THE UNIVERSITY OF MANITOBA

A STUDY OF THE IEMG AND TORQUE RELATIONSHIP OF THE ULNAR DEVIATOR MUSCLES OF THE WRIST IN NORMAL SUBJECTS AND PATIENTS WITH RHEUMATOID ARTHRITIS.

by

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A dissertation submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

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ABBREVIATIONS AND SYMBOLS

R.A. - Rheumatoid Arthritis.

ECU - Extensor Carpi Ulnaris Muscle.

ECRL - Extensor Carpi Radialis Longus Muscle.

ECRB - Extensor Carpi Radialis Brevis Muscle.

EDC - Extensor Digitorum Communis Muscle.

FCU - Flexor Carpi Ulnaris Muscle.

FCR - Flexor Carpi Radialis Muscle.

FDP - Flexor Digitorum Profundus Muscle.

FDS - Flexor Digitorum Superficialis Muscle.

PIP - Proximal Interphalangeal Joint.

MCP - Metacarpal Phalangeal Joint.

CMC - Carpometacarpal Joint.

EMG - Electromyography

IEMG - Integrated EMG signal, given in millivolts.

N - Newton, the measure of force.

T - Torque, given in Newton metres.

F - Muscle Force, given in Newtons.

X - The lever arm (perpendicular distance from the muscle tendon to the centre of wrist rotation), given in metres.

β - Slope of the straight line relationship of the log IEMG and T.

β X - Product of the slope and lever arm, given in log IEMG / Newton.

ABSTRACT

Weakness of the ulnar deviator muscles of the wrist has been hypothesized as a cause for the radial rotation deformity occurring in the wrist joint of patients with Rheumatoid Arthritis (Shapiro, 1970).

This investigation studied the integrated electromyogram (IEMG) and torque (T) relationship of the Extensor Carpi Ulnaris (ECU) and Flexor Carpi Ulnaris (FCU) muscles in normal subjects and patients with Rheumatoid Arthritis (R.A.). Twenty experiments were conducted on 14 normal subjects, and 6 patients with R.A. The total isometric torque of the combined ulnar deviators was measured during repeated contractions of varying magnitudes. The corresponding electromyographic signals from the two muscles were integrated. Each experimental period consisted of 4 test runs, with each run consisting of 12 to 16 contractions.

The results showed that a semi-logarithmic plot of the IEMG - T data was superior to the usual linear plot over all the torque ranges. The FCU contributed the greatest torque, but the ECU initiated the torque in at least 12 of the 20 subjects. The R.A. subjects showed a significant decrease in the maximal total torque (P=.05), due mainly to a decrease in the maximal FCU force (P=.01), while the ECU maximal force was not significantly reduced. The log IEMG / unit of force for the FCU was greater in the R.A. subjects (P=.05), and it was concluded that this was due to decreased FCU muscle bulk, with no change in the intrinsic properties of the muscle.

ACKNOWLEDGEMENTS

A number of people have contributed to this thesis, but I would especially like to thank Mr. R. Daher, for his assistance in the design and construction of the hand apparatus, Mrs. Gwen Smith for a tremendous job of typing, and to all the staff and patients who gave so willingly of their time to act as subjects for this study.

I would also like to thank Miss Joan Edwards for her endless patience and encouragement throughout this past year, and the Canadian Arthritis Society, for without their assistance this work could not have been undertaken.

Last, but by no means least, I would like to express my appreciation to my advisor, Dr. N. F. Clinch, his continued guidance, combined with an innate ability to demonstrate simplicity even in the most difficult areas, has made this a very enjoyable and memorable learning process.

DEDICATION.

I wish to dedicate this thesis to the memory of my father, Fredrick H. Dandy, who believed that "there was only one way to do a task - the right way".

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1.0. INTRODUCTION

"Man's place in nature is largely writ upon his hand, and many of the simplest and most familiar details of homely knowledge become important when we examine our hands with the critical spirit we would adopt towards the members of some strange and uncommon beast", (Wood-Jones, 1920).

