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MRI Investigation of Computer Users With and Without Chronic Wrist Pain

Ronald A. Burgess
Western University

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Ronald A. Burgess

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Certificate of Examination

Supervisor	Examiners
Dr. R. Terry Thompson	Dr. Peter Canham
Advisory Committee	
Dr. Peter Canham	Dr. David Holdsworth
Dr. Gary Rollman	Dr. Said Osman
Dr. James Johnson	Dr. Peter Keir

The thesis by

Ronald Alan Burgess

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Abstract

Epidemiological studies have demonstrated a link between repetitive work and upper extremity musculoskeletal disorders (UEMSD), but the issue of causality remains controversial. Studies have shown an association between restricted wrist range of motion (ROM) and UEMSD in computer users. These restrictions were attributed to increased antagonist muscle tension.

The first objective of this thesis was to compare wrist flexion ROM in computer users with UEMSD versus asymptomatic individuals with minimal computer exposure. The UEMSD group exhibited significantly reduced wrist flexion compared to controls. Measures of flexion with a supine forearm posture reduced wrist flexion in both groups, but the reduction was approximately 100% greater in the UEMSD group. The effect of a supine forearm posture on wrist flexion is consistent with known biomechanical changes in the distal extensor carpi ulnaris tendon. We infer from these results that wrist extensor muscle tension may be elevated in UEMSD subjects compared to controls. Increased antagonist muscle tension would alter the dynamics of the wrist joint and may alter carpal kinematics.

The second objective of this thesis was to compare wrist flexion ROM and carpal flexion kinematics between UEMSD and asymptomatic computer users. The UEMSD group exhibited significantly reduced wrist flexion compared to controls. In both groups, maximum active wrist flexion decreased at the supine forearm posture compared to the prone posture. However, there was no significant difference in carpal flexion kinematics between groups. Abnormalities were tentatively identified in the kinematics MRI of 7 symptomatic wrists, and

subsequent clinical imaging confirmed presumably post-traumatic injuries in 3 wrists and 12 intraosseous ganglia in 3 wrists. The etiology of intraosseous ganglia is controversial, but several theories implicates chronic ligament stress. The prevalence of abnormalities in the symptomatic wrists suggests that MRI may be useful in detecting wrist abnormalities related to UEMSD symptoms.

The third objective of this thesis was to compare wrist flexion range of motion and the prevalence of MRI-identified abnormalities between UEMSD and asymptomatic computer users. The UEMSD group exhibited significantly reduced dominant wrist flexion compared to controls. There was no significant loss of flexion between the prone and supine forearm postures in either group, which may be related to the frequent finding of subluxation or dislocation of the distal extensor carpi ulnaris tendon. Extraosseous ganglia were common in both symptomatic and asymptomatic wrists, and there was no significant difference in their size. Intraosseous ganglia were more frequent and significantly larger in the symptomatic wrists. The symptoms associated with intraosseous ganglia may be related to their size, and symptoms were often well-localized to the sites of the larger intraosseous ganglia. The results of this study suggest that increased antagonist muscle tension in symptomatic computer users may increase ligament stress. To the best of our knowledge this is the first MRI study comparing the prevalence of wrist abnormalities between symptomatic and asymptomatic computer users.

Keywords

Repetitive strain injury, cumulative trauma disorder, upper extremity musculoskeletal disorder, magnetic resonance imaging, range of motion, wrist, intraosseous ganglia, extraosseous ganglia, muscle tension.

Co-Authorship

The following thesis contains material from one review paper in preparation for submission to Rheumatology Reviews (Chapter 2), one manuscript published in Biomed Central Musculoskeletal Disorders (Chapter 3), one manuscript that is being revised for resubmission to Biomed Central Musculoskeletal Disorders (Chapter 4), and one manuscript that is being revised for publication in Biomed Central Musculoskeletal Disorders (Chapter 5).

All of the data acquisition and analysis presented in this thesis were completed by Ronald A. Burgess. Clinical assessment of magnetic resonance images were completed by Dr. William Pavlosky and Dr. Greg Garvin.

The original manuscript presented in Chapter 2 was written by Ronald Burgess and R. Terry Thompson. The original manuscript presented in Chapter 3, was written by Ronald Burgess, R. Terry Thompson and Gary Rollman. The original manuscript presented in Chapter 4 was written by Ronald Burgess, R. Terry Thompson, and Greg Garvin. The original manuscript presented in Chapter 5 was written by Ronald Burgess, William F. Pavlosky, and R. Terry Thompson.

The business of a man of science in this world is not to speculate and dogmatize, but to demonstrate. To be sure, he sometimes needs the aid of hypothesis, but hypothesis, at best, is only a pragmatic stop-gap, made use of transiently because all the necessary facts are not yet known. The appearance of a new one in contempt of it destroys it instantly. At its most plausible and useful it simply represents an attempt to push common sense an inch or two over the borders of the known. At its worst it is only idle speculation, and no more respectable than the soaring of metaphysicians. *H.L. Mencken*

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List of Abbreviations

α angle of departure of M_z from z-axis towards xy-plane(tip angle)

ANOVA analysis of variance

B_1 time-varying magnetic field

B_o static magnetic field

cc cubic centimeter

CTD cumulative trauma disorder

df degrees of freedom

ECRB extensor carpi radialis brevis

ECRL extensor carpi radialis longus

ECU extensor carpi radialis

EOG extraosseous ganglia

F frequency

FCR flexor carpi radialis

FCU flexor carpi ulnaris

FID free-induction decay

FS fat saturation

FT Fourier Transform

γ nuclear gyromagnetic ratio

GRE gradient recalled echo

G_x, G_y, G_z magnetic-field gradient in x, y and z dimensions

Hz Hertz

IOG intraosseous ganglia

kHz Kilohertz

μ nuclear magnetic moment

MHz Megahertz

mm millimeter

M_o net magnetization along z-axis (unperturbed)

MRI magnetic resonance imaging

ms millisecond

M_{xy} transverse (xy) plane magnetization

M_z longitudinal magnetization along z-axis

p statistical power (significance level)

ppm parts per million

r correlation coefficient

RF radio-frequency

RSI repetitive strain injury

s second

SD standard deviation

SNR signal to noise ratio

STIR short-tau inversion recovery

SPSS Statistical Package for the Social Sciences

T Tesla

t time

T_1 time-constant for recovery of M_z along z-axis

T_2 time-constant for M_{xy} decay (Bo-independent)

T_2^* time-constant for M_{xy} decay (Bo-dependent)

TE echo-time

TR RF pulse repetition time

UEMSD upper extremity musculoskeletal disorders

μs microsecond

VOI voxel of interest

ω precessional frequency

ω_o Larmor precessional frequency

x', y', z' rotating frame of reference coordinates

x,y,z laboratory or "fixed" frame of reference coordinates

Chapter 1

Investigation of Chronic Wrist Pain Associated with Computer Use

1.1 Introduction to Thesis

Wrist dysfunction associated with overuse is considered part of a spectrum of musculoskeletal disorders known as upper extremity musculoskeletal disorders (UEMSD). Also known as repetitive strain injury (RSI), or cumulative trauma disorder (CTD), this disorder is associated with exposure to activity that involves repetitive motion of the hand and arm, and is not a new phenomenon.

"The maladies that afflict the clerks . . . arise from three causes: First, constant sitting, secondly the incessant movement of the hand and always in the same direction, thirdly the strain on the mind from the effort not to disfigure the books by errors or cause loss to their employers when they add, subtract, or do other sums in arithmetic... Incessant driving of the pen over paper causes intense fatigue of the hand and the whole arm because of the continuous and almost tonic strain on the muscles and tendons, which in course of time results in failure of power in the right hand. . . .

Bernardo Ramazzini, 1713 [1]

UEMSD encompass a variety of pain symptoms and dysfunction affecting the muscles, tendons, joints, nerves, and vasculature of the shoulder, arm, and hand

[2,3]. Although some symptoms of UEMSD can be attributed to specific causes (e.g. median nerve abnormalities, tendinopathies) with diagnostic testing, the majority of subjective symptoms cannot be verified [4,5]. This lack of objective clinical evidence often makes the diagnosis and treatment of UEMSD problematic. There are some who suggest that this lack of evidence, and the fact that women are most often affected, is indicative of a non-organic illness [6].

The overall prevalence of UEMSD is uncertain, due largely to the wide range of diagnostic criteria and nomenclature used to define the disorder [7,8]. Clinical studies have demonstrated a strong link between specific hand-intensive occupations and UEMSD. For instance, a sample of workers from various industries requiring repetitive low-load hand use found that 54% had evidence of musculoskeletal disorders [9]. The prevalence of at least one musculoskeletal symptom has been reported as 93% in dental hygienists [10], and 81% in computer users [11]. The risk of hand and wrist tendinopathies for people who perform repetitive, high-force tasks is 29 times greater than those who do not [12]. Repetitive, but low-force tasks such as computer use are also associated with a greater incidence of hand and wrist problems [11-14] .

1.2 UEMSD Symptoms

UEMSD symptoms may include forearm muscle fatigue and pain; tendon or tendon sheath pain; paraesthesia and/or pain in the wrist or hand [15]. Although, in some cases, the symptoms may be consistent with a specific clinical diagnosis (e.g., carpal tunnel syndrome, tendinopathies, lateral epicondylitis), in many cases the clinical findings are non-specific. According to McDermott [16]: “There is no agreement concerning the cause; the pathology is unknown; the clinical features are diffuse; there are no useful diagnostic investigations; and the prognosis is uncertain.”

The multitude of symptoms in UEMSD and the apparent differential susceptibility between individuals exposed to the same workload is suggestive of a complex multi-factor etiology. Although repetitive work appears to be the primary risk factor for UEMSD, other risk factors have been implicated including gender, stress, and other psychosocial factors [13,17-19]. Unfortunately, even when the predominant risk factor (i.e., repetitive work) is discontinued, many UEMSD patients continue to experience symptoms, with some becoming permanently disabled. This may be interpreted as evidence that the causal factor is not work-related, or that the work produced a chronic, but currently unidentifiable disorder.

1.3 UEMSD Risk Factors

1.3.1 Computer Use

The question of a causal relationship between computer use and UEMSD has not yet been resolved. Studies to date have been criticized as they are largely cross-sectional, often rely on surveys, few measure exposure, and objective clinical evidence is limited [20]. In a recent review Waersted et al. [20] examined the results of 22 studies that related computer exposure to objective clinical evidence of UEMSD and concluded that there is limited evidence for a causal relationship with regards to wrist tendinitis. However, the authors do note that several studies, both cross-sectional and prospective, demonstrate a relationship between computer use and wrist pain. The authors emphasize the need for studies examining the possible effect of computer use on the musculoskeletal system.

1.3.2 Gender

Females are 2 to 3 times more susceptible to develop UEMSD than males [21,22]. Lundberg [23] suggests that this overrepresentation of females may relate to the higher proportion of women engaged in occupations with known risk factors. The female tendency to develop UEMSD may also be due to a

cumulative effect related to the additional load of domestic duties [23,24]. However, there are some indications that females possess increased pain sensitivity compared to males [21].

1.3.3 Psychosocial Factors

There is some evidence that psychosocial factors may play a role in the development and maintenance of UEMSD. Bongers et al. [17] conducted a meta-analysis of 28 epidemiological studies to determine what psychosocial risk factors were most likely to contribute to UEMSD. The authors found that stress, both work-related and non work-related were the factors most strongly associated with the development of UEMSD. High job demands without perceived control, were also associated with the development of UEMSD.

However, it is difficult to dissociate stress from any chronic illness, especially illness that has chronic pain as its defining feature. Only one prospective study was included in Bonger's meta-analysis, which examined the incidence of eye and musculoskeletal problems in video display terminal operators over a six-year period [25]. The authors found that increased keyboard use increased the risk of hand/wrist problems (RR 2.7, CI 1.3, 5.6). However, the study's authors did not examine psychosocial factors that may have contributed to the development of the hand/wrist problems, and thus adds little to what is known, or suspected regarding the influence of psychosocial factors in the etiology of UEMSD.

A more recent prospective study which measured psychosocial factors looked at the incidence of forearm pain over a two-year period in people randomly sampled from a general medical practice [18]. The authors found that work-related exposure was associated with higher risk of forearm pain as expected. However, the risk ratios for repetitive wrist motion (RR = 3.4, CI = 1.3, 8.7), and repetitive arm motion (RR = 4.1, CI = 1.7, 10) were exceeded by dissatisfaction with support from colleagues or supervisors (RR = 4.7, CI = 2.2, 10).

Obviously, in every case of UEMSD related to work there exists an activity to which the disorder can be attributed. It appears that stress and anxiety are potential causative psychological factors in the development of UEMSD and it is worthwhile to consider their potential physiological effects.

1.3.4 Stress and Anxiety

Individuals react to adverse situations differently, depending on whether they perceive a given situation as potentially threatening. Their perceptions can be influenced by their current mood, and prolonged stress can produce psychological stress reactions that are known to have detrimental effects on the body. In a review of UEMSD and stress, Lundberg [23] suggests that there may be an association between perceived stress, stress hormone release, and prolonged muscle activity. The author suggests that increased muscle tension may result from a combination of physical and mental factors. Increased trapezius muscle activity has been associated with higher levels of perceived stress in a study of female supermarket cashiers of whom 70% report neck and shoulder disorders [26]. Lundberg [23] suggests that prolonged muscle activity may play a primary role in the development of chronic musculoskeletal disorders.

It is known that prolonged muscle activation can produce muscle fatigue and ischemia, and the resulting pain is thought to occur due to the lowering of the activation threshold of nociceptors located in the muscle interstitium [27]. It is also thought that increases in the concentration of muscle metabolites within the interstitial fluid lowers the threshold of affected nociceptors [28]. However, muscle fatigue usually requires high levels of muscle activation, not the low levels of activation common to many UEMSD.

Elevated agonist muscle activity has been demonstrated in individuals with UEMSD [29,30]. Mackinnon and Novak [31] suggest that prolonged abnormal postures may affect muscle tension due to muscle length adaptation. It has been

shown that animal skeletal muscle immobilized in a shortened state shortens due to the loss of serial sarcomeres, and the rate of such loss increases when the muscle is chronically activated [32,33].

1.4 Functional Limitations in UEMSD

A common clinical finding in chronic UEMSD patients is decreased wrist range of motion (ROM) [13,14]. This decreased ROM is thought to be the result of wrist muscle shortening and/or co-contraction, rather than pain avoidance. Pascarelli & Kella [14] suggest that decreased wrist ROM could be useful as a diagnostic test for UEMSD, and may be responsible for the various symptoms. Barthel et al. [13] go further in suggesting that agonist-antagonist muscle shortening may be a possible pathomechanism in UEMSD, but do not elaborate further.

Presumably, increased antagonist co-contraction or shortened musculature could impart increased forces within the wrist joint, resulting in pain and dysfunction. It could also be argued that people with UEMSD develop muscle shortening because of their efforts to avoid pain by limiting the extent of their wrist motion. The individual simply learns to avoid excessive flexion or extension in order to avoid eliciting painful symptoms. Wrist range of motion is known to decrease with age, and wrist disorders are not uncommon in the elderly [34].

Although several authors [13,14] have described restrictions in wrist ROM in UEMSD patients, these restrictions were not quantified, nor were they compared with asymptomatic individuals exposed to the same occupational demands. Restricted wrist ROM may be due to internal derangement or abnormalities in the motion (kinematics) of the wrist joint. There do not appear to be any studies examining the possibility of altered wrist kinematics in UEMSD subjects.

1.5 Magnetic Resonance Imaging Studies of UEMSD

Although UEMSD is considered to be a soft-tissue disorder, the use of magnetic resonance imaging (MRI) in UEMSD has been limited to specific clinical entities such as lateral epicondylitis [35], or carpal tunnel syndrome [36-39]. There do not appear to be any studies using MRI to examine non-specific UEMSD, despite the demonstrated clinical usefulness of MRI in diagnosing disorders of the wrist [40]. Also, there do not appear to be any MRI studies investigating UEMSD related to specific occupations, which could potentially identify patterns of abnormalities relating to stereotypical hand use.

1.6 Wrist Anatomy and Biomechanics

There appears to be an association between UEMSD and repetitive motion of the wrist. The obvious question of whether the biomechanics of the wrist are abnormal in individuals with UEMSD has not, to our knowledge, been investigated. The biomechanics of the wrist are not fully understood, due largely to the wide spectrum of carpal bone motion observed in the normal wrist. However, it would be useful to determine whether carpal bone motion in individuals with UEMSD lies at one end of this spectrum.

Anatomically, the wrist encompasses the distal radius and ulna, the eight carpal bones, and the proximal bases of the five metacarpals as shown in Figure 1-1.

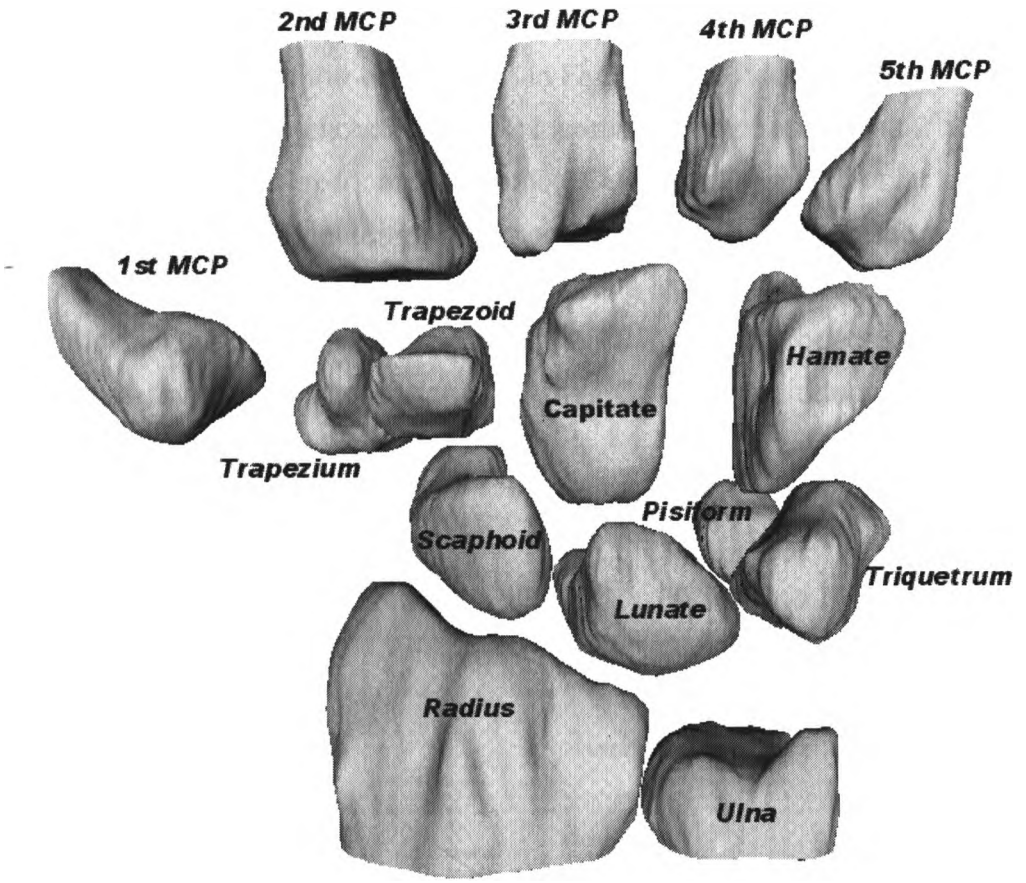


Figure 1-1. Carpal bone anatomy.

Computed tomography image showing a dorsal view of the right wrist showing the radius, ulna, carpal bones and metacarpal bases. The original image [41] was expanded to show the complex shapes of the various bones.

The carpal bones articulate with each other and with the radius to allow movement of the wrist. Wrist motion is controlled by the wrist motor tendons, of which there are three extensor tendons inserting onto the dorsal bases of the metacarpals as shown in Figure 1-2(a). The distal tendon of the extensor carpi ulnaris (ECU) is contained within a tendon sheath that is attached to the distal ulna proximally and the dorsal aspect of the triquetrum distally. Tensioning of the of the ECU tendon produces wrist extension and ulnar deviation, while tensioning of the extensor carpi radialis longus (ECRL) and brevis (ECRB) tendons produces wrist extension and radial deviation [42].

On the volar aspect of the wrist shown in Figure 1-2(b) there are two flexor tendons; the flexor carpi radialis (FCR) inserting on the base of the 2nd metacarpal, and the flexor carpis ulnaris (FCU) inserting on the volar aspect of the pisiform. The pisometacarpal ligament (LPM) attaches the pisiform to the base of the 5th metacarpal, and the pisohamate ligament attaches the pisiform to the "hook" of the hamate. Tensioning of the FCR tendon produces wrist flexion and radial deviation, while tensioning of the FCU tendon produces wrist flexion and ulnar deviation.

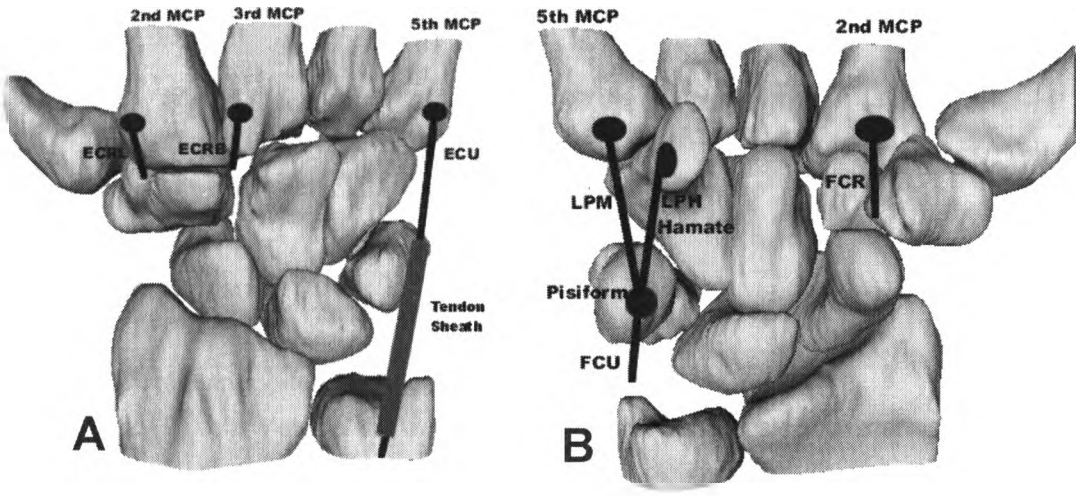


Figure 1-2. Carpal bone anatomy and tendon insertions.

Dorsal (a) and volar (b) views of the right wrist showing the insertions of the extensor tendons (ECRL, ECRB, and ECU) on the bases of the metacarpals (MCP); the insertion of the flexor carpi radialis (FCR) tendon on the 2nd MCP base, and flexor carpi ulnaris (FCU) tendon on the pisiform. The original image [41] was modified to show the ligament insertions.

The distraction forces transmitted by the flexor and extensor tendons create compression and result in complex motions of the carpal bones, or carpal kinematics. Carpal kinematics is governed by the tendon forces, morphology of the articulating surfaces, and the carpal ligaments. Carpal ligaments are classified into two types: Those attaching between adjacent carpal bones are known as intrinsic ligaments, while those attaching between non-adjacent carpal

bones or between carpal bones and the distal radius/ulna are known as extrinsic carpal ligaments. Figure 1-3 shows several of the numerous intrinsic and extrinsic carpal ligaments whose names, functions, and existence continue to be debated [43,44].

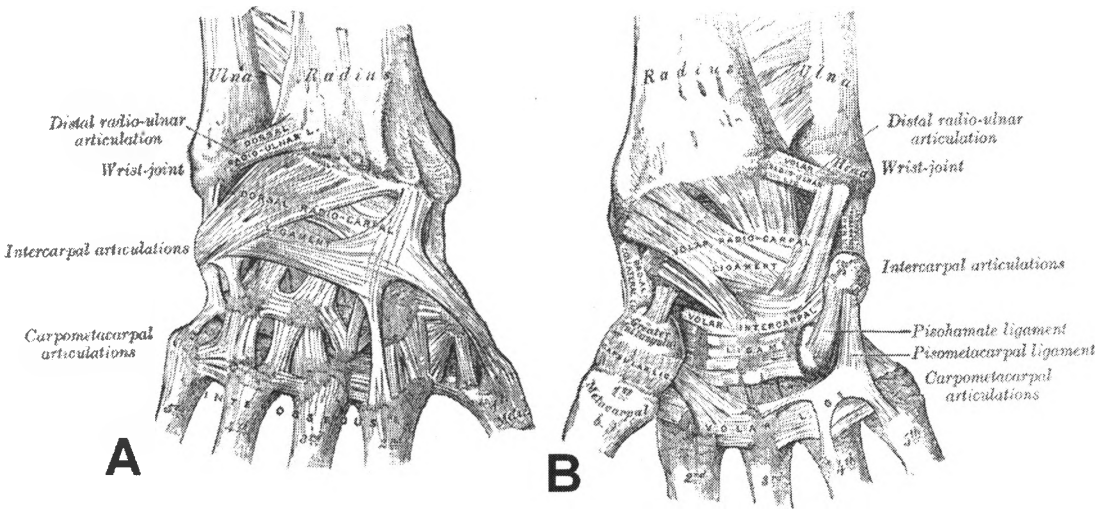


Figure 1-3. Dorsal and volar views of the carpal ligaments.

(A) Dorsal and (B) volar views of the wrist showing some of the intrinsic and extrinsic carpal ligaments [45].

