THE BIOMECHANICAL ANALYSIS OF THE HAND IN RHEUMATOID ARTHRITIS PATIENTS WITH MCP ARTHROPLASTY

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ABSTRACT

Rheumatoid arthritis (RA) is a chronic inflammatory disease, causing extreme deformity, pain and swelling of joints, severely affecting quality of life. Arthroplasty has had considerable success in larger joints such as the hip. The most frequently used artificial finger joints rely on a silicone elastomer component for their flexibility. However, success of these implants has been mixed; with fracture rates for the elastomer component reported to be up to 82%. It is currently unknown why fracture of the elastomer occurs so frequently. Motion analysis was used to determine range of motion (ROM) of the metacarpophalangeal (MCP) joints in patients with rheumatoid arthritis, both without and with arthroplasty, to determine how the procedure affects motion of the joint. A 12 camera motion capture system was used to capture hand kinematic data. Preliminary experiments determined the best positions for reflective markers for measuring motion. Subjects consisted of a control population (20) and a patient population (10 without surgery and 10 with). Data were processed to give maximum, minimum and ROMs of flexion/extension and abduction/adduction at all MCPs during four movements: pinch grip, key grip, fist clench and hand spread. Results showed ROM was decreased by ageing, further by RA, and further again by replacement surgery. MCP surgery patients produced significantly lower ROMs than all other groups, suggesting the implants may not restore movement.

ACKNOWLEDGEMENTS

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1. INTRODUCTION

The crippling joint disease of rheumatoid arthritis often affects the wrist and hand causing significant inflammation, deformity, pain, and loss of function. Treatment can involve arthrodesis, where articular cartilage and soft tissue are removed resulting in one solid bony mass. This procedure is successful in removing pain; however it causes loss of movement and, therefore, limits hand capabilities considerably. The other option is arthroplasty, where a replacement is implanted so movement and function are still possible.

However the success of these implants has been mixed and fracture rates have been reported anywhere from 0-82%. Goldfarb and Stern (2003) evaluated 208 arthroplasties, an average of 14 years postoperatively, 63% were broken, with an additional 22% deformed. Kay et al., (1978) report the highest fracture rate of 82% in Swanson prostheses followed for 5 years. Of 34 joint replacements, 17 were definitely fractured, with 11 probable cases. After fracture the implant may not support repetitive loading or movements so may not function as well and can cause further pain and swelling. Revision operations are possible but are an obvious unwanted complication and more difficult than the initial implantation. Therefore finger implants need to be improved to prevent fracture occurring so frequently or at least extend the life span of the prostheses.

Clues as to why implants are fracturing in such a manner could be provided by determining the movements that occur at the hand joints. It has been suggested that failure of arthroplasties may be due to twisting and turning forces at finger joints, experienced in everyday activities such as opening containers, getting dressed, grasping a pen and many more. Motion analysis enables the most accurate and complete analysis of movement, but current marker sets may be too simple and a more complex model may allow a more detailed understanding of the movement of finger and wrist joints. Furthermore limited detailed research using motion analysis currently exists on not only rheumatoid hands but also on normal hand movement.

Therefore the aim of this project is to accurately measure movement at the metacarpophalangeal (MCP) joint, the most commonly affected in RA, tAnd thereforehereby also attempting to gain a more detailed understanding of finger movement in both "normal" control subjects and arthritic patients. It is not realistic to attempt to give patients a range equivalent to non diseased hands and neither is it necessary. What needs to be determined is what functional range of movement is needed to improve the quality of life.

Understanding the movements hands are subjected to in everyday life more accurately and also investigating what degree of movement might be needed should help substantially when designing new prostheses.

The project will initially focus on determining if a new complex hand marker model is possible or necessary to understand hand movement further. This new marker system is intended for use when testing normal subjects in several simple hand movement tasks and to study the effect of ageing. The same marker set and tasks will then be used to test patients with rheumatoid arthritis and also those who have had MCP replacement surgery to investigate any differences between the movements possible. The main outcomes are therefore: (i) the creation of a new more accurate marker set and (ii) determining average range of hand movement in a normal population, those with rheumatoid arthritis and patients who have had replacement surgery.

2. BACKGROUND INFORMATION

2.1 Rheumatoid Arthritis

2.1.1 Introduction

Arthritis is a crippling joint disease, with unknown cause. It affects millions of people worldwide, causing sufferers extreme pain and loss of joint movement and function. With no cure available arthritis patients experience many difficulties; consequently quality of life can be affected considerably.

Rheumatoid arthritis (RA) is a chronic inflammatory disease, with the primary manifestation in the synovium and so can affect any synovial joint but most commonly the hands and feet (Grassi et al., 1998). Dramatic swelling and distortion of joints is observed with tenderness, pain and increased temperature at these locations (Lee &Weinblatt, 2001). These symptoms cause not only great discomfort but also loss of movement at joints, therefore restricting ability to perform everyday tasks and limiting quality of life. Loss of job can cause further problems, with a considerable percentage of sufferers becoming disabled and unable to work (Sokka, 2003). This work disability results in loss of income, and when coupled with the medical costs of the disease can lead to financial difficulty. Life span of those with RA is shortened from 3-18 years, depending on disease severity and age of onset (Alamanos &Drosos, 2005)

2.1.2 Prevalence

Rheumatoid arthritis affects between 0.5-1.0% of people worldwide (Silman &Pearson, 2002). However the occurrence of the disease ranges between different countries quite drastically (McCarty &Koopman, 1993). In the UK adult population in 2000 it was estimated that 386,600 cases existed (Symmons et al., 2002). RA prevalence increases with age (Lee &Weinblatt, 2001), with the peak onset occurring between 40-60 years of age. Interestingly in all populations and ages, women are reported to be 2-3 times more likely to develop RA (Symmons et al., 2002)

2.1.3 Etiology

The cause of RA is currently unknown. Many possibilities have been investigated, including occupational, geographical, metabolic, nutritional, genetic and psychosocial factors (Alamanos &Drosos, 2005). Current consensus is that RA is a multifactorial disease and due to an interaction between environmental and genetic factors. Other factors involved include ethnicity, the role of hormones (Hazes &Van Zeben, 1991) and smoking (Sagg et al., 1997). Genetic factors are among the most popular of possibilities, with first degree relatives and siblings of severe RA patients at a greater risk of developing the disease themselves (Deighton et al., 1992). Furthermore twin studies provide additional evidence, reporting that if one twin has RA a monozygotic twin has a 15.4% chance of developing the disease compared with only a 3.6% likelihood if the twin is dizygotic (Silman et al., 1993). Rheumatoid arthritis development is associated with the class II major histocompatibility complex (MHC), in particular, the human leukocyte antigen-D (HLA-D) region. Strong links have been continuously publicized with the HLA-DR4 epitope, (Olsen, 1988). Much research has been conducted to date on the role of genetics in RA, with the "shared epitope" theory a popular suggestion (Morel et al., 1990). It is clear from the research that there is a significant risk to individuals possessing certain gene epitopes or regions. The exact region or sequence is still being investigated and may still only be the cause in some cases or populations. Other possible causes need to still be considered.

2.1.4 Symptoms and classification

Symptoms of RA include pain and stiffness around the joint, often initially in only one joint but as the disease develops it begins to affect multiple joints (Rindfleisch &Muller, 2005). The body's immune system begins to attack the healthy joints leading to inflammation of joint linings and considerable swelling and pain. Fever, weight loss, fatigue and anaemia are also often found to accompany RA making the disease all the more debilitating (Hakim &Clune, 2002).

The criteria for classifying rheumatoid arthritis were revised in 1987 by The American Rheumatism Association (ARA) replacing the original criteria of 1958 (Arnett et al., 1988). RA is defined by the presence of 4 or more of the criteria in table 2.1. However there is at present no clinical test that can definitively confirm the presence of RA. The American College of Rheumatology Subcommittee on Rheumatoid Arthritis (ACRSRA) recommend

baseline measurements should be taken from patients to give clues that aid diagnosis (Arnett et al., 1988).

Table 2.1 ARA classification for Rheumatoid arthritis

1	Morning stiffness in and around joints (lasting at least one hour)	*
2	Soft tissue swelling (three or more joints)	*
3	Swelling of PIP, MCP or wrist joints	*
4	Symmetric swelling	*
5	Existence of rheumatoid nodules	
6	Presence of rheumatoid factor	
7	Radiographic changes showing erosions (particularly in hands and feet)	

* Criteria 1 - 4 need to have been present for a minimum of 6 weeks

2.1.5 Pathogenesis

The exact cause of RA is unknown, but it is has been suggested that a trigger is needed, usually autoimmune or infectious agents e.g. parvovirus, rubella, and others (Alamanos &Drosos, 2005). The early effects show synovial macrophage cell proliferation and microvascular damage, involving occlusion of blood vessels by small clots or inflammatory cells. As the disease progresses the synovium protrudes into the joint cavity as it grows. Proliferation and destruction continues and the inflamed synovial tissue grows irregularly, resulting in the formation of pannus tissue; a membrane that covers the normal surface of the articular cartilage. This pannus tissue invades cartilage and bone and begins to destroy them and the joint capsule (Rindfleisch &Muller, 2005, Lee &Weinblatt, 2001). Rheumatoid arthritis can affect all the synovial joints, but most commonly small joints of the hands and feet. Focusing on the hand, the wrist, metacarpophalangeal (MCP), distal interphalangeal (DIP) and proximal interphalangeal (PIP) joints as seen in Fig 2.1 can all be affected.

Fig 2.1 anatomy of the hand (Cerveri et al., 2003)

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RA often causes deformity at the MCP joints, commonly dorsal swelling may occur, and so stretch collateral ligaments. This causes the fibrocartilageinous plate to which the ligaments are attached to drops towards the palm. The flexor muscles in the hand then pull the proximal phalanx palmward too, this leads to volar sublaxation and ulnar deviation of the fingers, two common characteristics of RA hands, shown in Fig 2.2.

Fig 2.2 Ulnar deviation (Kirschenbaum et al., 1993)

RA can also affect the PIP and DIP joints of the hand. The PIP joints may become hyperextended in RA due to contracting of the interosseous and lumbrical tendons, this is sometimes termed the grasshopper deformity. When the PIP joints are in permanent flexion coupled with hyperextension of DIP joints it is termed boutonniere deformity (Fig 2.3).

Fig 2.3 Boutonniere deformity of left index finger. Dislocation and destruction of right index and middle finger MCP joints (Flatt 1961)

Damage to soft tissue and destroyed ligaments and tendons on one side of the hand may also cause Swan neck deformity, which is characterised by hyperextension at the PIP joint and flexion at the DIP joint, as seen in Fig 2.4. The fingers become twisted round to one side and patients are unable to pull them back.

Fig 2.4. Swan-neck deformity and destruction at PIP joints in both hands (Flatt 1961)

2.1.6 Treatment

There are no cures currently available for RA; treatment focuses on improving function, appearance and pain relief (Brooks, 2002). Management of the disease requires a multidisciplinary approach. Basic therapy when the patient is first diagnosed consists of patient education, physical therapy and rest (Strand, 1999). Pain relief is one of the main goals of treatment, there are several possibilities aimed at achieving this and also attempting to improve the quality of life of RA sufferers; both non surgical and surgical measures. Non surgical treatment includes using drugs, splints and steroids as well as acupuncture, occupational therapy, physiotherapy and anti- TNF therapy.

During initial stages of the disease aspirin, non steriodal anti-inflammatory drugs (NSAIDs) and corticosteroids injections are used as they have an immediate action and bring about the desired outcome of reducing pain and swelling. However there are several common adverse side effects (Rindfleisch &Muller, 2005). Disease modifying antirheumatic drugs (DMARDs) are offered to prevent or hopefully reduce further destruction of the joints. Common DMARDs include hydroxychloroquine (HCQ) and methotrexate. The main disadvantage of DMARDs is their effect is slow acting, (up to 6 months), with unpredictable effectiveness, and variability in duration (Hakim &Clune, 2002, McCarthy &Koopman, 1993).

Surgical measures are used in the more advanced stages of the disease, when non surgical methods were not successful or if the arthritis was not detected early enough. Early procedures are used for mild to moderate morphological and structural damage. Possibilities include synovectomy, tenosynovectomy, distal radioulnar joint synovectomy and tendon surgery (Burge, 2003). When the joint has almost or complete destruction then other procedures are necessary; either complete arthrodesis or arthroplasty. Arthrodesis involves articular cartilage and soft tissue removal resulting in one solid bony mass, with plates and intramedullary pins often used to maintain the position. This procedure is successful in removing pain but causes loss of movement at the joints therefore limits hand capabilities substantially. The other available option is arthroplasty, where an artificial replacement is implanted so pain is reduced, deformities are lessened but movement is also possible and improved. At the wrist joint arthrodesis is a popular option for RA patients (Burge, 2003). However in the finger joints fusing is not generally used as will cause extreme loss of function. Arthroplasty is a much more common treatment in more severe RA finger cases.

2.2 Finger arthroplasty

2.2.1 Introduction

Arthroplasty of the finger joint usually refers to MCP joint replacements; however DIP and PIP joint implants do exist (Trail, 2006). Most patients will be in later chronic stages of rheumatoid arthritis with surgery their last option. The prostheses are designed to relieve pain, restore functional range of movement (ROM), correct existing/prevent future deformity and improve cosmetic appearance (Beevers &Seedhom, 1995). Three basic designs have been developed so far; hinged, flexible and third generation prostheses.

2.2.2 Hinged

The earliest developed implants were all hinge designs composed of two or three metal components. Due to the design of these implants abduction and adduction movements are not possible. The first MCP joint prosthesis proposed was by Brannon and Klein in 1953. The implant (Fig 2.5) consists of two components joined together by a hinge joint, locked by a half threaded rivet screw. The hinge joint is finely bevelled to reduce irritation or abrasion of soft tissue during movement. Each section has an intramedullary stem inserted into the finger bones, these are triangular in shape to prevent rotation of the finger after insertion. Modifications from the initial design saw the introduction of staples through both stem and hub sections in an attempt to prevent sinking of the prosthesis into the phalanx when bone resorption occurs. All components are made from titanium, originally stainless steel. Results of the clinical trial (Brannon &Klein, 1959) are limited as only 2 implants were reviewed after 2 years, ROM ranged from 32.5-75 degrees, however this decreased greatly over the years and shortening of the finger also occurred. One of the prosthesis suffered bone resorption, sinking into the bone 10-12 months post surgery. Therefore although this initial prosthesis was not very successful it did pave the way for further implants and possibilities.
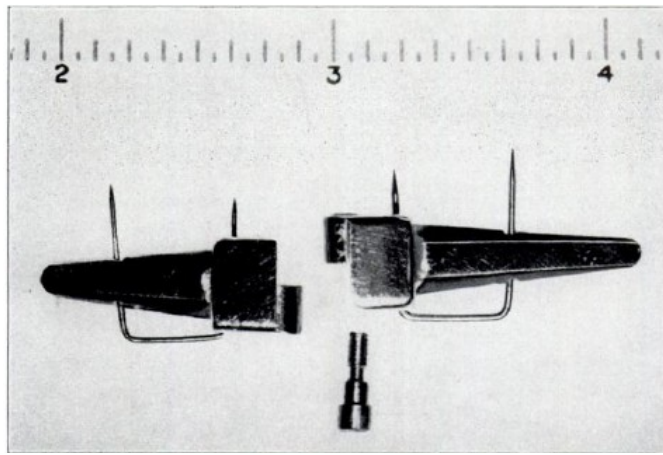
Fig. 2.5 The Brannon and Klein prosthesis (Brannon and Klein, 1959)

Consequently, the Flatt prosthesis was developed in 1961 (Fig 2.6) with three extra low carbon vacuum melt stainless steel components. There is a two pronged intramedullary stem to allow bone ingrowth and prevent rotation and sinking that was encountered with the Brannon and Klein prosthesis. A newer version developed a few years after incorporated a flexion-extension axis in a more volar position in relation to the plane of the stem aimed to provide better function. Four different sizes were available for the surgeon to pick the suitable size for each individual patient and the stems could be cut to shorten length

Fig. 2.6 Flatt metacarpophalangeal prosthesis in the right index and middle fingers. Five and a half months post operation, (Flatt, 1961).

Research reported the Flatt prosthesis gave a postoperative average range of motion of 24 degrees, which decreased at 5-14 years to 16 degrees (Flatt &Ellison, 1972). Although these average arcs of motion were decreased in each finger the arcs were in a more functional position. Furthermore the motion of the associated PIP joints not operated on tended to increase as a result of the reciprocal interaction between the joints. As a result Flatt and Ellison observed that hands could open to a greater extent and patients could perform a noticeably larger variety of functions compared to pre operative state.

However complications were reported; Blair et al., (1984b) reviewed 115 implants followed over an average of 54 months and state ulnar drift recurred in 43% and fracture in 21%. Further long term studies support these findings (Blair et al., 1984a). 41 Flatt arthroplasties were studied over an 11.5 year follow up, finding fractures in 47.7%, recurring ulnar drift in 57.5% and infection in 12.2%. Poor host bone tolerance was also shown, with 87% of radiographs showing a gap between the bone and the prosthesis, this will cause loosening of the implant and then migration down the metacarpals and proximal phalanges. Net bone resorption caused migration of the prosthesis, perforation of the metacarpal or proximal phalanx cortex in 44% and 59% of cases respectively. In addition, 50% of patients had fingers that did not rotate properly. Therefore these disadvantages led to development of other implants to reach higher success levels.

After the failure of the Brannon and Klein and Flatt prosthesis, second generation implants were developed. In 1973 the first of these, the Griffith –Nicolle implant was introduced. It has a roller and socket type design with two components. The roller component of the proximal phalanx is made from steel with the metacarpal cup component composed of polypropylene. A silicone rubber hemispherical capsule is attached to cover the hinge mechanism, attempting to minimise soft tissue irritation. Varma and Milward (1991) present clinical trial data on 101 implants after a follow up of 3.3 years on average, although fracture rate was very good (0%) recurrent ulnar deviation was the main persistent problem encountered, 27 degrees on average. In addition 4% of joints were removed due to infection.

Other second generation prostheses introduced include the Schetrumpf, Schultz, Steffee and St Georg-Buchholtz. All are ball and socket or roller and socket type designs, shown in Fig 2.7. However there are limited studies available (Schrumpf, 1975, Adams, 1990) and due to high fracture rates and limited success are often not used. The use of cement for fixation is believed to be the reason for the high fracture rates, as it causes higher loading on the joint mechanism and the prosthesis is not strong enough to transmit the forces caused by the flexor tendons. Therefore these prostheses are discounted also due to high fracture rates.

Schultz   Steffee   St. Georg-Buchholtz
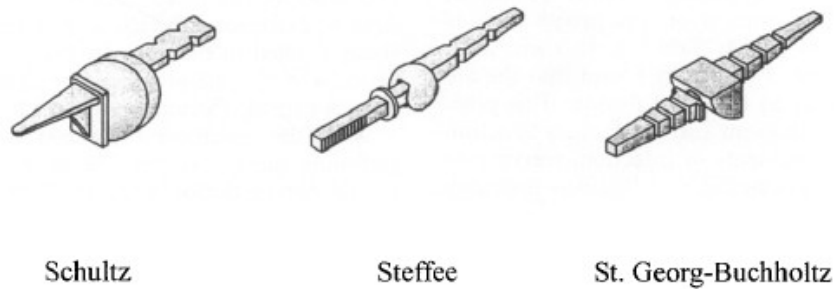
Fig. 2.7 The Scultz, Steffee and St Georg-Buchholtz implants, from Beevers &Seedhom (1995)

In addition some ceramic implants were also developed, the first being the KY Alumina ceramic prosthesis, followed by the Minami alumina ceramic implant. Both had metacarpal stems of polycrystal alumina with proximal phalanx stems composed of single crystal alumina and a bearing component of high density polyethylene. Results from Minami et al., (1988) revealed that ROM was too small for functionality, with extension limited on average at all joint by 18 degrees. Therefore ceramic implant design has been abandoned and focus has remained on other possibilities.

2.2.3 Flexible

Following limited success of the metallic hinge joint implants and the ceramic attempts, flexible silicone prostheses became popular as they provided more movement. The first model was developed by Swanson (1962) a flexible, heat-molded joint implant made of silicone rubber called "Flexspan", shown in Fig 2.8. Fixation was achieved by the concept of encapsulation; the prosthesis itself acts as an internal mold that maintains the correct joint alignment. The prosthesis is surrounded by a fibrous capsule that adapts and changes orientation due to motion immediately postoperatively. This method of fixation allows the stems to move up and down the bone canals as they are not fixed to the bone. Furthermore the gliding principle spreads the stresses over a larger area of the implant inflicting less stress on surrounding bone. Gliding is also aimed at giving an increased ROM and was intended to increase the life span. However this sliding movement can cause erosion and therefore loosening of the implant. There are many studies reporting the success and complications of Swanson implants over a range of follow up periods. These are summarised in Table 2.2. The main problem with the Swanson is the fracture rates, although these vary greatly with different studies.

