechanisms. One is known as a conversion reaction and the other consists of a psychophysiologic process in which symptoms are thought to arise from increased muscle tension.\textsuperscript{68}

**Conversion Reaction**

It is thought that a patient who has conflict about expressing by word or action some wish, idea, or feeling that is distasteful or unacceptable to his or her moral standard substitutes a bodily symptom.\textsuperscript{69} This symptom may represent an imagined consequence or punishment of a particular distasteful reaction. For example, a patient's neck pain may represent intense anger directed toward a close figure who is demanding and burdensome. The pain is a figurative translation of the wish to inflict suffering and also of punishment for having such a wish. Such a conversion "pain in the neck" may also represent the patient's perception of the other person.\textsuperscript{70}

Clinicians have observed that conversion reactions may also take the form of symptoms from a previous illness or the observation of illness in others. It is well-documented that a patient whose mother had crippling RA may identify with and take on the pain of such a loved person without demonstrating the corollary signs of the disease.\textsuperscript{71}

The patient with a conversion reaction describes symptoms in a vague, imprecise fashion with descriptive elaboration which differs from familiar patterns of disease. The accompanying emotional expression may be either overconcerned or unusually stoic. The past history may include illnesses
which were clearly conversion reactions. Exploration of the current life setting frequently brings to light recent stress-inducing circumstances. Additionally, conversion reactions may coexist with symptoms of an organic nature as in patients with unusually large amounts of pain in otherwise mild conditions. In a true conversion reaction, however, placebo therapy often relieves symptoms but nearly always is accompanied by side effects such as headache and nausea.\textsuperscript{72}

Conversion reactions may involve any part of the body under voluntary nervous control. Examples include paralysis, tics, blindness, skin hypesthesia, hysterical bent knee, hysterical bent back (camptocormia), inability to use one or both hands (writer's syndrome) and pain.\textsuperscript{73}

In the contemporary medical setting, pain is one of the most frequent forms of conversion. Some patients whose conversion takes this form have a life-long history of different pains, numerous surgical procedures, and accidental injuries. For such persons, the clinician finds other evidence that pain and suffering constitute a way of life. Consequently, the musculoskeletal system lends itself well to conversion symptoms because it plays such a large role in everyday expression.\textsuperscript{74}

Conversion reactions involving pain are frequently seen in patients with hysterical personalities and are common in depressed patients. However, they may be found in any personality with an underlying psychological disorder.\textsuperscript{75}
Psychophysiologic mechanism

This phenomena consists of prolonged muscle tension giving rise to symptoms such as pain, soreness, and stiffness. Pain is caused by altered muscle tension from body postures or physiological mannerisms that may reflect long-standing emotions. Examples include the "jaw clenched in anger" or the "rigidly held neck in stubbornness". Physicians have reported that this phenomena is lessened considerably after psychological counseling or eradication of stressful stimuli.

Rheumatoid Arthritis

Psychological causations of RA have been investigated since the turn of the century. These causations are grouped around three issues. The first is whether individuals who develop RA are characterized by identifiable psychological traits and personality patterns. The second issue is whether identifiable psychological responses in patients with RA influence the course of the disease. The third issue is whether there is a significant association between certain kinds of life experiences or psychological states and the timing of onset of rheumatoid arthritis.

In 1955, a systematic review of all data related to the psychological implications of RA was undertaken. In this review, it was noted that characteristic personality patterns of patients with RA included inhibition of hostility, inability to show anger, inability to form close relationships with a significant other, conflicts in sexual identity and adjustment, expressed "good" behavior, shyness, and depres-
sion. 78-81

Only two studies up to this date utilized control subjects in forming these conclusions. 82,83 Most studies used the Rorschach and other projective techniques or psychoanalytic therapy sessions.

A typical example of a "pre-1955" investigation of RA is an article written in 1936 by Dr. Giles Thomas. 84 Using a psychoanalytic approach, Thomas took careful case histories of 20 persons diagnosed as having RA. Reviewing the article indicates that there is much room for error. First, a diagnosis of RA was solely based on three criteria, which included:

1. Have you ever had arthritis or rheumatism?
2. Have you ever had swelling in any joints?
3. Do you wake up with stiffness or aching in any joints or muscles?

Current investigations indicate that this is not enough information upon which to make an accurate diagnosis of RA.

Second, researchers in that era associated gastrointestinal disturbances with psychological causation to the exclusion of physiological causation.