A full understanding of carpal kinematics has proved challenging due to the inherent complexity of the wrist. The anatomist, William Cheselden (1795) commented, perhaps prophetically, that "*the carpus is composed of eight bones of very irregular forms, undoubtedly the properest that can be, yet why in these forms, rather than any other, no one has been able to shew.*"

Analysis of carpal kinematics can be simplified somewhat due to the limited mobility between the trapezium, trapezoid, capitate, and hamate by virtue of their stout intrinsic ligaments [46]. These four bones are collectively referred to as the distal carpal row. In contrast, there is greater mobility between the scaphoid, lunate, triquetrum, and pisiform, which comprise the proximal carpal row due to

greater laxity of their intrinsic ligaments. The distal carpal row articulates with the proximal carpal row at the mid-carpal joint, while the proximal carpal row articulates with the radius at the radiocarpal joint as shown in Figure 1-4.

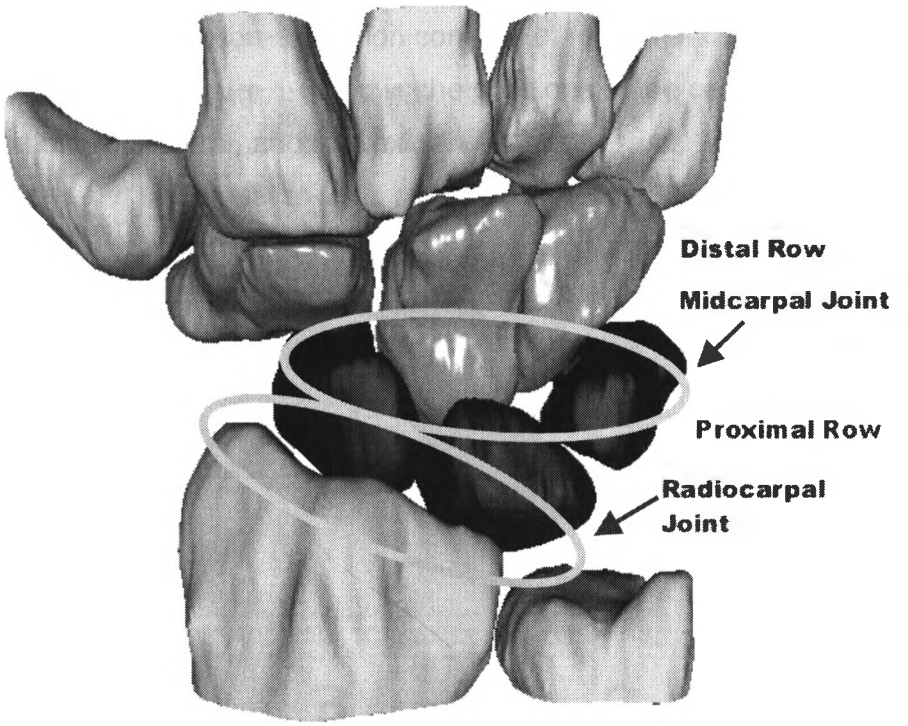


Figure 1-4. Dorsal view of the proximal and distal carpal rows.

Dorsal view of right wrist showing the proximal and distal carpal rows, as well as the radiocarpal and midcarpal joints.

1.6.1 Wrist Flexion-Extension Kinematics

Studies of carpal kinematics have focused primarily on two types of motion: flexion-extension and radioulnar deviation. The normal range of flexion is 70-90 degrees, while extension ranges from 60-80 degrees [47]. Wrist flexion-extension is normally described as the angle of the 3rd metacarpal with respect to the long axis of the radius. The 3rd metacarpal and capitate are tightly bound by stout intrinsic ligaments and move as a unit. In kinematics studies, radiocarpal

joint motion is often described in terms of motion of the lunate with respect to the radius (radiolunate joint), while midcarpal joint motion is described in terms of motion of the capitate with respect to the lunate (capitolunate joint).

Numerous ex vivo and in vivo studies of carpal kinematics in normal wrists have shown that wrist flexion-extension consists of motion at both the radiocarpal and midcarpal joints. Figure 1-5 shows the positions of the carpal bones in (a) extension, (b) neutral, and (c) flexion.

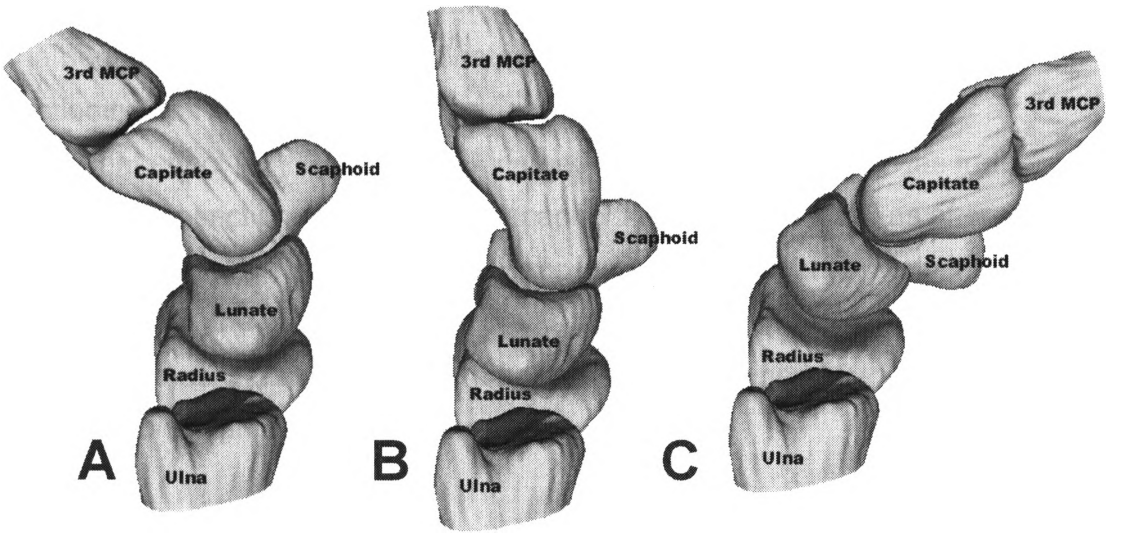


Figure 1-5. View of the central carpus (right wrist) in flexion and extension.
Sagittal view of the bones of the central carpus in (A) extended, (B) neutral, and (C) flexed postures.

Significant variations in the proportion of radiolunate versus capitolunate joint motion during wrist flexion-extension have been observed in normal wrists [48]. In some studies equal radiolunate (radiocarpal) and capitolunate (midcarpal) joint motion has been observed, while in others motion at the radiolunate or capitolunate joint predominates [49]. Although these differences between studies may relate to technique, there are large individual variations observed within

studies. Ferris [48] found that lunate motion during flexion-extension in 34 normal wrists varied considerably and suggested a bi-modal distribution.

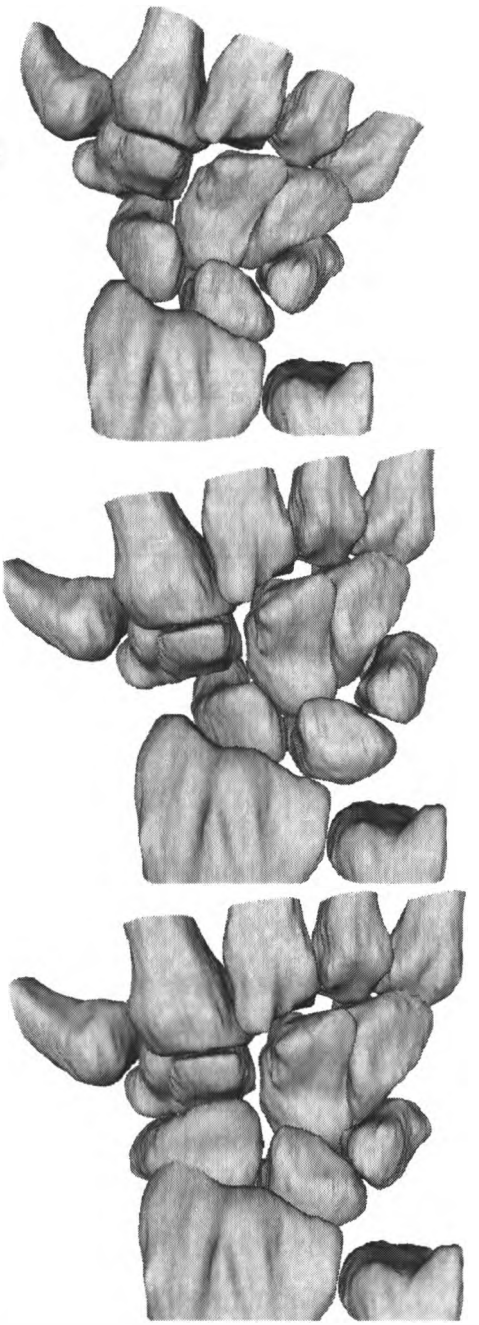
Motion coupling between the lunate and capitate is thought to be dependent on the integrity of the scapholunate ligament, as the scaphoid is considered to be the stabilizing link between the proximal and distal carpal rows. A kinematics model proposed by Kauer [50] suggests that during wrist flexion, the flexion forces of the distal carpal row are transmitted to the distal scaphoid via its articulations with the trapezium and trapezoid. The resulting rotation of the proximal pole of the scaphoid creates a flexion torque on the scapholunate ligament causing flexion of the lunate. However, ex vivo studies before and after sectioning of the scapholunate ligament showed either no effect [51], or a significant effect [52,53] on lunate kinematics. Lunate kinematics was further affected after sectioning of other periscaphoid ligaments, as well as after 1000 cycles of repetitive loading [52,53]. Cyclic loading is thought to produce plastic deformation of the remaining structures stabilizing the joint [53]. It appears that carpal kinematics during flexion are not entirely determined by the integrity of the periscaphoid ligaments.

Results from in-vivo studies have also shown mixed results. Wolfe [54] compared carpal kinematics between 5 healthy and 3 injured wrists with confirmed scapholunate ligament disruption. The authors found that relative midcarpal joint motion during wrist flexion was greater in the injured wrists compared to the normal wrists. Crisco [55] compared carpal kinematics in the injured and uninjured wrists of 8 subjects with confirmed scapholunate ligament disruption versus both wrists of 10 normal subjects. The authors reported that the relative radiocarpal joint motion was greater in both the injured and contralateral uninjured wrists compared to the normal subjects.

1.6.2 Wrist Radioulnar Deviation Kinematics

Radioulnar deviation is quantified as the angle of the 3rd metacarpal with respect to the long axis of the radius, and normal values are 15-20 degrees radial and 30-40 degrees ulnar deviation [47]. As the 3rd metacarpal and capitate essentially move as a unit it is convenient to describe radioulnar deviation kinematics in terms of the motions of the capitate and lunate with respect to the radius. Radioulnar deviation kinematics is complex due to out-of-plane motions that occur within the proximal carpal row.

As shown in Figure 1-6, when moving from (a) ulnar to (b) radial deviation the capitate translates radially, angulates radially, and extends while both the lunate and scaphoid translate ulnarly, angulate radially, and flex. Flexion of the scaphoid is thought to induce lunate flexion via the scapholunate ligament. In vivo studies have shown that wrist kinematics is highly variable between individuals. Craigen et al. [56] found that there is a spectrum of radioulnar deviation kinematics in both normal wrists and symptomatic, but arthroscopically normal wrists. Wrists with increased lunate translation had decreased scaphoid flexion (defined as “row” type) as shown in Figure 1-6.b, while wrists with decreased lunate translation had increased scaphoid flexion (defined as “column” type), as shown in Figure 1-6.c .



A. Ulnar Deviation

Translation of the proximal and distal carpal rows

B. Radial Deviation ("Row" type)

Primarily translation of the proximal carpal row
Minimal flexion of the scaphoid

C. Radial Deviation ("Column" type)

Primarily flexion of the scaphoid
Minimal translation of the proximal carpal row

Figure 1-6. "Row" versus "column" type wrist.

(A) Dorsal view of wrist in ulnar deviation; (B) "Row" type wrist in radial deviation; (C) "Column" type wrist in radial deviation.

Garcia-Elias et al. [57] confirmed Craigen & Stanley's results in a radiographic study of scaphoid motion in 60 normal wrists. They found that wrists with decreased scaphoid translation had increased flexion and vice-versa. They also demonstrated a linear relationship between measures of global wrist joint laxity and scaphoid motion. Lax wrists had more scaphoid flexion and less translation during radioulnar deviation. The authors suggest that the variability in kinematics may be due to ligamentous laxity, articular surface shape, and "the constraining effects of the muscles surrounding the joint" [57].

The possible role of variations in bone morphology on wrist kinematics has also been investigated. Two types of lunates have been identified: type II which has a facet that articulates with the proximal pole of the hamate, and type I which does not [58]. Nakamura [59] found that wrists with a type II lunate exhibited more proximal row translation during radial deviation than wrists with a type I lunate. However, Galley [60] observed the opposite effect; wrists with a type I lunate exhibited more proximal row translation during RUD than wrists with a type II lunate.

1.6.3 Carpal Kinematics and Tendon Loading

The effect of tendon loading on carpal kinematics has received little attention, and it is generally assumed that both agonist and antagonist muscle tensions are minimal during motion of the unloaded wrist. Moojen [49] suggests that differences in carpal kinematics between in vivo and ex vivo studies may be related to differences in tendon loading. The degree of tendon loading used in ex vivo studies ranges from 4 N (0.4 Kg) to 180 N (18.4 Kg) [49]. Short et al. [52] point out that the degree of tendon loading in in vivo studies is uncertain, and may vary between individuals.

The forces on the carpus are the product of the flexor and extensor tendon tensions and their respective moment arm lengths. The moment arm length is the perpendicular distance between the joint axis of rotation and the tendon. It has

been shown that the wrist flexor/extensor tendon moment arms are not fixed, but vary with wrist position [61]. This effect is due to changes in the joint axis of rotation as well as displacement of the tendons [61].

There are few studies examining the effect of changes in carpal kinematics on flexor/extensor moment arm lengths. A cadaveric study by Tang et al. [62] examined wrist flexor/extensor tendon excursions before and after sectioning of the scapholunate ligament and artificial widening of the scapholunate gap. Changes in both flexor and extensor moment arm lengths were observed, but no carpal kinematics data was acquired. Without experimental evidence one can only speculate what the effect of changes in carpal kinematics has on moment arm length.

As the capitolunate joint possesses a smaller radius than the radiolunate joint, motion at the midcarpal joint would presumably result in smaller flexion/extension moment arms than motion at the radiocarpal joint. As tendon excursion is the product of the moment arm length and the joint angle (in radians) it appears that motion at the midcarpal joint would reduce wrist flexor/tendon excursions, while motion at the radiocarpal joint would increase tendon excursions. This assumes that displacement of the tendons is not impeded. If increased motion at the capitolunate joint reduces tendon excursion then the corollary may also be true; reduced tendon excursion may increase capitolunate joint motion. For example, if the wrist extensor tendons were under increased tension, then motion at the capitolunate joint may increase during wrist flexion. This assumes that the resulting forces are sufficient to overcome the ligamentous constraints, an outcome that may only occur after many cycles of repetitive loading.

1.6.4 Summary

The kinematics of the wrist is complex and exhibits considerable variability in normal wrists. This variability relates to the relative proportions of motion at the

radiocarpal and midcarpal joints. Carpal kinematics is highly dependent on the integrity of the carpal ligaments, less dependent on bone morphology, and is affected by repetitive loading. The possible effects of repetitive loading combined with increased tendon load in vivo have not been investigated.

1.7 Magnetic Resonance Imaging

Plain film X-ray, X-ray computed tomography (CT), and magnetic resonance imaging (MRI) have all been used to acquire wrist kinematics data. Both MRI and computed tomography are superior to X-ray as they provide 3-dimensional data. MRI can produce the spatial resolution of CT; (0.2 mm), but the long scan times required can introduce artifacts due to patient motion. However, with MRI there is no exposure to ionizing radiation.

MRI is a noninvasive imaging modality that is based on the detection of a time-varying magnetic field produced by hydrogen nuclei (protons). MRI is the preferred modality for imaging the wrist due to the high soft-tissue contrast obtainable. This contrast is determined by acquisition dependent and tissue-specific factors governing the amplitude of the magnetic resonance (MR) signal. Spatial encoding localizes the MR signal to regions or voxels within the imaging volume, and the signal amplitudes are converted to pixel intensities on a display monitor. The differences or contrast between pixel intensities are generally representative of biological differences in tissue type, although other factors may contribute. The following sub-sections provide a brief description of nuclear magnetic resonance and MRI.

1.7.1 The Proton Dipole

The hydrogen nucleus consists of a single proton that possesses a nuclear magnetic moment due to its nuclear spin. In the presence of an external magnetic field, this magnetic moment experiences a torque that causes the axis

of the moment to precess around the external magnetic field, similar to the precession of a gyroscope due to the influence of gravity. The frequency of this precession is known as the Larmor frequency (Equation 1.1).

Equation 1-1. Larmor precession frequency.

$$v_0 = \frac{\gamma}{2\pi} B_0$$

Where υ_0 is the precession or Larmor frequency in Hertz, γ is the gyromagnetic ratio in radians/second/Tesla, and B_0 is the strength of the external magnetic field in Tesla. At a typical clinical field strength of1.5 Tesla (T) the Larmor frequency for protons is 63.86 MHz.

1.7.2 MR Signal Induction

Proton dipoles have a tendency to align with or against the external magnetic field (B_0). A small excess (1 ppm) of protons align with B_0 creating a net magnetization vector known as the longitudinal magnetization (M_z.). With respect to a constant or laboratory frame of reference, these protons precess around B_0. Although the protons are precessing, the magnitude of M_z remains constant because of the lack of phase coherence between the protons comprising M_z.

The application of another magnetic field B_1 (i.e., an RF field oscillating at the Larmor frequency) oriented perpendicular to B_0 will sum with B_0 to create a new effective magnetic field. As the frequency of B_1 is equal to the protons' precession frequency, the magnetic field experienced by the protons is constant. It is therefore convenient to describe the behavior of the protons using a frame of reference rotating around B_0 at the Larmor frequency, which is known as the rotating frame.

With respect to the rotating frame, the application of B_1 causes M_z to tip away from its alignment with B_0 towards the transverse plane. This creates a net magnetization vector with a component in the transverse plane. The net magnetization vector will now precess around the effective magnetic field at a frequency proportional to the strength of B_1. After the B_1 field is removed, the transverse component of the net magnetization vector, which is referred to as the transverse magnetization (M_t), will begin precessing around B_0 in the transverse plane at the Larmor frequency,

The transverse magnetization is composed of protons precessing in phase, which creates a time-varying magnetic field that will induce a current in a receiver coil to create the MR signal. The amplitude of the MR signal depends on several factors including the number of protons in the sample (proton density or PD); the magnitude of M_z prior to tipping, which is dependent on the strength of B_0; the tip angle, which is dependent on both the strength of B_1 and its duration (length of time that B_1 is kept on); and the phase coherence of the protons composing the transverse magnetization.

1.7.3 Free Induction Decay

After the B_1 field is removed,the MR signal begins to decay, which is known as free induction decay or FID (i.e., "free" of B_1). Without the influence of B_1 the protons will begin to simultaneously dephase and realign with B_0, Proton dephasing reduces the magnitude of the transverse magnetization, which is known as transverse relaxation or T_2 decay. The simultaneous regrowth of the longitudinal magnetization is somewhat confusingly known as longitudinal relaxation or T_1.

Although it appears that the T_2 and T_1relaxations are reciprocal and would occur at the same rate, this only occurs in samples of pure water. The magnitude of the transverse magnetization vector is dependent not only on the number of

precessing protons, but also on their degree of phase coherence..The loss of phase coherence occurs more rapidly than the regrowth of the longitudinal magnetization, therefore T_2 effects dominate.

1.7.4 Transverse (T_2) and Longitudinal (T_1) Relaxation

Transverse Relaxation (T_2)

The time constant T_2 defines the rate of decay of the transverse magnetization vector M_t, which decays exponentially from its initial value $M_t(0)$ according to Equation 1.2.

Equation 1-2. Transverse relaxation (T_2).

$$M_t(t) = M_t(0)e^{-t/T_2}$$

After a time T_2, M_t decays to approximately 37% of its initial value $M_t(0)$. There are essentially two mechanisms contributing to T_2 decay; a loss of phase coherence between the precessing protons, and a decrease in the number of protons comprising M_t as protons realign with B_0. Proton dephasing can result from a number of mechanisms. Changes in the precession frequency (and phase) can result from changes in the local magnetic field due to slow interactions with other spins, changes in the chemical environment, or energy exchange between two spins [63].

These dephasing mechanisms are inherent properties of the tissue and determine its "true" T_2 value. In practice, there is an additonal dephasing mechanism due to inhomogeneities in the main magnetic field. These field inhomogeneities can be due to intrinsic defects in the uniformity of the field, or deformation of the field due to the presence of materials with different magnetic susceptibilities. The effective or apparent T_2 is known as T_2^*, and is primarily

what determines the rate of decay of the MR signal. A smaller contribution to the rate of decay is due to protons comprising M_t realigning with B_0, which is known as longitudinal relaxation (T_1) [63].

Longitudinal Relaxation (T_1)

The time constant T_1 defines the rate of growth of the longitudinal magnetization vector M_z , which approaches its thermal equilibrium value (M_0) exponentially according to the equation:

Equation 1-3. Longitudinal Relaxation (T_1).

$$M_z(t) = M_0\left(1 - e^{-t/T_1}\right)$$

After a time interval of 5 T_1, longitudinal relaxation is essentially complete, as M_z has reached 99% of the thermal equilibrium value. Longitudinal relaxation occurs when there is an energy exchange between two protons or between a proton and an electron. This energy exchange only occurs when there is a fluctuation in the magnetic field, which results from motion of the molecules containing the protons and electrons. Energy exchange only occurs when the fluctuations are at or near the Larmor frequency. Thus the value of T_1 is determined by the proton's local magnetic, chemical, and physical environment. The value of T_1, and to a lesser extent T_2 are dependent on the strength of B_0. It is the differences in T_1 and T_2 that exist between tissue types that provide image contrast in MRI.

1.7.5 MRI Magnetic Fields

Three types of external magnetic fields are used in MRI: the static main magnetic field (B_0), which creates the longitudinal magnetization; the radiofrequency magnetic field (B_1), which creates the transverse magnetization;

and the gradient magnetic fields (Gx, Gy, Gz), which provide spatial encoding, as well as allow manipulation of the magnetization vectors.

Main Magnetic Field (B_0)

The purpose of the main magnetic field is to create the excess of precessing protons aligned with the field. In a typical clinical scanner a superconducting electromagnet is used to create a homogeneous magnetic field. A field strength of 1.5 Tesla is commonly used, but higher field scanners are available. The primary advantage of higher field is an increase in number of protons aligned with the field resulting in an increased signal to noise ratio [63].

Radiofrequency Magnetic Field (B_1)

To permit signal detection, the longitudinal magnetization vector M_z must be directed away from its alignment with B_0 (by convention the Z-axis) onto the transverse plane (XY-plane). This is done by the application of another magnetic field (B_1) oriented perpendicular to B_0. The B_1 field is several orders of magnitude smaller than B_0, but exerts an appreciable effect because of the resonance condition as it is oscillating at the Larmor frequency. When M_z is tipped away from alignment with B_0, a component vector projects along the transverse plane referred to as the transverse magnetization (M_t). After the B_1 field is removed, M_t will begin to realign with B_0 as it precesses around B_0. As the direction of M_t is time-varying (at the Larmor frequency) it is capable of inducing a current in a receiver coil tuned to the Larmor frequency.

The B_1 field is generated by a radiofrequency (RF) pulse whose amplitude and duration (τ) establishes the resulting angle between M_t and B_0. This angle is known as the tip angle (α), and is calculated using Equation 1.4.

Equation 1-4. RF tip angle.

$$\alpha = 2\pi\gamma\tau B_1$$

Where α is the tip angle, γ is the gyromagnetic ratio in radians, τ is the duration of the RF pulse in seconds, and B_1 is the magnetic field strength of the radiofrequency pulse in Tesla. A tip angle of $\pi/2$ radians or 90 degrees is often used in MRI as it results in a maximum transverse magnetization and maximum MR signal amplitude. However, the tradeoff is that the proton "reservoir" M_z is depleted, and will not be completely replenished until 5 x T_1, which limits the rate of repeated acquisitions.

Magnetic Field Gradients and Spatial Localization

The MR signal contains contributions from all of the protons within the volume of interest, and therefore cannot provide localization of the various tissue types present within the volume. Spatial localization is achieved by frequency and phase encoding of the protons in 3 dimensions by the application of magnetic field gradients, which in some applications are combined with a B_1 field. Reconstruction of the image from the encoded MR signal is possible with conversion from the frequency/phase domain to the spatial domain using the Fourier transform.

In conventional 2-D MRI spatial encoding in the 3rd dimension is accomplished by applying a magnetic gradient along one axis (for example, the Z-axis). This "slice-select" gradient creates a spectrum of precession frequencies oriented along the Z-axis. Selective excitation of this slice along the Z-axis is accomplished with the simultaneous application of an RF pulse whose bandwidth defines the extent or thickness of the slice. Only the protons within the excited slice contribute to M_t, meaning that only these protons will eventually contribute to the MR signal. Spatial encoding in the remaining 2 dimensions (in this case X and Y) is accomplished using gradients in a similar manner, but without the application of an RF pulse.