Table 2.2. Comparisons of the complications and successes of hand joint arthroplasties

Study	type of implant	no. of implants	Av. follow up time	fracture rate	infection rate	revision rate	method of assessment	ulnar drift	ROM (post op)	ROM (pre op)
Kirschenbaum (1993)	Swanson	144	102 mnths	10%	1%	2%	radiographs		16-59	
Swanson (1972) –Grand Rapids	Swanson	220	2-5 yrs	3.10%	0.60%		radiographs	3.2	2.5-64	
Swanson (1972) - Field clinic	Swanson	3409	5 yrs	0.88%	0.70%		questionnaire	1.9	4.0-57	
Mannerfelt &Andersson (1975)	Swanson	144	2.5 yrs	2.80%	0.70%		radiographs		9.0-49 (40)	35
Ferlic et al., (1975)	Swanson	162	38 mnths	9%	1%	1.80%	radiographs	8.9		
Beckenbaugh et al., (1976)	Swanson/ Niebauer	186/16	32 mnths	26.2/ 38.2%		2.40%	clinical & radiographs	11.3	10.0-48	
Bieber et al., (1986)	Swanson	210	5.25 yrs	0%			radiographs		22-61	
Blair et al., (1984)	Swanson	115	54 mnths	21%	3%		radiographs	43%	13-56 (43)	60-86 (26)
Blair et al., (1984)	Flatt	41	138 mnths	47%			radiographs	58%	16-40 (24)	
Goldfarb &Stern(2003)	?	208	14 yrs	63%			radiographs		46	30
Vahvanen &Viljakka (1986)	Swanson	107	45 mnths	4%				31%	7-41 (34)	
Hansraj (1997)	Swanson	170	5.2 yrs	7%	0.00%	6.40%	radiographs		27	38
Wilson (1993)	Swanson	375	9.5 yrs	17%	1%	3%	radiographs &questionnaire	43%	21-50	
Schmidt (1999)	Swanson	151	3.9 yrs	9%			radiographs			
Gellman (1997)	Swanson	901	8 yrs	14%	3%				10-60 (50)	40
Flatt (1972)	Flatt	242	15 yrs	2%	0.80%	10.70%	radiographs		15-31 (16)	47-71 (24)
Delaney et al., (2005)	Neuflex/ Swanson	40/37	2 yrs	0%	0%	0%	radiographs		16-72 (56)/ 19-59 (40)	47-79 (32)/ 51-80 (29)
Kay et al., (1978)	Swanson	34	5 yrs	50%+32%prob			radiographs			
Joyce et al., (2003)	Sutter	41	42 mnths	27%			radiographs then removal			
Radmer et al., (2003)	WEKO	28	15 mnths		0%	100%	radiographs		22-35 (30)	15-40 (30)
Minami et al., (1988)	ceramic	82	38mnths	0%			radiographs		18-48 (30)	
Varma &Milward (1991)	Nicolle	101	10yrs	0%	4%	4%			30%	
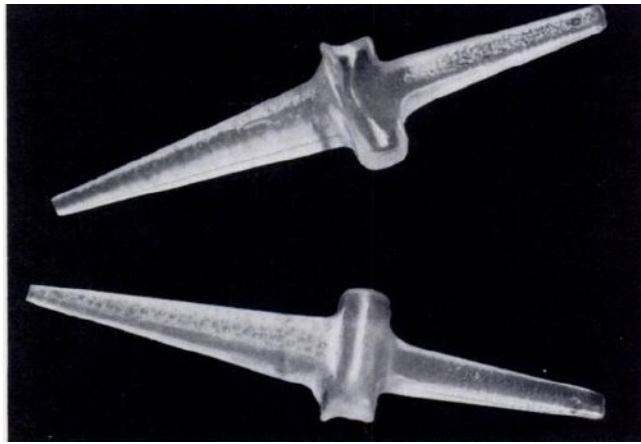
Fig 2.8 Swanson implant (Swanson 1972)

Another silicone implant, the Neibauer first used in 1966, was reinforced both internally and externally with Dacron, for strength and fixation respectively. However these two materials differ in mechanical properties and so results in stress at the interface between the two and so the softer material inevitably deforms. Both Hagert (1975) and Beckenbaugh (1976) report relatively high fracture rates, 53.7% and 38.2% respectively, suggesting the prosthesis is not strong enough to withstand the forces it is subjected to.

The Sutter metacarpal prosthesis was designed to be an improvement on the Swanson implant. Designed in 1987, it comes in seven different sizes to fit different fingers. The Sutter is made from a material called "Silflex", claimed to give greater range of movement than the Swanson. The centre of flexion is palmar to the implant's longitudinal axis, suggested to make extension easier. Joyce et al., (2003) reviewed 41 implanted Sutter prostheses, twelve were removed after an average of 42 months post surgery. Of these removed, eleven had fractured, ten completely (shown in Fig 2.9). These ten fractures all occurred at the junction between the distal stem and the hinge region, the same area that Swanson implants are known to fracture.

Fig 2.9 Fractured Sutter prostheses (Joyce et al., 2003)

Joyce et al., (2003) also conducted simulator tests on two Sutter prostheses. One completed just over 10 million cycles of flexion-extension, the other 5.3 million. Both fractured in the same place as those removed from patients, this is also the same region found to be the location of fracture in the Swanson implants. Of the retrieved implants many had a rectangular shaped fracture face, suggesting that the silico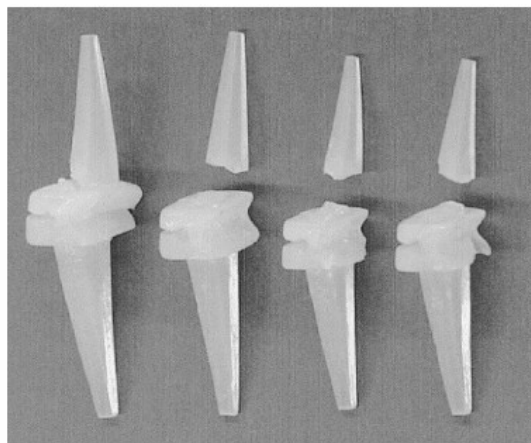ne had torn along the small radii at the junction between the stems and central hinge. This lead to the proposal that as the prosthesis is made of silicone it will be bending not only at the hinge but at the stem as well, and as these have a small cross sectional area and can not withstand the forces, the majority of fractures occur here.

Other flexible implants include the Helal, with a dorsal-ulnar flap attempted to overcome ulnar drift, and the Calnan-Reis prosthesis; a single polyethylene component fixed by cement (Calnan &Reis 1968). Neither showed outstanding results, with the Swanson implant still deemed superior.

2.2.4 Third generation

Third generation implants developed more recently are so called "total" implants, compromising several components. These include the Kessler (1974), Hagert (1986), Beckenbaugh (1983) and Ludborg (1993) implants shown in Fig 2.10 (Beevers &Seedhom, 1995) all made from different materials.
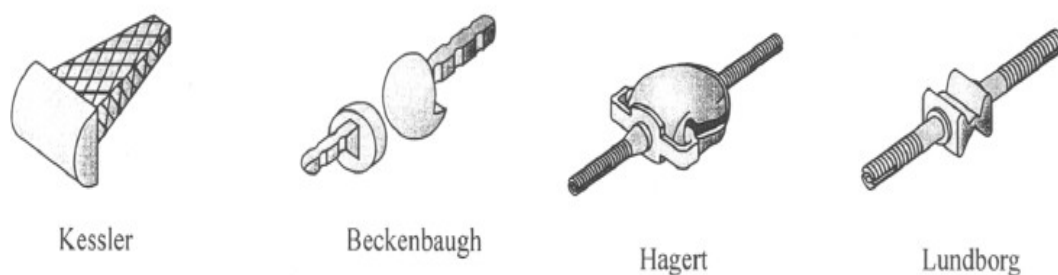
Fig 2.10 Third generation implants (Beevers &Seedhom, 1995)

With all these implants longer follow up studies are needed to give a better understanding of the success and possible complications that may occur. These implants are not suitable for severe RA patients with bone erosions and considerable deformity as ligaments and muscles are needed for stability of the implant. The Swanson implant remains the most commonly used and preferred, due to the ease of implanting and also removal if necessary and also the low cost of the prosthesis (Beevers &Seedhom, 1995)

The Neuflex is the newest prosthesis on the market, developed in 1998 by DePuy. Its major design feature is the 30 degree neutral angle, intended to replicate the hands natural resting position, therefore supposedly reducing stresses on the implant and in particular the central hinge region. Furthermore manufacturers state that it will optimise comfort and require less force to flex the fingers. It is a single piece silicone prosthesis, of Anasil silicone and seven possible sizes have been made to suit all individuals. Delaney et al., (2005) compare 10 Swanson and 12 Neuflex implants in a random allocation study after 2 years post surgery. Although there were no observed fractures, silicone synovotis or infection reported, they found that the Neuflex had a 13 degree greater flexion range than the Swanson. However, they discovered no differences between function, grip strength or ulnar deviation recurrence. Joyce and Unsworth (2005) tested the Neuflex in vitro using a single station simulator. Testing 3 size 30 implants they found them capable of 9.4, 10.3 and 19.9 million flexion-extension cycles before fracture occurred. All three fractured along the pivot of the central hinge region. This compares to the Sutter that fractured at just over 10 million and 5.3 million cycles (Joyce et al., 2003) and the Swanson that reportedly survived 400 million cycles with no problems (Swanson, 1972).

2.2.5 Complications

As highlighted above, success of the implants has been mixed and some reported revision rates are quite high. Data varies greatly, and fracture rates have been reported anywhere from 0 up to 82%. A summary of the different findings is shown in table 2.2. Goldfarb and Stern (2003) evaluated 208 arthroplasties, an average of fourteen years postoperatively. Of these, 63% were broken, with an additional 22% deformed at the time of final follow-up. Kay et al., (1978) report the highest fracture rate of 82% in Swanson prostheses followed for 5years. Out of 34 joint replacements, 17 were definitely fractured, with 11 probable cases. The most frequent facture location was the base of the distal stem.

Patients may not be aware when their prosthesis has fractured as it often does not cause pain and range of motion may not be greatly affected either (Beckenbaugh, 1976, Kay, 1978, Kirschenbaum, 1993). Therefore this could be one reason why reported fracture rates differ and true rates may in fact be even larger. A further reason for the variation in reported rates may be the methods of assessment; clinical assessment is unlikely to detect fracture, and even radiographs are difficult to interpret fractures and may miss some. The only definitive way to determine implant fracture is to remove and then carefully study it.

It is clear in many cases that the implant may have broken but if it is not causing any pain and is still providing functionality then it would not be appropriate to remove as would subject the patient to further unnecessary surgery and pain. Out of twelve removed Sutter prostheses eleven were fractured after 42 months (Joyce et al., 2003). However, after fracture the implant may be unable to support repetitive loading patterns that are experienced during every day activities (Fowler &Nicol, 2002).

Joyce et al., (2003) suggest an alternative explanation for fracture; based on the nature of loading in MCP joints of rheumatoid patients, where subluxing forces often dominate. This can lead to the cortical bone of the proximal phalanx to rub on the distal stem of the prosthesis. Any small abrasion may result in production of a stress concentration, followed quickly by fatigue failure at the junction between the distal stem and hinge of the implant. This theory is supported by their findings on the Sutter implant and also the Swanson.

A further problem that can occur with silicone implants is silicone synovitis (shown in Fig 2.11). This is caused by repeated rubbing of the implant against bony or sharp surfaces leading to silicone wear particles inducing an immune response, causing release of multinucleated giant cells and synovial hypertrophy (Lanzetta et al., 1994). Characteristic radiological changes including the development of cysts in adjacent bones may occur without symptoms, whereas others will encounter pain, joint stiffness, loss of motion and swelling of soft tissue (Khoo et al., 2004). To reduce this problem titanium (Ti) grommets were introduced to prevent abrasion of the silicone. These are additional titanium sleeves which are fixed to the implants to reduce wear of the silicone from sharp bone surfaces. Grommets have been shown to decrease fracture and osteolysis (Schmidt, 1999) with grommets 0% of prostheses fractured, compared to without the grommets where a fracture rate of 15% was observed. However Ti grommets may also result in further problems as then the titanium is worn and debris also causes inflammatory responses (Khoo et al., 2004).
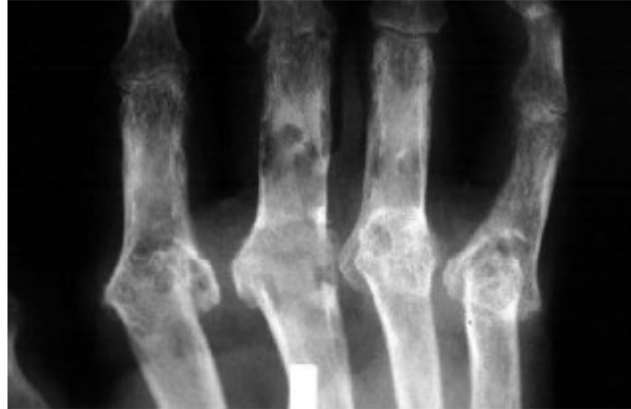
Fig. 2.11 Silicone synovitis (from Trail, 2006)

A further reported complication of implants is infection, which is much rarer; with some reported rates shown in table 2.2. Further problems include skin necrosis immediately post surgery due to the thin nature of arthritic skin and treatment of steroids which can contribute to poor healing. Dislocation of stems has also been noted, with swelling of the palm observed. Another issue to consider is recurrent ulnar drift, Trail (2006) suggests this is in fact inevitable, rates of ulnar drift are also shown in table 2.2.

It is currently unknown why fracture occurs so frequently. It has been suggested that turning and rotation at the wrist joint can cause wrist implants to become damaged after repeated twisting which they are not designed for (Palmer et al., 1985). It may be that the same applies at the finger joints which are assumed to only use two planes of movement but may in fact need to allow for rotation also. The movement analysis needs to be reviewed in order to determine what range of movement occurs at these joints and furthermore what range of movement is needed for arthritic patients. It is not realistic to attempt to give them a range equivalent to non diseased hands and neither is it necessary. As has been suggested in wrist implants, designs should focus on a more limited, applicable range of motion, rather than attempting to restore a complete normal range (Shepherd, 2002). What needs to be determined is what functional range of movement is needed to improve the quality of life. Therefore in order to design a better implant with a lower fracture rate the movement at the finger joints needs to be examined in greater detail.

2.3 Material properties of silicone

2.3.1 Introduction

The materials from which finger implants are made could provide further clues as to why these devices are fracturing with relative frequency. Silicones will be discussed in this chapter as they remain the most common material used as mentioned in the previous chapter.

2.3.2 Structure

Silicones are all composed primarily of molecules containing a backbone of alternate silicon and oxygen atoms with some organic side groups, most commonly the methyl group when it is known as poly(dimethyl siloxane) (PDMS), the structure for which can be seen in Fig 2.12. However different organic side groups are also found (Lambert, 2006). Silicone polymers can be transformed into elastomers by cross linking reactions forming chemical bonds between adjacent chains (Colas & Curtis, 2005).
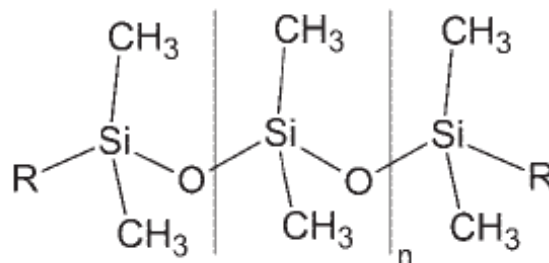
Fig 2.12 Basic structure of PDMS (Lambert, 2006)

2.3.3 Properties

There are many properties of silicones that make them an excellent choice for use as an implant. Not least of all their biocompatibility, with low toxicity and non reactive nature the silicone implants are generally well tolerated by the human body and will not cause any harm or unwanted response. It is silicone's semi-inorganic structure that allows it to be placed in the body without being absorbed and also means the mechanical properties will not be affected (Yoda, 1998) again of great importance for use as an implant.

The flexible, elastic properties of silicones allow movement of the arthroplasty. Due to the low glass transition temperature (Tg) as a result of low intermolecular interactions (Colas &Curtis, 2005), silicone implants will be rubbery at body temperature and not will not experience any temperature that will degrade them or effect their physical properties.

2.3.4 Failure

Initiation of fractures in silicone prostheses can be caused by several possibilities; the first being accidental scratching during implantation of the arthroplasty as a result of the surgical technique (Hutchinson et al., 1997), any nick can then act as an initiation site for cracks. After studying the surgical technique Weightman et al., (1972) suggested that poor surgical technique could create a step off point and therefore increase the stress in the bending element of the device enough to cause fracture. Sharp edges of the bone may also rub against the silicone implant, especially during flexion due to subluxing forces of the rheumatoid hand (Joyce et al., 2003) again causing a crack initiation site. It has also been suggested that the cross links may not be uniform throughout the silicone and these local inhomogenities can then act as microvoid initiation sites (Kinloch &Young, 1988). Once created by any of these possibilities, these crack initiation sites will then grow under certain conditions; primarily repeated dynamic loading (Kinloch &Young, 1988) such as with use in the finger joint. Once an initiation site has been introduced, it has been shown that even under low strains of 10% crack growth rate can be 2.5×10^{-5} mm/cycle in medical grade silicones when tested using pure shear tests. During flexion strain is believed to be much greater meaning crack growth will be even quicker (Leslie et al., 2008).

Failure may also occur as a result of the environment into which the implant is placed so that over time its mechanical and physical properties are altered and it will not function as initially intended. Many experiments have been carried out to investigate different environmental conditions. Swanson and Lebeau (1974) implanted silicone rubber specimens in beagles then removed and studied the physical properties. After 2 years the tensile strength decreased by 8%, elongation by 15%, and the elastic modulus increased by 16% showing how the implants performance could be reduced over time and how it could be more susceptible to fracture. Leslie et al., (2007) found placing samples of medical grade silicones in mild environmental conditions at body temperature caused true stress at failure to be reduced over time, showing reduced strength. With an elevated temperature this effect was even greater; suggesting the mechanism that reduces strength could be thermally activated.

It is however still unclear exactly how the material changes. Evidence from fourier transform infra-red (FTIR) spectroscopy and gel permeation chromatography (GPC) did not support the proposed theory that continued cross-linking affects properties. However, Leslie et al., (2008) did find other support for the assumption; suggesting that the changing absorbencies found with FTIR analysis may be indicating competing processes are taking place, possibly continued cross-linking is occurring but alongside oxidation. Support for this comes from findings of more pronounced changes in properties of the samples aged in air compared to distilled water and Ringer's solution. However it has also been shown that cyclic testing in vitro at 37 degrees did not cause finger implants to fracture after 10 million cycles (Weightman et al., 1972). Although discoloration of the prostheses was seen at the point of bending, suggesting continued stress concentration could lead to fracture eventually.

A further problem and possible source of failure comes from silicones lipophilic nature so they can be swollen by lipids absorbed from the body. Swanson and Lebeau (1974) reported maximum weight gain over the two years after silicone implantation was 0.91%, and was due mainly to lipid absorption. However lipid and fatty acid absorption was found to be much lower in finger implants and furthermore was not related to duration of implantation, failure or cracks observed after removal (Meester &Swanson, 1972). Lipid absorption was also noted by Weightman et al., (1972) with significant amounts of triglycerides and cholesterol found on fractured prosthesis, but they suggest that if inserted properly the implant should be successful despite this.

To conclude, it appears that the properties of silicones have an important role in the success of finger implants. The main problem seems to be the fast rate of crack propagation once a small initiation site has been created. Reducing the chance of such a crack from being introduced seems to be of key importance, this can be achieved by careful surgery, both when using sharp implements but also in ensuring no jagged bone edges are left. The continued rubbing of bone on the implant may be unavoidable due to the subluxing nature of the rheumatoid hand, in which case the implant material needs to be improved to withstand such impingements. It is important to consider the materials properties and behaviours in conjunction with the information about the forces and movements that prostheses are subjected to once implanted.

2.4 Methods to assess hand movement

2.4.1 Introduction

A better understanding and more detailed information of hand movement is needed to provide some clues as to why finger implant fracture rates are so high and possibly even how they could be reduced. This includes more accurate angle measurements and in depth data on movement patterns. There are a variety of different methods available to measure the movement at joints, ranging from the very basic, such as visual estimation and composite finger flexion, which are less reliable (Ellis &Bruton, 2000), to much more complex options such as goniometry and motion analysis.