1.1. PURPOSE OF THE STUDY

Weakness of certain muscles of the wrist, namely the ulnar deviators, the Extensor Carpi Ulnaris (ECU) and the Flexor Carpi Ulnaris (FCU), has been suggested as one of the primary predisposing factors in the development of ulnar drift of the fingers in patients with Rheumatoid Arthritis, (Shapiro, 1970).

This investigation was undertaken to study the ECU and FCU muscles of human subjects, under isometric conditions, and to evaluate the relative contribution of each muscle to the development of ulnar deviation torque of the wrist.

The relationship between the electrical activity and force of both muscles was investigated in a group of normal subjects and a group of patients with Rheumatoid Arthritis (R.A.), to test Shapiro's hypothesis, and to attempt to determine whether both muscles were equally involved in the process.

1.2. REVIEW OF THE LITERATURE

1.2.1. THE NORMAL WRIST JOINT

The articulated forearm and hand are illustrated in Figure 1,1. The wrist joint (Radio-carpal) is composed of the radius, fibro-cartilagenous triangular disc covering the head of the ulna, and the bones of the proximal carpal row, which are shown in Figure 1,2.

The wrist joint has two degrees of freedom of movement, one about an antero-posterior axis, the other a transverse axis.

The movements of the wrist joint are supplemented by the mid-carpal articulation, between the proximal and distal row of carpal bones, which also has two degrees of freedom of movement. The mid position of the Radio-carpal joint corresponds to slight flexion and ulnar deviation, however when the mid-carpal and radio-carpal articulation are taken together, the combined mid-position is 12 degrees extension and 3 degrees ulnar deviation, (Recklinghausen, 1920).

The axis for lateral motion of the wrist is perpendicular to the radius, in an antero-posterior direction at the head of the capitate bone, and parallel to the shortest diameter of the articular surface of the radius. The axes for both antero-postero and lateral motion of the wrist are relatively constant, and distal to the centre of the wrist joint, resulting in a gliding element in the movement of this joint, (Steindler, 1955).

When the hand is abducted radially, the proximal carpal row glides to the ulnar side. When the hand is adducted ulnarly, the proximal carpal row glide to the radial side. Similarly in wrist flexion and