Spatial encoding in the X-direction or read direction is termed frequency encoding. Application of a magnetic gradient (read gradient) along the X-axis produces a spectrum of proton precession frequencies that are dependent on their spatial position along the X-axis. If the MR signal is acquired or read at this point in time, it is possible to localize the X position of the protons by transforming the MR signal from the frequency domain to the spatial domain using the Fourier transform. However, each value of X in the transformed data will contain signal contributions from all of the protons along the Y-axis.

Spatial encoding in the Y direction or phase direction is termed phase encoding. Phase encoding is accomplished with a magnetic gradient (phase gradient) that is applied along the Y axis prior to the application of the read gradient. When the phase gradient is applied the proton's precession frequency immediately increases or decreases depending on the direction of the gradient. The proton's precession phase will accumulate positive or negative phase over the duration of the gradient. After removal of the phase gradient, the proton's precession frequency returns to its original value, but the change in the proton's precession phase is retained. The degree of phase change is dependent on the strength and duration of the phase gradient. Multiple acquisitions of the MR signal at different levels of phase gradient amplitudes produces a frequency spectrum along the Y-axis that can be transformed to the spatial domain with the Fourier transform.

1.7.6 MRI Pulse Sequences

An MRI pulse sequence specifies the amplitude, duration, and timing of the RF pulses and magnetic field gradients used to manipulate the transverse magnetization, as well as the timing and repetition rate of the data acquisition. By modifying these parameters the image contrast between different tissue types can be enhanced based on their T_1 and T_2 values, as well as the proton density (PD). Contrast enhancement is of primary importance in MR imaging as diseased

or damaged tissue is often identified based on the changes in T1, T2, or PD that occur.

Contrast between tissue types is due to inherent differences in their T_1, T_2 and proton density values. For example, at 1.5 Tesla adipose tissue has a T_1 of 200-750 msec., a T_2 of 53-94 msec., and a PD of 50 – 100, while muscle tissue has a T_1 of 950-1820 msec., a T_2 of 20-67 msec., and a PD of 45-90. There is little difference in proton density between the two tissues, but significant differences in their T_1 and T_2 values.

Pulse sequences are described as being weighted towards a particular contrast mechanism. For example, if the repetition time (TR) of signal acquisition is less than the T_1 value of the tissue of interest, the signal amplitude is dependent on the tissue's T_1. The signal amplitude is also affected by the tissue's T_2, but this effect can be minimized by using a short echo time, which denotes the time period between the creation of the transverse magnetization and acquisition of the echo signal. The echo time (TE) determines the degree of signal loss that occurs due to T_2 (or T_2^*) decay, and pulse sequences with a short TR (relative to T_1) and a short TE (relative to T_2) are known as T_1-weighted. Tissue with a short T_1 will appear bright with a T_1-weighted sequence, while tissue with a long T_1 will appear dark.

A T_2-weighted sequence uses a TR that is longer (~3x) than the T_1 of the tissue of interest to minimize any T_1-weighting. Employing a long echo time relative to the T_2 of the tissue of interest creates T_2 (or T_2^*) weighting. Pulse sequences with a long TR (relative to tissue T_1) and long TE (relative to tissue T_2) are known as T_2-weighted. Tissue with a short T_2 will appear dark with a T_2-weighted sequence, while tissue with a long T_2 will appear bright.

Image contrast based solely on the proton density of tissues can be obtained by minimizing both T_1 and T_2 dependencies. A pulse sequence with a long TR

(relative to T_1) and a short TE (relative to T_2) is known as a proton density-weighted sequence. The MR image intensities for some common tissues are shown in Table 1-1 .

Table 1-1. Typical MR image intensities for various tissues.
A comparison of typical MR image intensities for various tissues using T_1 and T2 weighted sequences [64].

Tissue Type	T_1-Weighted	T2-Weighted
Cortical Bone	Low	Low
Fat (incl. fatty marrow)	High	Intermediate - High
Red marrow	Low	Low-intermediate
Muscle	Low	Intermediate
Tendon & ligament	Low	Low
Low protein fluid	Low	High
High Protein fluid	High	High

Most MRI pulse sequences acquire an echo of the FID rather than the FID signal. The FID echo is produced by either a gradient reversal (gradient-recalled echo) or a 180 degree RF pulse (spin-echo). The gradient recalled echo (GRE) sequence uses a dephasing lobe on the read gradient, which causes increased dephasing of the spins composing the transverse magnetization. Rephasing of the spins is then initiated by reversing the polarity of the read gradient resulting in a gradient-recalled echo. The gradient echo signal is diminished in amplitude relative to the original FID signal due to T2* relaxation.

The spin-echo sequence employs an additional RF pulse to create an echo of the FID signal. Application of the RF pulse causes the established transverse magnetization projecting along the positive X axis in the X-Y plane to rotate through the Z-Y plane to project along the negative X axis in the -X-Y plane. In other words the transverse magnetization is rotated or flipped 180 degrees. The

effect of this flipping is that any static inhomogeneities in the magnetic field that caused spin dephasing will now cause spin rephasing resulting in formation of spin-echo. As a result, the spin-echo signal is diminished relative to the FID signal due to T2 rather than T2* relaxation. This results in a higher signal to noise ratio than GRE, but at a cost of longer acquisition times as the acquisition of the signal must be delayed until the spin-echo forms. The time required for the echo to form (TE) must be twice the time interval between the 90 degree pulse and the 180 degree pulse for total rephasing to occur. This time interval (TE/2) must be at least as long as the duration of the phase encoding gradient interposed between the 90 and 180 degree pulses. Although the time until signal acquisition in a GRE sequence is also delayed by the duration of the phase encoding gradient it is not necessary to wait an additional TE/2 for the echo to form.

Another common MRI sequence is the short-tau inversion recovery sequence (STIR), which provides signal nulling of specific tissues based on their T1 value. STIR is usually implemented as a spin-echo sequence, and uses an initial 180 degree pulse to invert the longitudinal magnetization 180 degrees. After inversion, the longitudinal magnetization is allowed to regrow toward its equilibrium value over a time period known as the inversion time. When the inversion time (TI) is equal to .69 times the T1 of a tissue, the tissue will have zero longitudinal magnetization. Application of a 90 degree RF pulse at this time point will create transverse magnetization without any contribution from the nulled tissue. To effectively null tissue, the sequence TR must be greater than the T_1 of the tissue of interest. The STIR sequence is often used to null the signal contribution from fat, and tissue with a T_1 longer than that of fat (e.g., muscle) will appear bright in the image. The STIR sequence is also very sensitive in detecting the presence of fluid, which usually has a longer T_1 than fat.

1.7.7 MRI Artifacts and Artifact Suppression

MRI artifacts represent a lack of correspondence between the anatomy and the image and can lead to both false positive and false negative readings.

Chemical Shift Artifact – Fat Shift

A proton's resonant frequency is a function of the proton's local magnetic environment. Protons in fat are chemically shielded by their electron clouds and resonate at a slightly lower frequency (~3.5 ppm) than protons in water. As a result, the spatial position of fat will be misregistered in the frequency encode direction, as well as between slices (which are also frequency encoded). In the image, the fat will be shifted relative to water to produce light and dark regions that may mimic pathology. Fat suppression techniques include the STIR sequence, as well as saturating the fat with a spectrally selective RF pulse prior to start of the imaging sequence.

Blood Flow Artifacts

Blood flow can affect the magnitude of the MR signal to produce variations in image intensity within blood vessels that may mimic vascular pathology. There are at least three mechanisms that produce magnitude variations; in-flow of blood containing unsaturated (i.e. equilibrium longitudinal magnetization) protons, out-flow of blood containing protons with a transverse magnetization component, and intra-voxel phase dispersion due to blood flow along a magnetic field gradient. Although motion of protons along a magnetic gradient will normally produce a phase shift, a voxel of flowing blood that contains a range of velocities will experience a range of phase shifts leading to phase dispersion within the voxel [65].

Blood flow can also affect the net phase of the MR signal to produce spatial misregistration known as blood flow ghosting. This occurs when the range of flow velocities within a voxel is narrow (bulk flow) meaning that phase shift dominates phase dispersion. As phase is what determines a voxel's position on the phase encoding axis, changes in phase due to flow along a gradient will produce spatial

misregistration in the phase encoding direction. The location of the ghosting along the phase encoding axis is determined by the net phase shift, which is a function of the flow velocity. When blood flow is pulsatile, flow velocity varies with time. The combination of bulk flow within a voxel and variable flow velocity can produce numerous ghosts in the phase encoding direction. These pulsatile flow artifacts adversely affect image quality and may mask pathology, or produce the appearance of pathology. The effect on image quality can sometimes be alleviated by swapping the phase and frequency encoding directions to redirect the ghosting from the area of suspected pathology [65].

Two techniques are commonly used to minimize blood flow artifacts, and can be applied alone or in combination. Elimination or reduction of the signal from flowing blood is achieved by applying RF saturation pulses adjacent to the imaging volume. The effectiveness of this technique is dependent on the blood flow velocity – slow flow (e.g. < 1 cm/s) may not fill the imaging volume with saturated protons.

Phase shift can be minimized by using a technique known as gradient moment nulling. A conventional gradient has two lobes of equal area but opposite polarity. The zeroth moment of the bipolar gradient is equal to the gradient area, which in this case is equal to zero. This results in no net phase accumulation for protons that are stationary (zeroth moment). When a proton is moving with a constant velocity (first moment) the phase accumulation is proportional to the integral of its position as a function of time and the intensity of the magnetic field gradient. Phase accumulation will occur unless the first moment is nulled, which is achieved by adding a third lobe to the gradient. Gradient moment nulling can be applied to all three gradients [66].

Magic Angle Effect

The magic angle effect refers to increased MR signal intensity from highly ordered tissue such as tendon when the angle between the tendon and the main

magnetic field approaches 54.7 degrees. Normal tendon exhibits hypointense signal due to the strong dipole-dipole interaction between protons in the water molecules bound to the collagen resulting in a short T_2 (< 10 msec.). This dipole-dipole interaction is dependent on the distance between the dipoles and the angle the dipole-dipole displacement vector makes with the main magnetic field B_0 . When this angle approaches 54.7 degrees the dipole-dipole interaction falls to zero, but this effect is not normally observed as the interactions are averaged due to random motion of the protons. In tendon, motion of the protons is constrained by the collagen structure such that orientation of the tendon at 54.7 degrees to B_0 reduces the dipole-dipole interaction. The net effect is longer T_2 relaxation, which with short T_2-w sequences can produce hyperintense regions within the tendon that might be confused with a tendinopathy [67,68].

1.8 Mechanisms of MRI Contrast for the Structures of the Wrist

Magnetic resonance imaging is an ideal imaging modality for assessing the bones and soft tissues of the wrist. The detection of abnormalities is based on changes in morphology and/or changes in image intensity. In some cases, the specificity of MRI is not 100% and further characterization of abnormalities may require the use of other imaging modalities or biopsy. A standard clinical wrist series will usually include T1-weighted (T1-w), T2-weighted (T2-w), short tau inversion recovery-weighted (STIR-w), and proton density-weighted (PD-w) images acquired in one or more planes.

1.8.1 Bone

MRI can detect defects in cortical bone, abnormalities in the bone marrow, and misalignment between articulating bones. The protons within cortical bone are relatively immobile, which results in an ultra-short T_2 (< 1 msec.), thus cortical bone appears black in both T_1-w and T_2-w images. There are, however, large

numbers of mobile protons in the blood and fat within bone marrow, as well as in the surrounding articular cartilage and synovial fluid, which can provide good contrast with the interposing cortical bone with T_1-w imaging. Defects in cortical bone due to fracture or erosion can be readily detected due to the presence of fluid (plasma, blood, or synovial fluid), which appears bright with T_2-w fat-saturated or STIR sequences.

At birth, bone marrow contains both red hematopoietic marrow and yellow fatty marrow, but in the extremities the red marrow is replaced by yellow marrow by age 25. The T_1 of yellow marrow is short as it contains approximately 15% water, 80% fat, and 5% protein. In a T_1-w image, yellow marrow will be of high intensity, and moderate to high with T_2-w imaging. With T_2-w FS or STIR imaging the yellow marrow will have a dark signal intensity [69].

Changes in the image intensity of bone marrow may be indicative of a number of pathologies. Increased fluid content due to neoplasm, hemorrhage, edema, or cystic lesions will result in a low intensity signal with a T_1-w sequence and a high intensity signal with a T_2-w FS or STIR sequence. Decreased fluid content due to fibrosis, necrosis, or an enostosis (bone island) will result in a decrease in signal intensity with both T_1-w and T_2-w FS sequences.

MR imaging in 3 planes permits the detection of misalignment between articulating bones. As normal bone alignment is dependent on normal carpal ligament integrity, misalignment usually indicates stretching or tearing of one or more ligaments.

1.8.2 Cartilage

MRI can reliably detect defects in the articular cartilage of the knee, which is typically 2-3 mm thick. However, the articular cartilage of the carpal bones is

typically < 1mm thick and MRI at clinical field strengths has not been shown to reliably detect cartilage abnormalities [70,71] .

1.8.3 Ligament

Although ligaments are approximately 2/3 water, the water protons are tightly bound to the collagen fibers limiting their mobility and shortening the T_2. As a result, normal ligaments will appear black or dark gray in both T_1-w and T_2-w images, from which abnormalities such as attenuation or discontinuity can be detected. The presence of high signal within ligament may indicate complete or partial tears or mucoid degeneration, and can be best detected with a T_2-w FS or STIR sequence. Regions of high signal may also represent artifacts due to the magic angle effect.

1.8.4 Tendon

The mobility of water protons in tendon is restricted due to the presence of collagen, which shortens T_2, and tendon will appear dark gray or black in both T_1-w and T_2-w images. Indications of a tendinopathy include thickening, focal thinning, disruption, or increased signal intensity. Localized regions of high signal may also represent artifacts due to the magic angle effect. Increased signal intensity due to fluid accumulation within the tendon may be due to a complete or partial tear, degeneration (tendinosis), or inflammation (tendinitis). Increased signal intensity surrounding a normal appearing tendon indicates tenosynovitis [72] .

1.8.5 Joint Effusion & Synovitis

Excess synovial fluid within the joint capsule/cavity will exhibit a well-defined area of low signal intensity with T_1-w imaging and high signal intensity with T_2-w FS, or STIR imaging. Thickening of the synovial membrane is known as synovitis, and may appear similar to an effusion, but with less well-defined margins [73].

1.8.6 Extraosseous and Intraosseous Ganglia

A soft tissue mass may indicate a neoplasm, but the most common soft-tissue mass in the wrist is the extraosseous ganglion. An extraosseous ganglion is a collagen-walled sac containing "synovial-like" fluid, which has a well-defined margin and homogeneous low signal intensity with T_1-w imaging and a high signal intensity with T_2-w FS, or STIR imaging. An intraosseous ganglion defines an osteolytic cavity within the bone marrow that is histologically identical to an extraosseous ganglion, and has a well-defined margin and homogeneous low signal intensity with T_1-w imaging and a high signal intensity with T_2-w FS, or STIR imaging. In the MRI studies reported in this thesis extraosseous and intraosseous ganglia were frequently identified. The clinical significance, etiology, and pathogenesis of extraosseous and intraosseous ganglia are controversial, and a comprehensive review of ganglia is provided in Chapter 2.

1.9 Thesis Objectives and Hypotheses

1. Compare maximum active wrist flexion between symptomatic computer users and asymptomatic individuals with minimal computer exposure. It is hypothesized that wrist flexion will be significantly limited in the symptomatic computer users.

2. Compare maximum active wrist flexion between symptomatic computer users and asymptomatic computer users. It is hypothesized that wrist flexion will be significantly limited in the symptomatic group.

3. Compare the contributions of radiolunate and capitolunate joint motion to total wrist flexion between symptomatic computer users and asymptomatic computer users. It is hypothesized that capitolunate joint motion will be significantly increased in the symptomatic group.

4. Compare the prevalence of MRI-identified abnormalities (intraosseous and extraosseous ganglia, tendonosis, tendonitis, synovitis, effusion) and wrist range of motion (flexion and radioulnar deviation) between symptomatic computer users and asymptomatic computer users. It is hypothesized that the prevalence of abnormalities will be significantly greater, and the wrist range of motion significantly less in the symptomatic group.

1.10 Thesis Outline and Organization

Chapter 1 provides background on upper extremity musculoskeletal disorders, and a description of the basic anatomy and biomechanics of the wrist joint. The underlying principles of magnetic resonance imaging are described, as well as an explanation of some common MRI artifacts and their suppression. This is

followed by mechanisms of MRI contrast of wrist structures, and descriptions of some common wrist pathologies.

Chapter 2 contains a manuscript entitled "A review of extraosseous and intraosseous ganglia", which is being prepared for submission. This manuscript reviews the history of extraosseous and intraosseous ganglia, and the controversies regarding their etiology, pathophysiology, symptomology, and treatment options.

Chapter 3 contains a manuscript entitled "The effect of forearm posture on wrist flexion in computer workers with chronic upper extremity musculoskeletal disorders". This manuscript has been published online (Burgess RA, et al., 2008) and compares wrist range of motion between symptomatic computers users and individuals with minimal computer exposure.

Chapter 4 contains a manuscript entitled "A comparison of wrist range of motion and flexion kinematics in symptomatic versus aymptomatic computer users", which is undergoing revision for resubmission to Biomed Central Musculoskeletal Disorders. This manuscript compares wrist ROM and carpal flexion kinematics between symptomatic versus asymptomatic computer users, and describes a number of wrist pathologies identified in the symptomatic users.

Chapter 5 contains a manuscript entitled "MRI-identified abnormalities and wrist range of motion in asymptomatic versus symptomatic computer users", which is undergoing revision for publication in Biomed Central Musculoskeletal Disorders. This manuscript compares wrist ROM between symptomatic versus asymptomatic computer users, and compares the prevalence and physical size of wrist pathologies identified in both groups.

Chapter 6 summarizes the studies comprising this thesis, discusses their limitations, and suggests possible avenues for further research.

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Chapter 2

A Review of Extraosseous and Intraosseous Ganglia

2.1 Introduction

The term "ganglion" was first used by Hippocrates (as cited in McEvedy [1]) to describe a "knot of tissue containing mucoid flesh". Currently, the term extraosseous ganglion is used to describe an encapsulated fluid that is usually found adjacent to joints, also known as soft-tissue ganglia. The term ganglion was also used by Fisk [2] to describe a lesion that was histologically identical to an extraosseous ganglion, but located within the bone marrow. The term intraosseous ganglia was first employed by Crabbe [3] to describe ganglia within bone marrow. The etiology, pathophysiology, and clinical significance of both types of ganglia continue to be debated, while their prevalence appears to be increasing, perhaps a consequence of expanded use of diagnostic imaging.

2.2 Extraosseous Ganglia

Extraosseous ganglia (EOG) are encapsulated "synovial-like" fluid usually found in soft tissues adjacent to joints and tendon sheaths [4]. Histologically, EOG possess a relatively acellular collagen wall that is not lined with synovium, a feature differentiating EOG from synovial cysts. EOG may also be intraligamentous [5,6], intraneural [7], or intratendinous [8,9]. Similar lesions located adjacent to the distal interphalangeal joint are known as digital mucous cysts, but are histologically identical to EOG [10]. Histologically identical lesions

are also found within the adventitia of arteries and rarely, veins, and are known as adventitial cysts [11].

EOG may arise directly from a joint capsule, or tendon sheath, or be attached by a pedicle or stalk. The majority of EOG (50 -70%) occur in the hand and wrist and 60 – 70 % of these are located on the dorsum of the wrist [12]. The positive predictive value of MRI in detecting occult wrist ganglia, that were confirmed histologically, was 75 % [13]. EOG may arise and recede spontaneously, enlarge with activity, and are often asymptomatic [12,14]. EOG are identified most often in middle-age, but do occur in children [15,16]. They are associated with antecedent trauma in a minority of cases, and although repetitive minor trauma has been implicated, according to Angelides [12] an association with occupation is not obvious. Treatment of EOG by aspiration or surgical excision has not been shown to provide any significant long-term benefit compared to no treatment for both dorsal [17] and volar [18] wrist ganglia.

There is no consensus with regard to the etiology and pathophysiology of EOG. Theories include synovial herniation (Eller, 1746; cited in Psaila [19]), mucoid degeneration of connective tissue [20], or mucin production within connective tissue [1]. Based on the current evidence it seems likely that all three events may co-occur, and the existing controversy appears to be one of precedence. The primary questions that have been well-examined concern the nature of the fluid, presence of connective tissue degeneration, and evidence of communication with the joint cavity. The question of etiology remains undetermined.

2.2.1 Chemical Analyses of Synovial and Extraosseous Ganglion Fluid

Synovial fluid is found within synovial joints [21] and tendon sheaths [22], and is thought to provide nutrition to articular cartilage [23] and act as a lubricant [24]. Synovial fluid is a dialysate of blood plasma containing blood proteins (e.g. albumin, globulin) and a number of mucopolysacharrides, primarily hyaluronan (hyaluronate, hyaluronic acid) [25]. The concentration of hyaluronan in synovial

fluid is approximately 1000 times greater than that of blood plasma [26], while the concentration of blood proteins in synovial fluid is approximately 1/5 that of blood serum [27]. Hyaluronan is secreted by synoviocytes whose production increases in response to mechanical stimulation [28]. Hyaluronan is also present in soft connective tissue where it is secreted by fibroblasts [29], whose production increases in response to mechanical stimulation [30].

Chemical analyses of EOG fluid have identified hyaluronan and serum proteins in varying concentrations [20,31,32]. The concentration of hyaluronan in EOG is often greater than synovial fluid, which accounts for its increased viscosity [20]. Digital mucous cysts also contain hyaluronan and uncharacterized protein [33,34]. Hyaluronan is also found in adventitial cysts [35-38] and intraneural ganglia [39]. Chemical analysis has not been reported for intraligamentous or intratendinous ganglion fluid, and the diagnosis relies on the macroscopic appearance of the fluid contents [5,8,9,40,41].

2.2.2 Pathogenesis of Extraosseous Ganglia

The presence of hyaluronan in EOG has been attributed to mucoid degeneration of connective tissue [42,43]; stress-induced metaplasia of connective tissue cells within sub-capsular cysts [1]; trauma or irritation of mesenchymal cells, fibroblasts, or modified synoviocytes at the synovial-capsular interface [12]; secretion by fibroblasts or mesenchymal stem cells embedded within the ganglion wall [10,19,43]; extrusion of joint/tendon sheath synovial fluid [44]. Other theories propose that intraligamentous EOG are due to irritation of ligament synovium [45], while advential cysts/ganglia result from the inclusion of synovial cell rests during development [46] or from pulsatile motion of the arterial wall [47].

The high concentration of hyaluronan in EOG may be due to a pressure gradient within sub-capsular cysts restricting the inflow of plasma [1], or ultrafiltration of

joint synovial fluid thru the cyst wall [44,48]. Both mechanisms are capable of explaining the relatively low viscosity of fluid in new or recurrent ganglia.

2.2.3 Evidence of Connective Tissue Degeneration

The theory of connective tissue degeneration in the pathophysiology of EOG was popularized by Carp and Stout [42]. The authors thought that communication with the joint cavity was a rare and secondary event, resulting from complete degeneration of the joint capsule. They found that the normally broad and thick collagen fibrils in the cyst wall and surrounding tissue were broken up into fine threads. The wall of the cyst appeared to be of thickened collagen, which was attributed to a proliferative process. Soren [20] postulated that ganglion formation involved several stages: thickening of some fibers, thinning, bifurcation and fragmentation of other fibers; liquifaction of the fibers and formation of cavities containing mucoid substances; development of an apparent cyst wall from compression of collagen fibrils due to the expanding fluid; creation of a true cyst wall by fibrocytes and fibroblasts.

It is clear that connective tissue degeneration is intimately associated with EOG, and would be a necessary prerequisite for extrusion of synovial fluid from the joint or tendon sheath. Opinions differ on the prevalence of communication in EOG.

2.2.4 Evidence of Communicating Extraosseous Ganglia

Eller's original theory (cited in Psaila [19]) of synovial herniation was initially abandoned due to the absence of synovial tissue within EOG, and a lack of demonstrable communication with the joint cavity [1]. McEvedy [1] found no evidence of cyst to joint communication in EOG when the cysts were injected with contrast medium, although the main cyst always communicated with small sub-capsular cysts. Andren & Eiken [44] demonstrated one-way joint to cyst communication in 37 of 59 wrist ganglia after injecting contrast medium into the

joint cavity, although it is not known whether they injected the radiocarpal joint , midcarpal joint, or both. Angelides et al. [49] reported on their surgical treatment of 500 dorsal wrist ganglia; all of which connected to the scapholunate ligament via a stalk or pedicle. While only 3 of 8 ganglia examined arthrographically exhibited joint-cyst communication, microscopic examination of 64 wrists after ganglion excision afforded a view of the scapholunate joint through sub-millimeter openings in the sectioned portion of the scapholunate ligament [49].

Although a lack of demonstrable arthrographic communication may signify a sub-capsular origin for some EOG, the results may depend on technique and imaging modality. In an arthrographic study [50] of EOG of the knee, joint to cyst communication was evident in only 2/20 cysts immediately after contrast injection, but increased to 6/11 cysts after 1 – 2 hours, both assessed with plain film radiography. CT imaging after a 1-2 hour delay showed communication in 15/15 cysts [50], indicating that delayed CT imaging is more sensitive than plain film radiography. Communication has also been demonstrated between EOG and tendon sheaths [51,52], and a study using dye injection into the distal interphalangeal joint showed communication in 48/54 digital mucous cysts [53].

Recent magnetic resonance imaging (MRI) studies have shown that both intraneural [7,54-56] and adventitial [55,57,58] ganglia connect with the joint, and arthrographic studies have confirmed communication with the joint cavity in intraneural [50,59], adventitial, and intraligamentous [50] ganglia.