2.4.2 Goniometery

The goniometer is an extremely useful tool to measure range of movement (shown in Fig 2.13). It is quick and easy, lending itself well to use in large clinical studies. Reliability of the goniometer is relatively high (Ellis &Bruton, 2000), making it more effective than basic measurements. However the reliability is dependent on the tester; factors such as experience and technique can affect angles recorded. If measured by different testers, joint angles at the hand can vary by ±7–9 degrees, compared to ±4–5 degrees if the same person is taking measurements (Ellis &Bruton, 2000). However, goniometers do not provide very accurate data, and give limited information about how different joints move to perform everyday tasks or activities. These and other more comprehensive details would be necessary to understand the specifics for implant design. Goniometry also only tests one joint of one finger at a time, in a fixed position, therefore not giving active ROM and, in order to calculate an average value for each joint of the hand, considerable time would be required. Another disadvantage is the examiners could influence angles achieved by forcing movements that would not be performed in everyday tasks so would not accurately represent the true nature of natural movement. The main limitation of using a goniometer to investigate hand movement in diseased hands is that they are only able to measure in 2-D and therefore errors would arise from the disfigured joints and data would not be representing the movement accurately.
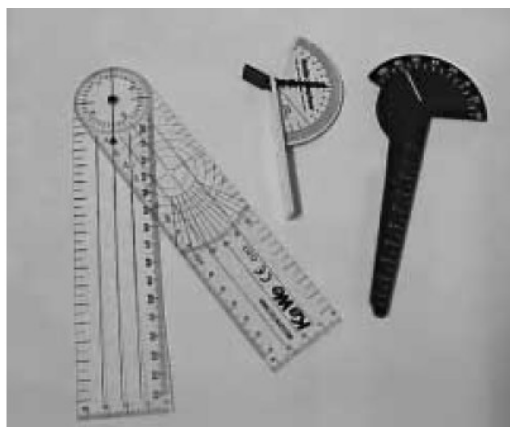
Fig 2.13 several different goniometers (Stam et al., 2006)

2.4.3 Gloves

The use of gloves has also been put forward as a measuring tool for hand movement. One example, the CyberGloveTM (Virtual Technologies Inc, 1992), has a mean error less than 6 degrees for all flexion and abduction angles (Kessler et al., 1995). However, error ranged from 0.3 degrees at the middle finger to 5.5 degrees at the index finger MCP joint (Yun et al., 2002). The SIGMA (Sheffield Instrumented Glove for Manual Assessment) glove, shown in Fig 2.14, has also been developed (Williams et al., 2000). Error for finger flexion was found to fall between ±5 degrees, again comparable to goniometry. Along with problems in accuracy at different joints, the glove has several other disadvantages, mainly that the sizes of gloves will not fit every hand in the same way and therefore one can not guarantee that the fibres of the gloves are accurately placed over the anatomical landmarks required. This would be even more apparent in diseased or injured patients, where the glove is very unlikely to fit inflamed or deformed hands and could cause considerable pain if forced. As no single deformity is the same, it is unlikely this problem could be overcome or standardised. In addition the glove may in fact restrict normal movement.
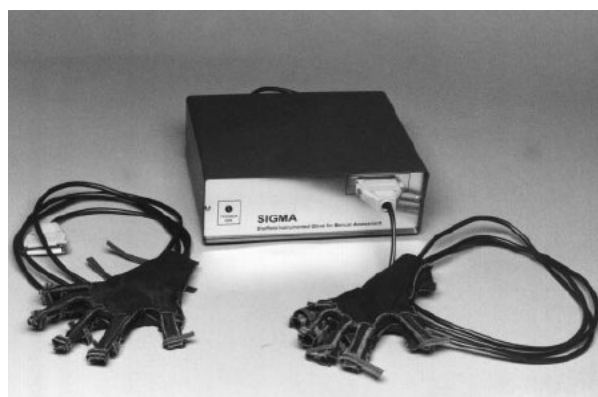
Fig 2.14 SIGMA glove and interface box (Williams et al., 2000)

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2.4.4 Three dimensional motion analysis

Three dimensional motion analysis is a further, more complex method available for measuring hand movement. The system utilises high resolution cameras with LED strobe lights around the lens. Subjects wear retro-reflective markers placed in pre-defined landmarks and as they move in the capture volume light is reflected back into the camera lens, strikes a light sensitive plate within and so creates a video signal. Motion analysis can therefore capture the active ranges of motion (AROM) of hand joints so recording changes in angles at all three finger joints continuously during movement of the finger. Rash et al., (1999) showed markers placed on the dorsal aspect of the hand and fingers can be used to accurately measure joint angles using motion analysis. Therefore, motion analysis presents a major advantage in its ability to provide more information than conventional goniometer measurements as it demonstrates the dynamic changes in the finger joints during motion. This method also produces much more information about movement at the individual joint, it allows angles to be measured in more than one plane, so can investigate flexion, extension, adduction, abduction and rotation all at the same time. Chiu et al., (1998) have shown it is possible to measure the angles of finger joints during motion analysis evaluation by adding more reflective markers and the data derived are comparable to the measurements obtained with a conventional goniometer.

However 3-D motion capture still has disadvantages; it can be considerably time consuming, because accurate placing of markers, one-by-one, is slow. The main disadvantage is that during movement muscle deformations and skin sliding will inevitably occur, particularly with older skin. The severity of this problem depends on where the markers are placed and will be discussed with the relevant marker sets.

Despite some disadvantages, motion analysis still remains the most accurate method to assess joint movement in the hand, although time consuming the benefits in terms of accuracy and information captured, far outweigh this.

2.4.5 Marker sets

Current marker systems used for motion analysis often place only a single marker on each phalanx which does not accurately define a segment. Three markers per segment are required to provide data on rotation at a joint. There is however no standardised set of marker positions, although suggestions have been made by the International Society of Biomechanics (Wu et al., 2005). There are several different approaches that have been taken by different research teams.

Simple marker systems have been proposed by Cerveri et al., (2007), shown in Fig 2.15 and also Carpinella et al., (2006). Markers are placed directly over the joint centres and on the finger tips on the distal border of the nail. This marker set may provide much quicker testing durations as less markers have to be attached; therefore would be very useful for clinical research on a large scale. Using fewer markers could also be of benefit when testing hand motion in children where there is not a large enough surface area to place more markers. However having so few markers prevents complex or accurate information from being obtained. The main disadvantage with this system is placing the markers directly over the joints where skin movement will be greatest. This causes markers to move non-rigidly with respect to the underlying bones, the markers will then no longer correspond to their pre-determined locations. Therefore this marker set will produce angle data that does not accurately represent the movement of the joints. Consequently, other marker sets have been proposed to improve the accuracy of measurements and limit the effect of skin movement by placing markers in alternative positions.

Fig 2.15 Ceveri et al., (2007) simple marker set

Chiu et al., (1998) and Su et al., (2005) both place two markers on each phalanx, shown in Fig 2.16, except at the distal phalanx were a single marker is used. This means when calculating the angle measurements at the PIP and DIP joints accuracy will be compromised. Su et al., (2005) report an accuracy of up to 0.1% in position and 0.2 degrees in angle measurement. The only issue with these more complex marker systems is the increased assessment time; both the accurate placing and the analysis of more markers creates a more time consuming process.
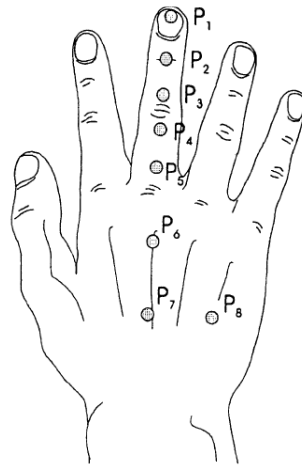
Fig 2.16 marker set, Chiu et al., (1998)

Floating marker clusters have also been used (Fowler &Nicol, 1999, 2001, 2002 and Degeorges et al., 2005). They consist of three carbon fibre pins protruding from a base forming a triad arrangement, with markers attached to the ends as shown in Fig 2.17. This method allows more markers to be used to gain information about the joint, without concern about fitting enough markers on each segment. However if floating clusters were used for every finger there would be too many markers for such a small capture volume. Markers could knock each other during movement and occlude others from the cameras. Furthermore these types of markers may not be appropriate when testing RA patients as severe swelling and deformities could cause one cluster to protrude onto another if placed on every finger, and large clusters may not be suitable for children with smaller hands either. However, unlike the other marker systems, floating clusters can give information about rotation at the joints
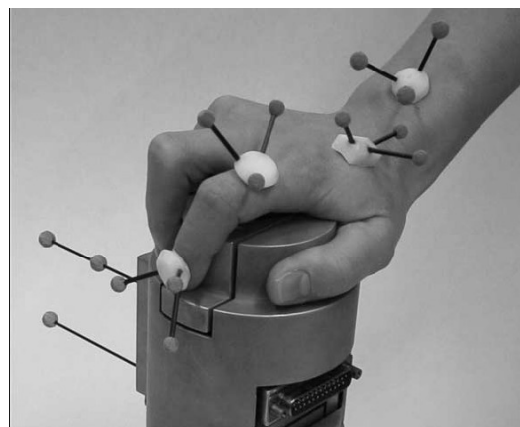
Fig 2.17 Floating markers, Fowler & Nicol (2001)

To conclude, although the more complex marker sets (Chiu et al., 1998, Su et al., 2005) with two markers on each phalanx give more accurate data on flexion/extension and abduction/adduction, they are still lacking two markers on the distal phalanx. Certainly adding extra markers to the finger tips needs to be tested to see if it is achievable in such a small volume. The current marker sets, with exception of the floating clusters, are also unable to provide rotational data. For rotation to be measured more markers need to be added to the fingers, which may not be possible as the error of the movement may be too great for the small degree of rotation actually occurring at the joints, but this possibility needs to be tested also.

The use of motion analysis on rheumatoid hands is also limited, with the only study to my knowledge conducted by Fowler and Nicol (2001), using their floating markers on eight RA patients and eight controls and then repeated with eight post MCP replacement patients (2002). However as discussed this marker set may cause problems when assessing the whole hand and using another marker set may give more accurate results.

3. EXPERIMENTAL METHODS

3.1 Ethical considerations

As the study involved using healthy volunteers and NHS patients strict ethical guidelines had to be followed. Ethical approval was granted by the University of Worcester for use of the staff and students as participants. The NHS ethical application process involved a 34 page document completed on line (Appendix 1) and then sent to the Warwickshire Research Ethical Committee to review. After attending a committee hearing, and making small changes to the patient information and consent forms, the study was granted favourable ethical approval (Appendix 2). Approval was then also given by the local R and D department.

3.2 Subjects

Four experimental groups were used, each consisting of ten subjects. Two control groups of young adults (age 23 ± 3.6years) and older adults (age 56 ± 7.4years) were recruited from the University environment. Both control groups went through screening to ensure they showed no symptoms of hand disease or previous injury/surgery that would affect joint movement. The screening questionnaire (Appendix 3) was completed by participants after reading the participant information sheet (Appendix 4) and giving informed consent (Appendix 5) before being tested.

Two patient groups were used, both suffering from rheumatoid arthritis. One group (age 60± 9.2years) consisted of stable rheumatoid arthritis patients with no history of surgery and the other group (age 67± 12.8years) had Swanson Metacarpophalangeal (MCP) arthroplasty in all four MCP joints, at least two years previously. Patients were excluded if they had any other surgery on the hand or if the implant showed signs of fracture determined by radiographs. Patients were also excluded if they had a current acute flare up. All patients were currently attending routine out-patients clinics. Suitable subjects received an invitation letter (Appendix 6) to ask them to participate. Any patients who indicated their interest were contacted via telephone and sent further information (Appendix 7). Those who agreed to participate in the study were then asked to give informed consent (Appendix 8) and a letter sent to inform their GP (Appendix 9). Patients were not given questionnaires or asked any specific questions during testing, but often they were keen to discuss their disease or their finger replacements.

All subjects were right hand dominant and only right hands were studied. All participants used were female. Subject characteristics are shown in tables 3.1a and b. All testing took place at the Motion Analysis Research and Rehabilitation Centre (MARRC), University of Worcester.

Clinical data collected included a recently taken Disease Assessment Questionnaire (DAS) (Appendix 10) and blood tests from a maximum of two weeks prior to testing to ensure validity. X-rays of patient's hands were also available for review.

Table 3.1a Subject characteristics of control subjects

Subject	Age	DOB		Subject	Age	DOB
YN01	21	23/05/1986		EN01	61	04/02/1947
YN02	30	07/04/1977		EN02	51	20/01/1957
YN03	27	28/10/1981		EN03	53	05/03/1955
YN04	21	28/09/1986		EN04	63	23/11/1945
YN05	21	15/09/1986		EN05	41	23/02/1967
YN07	21	23/02/1987		EN06	64	28/08/1944
YN08	22	27/11/1985		EN07	62	11/07/1946
YN09	22			EN08	58	19/09/1950
YN10	25	12/02/1983		EN09	61	20/09/1947
YN11	29	21/10/1979		EN10	50	26/09/1958
Average	23.9				56.4	
SD	3.57				7.43	

Table 3.1b Subject characteristics of patient subjects

Subje	Subject	Age	DOB	DAS score		Subject	Age	DOB	DAS score	Yrs post OP
	RA01	49	26/10/1959	4.11		MCP1	48	01/05/1960	3.72	4.90
	RA02	61	01/08/1947	2.55		MCP2	76	26/01/1932	4.77	4.50
	RA03	65	21/01/1943	4.32		MCP3	74	11/11/1933	2.09	2.60
	RA04	71	31/05/1937	3.84		MCP5	71	31/01/1937	2.09	4.30
	RA05	61	05/09/1947	4.1		MCP6	78	07/12/1929	4.21	3.60
	RA06	40	14/01/1968	3.79		MCP7	88	06/01/1920	3.04	3.80
	RA07	68	07/02/1940	3		MCP8	58	26/10/1950	2.78	4.80
	RA08	61	05/02/1947	3.47		MCP9	71	01/02/1937	4.03	5.40
	RA09	63	28/06/1945	3.53		MCP10	59	23/02/1949	1.64	3.50
	RA10	64	25/09/1944	4.18		MCP12	51	17/11/1957	1.69	1.90
	Average	60.3		3.81			67.4		3.01	3.93
	SD	9.17		0.45			12.84		1.13	1.03

3.3 Motion analysis

A Vicon M2 (624) (Vicon Peak, Oxford, UK) motion capture system was used to capture hand kinematic data. Twelve Vicon cameras and one video camera were positioned in an arc around the working volume at varying heights, exact camera set up is shown in Figs 3.1 and 3.3. This camera set up was developed during pilot testing, using trial and error over several months. Many of the various factors involved were constantly changed and many different combinations trialled. These included the distance from the cameras to the hand, the height, position and number of cameras, and also the degree of the arc that the cameras made around the hand. Once a capture of good enough quality was achieved the exact set up was recorded. Cameras have a ring of LED strobe lights fixed around the lens so that they can be adjusted. The chair was placed in the centre of the arc, with the right front leg placed over a marker on the floor to standardise placement. This marker is also used as the central point to position the cameras, using the distances in table 3.2.

Fig 3.1 Camera set up

Static and dynamic calibrations were performed using specially designed smaller calibration frames (Fig 3.2), using 9.5mm reflective markers. The static triangular frame used four markers; the first in the corner defining zero, with the others 50mm in the y direction, and 86 and 46mm in the x direction from this point. Static calibration involves placing the frame in the centre of the camera arc to allow calculation of the centre of capture volume and determination of the orientation of 3D workspace. The dynamic frame, shaped like a T-bar used three markers, one on the far end, the other two positioned 35 and 87.5mm along the bar.

Dynamic calibration is performed by waving the wand within the capture volume to allow the system to calculate the positions and orientations relative to one another. Calibration residuals of 0.6mm or less were achieved each time with sampling carried out at 60 Hz.
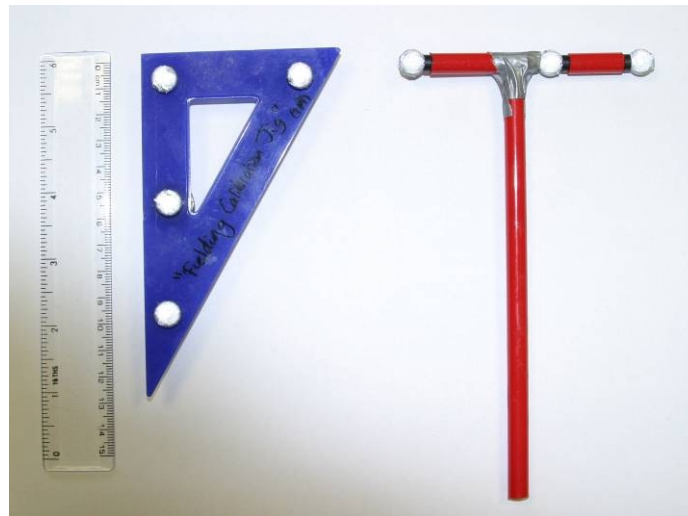
Fig 3.2 Small static and dynamic calibration frames

'Centre' point for hand position
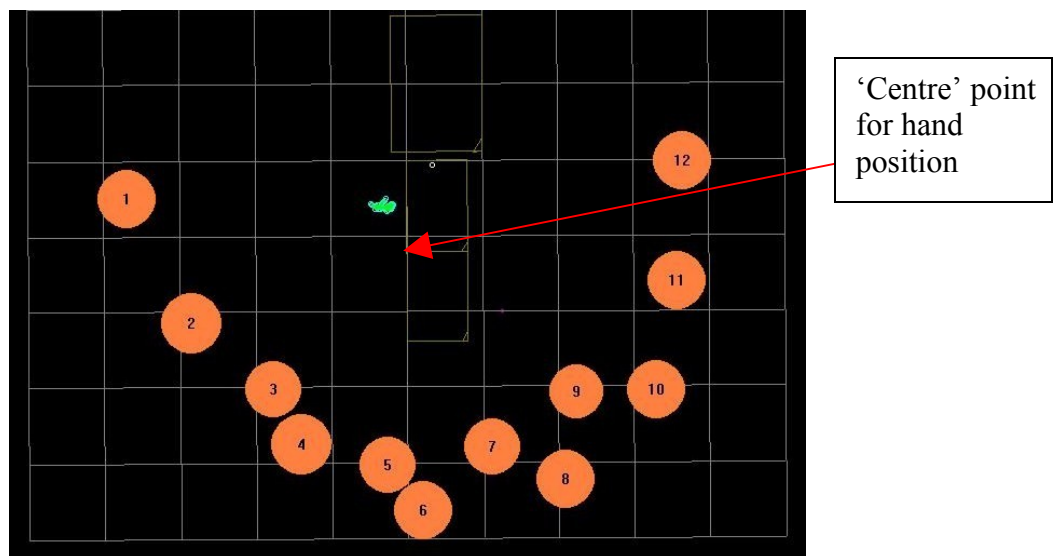
Fig 3.3 Overhead view of camera positions

Strobe intensity, i.e the level of light from the LEDs, for all cameras was set between 4-5 for each session. The sensitivity recorded in table 3.2 was used as a starting point with small adjustments made as necessary. Each session the cameras were all carefully focused on a mock hand consisting of 24 markers attached to wooden splints to ensure that data collected would be successful.

Table 3.2 Camera set up

Position	Cam number	Distance from centre (m)	Sensitivity	Gain
1[*]	12	1.8	6.5	5
2	13	1.9	5.5	5
3 (L)	7	1.65	6.5	5
4	8	2.1	7.5	5
5(L)	14	1.75	7.0	5
6	10	2.1	6.5	5
7(L)	9	1.75	6.3	5
8	5	2.1	8.0	5
9(L)	15	1.75	6.5	5
10	16	2.15	6.8	5
11	4	2	6.8	5
12	1	1.8	7.0	5

[*]Corner away from garage

(L) lower cameras

34 retro-reflective hemispherical markers (Vicon, Oxford, UK) were placed on the dorsal aspect of the hand. Four markers on the wrist (8.5mm in diameter), six markers to define the hand (5mm diameter) and twenty four markers on the fingers (3mm diameter). Several marker sets were tested during pilot testing (Appendix 11) with varying numbers and positions of markers. Three volunteers were used for pilot testing, trying many different combinations of marker positions, as well as different numbers and sizes of markers. The position of the hand within the capture volume was also altered several times to find the best angle to capture all markers as much as possible throughout the movements. The marker set used is shown in Fig 3.4, with the anatomical positions described in Appendix 12. For error analysis of the three main marker sets one female volunteer was used. The distance between pairs of markers at the proximal, middle and distal phalanxes of the index finger during movement was recorded. Results (Appendix 11) showed this marker set gave the lowest standard deviations of distance between the markers over nine repeats of a pinch grip. Therefore it showed the lowest level of skin movement artefact and greatest accuracy compared to the other models tested.

Marker positions were identified using non permanent marker pen before the markers were attached using "pre-tape" adhesive spray (Mueller, USA) of type designed to secure dressings. A small test patch on the lower arm was used to check for adverse reactions; if no reaction occurred the hand was then sprayed through a stencil and markers placed at set positions. No reactions occurred but if the spray failed to attach markers on any individual then double sided sticky tape was used and again tested on the arm in case of reactions.

Fig 3.4. 2 Markers per phalanx marker set

3.4 Trials

Subjects were seated in the centre of the lab with the arc of cameras surrounding them. The video camera was positioned so only the participants hand and torso was visible, the face was not captured to preserve anonymity. In the first static trial, subjects sat still with their elbow resting on the chair arm, and were asked to assume a relaxed position, they were then asked to raise their hand so the cameras could see and capture the resting position of the hand and fingers. Four dynamic trials then followed, each starting with fingers relaxed: 1) pinch grip 2) key pinch 3) making a fist 4) fingers spread. In the first task, subjects were asked to flex the hand until the thumb touched the index finger then extend as fully as possible or until they experienced considerable pain. The second task required subjects to flex the fingers so that the thumb meets the middle of the index finger as if holding a key and then fully extending. The third task involved subjects making a fist and then fully extending. The fourth trial involves fingers being abducted as much as possible and then adducted back.

Each action was completed 3 times per set, with 3 sets completed and a short rest time was allowed in between each set. Each participant completed trials in the same set order; completing tasks 1, 2, 3, and then 4. Subjects were asked to complete as much of the movement as possible but not to do anything that caused considerable pain.

3.5 Analysis

The data collected from the camera were then reconstructed using pre-determined parameters (Max acceleration; 5, Max noise factor; 1, Intersection limit; 2, residual factor; 0.5, Predictor radius; 3.) to produce a trajectory for each marker. These trajectories were then labelled according to the corresponding landmarks. Labelling of each trial was performed by first manually creating an auto label of the static trial for each subject that would then be used to speed up labelling of the dynamic trials. To create an auto label each marker was selected and manually labelled to correspond to the anatomical landmark that is represents, this set of labelled markers and relative positions would then be saved and can be applied to each trial of that subject. Any missed markers after the autolabel had been run were manually labelled. Trajectories were then defragmented and any gaps, therefore occlusion of markers, up to 6 frames long were auto-filled. Trials were then further cleaned if any crossover appeared where markers were getting swapped over, to perform this, the wrong data points needed to be snipped before being defragmented and the new trajectory labelled correctly. Some larger gaps on the hand were filled using Vicon GenPatch (Appendix 13) and Replace4 (Appendix 14) models as appropriate. As long as all other markers in the set are present it uses the information on the distances among these to determine where the missing marker should be. Data was then modelled using the missing data model (Appendix15) to locate where the gaps were and record this information to ensure these data points would not be used to determine crucial peak angle results. All gaps in the data were then filled to allow smoother filtering. A Butterworth filter with a cut-off frequency of 1Hz was then run, before modelling using the 2 markers per phalanx marker model (Appendix 16) to calculate angles at the finger joints. Flexion/extension and adduction/abduction are calculated at all the MCP, PIP and DIP joints and selected angles exported to Vicon Polygon to create reports and view the results (examples of which can be seen in Appendix 17). Angle data was also exported into excel to manipulate data. The three peaks and three troughs of each trial were selected and then results collated for each subject and group.

