1998

Rheumatoid Arthritis: Minimizing Deformity and Preserving Function in the Hand

Cindy Gillespie
University of North Dakota

RHEUMATOID ARTHRITIS: MINIMIZING DEFORMITY AND PRESERVING FUNCTION IN THE HAND

by

Cindy June Gillespie
Bachelor of Science in Physical Therapy
University of North Dakota, 1997

An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

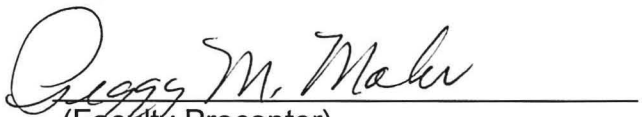
in partial fulfillment of the requirements

for the degree of

Master of Physical Therapy

Grand Forks, North Dakota
May
1998

This Independent Study, submitted by Cindy June Gillespie in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.



(Faculty Preceptor)

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ABSTRACT

Rheumatoid arthritis (RA) is a chronic disease of the synovial joints. The hand and wrist are the most commonly involved. The disease is characterized by pain, inflammation, limited range of motion, and destruction of bone and articular structures. In the later stages of RA, the person usually exhibits deformity of the affected joints. This can result in the inability to use the hand in a functional manner.

Rheumatoid arthritis affects approximately one percent of the population. A person with RA usually has increased medical expenses. It is likely that the person will experience a loss of productivity and income due to disability. The mortality rate of individuals with RA is increased when compared to the general public.

The purpose of this paper is to present an overview of RA including a history of the disease, prevalence, cost and work disability, and mortality that results from RA. In addition, the etiology and pathology of RA and common deformities of the wrist and hand that result from the disease process will be discussed. Physical therapy treatment of RA including patient education, splinting, surgery, exercise and modalities is reviewed.

The physical therapist can be instrumental in educating the patient about their disease, appropriate joint protection and energy conservation techniques, and correct use of exercise and modalities to relieve pain and improve hand function.

CHAPTER I

INTRODUCTION

Rheumatoid arthritis is a chronic disease of the peripheral joints but systemic symptoms may also be present.[1] Fleming et al[2] identified the most common sites of initial involvement as the hand and wrist (36%), multiple joints (29%), foot and ankle (19%), and other joints (16%). Gordon et al[3] found that 76% of individuals with a mean disease duration of 11.5 years (range = 1 to 31 years) had extra-articular manifestations of the disease. The most common extra-articular symptoms were subcutaneous nodules (53%) and pulmonary fibrosis (20%). Pericarditis was the least common feature. Other manifestations of the disease may include vasculitis, skin ulcerations, lymphadenopathy, neuropathy, amyloidosis, and generalized osteoporosis.[3,4] The presence of extra-articular manifestations was seen more frequently in patients with high rheumatoid factor titers and those with severe articular destruction as seen on x-ray.

The onset of RA may be sudden or insidious[2] and the rate at which deformities develop may be fast or slow depending on the individual.[5] The degree of severity exists on a continuum from mild and intermittent to severe and continuous.[6] RA has a variable course so that a person can have symptoms at one point in time and be symptom-free at later examination.[7] The disease is

characterized by pain, stiffness, limited range of motion, muscle atrophy, destruction of cartilage and bone, and attenuation of supporting structures such as ligaments and capsules which leads to subluxation and dislocation.[5]

Although the disease can occur at any age, onset is most common between 25 and 50 years of age.[1] Juvenile RA (JRA) begins before the child is 16 years old.[1,8] It is considered to be a distinct disease, separate from adult RA. There are three subtypes of JRA: systemic (Still's disease), pauciarticular (≤ 4 joints), and polyarticular (≥ 5 joints), each presenting differently. Since JRA and adult RA are considered to be different entities, further discussion will be directed toward adult RA.

History

The first written record of RA is attributed to Augustin-Jacob Landré-Beauvais, a French medical student.[9] In 1800, as the topic of his doctoral thesis, Landré-Beauvais wrote about his observations of a previously unrecognized disease that he called primary asthenic gout. The disease affected primarily the small joints and was characterized by pain, swelling, loss of range of motion, destruction of cartilage and bone, reduction of bone mass, and manifestation of extra-articular symptoms. In addition, primary asthenic gout had residual symptoms – swollen and deformed joints – that remained after the other symptoms of the disease had resolved. Landré-Beauvais compared his findings to classic gout, a recognized affliction of the era. He noted that primary asthenic gout was seen more often in females and the poor, was less painful and of longer duration than classic gout. In addition, classic gout usually occurred in a single

joint whereas primary asthenic gout involved more than one joint. Primary asthenic gout also lacked the deposits of sodium urate seen in the tissues of individuals with classic gout. Fifty-nine years later, Sir Alfred Baring Garrod introduced the term rheumatoid arthritis into the medical literature as the new name for primary asthenic gout.

Although Landré-Beauvais is credited with recognizing the existence of a separate disease,[9] there is information suggesting that RA occurred prior to the 1800's. The writings of Thomas Sydenham from the late 1600's describe his observations of a chronic disease with characteristics similar to those of RA. Rothschild et al[10] examined 84 skeletons (3,000 to 5,000 years old) that were excavated from an area in northwestern Alabama. Four females and two males displayed evidence of symmetrical involvement and destruction of the synovial joints. Rothschild and Woods[11] analyzed the skeletons of 129 Archaic Indians (4,050 to 4,300 years old) that were found in Kentucky. The researchers found evidence of decreased bone density and symmetrical, erosive arthritis of the synovial joints in seven skeletons, five females and two males. Although this evidence is not conclusive for the existence of RA prior to 1800, the characteristics that the skeletons[10,11] displayed are common to individuals with RA.

Dequeker[12] and Appelboom et al[13] also found suggestions of the existence of RA prior to the 19th century. Dequeker[12] inspected Flemish portrait paintings from the 15th through the 18th centuries and discovered that the paintings revealed hand deformities similar to those seen in RA, including

swan-neck and boutonniére deformities. After examining the 17th century paintings of Peter Paul Rubens, an European artist, Appelboom et al[13] concluded that the paintings displayed the progression of deformity seen in RA.

There are several theories regarding the origin of RA. Charles Short[9] theorized that ankylosing spondylitis was the initial form of rheumatic disease and that RA evolved from this condition. Due to a lack of archeological evidence suggesting the existence of RA in the Old World prior to 1800, Rothschild et al[10] proposed that RA originated in the New World and was transmitted to the Old World – possibly by a vector, bacteria, or virus – with the advent of overseas shipping and trade. Although there have been differing theories regarding the origins of RA, to date, there have been no definitive conclusions. The possible etiology of RA will be discussed further in chapter two.