These studies indicate that all forms of EOG may exhibit communication with a synovial fluid source (i.e. the joint cavity, or tendon sheath). Whether such communication develops from the joint to the ganglion or vice versa [46] has not been proven. If communication proceeds from the ganglion to the joint cavity, it would be reasonable to expect evidence of communication or connection between venous and arterial adventitial ganglia, or between adventitial ganglia and intraneural ganglia, which has not been demonstrated.

2.2.5 Etiology of Extraosseous Ganglia

It is evident that many types of EOG communicate with the joint capsule, which is often degenerate and in some cases the degeneration extends to the joint cavity. What initiates this degeneration remains unclear. Connective tissue stress is often proposed as the initiating factor [12], despite the low incidence of prior trauma [60]. To paraphrase Carp and Stout [42], most cases of EOG are atraumatic, and most cases of trauma do not result in EOG.

Extraosseous ganglia of the carpus have been associated with ligament abnormalities in symptomatic patients with either no history [61], or an unspecified history of acute trauma [62]. Conversely, carpal ganglia have also been associated with acute trauma, but with no evidence of ligament abnormalities [63]. A role for chronic repetitive trauma is often suggested in the etiology of EOG [64], but it is not obvious why only a subset of individuals exposed to the same workload develop ganglia. Soren [65] suggests that the degenerative process might be related to tissue strain in those whose occupational demands (typists, musicians, athletes, and labourers) combine with a constitutional inferiority in their connective tissue to make them selectively susceptible to the development of EOG.

There are some anecdotal reports of EOG in gymnasts [66], and tennis players [67], but there are very few studies relating EOG to occupation. Studies of instrumental musicians have found the prevalence of EOG ranges from 2.3 % to 7 % [68-70]. A case report of multiple digital mucous cysts in a patient whose work involved repetitive pressure on the finger tips was thought to be related to occupation [71]. In a study [72] of flexor tendon sheath ganglia, 11/40 patients were manual typists whose ganglia were always located over the middle of the proximal phalanx of the middle finger. The authors speculated that elevated stress of the tendon sheath occurred with finger flexion during typing [72], but it was not discussed why only the middle finger was affected. A more recent study of flexor tendon sheath ganglia [73] found only 3/57 patients were computer

keyboard users, and the authors conclude that no clear relationship to occupation exists. It is also conceivable that flexor tendon stress is reduced with computer keyboard use compared to manual typewriter use. The few reports of occupational associations are limited to palpable EOG, and little is known with regards to occult ganglia.

The predilection for EOG to arise at specific sites has been interpreted as evidence for locally elevated tissue stress. The most common origins for wrist ganglia are the scapholunate ligament and the scaphotrapezialtrapezoidal (STT) ligaments. The etiology of periscaphoid EOG has been attributed to an underlying ligamentous injury [74], or chronic mechanical stress of the periscaphoid ligaments [12,64,75].

2.2.6 Summary

An extraosseous ganglion is an encapsulated mass of synovial-like fluid that may communicate with the joint cavity or tendon sheath via degenerate connective tissue. The ganglion fluid may be derived from synovial fluid, or may be produced by cells within the connective tissue. The etiology may be related to mechanical stress of connective tissue. Symptoms are thought due to a mass effect, but treatment has not shown significant benefit compared to no treatment in relieving symptoms.

2.3 Intraosseous Ganglia

Intraosseous ganglia (IOG) are so-called because they are histologically identical to EOG, but are located within the bone marrow [2,3]. The term intraosseous ganglion cyst is also used interchangeably, but they are not true cysts as they lack an epithelial or synovial lining. IOG are found in many joints including the ankle, knee, hip, elbow, and wrist, and are identified most often in middle-age [76,77]. Although previously thought to be relatively rare and a likely source of pain, both symptomatic and asymptomatic IOG are increasingly being identified.

The symptoms associated with IOG are described as localized pain that is aggravated by activity [76,78,79]. In a study of 88 patients with IOG in various joints, 60 % were associated with symptoms [76]. IOG of the carpal bones have been associated with symptoms in 15 % [78] and 62 % of cases [80,81]. Symptoms have been attributed to increased intraosseous pressure due to development of the ganglion wall [78], although a ganglion wall can be present in asymptomatic ganglia [81]. Waiznegger [82] suggests that an intraosseous ganglion expanding through a breach in the cortex may stretch the capsular and ligamentous tissue eliciting pain. Uriburu [81] noted that cortical defects were present in 4 asymptomatic and 7 symptomatic cases. Increased radiotracer uptake, presumed due to increased osteocyte activity, is associated with symptomatic IOG, but is not a consistent finding [81,83,84]. Treatment of IOG by curettage and bone grafting has been shown to be totally [81,83,85,86], or highly [78,82,87] effective in relieving symptoms.

Histologically, IOG are osteolytic cavities within sub-chondral bone that are usually surrounded by a wall of regenerated bone, and less frequently necrotic bone [78,88,89]. The osteolytic lesion has been described as uni or multilocular, and may or may not possess a fibrous collagen lining or membrane [76,78,88]. The ganglion lining has been described as poorly vascularized [88], and incompletely lined by flattened connective tissue cells [88,89] also described as fibroblasts [76,78]. Cells resembling mesenchymal cells have been identified within the ganglion wall [90]. The adjacent bone marrow has been described as fibrotic, but not necrotic [89], or showing some evidence of bone resorption but new bone formation predominates [76]. Schajowicz [76] noted that a fibrous membrane surrounded only “mature” cysts, and was absent in smaller newly formed cysts. According to Eiken [78], IOG without a fibrous lining contain a “thin, slightly viscid fluid”, while those with a lining contain a “thick gelatinous material”.

The fluid contents of IOG are macroscopically similar to EOG, but there is some confusion in the literature regarding the chemical constituents of IOG fluid. Several authors claim that the contents of IOG are chemically identical to that of EOG, but Feldman [88] miscites a study of EOG by Soren [20], while a number of authors [87,91,92] miscite a study of EOG by Young [93]. Histochemical analysis of an intraosseous ganglion identified hyaluronic acid on the inner surface and within the ganglion wall, as well as within the cytoplasm of immature mesenchymal cells [90]. The authors propose that hyaluronic acid in intraosseous ganglia results from an active cellular process due to an unknown stimulus, rather than degeneration of connective tissue [90].

Radiographically, IOG are well-marginated with an ovoid, round, or hourglass shape, and are usually surrounded by a thin sclerotic border. With T1-weighted magnetic resonance imaging (MRI), IOG are usually isointense with respect to muscle, but may also be hypo or hyper-intense [77]. With T2-weighted or inversion recovery-weighted MRI, IOG are always hyperintense with respect to muscle [77].

The imaging diagnosis of IOG is based primarily on the absence of associated osteoarthritic changes, as IOG are histologically and radiographically identical to sub-chondral cysts associated with osteoarthritis known as osteoarthritic (OA) cysts [77]. OA cysts are thought to develop either as a result of synovial fluid penetration via fissures in the cartilaginous cortical surface [94,95], or as a degenerative process within intramedullary bone [96]; possibly resulting from transmission of high contact stresses at the articulating surface due to cartilage loss [97,98]. Williams [77] argues that differentiating between IOG and OA cysts is a matter of semantics, as they may have different etiologies but that the end result is the same. IOG, unlike OA cysts, are predominantly located in regions of bone not normally exposed to compressive load (i.e. non-articulating). In the majority of cases there is no history of antecedent trauma or evidence of

cartilage defects [78,81,88,89]. These facts appear to rule out compressive bone stress, or synovial fluid penetration via cartilage defects in the etiology of IOG.

2.3.1 Pathogenesis

The pathogenesis of IOG is uncertain, but possible mechanisms that have been proposed include: a primary intraosseous process (idiopathic), or secondary to an extraosseous process (penetrating). The idiopathic theories propose that IOG result from intramedullary metaplasia and proliferation of fibroblasts followed by an accumulation of hyaluronic acid producing pressure atrophy of the trabeculae [88], activation of immature mesenchymal cells [90], or aseptic bone necrosis due to a local vascular disturbance [76,78,99,100]. Penetration theories include herniation of synovial membrane into bone [100]; intrusion of an extraosseous ganglion [101] or ganglia-like tissue [89]; intrusion of synovial fluid via an articular crack (Nigrisoli, 1971, as reported in Kambolis [89]); intrusion of degenerate ligament fluid followed by intramedullary metaplasia and myxoid degeneration and secretion [102].

There is currently no consensus on what pathomechanism(s) may be involved, or the underlying etiology. The theory of metaplasia has been criticized as it begs the question of what initiates this deviation from normal in adulthood [103]. The theory of penetration by an extraosseous ganglion lacks support when EOG are not present, as was the case in 54 % of cases reviewed by Nishimura [104]. The theory of vascular disturbance has been criticized because IOG are located in well vascularized bone that is usually not necrotic [105]. The cause of the vascular disturbance has been ascribed to mechanical factors and repetitive trauma [76,78,99], despite the predominant location of IOG in non-articulating bone. Theories of fluid penetration, of either a synovial or degenerate ligament origin have their basis in the frequent identification of communication with the joint cavity, or the proximity of IOG to ligament attachment sites.

2.3.2 Evidence of IOG Communication

Evidence of IOG communication with the joint cavity or EOG has been identified intraoperatively [76,81,87,106], histologically [107], cystographically [106], and radiographically [78,82,108]. Evidence of communication between IOG and the joint cavity is usually absent with plain radiographs or MRI, but is more apparent with computed tomography. A communicating defect or channel was identified in 7.8% [78], 33 % [81] and 57 % [108] of IOG with computed tomography. Opinions differ on whether communication develops from the inside-out [78,99] or the outside-in [108].

Histological evidence of IOG in communication with the joint cavity was demonstrated by Crane et al. [107] who found a defect in the dorsal radial aspect of an excised lunate that emitted a gelatinous fluid. A gelatinous material was also found on the ulnar aspect of the scaphoid and its ligament attachment. The authors suggested either extrusion of fluid from the bone, or proliferation of synovial membrane into the bone as possible causes [107]. In two case reports [109,110], bilateral IOG in the radial aspect of the lunate were considered to be idiopathic due to the absence of communication with the joint cavity at operation [110], or in the excised biopsy specimen [109]. In a case report [111] of bilateral IOG of the scaphoid and lunate, the ganglia were attributed to penetration of the scapholunate ligament into the adjacent bones. A case report [112] of bilateral IOG of the distal scaphoid attributed the ganglia to impaired vascular circulation due to compressive bone stress. The author speculates that the level of vascular impairment may have been sufficient to produce mucoid degeneration, but not bone necrosis [112].

These various interpretations of similar pathology were influenced by the presence or absence of associated ligamentous degeneration. The predilection of IOG to develop in proximity to capsular or ligamentous attachment sites has been noted by several authors [3,100,102]. This proximity has been interpreted as the site for herniation of synovial membrane [100], intrusion of synovial fluid

[3], or intrusion of degenerate ligament fluid [102]. In a cadaveric study, Schrank et al. [102] demonstrated communication between IOG and ligament attachment sites histologically, and noted that the ligaments adjacent to IOG were in various stages of degeneration. The authors suggested that intrusion of degenerate ligament fluid was followed by intramedullary metaplasia and myxoid degeneration and secretion [102].

In a canine study [113], it was found that cyst-like lesions adjacent to the caudal cruciate ligament attachment on the tibia developed after sectioning of the cranial cruciate ligament. Histologically, the lesions contained a loose fibro-reactive marrow, increased hemato/myelopoietic elements, vascular congestion, and trabecular bone remodeling. The authors propose that the lesions were due to bone resorption, and that synovial fluid intrusion, if present, was a secondary event. The authors suggest that bone resorption was due to altered biomechanics in an unstable joint, producing either abnormal stress on the intact caudal cruciate ligament or abnormal contact between the femoral condyle and tibia [113]. Several authors have proposed that ligament stress may be a factor in the development of intraosseous cyst-like lesions at the cruciate ligament attachment sites in the human knee [114-116].

2.3.3 Etiology of Extraosseous and Intraosseous Ganglia

Although EOG and IOG frequently co-exist [80,104], some authors question the ability of EOG to penetrate dense cortical bone and suggest that communicating EOG result from expansion of IOG through the cortex [77,78]. In the absence of demonstrable communication, with either an extraosseous ganglion or the joint cavity, another mechanism is needed to explain ganglion fluid in bone that, by definition, possesses normal articular cartilage. The fact that IOG are usually located in regions of bone not normally exposed to compressive load (i.e. non-articulating and juxta-entheseal), suggests a distractive rather than compressive stress mechanism. The frequent association between IOG and EOG suggests

that both types of ganglia are different manifestations of ligament stress [64,80,117]. Although acute trauma is noted in some cases, its effect may be indirect. For example, it is likely that stretch or rupture of one ligament (e.g. anterior cruciate ligament of the knee) alters the dynamics of the joint resulting in chronic overloading of another ligament (e.g. posterior cruciate ligament). In the majority of cases ganglia are not associated with previous trauma, and it is presumed that the dynamics of the joint is normal.

The frequency of carpal ganglia in proximity to the scapholunate and dorsal intercarpal ligaments has been attributed to the concentration of forces in this region [80], particularly shear stress of the scapholunate ligament during heavy use [64]. Schrank [102] believes that the predilection for IOG to develop in the capitate and lunate may be due to increased tension load in the central carpus during complex wrist movements. Neither theory explains the apparent difference in susceptibility between individuals performing the same tasks. This may be due to a constitutional inferiority as proposed by Soren [65], but there is paucity of imaging studies on the prevalence of both forms of ganglia relating to specific vocational or avocational activities.

Although it is assumed that joint dynamics are normal in atraumatic cases, there is considerable variability in the kinematics of the normal wrist [118-120]. The cause of this variability is unknown, but differences in bone morphology or ligamentous laxity have been proposed [118]. The importance of tendon loading has been emphasized with regards to approximating the in vivo condition in ex vivo kinematics studies [118,121]. However, the implicit assumption that in vivo tendon loading is consistent among individuals has been questioned [122]. An increase in tendon loading would alter the dynamics of the joint, but whether differences in tendon loading, or differences in bone morphology or ligamentous laxity translate into a susceptibility to develop ganglia is unknown.

2.4 Summary

Extraosseous ganglia are encapsulated masses of synovial-like fluid that frequently communicate and likely originate from the joint or tendon sheath. Intraosseous ganglia are osteolytic lesions, usually encapsulated and containing a synovial-like fluid, that arise adjacent to ligament attachment sites.

Extraosseous and intraosseous ganglia are identical or very similar in most respects. Both arise in otherwise normal joints, frequently co-exist in the same joint and often at the same site. Both types of ganglia may or may not be symptomatic, and treatment appears more effective in relieving symptoms associated with IOG than those associated with EOG. Although there are many different theories regarding their pathogenesis, there is some general consensus that their etiology may be related to mechanical stress of capsular or ligamentous tissue. A history of acute trauma is infrequent, suggesting a role for chronic ligament stress in their etiology.

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Chapter 3

The Effect of Forearm Posture on Wrist Flexion in Computer Workers with Chronic Upper Extremity Musculoskeletal Disorders

3.1 Introduction

Occupational hand use has been associated with chronic upper limb pain and dysfunction, but the issue of causality remains controversial [1]. However, recent reviews of the epidemiological literature have concluded that there is a moderate, but consistent association between computer use and hand/arm symptoms [2,3]. Some symptoms may be consistent with specific clinical diagnoses (e.g., carpal tunnel syndrome (CTS), lateral epicondylitis), but frequently the symptoms are non-specific (e.g., myalgia) [4]. A prospective study of computer users found that 50% of hand/arm symptoms were non-specific [5]. This collection of specific and non-specific symptoms is known by a variety of labels including repetitive strain injury (RSI), cumulative trauma disorder (CTD), or work-related upper extremity disorder (WRUED). These labels are controversial, as they either imply causation or signify the presence of a disorder for which objective signs of pathology are often lacking. While we appreciate that no single term adequately encompasses the various specific and non-specific upper limb symptoms encountered in clinical practice [6], in this paper we use upper extremity musculoskeletal disorder (UEMSD) only to categorize the symptomatic subjects enrolled in our study. Current hypotheses ascribe the symptoms of UEMSD to muscle/tendon/soft-tissue damage, neurogenic disorders, or psychogenic causes [7,8], presumed to arise from biomechanical or psychosocial stressors [1].

The proliferation of computers in the workplace has coincided with an increase in the number of people reporting UEMSD attributed to computer use. Estimates of the prevalence of computer-related UEMSD vary depending on the diagnostic criteria used [9-11], but the fraction of computer workers who develop severe or disabling symptoms does not appear to be large. This implies differences in individual susceptibility or the influence of other etiological agents. There can be little doubt that computer use exacerbates upper limb symptoms, but whether these symptoms arise as a consequence of an unidentifiable injury sustained in the workplace or elsewhere is unknown. Hadler [12] has proposed that the wrist is susceptible only to "violence from without" and violence from "performance at the extremes of tissue tolerance". However, there exists a third possibility: that the wrist is susceptible to violence (or stress) from *within*. Individuals with UEMSD who engage in repetitive work (computer users, factory workers and instrumental musicians) often have limited wrist range of motion (ROM), which has been attributed to increased antagonist muscle tension [13-17]. Increased antagonist muscle tension in the upper limb may be a source of biomechanical stress during both occupational and non-occupational activities.

In the previous studies, increased antagonist muscle tension was inferred from limitations in wrist ROM [13-17]. Pain is known to have a limiting effect on joint mobility, but pain elicited during testing was mentioned in only one study [14]. There seems to be general agreement by the various authors that wrist ROM was limited by increased antagonist muscle tension [13,14,16,17] or ligament/muscle/tendon shortening [15] although there is no consensus on whether this represents an increase in active or passive tension. There is some evidence for increased agonist muscle activity in upper extremity disorders [18,19] as well as a positive relationship between antagonist muscle activity and trait anxiety [20], but we are not aware of any studies demonstrating increased antagonist muscle activity in UEMSD patients. It has also been suggested that the increase in antagonist muscle passive tension resulted from replacement of Type II muscle fibers with shorter Type I fibers in response to chronic muscle

activity [13]. However, the results of studies relating changes in muscle fiber-type distribution to work-related myalgia have been contradictory [21-23].

Despite the uncertainty regarding the underlying mechanism, an increase in antagonist muscle tension would clearly affect wrist joint dynamics. Wrist flexion in affected computer users is often impaired compared to normative values or to the unaffected wrist [13,16,17]. Increased wrist extensor muscle tension would demand increased flexor muscle activity during wrist flexion, thereby increasing muscle and wrist joint loading. This increased loading may produce sufficient stress to elicit symptoms in the joint and muscle, particularly at the extremes of wrist flexion. Although computer use involves primarily wrist extension rather than flexion, other domestic or recreational activities that require repetitive or forceful wrist flexion may strain the joint or muscles, the symptoms of which are exacerbated by and then attributed to occupational computer use.

Wrist ROM is normally measured with the forearm prone and the elbow flexed 90 degrees [24], which is the posture normally assumed during computer use. Several of the previous studies did not specify the forearm postures used during flexion testing, particularly the study noting an increase in pain reported during testing [14]. Recreational or domestic activities involving a variety of forearm postures with the elbow fully extended may affect joint loading and symptoms. Therefore, it would be useful to quantify wrist flexion at several forearm postures with the elbow extended. Techniques for measuring joint ROM may be passive—using examiner applied force, or active—using subject generated force. The previous studies of wrist ROM [13-17] used either one or both techniques, and passive flexion was always greater than active flexion. We used active measures of wrist ROM to minimize any potential discomfort to the subjects. The objective of this study was to determine whether changes in forearm posture would affect pain reports during maximum active wrist flexion, or whether pain would have a limiting effect on flexion angle.

3.2 Methods

3.2.1 Subjects

5 subjects (4 female, 1 male, mean age = 43, SD = 5.1 yrs.) who reported chronic (> 1 year duration) UEMSDs that they attributed to occupational computer use were recruited from a local RSI support group and the community. Two subjects reported bilateral symptoms, and three reported unilateral dominant-side symptoms. All subjects reported forearm muscle pain, and all but one reported wrist pain and/or pain in the hand or fingers. These symptoms are consistent with UEMSD, and the subjects had received a variety of diagnoses (RSI, tendonosis, CTS) by either their family physician or a specialist. No UEMSD subjects reported any previous trauma, and all were currently working except for one female subject who was off work due to symptoms. The control group consisted of thirteen subjects (7 female, 6 male, mean age = 46, SD = 17 yrs.) with no history of wrist or forearm symptoms. Only two of the control subjects used computers extensively in their occupations. All subjects were right-hand dominant except for one male control subject. Our Institutional Ethics Review Board approved the study, and informed consent was obtained from each subject.

3.2.2 Materials and Procedure

A custom wrist support (Figure 3-1) was used to maintain the elbow in full extension, the wrist in neutral radioulnar deviation, and the forearm prone, neutral, or supine. The support was designed to contact only the wrist and elbow to prevent compression of the forearm musculature. Neutral radioulnar deviation was maintained with an adjustable guide bar during testing with the forearm supine, by contact with the hand support with the forearm neutral, and visually with the forearm prone.

A B C

Figure 3-1 Image of custom wrist support.

Images of the wrist support used to measure flexion with the forearm (A) prone, (B) neutral, and (C) supine.

The wrist support's height was adjusted to align the forearm parallel to the floor at shoulder height with the subject seated. A 6" universal goniometer (Medelco model DIA 503) was used to determine maximum active wrist flexion as the angle formed between the midline of the long axis of the radius and the dorsal aspect of the 3rd metacarpal. This deviation from the normal testing procedure [24], where the dorsal aspect of the radius anchors one arm of the goniometer, was necessary as the wrist support interfered with this positioning when the forearm was supine. This technique has been previously shown to be reliable for measures of wrist extension [25]. The subjects were instructed to relax their fingers and flex only their wrists as far as possible while maintaining neutral radioulnar deviation, and to report any symptoms that arose or were exacerbated during the maneuver. The sequence was randomized with respect to the initial wrist measured and forearm posture. Maximum active wrist flexion was measured once at each of the three forearm postures. The wrist flexion data were analyzed with a 2 x 2 x 3 (Group x Side x Posture) mixed analysis of variance using SPSS version 16, (SPPS Inc., Chicago, IL) with side and posture as the repeated measures. Any significant effects (two-tailed) were followed by a

series of *post hoc* means comparisons adjusted using Tukey's HSD for within group comparisons, and Tukey-Kramer for between group comparisons.

3.3 Results

The mean wrist flexion angles for the non-dominant and dominant wrists at each forearm posture for both groups are shown in Figure 3-2. Dominant wrist flexion in the UEMSD group was significantly restricted compared to the control group at all forearm postures and, in the non-dominant wrist, at the neutral and supine forearm postures. In the dominant wrist, UEMSD group mean flexion angle versus control group mean flexion angle (± 1 standard deviation) was 45.8 (20.6°) versus 66.0 (7.7°), $p < .05$ with the forearm prone; 36.6 (17.2°) versus 61.1 (9.2°), $p < .01$ with the forearm neutral; and 22.2 (8.5°) ve rsus 48.3 (8.7°), $p < .01$ with the forearm supine. In the non-dominant wrist, the UEMSD group's mean flexion angle versus the control group's mean flexion angle was 59.2 (4.1°) versus 64.4 (6.7°), *ns* with the forearm prone; 44.8 (17.9°) versus 64.1 (7.7°), $p < .01$ with the forearm neutral; and 30.0 (12.7°) vers us 50.5 (11.4°), $p < .01$ with the forearm supine.

The differences in mean flexion between the three forearm postures within each group are shown in Table 3-1. Within the control group, mean flexion decreased significantly ($p < .01$) from prone to supine, and from neutral to supine for both wrists. Within the UEMSD group, mean flexion decreased significantly (dominant, $p < .05$; non-dominant, $p < .01$) from the prone to supine forearm posture in both wrists. There were no significant differences in flexion at equivalent forearm postures between the dominant versus non-dominant wrist in either group. None of the UEMSD subjects reported an increase in pain symptoms during the testing procedure.

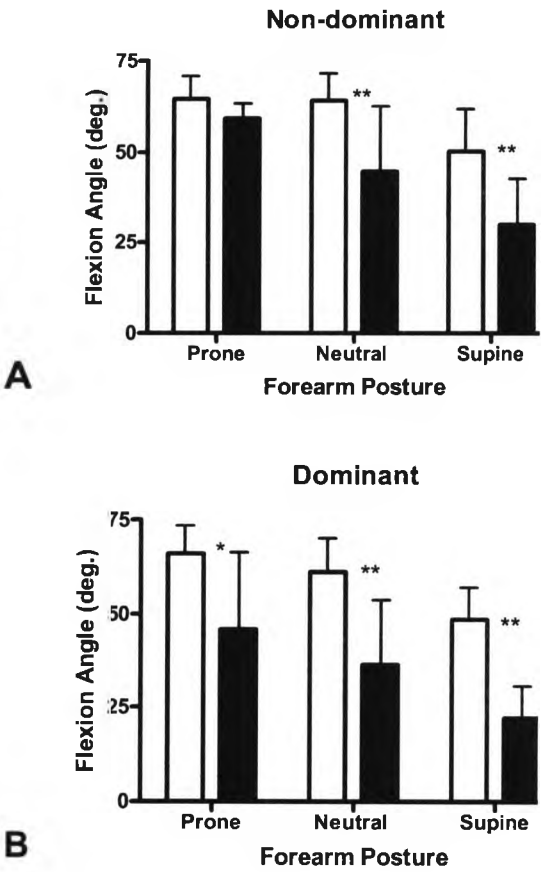

Figure 3-2. Wrist flexion versus forearm posture.