Third, parts of patients' histories were given more importance than others. For example, each patient had infected tonsils before or during the onset of RA. Also, one person had an attack of hives before the disease symptoms appeared. These incidences lend credence to the infectious or immunological nature of RA, but were glossed over in favor of symptoms which could be explained by the "current thought" of that era, which was basically psychoanalytic.
Consequently, each case history revealed sexual adjustment problems, evidence of contained hostility, and depression or emotional trauma precipitating onset of RA.

Contained Hostility

The question of contained hostility piqued the interest of researchers and a large amount of data has been accumulated in that area.

Data which indicated that patients with RA regarded their parents as mean or threatening was interpreted as a situation which could breed long-term hostility. Additionally, since depression was evident in many patients with RA, and since depression may be a manifestation of inward hostility, this hypothesis was theoretically sound. However, RA is experienced by patients in lower social classes "whose open expression of anger or hostility may be more culturally acceptable." The hypothesis of the importance of contained hostility later received affirmation by several groups of workers on the basis of projective testing. The specificity of the concept of contained hostility for rheumatoid arthritis can certainly be questioned, since patients with ankylosing spondylitis, peptic ulcers and ulcerative colitis produce similar test results.

Early studies indicated that patients with RA were more likely to get divorced. This was interpreted to be additional evidence that might support the concept of unexpressed hostility. However, this data could not be substantiated by studies in Jerusalem, or Great Britain.
The Minnesota Multiphasic Personality Inventory (MMPI) has been used to measure personality profiles in patients with RA. Findings consistently show elevation in the areas of hypochondriasis, depression and hysteria. However, these areas are also elevated in patients with multiple sclerosis or spinal cord injuries. 92

An excellent expression of the theoretical relationship between hostility and rheumatoid arthritis has been made by Fisher and Cleveland in their book, *Body Image and Personality*.

"We were struck by how much difficulty arthritis patients had in expressing anger. They rarely lost their tempers and even in situations of considerable frustration maintained an affable attitude. It seemed as if they were afraid to express anger and felt it necessary to contain it inwardly in a tightly controlled fashion. . . one could conceive of the arthritic as a person who has certain unacceptable impulses over which he is so fearful of losing control that he has found it necessary to convert his body into a continuing vessel whose walls would prevent the outbreak of these impulses . . .selectively utilizing a particular layer of his body (striate musculature) to achieve a protective wall about himself. His muscle stiffness seemed to be equated with inhibition and making his body exterior tough and resistant." 93

This attitude is somewhat supported by the authors of *Primer on Rheumatic Diseases* who state that:

"pleasure in movement, which underlies life-long aspirations of independence and achievement, contributes to the preferred self-image of most patients. At times of crisis, with actual or impending invalidism, attempts to maintain this self-image are threatened because the usual style of coping through movement leads to further pain. The temptation arises to abandon the emphasis on activity, to avoid further pain, and to be cared for. Such temptation is accompanied by considerable shame. Succumbing to the temptation, however, is also facilitated and justified by the existence of painful illness and demonstrable crippling. These circumstances lead to a mental conflict between a more primitive wish for pain reduction and nurturance and an adult wish to restore independence and achievement. The
outcome of this conflict is determined by the willingness to tolerate pain now, and to avoid dependency, for the long-range expectation of resuming autonomous activity."94

These quotations support the hypothesis that the rigid or hostility-contained personality of a patient with RA is a manifestation of the disease, rather than a causative factor. However, methodological problems have thus far precluded testing individuals before the onset of RA. It is possible that future testing may incorporate predetermination of RA by biological methods. Once this is accomplished, researchers may be able to more fully determine the role of personality profiles in the onset of RA.

Disease Course and Personality

A review of the literature indicates that there is little doubt that disease course and personality interact. Moldofsky (1970) identified a sub-group of "paradoxical responders" among rheumatoid patients, whose moods worsen as their inflammation lessens. Such patients arrived seemingly cheerful, brave, and compliant, but developed depression and new complaints as therapy progressed. They, therefore, selected themselves for alternative therapies, and were more likely to receive prolonged hospitalization, extensive investigation, systemic steroid and surgical therapies. This response was seen in nearly half of the hospitalized rheumatoid patients. It was hypothesized that the "paradoxical" patients had a tremendous psychological need to hold family, friends, and therapists close to them. Consequently, they had "much to fear from restored health."95

MMPI test results also showed that psychological
factors may influence the course of the disease. Patients with slow progression scored higher on scales reflecting compliance, perfectionism, subservience, denial of hostility, and capacity for status and social response than did those with fast progression of the disease. Additionally, patients with fast disease progression scored higher on scales reflecting physical malfunctioning, general maladjustment and manifest anxiety. 96

These results were interpreted to indicate that patients with rapid disease progression understandably seemed to be making a strong but unsuccessful attempt to keep their impulses under control. It was pointed out that patients with rapidly advancing rheumatoid arthritis, as well as those with rapidly growing carcinomas, tend to expend a large proportion of energy in ego-defense. As the objective evidence of disease progression becomes increasingly obvious in such individuals, they naturally begin to demonstrate ego-defense failure, as indicated by increased manifest anxiety and overt depression.