Prevalence

The reported rates of definite RA range from 0.2% to 2.3% among different countries and different racial backgrounds.[14-18] The female:male ratio ranges from 2:1 to 3.75:1[14-17,19-23] and there is an increased prevalence with increased age.[16,17,19-22,24-27]

Results of the National Health Examination Survey (NHES) – 1960 to 1962[22] determined the prevalence of probable, definite, and classical RA to be 3.2% overall, 4.6% in women, and 1.7% in men. The percentage of people affected decreased to 1.0% overall, 1.4% for women, and 0.5% for men when cases of only definite and classical RA were considered. With regard to ethnicity,

there was an equal prevalence among Caucasians and African-Americans with higher rates among other races.

There is a widespread variance in the prevalence of RA according to age. Moolenburgh et al[24] discovered that in Lesotho, S. Africa there were no cases of definite RA in individuals under the age of 55. In contrast, the prevalence of definite RA in female Yakima Indians from Washington state, ages 18 to 34, was 3.2%.[25] Other studies of this young age group had rates of less than 1%.[16,19,21,24]

Comparability between studies is difficult because different criteria have been used to classify cases of the disease.[14,19,20,24,25,26,28] There are four criteria that frequently appear in the research – the American Rheumatism Association (ARA) 1958 revised criteria,[29] the ARA 1987 revised criteria,[30] the Rome criteria,[31] and the New York criteria.[32] Comparisons of these criteria result in dramatically different outcomes.[20,26] In addition, the various ages of subjects, dissimilar methodologies, and use of different time frames makes comparison of studies difficult or impossible.[14,15,17,19,21,25]

The fluctuating nature of RA, with remissions and exacerbations, makes the prevalence of the disease difficult to measure.[6] As a part of the Tecumseh Community Health Survey, Mikkelsen et al[21] conducted a study of all residents in the community. Of the participants who were at least 16 years of age, the prevalence of possible, probable, and definite RA combined was 7.6% overall, 10.0% for women, and 4.9% for men.

Four years later, 71.4% of the participants who were suspected of having RA in the original Tecumseh study were reexamined.[7] Of the individuals

diagnosed with definite arthritis in the original study, 21% had no evidence of RA, and 58% were diagnosed as having either a less severe classification of RA or tested positive for rheumatoid factor without any other signs of the disease. The individuals who were originally diagnosed with either possible or probable RA displayed similar indications of remission. Similar results were reported by Wolfe and Hawley,[33] who determined that there was an 18.1% remission rate among persons having either definite or classical arthritis. Cathcart and O'Sullivan[34] found that 1/3 to 1/2 of those having either probable or definite RA during their original study[20] were symptom-free at follow-up four years later. As an explanation, the researchers suggested the possibility that early RA may be a benign, self-limiting process.

Cost and Work Disability

The financial burden of RA is considerable. Stone[35] estimated the lifetime costs of RA including medical costs and productivity loss. The average lifetime cost was ascertained to be $20,412 per person diagnosed with RA. Due to their longer life expectancies, the dollar amount for individuals under 45 years of age was $60,000 for men and $26,000 for women. Meenan et al[36] found that the cost incurred by individuals with stage III[37] RA, as defined in Table 1, averaged $7,077 annually for those requiring hospitalization and $647 annually for others who were not hospitalized[36].

The cost of RA in terms of lost productivity and income is substantial. Liang et al[38] found that RA limits a person's activity an average of 6.8 days each month, including 2.5 days of lost employment and 1.3 days of bed rest. Yelin

Table 1. – New York Rheumatism Association Classification of Stages of Rheumatoid Arthritis*

Stage I	No bone or articular cartilage erosion evident on x-ray** Evidence of osteoporosis may be evident on x-ray
Stage II	Evidence of osteoporosis on x-ray, with or without slight bone erosion, slight destruction of cartilage may be present** Lack of joint deformity, with or without limited joint mobility** Adjacent muscle atrophy Extra-articular soft tissue lesions may be present
Stage III	Evidence of cartilage and bone destruction on x-ray; evidence of osteoporosis** Joint deformity, i.e. subluxation or ulnar drift, without fibrous or bony ankylosis** Extensive muscle atrophy Extra-articular soft tissue lesions may be present
Stage IV	Fibrous or bony ankylosis** Presence of criteria in stage III

* adapted from Steinbrocker et al.[37]

** these criteria are required for classification

et al[39] discovered that 10% of the individuals who were working at the time of diagnosis had stopped working within one year. By the fifth year, the percentage had increased to 33% and 60% had ceased working after 15 years.

Yelin et al[40] determined that for persons with stage I or II[37] RA, the probability of becoming disabled was 0.44[40]. The probability increased to 0.72 for individuals with stage III or IV[37] disease[40]. The probability was not affected by age, sex, race, or educational level. Single people had a 0.33 probability of work loss compared to 0.67 for married individuals. The amount of autonomy experienced at work also affected disability status. Individuals who were either self-employed or able to adapt their work activities as needed were less likely to become disabled. Reisine et al[41] found that the likelihood of a person becoming disabled increased by 36 times if they experienced a lack of autonomy on the job.

During a year-long study, Meenan et al[36] determined that 66% of individuals with stage III[37] RA made changes in their work situations and 86% of these people became totally disabled[36]. The individual's occupation did not have a significant effect on disability. In contrast, Reisine et al[41] found that individuals were 6.8 times more likely to become disabled if their jobs were physically demanding.

In summary, the loss of productivity and income due to disability is considerable.[35,36,38-41] The severity of the disease, a person's marital status, and the amount of autonomy in one's work environment appear to be important determining factors in the development of disability due to RA. Disability not only

affects the individual with RA and his or her family but it also has an effect on society in terms of lost labor force and increased medical expenses.