Mean wrist flexion angle versus forearm posture for the non-dominant (A) and dominant (B) wrists of the UEMSD group (n=5) and control group (n=13). *$p < .05$, **$p < .01$. Y-bars = 1 *SD*.

Table 3-1. Differences in Mean Wrist Flexion between Forearm Postures.

Group	Side	Posture Comparison	Mean Difference	SD (°)	*q*	*df*	Tukey's HSD
Control	Non-Dominant	Prone-Neutral	0.3	8.4	0.13	3,72	ns
		Prone-Supine	13.9	10.0	5.77	3,72	$p < .01$
		Neutral-Supine	13.6	13.2	5.64	3,72	$p < .01$
	Dominant	Prone-Neutral	4.9	10.3	2.04	3,72	ns
		Prone-Supine	17.7	11.4	7.33	3,72	$p < .01$
		Neutral-Supine	12.8	8.1	5.29	3,72	$p < .01$
UEMSD	Non-dominant	Prone-Neutral	14.4	14.5	2.19	3,24	ns
		Prone-Supine	29.2	11.0	4.45	3,24	$p < .01$
		Neutral-Supine	14.8	11.8	2.25	3,24	ns
	Dominant	Prone-Neutral	9.2	8.1	1.40	3,24	ns
		Prone-Supine	23.6	16.6	3.60	3,24	$p < .05$
		Neutral-Supine	14.4	11.3	2.19	3,24	ns

3.4 Discussion

We compared maximum active wrist flexion in UEMSD subjects versus control subjects at three forearm postures. None of the UEMSD subjects reported any increase in pain symptoms during testing, indicating that their wrist flexion was not limited by pain. Mean wrist flexion in the UEMSD group was restricted compared to the control group in both wrists at all forearm postures with the exception of the non-dominant wrist with the forearm prone. These flexion limitations are consistent with previous studies of individuals with UEMSDs [13-17]. However, the most striking result was the decrease in wrist flexion at the supine forearm posture compared to the prone forearm posture in both wrists of both groups.

The control group's mean flexion data for both wrists at the prone and neutral forearm postures was comparable to previous studies of active wrist flexion [24-26]. The control group's flexion decreased at the supine posture compared to the prone or neutral postures in both wrists. Wrist flexion in the UEMSD group also decreased between the prone and supine forearm postures, but to a greater extent than in the control group. Overall, wrist flexion between the prone and supine forearm postures in the UEMSD group decreased 51.5 % in the dominant and 49.3 % in the non-dominant wrist, and in the control group decreased 26.8 % in the dominant and 21.6 % in the non-dominant wrist.

We are aware of only one previous study conducted by Hewitt in 1928, who found that mean active wrist flexion decreased approximately 12° between the prone and supine forearm postures in normal subjects [27]. However, in Hewitt's study active wrist flexion was approximately 10° - 15° greater than that shown in previous studies or our study [25,26]. We suspect that this discrepancy may be due to the comparatively young age (mean age < 20 yrs.) and exclusively female gender of the subjects in Hewitt's study, both of which have been associated with increased wrist flexion [28]. Hewitt [27] did not explicitly address the decrease in flexion between the prone and supine forearm posture observed in her study, but suggested that agonist muscle efficiency may be greater with the forearm prone versus supine.

Another study on the effects of forearm posture found that maximum active wrist flexion decreased slightly (< 5 degrees) when the forearm was semi-prone (45°) versus fully prone (90°) [26]. This decrease was at tributed to possible changes in the articular contact of the carpal bones, or increases in trans-carpal ligament tension [26]. Our results did not show a significant decrease in wrist flexion between the prone and neutral forearm postures in either group. However, the decrease in wrist flexion between the prone and supine forearm postures that is evident in both groups cannot be readily explained by changes in articular contact or trans-carpal ligament tension. The results of a cadaveric study [29]

examining wrist tendon excursion demonstrated that the normal wrist is capable of 70 degrees of flexion at the prone, neutral, and supine forearm postures when the proximal wrist motor tendons are severed from their origins. Presumably both cadaveric and live wrists possess equivalent trans-carpal ligament and articular contact characteristics.

Forearm posture may exert an effect on wrist flexion due to biomechanical changes that occur with forearm supination. Several cadaveric wrist studies have shown that excursion of the distal tendon of the extensor carpi ulnaris (ECU) muscle during wrist flexion increases approximately three-fold between the prone and supine forearm postures [29-31]. These results have been attributed to an increase in the length of the distal ECU tendon's moment arm due to dorsal displacement of the distal ECU tendon relative to the carpus during forearm supination [29-31]. A three-fold increase in the length of the distal ECU tendon's moment arm corresponds to a three-fold increase in distal ECU tendon excursion to achieve a given wrist flexion angle. It follows that decreased extensibility of the ECU muscle would resist ECU tendon excursion, limiting wrist flexion to a greater degree with the forearm supine versus prone. Decreased extensibility of the ECU muscle would explain the greater loss of wrist flexion with the forearm supine that we observed in the UEMSD group compared to the control group. This apparent decrease in ECU muscle extensibility may be due to increases in active and/or passive tension.

The presence of ECU muscle tonus and an intact connection to the proximal tendon's origin in live subjects might explain why a supine forearm posture limited wrist flexion in our control subjects, but not in studies of cadaveric wrists [29-31]. Similarly, an increase in ECU muscle activity in the UEMSD group compared to controls might also explain why the decrease in flexion between the prone and supine forearm postures in the UEMSD group was approximately double that in the control group. We are not aware of any studies demonstrating increased wrist extensor muscle activity during wrist flexion in UEMSD subjects,

although such increased activity may occur. However, it appears that even maximum contraction of the ECU muscle would be incapable of resisting maximum wrist flexion torque.

A study of normal wrists [32] found that maximum wrist extension torque was less than 60% of maximum flexion torque over the range of flexion angles we measured in our study. Other studies have confirmed the superiority of wrist flexion torque, although to lesser degrees, and all were conducted with the forearm prone [33-35]. Presumably the extension torque capability of the ECU muscle would be enhanced when the forearm is supine due to the increase in its moment arm length[29-31].

We are aware of only one study that examined wrist extensor torque with the forearm supine. Ketchum [36] used measures of wrist extensor muscle masses and tendon excursions from cadavers combined with flexion torque from live subjects to estimate the force generated by each muscle. The tendon excursions were measured with the forearm supine, but the ECU muscle was estimated to contribute less than 30% of the total extensor force [36].

It is also possible that the reduction in wrist flexion with the forearm supine was due to a decrease in wrist flexion torque. However, wrist flexor tendon excursion has not been shown to vary significantly between the prone and supine forearm postures [29,30], suggesting that flexion torque would not decrease. As the ECU muscle is only one of three muscles contributing to wrist extension torque it appears unlikely that even maximum activation of the ECU muscle would be capable of opposing wrist flexion torque. Therefore, we infer that the limited wrist flexion exhibited by the UEMSD group compared to the control group with the forearm supine is due to increased ECU muscle passive tension.

Maximum active wrist flexion in the dominant wrist of the UEMSD group was also below normal with the forearm prone, in agreement with previous studies

[13,16,17]. However, we are unable to attribute this flexion restriction to increased ECU muscle passive tension. It follows from the studies of wrist motor tendon excursion previously discussed [29-31] that the required ECU tendon excursion for a given wrist flexion angle is reduced three-fold when the forearm is prone versus supine. Consequently, one would expect the wrist flexion angle to increase three-fold with the forearm prone versus supine if ECU muscle passive tension was the limiting factor. Our results showed that dominant wrist flexion in the UEMSD group increased approximately two-fold between the supine and prone forearm posture, and remained below normal. The fact that wrist flexion increased only two-fold and remained below normal despite a three-fold decrease in the required excursion of the distal ECU tendon indicates that ECU muscle tension is not limiting wrist flexion with the forearm prone.

The limited wrist flexion may have been due to a general impairment in dominant wrist flexor muscle strength in the UEMSD group. A study of subjects with medial and lateral epicondylitis found that peak wrist flexion torque was reduced by 13% in the affected versus the unaffected arm [37]. However, the same study found that peak wrist extension torque was still less than 60% of peak flexion torque in both arms [37]. These results suggest that substantial wrist flexor impairment would be required to limit wrist flexion in conjunction with maximum extensor muscle activation. Instead, we propose that dominant wrist flexion in the UEMSD group with the forearm prone is limited by decreased extensibility of the radial wrist extensor muscles: extensor carpi radialis brevis (ECRB) and extensor carpi radialis longus (ECRL). We infer that this is due to an increase in passive rather than active tension because, as described previously, wrist flexion torque exceeds wrist extension torque over the range of wrist joint angles measured in our study [32].

Our findings of restricted wrist flexion in UEMSD subjects compared to normal controls is consistent with the idea that wrist extensor muscle passive tension is elevated in this group [17]. This interpretation agrees with a previous report of

palpable increases in wrist extensor muscle tone with wrist flexion in UEMSD patients [13]. The modulation of maximum active wrist flexion by forearm posture is readily explained by known changes in the length of the distal ECU tendon's moment arm [29-31]. The existence of flexion restrictions in the dominant wrist of the UEMSD group at both the prone and supine forearm postures implicates both the ulnar (ECU) and radial wrist extensor muscles (ECRL and ECRB).

Increased wrist extensor muscle passive tension would affect wrist joint dynamics during flexion. We had initially presumed that, as wrist flexion during computer use is minimal, increased joint and muscle/tendon stress might have occurred during domestic or recreational activities, the symptoms of which were then exacerbated by, and perhaps attributed to computer use. However, the evidence for increased ECU muscle passive tension suggests that increased stress could also occur during radial deviation of the wrist, as the ECU muscle despite its name, functions primarily as an ulnar deviator [30,31].

The apparent increase in extensor muscle passive tension exhibited by the UEMSD group may be related to computer use. Mackinnon and Novak [7] suggest that prolonged abnormal postures may affect muscle tension due to muscle length adaptation. It has been shown that animal skeletal muscle immobilized in a shortened state shortens due to the loss of serial sarcomeres, and the rate of such loss increases when the muscle is chronically activated [38,39]. However, the increase in muscle passive tension that follows immobilization has been attributed to qualitative and quantitative changes in the connective tissue surrounding the muscle, and these changes have been shown to precede sarcomere loss [40]. Conceivably, prolonged wrist extension during computer use could result in muscle length and/or connective tissue changes that increase extensor muscle passive tension.

The cross-sectional design of the current study limits our ability to infer causality, particularly as flexion limitations were also evident in the unaffected wrist of the

unilaterally affected UEMSD subjects. However, the results of this study suggest a role for elevated extensor muscle passive tension as a factor in the development of UEMSD symptoms in computer users. Computer usage in the control group was minimal, so we cannot determine from this study whether wrist flexion is similarly affected in asymptomatic computer users. The limitations of this study include a small sample size, and a lack of clear case definitions for the UEMSD subjects. The control group was not age and gender matched to the UEMSD group; however, 6 of the 7 female control subjects were within the age range of the female subjects in the UEMSD group.

3.5 Conclusions

The UEMSD group exhibited reduced active wrist flexion compared to the control group that did not appear to be pain related. A supine versus prone forearm posture reduced wrist flexion in both groups, but the reduction was approximately 100% greater in the UEMSD group. We infer that these results are consistent with increased wrist extensor muscle passive tension, particularly in the extensor carpi ulnaris muscle. The effect of a supine forearm posture on wrist flexion, particularly in highlighting flexion restrictions that were not evident with the forearm prone, suggests that this technique may be a useful addition to the standard wrist ROM testing procedure.

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Chapter 4

No Difference in Carpal Flexion Kinematics in Symptomatic vs Asymptomatic Computer Users. Abnormalities Identified in a Subset of Symptomatic Wrists.

4.1 Introduction

Computer use has been associated with an increased prevalence of upper extremity musculoskeletal disorders (UEMSD). Previous studies have shown that computer users with UEMSD have limited wrist range of motion that was attributed to elevated antagonist muscle tension [1,2]. In a Chapter 3 we showed that maximum active wrist flexion was limited in computer users with UEMSD compared to control subjects who had minimal computer exposure. Wrist flexion was modulated by forearm posture in both groups, decreasing between the prone and supine forearm postures, but the decrease was approximately 100% greater in the UEMSD group. We inferred from the known increase in the flexion moment arm of the distal extensor carpi ulnaris (ECU) tendon that occurs with forearm supination [3-5], that ECU muscle tension was elevated in the UEMSD group. Dominant wrist flexion in the UEMSD group remained below normal with the forearm prone, which we attributed to elevated tension in the radial extensors (extensor carpi radialis brevis: ECRB, and extensor carpi radialis longus: ECRL). We further concluded that the mechanism of increased muscle tension was passive rather than active, as wrist flexion torque has previously been shown to exceed extension torque over the range of flexion angles we measured [6,7].

Increased wrist extensor muscle tension would alter joint dynamics during wrist flexion or radial deviation, and may influence joint kinematics. Wrist flexion involves both radiolunate and capitolunate joint motion, and the relative contribution of each has been shown to vary considerably in normal wrists [8,9]. Possible sources for this variability include differences in methodology, ligament laxity, bone morphology, and muscle tension [9,10]. As the radius of the capitolunate joint is smaller than that of the radiolunate joint [11], flexion that occurs primarily at the capitolunate joint would presumably require less extensor tendon excursion than flexion occurring primarily at the radiolunate joint. Therefore, increased extensor tension may produce increased capitolunate joint motion during flexion, assuming the normal ligamentous constraints are overcome. The purpose of this study was to compare wrist flexion range of motion and capitolunate joint motion in computer users with UEMSD versus asymptomatic computer users. We hypothesized that wrist flexion will be decreased and capitolunate joint motion increased in the symptomatic group compared to the asymptomatic group.

4.2 Methods

4.2.1 Subjects

Our institutional ethics review board approved the study, and informed consent was obtained from each subject. Eight female control subjects (mean age = 39.4, SD = 12.3 yrs.) with no history of upper limb trauma or symptoms were recruited from the local community. 7 of the 8 control subjects used computers extensively in their occupations. Six female UEMSD subjects (mean age = 43.7, SD = 5.0 yrs.) who reported a variety of symptoms that they attributed to occupational computer use were recruited from a local RSI support group. UEMSD symptoms were bilateral in four subjects and included wrist joint pain, hand/finger paraesthesia, and forearm muscle pain (myalgia). Four of the six UEMSD subjects had been off work for periods ranging from 2 to 6 years due to their symptoms. Despite the long work absences, the UEMSD subjects reported

continuing symptoms that interfered with normal daily activities. Two UEMSD subjects had experienced prior wrist trauma and had undergone x-ray imaging, the results of which were negative (based on anecdotal evidence). All subjects were right-hand dominant with the exception of one ambidextrous control subject.

4.2.2 Materials and Procedure

We measured maximum active wrist flexion of both wrists using a plastic goniometer with the forearm in the prone, neutral and supine postures as described previously [12] . MR images of both wrists in neutral posture (neutral flexion/extension and radioulnar deviation), and flexed to approximately 50 degrees were obtained using a custom 1.89 Tesla MRI system (Surrey Medical Imaging Systems (SMIS) console); 16 element quadrature birdcage RF coil; Magnex 32cm horizontal bore magnet). A custom wrist support was used to position the subject's forearm prone and the wrist in neutral flexion/extension by aligning the dorsal aspect of the third metacarpal with the dorsum of the forearm. T1-weighted coronal images (TR/TE = 675/17, matrix = 256 x 128, FOV = 70 mm, 6 x 3 mm contiguous slices) were acquired to ensure neutral radioulnar deviation by ensuring alignment of the long axis of the capitate with the long axis of the radius. T1-weighted sagittal images (TR/TE = 675/17, matrix = 256 x 128, FOV = 70 mm, 18 x 3 mm contiguous slices) were then obtained with the wrist neutral, and flexed to approximately 50 degrees. Wrist positioning in neutral radioulnar deviation was maintained visually during wrist flexion.

The angles of the neutral and flexed capitate and lunate with respect to the palmar tilt of the radius were extracted from the sagittal MR images using SMIS analysis software as shown in Figure 4-1. A line parallel to the anterior and posterior distal poles of the lunate determined the angle of the lunate, while the angle of the capitate was determined by connecting a line between the center of the capitate head to the midpoint of its articulation with the third metacarpal. Motion of the lunate and capitate was determined by subtracting the measured

angle in the neutral wrist posture from that of the flexed wrist posture. Capitate motion relative to the radius represents total wrist flexion. Capitolunate joint motion was calculated by subtracting lunate motion from capitate motion.

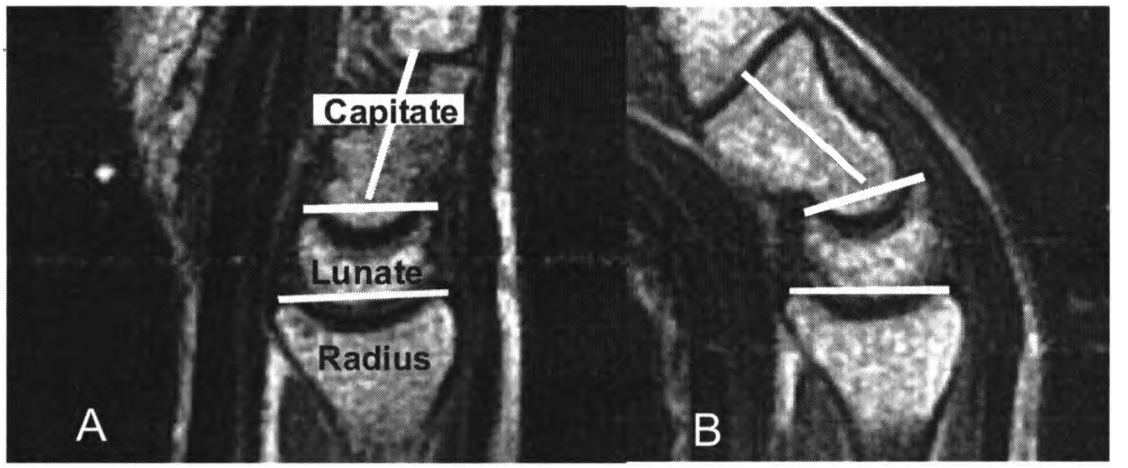

Figure 4-1 Sagittal T1-w images of the wrist.
Sagittal T1-w images of the wrist in (A) neutral and (B) flexed posture.

A staff radiologist reviewed any suspect MR images and two asymptomatic control subjects showed evidence of degenerative changes (abnormal scapholunate gap in one, degenerative cysts in the other). No further intervention was advised for these subjects, and their data was not used in the present study. The remaining control group was composed of 6 female subjects (mean age = 36.3, SD = 11.8 yrs.). MR images of four UEMSD subjects showed suspected abnormalities, and standard clinical scans were acquired of 6 UEMSD wrists with the following parameters: Coronal and axial TI-weighted (TR/TE, 630/22; image matrix, 256_512; number of acquisitions, 2), proton density (2000/22; 256_512; 1), T2-weighted (2000/80; 256_512, 1); Sagittal, coronal and axial short-tau inversion-recovery (STIR-w) 128_512, 2 (2000/23/160) (TR/TE/TI). The MR images were assessed by one experienced musculoskeletal radiologist with 14 years of experience who was blinded to the subject's symptoms. From these clinical scans there was evidence of post-traumatic injury in two wrists for which

a history of trauma was reported. The data for these wrists were excluded from the UEMSD group analysis.

The following criteria were used to interpret the clinical images:
A well-marginated area within subchondral bone that is hyperintense using a STIR-w sequence, and hypointense using a T1-w sequence usually indicates a fluid-filled lesion, and will be termed an intraosseous ganglion. A well-marginated area in soft tissue that is hyperintense using a STIR-w sequence, and hypointense using a T1-w sequence usually indicates a fluid-filled lesion, and will be termed an extraosseous ganglion. A poorly marginated area within subchondral bone that is hyperintense using a STIR-w sequence, and hypointense using a T1-w sequence will be termed an edema-like lesion.

Statistical analysis was performed using SPSS version 16 (SPPS Inc., Chicago, IL). The wrist flexion data were analyzed with a 2 x 2 x 3 (Group x Side x Posture) mixed analysis of variance with side and posture as the repeated measures. Wrist flexion means were compared with t-tests using an alpha level of .05. The carpal bone motion data was analyzed with a 2 x 2 (Group x Side) repeated measures analysis of variance, and means were compared using t-tests with a significance level of .05 adjusted with Tukey's HSD.

4.3 Results

4.3.1 Wrist Flexion ROM

Mean group wrist flexion for the right and left wrists at each forearm posture are shown in Figure 4-2. Left wrist flexion in the UEMSD group was significantly less than the control group with the forearm prone (Mean (M) = 57.2°, SD = 9.4°) versus (M = 72.8°, SD = 8.5°), $p < .05$); and neutral (M = 40.4°, SD = 15.1°) versus (M = 70.0°, SD = 5.9°), $p < .01$. Right wrist flexion in the UEMSD group was significantly less than the control group with the forearm prone (M = 55.3°, SD = 14.4°) versus (M = 70.8°, SD = 4.0°), $p < .05$, neutral (M = 39.8°, SD = 16.6°) versus (M = 66.8°, SD = 9.4°), $p < .01$, and supine (M = 39.3°, SD = 11.5°) versus (M = 54.3°, SD = 16.4°), $p < .05$. Mean wrist flexion decreased between the prone and supine forearm postures in both wrists of both groups but this was significant only in the right wrist of the control group (Mdif. = 12.5°, SD = 11.4°, $p < .05$). There were no significant differences in flexion at equivalent forearm postures between the right versus left wrist in either group. None of the subjects reported pain as a limiting factor during flexion testing.

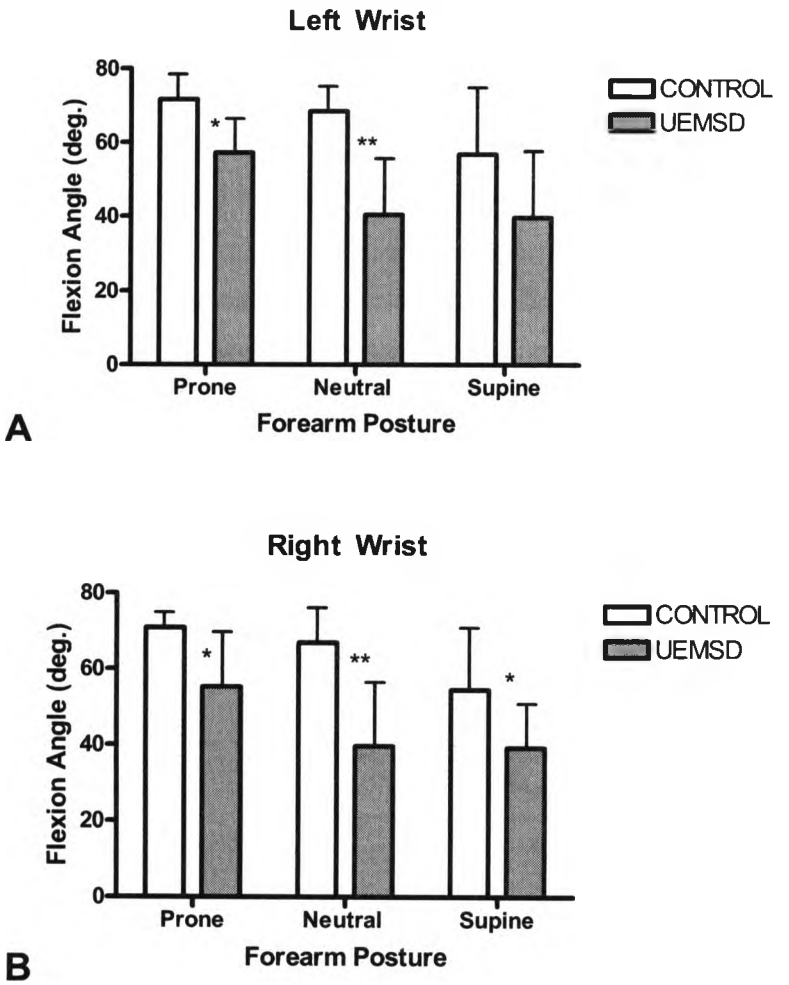

Figure 4-2. Wrist flexion versus forearm posture.

Mean wrist flexion angle versus forearm posture for the left (A) and right (B) wrists of the UEMSD group (left n = 5, right n = 4), and control group (n = 6). *$p < .05$, **$p < .01$ Y-bars = 1 SD.

4.3.2 Radiolunate and capitolunate joint kinematics

The scatterplot in Figure 4-3 shows the radiolunate and capitolunate motion for the individual wrists. There was no difference in the mean capitolunate joint motion as a percentage of total wrist flexion in the control group (M = 66.6°, SD = 11.9%) compared to the UEMSD group (M = 54.1°, SD = 11.8 %), *ns*.

The linear regression of radiolunate motion on capitolunate motion is significant for the control group (r^2 = 0.58, $p < .01$), but not for the UEMSD group (r^2 = 0.0004, ns).