Another study attempted to analyze correlations between pain and the individual patient's mood patterns. It was found that patients with RA could be divided into two basic pain-mood profiles. In one group, a synchrony was recorded between mood changes, particularly within a spectrum of anxiety or hostility, and concurrent fluctuations in joint tenderness and symptoms. A second group was observed in whom there appeared an inverse relationship between the intensity of joint pain and a sense of despair or hopeles-
ness. Follow-up on this study indicated that those patients who demonstrated an inverse relationship had a "less favorable course and more severe relentless disease."⁹⁷

Life Experiences and the Onset of RA

One investigator, Rimon (1969), studied 100 female patients with RA. Of the 100, he focused primarily on 10 with "malignant cases of RA". Of these 10, 5 had a personal crisis at the onset of the disease and 7 of the 10 were economically very poor.⁹⁸

Studies in identical twins discordant for RA suggested that stressful life events may be important.⁹⁹ However, a controlled study of circumstances surrounding the onset of RA showed equal frequency of stressful events prior to disease onset in 532 patients and in matched controls.¹⁰⁰

Many of the earlier investigations of RA indicated a personal crisis close to the onset of the disease. However, it is doubtful that this is a sole factor in the onset of RA, because most people have personal crises and many do not develop RA.

Other Psychosomatic Factors

Physiologic differences have been identified, including a lower blood pressure but faster pulse, increased sweating, and colder extremities, with exaggeration when the subjects were stressed by angry criticism.¹⁰¹

An interesting study investigated the possibility that various forms of organic illness may not just randomly occur in a person's life, but that life experiences, psychosocial forces and individual personality traits appear to influence
an individual's susceptibility to various illnesses.  

Conclusion

Although psychological causation of RA is yet unproved, it is a factor in the exacerbation of the disease. For example, rheumatologists are the first to admit that stress or personal crisis will increase the severity of joint symptoms. Since emotional stress can influence the disease process of RA, further research is warranted that would focus on the interaction of biological and psychological factors.

An investigation of the interaction effects of psychological and biological factors appears warranted. The psychological factors to be examined would include hostility, inability to show anger, inability to form close relationships with a significant other, conflicts in sexual identity and adjustment, expression of "good" behavior, shyness, and depression. These are the most common personality characteristics seen in patients with RA.

The biological factors to be examined would include the presence or absence of infection, metabolic and biochemical abnormalities, autoimmunity reactions, and possible genetic determinants.

The subjects to be considered would be siblings, parents and grandparents of rheumatoid patients. Additionally, the spouses and children of each sibling, parent, and grandparent would be considered. The rheumatoid patients must be accurately diagnosed by fulfilling five or more of the RA criteria.
The subjects in this experiment would be given personality inventories and biological screenings during an extended time period covering many years.

The personality inventories may include the California Personality Inventory and Cattel's questionnaire. These may be given separately or in a combined manner. However, it may be advisable to design a new personality inventory that would specifically measure the common personality characteristics presently identified in rheumatoid patients. This could be accomplished by rheumatologists and psychologists who would establish criteria to be used for the detection of these personality characteristics.

Additionally, biological testing would incorporate procedures to detect infection, metabolic and biochemical abnormalities, autoimmunity reactions, and genetic determinants.

A format for administration of the psychological and the biological tests would be established and a procedure for data collection would be implemented.

This data then could be analyzed during and at the conclusion of the time period of the investigation.

The following points support the solidarity of this research suggestion:

1. The nature of the experiment allows a "family tree" to be constructed which may or may not indicate a pattern of genetic inheritance.

2. If proper biological tests are used, the presence or absence of infection, metabolic and
biochemical abnormalities, and autoimmunity factors may be indicated. Additionally, the factors may be discovered before the actual onset of the disease. If this is the case, a biological causation may be substantiated.