Mortality

The mortality rate in individuals with RA has been reported to be 1.5 times higher than in the general population.[23,42] Cardiovascular disease has been reported as the most common cause of death[23,42-44] with rates similar to that of the general population.[42,43] Other causes include infectious (pulmonary, septicemia, peritonitis), respiratory, renal, gastrointestinal (bleeding, perforations), and neoplastic diseases and urogenital sepsis.[23,42-44] Complications of RA such as decubitus ulcers, spinal cord compression, cachexia (general ill health and malnutrition), vasculitis, renal failure due to amyloidosis, and rheumatoid heart disease have also been reported as causes of death.[43,44]

Vandenbroucke et al[43] concluded that the proportion of individuals with RA who died from infectious causes was 3.7 times higher than that of the general population. Uddin et al[42] found that there was an increase of 7 to 10 times that of the general population and hypothesized that the debilitating nature of the disease may make the RA patient more susceptible to infection.

Studies also show that life expectancy of the individual with RA is decreased.[23,42,43] Vandenbroucke et al[43] found that the median life expectancy was three years less for women and seven years less for men when compared to the general population. In contrast, Mitchell et al[23] estimated the median age at the time of death to be ten years younger for women and four years younger for

men. People that died of causes unrelated to RA had a longer life expectancy (71.9 years) than those whose death was either directly related to or complicated by RA (63.7 years).[44]

In summary, RA has a negative impact on the patient, his/her family, and society in terms of increased medical expenses, work disability, and higher mortality rate among individuals with RA.[23,35,36,38-44] The management of RA is an important factor in minimizing the effect of the disease on all people involved. The chapters that follow will discuss the etiology and pathology of RA, common deformities of the wrist and hand that result from the disease process, and some of the methods used to treat the disease.

CHAPTER II

ETIOLOGY AND PATHOLOGY

Etiology

The etiology of RA is unknown.[45] However, several substances including various viruses have been investigated as possible causative factors. Schumacher[46] suggested viruses as a possible agent when he discovered virus-like particles in the synovial capillary walls of some RA patients. Further support for the role of viruses as possible initiators of the disease process was offered by Tosato et al.[47] These researchers observed the lymphocyte (non-phagocytic white blood cells present in the lymph and blood – T and B cells) activity of individuals with and without RA. All of the participants had been previously infected with and successfully recovered from the Epstein-Barr virus (EBV). It was concluded that the B and T cells of the people without RA responded with an initial increase in the secretion of antibody followed by suppression of lymphocyte activity after the antigen was destroyed. In patients with RA, this suppression did not occur. The lymphocyte activity was suppressed by 94% in the group without RA compared to 17% suppression in persons with RA. The results of this study suggested that defective suppression of lymphocyte activity may result in unregulated antibody production.

Rheumatoid factor has also been mentioned as a possible causative factor in RA. Rheumatoid factor (RF), an autoantibody that is produced by the immune system,[48] is present in 70% to 80% of individuals with RA.[45] It is found more frequently in older individuals[19,21] and is the most abundant antibody in the immune complexes of RA patients.[49] Jasin[48] analyzed cartilage samples from people with RA and found that they contained 14 to 37 times more antibodies than the cartilage of individuals without RA. In addition, RF-antibody complexes were present in 81% of the individuals with RA but were absent in the control group. Vaughan[49] proposed that aggregates of RF may be capable of initiating the immune response in RA patients.

It has also been suggested that RF may be an indicator for increased susceptibility to[7] and severity of RA.[50] Alarcón et al[51] found that subcutaneous nodules and vasculitis were present only in individuals with RF. In addition, individuals who were RF(-) did not have the radiological changes that are common with a diagnosis of RF(+) RA.[51,52] Rheumatoid factor appears with equal frequency in men and women but women are diagnosed with RA two to three times more often than men, [16,19,21] giving support to the hypothesis that RF is important in the susceptibility to rather than being the cause of RA.

The presence of RF does not necessitate a diagnosis of RA. The NHES[22] revealed that 2.6% of individuals were RF(+) but had no other signs or symptoms of RA. Mikkelsen et al[21] obtained similar results. In addition, not all persons who have symptoms of RA test positive for RF.[21,22] Calin and Marks[52] stated that the symptoms described in the first five criteria used to classify cases of RA[29] can

also be experienced by individuals with degenerative joint disease or ankylosing spondylitis, conditions in which RF is usually absent.[52] It has also been shown that a person may test RF(+) initially and test RF(-) at a later date.[7]

Genetics have also been implicated in the cause and severity of RA but there is conflicting data in this regard. Aho et al[53] determined that there was no difference in the overall prevalence rate between monozygous and dizygous twins. The researchers concluded that the participation of genetics in determining the susceptibility to RA was minimal and that hereditary factors may be more important in determining disease severity rather than susceptibility.

Wasmuth et al[54] discovered that the prevalence of RA was significantly increased ($p<0.001$) in the first and second degree relatives of probands – the persons who were initially diagnosed with RA – compared to the probands' spouses. There was also a greater prevalence of RA among relatives when the proband developed the disease early in life (between the ages of 15 and 34) rather than later (between the ages of 55 and 74). From these results, the researchers concluded that there may be a genetic component involved in the development of RA. In addition, the spouses of probands had increased rates of RA when compared to the spouses of relatives, leading the authors to postulate that an environmental factor may also play a part in the acquisition of RA.

Certain human leukocyte antigens have also been seen more often in individuals with RA compared to the general population.[51,55] Human leukocyte antigens (HLAs) are molecules (receptors) on the surface of cells.[56] The receptors identify cells as being a part of the body rather than foreign

substances. The HLA antigens, specifically the HLA-DR class, have been linked to an increased risk of developing RA and to a more destructive course of the disease.[51,55]

Alarcón et al[51] discovered that the distribution of HLA-DR4 was increased (56.25% vs. 13.82%) in RF(+) patients compared to controls. However, when comparing individuals having RF(-) RA to the control group, the distribution of antigens was similar. The researchers found that RF(-) individuals who carried the HLA-DR4 antigen [DR4(+)] were at higher risk for developing destructive disease than RF(-) individuals who did not carry the antigen [DR4(-)]. Individuals who were RF(-) and DR4(+) also had more severe grades of erosion on x-ray films, increased use of remittive drug therapy such as gold, antimalarials, and immunosuppressive medications, and greater amounts of destructive disease than RF(-) individuals who were DR4(-). Calin et al[55] had similar results.

Nepom et al[57] examined individuals for the presence of HLA-Dw4 and HLA-Dw14, alleles of the HLA-DR4 gene. One or both alleles were found in all individuals with DR4(+), RF(+) RA. Sixty percent of the DR4(-) patients with classic RA had a DR1 gene with a nucleotide sequence identical to the Dw14 allele. The researchers concluded that the presence of Dw4, Dw14, and the DR1 nucleotide sequence indicated a predisposition to RA in both DR4(+) and DR4(-) patients.