The angles were defined as shown in Fig 3.5, with the black line representing a zero value. Therefore a negative value for measurements in the y direction is representing extension, and positive values representing flexion angles. For movements in the z direction, when the fingers moved left of the central line they became positive and to the right become more negative.
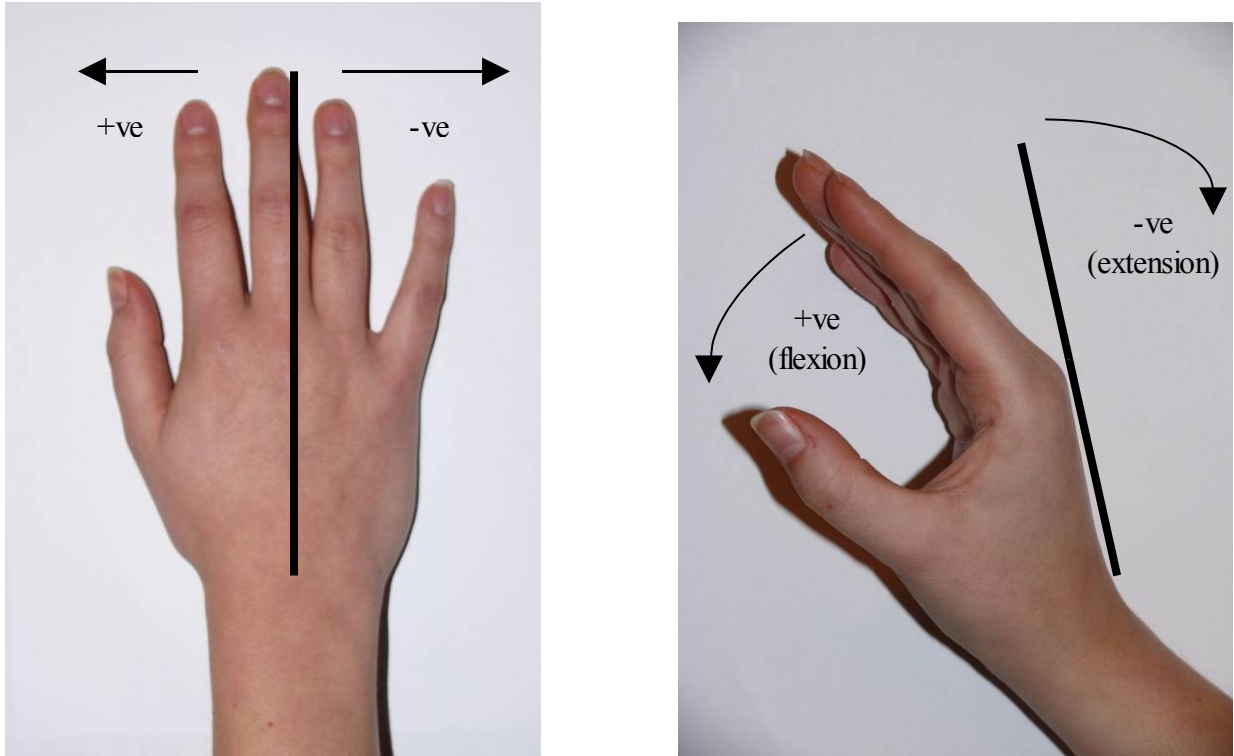
Fig 3.5. Definitions used to determine the values of hand movements in the z and y directions.

3.6 Statistical analysis

Descriptive statistics were used to analyse data, including mean, median and standard deviation of angles and the variations at different joints, fingers and within different groups. The data from all four MCP joints was selected to be analysed for all dynamic trials.

Normality of the data sets collected for normal, pre and post operative patients was assessed using an Anderson- Darling test. The different group data was then compared using Man-Whitney tests as not all the data sets were normally distributed.

MINITAB 15 statistical software (E-academy, Ontario, Canada) was used for all statistical analysis.

4. RESULTS

4.1 Introduction

Data from all the subjects; young normals (YNs), elderly normals (ENs), rheumatoid patients (RAs) and MCP replacement patients (MCPs) can be found on the results CD (Appendix 18). This includes the minimum and maximum values for y and z direction movements at the index, middle, ring and little finger MCP joints, for all four movements, for all 40 subjects used. Data is presented on the average minimum and maximum values plus ROMs for each group in the tables, looking at each movement in turn, with the graphs illustrating the differences in average ROMs for each group.

4.2 Pinch grip

Fig 4.1 Average ROMs for all subject groups when performing the pinch grip.
Error bars represent ± 1 standard deviation. Results are statistically significant (p < 0.05)
from YNs(*) ENs(▲) and RAs (●)

At all fingers average ROMs were significantly lower for the MCP patients (p < 0.05) compared to all other subject groups. Although in Fig 4.1 the elderly controls appear to show more limited movement than the young controls this was not significant, and again the rheumatoid patients were not significantly worse compared to the ENs although results suggest a difference. Table 4.1 shows that during the pinch movement the MCP subjects on average were not able to achieve any degree of extension at any of the fingers, as none of the minimum y values are negative.

4.3 Key grip

Fig 4.2 Average ROMs for all subject groups when performing the key grip.
Error bars represent ± 1 standard deviation. Results are statistically significant (p < 0.05) from YNs(*) ENs(▲) and RAs (●)

Again the MCP subjects showed significantly lower average ROMs (p < 0.05) compared to both normal groups for all fingers and smaller than RAs for index and middle fingers. Although results suggest other trends between groups none of these were found to be significant.

4.4 Fist

Fig 4.3 Average ROMs for all subject groups when making a fist.
Error bars represent ± 1 standard deviation. Results are statistically significant (p < 0.05) from YNs(*) ENs(▲) and RAs (●)

When making a fist, EN subjects' average ROM was significantly reduced compared to the younger controls. RAs showed significantly lower average range of movements compared to the younger and also elderly controls, with a further significant decrease found for the MCPs at the index and middle fingers (p < 0.05).

The first three movements all show the same pattern occurring, with the YNs capable of producing the greatest ROM for the pinch, key and grip movements, with highest values seen during the fist grip. There then appears to be an ageing effect, as the ENs produce lower values for all movements at all fingers, although only significant at the fist. The rheumatoid patient's movement is restricted to an even greater extent, with values lower than both normal populations, again only significant when forming a fist. The MCP replacement patients show the lowest ROM for all movements and at all fingers, significant at most fingers during all movements, suggesting that the implants were unable to restore movement to that of rheumatoid, let alone elderly normals. This pattern of decreasing movement repeats itself at all fingers across these three movements.
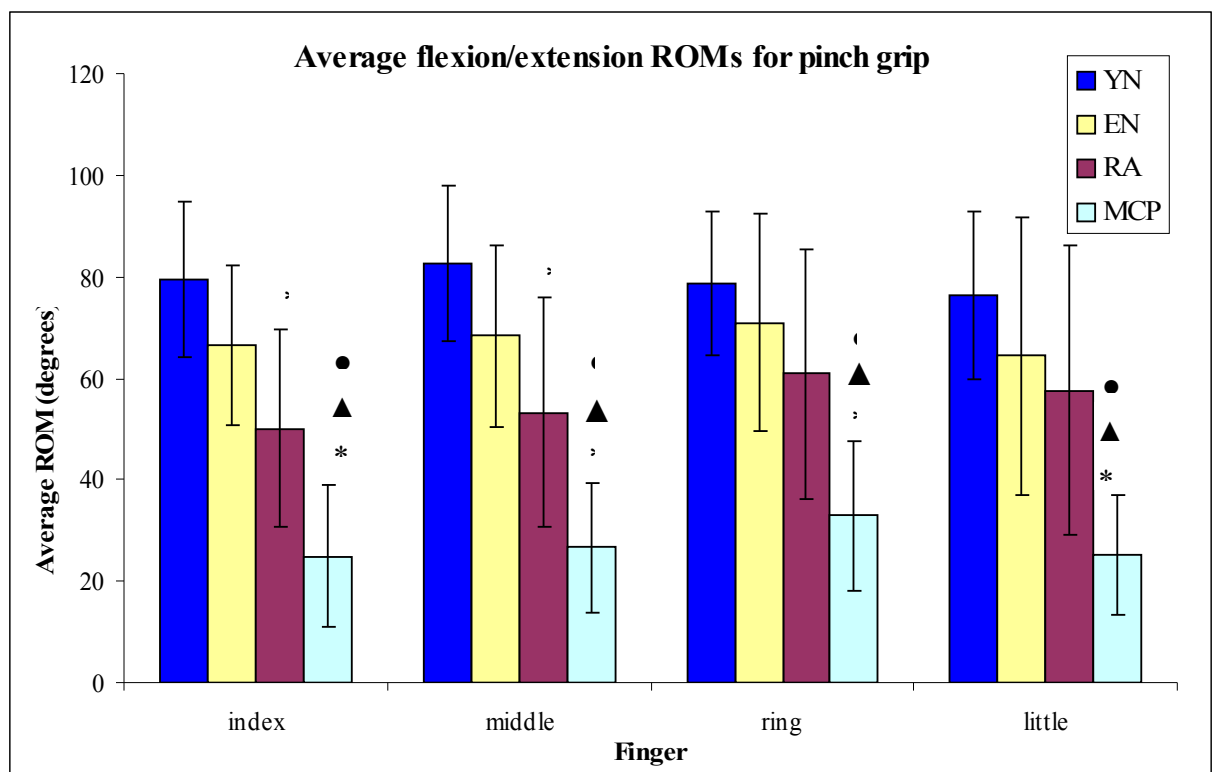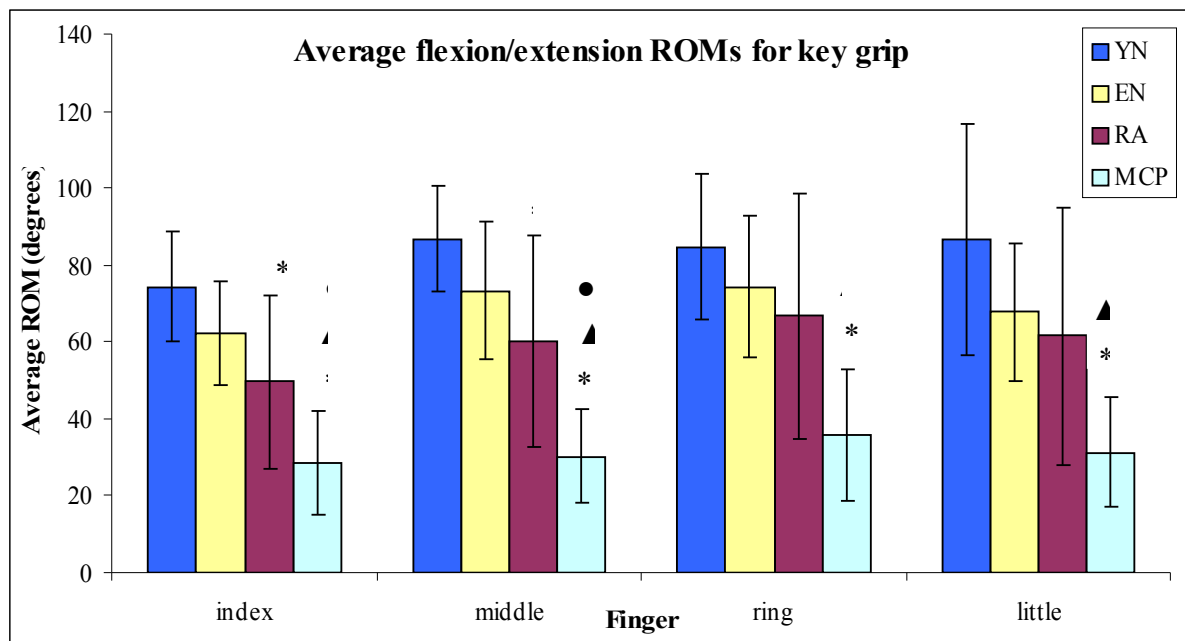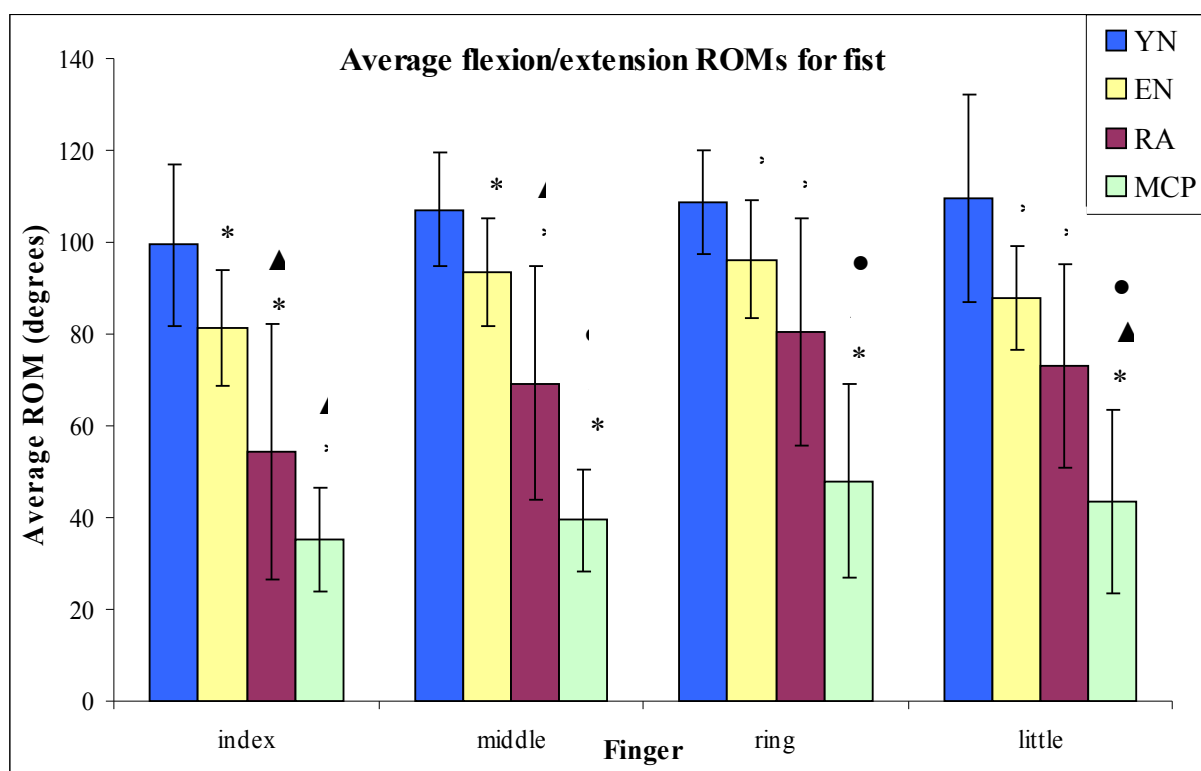
4.5 Spread
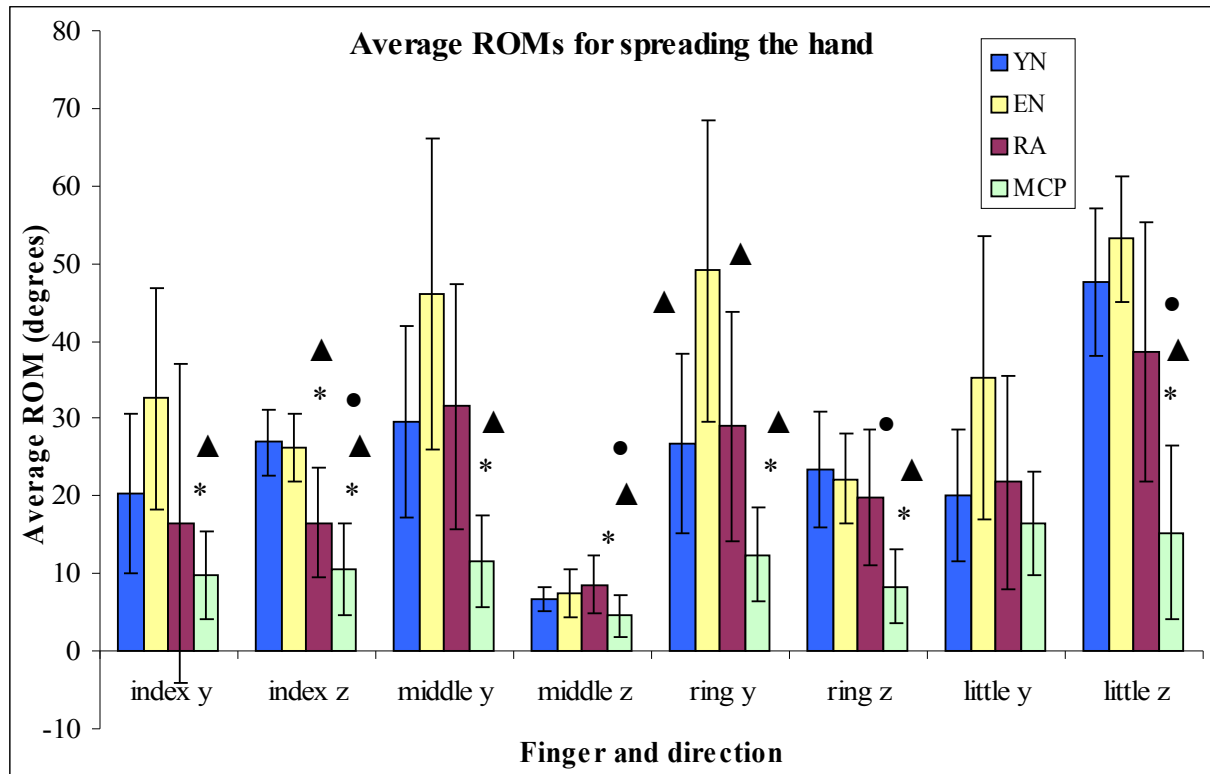
Fig 4.4 Average ROMs for all subject groups when spreading out the hand.
Error bars represent ± 1 standard deviation. Results are statistically significant (p < 0.05)
from YNs(*) ENs(▲) and RAs (●)

The ROMs for the spread movement do not repeat the pattern seen in the other movements, although in general the control subjects are still producing higher ROMs at all fingers there are a few exceptions and the results are not as clear as in the other graphs. When spreading out the hand, movement in the y direction (i.e flexion/extension) was significantly lower for MCP patients compared to both control groups (p < 0.05), and although results suggest a reduction in ROM compared to the RAs this was not found to be significant. Interestingly, the ENs' movement in the y direction was the highest at all the fingers, seen clearly in Fig 4.4, and movement was significantly greater at the ring finger (p < 0.05). This suggests in order to carry out this spreading movement ENs are needing to extend the fingers backwards and also flex fingers to a greater extent at the MCP joints (as seen in table 4.4) so are unable to keep the fingers straight as asked. In the z direction results were similar to the other movements, with the MCPs again showing significantly reduced ROMs at all fingers (p < 0.05) compared to all other subject groups. The RAs also appear to show reduced movement in this direction, although it is significantly so only at the index finger.

Table 4.1 Average max, min and ROMs (degrees) and standard deviations of projected angles for pinch grip

	Index max y	(SD)	Index max z	(SD)	Index min y	(SD)	Index min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	62.99	11.86	4.32	5.67	-16.43	9.69	-10.99	3.70	79.42	15.24	15.31	5.11
EN	53.83	12.21	1.55	7.15	-12.68	7.97	-12.31	5.29	66.51	15.65	13.86	5.78
RA	43.32	9.76	-7.11	15.16	-7.54	18.71	-21.96	13.18	50.11	19.53	14.84	5.72
MCP	45.65	16.56	-5.98	8.98	20.78	12.30	-13.44	9.25	24.87	14.01	7.47	2.67
	Middle max y	(SD)	Middle max z	(SD)	Middle min y	(SD)	Middle min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	62.29	13.19	-1.36	4.11	-20.26	9.44	-9.43	4.94	82.54	15.45	8.07	3.41
EN	54.00	11.81	-3.25	4.93	-14.28	11.53	-11.81	4.03	68.28	17.97	8.56	4.00
RA	50.27	15.29	-12.63	14.07	-3.36	22.49	-23.76	12.71	53.29	22.54	11.13	5.84
MCP	47.60	23.56	-2.64	7.33	21.00	18.05	-7.37	6.88	26.60	12.74	4.73	2.76
	Ring max y	(SD)	Ring max z	(SD)	Ring min y	(SD)	Ring min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	61.85	13.23	-0.34	3.94	-16.75	9.07	-18.55	4.23	78.60	14.17	18.21	5.46
EN	54.30	14.85	-4.34	6.44	-16.65	10.97	-17.93	5.52	70.95	21.48	13.59	4.43
RA	50.11	17.62	-15.28	11.89	-10.73	17.82	-31.91	12.76	60.84	24.68	16.64	8.99
MCP	41.88	15.69	-5.15	8.60	8.98	15.52	-12.37	10.19	32.90	14.79	7.22	2.18
	Little max y	(SD)	Little max z	(SD)	Little min y	(SD)	Little min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	65.44	14.08	8.52	7.77	-10.87	11.20	-41.28	8.54	76.31	16.45	49.80	10.94
EN	54.53	19.56	-0.23	13.87	-9.94	10.45	-39.94	9.44	64.47	27.37	39.71	14.31
RA	61.07	16.05	-9.14	16.03	3.46	18.87	-48.70	5.62	57.61	28.55	39.56	16.22
MCP	36.98	22.25	-2.47	9.86	11.79	22.31	-12.32	9.73	25.20	11.88	9.85	3.64

Table 4.2 Average Maximum, minimum and ROMs with standard deviations of projected angles for key movement.