3. The consecutive utilization of personality inventories would enable the researcher to suggest that the typical RA personality precedes or is a result of the disease.

Finally, other results may surface which indicate that specific biological and psychological factors interact in RA etiology.

EXPLOITIVE COMMERCIAL TECHNIQUES USED
ON THE RHEUMATOID PATIENT

The rheumatoid patient is especially susceptible to exploitation by quacks and charlatans because the pains that accompany the disease "make him desperate in his never-ending search for relief and in his willingness to try anything that promises it."\(^{103}\)

Formerly, the market was flooded with gimmicks such as the "Miracle Health Relaxer", which sold for $9.95 and promised relief for both arthritis and constipation. A Vryllium Tube that was chemically analyzed and found to contain approximately two cents worth of salt sold for $250.00.

Among the most popular devises were vibrators. One model came with five attachments, including one guaranteed to banish dandruff.\(^{104}\)

The above-mentioned gimmicks have finally been pulled
off of the market by the Food and Drug Administration (FDA). However, they have been replaced by worthless diet cures, and expensive over-the-counter (OTC) pain relievers.

When the "Arthritic's Cookbook" sold well, out came a sequel, "New Hope for Arthritics" which contained the same diet. Many arthritic diets and cookbooks inundated the market until the Arthritis Foundation, which maintains a list of books not recommended for arthritics, had to add a special section for the growing assortment of diet cure-alls.  

The truth is that there is no scientific evidence that any food or vitamin has anything to do with causing or curing arthritis. "The proper diet for someone with arthritis", advises a local rheumatologist, "is a normal, well-balanced, nourishing diet - the same things people without arthritis should eat." One exception, however, is gouty arthritis, which calls for a diet to limit the uric acid levels in the blood.

Plain aspirin, which is one of the most effective drugs used in initial RA treatment, is relatively cheap. However, the makers of Anacin, claim that their product is "the pain medication doctors prescribe most for arthritis" and that it has special anti-inflammatory action. Upon closer investigation, it is noted that Anacin is simply a plain aspirin tablet with small amounts of caffeine or antacid added. Arthritis Strength Bufferin also falls in the same category and is also several times more expensive than aspirin.  

Liniments, ointments, and body rubs are also heavily promoted for relief of aches and pains. However, medical
personnel warn that indiscriminate use of external analgesics with high methyl salicylate content, such as Exocaine, Musterole Deep Strength, and Panalygesic can cause poisoning. 107

A partial list of known quack remedies include: wearing copper around an inflamed joint, applying a concoction of boiled poisonous leaves, drinking mineral water, leaving all windows open at night, inner shoe plates made of copper and sprinkled with sulphur, implanting beans under the skin, and soaking in mud baths or uranium mines. 108
FOOTNOTES


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22 Ibid.


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27 Ibid.

28-35 Ibid.


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39 Ibid.

40 Rodnan, Gerald R., editor. Primer on the Rheumatic Diseases, p. 35.

41 Ibid.
42-48 Ibid.


52 Steinberg, Alfred D. and Decker, John L. "Immunoregulatory Drug". Arthritis and Allied Conditions, p. 382.

53 Better Homes and Gardens, "Arthritis Progress", p.34.

54 Ibid.

55 Ibid.


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59 Ibid.


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69. Ibid.

70. Ibid.

71. Ibid.

72. Ibid.


75. Ibid, p. 127.

76. Ibid, p. 126.

77. Green, Norman M.D. Personal communication.


88. Ibid.


94. Rodnan, Gerald R. Primer on the Rheumatic Diseases, p.128.


101Green, Norman, M.D. Personal communication.


103Kitay, William, Overcome Arthritis, p. 142.


105Ibid.


107Ibid.

108Kitay, William, Overcome Arthritis, p. 142, 143.


Felty, A.R. Bulletin from John Hopkins Hospital. 35:16, 1924.


Shoulder flexion ("wand" exercise). A stick is held with both hands approximately a shoulder's width apart. The "good" arm through the stick or wand assists the stiff shoulder in stretching. This is used for subacute or chronic shoulder contractures.

Shoulder circumduction (Codman). A pendulum rotary motion is assisted by gravity and by holding a one-kilogram weight. This exercise for acute or severe restriction of shoulder motion can also be performed with the patient lying prone extending the arm over the side of the bed.

Wrist extension (dorsiflexion). Hand is placed flat on table. Wrist is extended by body leaning over table.

From: Arthritis and Allied Conditions, p. 526-527.