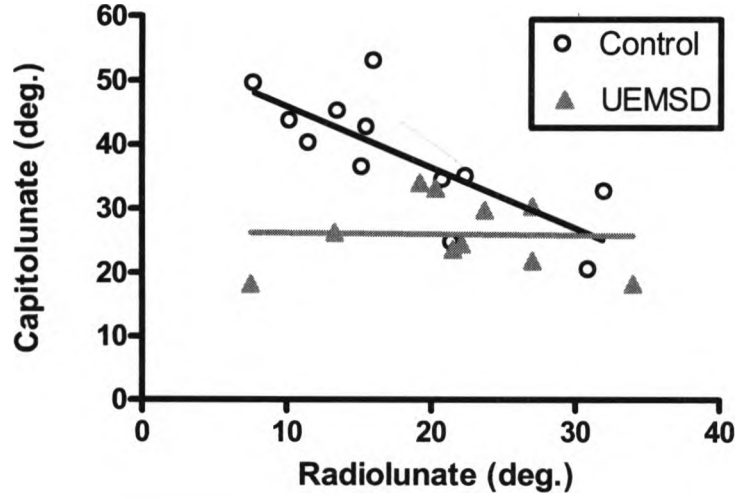

Figure 4-3. Plot of radiolunate and capitolunate joint motion.

Scatterplot of the radiolunate and capitolunate joint motion for the individual wrists in the control group (n=12) and UEMSD group (n=10).

4.3.3 Clinical Imaging

Case 1: 47 year-old female secretary with a 3-year history of bilateral dorsoradial wrist pain and forearm muscle pain/lateral epicondylitis with no history of previous trauma. A coronal T1-w and sagittal STIR-w image of the right wrist (Figure 4-4) show an intraosseous ganglion within the ulnar volar aspect of the distal pole of the scaphoid adjacent to the attachment of the scaphocapitate ligament.

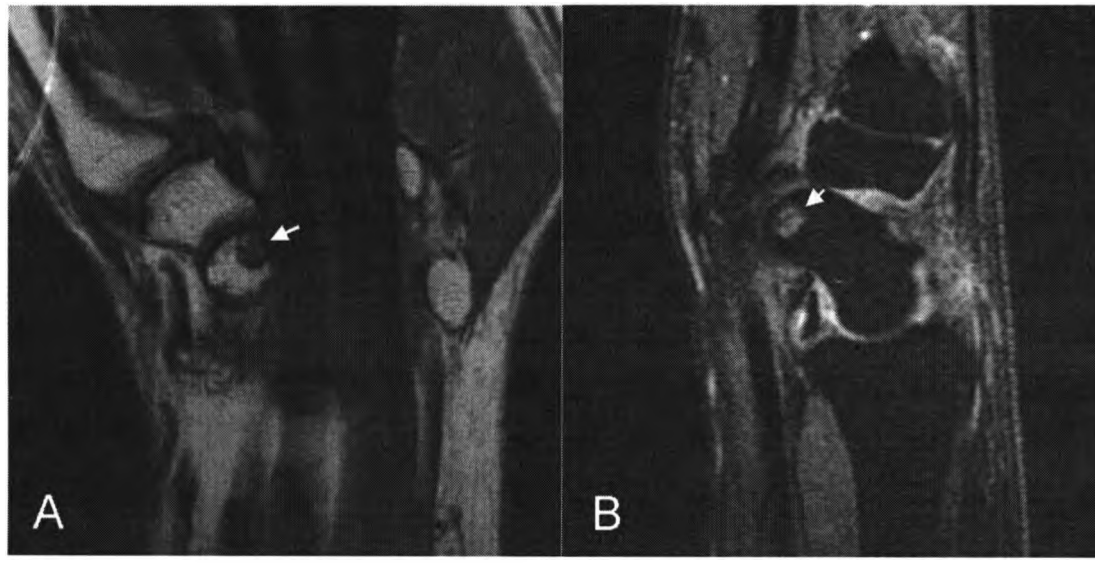

Figure 4-4. Case 1 right wrist MR images.

(A) Coronal T1-w and (B) sagittal STIR-w image of the right wrist showing an IOG (arrows) in the ulnar aspect of the distal volar pole of the scaphoid adjacent to the attachment of the scaphocapitate ligament.

Coronal STIR-w and axial T2-w MR images of the left wrist (Figure 4-5) show a multilobulated extraosseous ganglion dorsal to the articulation of the capitate, lunate and scaphoid, and synovitis surrounding the lateral aspect of the scaphoid.

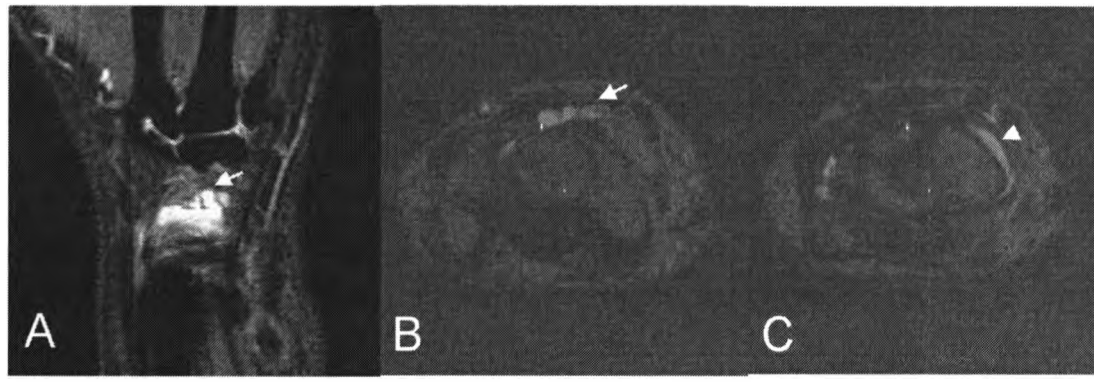

Figure 4-5. Case 1 left wrist MR images.

(A) Coronal STIR-w image of the left wrist demonstrating a multilobulated extraosseous ganglion (arrow) dorsal to the intersection of the scaphoid, capitate, and lunate; (B) axial T2-w images showing the ganglion (arrow) dorsal to the scapholunate ligament; (C) synovitis (arrowhead) surrounding the lateral aspect of the scaphoid.

Case 2: 50 year-old female clerical worker with a 10-year history of chronic right wrist pain in the ulnar aspect that developed approximately 1 year after a fall on the right hand, as well as tingling & pain in the ulnar aspect of the left wrist/forearm that began approximately 1 year prior to the right wrist trauma. The subject had been off work due to symptoms for six years prior. An MR image of the right wrist (Figure 4-6) showing a grade II/III dorsal subluxation of the distal ulnar head suggestive of distal radioulnar joint (DRUJ) instability.

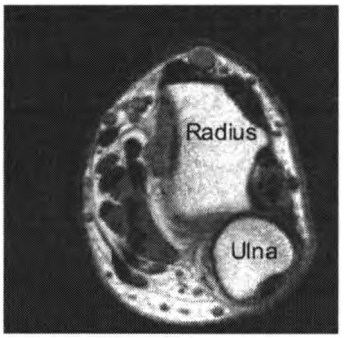

Figure 4-6. Case 2 right wrist MR image.
T1-w axial image of the right wrist showing grade II-III dorsal subluxation of the distal ulnar head.

T1-w MR images (Figure 4-7 a-c) of the left wrist showing a sclerotic lesion within the ulnar aspect of the lunate at its articulation with the distal ulna, and a mushroom-shaped lesion within the triquetrum at the insertion of the distal ECU tendon sheath. STIR-w MR images of the left wrist (Figure 4-7 d-f) show a dorsal extraosseous ganglion at the radioscaphoid joint; several extraosseous ganglia proximal to the pisotriquetral joint; intraosseous ganglia within the distal capitate adjacent to the attachment of the capitohamate ligament and within the proximal dorsal and volar aspects of the lunate adjacent to the lunotriquetral ligament attachment. There is also fluid intrusion within the ulnar aspect of the triquetrum adjacent to the attachment of distal ECU tendon sheath.

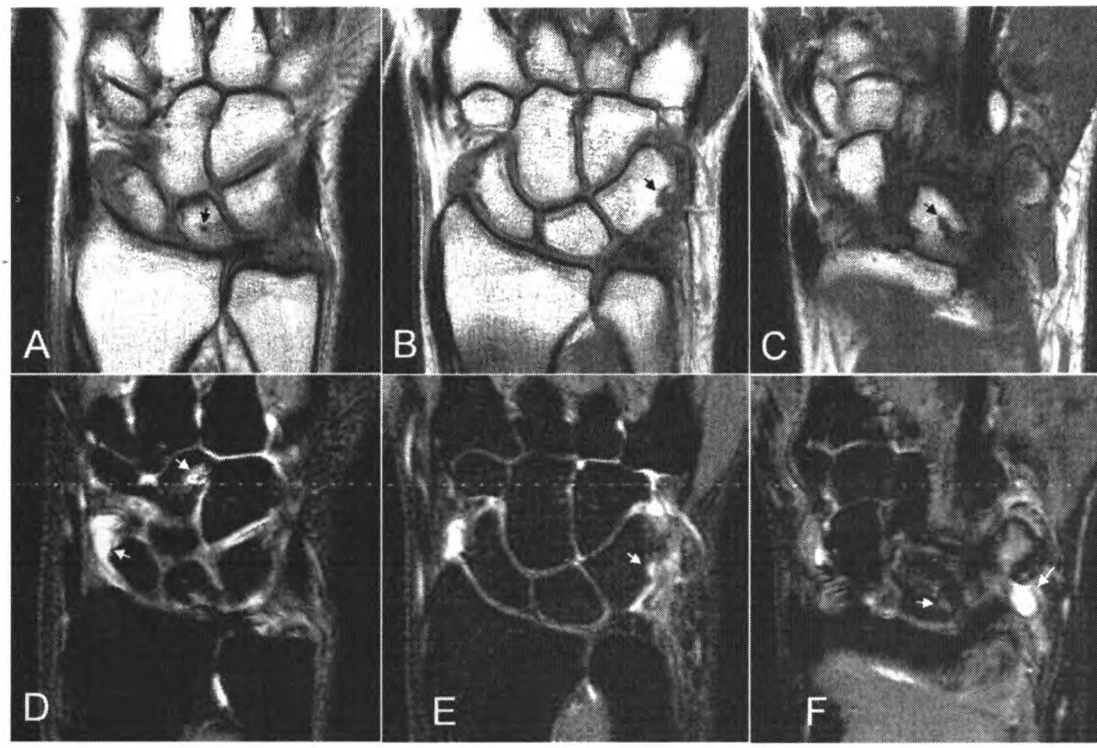

Figure 4-7. Case 2 left wrist MR images.

Coronal T1-w images of the left wrist showing cystic lesions (arrows) within the (A) dorsal and (B) volar ulnar aspect of the lunate; (B) a mushroom shaped lesion within the triquetrum at the insertion of the distal ECU tendon sheath. Coronal STIR images showing (D) extraosseous ganglia distal to the radioscaphoid joint and (F) proximal to the pisotriquetral joint. Intraosseous ganglia within the (D) distal capitate adjacent to the insertion of the capitohamate ligament; (E) within the triquetrum adjacent to the attachment of distal ECU tendon sheath; within the proximal (D) dorsal and (F) volar aspects of the lunate adjacent to the lunotriquetral ligament insertion.

Case 3: 40 year-old female clerical worker with a 3-year history of dorsoradial right wrist pain with no history of previous trauma. The subject had been off work due to symptoms for two years prior to the study. MR images of the right wrist were acquired using the following sequences: T1-w (coronal, axial); STIR-w (coronal, sagittal, and axial); Pd/T2-w (coronal, axial). An MR image of the right wrist (Figure 4-8) shows a small intramedullary sclerotic focus within the waist of the scaphoid measuring 5 mm in maximal dimension that was hypointense with STIR-w imaging.

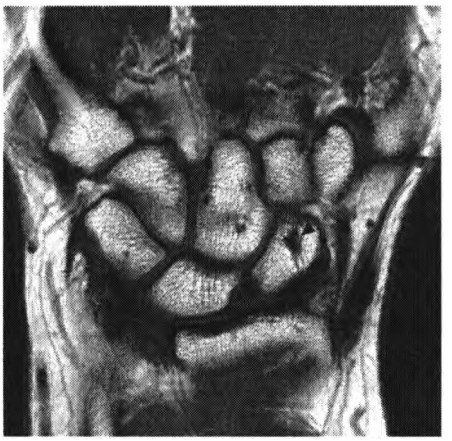

Figure 4-8. Case 3 right wrist MR image.

Coronal T1-w image of the right wrist in Case 3 showing a sclerotic lesion (arrowhead) within the waist of the scaphoid.

Case 4: 39 year-old female clerical worker with a 5-year history of bilateral wrist pain (central and ulnar) who had sustained trauma to the lateral radial aspects of both wrists approximately 1 year prior to the development of chronic symptoms. The subject reported that previous X-ray imaging of the right wrist post-trauma was negative.The subject had been off work due to symptoms for 3 years prior to the study. MR images of the right wrist (Figure 4-9) show edema-like lesions in the lunate, capitate, 3rd metacarpal base, and ulnar aspect of the trapezoid, as well as a sclerotic lesion in the proximal ulnar aspect of the lunate.

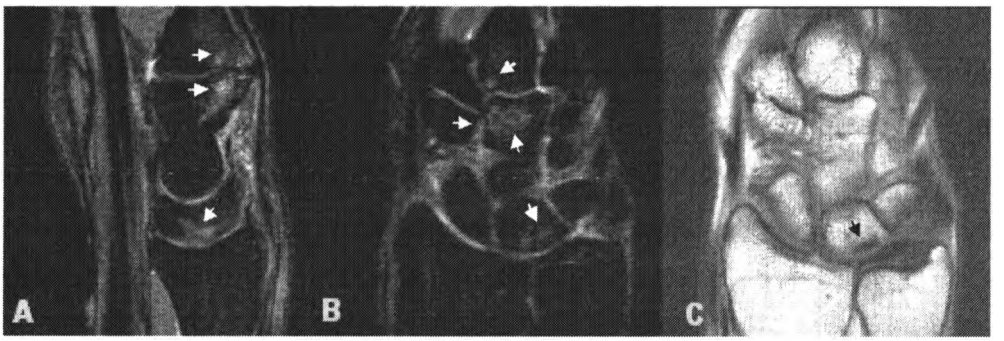

Figure 4-9. Case 4 right wrist MR images.

(A) Sagittal STIR-w MR image of the right wrist (Case 3) showing edema-like lesions (arrows) in the lunate, capitate, and 3rd MCP base. (B) Coronal STIR image showing edema-like lesions in the lunate, capitate, 3rd MCP base, and ulnar aspect of the trapezoid. (C) Coronal T1-w MR image showing a sclerotic lesion in the proximal ulnar aspect of the lunate.

4.4 Discussion

Mean wrist flexion in the UEMSD group was restricted compared to the control group in both wrists at all forearm postures with the exception of the left wrist at the supine forearm posture. These results are consistent with those of our previous study [12] and we infer that wrist extensor muscle passive tension is elevated in symptomatic but not asymptomatic computer users. Although wrist flexion was limited in the UEMSD group there was no increase in capitolunate joint motion when compared to the control group. We infer from these results that although joint dynamics was altered in the UEMSD group this had no effect on

carpal flexion kinematics, contrary to our hypothesis. The linear regression of capitolunate motion on radiolunate motion was significant for the control group, but not the UEMSD group. This result is due to wrist flexion below the targeted 50 degrees in two UEMSD wrists.

There was a reduction in mean wrist flexion between the prone and supine forearm postures in both groups, but this was only significant for the dominant wrist of the UEMSD group. In our previous study we found significant reductions in wrist flexion between the prone and supine forearm postures in both groups. We suspect that the effect was less pronounced in the present study due to the smaller size of the control group, and the fact that 4 of the UEMSD subjects had been off work for long periods compared to 1 subject in the first study. In addition, in the present study the symptoms in 3 wrists were consistent with MRI evidence of post-traumatic injury, and wrist flexion was not restricted in these wrists.

The clinical MR images of 4 UEMSD subjects showed evidence of post-traumatic injuries in two symptomatic wrists, and numerous intraosseous ganglia in 3 symptomatic wrists. The term intraosseous ganglion denotes the presence of "synovial-like" fluid/gel within an osteolytic cavity lined with fibrous collagen tissue [13,14]. Intraosseous ganglia are located in the epiphyseal region of bone, and are most often identified in the ankle, knee, hip, and wrist [15,16]. Intraosseous ganglia are histologically identical to extraosseous ganglia [13,17-21], the latter being almost always located adjacent to ligaments and tendon sheaths [22].

Intraosseous ganglia may be associated with symptoms, but may also be incidental findings. In a study of 280 elderly cadaveric wrists, for which the clinical data was unavailable, the prevalence of IOG confirmed histologically was 9.6 % [23]. The prevalence of extraosseous ganglia has been reported as 19 % in symptomatic wrists [24], and 51 % in asymptomatic wrists [25]. Extraosseous and intraosseous ganglia frequently coexist. Van den Dungen et al. [26] found

intraosseous ganglia in 47 % of patients with dorsal extraosseous ganglia of the wrist . We found extraosseous ganglia in all of the symptomatic wrists with intraosseous ganglia.

The pathogenesis of both types of ganglia is unknown, but most current theories propose that they result from either a degenerative or proliferative process, possibly initiated by biomechanical stress [13,14,27-29]. In a study of 122 extraosseous ganglia of the wrist, 30 % were associated with intercarpal ligament tears [24]. In a cadaveric study, Schrank et al. found that 43 of 47 intraosseous ganglia were located adjacent to the site of ligament attachment, the ligaments of which were in varying stages of degeneration [23].

We hypothesize that the IOG may be due to chronic carpal ligament stress based on the associated restrictions in wrist flexion. If this proposed etiology is correct, the chronic wrist symptoms may be related to chronic ligament stress rather than the presence of the ganglia. This study demonstrates that MRI may be useful for evaluating chronic wrist symptoms associated with computer use.

4.5 Conclusions

Wrist flexion was restricted in computer users with UEMSDs compared to asymptomatic computer users. There was no difference in wrist flexion kinematics between the two groups. Some symptoms appear to be related to previously undiagnosed post-traumatic injuries, while others may be related to the presence of intraosseous ganglia.

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Chapter 5

MRI-identified Abnormalities and Wrist Range of Motion in Asymptomatic Versus Symptomatic Computer Users

5.1 Introduction

Computer use is often associated with an increased prevalence of hand and wrist disorders [1-6], but the relationship remains controversial due to the frequent absence of identifiable pathology [7-12]. Symptoms are thought due to soft-tissue injury resulting from prolonged repetitive hand use, and have been referred to as repetitive strain injury, cumulative trauma disorder, occupational overuse syndrome, or work-related upper extremity musculoskeletal disorder. Symptoms may be associated with specific clinical entities such as peripheral nerve entrapment, extraosseous ganglia, tendon or muscle disorders, or may be non-specific [13-15]. Magnetic resonance imaging (MRI) has been used to study heterogeneous groups of patients with specific disorders, such as carpal tunnel syndrome [16-19], and lateral epicondylitis [20], as well as unexplained wrist pain [21,22]. To the best of our knowledge there are no previously published MRI studies of computer users with chronic wrist pain.

A number of risk factors other than repetitive hand use have been implicated including gender, psychological stress, and other psychosocial factors [23-25]. Previous studies have found that symptomatic computer users exhibit limited wrist range of motion attributed to increased antagonist wrist motor muscle tension [4,23,26]. Increased wrist motor muscle tension increases biomechanical stress within the wrist joint, and this additional stress may become clinically symptomatic [4].

The aims of this study were twofold:
1). Compare the prevalence of MRI-identified abnormalities in the wrists of symptomatic versus asymptomatic computer users. The hypothesis is that the prevalence of abnormalities will be greater in the symptomatic wrists compared to the asymptomatic wrists.
2). Compare wrist range of motion between symptomatic and asymptomatic computer users. The hypothesis is that wrist flexion will be decreased in the symptomatic group.

5.2 Methods

The study was approved by the local ethics review committee, and all subjects provided informed consent. Subjects were recruited from the community and a local RSI support group.

Inclusion criteria: 1) Daily computer use for a minimum of 4 hours per day for at least 3 years. 2) No history of upper limb symptoms to define asymptomatic. 3) Wrist pain for at least 6 months to define symptomatic.

Fourteen symptomatic computer users (10 female, 4 male), mean age = 38.4 (± 8.4 yrs.), range = 25 – 49 yrs.), and 10 asymptomatic computer users (8 female, 2 male) mean age = 39.4 (± 8.7 yrs.), range = 28 - 50 yrs.) were recruited for the study. The majority of the participants' occupations involved clerical work (n = 14) or computer programming (n = 5), and all subjects were right-dominant. The participants were asked to identify the nature, location, and duration of any upper limb symptoms on a hand/forearm diagram, and report any previous upper limb trauma or surgery. MR images of both wrists were acquired with a Siemens Espree 1.5 T scanner using an 8-channel wrist coil. The participants were imaged in the "superman" position with the arm over the head while lying prone.

The wrist was in neutral radioulnar and flexion/extension posture, the elbow posture was in extension, and the forearm posture varied between neutral and supine.

The following fast spin echo (FSE) and gradient echo (GRE) imaging sequences were used:

Axial T1 (TR 495/TE 16 ms); 320 x 320 matrix; 3 mm thick; 0.3 mm gap
Coronal T1 (TR 545/TE 16 ms); 320 x 320 matrix; 2 mm thick; 0.2 mm gap
Coronal T2 (TR 564 /TE 24 ms); 230 x 256 matrix; 2 mm thick; 0.2 mm gap; (GRE)
Coronal proton density with fatsat (TR 3700 TE 33 ms); 320 x 320 matrix; 2 mm thick; 0.2 mm gap
Axial Inversion Recovery (TR/TE/TI = 5740 /39 /140 ms); 256 x 256 matrix; 3 mm thick; 0.3 mm gap
Coronal Inversion Recovery (TR/TE/TI = 6310 /39/140 ms); 256 x 256 matrix; 2 mm thick; 0.2 mm gap
Sagittal Inversion Recovery (TR/TE/TI = 5480 /35 /140 ms); 272 x 320 matrix; 3 mm thick; 0.3 mm gap

A field of view of 90 mm x 90 mm was used for all sequences. The MR images were assessed by a radiologist (W.P.) with 25 year's experience.

Measures of maximum active wrist range of motion during flexion and radioulnar deviation were acquired with the forearm prone and elbow fully extended. Maximum wrist range of motion was taken as the maximum of three consecutive measures. Single measures of maximum active wrist flexion were acquired with the elbow flexed approximately 90 degrees and the forearm prone and supine. Range of motion measures were acquired using a Biometrics (Gwent, UK) SG65 dual axis goniometer and ADU301 angle display unit.

The distal end of the goniometer was affixed to the dorsum of the hand aligned with the third metacarpal. The proximal end was affixed to the dorsum of the forearm aligned with the radius for measurements with the forearm prone. For measurements with the forearm supine, the proximal end was affixed along a line extending from the 3rd metacarpal to the midline of the olecranon. The display unit was zeroed with the forearm in neutral flexion/extension and radioulnar deviation. The subjects were instructed to flex their wrist as far as possible while keeping their wrist in neutral radioulnar deviation by maintaining the degree of radioulnar deviation on the angle display unit as close to zero as possible. The subjects were then instructed to radioulnar deviate their wrist as far as possible while keeping their wrist in neutral flexion/extension by maintaining the degree of flexion/extension displayed on the angle display unit as close to zero as possible. The goniometer data was digitized with a Dataq Instruments (Akron, OH, USA) DI-158U analog to digital converter.

Statistical analysis was performed using SPSS (ver.17) with an alpha level of .05 for all tests. The maximum area of intraosseous and extraosseous ganglia was measured using Onis Dicom Viewer (Japan, ver. 2) by manually delineating the outer boundary of the ganglia in the imaging plane showing the greatest area. Comparison of ganglia size between groups was performed with the Mann-Whitney U test. Computer exposure and wrist range of motion data was analyzed with independent t-tests. Comparisons of prevalence were performed with the chi-square test.

5.3 Results

There was no significant difference in the number of years of computer exposure between the control group (13.1 ± 6.5 yrs.) and the symptomatic group (10.3 ± 5.1 yrs.), $p = .35$. The symptomatic subjects reported chronic wrist pain with a mean duration of 48 ± 36 months that was bilateral in 10 subjects and right-side

only in 4 subjects, for a total of 24 symptomatic wrists. Only 1 subject was not working (6 years) due to symptoms. Previous adolescent wrist fracture/sprain was reported by 2 asymptomatic and 3 symptomatic subjects. Two symptomatic subjects had previous right wrist surgery: extensor carpi radialis brevis and flexor carpi radialis tendon release in one, and flexor retinaculum release in the other.

Pain symptoms were rated as mild, moderate, or severe, and were always more pronounced in the dominant wrist. Dorsal pain sites were reported in 15 wrists (8 radial, 5 central, 4 diffuse). Volar pain sites were reported in 11 wrists (6 diffuse, 2 central, 3 ulnar). Paresthesia in one or more of the digits 2-5 was reported in 11 wrists, and thenar paresthesia was reported in 5 wrists. Wrist extensor myalgia was reported in 10 wrists and wrist flexor myalgia was reported in 2 wrists.

5.3.1 Intraosseous Ganglia

Intraosseous lesions exhibiting a fluid-like MR signal and located adjacent to ligament attachment sites (entheses) were identified in 11/24 asymptomatic wrists (25 lesions) and 18/24 symptomatic wrists (32 lesions). The imaging characteristics of these lesions are consistent with intraosseous ganglia (IOG). The prevalence of intraosseous ganglia in the symptomatic wrists (75 %) was significantly greater than in the asymptomatic wrists (45.8 %), χ^2 (1, n = 48) = 4.27, p = .039. As shown in Table 5-1, the distribution of intraosseous ganglia was similar between groups, and the majority were located in the lunate and capitate in both the asymptomatic (60 %) and symptomatic (57.5 %) wrists. One asymptomatic subject had 14 intraosseous ganglia (8 right), and one symptomatic subject had 7 intraosseous ganglia (5 left).