	Index max y	(SD)	Index max z	(SD)	Index min y	(SD)	Index min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	56.24	9.64	1.17	4.80	18.13	11.07	-16.59	3.76	74.36	14.35	17.76	6.37
EN	51.26	11.00	-1.27	7.56	10.84	7.90	-17.46	5.39	62.10	13.54	16.19	7.18
RA	42.02	12.91	-11.54	18.22	-7.54	18.39	-26.86	13.04	49.55	22.64	15.32	10.31
MCP	47.71	16.82	-6.49	9.32	19.10	11.71	-15.87	8.94	28.61	13.33	9.37	3.97
	Middle max y	(SD)	Middle max z	(SD)	Middle min y	(SD)	Middle min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	67.07	10.97	-4.35	4.09	19.59	9.68	-11.99	3.85	86.66	13.74	7.63	3.03
EN	61.21	11.93	-5.56	3.21	12.08	12.65	-14.17	4.15	73.29	17.86	8.61	4.04
RA	57.41	12.19	-13.98	12.18	-2.85	23.05	-24.19	13.44	59.98	27.53	7.79	9.63
MCP	50.61	20.76	-3.80	6.89	20.33	18.35	-11.36	6.36	30.29	12.10	7.55	3.79
	Ring max y	(SD)	Ring max z	(SD)	Ring min y	(SD)	Ring min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	66.37	15.25	-1.70	4.35	18.21	8.55	-18.40	5.26	84.57	18.90	16.70	5.78
EN	60.91	12.32	-4.87	2.95	13.34	12.82	-19.87	5.96	74.25	18.45	15.00	5.96
RA	57.76	19.14	-14.43	13.04	-8.91	19.37	-34.40	13.17	66.67	31.72	19.97	9.35
MCP	44.02	10.45	-5.35	8.71	8.29	14.81	-14.55	9.45	35.73	17.21	9.20	4.57
	Little max y	(SD)	Little max z	(SD)	Little min y	(SD)	Little min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	69.25	20.58	6.09	8.24	17.32	12.14	-40.93	9.00	86.57	30.12	47.02	11.84
EN	61.28	13.13	-0.21	7.55	-6.39	9.48	-40.46	12.85	67.67	17.90	40.26	14.84
RA	68.37	17.82	-7.20	15.63	6.79	20.01	-49.17	6.23	61.58	33.55	41.97	19.16
MCP	39.37	17.58	-5.24	8.25	8.04	21.16	-15.41	8.23	31.33	14.18	10.17	4.11

Table 4.3 Average Maximum, minimum and ROMs with standard deviations of projected angles for fist movement

	Index max y	(SD)	Index max z	(SD)	Index min y	(SD)	Index min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	80.78	14.10	2.09	4.82	-18.59	12.40	-14.72	4.47	99.37	17.62	16.80	6.57
EN	70.36	13.75	2.16	6.22	-10.90	6.58	14.75	3.74	81.26	12.70	16.91	6.23
RA	49.49	10.81	-10.00	13.99	-5.02	21.99	-27.37	9.61	54.52	27.87	17.37	6.14
MCP	54.17	14.14	-6.31	9.60	18.75	11.09	-14.06	9.13	35.42	11.31	7.75	3.31
	Middle max y	(SD)	Middle max z	(SD)	Middle min y	(SD)	Middle min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	86.74	10.28	-0.62	3.28	-20.36	9.77	-10.40	2.59	107.10	12.50	9.78	3.76
EN	79.91	12.47	-2.47	2.90	-13.58	11.01	-14.11	3.19	93.48	11.77	11.64	3.51
RA	66.69	9.58	-13.43	10.41	-3.65	23.19	-24.65	12.24	69.29	25.49	10.89	3.62
MCP	59.53	18.34	-3.11	7.42	20.18	17.72	-10.55	7.15	39.35	11.17	7.44	3.33
	Ring max y	(SD)	Ring max z	(SD)	Ring min y	(SD)	Ring min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	89.74	10.37	3.56	3.26	-18.95	8.12	-18.55	4.02	108.68	11.19	22.10	5.05
EN	81.20	14.81	-0.51	2.62	-15.08	11.74	-19.32	5.05	96.29	12.98	18.82	6.29
RA	72.69	14.04	-10.68	9.83	-8.84	16.51	-35.05	13.67	80.33	24.72	25.79	10.67
MCP	55.45	14.51	-3.41	7.41	7.43	15.40	-13.99	9.17	48.02	20.99	10.58	5.62
	Little max y	(SD)	Little max z	(SD)	Little min y	(SD)	Little min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	91.35	13.98	15.57	6.77	-18.22	11.72	-39.73	9.27	109.57	22.68	55.31	11.12
EN	81.13	10.38	12.04	5.66	-7.49	10.66	-42.34	11.70	87.88	11.37	54.38	12.39
RA	80.04	9.18	2.43	11.98	7.11	16.99	-50.12	4.91	72.93	22.09	52.55	13.58
MCP	51.68	19.17	-1.88	7.38	8.98	20.82	-16.33	8.23	43.61	20.00	14.46	6.25

Table 4.4 Average maximum, minimum and ROMs with standard deviations for projected angles during spread movement

	Index max y	(SD)	Index max z	(SD)	Index min y	(SD)	Index min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	23.54	11.41	13.36	3.07	3.22	8.75	-13.56	4.44	20.32	10.31	26.91	4.34
EN	42.26	12.41	12.03	4.95	9.61	13.31	-14.20	4.92	32.65	14.24	26.23	4.29
RA	31.08	16.09	-0.53	15.58	14.64	17.27	-19.00	14.04	16.45	20.50	16.57	7.01
MCP	34.83	14.10	-1.66	12.90	24.97	12.42	-12.15	10.60	9.85	5.65	10.49	5.90
	Middle max y	(SD)	Middle max z	(SD)	Middle min y	(SD)	Middle min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	24.78	12.19	-0.30	4.30	-4.81	5.06	-6.94	4.26	29.60	12.36	6.64	1.52
EN	43.38	14.59	-1.57	4.63	-2.63	14.65	-9.08	2.09	46.01	20.04	7.51	2.98
RA	39.04	12.02	-5.58	12.98	7.49	17.32	-14.22	13.42	31.55	15.77	8.64	3.64
MCP	35.10	18.75	-2.15	7.78	23.47	18.34	-6.68	7.66	11.63	6.00	4.54	2.73
	Ring max y	(SD)	Ring max z	(SD)	Ring min y	(SD)	Ring min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	21.66	11.55	1.02	4.18	-5.10	5.40	-22.35	5.64	26.76	11.57	23.38	7.41
EN	43.29	12.84	-2.37	4.02	-5.76	13.15	-24.62	4.64	49.05	19.36	22.26	5.82
RA	34.22	11.83	-7.61	15.50	5.25	17.29	-27.43	15.02	28.97	14.88	19.82	8.78
MCP	32.03	19.59	-3.20	9.95	19.63	23.04	-11.57	10.26	12.40	6.07	8.37	4.81
	Little max y	(SD)	Little max z	(SD)	Little min y	(SD)	Little min z	(SD)	ROM y	(SD)	ROM z	(SD)
YN	17.59	10.82	3.29	3.53	-2.53	8.16	-44.31	9.20	20.12	8.47	47.60	9.62
EN	40.96	15.14	3.60	3.09	5.73	16.12	-49.58	8.32	35.23	18.32	53.18	8.07
RA	39.64	15.65	-3.64	12.13	17.80	19.14	-42.28	7.38	21.84	13.80	38.64	16.62
MCP	30.30	21.78	-3.01	8.74	13.78	21.33	-18.28	11.39	16.52	6.62	15.26	11.22

4.6 Patient feedback

Although no questionnaire was given to patients who took part in the study many were keen to volunteer information and often wanted to talk about their disease or the surgery they had had. All the post surgery patients who talked about their procedure were extremely happy with the results of their operation and keen to tell me how much better it had made their lives. The focus of the improvement differed, with several commenting on the improved appearance of the hand, especially straightening of the fingers. Others were more relieved about the lack of pain they now experience and their ability to do many more everyday tasks since their operation. So from the information they chose to volunteer it is clear that they all felt the procedure extremely worthwhile and effective, indeed many had been so pleased with results they had the other hand operated on as well. When the rheumatoid patients talked of their suffering, again focus was often on the physical appearance of their hands and how self conscious many of them were about how their deformities made them look. There were also comments on the pain and inability to perform tasks; varying from getting dressed properly and stitch work to not being able to shake someone's hand.

4.7 DAS scores

Only descriptive statistics were used on the DAS scores (data in table 3.1b). The average score was slightly lower in the rheumatoid population compared to the MCP replacement group; however the standard deviation in the MCP group was much higher, suggesting greater variation. The DAS scores meanings and derivation are explained further in the next section.

To summarise, ROM at the MCP joint appears to be negatively affected by ageing, with a further decrease seen if a patient is suffering from RA. The effect of having replacement surgery did not appear to improve movement, in this study in fact patients showed lower ROMs compared to those in the diseased state. However this does not take into account the ROM of subjects before the operation, so it is unknown what affect the surgery had on movement of the individuals.

5. DISCUSSION

5.1 Introduction

This study produced a large amount of data, a fraction of which was chosen for analysis. The main aim was to gain a better understanding of hand movement in both normal, diseased and MCP replacement populations. The results give a good understanding of how movement is affected by ageing, disease and surgery; there is, however, much more that can be done. As the study was of a preliminary nature it leads the way for many more investigations.

5.2 Control population

Understanding what is "normal" hand movement is a key question needing to be answered in the implant design process and more work needs to be carried out on this. Both control groups showed higher average ROMs compared to both patient groups, as expected they are able to move to a greater extent. The control group itself needs expanding to include male subjects of all ages to see if there are any gender differences. As an ageing affect was revealed in the results (significant during the fist movement, Fig 4.3), older elderly controls (possibly 60+ years of age) should be investigated to see if this age range has an even greater effect on hand movement and stiffness causing reduced range of movement. Also then there would be a more comparable age control match to those who have had the MCP replacement surgery who on average were older then those suffering from RA. Extending the control database would also enable testing and showing repeatability and reliability and could possibly lead to the model being validated for use in a clinical setting, as had been done recently with the Oxford foot model (Carson et al., 2001), as currently no hand model is available.

Within the normal population, the neutral or natural position could also be investigated. The data collected in this study, in the static trials, could be used to see how much flexion/extension of the hand is occurring when a subject is holding the hand in a relaxed neutral position. Determining what is a neutral position is a difficult question to answer. Is this the position one naturally holds their hand when relaxed, in which case suggestion have been made that this is closer to 30 degrees of flexion (DePuy, 2009), or is it anatomically neutral? The only way to determine the position definitively is to place

all markers on the back of the hand and then take x-rays or use bone pins as was done extensively with gait analysis to find what is straight/natural (Leardini et al., 2005). It may then be interesting to see how this position changes with age as it appears, from my data, that there is a natural progression to a more flexed natural state and furthermore how this is again affected by disease state and surgery as many studies suggests a more functional arc of motion post-surgery (Blair et al., 1984, Beckenbaugh et al., 1976, El-Gammel and Blair, 1993, Mannerfelt and Anderson, 1975). For abduction/adduction movements it is also hard to determine what the neutral position is, and for increased accuracy may need to be calculated for each finger.

Another factor to be considered is people within the normal population who may have hypermobility, criteria include passive dorsiflexion of the little finger over 90 degrees and hyperextension of the elbows and knees over 100 degrees (Beighton et al., 1973) This condition may have been affecting at least one of our control subjects and results in very large ROMs due to the larger extension angles achievable. YN03 and YN08 both had very large ROMs across all movements compared to the other subjects in the group (Appendix 18). There have been suggested links to hypermobility and an increased risk of developing osteoarthritis in later life (Bridges et al., 1992; Bird et al., 1978). Perhaps in future control subjects should also be screened for this as they may not represent "normal movement".

Previous work on a control population of similar age to my YN group found comparable ROMs (table 5.1). Chiu et al., (1998) asked subjects to perform a pinch like grip and used a similar marker set to mine. The average ROM at the MCP joints were 77 ±9 degrees, 85 ±13 degrees, 87 ±9 degrees and 70 ±17 degrees for the index, middle, ring and little finger respectively. Compared to my results for the young normals of 79 ±15 degrees, 82 ±15 degrees, 78 ±14 degrees and 76 ± 16 degrees for the same fingers. Fowler and Nicol (2001b) also report a similar average ROM of 79 degrees for controls using an instrumented glove worn by subjects for 6 hours over a 3 day period.

5.3 Rheumatoid patients

Results from this study showed that average ROMs for RA patients were significantly reduced from young and in some cases elderly controls as well (Figs 4.1-4.4), meaning that movement is not as effective and some tasks would be difficult with a more limited range. The high standard deviations (tables 4.1-4.4) within the rheumatoid population are not surprising given the large variation between the subjects used in terms of the deformities seen and possibly the different stages of the disease they were suffering from. There are 2 or 3 patients with considerably lower ROMs compared to all the others, particularly RA05 and RA08 (Appendix 18). Movement may not necessarily be affected by the number of years they have been suffering from the disease, it could be related to the disease activity. Disease activity scores (Appendix 10) are generally used to assess the activity of rheumatoid arthritis (van der Heijde et al., 1990, 1992) and include a combination of c-reactive protein (CRP), erythrocyte sedimentation rate (ESR) and rheumatoid factor measures. The score also incorporates the patients own assessment of global health at that time (where they rate themselves on a scale of 1-100) and then a count of tender and swollen joints. These factors are all fed into a formula to give the overall DAS score. Another factor affecting ROM will be at what stage the RA was discovered and what treatment was given which will affect the amount of deterioration and bone erosions, this could be investigated by looking at the x-rays of the subjects. It may be of interest to use DAS scores to select subjects and just use more advanced stages or test 10 subjects from each stage to look at how varying disease activity affects the movement the patient is then capable of. Other possibilities include using the stages of RA; either the 4 stages of anatomic changes of joint destruction or the 5 stages of pathogenesis of RA (see Pope, 1996). Furthermore the Ritchie articular index could be used, which is an assessment of joint tenderness, given as a sum of the grades at various locations (Ritchie et al, 1968).

There is, therefore, a definite need to expand the RA group; firstly by using more than 10 subjects and also including male subjects to see if there are any gender differences and also to get a more accurate idea of movement for RA sufferers. Within the RA group used in this research, many subjects had deformities at PIP and DIP joints either as well as MCP joints or instead. The location of the patient's deformity is likely to affect the results. Although it is expected that hand movement in general will be reduced it is understandably greater affected at the joints showing swelling and deformity. It may be useful to study subjects with only MCP deformities to determine the extent of restriction to these joints caused by RA.

The results obtained in this study can be studied in more detail, looking at the PIP and DIP angles of certain patients with deformities at these joints could help replacements at these locations. More in-depth analysis may also reveal that patients may have adapted their hand movements to carry out certain everyday tasks and so use the MCP joints to a greater extent to compensate for other joints affected. For example it may be the case that deformity at one joint, say the MCP, could cause movement to be greater than expected at the PIP or DIP joints to allow them to still be able to carry out the movement successfully, or vice versa. It may require selection of RA patients with more severe deformities at certain joints to investigate this further.

To date there appears to be no literature from motion analysis available to compare the results from the rheumatoid subjects in this study; this highlights the need for further work to understand the complex nature of movement in the rheumatoid hand.

5.4 MCP replacement patients

MCP patients produced the lowest ROMs at all fingers for all movements (Figs 4.1-4.4) and this effect was significant in most cases, showing the extent of the reduction in movement. The results suggest that having replacement surgery does not restore movement to anywhere near normal, although this may not really be necessary. However movement is in fact reduced significantly from subjects with RA, implying range of movement is reduced by having the replacement.

The standard deviations within the MCP replacement patients were also very large (tables 4.1-4.4), as again the ROMs differed greatly (Appendix 18) with MCP 08 producing very high values showing very good movement, however MCP 01 had very poor movement, capable of ROMs of only 7.4 and 4.3 degrees at the index and middle finger respectively during the pinch grip, compared to the averages for the MCP group which are 24.8 and 26.6 degrees respectively. The results obtained from the MCP patients may be affected by number of years post surgery, although when looking at subject characteristics (table 3.1) there does not appear to be any affect, this may again be due to the small sample size used. Using a greater number of patients from a large range of years post surgery could reveal an effect. Although other factors could also affect the movement these patients can achieve including how much they comply with the post operation stretching and exercises given, how much they continue to use their hands and how active they are.

To investigate this further the group needs to be extended to look at differences over the years post surgery, possibly to see if there is a more specific point when the implants become less effective. Again male subjects need to be included in the patient group as well.

Literature from motion analysis using patients with MCP replacements is limited. Fowler and Nicol (2001a, 2002) have studied the forces and movements of rheumatoid hands after replacement using the floating marker clusters as mentioned previously; however in the first publication only present data on resultant moments, where results from MCP replacement patients are on average 41% of that of control subjects (Fowler and Nicol, 2001a). In 2002 they report an average MCP flexion angle of 54 degrees for a key turn, this compares to our results of the MCPs key turn of 28, 30, 35 and 31 degrees at the index, middle, ring and little fingers respectively (31 degrees average). Some studies on replacements report pre and post operation average ROMs measured using goniometers, also shown in table 5.1. Blair et al., (1984) report active ROM of 43 degrees post operation compared to 26 degrees pre operation. Mannerfelt and Anderson (1975) and Beckenbaugh et al., (1976) found similar ROMs for MCP replacement patients 2.5 years post surgery of 40 and 38 degrees respectively. El-Gammel and Blair (1993) found that ROM was increased 2 years post operation but then showed a gradual decline as the follow-up period increased. In all these clinical studies the arc of motion appears to change position so that the active ROM is in a more extended position, compared to a more flexed arc as seen pre operatively. This may allow more functional movement. It may be interesting to review the results of this study further to look at the arcs used by the different subject groups to see if this differs and also if it changes as the number of years post op increases.

Although the results from this study appear to suggest that having replacement surgery reduces ROM this may not be the case. The values for the pre-operative ROMs are unknown; therefore, it is not possible to say if movement has increased or decreased. It may be that movement is in fact greater as they may have had very poor movement before surgery. A further interesting study and only way to determine this, would be to investigate patients, selected from waiting lists for MCP replacements, before and after surgery. Another avenue would be following the same subject from early to late arthritis testing their movement as the disease progresses.

Table 5.1 Comparison of results of average ROM for normal and patient subjects

Study	Method of evaluation	Subjects	Normal ROM (°)	Pre op ROM (°)	Post op ROM (°)
This study*	Motion analysis	10 young normal 10 elderly normal 10 RA 10 MCP replacements	80 68	55	27
Chiu et al., *	Motion analysis	32 controls	79		
Fowler & Nicol	Instrumented glove	8 controls 8 MCP replacements	79		52
Blair et al.,	goniometer	28 MCP replacements		26	43
Mannerfelt & Anderson		MCP replacements after 2.5yrs			40
Beckenbaugh et al.,		MCP replacements after 2.5yrs			38

*Average ROM for this study and Chiu et al., (1998) calculated as an average of all four fingers for the pinch movement.

5.5 Rotation

The role of rotation on implant fracture is still unknown as the marker set used in this study and others (Su et al., 2005, Cerveri et al., 2007; Chiu et al., 1998) is not comprehensive enough to provide information on rotational angles. Although tested in preliminary testing (Appendix 11) the angles produced were not deemed great enough compared to the error of the system to be confident we were measuring precise results. A method to measure rotation needs to be developed to see if this could be affecting the implants. One possibility is to use a model similar to the floating clusters (Fowler and Nicol, 1999, 2001a, 2002) but using smaller markers and therefore smaller clusters now that I have shown that very small markers can be used to precisely measure movement in 3D motion analysis. This could then allow more than one finger or joint to be measured at the same time, unlike with the previous floating clusters.

5.6 Forces

As well as further testing on the angular measurements achievable by RA and MCP replacement, force measurements may provide further insight as to why fractures occur. Loads applied during everyday activities may contribute to the failure of the implants. Fowler and Nicol (1999) have developed a force transducer to measure external loading data and used it to test movements in control and MCP replacement subjects (Fowler and Nicol, 2001a, 2002). Results found that on average MCP replacement subjects were capable of exerting forces that were only 38% of those exerted by the control subjects (Fowler and Nicol, 2001a).

They also report that MCP implants are subjected to higher normalised muscle/tendon forces, which have a resultant contact force 22% larger on average compared to the normalised control data. Fowler and Nicol (2002) go on to suggest that the loading systems encountered could be contributing to the failure of the implants by causing high stress concentrations at the hinge region that tend to lead to the occurrence of ulnar drift. Implants, therefore, may not be strong enough to withstand these forces, especially in younger patients with higher grip strengths. Pressure sensors could be used on the fingertips of the subjects to measure the force they are applying to everyday objects, a simple cylinder or a pen maybe, as they are grasped. Combining information on the forces applied with the angles and possibly the velocity of the movement would provide much more detailed information on the exact conditions to which implants are subjected. Again control, rheumatoid and replacement surgery patients should be used to investigate any differences, especially patient hands as they are likely to be experiencing different forces due to the nature of the disease.