In summary, there is support for the theory that there is a genetic predisposition to RA[51,57] and for the hypothesis that genes determine the severity of RA.[51,55] Environmental factors may also play an important part.[54]

Pathology

According to Harris,[45] there are five pathologic phases of RA. The first stage is characterized by the presence of the causative factor (antigen) of RA. There is development of capillaries and attraction of neutrophils (phagocytic white blood cells) to the joint. The initial symptoms of pain and stiffness occur when the macrophages release cytokine. Fatigue and malaise may be present before the manifestation of joint symptoms.

Stages two and three are differentiated by the severity of the disease.[45] There is proliferation of T and B cells, production of antibodies by plasma cells and activation of the complement system. Angiogenesis continues and plays a role in the development of synovitis. Joint effusion, morning stiffness, and destruction of cartilage begin during stage two but systemic symptoms are usually not seen until the third stage. Medications are usually started during the second stage in order to slow the progression of the disease.

During stage four, synovial tissue continues to proliferate and irreversible damage occurs.[45] The cartilage, ligaments and bone are all involved at this stage. Stage five is a continuation of the process and medication will have little effect on the disease during this stage.

The pathologic process of RA involves both the cell-mediated and antigen-antibody immune responses.[45] Cell-mediated immunity involves the recognition of antigen by macrophages. Once the antigen has been recognized, the macrophage phagocytizes it. T lymphocytes bind to the macrophage-antigen complexes, a process that stimulates the production of the cytokine interleukin-1

(IL-1) by the macrophage.[56] Production of IL-1 leads to the differentiation of specialized T cells – T-helper, T-suppressor, and T-cytotoxic cells. T-helper cells produce the cytokine interleukin-2 (IL-2) which in turn stimulates several processes including production of increasing numbers of T-helper cells, activation of T-cytotoxic cells that lyse the macrophages containing the antigen, and activation of B lymphocytes. The B cells differentiate into plasma cells that participate in the antigen-antibody immune response known as complement.

In addition to IL-1 and IL-2, there are other cytokines including tumor necrosis factor, gamma-interferon, and colony stimulating factor.[45] Cytokines have several functions including the stimulation of: synovial cell proliferation, release of prostaglandin and collagenase, and resorption of bone. They may also be responsible for the fatigue and malaise that are often present before symptoms of joint pain and stiffness appear. In addition, cytokines can induce fever and pain and act as chemoattractants, bringing phagocytes to the injured area.[56]

Eastgate et al[58] found that the blood level of IL-1 was significantly higher ($p<0.001$) in rheumatoid patients compared to control subjects. The researchers also discovered that the increased level of IL-1 was positively correlated with the number of affected joints, duration of morning stiffness, joint pain, and erythrocyte sedimentation rate. The level of IL-1 was negatively correlated with the hemoglobin level. The researchers postulated that IL-1 might contribute to the development of anemia by inhibiting the production of erythropoiesis. The complement system is made up of a series of proteins.[56] Once an antibody

recognizes and binds with an antigen, the first protein attaches to the antigen-antibody complex and activates the system. The proteins are activated sequentially and each has a function. These functions may include capillary vasodilatation, chemoattractant for phagocytes, or cytolysis – all of which result in inflammation. Regulatory proteins are responsible for inactivating the system when the antigen is destroyed.

The synovial tissue contains plasma cells that synthesize antibodies and autoantibodies.[48] These substances are capable of forming antigen-antibody immune complexes. Although the significance of these complexes is not known, they may be responsible for the proliferation of diseased synovium and destruction of cartilage and bone. Ruddy et al[59] demonstrated that the synovial tissue produces proteins that are involved in the complement system. It can be seen that, if the proteins necessary to activate the system are produced in the synovial tissue and the antigen (which may be one's own tissues) is available to bind with antibody, the system could easily become self-perpetuating.

During the disease process, the vasculature of the rheumatoid synovial tissue is altered. Stevens et al[60] found that the density of capillaries in rheumatoid synovium was one-third that of normal synovium. The vessels were also located deeper within the tissue than usual. These conditions resulted in a decreased availability of nutrients and oxygen to the tissue. The researchers concluded that the hypoxic condition resulted in injury to the tissue and postulated that the lack of oxygen was a factor in sustaining the inflammatory

process. The antigen-antibody complexes that are formed are also capable of causing the microvascular injury seen with RA.

The cells of rheumatoid synovium produce prostaglandins[61-63] and collagenase.[62] Prostaglandins produce inflammation, vasodilitation, pain, and edema,[63] and stimulate bone resorption.[61,63] Collagenase has been implicated in the destruction of articular cartilage.[62] Woolley et al[64] discovered that the presence of collagenase in the joint was restricted to the cartilage-pannus junction. Collagenase was found in the synovial tissue only 2% of the time. This finding seems to correlate with Martel et al[65] who found that bone erosions are most extensive at the joint margins where the bone is not covered by articular cartilage. Once the articular cartilage has been destroyed, the bones are more susceptible to compressive forces.

In summary, there are several theories regarding the etiology of RA including viruses, RF, and genetics.[46-49,54] It is possible that environmental and physiological factors contribute to an individual's susceptibility to the disease.[7,39,44,50-53] Regardless of the etiology of RA, it is known that the disease process results in pathological changes within the joint and causes pain, inflammation, and destruction of bone and articular cartilage.[45]

CHAPTER III

COMMON DEFORMITIES IN RHEUMATOID ARTHRITIS

In the wrist, the first sign of RA may be the appearance of tenosynovitis of the extensor carpi ulnaris (ECU) tendon sheath followed by involvement of the distal radioulnar joint.[66] The pathological process leads to destruction of the triangular fibrocartilage (TFC) and distal subluxation of the ulna. Resnick[67] found that erosions of the distal radius and ulna were related to distal subluxation of the ulna and perforation of the TFC, which allows communication between the radioulnar and radiocarpal joints.

Martel et al[65] found that erosion of the ulnar styloid process occurred in 45% of persons with RA and separation of the distal radioulnar joint occurred in 41% of the individuals. Erosion of the ulnar notch of the radius occurred in 21% of the people and was attributed to compressive forces resulting from destruction of the TFC.

The TFC provides support for the ulnar carpus, stabilizes the distal radioulnar joint to prevent dorsal subluxation,[68] and transfers axial forces from the radius to the ulna.[68,69] Destruction of the TFC allows the carpus to make contact with the ulna. Direct articulation between the carpus and ulna prevents full pronation and supination from taking place.