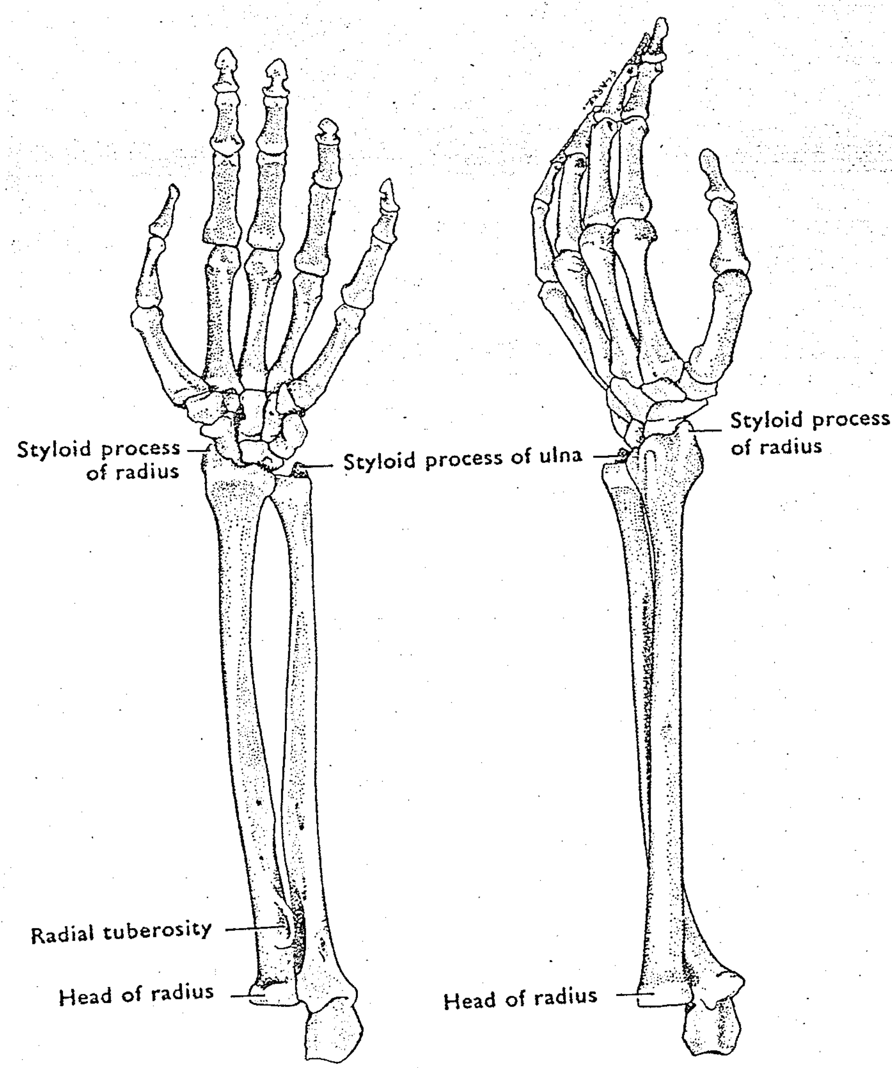
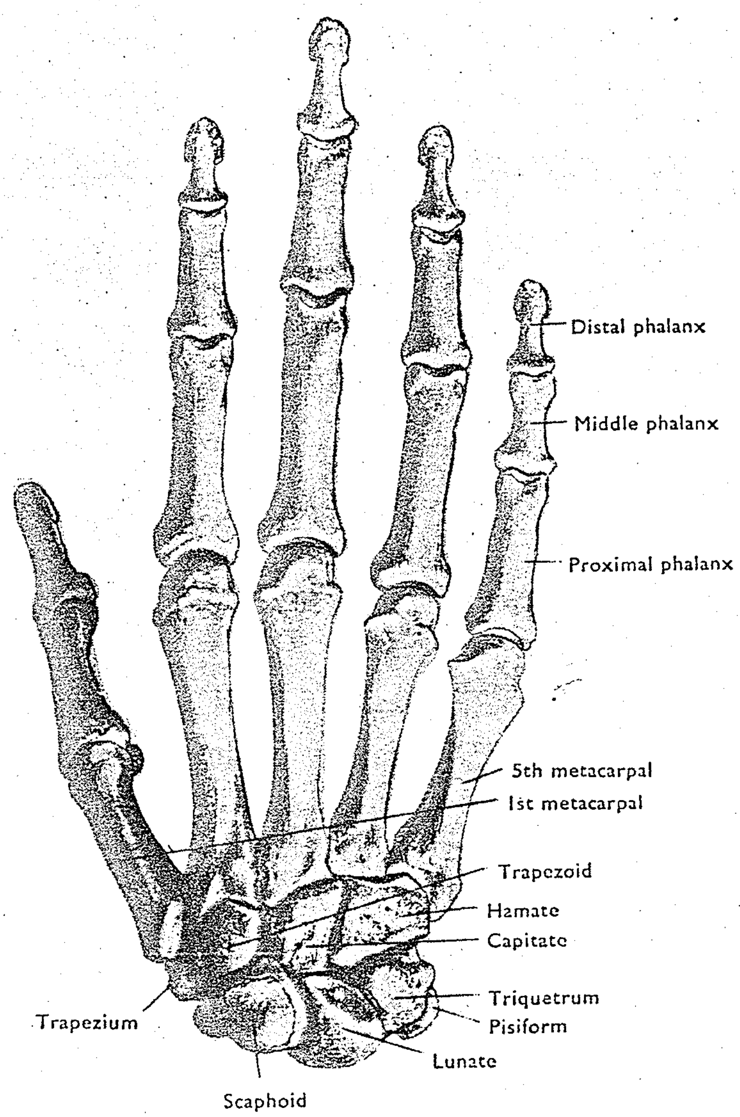


Figure 1,1. THE LEFT FOREARM AND HAND IN SUPINATION & PRONATION.