5.7 Mechanical testing

Joyce (2003) is a strong advocate for the need to test implants in vitro before they are inserted and used in vivo. His work with the finger simulator as mention previously (Joyce et al., 2000, 2003,) is one such possibility. Using such machinery to test implants to failure could give vital indicators for improving current designs to prevent or at least prolong such failure. Any increase in longevity of implants will be immensely beneficial to patients if it can reduce revision procedures. When testing Sutter implants with the simulator, Joyce et al., (2003) found the fracture location and type to be the same as those in implants which they removed from patients showing that the machine replicated the conditions experienced in the hand. The machine put implants through 112 cycles per minute, with 3000 flexion/extension dynamic loads of 10-15 N through a 90 degree range, followed by a static pinch load of 160N. Other mechanical tests could be considered as a direct result of the more detailed biomechanics discovered. For example now testing through a smaller ROM in the flexion extension cycles as this is what the RA hand is subjected to in everyday tasks.

The implants could also be tested to see if they withstand rotational forces and if this causes fracture. If rotation does indeed cause fracture but it is not at the junction of the distal stem and hinge region, then it is unlikely to be the main cause of fracture found so far. However would not rule out that it could have a role as a contributing factor.

6. CONCLUSIONS

Due to the preliminary nature of this study it has undoubtedly raised more questions than it has answered and paves the way for much more research into discovering the details of hand movement. One important result to come out of this project has been to show that it is possible to use motion analysis accurately and effectively in measuring hand movement not only in a "normal" control population but also in diseased and post-surgery hands with severe deformities. The study has shown that more markers could be added to existing markers sets and smaller individual markers could be used and so increase the accuracy as skin movement will play less of a role. Now such a marker set has been developed, it can be used to get a substantial database from all populations to understand hand movement to a greater extent than we do currently and therefore aid in replacement design. The marker set can now also evolve and possibly incorporate even more markers to determine any rotational movements involved.

The other main finding of this study was that ROM at MCP joints was affected by ageing, reduced again by rheumatoid arthritis and significantly lowered further by MCP replacement. This therefore suggests movement may not be restored by implants to that which other rheumatoid patients are capable of. Although, the surgery is reducing pain and may provide improved movement compared to what the patient experienced before.

Designs for improved finger implants are unlikely to come from just one area of research; it is more likely to be many contributing factors that influence the fracture of these prostheses. Understanding the movement to which they are subjected to, especially when implanted in a rheumatoid hand, is one important area but results need to be considered along side other information available such as the materials used and also the forces experienced.

7. APPENDICES

Appendices 1 to 8 are not available in this web version of the thesis

Appendix 9 – Letter to GP

UNIVERSITY OF BIRMINGHAM

Department of Mechanical and manufacturing Engineering

Version1 30/03/08

TITLE OF STUDY: A biomechanical analysis of the rheumatoid hand after MCPJ replacement.

Dear Dr

RE: Patient ……………………………….. **DOB** …………………………………

I write to inform you that your patient will be a participant in a medical research study. The aim is to understand hand movement in people with rheumatoid arthritis. We plan to study what ranges of movements are needed to perform essential tasks. The purpose of the study is to assess how the rheumatoid hand moves after a patient has had their metacarpal-phalangeal joints (MCPJ) replaced. This will be compared to patients with rheumatoid arthritis who have not had this operation, and to healthy volunteers.

The Chief Investigator is Miss Louise Lester from the University of Birmingham

The local collaborator at the Worcestershire Acute Hospitals is Miss Helen Whalley

The members of staff from the Rheumatology and Orthopaedic departments will assist in this study. These patients are normally under the care of Professor Rai (Consultant Rheumatologist and visiting Professor at University of Worcester) and Mr Arafa (Consultant orthopaedic surgeon) at the Worcestershire Acute Hospitals NHS Trust.
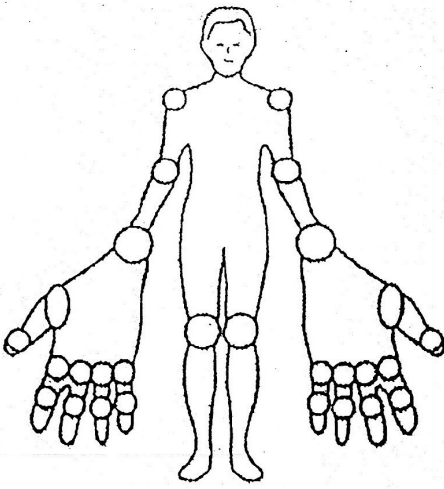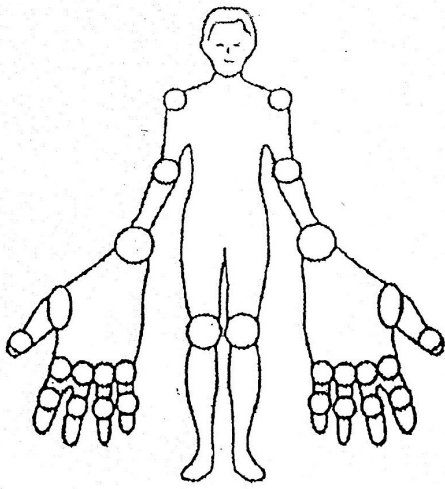We hope to publish the results of the study in the future.

If you have any queries, please do not hesitate to contact:

Miss Helen Whalley
Orthopaedic SpR
Dept of Orthopaedics
Worcestershire Acute Hospitals NHS Trust
Worcester

Yours sincerely

Miss Helen Whalley

Appendix 10 -Disease activity score (DAS) questionnaire

Patients Name	Unit Number

Scoring sheet for DAS 28, Anti TNF Therapy	Date

CRP [   ]   (check FBC, etc also)

ESR [   ]   Rh F [   ]

Patient's Global Assessment of Disease Activity

Considering all the ways your arthritis affects you on average how have you been doing in the past week?:

Disease as   Disease as
Well as can   _____   bad as
Be expected   imaginable

Joint Counts

Swollen Joints   Tender Joints

Number [   ]   Number [   ]

Appendix 11 – preliminary testing

Preliminary testing of different marker sets and modelling approaches

Vicon Model
The Vicon Marker set/Model defines the Index finger segments as follows;

Proximal Phalanx   RH2 – RIF1
Middle Phalanx   RIF1 – RIF2
Distal Phalanx   RIF2 – RIF3

Markers are placed as follows

RH2 – Distal head of the 2nd Metacarpal
RIF1 – Distal head of the 2nd Proximal Phalanx
RIF2 – Distal head of the 2nd Middle Phalanx
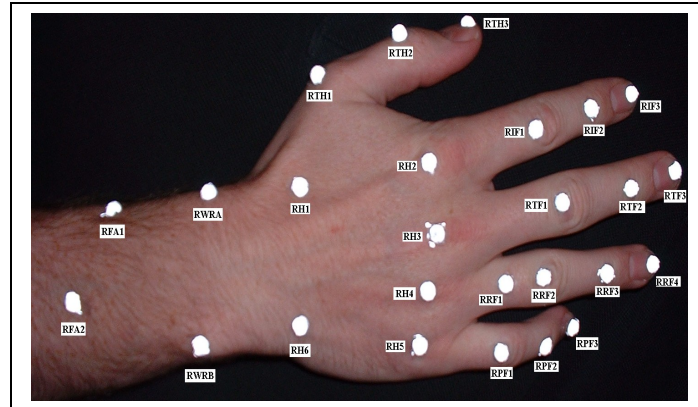RIF3 – Distal head of the 2nd Distal Phalanx

Fig 1. Vicon model marker set

MARRC 2 Phalanx Marker Model
Finger segments based on the following index finger segment definitions;

Proximal Phalanx   RIPP1 – RIPP2
Middle Phalanx   RIMP1 – RIMP2
Distal Phalanx   RIDP1– RIDP2

Markers are placed as follows;

RIPP1 – Proximal head of the Proximal Phalanx
RIMP1– Proximal head of the Middle Phalanx
RIDP1– Proximal head of the Distal Phalanx
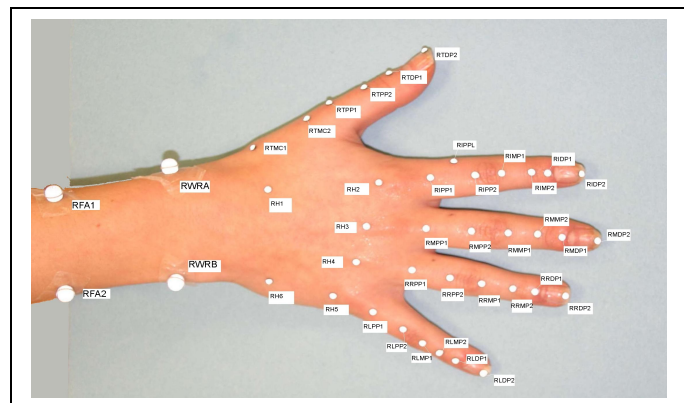
Fig 2. MARRC 2 Phalanx marker set

MARRC Model – Joint Centres
Markers are placed over the joint centres and the finger segments are defined as follows.

Proximal Phalanx   RIPIP-RIMCP
Middle Phalanx   RIDIP-RIPIP
Distal Phalanx   RIDPT-RIDIP

RIMCP - Right Index Finger MCP Joint Centre
RIPIP   - Right Index Finger PIP Joint Centre
RIDIP   - Right Index Finger DIP Joint Centre
RIDPT   - Right Index Finger Distal Phalanx Tip

Fig 3. MARRC model joint centres marker set

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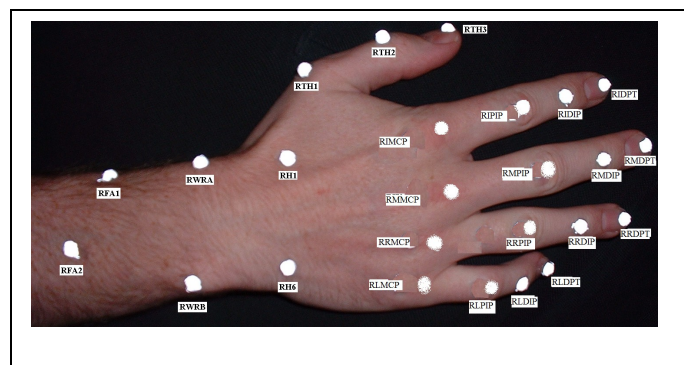The following tables present the data from the modelling of the right index finger during a pinch grip repeated nine times by the same subject. Using the original Vicon Hand model, a "MARRC Model 2 Phalanx Marker" model and a "MARRC Joint Centre" model.

Output Angle Data.

Joint	Vicon Model		MARRC Model 2 Phalanx markers		MARRC Model Joint Centre	
	Mean Peak Angle at Flexion	Mean Peak Angle at Extension	Mean Peak Angle at Flexion	Mean Peak Angle at Extension	Mean Peak Angle at Flexion	Mean Peak Angle at Extension
MCP	47.7°	22.3 °	52.5 °	29.0 °	56.0 °	31.7 °
PIP	38.2 °	13.0 °	51.0 °	15.3 °	44.3 °	5.8 °
DIP	35.2 °	8.2 °	25.2 °	9.3 °	21.5 °	16.0 °

Marker Variability (Skin movement artefact)

Vicon Model				
	Phalanx Segment (Vector Definition)	Mean Distance between Markers(mm)	SD	Range (mm)
Proximal Phalanx	RH2 – RIF1	36.4	2.30	32.5-39.5
Middle Phalanx	RIF1 – RIF2	28.2	1.23	26.5-30.5
Distal Phalanx	RIF2 – RIF3	22.0	0.48	21.5-23.0

MARRC Model "2 Phalanx Marker"				
	Phalanx Segment (Vector Definition)	Mean Distance between Markers (mm)	SD	Range (mm)
Proximal Phalanx	R2PP – RIF1	22.8	0.92	21.5-24.0
Middle Phalanx	R2DP – RIF2	14.1	0.55	13.5-15.25
Distal Phalanx	R2MP – RIF3	13.2	0.30	12.75-13.75

MARRC Model "Joint Centre"				
	Phalanx Segment (Vector Definition)	Mean Distance between Markers (mm)	SD	Range (mm)
Proximal Phalanx	RIPIP-RIMCP	37.9	3.2	34.5 – 43.0
Middle Phalanx	RIDIP-RIPIP	24.1	1.508	22.6 – 27.0
Distal Phalanx	RIDPT-RIDIP	16.9	0.732	16.5 – 17.5

Rotational data

On the MARRC 2 markers per phalanx model in certain trials an additional lateral marker was added to the index finger (labelled RIPPL on fig 2) to study rotational angles.

The following graphs show data collected during testing, with from top to bottom; X - flexion/extension, Y- abduction/adduction, Z- rotation

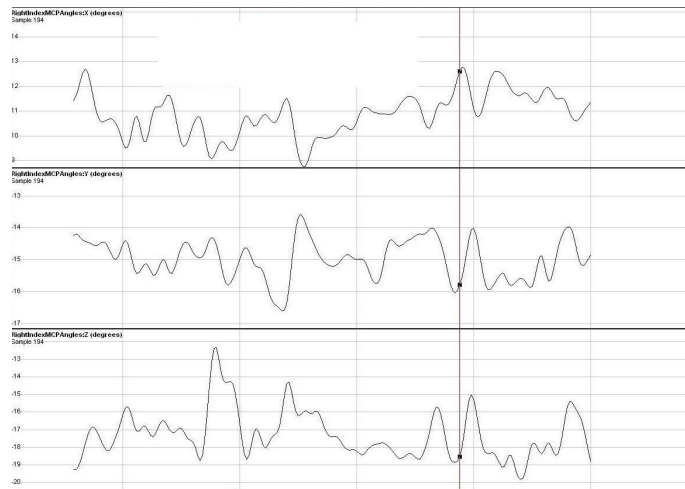Fig 4. Static trial, showing the variability of the measurements at the MCP joint.
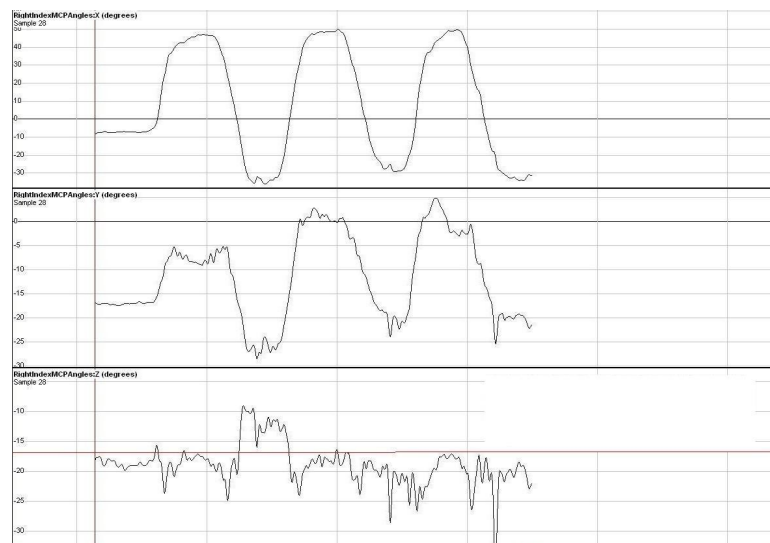

Fig 5. Dynamic trial, angles generated at MCP during pinch grip

The red line added to the rotational plot represents "zero", this 17 degree point is where the hand is at neutral in the static trials. Therefore angles less than 17 degrees represent external rotation and angles greater than 17 degrees represent internal rotation.
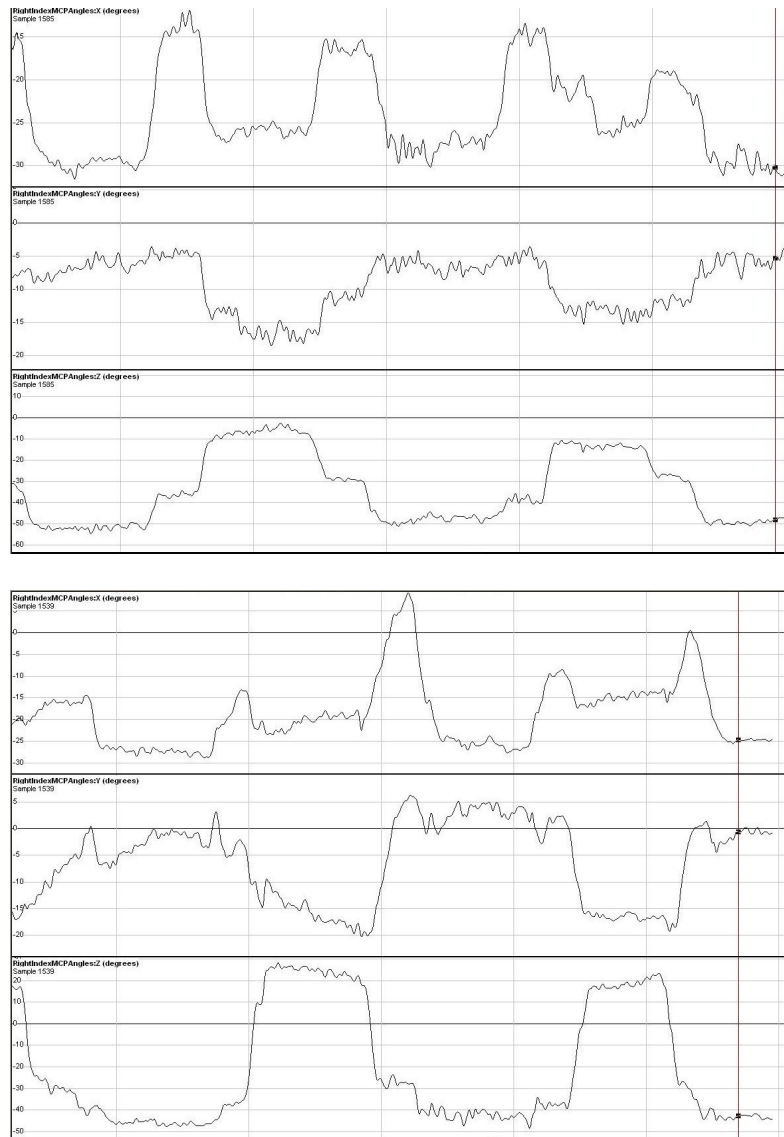
Fig 6a and b. MCP angles during forced rotation.

The final two trials investigated the amount of rotation when the finger was forced to rotate using external force to twist the MCP joint as much as possible. Fig 6a shows data when the tip of the finger is twisted with the finger staying straight and Fig 6b shows angles produced when the PIP and DIP joints are flexed and then external force is applied.

The rotational data is inconclusive and gives no real indication of actual rotation occurring as skin error is too high. The variability of the angle when the hand was held still was approximately 5 degrees (as seen in Fig 4) and the rotation measured during a dynamic trial was only approximately 7 degrees (Fig 5). It is therefore difficult to differentiate between rotation of the bone and skin movement, so angles produced are unlikely to be a true reflection of rotation. The forced rotation data gives much higher values showing that measurement of rotation is perhaps possible even given the errors. However in everyday movements and activities rotation is unlikely to reach such high values. Consequently when testing common hand function tasks the error is likely to be too high to give a significant result.

Appendix 12 -Marker positions

RFA1	Right lower forearm thumb side, (third of the way up in line with RWRA)
RWRA	Right wrist bar thumb side
RWRB	Right wrist bar pinkie side
RFA2	Right lower forearm pinkie side, (third of the way up in line with RWRB)
RH1	Top of second metacarpal (just under index knuckle) (Right Hand)
RH2	Head of second metacarpal (base of index finger)(Right Hand)
RIPP1	Right Index Finger Proximal Phalanx 1 (i.e. proximal end of the prox. phalanx)
RIPP2	Right Index Finger Proximal Phalanx 2 (i.e. distal end of the prox phalanx)
RIPPL	Right Index Finger Proximal Phalanx Lateral (placed on the side of the phalanx to create a segment)
RIMP1	Right Index Finger Middle Phalanx 1 (proximal end of middle phalanx)
RIMP2	Right Index Finger Middle Phalanx 2(distal end of middle phalanx)
RIDP1	Right Index Finger Distal Phalanx 1 (proximal end of distal phalanx)
RIDP2	Right Index Finger Distal Phalanx 2 (distal end of distal phalanx)(on finger tip)
RH3	Head of third metacarpal (base of third finger) (Right Hand)
RMPP1	Right Middle Finger Proximal Phalanx 1 (proximal end of proximal phalanx)
RMPP2	Right Middle Finger Proximal Phalanx 2 (distal end of proximal phalanx)
RMPPL	Right Middle Finger Proximal Phalanx Lateral
RMMP1	Right Middle Finger Middle Phalanx 1(proximal end of middle phalanx)
RMMP2	Right Middle Finger Middle Phalanx 2(distal end of middle phalanx)
RMDP1	Right Middle Finger Distal Phalanx 1 (proximal end of distal phalanx)
RMDP2	Right Middle Finger Distal Phalanx 2(distal end of distal phalanx)(on finger tip)
RH4	Head of fourth metacarpal (base of ring finger)(Right Hand)
RRPP1	Right Ring Finger Proximal Phalanx 1 (proximal end of proximal phalanx)
RRPP2	Right Ring Finger Proximal Phalanx 2(distal end of proximal phalanx)
RRPPL	Right Ring Finger Proximal Phalanx Lateral
RRMP1	Right Ring Finger middle Phalanx 1(proximal end of middle phalanx)
RRMP2	Right Ring Finger middle Phalanx 2(distal end of middle phalanx)
RRDP1	Right Ring Finger Distal Phalanx 1(proximal end of distal phalanx)
RRDP2	Right Ring Finger Distal Phalanx 2(distal end of distal phalanx)(on finger tip)
RH5	Head of fifth metacarpal (base of pinkie)(Right Hand)
RLPP1	Right Little Finger Proximal Phalanx 1(proximal end of proximal phalanx)
RLPP2	Right Little Finger Proximal Phalanx 2(distal end of proximal phalanx)
RLPPL	Right Little Finger Proximal Phalanx Lateral
RLMP1	Right Little Finger middle Phalanx 1(proximal end of middle phalanx)
RLMP2	Right Little Finger middle Phalanx 2(distal end of middle phalanx)
RLDP1	Right Little Finger Distal Phalanx 1(proximal end of distal phalanx)
RLDP2	Right Little Finger Distal Phalanx 2(distal end of distal phalanx)(on finger tip)
RH6	Top of fifth metacarpal (just under little finger knuckle)(Right Hand)

Markers attached as close as possible to these bony landmarks; identified by lightly pressing on subjects hand. At wrist RWRA and RWRB can be located by gentle flexion and extension of wrist.