The ligaments of the wrist provide stabilization while allowing controlled motion between the carpal bones.[70] The destruction caused by RA – erosion of bone and articular cartilage, and attenuation of ligaments and supporting structures – causes narrowing of the joint spaces and results in restriction of all motions at the wrist.[71] Attenuation of the ligaments results in abnormal movement of the carpals.[72] As an example, when the intercarpal ligaments between the scaphoid and lunate are destroyed, these bones will separate. This allows the capitate to settle between the two bones and results in the collapse of the midcarpal joint. Kauer[73] demonstrated that movement occurs at both the radiocarpal and midcarpal joints and that each joint is equally important.

Another possible consequence of carpal dislocation is rupture of the flexor pollicis longus and flexor digitorum profundus tendons to the index finger.[72] If the intercarpal ligaments between the scaphoid and lunate are attenuated, the scaphoid has a tendency to flex palmarly which allows the scaphoid tuberocity to penetrate the palmar radiocarpal ligament, contact the muscles and weaken them through attrition.[71]

Ulnar Drift

Ulnar drift of the phalanges is a deformity that is often seen in individuals with RA and there are several theories regarding its development. Fearnley[74] proposed that ulnar drift of the phalanges occurs due to active use of the hand, gravitational pull on the fingers, and muscle imbalances – ulnar dislocation of the extensor tendons at the metacarpophalangeal (MP) joints and altered strength of the intrinsic muscles.

Vainio and Oka[75] developed a similar hypothesis as to the formation of this deformity. When the fingers of the normal hand are extended, the phalanges and the metacarpals form a straight line. When the fingers are flexed at the MP joints, a small amount of ulnar drift occurs. It was hypothesized that as the ligaments and capsule become attenuated, the radial collateral ligaments are more vulnerable to stretching. The natural tendency of the fingers to assume an ulnarly deviated position, use of the hand, and the integrity of the collateral ligaments were found to be important in the development of deformity. Ulnar drift was found twice as often in women (28.6%) than in men (14.6%). This finding was attributed to the delicate bone structure seen in females, the different activities performed by the genders, and the tendency of women to continue with their household chores during periods of exacerbation. It was proposed that men had more opportunities to rest during the acute stages of the disease. Patients who were allowed to completely rest their joints during the acute inflammatory phase did not develop any deformity of the MP joint.

Shapiro[76] noted that hands displaying ulnar drift also had radial deviation of the carpus and that patients who developed ulnar drift during the course of the study had a greater amount of radial deviation at the study's inception. He concluded that radial deviation of the wrist initiated the process that resulted in ulnar drift deformity. The long flexor tendons were then in a position of advantage to exert an ulnar force on the MP joints and further exaggerate the deformity.

Although ulnar drift is commonly seen in all of the fingers, the fingers can also be affected individually.[74] The little finger acts as a block to the other fingers and limits the amount of drift that the other fingers can undergo. If the capsule and ligaments of the little finger are intact, only minor drift of the other fingers can take place. However, if the ligaments are weakened, ulnar drift can develop uninhibited and the ring, middle, and index finger are also free to drift.

Swan-Neck Deformity

Swan-neck deformity – hyperextension of the proximal interphalangeal (PIP) joint and flexion of the distal interphalangeal (DIP) joint of the fingers – also occurs in patients with RA.[77] With this deformity, patients experience a loss of grasp but are still able to utilize the fingertip pinch. Swelling of the PIP joint causes the lateral bands (the interosseous attachments) of the extensor hood to displace to the dorsum of the hand. In this position, the interossei muscles are unable to function independent of the extensor digitorum (ED) muscle. When the ED contracts, the PIP joint becomes hyperextended. The flexor tendons attempt to compensate for the loss of interosseous function and cause a secondary flexion deformity at the DIP joint.

Boutonniére Deformity

Boutonniére deformity – extension of the MP joint, flexion of the PIP joint and hyperextension of the DIP joint – is another common deformity that can occur in the patient with RA.[77] The ED tendon (central slip of the extensor hood) may rupture near its attachment to the base of the middle phalanx. If this happens, as the ED contracts in an attempt to extend the PIP, only the MP joint

extends. The PIP joint flexes and the DIP joint extends in order to maintain a straight line of pull. The interossei are unable to prevent this motion because the pathological condition of the extensor hood puts them at a mechanical disadvantage.

Thumb Deformities

As many as 50% of people who have RA of the hand experience thumb deformities.[77] Synovitis of the MP joint is often the first sign of RA in the thumb.[78] When the MP joint becomes inflamed, the extensor pollicis longus muscle becomes ulnarly displaced and the extensor pollicis brevis muscle looses its power to extend. The extensor muscles are placed at a mechanical disadvantage and a flexion deformity of the MP joint results. If the deformity is not corrected, the IP joint will become hyperextended.

Other deformities of the thumb are caused by synovitis occurring first at the carpometacarpal (CMC) joint.[78] Synovitis in the CMC joint may result in subluxation of the joint. The first metacarpal assumes an adducted position and the adductor pollicis muscle becomes contracted. When this occurs, the MP joint may become hyperextended with a compensatory flexion of the IP joint. Conversely, the MP joint becomes flexed and the IP joint becomes hyperextended. Regardless, deformities of the thumb can result in a loss of grasp and finger pinch. This makes the hand virtually nonfunctional.

Spontaneous Fusion

The combination of articular cartilage destruction and limited movement that occurs in RA can result in spontaneous arthrodesis (fusion) of an affected

joint.[71] The supporting structures of the PIP joint are more taut than those at the MP joint, allowing less room at the PIP joint for joint effusion.[74] This results in greater limitation of movement at the PIP joint and makes it more vulnerable to spontaneous fusion than the MP joint. The DIP joint is usually not affected.

In summary, the common deformities of the hand that occur in RA include collapse of the wrist joint, ulnar drift at the MP joints, swan-neck and boutonniére deformities, and various deformities of the thumb. In addition, the destruction of articular cartilage combined with lack of joint movement can result in spontaneous fusion of affected joints. A loss of function in the hand may occur due to the development of any of these deformities.

CHAPTER IV

TREATMENT

There is no cure for RA. Treatment of the disease involves control of the symptoms. This chapter will focus on education of the patient, splints and surgeries that can be used to minimize the deformities and improve function, and exercises that may help alleviate the muscle weakness and other complications that occur in RA.