Dorsal aspect of bones of right hand.

Figure 1,2. BONES OF THE DORSAL ASPECT OF THE RIGHT HAND.

extension the gliding element prevails, in flexion the proximal row glides dorsally, and in extension glides volarly, (Lanz and Wachsmuth, 1935).

In lateral motion of the wrist, the scaphoid undergoes the greatest shift, in ulnar deviation the scaphoid shifts radially as much as one centimetre, (Fick, 1911).

Braune and Fischer (1887) described the combined ranges of the wrist and mid-carpal articulations, noting that flexion occurred mainly at the wrist joint, and extension at the mid-carpal joint, with ulnar deviation occurring principally at the wrist joint, and radial deviation at the mid-carpal joint.

The general arrangement of the muscles acting on the wrist and hand are illustrated in Figure 1,3. The particular arrangement of the ulnar deviator muscles, the ECU and FCU are shown in Figure 1,4. In this Figure the elbow is flexed and the forearm pronated, which was the test position described in section 2.3.2.

The ECU occupies a superficial position on the dorsal aspect of the forearm, lying lateral to the posterior border of the ulna. It has an extensive origin from the lateral epicondyle of the humerus, the fascia and intermuscular septum overlying this area, and from a thick aponeurosis of the posterior border of the ulna, distal to the insertion of anconeus muscle. The muscle occupies the middle one-half of the ulnar border and the origin extends to 6 to 8 cms. above the ulnar styloid process. The entire muscle belly is enclosed in a fascial covering. The tendon arises at the middle of the forearm, but the fibres descend in a pinniform manner to just above the ulnar styloid process. Here, the

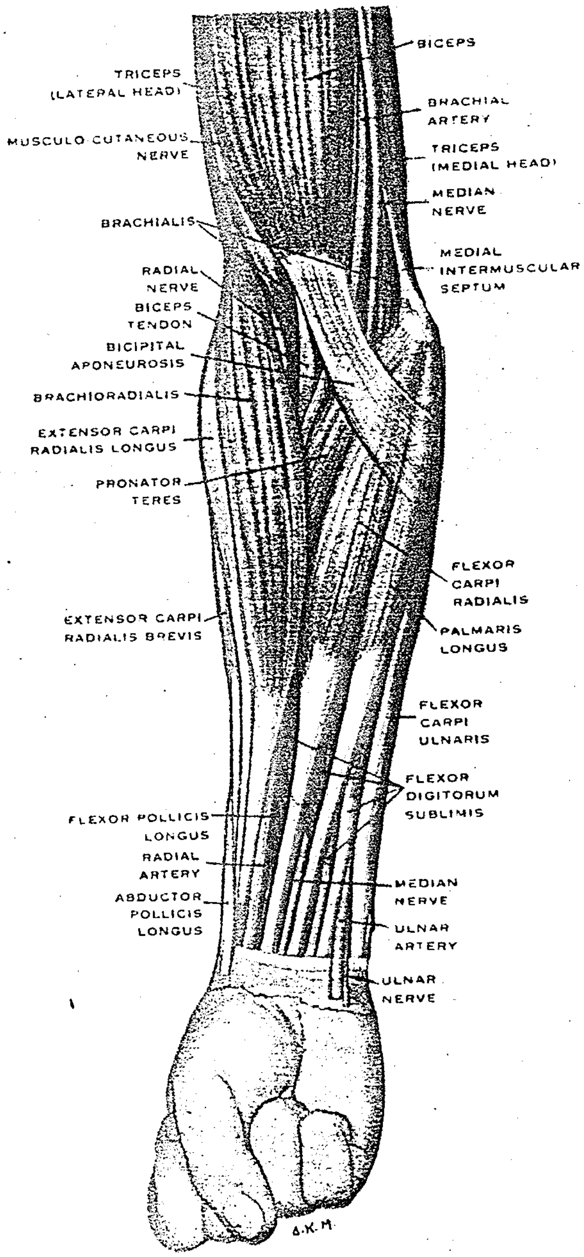


Figure 1,3. THE MUSCLES OF THE FOREARM.