Appendix 13 -GenPatch

```
{*VICON BodyLanguage (tm) model                              *}
{*========================================================= *}
{*Title:    Gen (Generic) Patch                              *}
{*Author:   Joe Bevins                                       *}
{*Date:     Nov 2006                                         *}
{*Purpose:  Patch missing markers on a segment               *}
{*Comments: Designed to patch missing markers on             *}
{*          a segment of four.  3 Markers must               *}
{*          be present in a given frame for the              *}
{*          fourth to be recreated.                          *}
{*                                                           *}
{*          MUST BE RUN ON A (COMPLETE i,e.                  *}
{*          with a complete segment) STATIC                  *}
{*          BEFORE USE ON A DYNAMIC TRIAL                    *}
{*                                                           *}
{*          Can be run on multiple segments                  *}
{*          simultaneously                                   *}
{*========================================================= *}


MACRO Patch4(M1,M2,M3,M4)

{*Optional Points*}
OptionalPoints(M1,M2,M3,M4)

{* Create replacement marker from static*}
DummySeg1 = [M4,M4-M2,M4-M1,xyz]
IF $STATIC==1
        M3#P=M3/DummySeg1
        PARAM(M3#P)
ENDIF
M3 = M3 ? M3#P*DummySeg1
OUTPUT(M3)

{* Create replacement marker from static *}
DummySeg2 = [M3,M3-M2,M3-M1,xyz]
IF $STATIC==1
        M4#P=M4/DummySeg2
        PARAM(M4#P)
ENDIF
M4 = M4 ? M4#P*DummySeg2
OUTPUT(M4)

{* Create replacement MT5 marker from static *}
DummySeg3 = [M3,M3-M2,M3-M4,xyz]
IF $STATIC==1
        M1#P=M1/DummySeg3
        PARAM(M1#P)
ENDIF
M1 = M1 ? M1#P*DummySeg3
```

OUTPUT(M1)

{* Create replacement marker from static *}
DummySeg4 = [M3,M3-M1,M3-M4,xyz]
IF $STATIC==1
        M2#P=M2/DummySeg4
        PARAM(M2#P)
ENDIF
M2 = M2 ? M2#P*DummySeg4
OUTPUT(M2)

ENDMACRO

{*Call the patch!*}
{*INPUT 4 Markers for the segment you want to patch*}

{*HAND segment*}
Patch4(RH1,RH6,RH2,RH5)

Appendix 14 -Replace 4

```
{*VICON BodyLanguage (tm) model                                    *}
{*===============================================                   *}
{*Title:    Replace 4                                               *}
{*Author:   MARRC                                                   *}
{*Date:     Nov 2006                                                *}
{*Purpose:   Patch missing markers on a segment                     *}
{*Comments:   Designed to patch missing markers on                  *}
{*          a segment of four.  All 4 Markers                        *}
{*          must be present at some point                            *}
{*          throughout the trial for the                             *}
{*          fourth to be recreated.                                  *}
{*                                                                   *}
{*          WARNING - some consideration                             *}
{*          should be given to the how "well"                        *}
{*          the missing marker is being                              *}
{*          reconstructed NB if it missing for                       *}
{*          most of the trial it would be better                     *}
{*          to patch from the static                                 *}

{*                                                                   *}
{*          Can be run on multiple segments                          *}
{*          simultaneously                                           *}
{*===============================================                   *}


MACRO REPLACE4(p1,p2,p3,p4)
{*Replaces any point missing from set of four fixed in a segment*}
        s234 = [p3,p2-p3,p3-p4]
        p1V = Average(p1/s234)*s234
        s341 = [p4,p3-p4,p4-p1]
        p2V = Average(p2/s341)*s341
        s412 = [p1,p4-p1,p1-p2]
        p3V = Average(p3/s412)*s412
        s123 = [p2,p1-p2,p2-p3]
        p4V = Average(p4/s123)*s123
        {* Now only replaces if original is missing  11-99 *}
        p1 = p1 ? p1V
        p2 = p2 ? p2V
        p3 = p3 ? p3V
        p4 = p4 ? p4V
        OUTPUT(p1,p2,p3,p4)
ENDMACRO


{* Enter required points here *}


{*HAND segment*}
```

REPLACE4(RH1,RH6,RH5,RH2)

REPLACE4(RH3,RH6,RH5,RH2)

REPLACE4(RH1,RH6,RH5,RH4)

{*REPLACE4(RH6,RH4,RH2,RH1)*}

REPLACE4(RH3,RH4,RH6,RH1)

REPLACE4(RH2,RH4,RH5,RH6)

{*SPARE*}
{*REPLACE4(P1,P2,P3,P4)*}

Appendix 15 -Missing data model

{*VICON BodyLanguage (tm) model*}

{*== *}
{*EDITED JAN 08 J Bevins ref L Lester. Modified from: *}
{*issued: January 2002   *}
{*Model RHand.MOD   TO ID FRAMES WITH MISSING DATA POINTS*}
{*   It is intended that this code is run prior to   *}
{*   the gap filling that will be required before we   *}
{*   filter the data*}
{*   *}
{*== *}

{*Start of macro section*}
{*======================*}

macro MISSINGDATA(MarkerID)
OptionalPoints(MarkerID)
IF
        EXIST(MarkerID)
        MarkerID#_Miss = {0,0,0}
ELSE
        MarkerID#_Miss ={1,1,1}
ENDIF
Output(MarkerID#_Miss)
endmacro

{*======================*}
{*End of macro section*}

{*Define optional marker points*}
OptionalPoints(RH1,RH2,RH3,RH4,RH5,RH6,RFA1,RWRA,RWRB,RFA2,RTPP2,RTH2,RTH3)
OptionalPoints(RIF1,RIF2,RIF3,RTF1,RTF2,RTF3,RRF1,RRF2,RRF3,RRF4,RPF1,RPF2,RPF3)
{*MORE OPTIONAL POINTS*}
OptionalPoints(RTMC1,RTMC2,RH3,RHNDV1,RH4,RHNDV2,RH5,RH6,RTMC1,RTMC2
,RTPP1,RTPP2
,RTDP1,RTDP2
,RIPP1,RIPP2
,RIMP1,RIMP2
,RIDP1,RIDP2
,RMPP1,RMPP2
,RMMP1,RMMP2
,RMDP1,RMDP2
,RRPP2,RRPP1
,RRMP2,RRMP1
,RRDP2,RRDP1
,RLPP1,RLPP2

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,RLMP1,RLMP2
,RLDP1,RLDP2)

MISSINGDATA(RFA1)
MISSINGDATA(RWRA)
MISSINGDATA(RWRB)
MISSINGDATA(RFA2)
MISSINGDATA(RH1)
MISSINGDATA(RTMC1)
MISSINGDATA(RTMC2)
MISSINGDATA(RTPP1)
MISSINGDATA(RTPP2)
MISSINGDATA(RTDP1)
MISSINGDATA(RTDP2)
MISSINGDATA(RH2)
MISSINGDATA(RIPP1)
MISSINGDATA(RIPP2)
MISSINGDATA(RIPPL)
MISSINGDATA(RIMP1)
MISSINGDATA(RIMP2)
MISSINGDATA(RIDP1)
MISSINGDATA(RIDP2)
MISSINGDATA(RH3)
MISSINGDATA(RMPP1)
MISSINGDATA(RMPP2)
MISSINGDATA(RMPPL)
MISSINGDATA(RMMP1)
MISSINGDATA(RMMP2)
MISSINGDATA(RMDP1)
MISSINGDATA(RMDP2)
MISSINGDATA(RH4)
MISSINGDATA(RRPP1)
MISSINGDATA(RRPP2)
MISSINGDATA(RRPPL)
MISSINGDATA(RRMP1)
MISSINGDATA(RRMP2)
MISSINGDATA(RRDP1)
MISSINGDATA(RRDP2)
MISSINGDATA(RH5)
MISSINGDATA(RLPP1)
MISSINGDATA(RLPP2)
MISSINGDATA(RLPPL)
MISSINGDATA(RLMP1)
MISSINGDATA(RLMP2)
MISSINGDATA(RLDP1)
MISSINGDATA(RLDP2)
MISSINGDATA(RH6)

Appendix 16 -2 markers per phalanx model

{*VICON BodyLanguage (tm) model*}


```
{*============================================================    *}
{*EDITED JAN 08 J Bevins ref L Lester. Modified from:              *}
{*issued: January 2002                                            *}
{*Model RHand.MOD                                                 *}
{*Use only with BodyBuilder V. 3.53 or later                      *}
{*Use only with RHand.MP parameters and RHand.MKR                 *}
{*                                                                *}
{* Model has been modified from the Vicon original                *}
{* To change the selection of markers that now define             *}
{* the finger vectors.  This is based on a new marker             *}
{* set.                                                           *}
{* Model now places 2 markers on each phalanx                     *}
{* And uses these to define the phalanx vectors                   *}
{*                                                                *}
{* NB the marker set also has provision for a side                *}
{* marker on the Proximal phalanx to allow a calc                 *}
{* of MCP rotation NB NOT YET IMPLEMENTED IN MODEL CODE           *}
{*                                                                *}
{*                                                                *}
{*============================================================    *}
```


{*This file is supplied to illustrate the normal operation of BodyLanguage.
Vicon Motion Systems accept no responsibility for its correct operation*}

{*Start of macro section*}
{*=====================*}

```
macro NORMALISE(Vec)
{* Normalises the vector Vec *}
len = 1(Vec)*1(Vec)+2(Vec)*2(Vec)+3(Vec)*3(Vec)
len = sqrt(len)
Vec = {1(Vec)/len,2(Vec)/len,3(Vec)/len}
endmacro

macro CROSSPROD(Vec1,Vec2,Result)
tmp =
{2(Vec1)*3(Vec2)-3(Vec1)*2(Vec2),3(Vec1)*1(Vec2)-1(Vec1)*3(Vec2),1(Vec1)*2(Vec2)-2
(Vec1)*1(Vec2)}
Result = tmp
endmacro

macro PROJECTION(line,segment,joint)
```
{* Calculates flexion/extension and abduction/adduction angles using technique of:
Cheng P.L., Pearcy M. (1998) A 3D Definition for the Flexion/Extension and
Abduction/Adduction Angles.

```
%line=(line+0(segment))/segment
output(%line)
RotY=acos(SQRT((1(%line)*1(%line))+(2(%line)*2(%line))))
RotZ=acos(SQRT((1(%line)*1(%line))+(3(%line)*3(%line))))
If 3(%line) > 0 Then RotY=-RotY Else RotY=RotY EndIf
If 2(%line) > 0 Then RotZ=RotZ Else RotZ=-RotZ EndIf
joint#ProjAngles=<0,RotY,RotZ>
output(joint#ProjAngles)

{*Alternative calculations using 'atan' and 'atan2' functions

RotY2=-atan(3(%line)/1(%line))
RotZ2=atan(2(%line)/1(%line))
joint#ProjAngles2=<0,RotY2,RotZ2>
output(joint#ProjAngles2)

RotY3=-atan2(3(%line),1(%line))
RotZ3=atan2(2(%line),1(%line))
joint#ProjAngles3=<0,RotY3,RotZ3>
output(joint#ProjAngles3)              *}

endmacro

macro SEGVIS(Segment)
ORIGIN#Segment=0(Segment)
XAXIS#Segment=0(Segment)+(1(Segment)*100)
YAXIS#Segment=0(Segment)+(2(Segment)*100)
ZAXIS#Segment=0(Segment)+(3(Segment)*100)
output(ORIGIN#Segment,XAXIS#Segment,YAXIS#Segment,ZAXIS#Segment)
endmacro

{*=====================*}
{*End of macro section*}

{*Define optional marker points*}
OptionalPoints(RH1,RH2,RH3,RH4,RH5,RH6,RFA1,RWRA,RWRB,RFA2,RTPP2,RTH2,R
TH3)
OptionalPoints(RIF1,RIF2,RIF3,RTF1,RTF2,RTF3,RRF1,RRF2,RRF3,RRF4,RPF1,RPF2,R
PF3)
{*MORE OPTIONAL POINTS*}
OptionalPoints(RTMC1,RTMC2,RH3,RHNDV1,RH4,RHNDV2,RH5,RH6,RTMC1,RTMC2
,RTPP1,RTPP2
,RTDP1,RTDP2
,RIPP1,RIPP2
,RIMP1,RIMP2
,RIDP1,RIDP2
,RMPP1,RMPP2
,RMMP1,RMMP2
,RMDP1,RMDP2
```

```
,RRPP2,RRPP1
,RRMP2,RRMP1
,RRDP2,RRDP1
,RLPP1,RLPP2
,RLMP1,RLMP2
,RLDP1,RLDP2)

{*Define Virtual Markers*}
RHNDV1=(RH1+RH6)/2
RHNDV2=(RHNDV1+RH6)/2

{*Define Hand Segment Frame*}
RHand1=[RHNDV1,RH3-RHNDV1,RH6-RH1,xzy]
SEGVIS(RHand1)

{*Define Segment Vectors*}
RightHandAxis1=RH2-RH1
RightHandAxis2=RH3-RHNDV1
RightHandAxis3=RH4-RHNDV2
RightHandAxis4=RH5-RH6

RightThumb1=RTMC1-RTMC2
RightThumb2=RTPP1-RTPP2
RightThumb3=RTDP1-RTDP2

RightIndexFinger1=RIPP2-RIPP1
RightIndexFinger2=RIMP2-RIMP1
RightIndexFinger3=RIDP2-RIDP1

RightMiddleFinger1=RMPP2-RMPP1
RightMiddleFinger2=RMMP2-RMMP1
RightMiddleFinger3=RMDP2-RMDP1

RightRingFinger1=RRPP2-RRPP1
RightRingFinger2=RRMP2-RRMP1
RightRingFinger3=RRDP2-RRDP1

RightLittle1=RLPP2-RLPP1
RightLittle2=RLMP2-RLMP1
RightLittle3=RLDP2-RLDP1


{*Normalise Segment Vectors*}
NORMALISE(RightHandAxis1)
NORMALISE(RightHandAxis2)
NORMALISE(RightHandAxis3)
NORMALISE(RightHandAxis4)
NORMALISE(RightThumb1)
NORMALISE(RightThumb2)
NORMALISE(RightThumb3)
NORMALISE(RightIndexFinger1)
NORMALISE(RightIndexFinger2)
```

NORMALISE(RightIndexFinger3)
NORMALISE(RightMiddleFinger1)
NORMALISE(RightMiddleFinger2)
NORMALISE(RightMiddleFinger3)
NORMALISE(RightRingFinger1)
NORMALISE(RightRingFinger2)
NORMALISE(RightRingFinger3)
NORMALISE(RightLittle1)
NORMALISE(RightLittle2)
NORMALISE(RightLittle3)

{*Calculate Cross-Products for Joint Angles*}
CROSSPROD(RightThumb1,RightHandAxis1,RTJ1Prod)
CROSSPROD(RightThumb2,RightThumb1,RTJ2Prod)
CROSSPROD(RightThumb3,RightThumb2,RTJ3Prod)
CROSSPROD(RightIndexFinger1,RightHandAxis1,RIFJ1Prod)
CROSSPROD(RightIndexFinger2,RightIndexFinger1,RIFJ2Prod)
CROSSPROD(RightIndexFinger3,RightIndexFinger2,RIFJ3Prod)
CROSSPROD(RightMiddleFinger1,RightHandAxis2,RTFJ1Prod)
CROSSPROD(RightMiddleFinger2,RightMiddleFinger1,RTFJ2Prod)
CROSSPROD(RightMiddleFinger3,RightMiddleFinger2,RTFJ3Prod)
CROSSPROD(RightRingFinger1,RightHandAxis3,RRFJ1Prod)
CROSSPROD(RightRingFinger2,RightRingFinger1,RRFJ2Prod)
CROSSPROD(RightRingFinger3,RightRingFinger2,RRFJ3Prod)
CROSSPROD(RightLittle1,RightHandAxis4,RPFJ1Prod)
CROSSPROD(RightLittle2,RightLittle1,RPFJ2Prod)
CROSSPROD(RightLittle3,RightLittle2,RPFJ3Prod)

{*Calculate First Joint Projected Angles*}
PROJECTION(RightThumb1,RHand1,RightThumbJ1)
PROJECTION(RightIndexFinger1,RHand1,RightIndexFingerJ1)
PROJECTION(RightMiddleFinger1,RHand1,RightMiddleFingerJ1)
PROJECTION(RightRingFinger1,RHand1,RightRingFingerJ1)
PROJECTION(RightLittle1,RHand1,RightLittleJ1)

{*Calculate Finger Joint Absolute Angles*}
RightThumbJ1AbsAngles=<asin(DIST(RTJ1Prod, {0,0,0})),0,0>
RightThumbJ2AbsAngles=<asin(DIST(RTJ2Prod, {0,0,0})),0,0>
RightThumbJ3AbsAngles=<asin(DIST(RTJ3Prod, {0,0,0})),0,0>
RightIndexFingerJ1AbsAngles=<asin(DIST(RIFJ1Prod, {0,0,0})),0,0>
RightIndexFingerJ2AbsAngles=<asin(DIST(RIFJ2Prod, {0,0,0})),0,0>
RightIndexFingerJ3AbsAngles=<asin(DIST(RIFJ3Prod, {0,0,0})),0,0>
RightMiddleFingerJ1AbsAngles=<asin(DIST(RTFJ1Prod, {0,0,0})),0,0>
RightMiddleFingerJ2AbsAngles=<asin(DIST(RTFJ2Prod, {0,0,0})),0,0>
RightMiddleFingerJ3AbsAngles=<asin(DIST(RTFJ3Prod, {0,0,0})),0,0>
RightRingFingerJ1AbsAngles=<asin(DIST(RRFJ1Prod, {0,0,0})),0,0>
RightRingFingerJ2AbsAngles=<asin(DIST(RRFJ2Prod, {0,0,0})),0,0>
RightRingFingerJ3AbsAngles=<asin(DIST(RRFJ3Prod, {0,0,0})),0,0>
RightLittleJ1AbsAngles=<asin(DIST(RPFJ1Prod, {0,0,0})),0,0>
RightLittleJ2AbsAngles=<asin(DIST(RPFJ2Prod, {0,0,0})),0,0>
RightLittleJ3AbsAngles=<asin(DIST(RPFJ3Prod, {0,0,0})),0,0>

{*Output Joint Angles*}

{*Sequence to remove data points filled*}
{*INDEX FINGER*}
IF   (RH1_Miss(1)==1 OR RH2_Miss(1)==1 OR RIPP1_Miss(1)==1 OR
RIPP2_Miss(1)==1)
        RightIndexFingerJ1AbsAngles = <-50,-50,-50>
ELSE
        RightIndexFingerJ1AbsAngles = RightIndexFingerJ1AbsAngles
ENDIF

IF   (RIPP1_Miss(1)==1 OR RIPP2_Miss(1)==1 OR RIMP1_Miss(1)==1 OR
RIMP2_Miss(1)==1)
        RightIndexFingerJ2AbsAngles = <-50,-50,-50>
ELSE
        RightIndexFingerJ2AbsAngles = RightIndexFingerJ2AbsAngles
ENDIF

IF   (RIMP1_Miss(1)==1 OR RIMP2_Miss(1)==1 OR RIDP1_Miss(1)==1 OR
RIDP2_Miss(1)==1)
        RightIndexFingerJ3AbsAngles = <-50,-50,-50>
ELSE
        RightIndexFingerJ3AbsAngles = RightIndexFingerJ3AbsAngles
ENDIF

{*SECTION TO ACCOUNT FOR THE PROJECTED ANGLE CALCULATIONS*}
{*MARKERS to exclude = RH3 RH6 RH1 RIPP1 & RIPP2*}

IF (RH3_Miss(1) ==1 OR RH6_Miss(1) ==1 OR RH1_Miss(1) ==1 OR RIPP1_Miss(1)
==1 OR RIPP2_Miss(1) ==1)
RightIndexFingerJ1ProjAngles = <-50,-50,-50>

ELSE
RightIndexFingerJ1ProjAngles = RightIndexFingerJ1ProjAngles
ENDIF
{*END INDEX FINGER*}

{*MIDDLE FINGER*}
IF   (RH1_Miss(1)==1 OR RH2_Miss(1)==1 OR RH3_Miss(1)==1 OR
RMPP1_Miss(1)==1 OR RMPP2_Miss(1)==1)
        RightMiddleFingerJ1AbsAngles = <-50,-50,-50>
ELSE
        RightMiddleFingerJ1AbsAngles = RightMiddleFingerJ1AbsAngles
ENDIF

IF   (RMPP1_Miss(1)==1 OR RMPP2_Miss(1)==1 OR RMMP1_Miss(1)==1 OR
RMMP2_Miss(1)==1)
        RightMiddleFingerJ2AbsAngles = <-50,-50,-50>
ELSE
        RightMiddleFingerJ2AbsAngles = RightMiddleFingerJ2AbsAngles