Patient Education

Education is an important component of the overall treatment plan of the patient with RA. Kay and Punchak[79] conducted a study among 100 RA patients recruited from a rheumatic disease center and found that patients were not adequately informed about their disease. Fifty-four percent of the participants said that they had received little or no information regarding the pathology, symptoms, and prognosis that could be expected and 67% said that they would like more information. Many stated that they used television, books, pamphlets, magazines, radio, and newspapers to gain information about their disease. The content of an educational program for people with RA might include information about the disease and its symptoms,[80,81] exercise techniques,[80-82] joint protection, relaxation,[80,82] diet, use of modalities such as heat and massage,[82]

social and functional difficulties, pain control,[80] control of anxiety, expression of emotions, coping and stress management skills,[81] and community resources.

Berg et al[80] found that patients reported improvement in their levels of physical activity, mobility, activities of daily living (ADLs), and social activity six months after attending patient education classes. There was a significant improvement ($p<0.001$) in the patient's knowledge of RA, compliance with joint protection and energy conservation techniques, and participation in an exercise program. Davis et al[81] also demonstrated the benefits of patient education as shown by increased knowledge and perceived self-efficacy, including ADLs, and symptom and pain control ($p<0.05$). Lorig et al[83] found that patients not only increased their knowledge and compliance with exercise and relaxation programs ($p<0.01$) but that pain was decreased by 20%, twice that of the control group that had not received instructions. The number of visits also declined although this result did not reach significance.

In contrast, Cohen et al[82] determined that there was little difference in the self-management behaviors of individuals after attending an educational course. These researchers found that exercise was the only behavior that significantly increased after attending six weekly education classes. Neither the degree of disability nor the patient's subjective pain levels was altered.

There is also contradicting evidence regarding the effect that education has on the patient's psychological status. Lorig et al[83,84] reported that not only did patient's increase their compliance with exercise and relaxation techniques ($p<0.01$), but they also experienced a decrease in their symptoms of depression

($p<0.05$). This study contrasts with the results obtained by Goeppinger et al[85] who determined that in spite of significant improvement in the patient's knowledge of RA, performance of self-care behaviors, and level of perceived helplessness, the level of depression experienced by these patients was unchanged.

The instruction style may not be as important as the fact that the information is made available to the individual. Lorish et al[86] conducted an experiment with patients who were in a hospital setting. Three teaching methods were used:

1. individualized (I) instruction containing content that was adjusted to the patient's knowledge of RA, physical condition, and emotional status
2. a routine (R) program that utilized formatted self-instruction and contained information about the disease and home care issues
3. no planned (NP) instruction which included instructions on how to correctly perform a home program without any additional information except responses to questions asked by the patient.

Although all of the programs demonstrated significance in the ability to convey knowledge, the I program was rated significantly higher when compared to the other two programs. All patients in the I program demonstrated a gain in their knowledge base compared to 65% of the patients in the R group and 78% in the NP program. Other studies[82,85] have also shown significant gains in knowledge utilizing various teaching methods including small groups, self-instructed programs, or the use of professionals versus lay instructors.

After a patient has been diagnosed as having RA, he or she may be referred to physical therapy. Because the therapist often spends a considerable amount of time with the patient, the patient may rely on his or her therapist as a source of information. The physical therapist can be an important resource and direct the patient to other professionals as needed.

Splinting

There are various types of splints available that can be used in the treatment of RA hand deformities.[87-104] Splints are used for a variety of reasons including 1) pain relief 2) decreasing inflammation 3) correcting deformities 4) creating functional deformities 5) post-surgical rehabilitation[87] and 6) increasing function.[88] Features to consider when choosing a splint include durability, simplicity of construction, comfort, lightweight, cosmesis, and ease of donning and doffing.[89]

Compliance should be considered when initiating a splinting program. Some of the reasons cited for non-compliance include a decrease in pain levels so the patient felt that splinting was no longer necessary, increased joint stiffness, inconvenience, and discomfort.[90] Callinan and Mathiowetz[91] compared hard resting hand splints (thermoplastic with straps) with soft resting splints (fabric outer cover with a thermoplastic insert) and found that compliance rates were 67% and 82%, respectively. Fifty-seven percent of the participants preferred the soft splint.

Splinting should minimize restriction by preventing faulty movements while allowing movements that can be performed correctly.[89] VanBrocklin[88] described

a cock-up splint that can be made to support the wrist only or a platform can be added that limits flexion of the MP joints or fingers. The points of pressure and the straps are placed such that they help to provide optimal alignment of the structures and reduce stress on the joints. Smith et al[92] suggested the use of a splint that supports the wrist and minimizes flexion at the MP joint. Bennett[89] described several splints that can be used either to prevent or correct deformities and restore hand function. He stated that the use of splints must be part of a comprehensive program that also includes maintenance of muscle strength and limitation of activity.

The ability to obtain correction of deformity is dependent on the integrity of the tissues surrounding the joint.[93] If the tissue has lost its flexibility, correction may not be possible. Extensive bone erosion may also result in faulty movement at the joint.[89] Early intervention is important. Convery et al[87] found that dynamic splints are an important component of the post-operative rehabilitation process but they are not beneficial in the prevention of deformity.

Nordenskiöld[94] compared two prefabricated elastic wrist orthoses and found that RA patients experienced as much as 52% less pain if the orthoses were worn during the performance of activities of daily living. Grip force was improved by up to 29% but was not restored to normal as defined by the control group. In contrast, Stern[95] found no significant difference in grip strength when a wrist splint was used. A disadvantage of this type of brace is that it can interfere with the performance of some activities[94] by reducing finger dexterity and hand function.[94,96] Stern[95] discovered that finger dexterity is affected by some types of

commercial wrist orthoses but is unaffected by others. Fingertip pinch can also be significantly improved by splinting.[93]

Surgery

Surgery is indicated for the treatment of RA in order to reduce pain, correct deformity, improve function, and for cosmetic reasons.[104] The objectives of surgery include removal of diseased synovium, prevention or repair of ruptured or adhered tendons, and providing stability to the involved joints.[66] Twenty-five percent of the surgeries performed for arthritis are performed on the hand.[105] This chapter will discuss the surgeries that are most commonly performed as treatment for RA.