The carpal ligaments located immediately adjacent to the intraosseous ganglia are listed in Table 5-2. The most frequent site was adjacent to the extrinsic ligament attachment site of the dorsal lunate. Three intraosseous ganglia in the triquetrum were located adjacent to the attachment of the sheath of the distal extensor carpi ulnaris (ECU) tendon. The median maximum area of the

intraosseous ganglia in the symptomatic wrists (Mdn = 7.5 mm^2) was significantly greater than the asymptomatic wrists (Mdn = 5.0 mm^2), U(1) = 526, Z = 2.02, *p* =.043.

Examples of intraosseous ganglia in the dorsal lunate adjacent to the attachment of the dorsal extrinsic ligament in symptomatic and asymptomatic wrists are shown in Figure 5-1. Also shown are the origins of a dorsal extraosseous ganglion in both symptomatic and asymptomatic wrists. The capitolunate angle in the asymptomatic subject (Figure 5-1f) was not abnormal (> 30 degrees). Figure 5-2 shows examples of intraosseous ganglia in the distal scaphoid adjacent to the attachment of the capitoscaphoid ligament of both symptomatic and asymptomatic wrists. Examples of intraosseous ganglia in the capitate and hamate for both symptomatic and asymptomatic wrists are shown in Figure 5-3. The subject with an intraosseous ganglion of the capitate shown in Figure 5-3a had previous surgical release of the flexor carpi radialis and extensor carpi radialis brevis tendons.

Table 5-1. Intraosseous ganglia distribution by bone.

Number, location, and mean maximum dimensions and area of intraosseous ganglia (IOG) in the asymptomatic and symptomatic wrists.

Bone	# IOG Asymptomatic	# IOG Symptomatic
lunate	7	13
capitate	8	6
scaphoid	2	4
trapezoid	3	2
triquetrum	2	2
trapezium	1	2
hamate	2	2
distal radius	0	1
1st MCP	1	0
Right Wrist	11	14
Left Wrist	14	18
Total	25	32
Mean Max. Dim. (mm)	1.9 ± 1.1	3.0 ± 3.2
Mean Max. Area (mm^2)	6.1 ± 3.2	11.2 ± 11.1
Median Max. Area (mm^2)	5	7.5
# Wrists	4 Right, 7 Left	9 Right, 9 Left

Table 5-2. Intraosseous ganglia distribution by ligament.

Name (number) of the ligaments located adjacent to the intraosseous ganglia in the asymptomatic and symptomatic wrists. (C-Tra, capitotrapezoid ligament; C-H, capitohamate ligament; C-S, capitoscaphoid ligament; S-L, scapholunate ligament; L-T, lunatotriquetral ligament; CMC, carpometacarpal ligament; ECU, attachment of ECU tendon sheath on the triquetrum).

	Asymptomatic			Symptomatic		
	Intrinsic Ligament	Extrinsic Ligaments		Intrinsic Ligament	Extrinsic Ligaments	
Bone		Dorsal	Volar		Dorsal	Volar
lunate	S-L(3)	3	1	LT(3)	10	
	C-Tra(2)			C-Tra(2);S-		
capitate	C-H(5)			C(2);C-H(2)		
scaphoid	C-S(2)			C-S(4)		
trapezoid	C-Tra(2)			C-Tra(3)		
triquetrum		ECU(2)			ECU(1)	
trapezium	CMC(1)			CMC(2)		
hamate	C-H(3)			C-H(2)		
distal radius					LRL(1)	
1st metacarpal	CMC(1)					

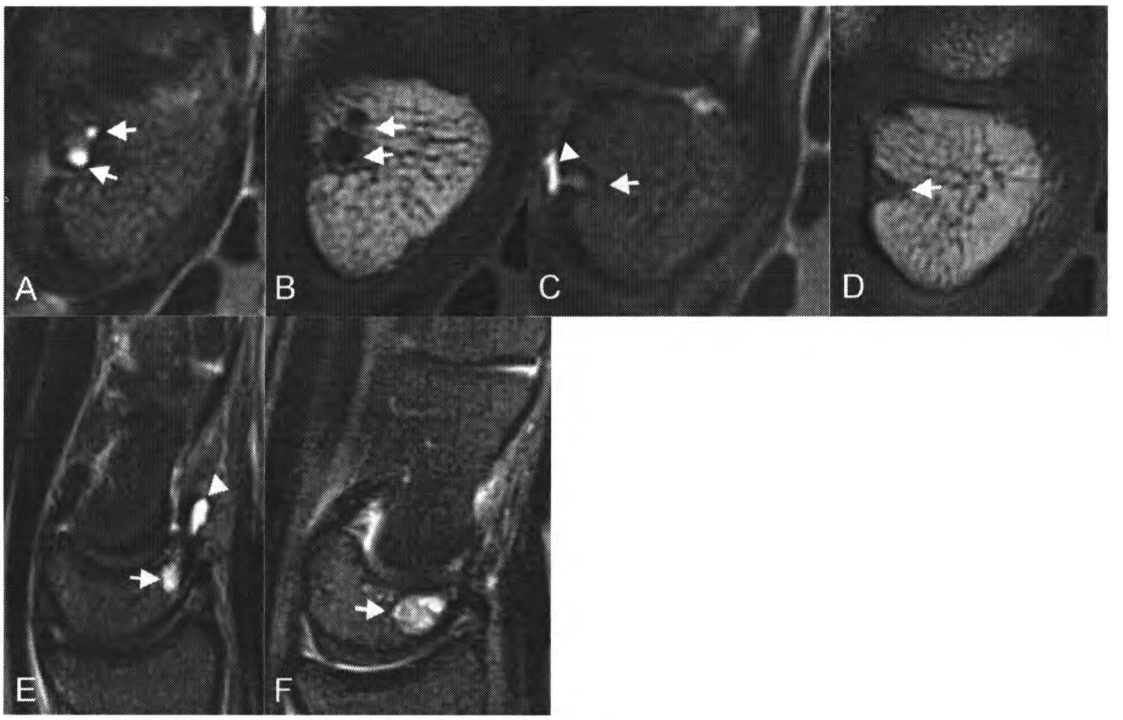

Figure 5-1. Intraosseous ganglia of the dorsal lunate.

Axial IR images (A, C) and axial T1-w images (B, D) of intraosseous ganglia (arrows) in the dorsal lunate at the attachment of the dorsal extrinsic ligaments. Sagittal IR images (E, F) of intraosseous ganglia in the dorsal lunate. Images (A, B, E) are from symptomatic wrists, and images (C, D, F) are from asymptomatic wrists. Dorsal extraosseous ganglia (arrowheads) originating at the same site are shown in C and E.

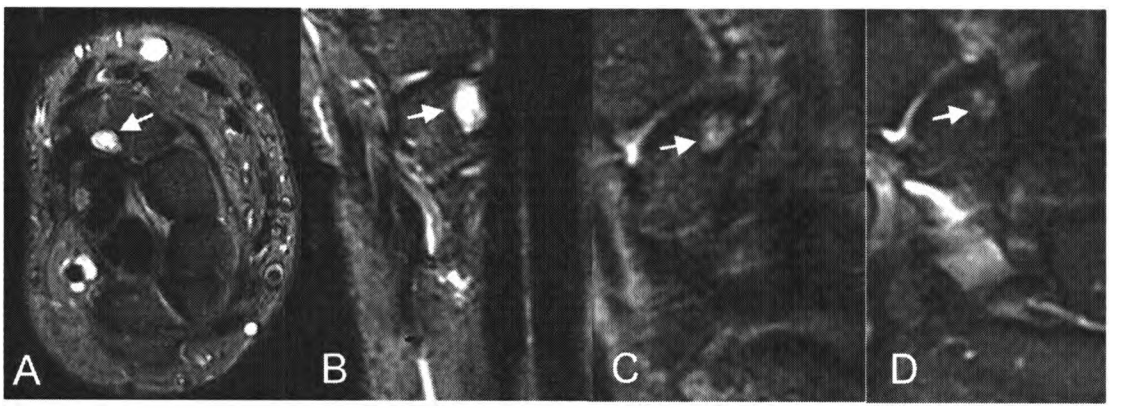

Figure 5-2. Intraosseous ganglia of the distal scaphoid.

(A) Axial and (B) coronal IR images showing an intraosseous ganglion (arrow) in the distal scaphoid adjacent to the capitoscaphoid ligament in a symptomatic wrist. Similar distal scaphoid ganglia are shown in coronal IR images of a (C) symptomatic and (D) asymptomatic wrist.

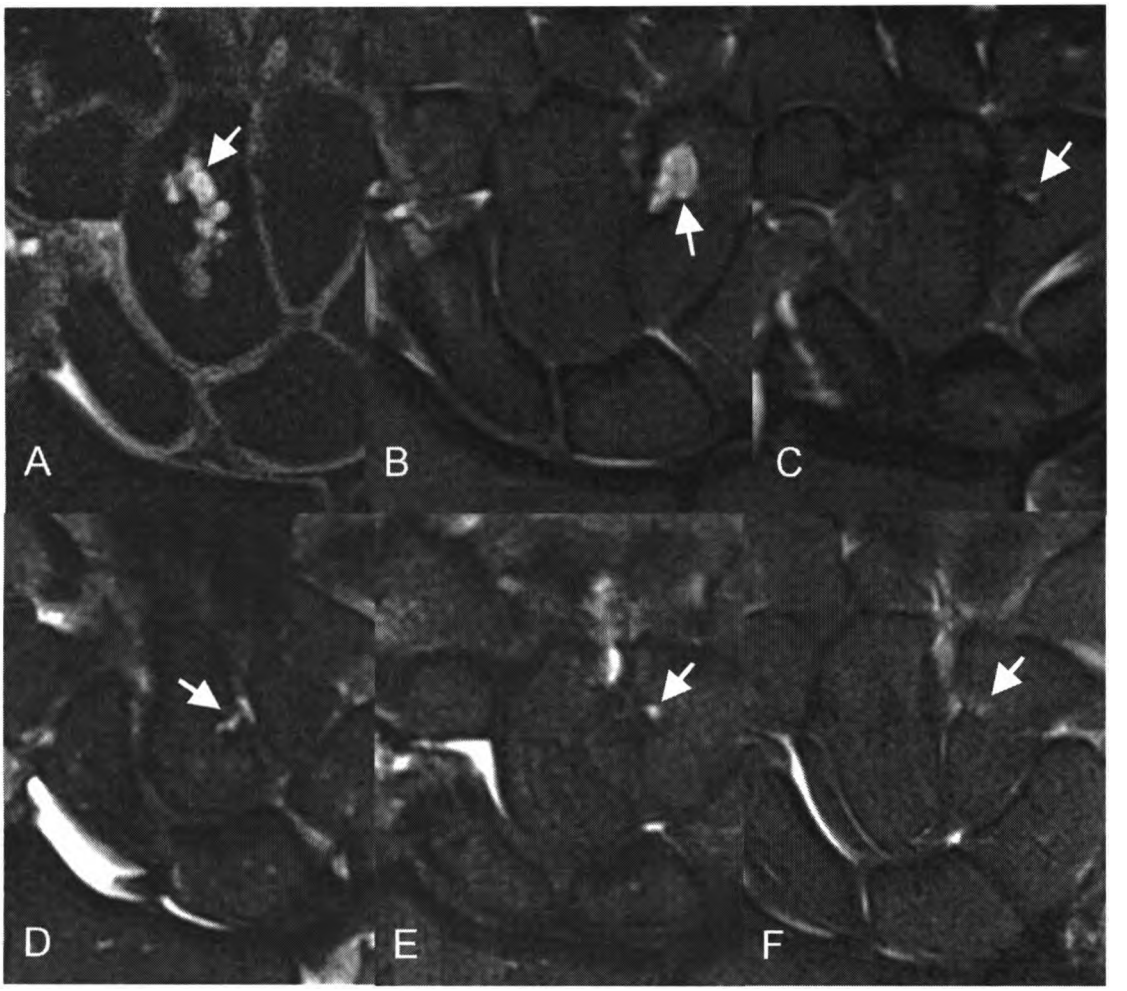

Figure 5-3. Intraosseous ganglia of the capitate and hamate.

Coronal IR images showing intraosseous ganglia (arrows) in the (A) capitate, and (B,C) hamate of symptomatic wrists; (D) capitate and (E,F) hamate of asymptomatic wrists.

5.3.2 Extraosseous Ganglia

Extraosseous ganglia were identified in 16 asymptomatic wrists (23 ganglia), and in 18 symptomatic wrists (23 ganglia) as shown in Table 3. An abnormal mass (12 x 6.0 mm) was identified in the dorsoradial aspect of one symptomatic wrist that was uncharacteristic of an extraosseous ganglion. At the time of this writing

it was not possible to determine the nature of the lesion and it was excluded from the analysis. There was no significant difference in the median maximum area of the extraosseous ganglia between the asymptomatic wrists (Mdn = 23.3 mm^2) and symptomatic wrists (Mdn = 23.6 mm^2), U(1) = 261 , Z = .182, p =.86. The most frequent location was adjacent to the pisotriquetral joint in the symptomatic wrists (26 %), and volar to the radioscaphoid articulation in symptomatic wrists (39.1 %). Intraosseous and extraosseous ganglia originating at the same site were identified in 1 asymptomatic and 4 symptomatic wrists.

Table 5-3. Extraosseous ganglia distribution and size.

Location	ASYMPTOMATIC	SYMPTOMATIC
DORSAL	5	10
VOLAR	15	12
DRUJ	3	1
# Wrists with EOG	6 right, 10 left	9 right, 9 left
# EOG in right wrist	9	16
# EOG in left wrist	14	7
Mean Max. Dimension (mm)	7.3 ± 4.0	8.4 ± 3.6
Mean Maximum Area (mm^2)	27.0 ± 17.9	32.9 ± 30
Median Maximum Area (mm^2)	23.3	23.6

5.3.3 Tendinopathies

Mild tenosynovitis of the wrist extensors was identified in 2 asymptomatic and 5 symptomatic wrists, and most often affected the extensor carpi radialis brevis and longus tendons. Subluxation of the distal ECU tendon was evident in 14 asymptomatic wrists and 14 symptomatic wrists. In 9 wrists (4 asymptomatic) > 80% of the distal ECU tendon was displaced volar to the ulnar border of the ulnar sulcus. In an additional 8 wrists (7 asymptomatic) the distal ECU tendon was

completely dislocated volar to the ulnar border of the ulnar sulcus as shown in Figure 5-4. Forearm posture during imaging varied between neutral and supine.

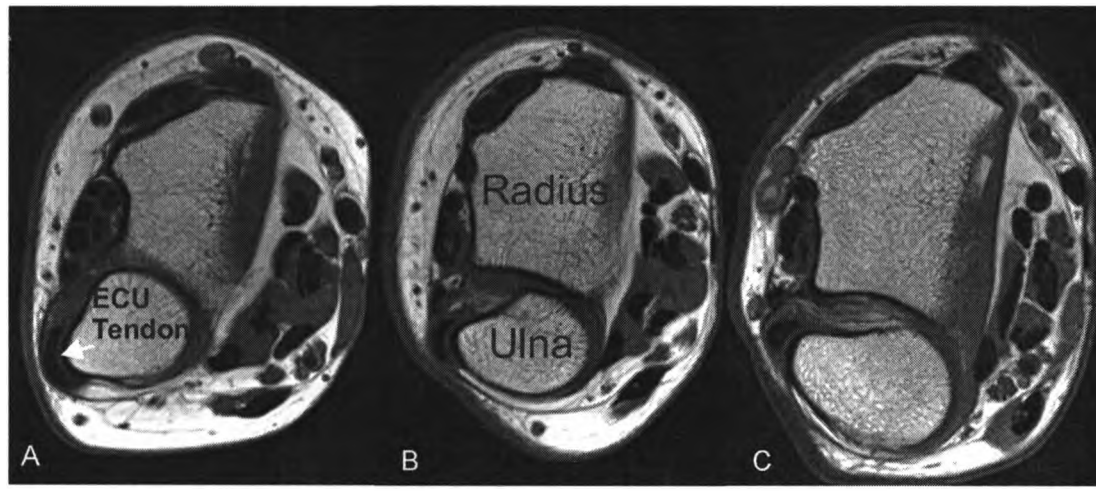

Figure 5-4. Subluxation of the distal extensor carpi ulnaris (ECU) tendon.

Axial T1-w images of 3 asymptomatic wrists showing distal ECU tendon dislocation with the forearm (A) neutral; (B) mid-supine; (C) supine.

Persistent Median Artery

A persistent median artery was identified bilaterally in 2 subjects, 1 asymptomatic and 1 with symptoms consistent with carpal tunnel syndrome. The artery was located adjacent to the median nerve in the asymptomatic subject, and within a bifurcation of the median nerve in the symptomatic subject as shown in Figure 5-5.

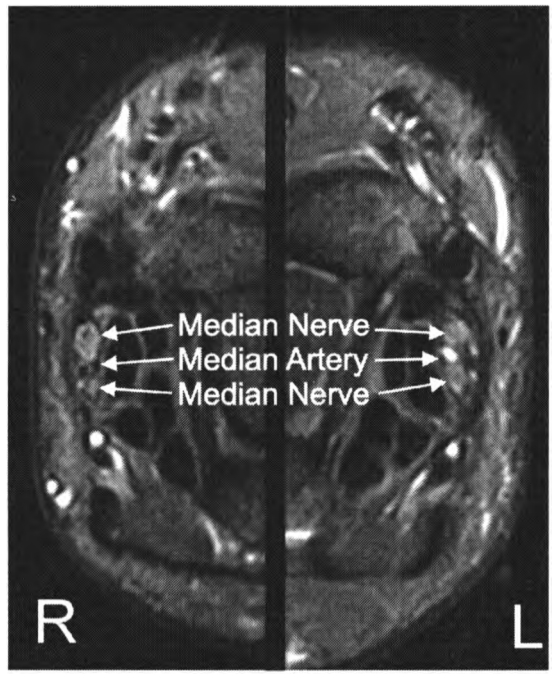

Figure 5-5. MR image of bilateral persistent median artery.

Axial STIR-w images of the right and left wrists of a subject with bilateral CTS symptoms showing a persistent median artery within the bifurcated median nerve.

5.3.4 Other MRI Findings

Other MRI findings included mild-moderate radiocarpal joint effusion (2 asymptomatic, 1 symptomatic wrist), perforation or central thinning of the triangular fibrocartilage (4 asymptomatic wrists), areas of edema and/or sclerosis (1 asymptomatic, 2 symptomatic wrists), low signal lesion in the volar lunate suggestive of granulation within a ganglion (1 symptomatic wrist), partial scapholunate ligament tear (1 symptomatic wrist), and humpback scaphoid (1 asymptomatic wrist). There was no evidence of degenerative joint disease in any wrist.

5.3.5 Wrist Range of Motion

The wrist range of motion data for the right and left wrists is shown in Table 4. The first entry lists the mean maximum active flexion measured with the forearm prone followed by the degree of ulnar deviation at the flexion limit. Listed next is the mean difference in flexion between the prone and supine forearm postures followed by the degree of ulnar deviation at the flexion limit with the forearm supine. The final entry lists the degree of radioulnar deviation. Mean right wrist flexion in the symptomatic group (60.7 ± 7.3 deg.) was significantly less than the asymptomatic group (68.8 ± 6.7 deg.), $p = .007$. During testing one symptomatic subject reported pain at the limit of wrist flexion.

Table 5-4. Wrist range of motion.

Wrist range of motion for the right and left wrists of the asymptomatic and symptomatic groups (± 1 SD; ** $p < .01$).

Wrist	Motion / Forearm Posture	Asymptomatic (n=10) (deg. ± SD)	Symptomatic (n=14) (deg. ± SD)
Right	Flexion / Prone	68.8 ± 6.7	60.7 ± 7.3**
	Ulnar Deviation / Prone	11.7 ± 6.4	8.9 ± 4.0
	ΔFlexion / Prone-Supine	3.5 ± 5.4	8.0 ± 11.9
	Ulnar Deviation	11.9 ± 7.9	10.1 ± 7.1
	Radioulnar Deviation / Prone	61.7 ± 9.3	60.1 ± 10.8
Left	Flexion / Prone	69.6 ± 9.2	61.7 ± 11.6
	Ulnar Deviation / Prone	11.7 ± 6.3	8.4 ± 6.1
	ΔFlexion / Prone-Supine	2.6 ± 8.4	8.3 ± 10.8
	Ulnar Deviation / Supine	10.6 ± 9.3	12.1 ± 9.3
	Radioulnar Deviation / Prone	62.2 ± 11.3	61.1 ± 8.8

5.4 Discussion

In this study we compared the prevalence of MRI identified abnormalities of the wrist and wrist range of motion between asymptomatic and symptomatic computer users. Similar abnormalities were frequently identified in both symptomatic and asymptomatic wrists, the majority of which indicate an excess of synovial or "synovial-like" fluid adjacent to ligaments (intraosseous and extraosseous ganglia), within the joint space (effusions), or within tendon sheaths (tenosynovitis). With the exception of intraosseous ganglia, these abnormalities have previously been associated with symptomatic computer users. Subluxation or dislocation of the distal ECU tendon was also common in both groups, and to our knowledge, has not previously been associated with computer use.

Symptoms were always more pronounced in the right wrist, which was the dominant wrist of all subjects. Dorsal symptoms were more frequent and focally localized than volar symptoms. In only one case could the symptoms be attributed to a specific abnormality. A female subject (age 35) with a 6-year history of bilateral symptoms consistent with carpal tunnel syndrome (CTS) has a persistent median artery within a bifurcation of the median nerve in both wrists (Figure **5-5**). The median artery normally involutes in fetal life, and previous studies have associated CTS with a persistent median artery [27,28]. Cases of persistent median artery with an associated bifurcated median nerve are reportedly rare [29]. The association between symptoms and abnormalities for the remaining wrists are less clear.

5.4.1 Distal ECU Tendon Subluxation

The stability of the distal ECU tendon at the distal ulna is dependent on the integrity of the surrounding sub-sheath, and subluxation can result from rupture

of the sheath and migration of the tendon, or stripping of the sheath's ulnar attachment and formation of a false sheath [30]. There was no evidence of rupture of the sub-sheath in any wrist in the present study, indicating that the subluxation was due to formation of a false sheath. Subluxation or dislocation of the distal ECU tendon was found in 58.3 % of both asymptomatic and symptomatic wrists, but no ulnar symptoms were reported by any of these subjects.

In normal wrists, ECU subluxation has been shown to occur with forearm supination, and increase with ulnar deviation and wrist flexion [31]. However, in that study no tendons were subluxed completely volar to the ulnar border of the ulnar sulcus, which indicates dislocation. In the present study wrist posture was neutral, but forearm posture varied between neutral and supine, presumably due to the "superman" imaging position. Nonetheless, in 8 wrists (7 asymptomatic) the ECU tendon was completely volar to the ulnar border of the ulnar sulcus. ECU tendon subluxation has been associated with a shallow ulnar sulcus [32], thought to be either congenital [33] or due to bone attrition [32]. We are not aware of an imaging standard for determining sulcus depth, but in the present study ECU tendon subluxation was associated with sulci that were comparatively shallow. The high prevalence of asymptomatic distal ECU tendon subluxation/dislocation observed in the present study suggests that ulnar-sided symptoms might be misattributed to this pathology in this occupational group. ECU tendon subluxation or dislocation is typically associated with sports such as golf and tennis. Whether ECU subluxation is related to computer use is a question that requires a prospective study.

5.4.2 Extraosseous Ganglia

An extraosseous ganglion is an avascular collagen-walled sac similar to a synovial cyst, but devoid of synovial cells. The chemical constituents of the fluid contents are similar to synovial fluid [34,35]. Extraosseous ganglia are usually

located adjacent to joints, and frequently communicate with the joint space [36-40]. The etiology of extraosseous ganglia is thought to be synovial herniation [41], mucoid degeneration of connective tissue [41], or proliferation of mucin producing cells [38].

In the present study, the prevalence of extraosseous ganglia was 66.6 % in the asymptomatic wrists and 75 % in the symptomatic wrists. The positive predictive value of MRI in detecting occult wrist ganglia that were confirmed histologically was 75 % [22]. The prevalence of extraosseous ganglia identified with MRI has been reported as 19 % in symptomatic wrists [42], and 51 % in asymptomatic wrists [43]. Symptoms may relate to the location of the ganglia, and were predominantly (86%) volar in a study of asymptomatic wrists [43]. In the present study, dorsal ganglia were identified in 47.8% of symptomatic wrists and in 34.8 % of asymptomatic wrists, but there was nothing that differentiated between them. Extraosseous ganglia may cause symptoms due to a mass effect, but there was no significant difference in mean ganglion size between the asymptomatic and symptomatic wrists.

Treatment of extraosseous ganglia by aspiration or excision has not been shown to provide any significant long-term benefit compared to no treatment for both dorsal [44] and volar [45] wrist ganglia. It has been suggested that persistent symptoms may be due to an underlying joint abnormality [42]. Previous studies have shown an association between extraosseous ganglia and ligament degeneration [46,47], and it has been suggested that ligament degeneration may result from acute trauma or chronic stress [47].

5.4.3 Intraosseous Ganglia

The term “intraosseous ganglion” describes a “synovial-like” fluid within an osteolytic cavity lined with fibrous collagen tissue [48,49]. They have been identified in many joints, including the foot, ankle, knee, hip, elbow, and wrist

[50]. Intraosseous ganglia are histologically similar to extraosseous ganglia [49,51-53], and although similar to degenerative cysts, intraosseous ganglia do not communicate with the joint space via eroded hyaline cartilage [54]. The etiology of intraosseous ganglia is unknown, but theories include vascular disturbance [50,55], proliferation of mucin producing cells [51], distractive bone stress at the ligament attachment [56], or ligament degeneration [54,57].