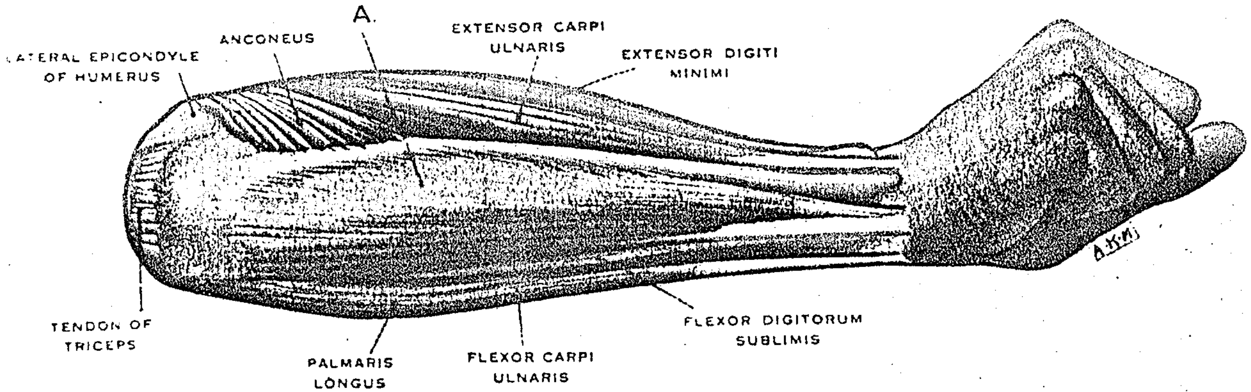


Figure 1,4. THE ECU AND FCU MUSCLES OF THE RIGHT FOREARM.

tendon, free of muscle fibres, passes deep to the extensor retinaculum, in a separate synovial sheath from the other tendons, and inserts into a tubercle on the base of the fifth metacarpal, towards the ulnar aspect of the base, (Gray, 1967). The ECU is penniform in shape with the fibres passing downwards and laterally to insert on the lateral side of the tendon, (Kaplan, 1965).

The nerve supply to the ECU is from the posterior interosseus nerve which enters the muscle 8 to 10 cms. distal to the lateral epicondyle of the humerus, (Brash, 1955). The blood supply enters the muscle through the same neurovascular bundle, however Kaplan (1965) noted that the blood supply to this muscle was less abundant than to the other muscles of the forearm.

The FCU is a flattened muscle, occupying the volar and ulnar aspect of the forearm. It is a superficial muscle arising by two heads, a humeral and ulnar head, connected by a tendinous arch. The humeral head is small, and arises from the medial epicondyle of the humerus, by the common extensor tendon. The ulnar head arises from the medial margin of the olecranon process, and the upper two-thirds to three-fifths of the posterior border of the ulna, by an aponeurosis common to it and the ECU muscle, and from the inter-muscular septum. The humeral fibres pass directly downwards to converge on the tendon, the ulnar fibres pass downwards and laterally, converging on the tendon on the medial side at the level of the wrist joint. The tendon is inserted into the pisiform bone, with additional ligamentous attachments to the hamate and fifth metacarpal (Gray, 1967).

The nerve supply to the FCU is from the ulnar nerve, usually two to three branches, one branch supplying the humeral head enter the muscle 2 to 3 cms. below the lateral epicondyle of the humerus. The second and third branches enter the muscle 3 to 4 cms. below the epicondyle, and occasionally a more distal branch enters the muscle 7 to 8 cms. below the epicondyle. The blood supply to the FCU is from the ulnar artery, entering the muscle at the same neurovascular hila, (Brash, 1955).

1.2.2. JOINT INVOLVEMENT AND MUSCLE WEAKNESS IN RHEUMATOID ARTHRITIS

Rheumatoid Arthritis has been defined as a sub-acute or chronic systemic inflammatory disease, manifested by joint involvement and muscle weakness, (Copeman, 1970). The etiology of the disease is unknown, the most widely accepted causative hypothesis being a defective auto-immune mechanism, (Hollander, 1972). The disease course is varied, with remissions and exacerbations of the inflammatory rheumatoid process, the prognosis is also varied, with 15 to 20% of patients suffering unrelenting disease progression, leading to wheelchair or bed-ridden existence, (Duthie et al., 1964).