Synovectomy and Tenosynovectomy – Synovectomy is a procedure that involves excision of the synovial tissue from a joint.[106] Tenosynovectomy is the removal of the sheath surrounding a tendon. The procedures are often performed at the same time[105] during the early stages of RA[104,107] when there is a minimal amount of deformity and conservative measures – medications and rest – have been unsuccessful in treating the patient's symptoms of pain and inflammation.[104,105,107] In addition to reduction of pain, synovectomy slows the destruction of cartilage and tendons and helps to improve function.[108] Straub and Ranawat[66] found that the success rate of synovectomy in controlling synovitis was 99.4% after one year and 92.5% after five years.

Synovectomy and tenosynovectomy are performed by cutting through the extensor retinaculum.[104] The synovium is then removed from the tendons and joint spaces. Because of its location, the synovium cannot be completely

removed.[104,105] Norris[104] stated that only 60% of the synovium can be removed in the wrist and hands. Following surgery, Straub[107] recommended post-operative splinting for 10 days to allow the soft tissues to heal.

Tenosynovectomy of the flexor tendons may be performed if median nerve compression is causing symptoms of carpal tunnel syndrome.[104] During this procedure, the bones of the carpal tunnel are inspected.[107] This helps to ensure that the bones are smooth in order to reduce the likelihood of tendon rupture. Tenosynovectomy of the finger tendons is rarely performed because the pain and stiffness that result from the procedure have limited its popularity.[108] If a flexor tenosynovectomy is performed, Straub and Ranawat[66] recommended that compressive dressings be applied for 24 to 48 hours with active range of motion being initiated 48 hours after surgery.

Darrach Procedure – This procedure involves excision of the distal ulna and is often performed simultaneously with synovectomy and tenosynovectomy.[105,107,108] If too much of the ulna is removed, the distal radioulnar joint will become unstable and fail to provide the support needed by the ulnar carpus.[107] After the joint spaces are cleared of synovium, the extensor tendon is placed under the extensor tendons, providing a smooth surface for the tendons to glide on, and any ruptured tendons are repaired. A loop can be made, using a portion of the extensor retinaculum, to correctly position the ECU tendon and hold it in place. A cast is worn for six weeks, after which active range of motion is started. A 60° arc of motion can be expected after performance of this procedure.

Arthroplasty – The goal of arthroplasty is to relieve pain, correct deformity, and gain stability and additional movement in the joint.[105] Synovectomy of the joint is performed prior to prosthetic placement.[108] The intramedullary canals are then reamed and the prosthesis is fit to the joint.

In the wrist, there are two types of prosthesis that are commonly used: the Meuli three component metal-polythene-mental prosthesis and the Swanson flexible silastic implant.[104] The metal components are cemented in place.[108] The silastic prosthesis is held in place by the capsule and ligaments surrounding the joint.[105] Both component types can loosen and dislocate.[104] Because it is not as strong, the silastic implant is recommended for those patient's who will be performing only light-duty activities.[105] After arthroplasty of the wrist, Ranawat and Rosenberg[108] recommended that the joint be immobilized for four weeks. Active exercise can then be initiated with an estimated return of a 60° flexion-extension arc of motion.

Ranawat and Rosenberg[108] suggested that arthroplasty of the MP joint be followed by five days of immobilization, after which the use of a dynamic splint and active exercise can be performed. Exercise and the use of a dynamic splint was also recommended by Altissimi and Ciaffoloni.[105] It is common to gain 50° to 70° of flexion in the MP joint after surgery. The protocol for arthroplasty of the proximal interphalangeal joint is similar.[108] Arthroplasty of the CMC joint of the thumb can also be performed and is usually accompanied by fusion of the first MP and IP joints. A thumb spica cast is used to immobilize the joint for four weeks before exercise is begun.

Arthrodesis – Arthrodesis (fusion) is performed when deformities, instabilities and pain are severe.[105] This procedure is also recommended as an alternative to wrist arthroplasty for people who will be involved in heavy-duty activities. Arthrodesis offers relief of pain and improvement of function.[108] The disadvantage of the procedure is that the joint range of motion is lost. For this reason, arthrodesis is not performed on the MP joints of the fingers or the CMC joint of the thumb.

In the wrist, tenosynovectomy,[66] synovectomy,[108] and ulnar head removal are performed as part of the procedure. The articular cartilage is then removed from the joint and the bones are smoothed. Bone grafts are placed in the joint spaces to further reinforce the joint.[66,108] Pins are used to secure the bones in proper position. The wrist is immobilized for six to eight weeks. Removal of the pins takes place after the bones are solidly united.[66] The PIP and DIP joints of the fingers and the MP and IP joints of the thumb can also be fused using a similar procedure.[108]

Tendon Surgery – Tendon surgery is performed to repair ruptured tendons and minimize deformity.[104] The tendon repair can be done end-to-end (suturing the tendon back together) or end-to-side (suturing the ruptured tendon to an adjacent tendon). The extensor pollicis longus and the extensor digitorum tendons of the ring and little fingers are ruptured most often.[66,104] The extensor indices can be transferred to the thumb to replace the extensor pollicis longus[105] but it is only strong enough to extend one digit. The flexor digitorum superficialis can be

transferred to the extensor surface to extend more than one finger if necessary.[104]

If there is minimal bone erosion of the MP joints, ulnar drift can be minimized by performing a synovectomy of the joints and relocating the extensor tendons so that they have the correct line of pull.[104] Crossed intrinsic transfer, a procedure in which the intrinsic tendons on the ulnar sides of the fingers are divided then reattached on the radial side of the adjacent finger, is also used to strengthen the pull of the muscles on the fingers in the radial direction. After insertion of MP prostheses to each of the fingers, the radial collateral ligament of the index finger is tightened and the crossed intrinsic transfer is performed to help correct the ulnar drift deformity. The joint is immobilized for two weeks in slight flexion and then a dynamic splint is utilized for six weeks to help maintain extension during the day. A resting splint is used at night. A radial abduction splint can be used as a prophylactic to resist ulnar deviation that might occur in the future.

Exercise

Amick[109] conducted electromyographic (EMG) studies on RA patients and normal controls. He found that the groups had similar readings for nerve conduction times, threshold intensity for stimulation, and duration and amplitude of motor unit action potentials. From these results, he proposed that the muscle atrophy commonly seen in patients with RA was due to disuse rather than lower motor neuron disease. Wozny and Long[110] obtained similar EMG results.