The prevalence of intraosseous "fluid-like" lesions with an MRI appearance consistent with intraosseous ganglia was greater in the symptomatic wrists (75%) compared to the asymptomatic wrists (45.8 %). Not all lesions were of the characteristic spherical or oval shape of intraosseous ganglia, but all were located adjacent to ligament attachment sites. We cannot be certain that these lesions are true intraosseous ganglia without histological confirmation, although identification with MRI appears to be highly specific. In a cadaveric wrist study, all of the intraosseous ganglia tentatively identified with MRI were subsequently confirmed histologically [54]. The prevalence of intraosseous ganglia in that study was 9.6 %, but the clinical history was unknown [54]. An MRI study of 30 asymptomatic subjects with a mean age of 31 years found 24 "bright osseous lesions" in 14 wrists, but there was no agreement on whether the lesions represented intraosseous ganglia, erosions, edema, or sub-chondral cysts [58]. The high prevalence of intraosseous ganglia identified in the present study may be due to the sensitivity of the inversion recovery sequence [59], as 21 of the 58 ganglia identified had a maximum dimension of less than 2 mm.

Intraosseous ganglia have been associated with non-specific wrist pain [21], but may also be incidental findings. We are aware of only one previous case report of a computer user with non-specific wrist pain that was attributed to an intraosseous ganglion [60]. It has been suggested that the symptoms associated with intraosseous ganglia may be related to their size [21,50,51]. In the present study, the intraosseous ganglia in the symptomatic wrists were significantly larger than those in the asymptomatic wrists. However, some larger intraosseous

ganglia were asymptomatic in both symptomatic and asymptomatic wrists. In some cases, the symptoms were well-localized to the sites of intraosseous ganglia, but in other cases the symptoms were diffuse. The distribution of the intraosseous ganglia was very similar to previous studies, with the majority being located in the lunate, capitate, and scaphoid [54,57]. However, in the study by Schrank et al. [54] all of the scaphoid ganglia were located in proximity to the scapholunate or palmar intrinsic ligaments, whereas in the present study all of the scaphoid ganglia were located in proximity to the capitoscaphoid ligament (Table 5-2). Scaphoid ganglia located in proximity to the capitoscaphoid ligament have been reported previously in symptomatic [21,60,61] and asymptomatic subjects [48]. Treatment of intraosseous ganglia with curettage and bone grafting is highly effective in relieving symptoms [21,60,62,63].

Schrank et al. [54] found an association between intraosseous ganglia and ligament degeneration, and suggest that intrusion of fluid from degenerated ligaments is the initiating event. An association has also been shown between intraosseous and extraosseous ganglia of the wrist [57,63,64], which in some cases may arise from the same site as shown in Figure 5-3. Some authors believe that intraosseous ganglia result from cortical penetration by extraosseous ganglia [65], or that extraosseous ganglia result from expansion of intraosseous ganglia thru the cortex [48], or that both mechanisms occur [50]. It has also been proposed that chronic ligament overload is the initiating factor for both [57]. In the present study, the prevalence of coexisting intraosseous and extraosseous ganglia was 25 % in the asymptomatic wrists and 58.3 % in the symptomatic wrists. Intraosseous and extraosseous ganglia originating at the same site were found in 1 asymptomatic and 4 symptomatic wrists.

We agree with Van den Dungen et al. [57] that both types of ganglia may be different manifestations of chronic ligament stress. The increased size of the intraosseous ganglia in the symptomatic wrists may reflect higher levels or longer durations of ligament stress. There was no difference in the mean years of

computer exposure between groups, although other work-related factors such as repetition rate, posture, or continuous daily exposure may have differed between groups. Non-work related factors including domestic and recreational activities may have also differed between groups.

5.4.4 Wrist Range of Motion

Mean wrist flexion in the symptomatic group was less than the asymptomatic group in both wrists, but this was only statistically significant for the dominant wrist with the forearm prone (Table 5-4). These results agree with those of our previous study where we found significant limitations in dominant wrist flexion in symptomatic computer users when measured with the forearm prone [26]. In that study we also found that wrist flexion decreased significantly when measured with the forearm supine in both wrists in both the symptomatic and control groups. We attributed this decrease to the increase in the length of the moment arm of the distal ECU tendon that occurs with forearm supination [66-68]. There was no significant decrease in wrist flexion with forearm supination in either group in the present study. Some of this may have been due to the subject's tendency to ulnar deviate during wrist flexion — motion that was constrained in our previous study. We speculate that the lack of a significant decrease may also be related to the frequent finding of ECU subluxation/dislocation, which would minimize/eliminate the increase in the distal ECU tendon's moment arm that normally occurs with forearm supination.

Wrist flexion restrictions in the dominant wrist of symptomatic subjects may be due to pain avoidance; however, in 3 of the 4 subjects with only dominant wrist symptoms, flexion in their non-dominant wrist was also restricted (< 55 deg.). Wrist flexion restrictions may be due to decreased extensibility of the wrist extensor muscles, but there was no significant difference in radioulnar deviation between groups. This apparently contradictory finding, given the restrictions in wrist flexion, has been reported in a previous study of symptomatic repetitive

workers with restricted wrist flexion [15]. One plausible explanation is that prolonged radioulnar deviation during computer use causes stress-induced creep of the carpal ligaments.

Restricted wrist flexion is a common finding in symptomatic computer users [4,15,23,26], which may be due to increased antagonist muscle co-contraction or myofascial shortening [4,23]. Although the flexion restrictions we observed were modest, they would create a substantial increase in the biomechanical loading of the muscles, tendons, and ligaments of the wrist during extremes of wrist flexion or radioulnar deviation. The associated wrist extensor tenosynovitis, wrist extensor/flexor myalgia, and intraosseous ganglia in the symptomatic wrists may all be indicative of increased biomechanical loading. Increased loading may occur during computer use, but presumably any repetitive activity demanding extremes of wrist flexion or radioulnar deviation could contribute.

5.5 Conclusions

This study found an increased prevalence of intraosseous ganglia, wrist extensor tenosynovitis and wrist motor muscle myalgia in symptomatic wrists compared to asymptomatic wrists. Intraosseous ganglia were significantly larger in the symptomatic wrists compared to the asymptomatic wrists, but size was not a consistent determinant of pain symptoms. Dominant wrist flexion was restricted in the symptomatic group compared to the asymptomatic group. Equal numbers of extraosseous ganglia of similar size were identified in both symptomatic and asymptomatic wrists. Subluxation of the distal ECU tendon was common in both groups, but was not associated with ulnar-sided symptoms.

5.6 Limitations

This study is limited by the use of a convenience sample of a small number of self-referred subjects whose computer usage per day may have varied, and the lack of histological confirmation of the intraosseous ganglia.

5.7 References

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Chapter 6

Discussion and Conclusions

6.1 Discussion

The primary objective of this thesis was to investigate the wrist kinematics and prevalence of wrist pathology in computer users with and without chronic wrist pain. Wrist symptoms related to computer use is a controversial issue, but such controversy is not uncommon with respect to musculoskeletal disorders attributed to occupation [1,2]. The controversy with respect to computer use stems largely from the lack of clear evidence on the relationship between exposure and its presumed health effects [3,4].

A very recent review of musculoskeletal disorders associated with computer use highlighted the need for research focusing on the possible effect of computer work on the musculoskeletal system [5]. The authors note that epidemiological studies to date have been based primarily on subjective measures of symptoms, and that there is limited evidence of a causal association between computer use and upper extremity symptoms [5].

The main findings of this thesis indicate that there are functional and pathological differences between asymptomatic and symptomatic computer users. The questions of whether the functional differences (i.e., ROM restrictions) are causal in the development of the pathologies, and whether these pathologies are responsible for the pain symptoms are yet to be answered. However, these objective measures may further our understanding of an individual's susceptibility to develop symptoms related to computer use, as well as the physiological basis of non-specific wrist pain.

To our knowledge, this is the first study employing MRI to assess wrist pathology in this specific occupational group. It is hoped that the results contained in this thesis will provide some direction for future studies.

The following sections summarize the observations, interpretations, limitations, and avenues for future work for the functional and pathological differences observed between asymptomatic and symptomatic computer users.

6.2 Functional Differences

Maximum active wrist flexion was limited in symptomatic computer users when measured with the forearm prone in all of the studies contained in this thesis In addition, there was a significant loss of wrist flexion between the prone and supine forearm postures in two studies comparing symptomatic computer users to individuals with minimal computer exposure (Chapter 3) or asymptomatic computer users (Chapter 4). In the first study (Chapter 3) both groups exhibited reductions in wrist flexion with the forearm supine, but the reduction was approximately 100% greater in the symptomatic group. In the second study (Chapter 4) the reduction in flexion between the prone and supine postures was only significant in the dominant wrist of the symptomatic group. In the same study, no differences were observed for carpal flexion kinematics between symptomatic and asymptomatic computer users. In the third study (Chapter 5), wrist flexion did not differ between the prone and supine forearm postures in either the symptomatic or asymptomatic computer users.

Interpretation

Reduced wrist flexion has been observed in previous studies [6,7] of computer users, and was attributed to increased antagonist (extensor) muscle tension.

Wrist flexion restrictions could also result from flexor muscle weakness or inhibition, but it is difficult to conceive how either mechanism would be affected by changes in forearm posture.

Increased wrist extensor muscle tension could be either passive or active. We infer that it is passive because previous studies have shown that wrist flexion torque exceeds wrist extension torque over the range of wrist joint angles measured in our study [32].

We attribute the reduction in wrist flexion between the prone and supine forearm postures exhibited by both symptomatic and asymptomatic subjects to the known increase in the moment arm of the distal ECU tendon that occurs with forearm supination. This reduction in flexion with forearm supination has been demonstrated in one previous study of normal individuals [8]. As the reduction in flexion in the symptomatic group was significantly greater than the asymptomatic group we infer that ECU muscle tension is elevated in the symptomatic group. Again, we infer that the tension increase is passive rather than active due to the superiority of wrist flexion torque.

In my third study (Chapter 5) there was no apparent effect of forearm posture on wrist flexion in either group. However, the frequent finding of ECU subluxation/dislocation in both groups indicates altered biomechanics of the distal ECU tendon. It is readily apparent that subluxation/dislocation would minimize/eliminate the increase in the distal ECU tendon's moment arm that normally occurs with forearm supination. Thus, we are unable to infer whether ECU muscle tension differed between groups.

A study of symptomatic musicians also found wrist range of motion limitations, which were attributed to undefined "biomechanical restrictions" [9]. We hypothesized that increased extensor muscle tension would alter carpal kinematics by increasing the contribution of the capitolunate joint to wrist flexion.

The results of our study described in Chapter 4 showed significant restrictions in wrist flexion in symptomatic versus asymptomatic computer users, but there was no significant difference in carpal flexion kinematics. From this result we assume that any biomechanical restrictions involve the musculature, and flexion restrictions represent a decrease in extensibility of the extensor tendons rather than the kinematics of the wrist joint.

There was no difference in wrist radioulnar deviation between groups in the third study (Chapter 5). This apparently contradictory finding, given the restrictions in wrist flexion, has been reported in a previous study of symptomatic repetitive workers with restricted wrist flexion [10]. One plausible explanation is that prolonged radioulnar deviation during computer use causes stress-induced creep of the carpal ligaments.

The observations from the studies contained in this thesis and from previous studies [6,7] indicate that wrist flexion restrictions are common in symptomatic computer users. Whether these restrictions are a consequence of computer use is unknown, as there do not appear to be any longitudinal studies examining wrist range of motion in computer users.

We infer that the flexion restrictions are due to increased wrist extensor muscle passive tension. It has been suggested that prolonged abnormal postures may affect muscle tension due to muscle length adaptation [11], a theory that is supported by animal studies [12,13]. However, the fact that only a segment of computer users with equivalent exposure exhibit flexion restrictions suggests the influence of additional factors. These factors may act to increase susceptibility or alternatively, may act to decrease susceptibility in certain individuals. The influence of psychological factors on muscle activation is well known, and ergonomic and other workload factors, as well as avocational activities may affect wrist extensor muscle passive tension.

Implications

Increased wrist extensor muscle passive tension would affect the dynamics of the wrist joint, particularly at the extremes of joint motion. Repetitive work that demands extremes of flexion or radioulnar deviation would increase tensile stress of the wrist motor muscles and tendons, and create an abnormal distribution of forces within the wrist joint. The dissipation of theses forces would increase compressive stresses of the carpal bones at the articulations, as well as tensile or shear stress of the carpal ligaments at their attachments.

Limitations

The major limitation of the studies contained in this thesis concern the small subject sample sizes, although the wrist ROM results were consistent with studies having larger sample populations. The cross-sectional design of the studies in this thesis precludes any inferences with regard to computer use and wrist flexion restrictions. The inference drawn with respect to increases in extensor muscle passive tension was not confirmed by any objective measure of extensor muscle passive tension.

Future work

Electromyographic measures of muscle activity would determine conclusively if wrist flexor muscle inhibition or increased extensor musce activation is occurring at the wrist flexion limit. A non-invasive, objective measure of wrist extensor muscle passive tension would confirm whether it is elevated in symptomatic computer users. Magnetic resonance elastography is a non-invasive technique that is showing promise as a method to quantify muscle tension, whether passive or active, in vivo. A prospective study of newly employed computer users utilizing both electromyography and MR elastography may also help determine whether increased muscle passive tension exists prior to, or as a consequence of computer exposure.

6.3 Pathological Differences

Wrist pathologies were identified in a subset of symptomatic computers users referred for clinical MRI examinations in the study described in Chapter 4. The MRI study described in Chapter 5 identified a significantly increased prevalence of pathologies in the wrists of symptomatic versus asymptomatic computer users.

6.3.1 Post-traumatic and congenital abnormalities

The identification of presumably post-traumatic injuries in two symptomatic subjects (DRUJ instability, bone edema), and a congenital abnormality in one subject (bilateral persistent median artery) indicate that MRI can detect clinically significant abnormalities in subjects with chronic wrist symptoms that are perhaps exacerbated by computer use.

6.3.2 Tendinopathies

The results of the study presented in Chapter 5 showed that distal ECU tendon instability was common in both groups. Mild tenosynovitis of the wrist extensor and flexor tendons was an infrequent finding, but more common in the symptomatic wrists.

Interpretation

The degree of distal ECU tendon subluxation is known to increase with forearm supination in normal subjects, but complete dislocation can be considered abnormal and is usually associated with hand intensive sports activities.The frequent finding of distal ECU tendon subluxation/dislocation without evidence of rupture of the tendon sub-sheath suggests an etiology involving chronic rather than acute stress. Previous reports indicate that this pathology is often associated with ulnar-sided wrist pain, particularly during forearm

pronosupination. The absence of ulnar-sided symptoms in the subjects in the present study may be related to infrequent demands for forearm pronosupination in these subjects.

Implications

As mentioned previously, instability of the distal ECU tendon would minimize or eliminate the increase in the distal ECU tendon's moment arm that normally occurs with forearm supination. This indicates that this pathology is a potential confound when comparing wrist flexion ROM between the prone and supine forearm postures. Cases of mild tenosynovitis were infrequent, but are indicative of chronic tendon stress.

Limitations

The true prevalence of ECU dislocation in this study may have been greater than what was observed, as not all of the subjects were imaged with the forearm fully supine. The possibility that distal ECU tendon instability develops as a consequence of computer use can only be ascertained with a prospective study.

Future work

MRI scanning with the forearm fully supine would provide a true measure of the prevalence of distal ECU tendon subluxation.

6.3.3 Extraosseous ganglia

The study described in Chapter 5 demonstrated that extraosseous ganglia were very common in both symptomatic and asymptomatic wrists. There was no difference in the prevalence or size of the extraosseous ganglia between the symptomatic and asymptomatic wrists. Based on the imaging findings, there was nothing to differentiate between the extraosseous ganglia in the symptomatic versus asymptomatic wrists. The origins of the extraosseous ganglia were

evident in only a few cases, as were the presence of concomitant intraosseous ganglia.

Interpretation

The prevalence of extraosseous ganglia shown in this study may be related to computer use, or avocational activities. A previous MRI study [14] of asymptomatic subjects identified a similar prevalence of extraosseous ganglia, although the occupations of the subjects were not reported. To determine whether extraosseous ganglia are related to computer use would require a prospective study, or at least a comparison with prevalence rates from cross-sectional studies that stratify the subjects by occupation.

As outlined in Chapter 2, the etiology and symptomology of extraosseous ganglia are controversial. The treatment of extraosseous ganglia does not provide any significant benefit compared to no treatment over the long-term [15,16]. The extraosseous ganglia identified in my study were remarkably similar between symptomatic and symptomatic wrists, suggesting that symptoms may be unrelated to their presence.

Limitations

The lack of histological confirmation makes the diagnosis tentative, but the MRI characteristics of the lesions were consistent with extraosseous ganglia. In only a few cases was it possible to identify the ganglion's connection to the underlying joint.

Future work

The use of 3-dimensional MRI could improve the detection of any connection between the extraosseous ganglia and the underlying joint structures. The potential benefits are twofold: identifying the true prevalence of concomitant intraosseous ganglia, and defining the joint structures presumably undergoing

stress, the latter of which may be useful for localizing symptoms to the ganglion's origin.

6.3.4 Intraosseous ganglia

The study described in Chapter 5 demonstrated that intraosseous ganglia were common in computer users, and that the prevalence and size of intraosseous ganglia were greater in the symptomatic wrists compared to the asymptomatic wrists.

Interpretation

The overall prevalence of intraosseous ganglia shown in this study may be related to computer use, but a prospective study is required. The increased prevalence and size of intraosseous ganglia in the symptomatic wrists suggests that they may be related to symptoms. Symptoms were often, but not always well-localized to the sites of larger intraosseous ganglia. As outlined in Chapter 2, the etiology and symptomology of intraosseous ganglia are controversial. Intraosseous ganglia may or may not be symptomatic, which may be related to their size, although this is not a consistent determinant. Increased radiotracer uptake, presumed due to increased osteocyte activity, is associated with symptomatic intraosseous ganglia [17-19].

Intraosseous ganglia arise adjacent to ligament attachment sites, whose ligaments are often degenerate, suggesting an etiology involving chronic ligament stress [20]. The flexion restrictions in the dominant wrist of the symptomatic subjects we infer to be indicative of increased extensor muscle passive tension. The effects of increased extensor muscle tension on the dynamics of the wrist joint may translate to chronic stress of carpal ligaments.

Limitations

The lack of histological confirmation makes the diagnosis tentative, but the MRI characteristics of the lesions were consistent with intraosseous ganglia. No information was available with respect to any associated degeneration of the carpal ligaments

Future work

Improved delineation of intraosseous ganglia with 3-D MRI could improve the localization of ganglia to the ligament attachment site, as well as identify communication with adjacent extraosseous ganglia. MRI utilizing an ultra-short T2 sequence could identify degenerate carpal ligaments, thus highlighting the regions of increased joint stress. Detection of ganglia and ligament degeneration may identify specific patterns of wrist joint stress in this occupational group.

Although a radiotracer may identify increased osteocyte activity in some symptomatic ganglia, the sensitivity and specificity of this technique has not yet been established. With the assumption that increased osteocyte activity results from expansion of the intraosseous ganglion due to fluid accumulation, it may be useful to characterize the ganglion fluid using T2 relaxometry. T2 mapping before and after wrist exercise could identify changes in ganglion fluid concentration that result from fluid intrusion, which may be specific to symptomatic ganglia.

6.4 Conclusions

In this thesis I showed that wrist flexion was limited in symptomatic computer users compared to both asymptomatic computer users and individuals with minimal computer exposure. We identified a number of wrist abnormalities in 3 subjects which appear to be post-traumatic or congential in nature. We found that extraosseous ganglia were common in both symptomatic and asymptomatic computer users. Intraosseous ganglia were more numerous and larger in the symptomatic computer users. The etiology of extraosseous and intraosseous

ganglia is thought to be related to chronic ligament stress, but this theory is controversial as the source of the stress is often not apparent. We speculate that restrictions in wrist flexion are due to increased extensor muscle passive tension, and may be a source of chronic ligament stress.

6.5 References

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APPENDIX A Ethics Approval

Office of Research Ethics

The University of Western Ontario
Room 00045 Dental Sciences Building, London, ON, Canada N6A 5C1
Telephone: (519) 661-3036 Fax: (519) 850-2466 Email: ethics@uwo.ca
Website: www.uwo.ca/research/ethics

Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. R.T Thompson
Review Number: 11042 **Revision Number:** 1
Protocol Title: Factors Affecting Wrist Motion In Repetitive Strain Injury
Department and Institution: Nuclear Medicine, St. Joseph's Health Care London
Sponsor:
Ethics Approval Date: November 28, 2005 **Expiry Date:** April 30, 2006
Documents Reviewed and Approved: Revised Study End Date
Documents Received for Information:

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (HSREB) which is organized and operates according to the Tri-Council Policy Statement and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted full board approval to the above named research study on the approval date noted above. The membership of this REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

This approval shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request Form.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the HSREB except when necessary to eliminate immediate hazards to the subject or when the change(s) involve only logistical or administrative aspects of the study (e.g. change of monitor, telephone number). Expedited review of minor change(s) in ongoing studies will be considered. Subjects must receive a copy of the signed information/consent documentation.

Investigators must promptly also report to the HSREB:
a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
b) all adverse and unexpected experiences or events that are both serious and unexpected;
c) new information that may adversely affect the safety of the subjects or the conduct of the study.

If these changes/adverse events require a change to the information/consent documentation, and/or recruitment advertisement, the newly revised information/consent documentation, and/or advertisement, must be submitted to this office for approval.

Members of the HSREB who are named as investigators in research studies, or declare a conflict of interest, do not participate in discussion related to, nor vote on, such studies when they are presented to the HSREB.

Chair of HSREB: Dr. John W. McDonald
Deputy Chair: Susan Hoddinott

Ethics Officer to Contact for Further Information			
☐ Janice Sutherland	☐ Karen Kueneman	☐ Susan Underhill	☑ Jennifer McEwen

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Office of Research Ethics

The University of Western Ontario
Room 4180 Support Services Building, London, ON, Canada N6A 5C1
Telephone: (519) 661-3036 Fax: (519) 850-2466 Email: ethics@uwo.ca
Website: www.uwo.ca/research/ethics

Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. R.T. Thompson **Review Level:** Expedited
Review Number: 15866E **Revision Number:** 3
Review Date: March 10, 2010 **Approved Local # of Participants:** 30
Protocol Title: Investigation of upper extremity musculoskeletal disorders associated with computer use
Department and Institution: Nuclear Medicine, St. Joseph's Health Care London
Sponsor: INTERNAL RESEARCH FUND-HOSPITAL
Ethics Approval Date: March 10, 2010 **Expiry Date:** June 30, 2010
Documents Reviewed and Approved: Revised study end date.
Documents Received for Information:

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced revision(s) or amendment(s) on the approval date noted above. The membership of this REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request Form.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the HSREB except when necessary to eliminate immediate hazards to the subject or when the change(s) involve only logistical or administrative aspects of the study (e.g. change of monitor, telephone number). Expedited review of minor change(s) in ongoing studies will be considered. Subjects must receive a copy of the signed information/consent documentation.

Investigators must promptly also report to the HSREB:

a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
b) all adverse and unexpected experiences or events that are both serious and unexpected;
c) new information that may adversely affect the safety of the subjects or the conduct of the study.

If these changes/adverse events require a change to the information/consent documentation, and/or recruitment advertisement, the newly revised information/consent documentation, and/or advertisement, must be submitted to this office for approval.

Members of the HSREB who are named as investigators in research studies, or declare a conflict of interest, do not participate in discussion related to, nor vote on, such studies when they are presented to the HSREB.

Chair of HSREB: Dr. Joseph Gilbert
FDA Ref. #: IRB 00000940

Ethics Officer to Contact for Further Information			
☐ Janice Sutherland	☐ Elizabeth Wambolt	☑ Grace Kelly	☐ Denise Grafton

This is an official document. Please retain the original in your files.

cc: ORE File
LHRI

Office of Research Ethics
The University of Western Ontario
Room 4180 Support Services Building, London, ON, Canada N6A 5C1
Telephone: (519) 661-3036 Fax: (519) 850-2466 Email: ethics@uwo.ca
Website: www.uwo.ca/research/ethics

Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. R.T. Thompson
Review Number: 15866E **Revision Number:** 2
Review Date: November 12, 2009 **Review Level:** Expedited
Protocol Title: Investigation of upper extremity musculoskeletal disorders associated with computer use
Department and Institution: Nuclear Medicine, St. Joseph's Health Care London
Sponsor: INTERNAL RESEARCH FUND-HOSPITAL
Ethics Approval Date: November 12, 2009 **Expiry Date:** March 31, 2010
Documents Reviewed and Approved: Revised Study End Date
Documents Received for Information:

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Western

Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. R.T. Thompson
Review Number: 15866E **Revision Number:** 1
Review Date: July 9, 2009 **Review Level:** Expedited
Protocol Title: Investigation of upper extremity musculoskeletal disorders associated with computer use
Department and Institution: Nuclear Medicine, St. Joseph's Health Care London
Sponsor: INTERNAL RESEARCH FUND-HOSPITAL
Ethics Approval Date: July 9, 2009 **Expiry Date:** December 31, 2009
Documents Reviewed and Approved: End Date Revision
Documents Received for Information:

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