ENDIF

IF   (RMMP1_Miss(1)==1 OR RMMP2_Miss(1)==1 OR RMDP1_Miss(1)==1 OR
RMDP2_Miss(1)==1)
        RightMiddleFingerJ3AbsAngles = <-50,-50,-50>
ELSE
        RightMiddleFingerJ3AbsAngles = RightMiddleFingerJ3AbsAngles
ENDIF

{*SECTION TO ACCOUNT FOR THE PROJECTED ANGLE CALCULATIONS*}
{*MARKERS to exclude = RH3 RH6 RH1 RMPP1 & RMPP2*}

IF (RH3_Miss(1) ==1 OR RH6_Miss(1) ==1 OR RH1_Miss(1) ==1 OR RMPP1_Miss(1)
==1 OR RMPP2_Miss(1) ==1)
RightMiddleFingerJ1ProjAngles = <-50,-50,-50>

ELSE
RightMiddleFingerJ1ProjAngles = RightMiddleFingerJ1ProjAngles
ENDIF
{*END MIDDLE FINGER*}

{*RING FINGER*}
IF   (RH1_Miss(1)==1 OR RH4_Miss(1)==1 OR RH6_Miss(1)==1 OR RH3_Miss(1)==1
OR RRPP1_Miss(1)==1 OR RRPP2_Miss(1)==1)
        RightRingFingerJ1AbsAngles = <-50,-50,-50>
ELSE
        RightRingFingerJ1AbsAngles = RightRingFingerJ1AbsAngles
ENDIF

IF   (RRPP1_Miss(1)==1 OR RRPP2_Miss(1)==1 OR RRMP1_Miss(1)==1 OR
RRMP2_Miss(1)==1)
        RightRingFingerJ2AbsAngles = <-50,-50,-50>
ELSE
        RightRingFingerJ2AbsAngles = RightRingFingerJ2AbsAngles
ENDIF

IF   (RRMP1_Miss(1)==1 OR RRMP2_Miss(1)==1 OR RRDP1_Miss(1)==1 OR
RRDP2_Miss(1)==1)
        RightRingFingerJ3AbsAngles = <-50,-50,-50>
ELSE
        RightRingFingerJ3AbsAngles = RightRingFingerJ3AbsAngles
ENDIF

{*SECTION TO ACCOUNT FOR THE PROJECTED ANGLE CALCULATIONS*}
{*MARKERS to exclude = RH3 RH6 RH1 RRPP1 & RRPP2*}

IF (RH3_Miss(1) ==1 OR RH6_Miss(1) ==1 OR RH1_Miss(1) ==1 OR RH4_Miss(1) ==1
OR RRPP1_Miss(1) ==1 OR RRPP2_Miss(1) ==1)
RightRingFingerJ1ProjAngles = <-50,-50,-50>

ELSE

RightRingFingerJ1ProjAngles = RightRingFingerJ1ProjAngles
ENDIF
{*END RING FINGER*}

{*LITTLE FINGER*}
IF   (RH1_Miss(1)==1 OR RH6_Miss(1)==1 OR RLPP1_Miss(1)==1 OR
RLPP2_Miss(1)==1)
        RightLittleJ1AbsAngles = <-50,-50,-50>
ELSE
        RightLittleJ1AbsAngles = RightLittleJ1AbsAngles
ENDIF

IF   (RLPP1_Miss(1)==1 OR RLPP2_Miss(1)==1 OR RLMP1_Miss(1)==1 OR
RLMP2_Miss(1)==1)
        RightLittleJ2AbsAngles = <-50,-50,-50>
ELSE
        RightLittleJ2AbsAngles = RightLittleJ2AbsAngles
ENDIF

IF   (RLMP1_Miss(1)==1 OR RLMP2_Miss(1)==1 OR RLDP1_Miss(1)==1 OR
RLDP2_Miss(1)==1)
        RightLittleJ3AbsAngles = <-50,-50,-50>
ELSE
        RightLittleJ3AbsAngles = RightLittleJ3AbsAngles
ENDIF

{*SECTION TO ACCOUNT FOR THE PROJECTED ANGLE CALCULATIONS*}
{*MARKERS to exclude = RH3 RH6 RH1 RLPP1 & RLPP2*}

IF (RH3_Miss(1) ==1 OR RH6_Miss(1) ==1 OR RH1_Miss(1) ==1 OR RH4_Miss(1) ==1
OR RLPP1_Miss(1) ==1 OR RLPP2_Miss(1) ==1)
RightLittleJ1ProjAngles = <-50,-50,-50>

ELSE
RightLittleJ1ProjAngles = RightLittleJ1ProjAngles
ENDIF
{*END LITTLE FINGER*}
{*END Sequence to remove "False" data points*}

output(RightThumbJ1AbsAngles,RightThumbJ2AbsAngles,RightThumbJ3AbsAngles)
output(RightIndexFingerJ1AbsAngles,RightIndexFingerJ2AbsAngles,RightIndexFingerJ3Ab
sAngles)
output(RightMiddleFingerJ1AbsAngles,RightMiddleFingerJ2AbsAngles,RightMiddleFingerJ
3AbsAngles)
output(RightRingFingerJ1AbsAngles,RightRingFingerJ2AbsAngles,RightRingFingerJ3AbsA
ngles)
output(RightLittleJ1AbsAngles,RightLittleJ2AbsAngles,RightLittleJ3AbsAngles)

{*Output Virtual Markers*}

output(RHNDV1,RHNDV2)

{*ADDITION CODE TO CALCULATE MCP ROTATION _ RIGHT INDEX ONLY*}

{*Define RIPP Segment - NB No axis seq is defined*}

{*RIPP =[RIPP1, RIPP2-RIPP1,RIPP1-RIPPL,XZY]

{*AXIS VIS*}
{*Define a VISUAL COOR Frame of the LCS for the RIPP segment*}
RIPPO = RIPP1
RIPPX = RIPPO+RIPP(1)*200
RIPPY = RIPPO+RIPP(2)*200
RIPPZ = RIPPO+RIPP(3)*200
OUTPUT(RIPPO,RIPPX,RIPPY,RIPPZ)*}

{*Define R "hand" segment*}

RHAND =[RH1,RH2-RH1,RH4-RH1,XZY]

RHANDO = RH1
RHANDX = RHANDO+RHAND(1)*200
RHANDY = RHANDO+RHAND(2)*200
RHANDZ = RHANDO+RHAND(3)*200
OUTPUT(RHANDO,RHANDX,RHANDY,RHANDZ)

{*Calculate the Angles*}
{*Child first*}
{*Seq to give Flex/Ext X, Add/Abb Y and Rot Z*}
{*
RightIndexMCPAngles = <RIPP,RHAND,YZX>
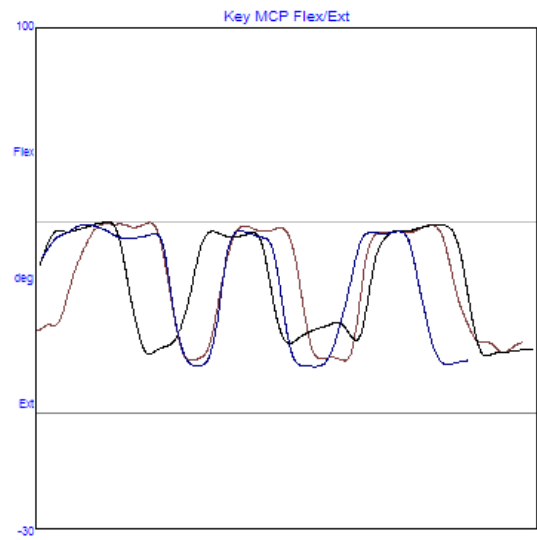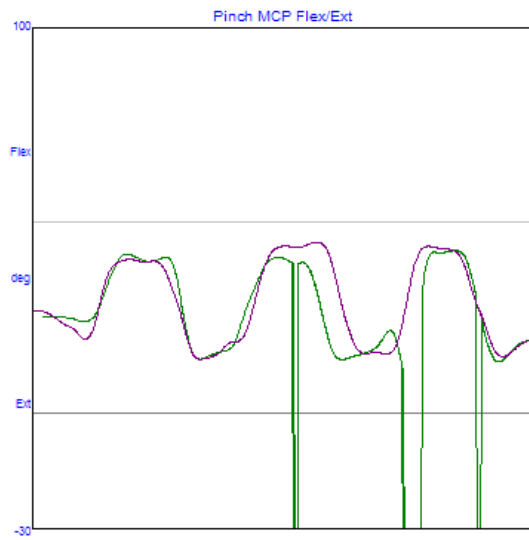*}
{*Output the calculated angles*}
{*OUTPUT(RightIndexMCPAngles)*}

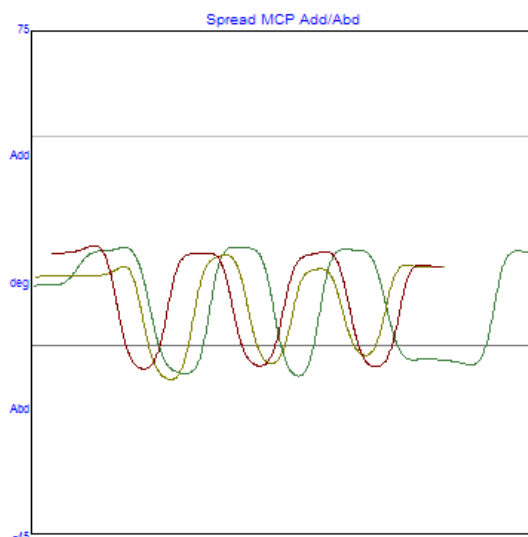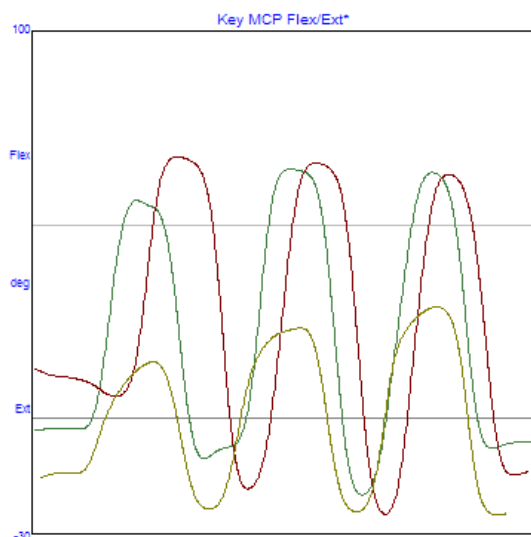**Appendix 17a - Example data plots from YN01 for all movements**
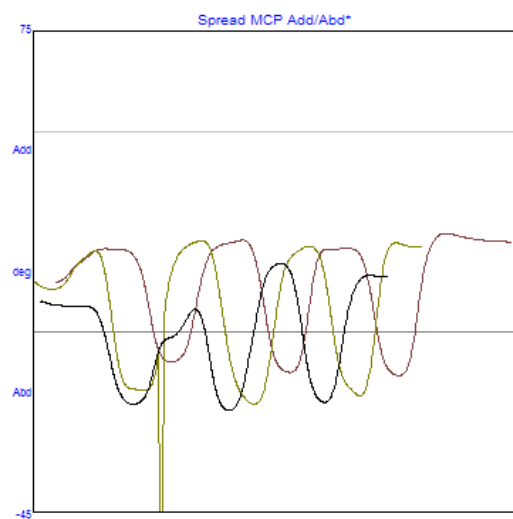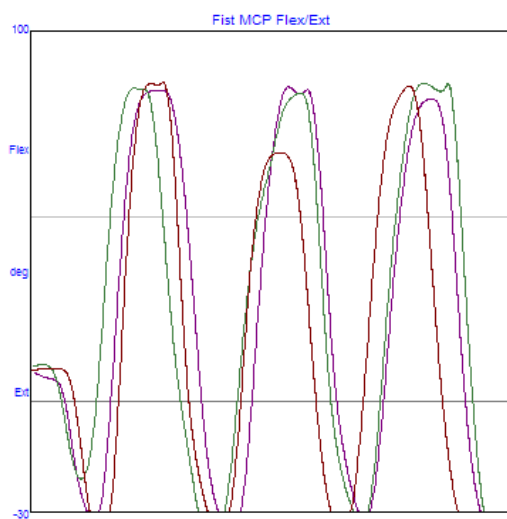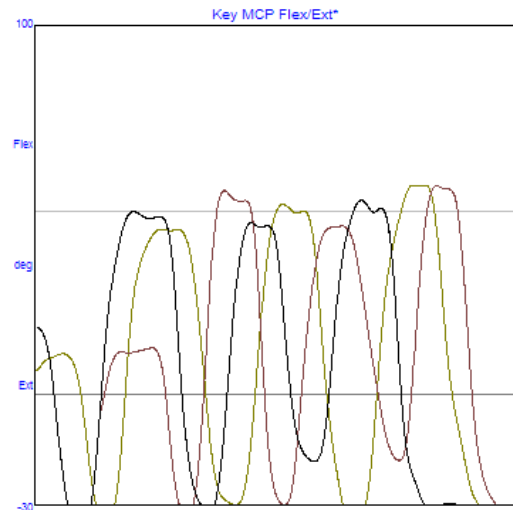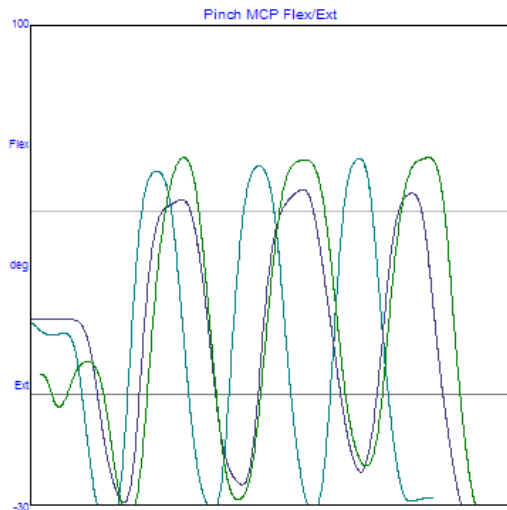
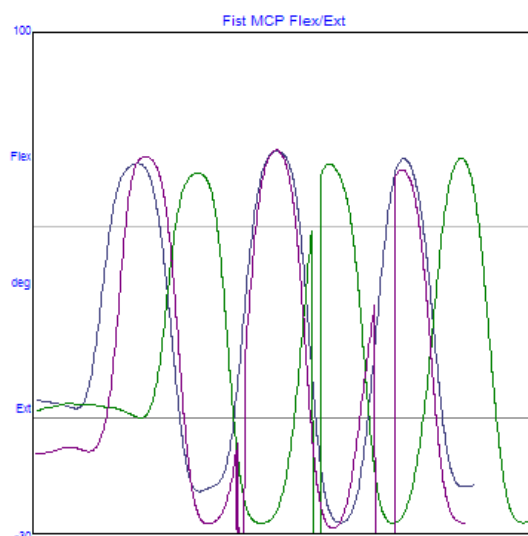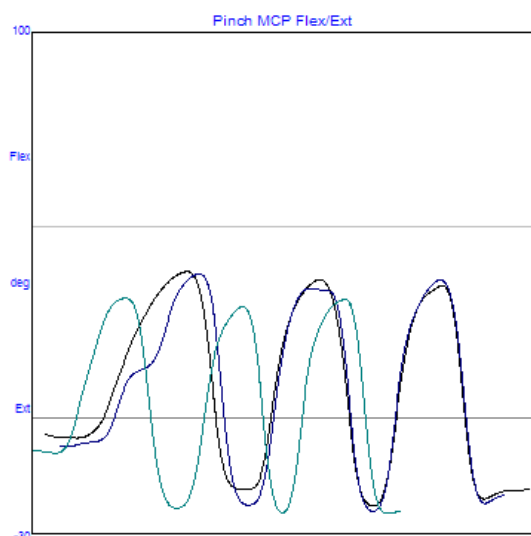Appendix 17b – Example data plots for EN01 for all movements
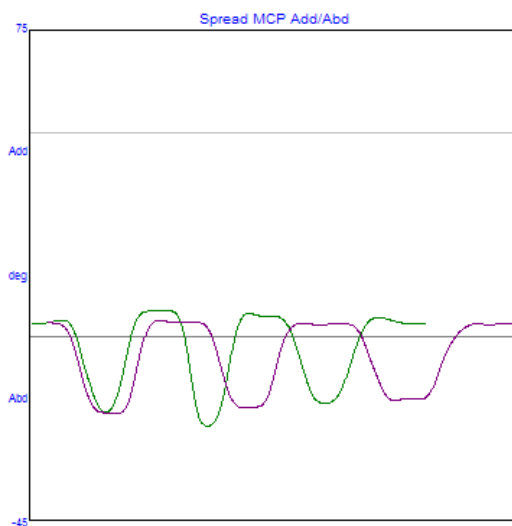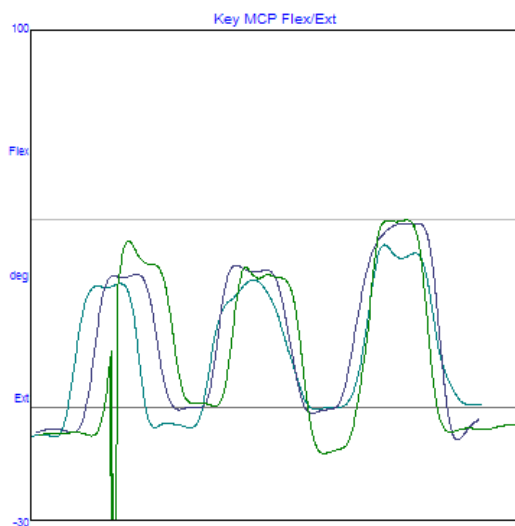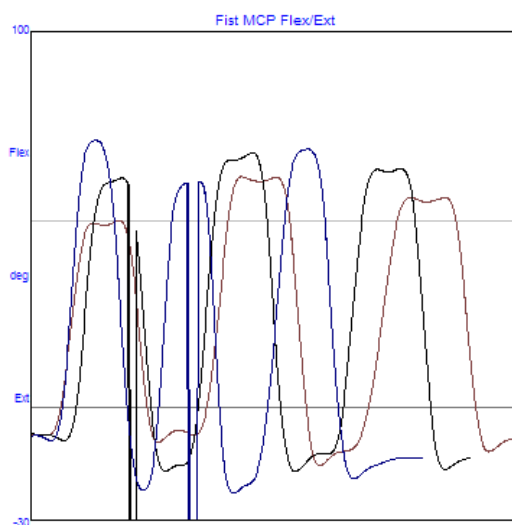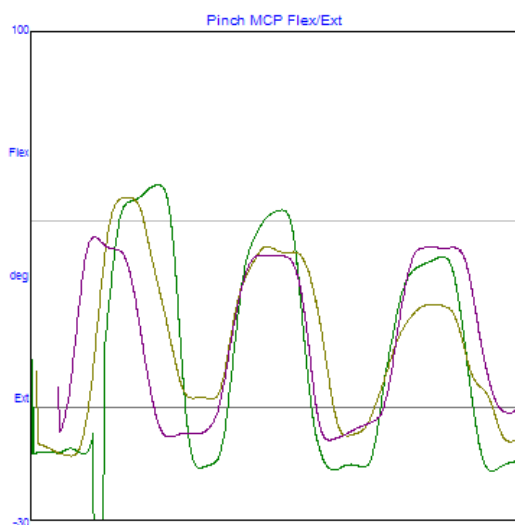
Appendix 17c – Example data plots for MCP01 for all movements

Pinch MCP Flex/Ext

Key MCP Flex/Ext

Fist MCP Flex/Ext

Spread MCP Add/Abd
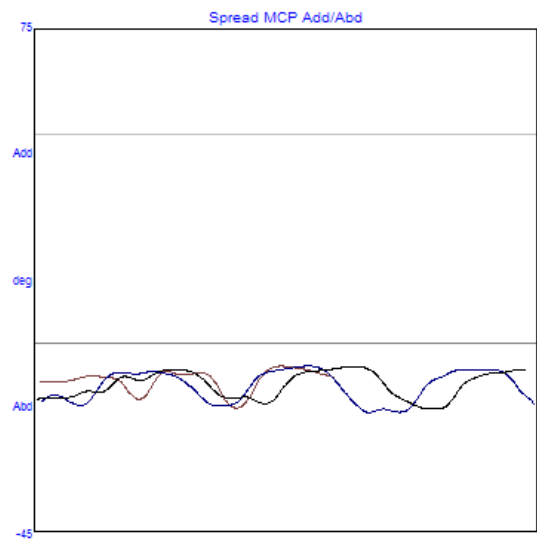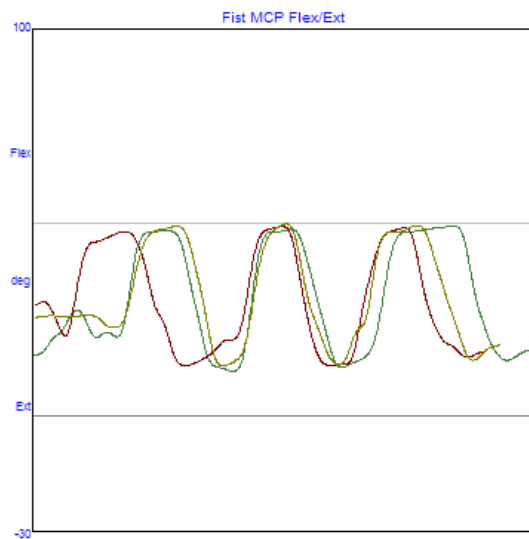
Appendix 17d – Example data plots for RA01 for all movements

Appendix 18 – Results of all subjects

8. REFERENCES

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