Bens et al[111] described a device called the "Hand Gym" that can help the patient perform hand exercises correctly. This apparatus assists the individual in maintaining the proper position needed to stretch and strengthen the intrinsic muscles of the hand. Schaufler et al[112] found that over a four-month period RA patients were able to increase PIP joint range of motion an average of 7.6° ($p<0.001$) by using the device. Grip strength, palmar pinch, and lateral pinch were also increased significantly ($p<0.001$ to <0.05). Brighton et al[113] discovered a significant increase in grip strength ($p<0.0001$) and pincher strength ($p<0.0005$) after participation in a two-year exercise program. Hoenig et al[114] also suggested that hand exercises may be beneficial.

Smith et al[92] suggested that the strong forces of the flexor tendons need to minimized and that this can be done by changing the way that the hand is used. These researchers proposed that the hand be used as a hook, such as when carrying a pail. This keeps the MP joints extended. Pinch and grasp should be avoided. Instead, the hand can be used in a scooping fashion to pick up objects or the objects can be grasped between the palms of both hands. The researchers suggested that the patient avoid squeezing activities as part of an exercise program since this would tend to further strengthen the flexor muscles.

The following exercises might be included in a stretching and strengthening program for the hands:

1. placing the hand flat on a table and lifting each finger off the table individually[113]
2. gathering and ungathering a towel[113]

3. pinching a sheet of paper between the thumb and each individual finger and attempting to pull the paper out[113]
4. placing the palm flat on a table with the MP joints at the edge of the table, then flexing and extending the fingers at the MP joints, repeating the exercise at the PIP joints[113]
5. resisted finger abduction and adduction[111]
6. flexing the PIP and DIP joints while maintaining the MP joints in extension[111]

Wehbé[115] described exercises to help prevent the flexor and extensor tendons from becoming adhered and stretch the intrinsic muscles. They include making a fist in three different ways:

1. with the MP, PIP, and DIP joints flexed
2. with the MP joint extended and the PIP and DIP joints flexed
3. with the MP and PIP joints flexed and the DIP joints extended

Dellhag et al[116] demonstrated that participation in an exercise program can significantly improve range of motion and grip strength in the hand. These researchers also concluded that a paraffin wax bath was effective in reducing pain and stiffness but did not result in increased hand function if used without exercise. Hawkes et al[117] tested three modalities – ultrasound, paraffin, and a faradic hand bath – followed by exercise. Each of the treatments showed significant reductions in pain and significant increases in grip strength and range of motion. Because each of these treatments was associated with a significant

increase in strength and range of motion, it would not seem inappropriate for patients to use modalities in addition to exercise for the relief of pain.

In summary, it appears that patient education can enhance the patient's knowledge about RA and have a positive effect on the level of participation in physical and social activities.[80] Education may increase the individual's ability to control the symptoms of the disease and enhance perceived self-efficacy.[81] The patient should also be educated in techniques of joint protection and energy conservation. Splinting is used for pain relief, correction of deformities, after surgery, to reduce stress on the joints, and to improve hand function.[87,88,104] Surgery is performed in the early stages of RA to remove the diseased synovium.[104,107] In the later stages of the disease, arthroplasty and arthrodesis are performed to correct deformity and relieve pain.[105] Exercise also plays an important part in maintaining and improving hand function.[112,116]

CHAPTER V

CONCLUSION

Rheumatoid arthritis is a chronic rheumatic disease that affects primarily the small synovial joints of the body. The joints of the hand and wrist are most commonly involved but any synovial joint can be affected.[2] The disease can develop at any age. Juvenile RA is defined as a disease that has its onset before 16 years of age.[1,8] The onset of adult RA is usually between 25 and 50 years of age and affects females more frequently than males.[1] The prevalence of the disease increases with increased age.

The etiology of RA is unknown.[45] Several theories have been postulated to explain the origins of and susceptibility to the disease including viruses, RF, genetics, and environmental factors.[7,39,44,46-54] The signs and symptoms of RA include pain, inflammation, joint effusion, limited range of motion, destruction of bone and articular cartilage, and attenuation of ligaments and joint capsules.[5] The disease can also present with systemic complications.[3,4] The symptoms can be mild or severe, intermittent or continuous.[6] Most individuals experience exacerbations and remissions of symptoms.[7]

The person with rheumatoid arthritis in its later stages usually experiences deformity in the affected joints.[74-78] In the hand, these deformities often result in a loss of function which can interfere with the person's lifestyle.[38] These

individuals have increased medical expenses, lost productivity at work, and higher rates of work disability than the general population.[35-41] There is also a higher mortality rate associated with RA.[23,42]

Physical therapy treatment of RA involves a variety of treatments including patient education, splinting, post-surgical rehabilitation, and exercise. Patient education can help the individual with RA understand the basic pathology of RA and management of the disease.[79-82] Splinting can be used to aid in maintaining correct joint alignment and reducing stress on the joints.[87,88] Surgery can also used to help minimize deformities.[66,104] After surgery, there is often a period of immobilization.[66,104,105,107,108] Physical therapy treatment may or may not be a part of the patient's post-surgical rehabilitation program. Exercise can be used to help maintain range of motion and strength.[112,116] Modalities can reduce pain and stiffness and have been shown to be beneficial when used in combination with an exercise program.[116,117] The exercise program should be tailored to meet the specific needs of the individual and help prevent complications of the disease.

The physical therapist (PT) often spends a considerable amount of time with the patient. It is this author's opinion that the relationship that develops between the physical therapist (PT) and the patient can be instrumental in helping the patient cope with his/her disease. If the patient feels comfortable confiding in the PT, information may be gained about the patient's lifestyle that can be used to help plan an appropriate treatment plan. If a treatment plan is not

conducive to the patient's lifestyle, it is likely that their will be a low level of compliance.

A treatment plan should include patient education about the pathology of the disease, exercise, joint protection, energy conservation, use of modalities, pain control, and coping techniques.[80-82] Vanio and Oka[75] found that continuing to use the hand during an exacerbation of RA symptoms resulted in increased deformity. Splints can be used to help rest the joints in proper alignment in order to minimize deformity. The PT can instruct the patient in the proper performance of exercise and energy conservation in order to protect the joints from injury. The PT can help the patient develop alternative methods for completing activities. This may enable the person to remain productive and reduce his/her independence on others.

Further research is needed to determine the etiology of RA and increase understanding of the disease process. Until a cure can be found, the PT can help the patient cope with the chronic manifestations of RA and perhaps provide methods of disease management that will enhance the patient's independence. A better understanding of RA will allow the development of more effective treatment methods.

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