1.2.2.1. Joint Involvement in R.A.

Copeman (1970) described the joint inflammatory pathology in three stages. The earliest change occurring in the synovial membrane, with increased formation of synovial fluid, effusion in the joint cavity, resulting in the eventual proliferation of synovial tissue. These changes also occur in the synovial tendon sheaths and bursa. The synovial membrane gradually becomes thickened and fibrotic, cartilage is eroded by the extension of granulation tissue, and adhesions are formed between the membrane and joint capsule, and between tendons and tendon sheaths. Finally, the cartilage may become completely destroyed, and with the added destruction of ligamentous attachments, the joint becomes completely unstable, or progress to fibrous or bony ankylosis.

The joints of the hands (PIP and MCP), feet, and wrists are commonly affected early in the disease, (Hollander, 1972). Copeman (1950) reported wrist involvement early in the disease process of 76% of R.A. patients studied. Flatt (1968) reported a 75% incidence of wrist involvement in R.A. Buchanan and Boyle (1971) in a study of 50 patients, reported a 63% incidence of PIP involvement, 80% incidence MCP joint involvement, 42% incidence CMC involvement, and an 83% incidence of wrist joint involvement.

The specific deformities are related to the location and intensity of the destructive process (Kessler et al., 1965; Ansell, 1969; Bywaters, 1969), though the deformity of ulnar drift of the fingers is one of the most consistent and visible hallmarks of the disease (Buchanan and Boyle, 1971).

Various etiological theories have been proposed to explain this deformity. Gravity, pressure, fatigue and weakness of the radial intrinsic muscles of the hand, and laxity of the capsule of the MCP joint are all factors which have been implicated (Fearnley, 1951; Lush, 1952; Rose and Wallace, 1952; Vaino and Oka, 1953; and Brewerton, 1957).

Smith et al., (1964, 1966) have postulated that the deformities of volar subluxation of the MCP joints, and ulnar drift, are the result of normally present forces acting on diseased joint restraints, specifically, the large volar forces generated by the flexor tendons during pinch and grasp, cause mechanical damage to the MCP collateral ligaments which serve as anchors for these forces. The radial collateral of the fifth finger is particularly subject to damage from the unopposed pull of the abductor digiti minimi muscle.

The intrinsic muscles of the hand have been further implicated as a causative factor by Boyes (1964) and Mannerfelt (1966), both authors emphasizing the possibility of spasticity of the ulnar intrinsic muscles as a primary etiological factor, however EMG studies by Wozny and Long (1966) appear to have disproved this hypothesis.

Hakstain and Tubiana (1966) suggested that the sloping contour of the MCP heads, combined with the lengthened radial collateral ligaments of the MCP joints may be the predisposing factor, in the development of ulnar drift.

All of the above mentioned authors centered their hypotheses on dysfunction of the hand, and in particular, the MCP joints and surrounding ligamentous and muscular structures.

Pekin and Zaifler (1963) noted that radial deviation of the wrist occurs in patients with R.A., and pointed out that although ulnar drift of the fingers is a recognized deformity, positional abnormalities of the wrist had not previously been emphasized.

Pahle and Raino (1969) studied 56 patients with R.A., in whom 69 wrists were fused. From radiological examination they demonstrated that when the wrist was fused in 5 degrees, or more, of radial deviation, 17 of 20 hands demonstrated ulnar drift of the fingers. Of 18 wrists fused in greater than 5 degrees of ulnar deviation, 14 hands showed radial drift of the fingers.

Shapiro (1970) demonstrated radial rotation of the wrist in a serial x-ray study of 100 hands of patients with R.A. Only those subjects with a minimum of 2 x-rays taken one year apart were included in the investigation, with 35 patients having a minimum of 3 serial x-rays at yearly intervals. The results showed that all the patients who had ulnar drift of the fingers, had radial wrist rotation, and radial deviation of the metacarpals. Shapiro suggested that weakness of the ulnar deviator muscles of the wrist was the probable cause for these findings.

The wrist is the key joint for proper hand function, the proximal carpal row being the link between the forearm and the hand (Swanson and Swanson, 1973).

The rheumatoid inflammatory process at the wrist follows the basic stages of joint inflammation (Bachdahl, 1967). Synovitis of the ECU tendon occurs early in the wrist pathology, and a synovitis between the head of the ulna and the triangular disc, and in the mid-carpal joints, can be readily demonstrated. With continued synovitis there is destruction of the ligaments of the distal radio-ulnar, radio-carpal, and mid-carpal joints. In advanced pathological states, the pannus invades all of the articular structures, leading to complete rupture of the triangular disc, and dorsal dislocation of the head of the ulna. Ligamentous destruction occurs with subluxation of the wrist and mid-carpal articulations (Bachdahl, 1967).

Confirmation of the ligamentous destruction has been provided by wrist arthrography. Harrison et al., (1971) demonstrated that in normal subjects the 3 compartments of the inferior radio-ulnar, radio-carpal, and mid-carpal joints are separate and do not communicate. However, their findings showed that in R.A. of the wrist, 90% of the 60 wrists examined had inferior radio-ulnar and wrist joint communications, and 70% had wrist and mid-carpal joint communications.

Pekin and Zaifler (1963) noted an ulnar shift of the scaphoid and lunate bones with rotation of the scaphoid into the palm. Since the proximal and distal carpal rows maintained their relationship, they noted that the effect of this shift was to rotate the hand upon the forearm in a radial and volar direction.

1.2.2.2. MUSCLE WEAKNESS IN RHEUMATOID ARTHRITIS

Rheumatoid Arthritis is manifested by pain, stiffness, joint inflammation and muscle weakness. The muscle weakness, and atrophy are early, constant and pervasive manifestations of the disease, that are evident virtually throughout the course of the disease and contribute significantly to the total disability, (Copeman, 1970; Hollander, 1972).

As early as 1873, Paget noted that the profound muscle wasting associated with inflammatory arthritis was considerably more than would be expected from disuse alone.

Myositis associated with R.A. was first described by Curtis and Pollard (1940), who noted nodules of cells from both muscle and nerve in patients with active R.A. Further work by Sokoloff et al., (1950) concluded that these lymphocytic cell infiltrations were not diagnostic of rheumatoid myositis. Cruickshank (1952) came to similar conclusions, however he reported a higher incidence of focal lesions in nerves than in muscles, with the lymphorrhagic foci in the muscle fibres located in the epimysium, on the surface of the muscle and tendons, and frequently related to arterioles, and venules, with the capillaries also containing lymphocytic foci.

Myopathy as defined by a marked reduction in muscle bulk, and a decrease in fibre size, has also been described as a characteristic pathological feature in the absence of other findings, (Haslock et al., 1970). In this study, group atrophy was also present, and often so striking as to suggest denervation. The motor innervation was normal to the level of the motor end-plates, which were often abnormal either as distal sprouting, or deformity. Haslock et al., (1970) found that the reduction in muscle fasciculi was accompanied by increased epimysial connective tissue.

Selective muscle fibre group atrophy has been reported in R.A. Haslock et al., (1970) found selective type 2 fibre atrophy, which was confirmed by Eldstrom and Nordemar (1974). Inactivity by immobilization is known to cause atrophy of both fibre types, (Karpati et al., 1968 and Patel, 1969). Bundschu (1971) demonstrated a mixed atrophy of muscle associated with joint dysfunction in the presence of arthrosis, Edstrom (1970a) showed a type 1 fibre atrophy following ligamentous knee injury. Selective type 2 fibre atrophy has been reported in other neuromuscular disorders. Brooke and Engel (1969) reported type 2 atrophy in Myasthenia Gravis, and Periodic Paralysis, and Eldstrom (1970b) has